PAINFUL STIFF SHOULDER
(FROZEN SHOULDER)

AND

SOFT TISSUE RHEUMATISM

IN THE UPPER LIMB

M.D. THESIS - DR ALLAN IVAN BINDER

UNIVERSITY OF CAPE TOWN

1985

My interest in soft tissue (non-articular) rheumatism was stimulated by my appointment as an Arthritis and Rheumatism Council (ARC) research fellow and honorary senior registrar in rheumatology attached to Dr Brian Hazleman's unit in Cambridge, England. Soft tissue lesions are common in clinical practice and an interest in the frozen shoulder had been present in Cambridge for many years.

The paucity of consistent data on the frozen shoulder led me to examine the pathogenesis, natural history and the response to treatment. I also sought to ascertain the value of clinical and laboratory features in the differentiation of sub-groups presenting with a painful stiff shoulder. As the geniune frozen shoulder is relatively uncommon, I expanded the study to include rotator cuff tendinitis of the shoulder and lateral epicondylitis of the elbow which it seemed might share a common pathology with the painful stiff shoulder.

INDEX

PART I

GENERAL CONCEPTS AND A REVIEW OF THE LITERATURE

CHAPTER 1

SOFT TISSUE RHEUMATISM

Lesions of tendons and their sheaths, fasciae, bursae, ligaments, joint capsules and the teno-periosteal junction (the enthesis) cause much morbidity. As biopsy and surgery are rarely employed for diagnosis and treatment, histological data is scanty and their pathologies are poorly understood.

While these lesions may occur in association with overt systemic disease as for example, inflammatory arthritis or infection, most occur in the absence of other features and are included under the term of 'Soft Tissue (Non-articular) Rheumatism'.

CLASSIFICATION

There is no universally accepted classification. For practical purpose the conditions can be divided into (A) generalised (Table 1:1) and (B) localised soft tissue lesions. Generalised soft tissue lesions will not be considered further.

The major structures involved in the localised soft tissue lesions (Table 1:2) and many of the commoner conditions (Figure 1:1) are shown. Some of the soft tissue lesions affecting the shoulder and elbow have been chosen for detailed study.

EPIDEMIOLOGY

The lack of an accepted classification and the differences in nomenclature of the soft tissue lesions has resulted in a paucity of information regarding the epidemiological and economic importance of these conditions. Wood et al (288) used the International Classification of Diseases, eighth revision (ICD, WHO 1967) (277) to assess the incidence of soft tissue lesions in the community and noted that over one third of the rheumatological consultations in general practice were for soft tissue lesions, making over 4 million visits in Britain each year. A new ICD introduced in 1977 (278) has not yet been used to study these lesions. The association between occupation and specific soft tissue lesions (16, 116, 212) may also produce regional variation in the incidence of some of these conditions.

ASSOCIATION OF SOFT TISSUE LESIONS

While many lesions result from activities related to specific occupations, sport or hobbies, their occurence is often unexplained and may result from individual susceptibility. This predisposition in supported by the documented associations of a variety of these lesions in the same patients. Rotator cuff tendinitis, frozen shoulder, tennis and golfer's elbow, carpal tunnel syndrome, Dupuytren's contracture and de Quervain's tenosynovitis have been found to occur more frequently in the same patients (22, 61, 102, 127, 195, 204, 216).

Niepal and Sitaj (203) proposed enthesopathy as a unifying concept for many of the soft tissue lesions including those affecting the shoulder and elbow. They did not however, offer any explanation for the susceptibility of the enthesis in these patients. The 'fibrositis syndrome' (252) which is characterised by musculoskeletal ache and tender sites at the entheses has been suggested ás a pain amplification syndrome. Its pathogenesis is unexplained and the association between this syndrome and a variety of soft tissue lesions in the same patient is uncertain. Cervical spine disease particularly cervical spondylosis can result in severe pain in the shoulder and elbow (102, 247) and may be confused with localised soft tissue lesions in the upper limb. Murray-Leslie and Wright (195) felt that the symptoms can be indistinguishable. A hereditary predisposition was suggested by Bulgen et al (30) who reported an increased incidence of HLA-B27 antigens in painful stiff shoulder patients. However, this association has not been confirmed (206, 234, 243) or reported in other soft tissue lesions.

TABLE 1:1

GENERALISED SOFT TISSUE LESIONS

A. WITH EVIDENCE OF INFLAMMATION

i. Polymyalgia rheumatica and cranial arteritis
ii. Prodrome of inflammatory arthropathies and connective tissue diseases
iii. Viral and bacterial infections

B. WITHOUT INFLAMMATION

i. Hypothyroidism
ii. Drug related painful states associated with steroid withdrawal, chronic barbiturate abuse and the contraceptive pill
iii. Dyskinetic phase of Parkinson's disease
iv. Chronic Brucellosis, Bornholm's disease
v. Associated with malignancy e.g. myeloma, carcinoma
vi. Osteomalacia
vii. 'Fibrositis', 'lumbago'

C. ASSOCIATED WITH WEAKNESS

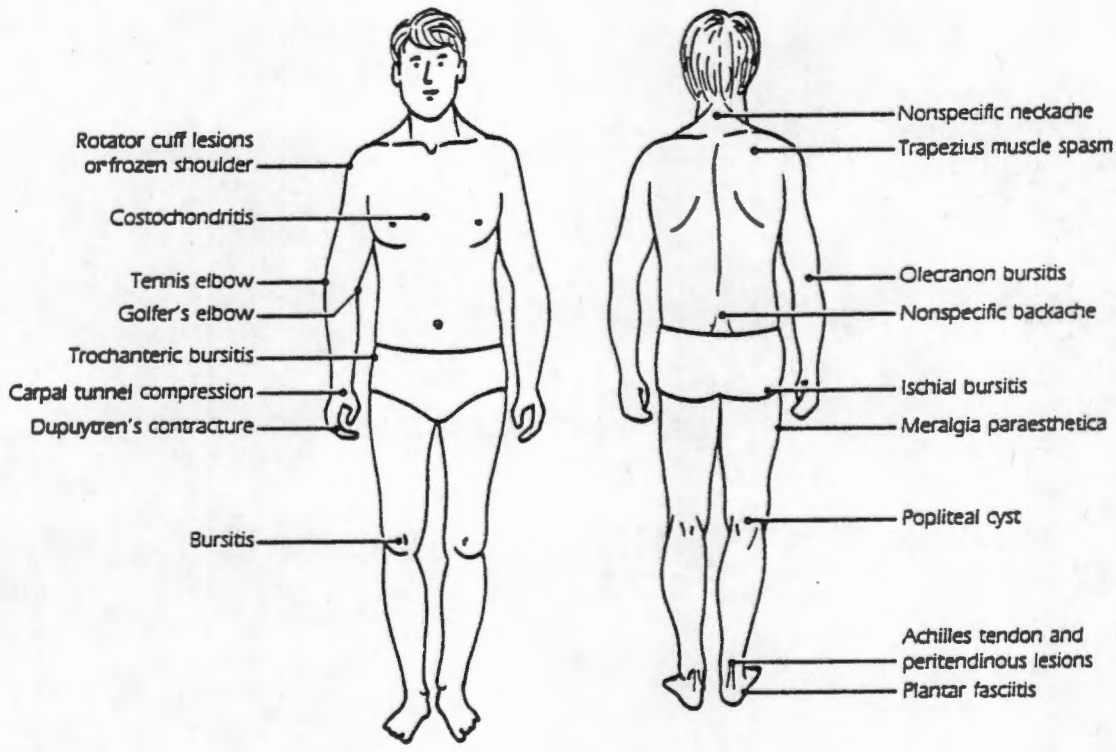
i. Prodrome of polymyositis or dermatomyositis
ii. Carcinomatous neuromyopathy (some forms)
iii. Hypokalemic states

D. 'PSYCHOGENIC' RHEUMATISM

TABLE 1:2

STRUCTURES AFFECTED AND ASSOCIATED SOFT TISSUE LESIONS

STRUCTURE	PATHOLOGY
Tendons	Rupture, degeneration, tendinitis
Tendon sheaths	Peritendinitis, tenosynovitis, ganglion
Bursae	Bursitis - acute or chronic
Tenoperiosteal junctions	Enthesopathy, apophysitis
Fasciae	Fasciitis, Dupuytren's contracture
Ligaments	Sprain, strain, tear



F I G U R E I : I. Common sites of soft tissue lesions.

CHAPTER 2

THE NORMAL TENDON, JOINT CAPSULE AND ROTATOR CUFF AND THE EFFECT OF AGING; FUNCTIONAL ANATOMY OF SHOULDER

THE NORMAL TENDON

EMBRYOLOGY: Tendons begin as mesenchymal condensations of fibroblasts which secrete parallel orientated collagen fibres (281). As these fibres increase in number, the cellularity of the structure reduces and the remaining fibroblast nuclei come to lie in parallel between the bundles of collagenous fibres. Tendons can usually be recognised in the embryo by the end of the second month.

The tendon develops independently of its muscle and is later linked by connective tissue which comes predominantly from the perimysium (108). This area remains vulnerable to rupture throughout childhood. Once muscle and tendon have been united, the force and the direction of stress affecting the unit, controls the further orientation of collagen fibres and the extent of its proliferation (230, 281). While most collagen fibres remain in parallel, some interweaving does occur (98) to provide additional stability to the structure. The wavy nature of the collagen bundles and the presence of scanty elastin fibres within the tendon (98, 107, 230), provide further longitudinal flexibility. Once the tendon has been fully developed, the remaining fibroblasts within the structure lose their ability to divide (107). If damage occurs, fibroblasts migrate from the surrounding loose connective tissue to the area of damage and are responsible for new collagen production and eventual tendon repair (29, 218).

HISTOLOGY: The parallel but wavy bundles of collagen fibres are grouped in fascicles separated from each other by small amounts of loose connective tissue (endotendineum) containing scanty blood vessels, nerves and elastin fibres. The fascicles are held together by loose connective tissue (peritendineum) which is continuous with the endotendineum. The fibrous sheath (epitendineum) merges with the surrounding loose connective tissue. A high elastin content in this region permits some tendon movement, but where greater movement is necessary, a double layered synovial-lined sheath is present. The cellular component of adult tendon is scanty with fibroblasts lying in parallel and between the collagen bundles. The lateral limits of these cells are indistinct. The nuclei are spindle shaped and

heterochromic with axes parallel to the collagen fibres. In cross-section, the nuclei are stellate shaped, lying between the collagen bundles. Occasional macrophages may also be identified.

ULTRASTRUCTURE: Electron microscopy of normal adult tendon (51) shows collagen fibrils ranging from 250 - 1400 angstrom which in cross-section are grouped in bundles separated by granular and filamentous precipitates. Longitudinal sections of the individual fibrils shows monotonously regular periodicity characteristic of the native type macro-molecular aggregated collagen. Scanty elastin fibres are also noted. The fibroblasts (tenocytes) appear to be metabolically active cells (136) with well developed rough endoplasmic reticulum, Golgi apparatus, mitochondria and large cytoplasmic ribosomes. Lysosomes and lipid vacuoles are also evident. The cell membranes show indentations caused by the surrounding collagen fibres.

HISTOCHEMISTRY: Tendons have traditionally been considered as metabolically inert structures (98, 214). Holloszy (1971) (126) reported very low levels of oxidative enzyme in rat tendon even after exercise. However, Tipton et al (1975) (264) showed experimentally that tendons were metabolically active and that the activity increased with exercise, although it remained 60 - 70% lower than the attached muscle. Heikkinen et al (1975) (114) demonstrated that chronic exercise could increase collagen turnover and selectively activate Kreb cycle enzymes in rat tendon. Suominen and Heikkinen (1975) (261) found similar effects in tendon biopsy specimens from older men engaged in a long standing exercise program. Jozsa et al (1979) (136) studied fresh human postmortem tendons and identified enzymes for oxidative and anaerobic metabolism which correlated with the ultrastructural evidence of the metabolic potential of adult tenocytes.

THE ENTHESIS: The point of attachment of tendons to bone (the enthesis) has long been recognised to be the site of maximum stress and an area which differed histologically from the rest of the tendon. Havers (1691) (112) and Kolliker (1853) (144) observed that some tendon fibres penetrated the cartilage and attached directly to bone. Sharpey and Ellis (1856) (244) stressed the importance of these perforating (Sharpey) fibres to the tensile strength of the enthesis.

Dolgu-Saburoff (1929) (70) described the 4 traditional zones of tendon, fibrocartilage, mineralised fibrocartilage and bone which constitute the enthesis. A distinct "blue-line" was attributed to the zone of mineralised

fibrocartilage. Cooper and Misol (1970) (51) confirmed the presence of these zones and added ultrastructural detail of this region. Smooth endoplasmic reticulum and intracellular lipids and lysosomes increased and the Golgi apparatus became more prominent. An electron-dense line (corresponding to the blue-line) was noted in the mineralised fibrocartilage zone with granular deposits becoming increasingly evident within the matrix. These deposits clumped and eventually merged with bone. This study suggested that the cells were both viable and metabolically active, but no histochemical studies have been performed to confirm this.

BLOOD SUPPLY TO THE TENDON: While early anatomists (119, 144) regarded the adult tendon as an avascular structure, Mayer (1916) (174) used an injection technique to demonstrate a vascular supply arising from muscle, bone and the surrounding connective tissues. Edwards (1946) (77) found a scanty superficial longitudinally orientated vascular network to sustain the tenocytes and Peacock (1959) (214), using a dog tendon model and radio-active phosphorus (^{32}p), showed that blood vessels from the muscle supplied the proximal third and vessels from the bone the distal quarter of the tendon. The rest of the tendon was supplied by vessels from he paratenon - these anastomosing segmentally with vessels from muscle and bone. Heiple et al (1967) (115) showed that ^{14}C - labelled collagen could be used to estimate the tendon blood flow in animal models and Piaggi and Mingione (1981) (217) that ^{133} Xenon could be used in a similar manner in clinical practice. Davies and Young (1954) (60) injected labelled sulphate into immature rats and found maximum radioactivity at the enthesis of tendons and ligaments. Similar observations were made by Ball (1971) (9) who used radioactive calcium. Peacock (1959) (214) and Rathbun and Macnab (1970) (225) also suggested that this region has a rich blood supply.

THE ROTATOR CUFF: Codman (1934) (45) described a musculo-tendinous cuff formed when the shoulder joint capsule fused with parts of the tendons of teres minor, infraspinatus, supraspinatus and subscapularis muscles. Palmer (1939) (211), Lindblom and Palmer (1939) (161) and Pettersson (1942) (215) called this structure the tendon aponeurosis of the shoulder joint. Moseley (1947) (188) introduced the term the "rotator cuff" which referred to the aforementioned muscles and their tendons. In the literature, the terms musculo-tendinous and rotator cuff are used to include both above concepts.

Skinner (1937) (251) and Withers (1949) (286) suggested that the tendinous component of the cuff was short and not fused with the joint capsule in the newborn. With growth, the tendons increased in length and eventually fused

with each other and with the joint capsule. Olsson (1953) (208) dissected a large series of shoulders and found the mean length of the tendinous portion of the cuff to be 19mm for the supraspinatus and 13mm for the infraspinatus and subscapularis tendons. He could not confirm any age related changes in the cuff. The rotator cuff lies between the glenohumeral joint and subacromial bursa, with no communication between the joint and bursa unless rupture of the cuff has occurred.

The supraspinatus muscle is the most important of the rotator cuff muscles. It arises from the supraspinatus fossa of the scapula and inserts into the greater tuberosity of the humerus, merging with the superior aspect of the capsule and forming part of the floor of the subacromial bursa. Its principal action is to assist abduction of the arm by fixing the head of the humerus against the glenoid. This is especially important in the initial 15 - 20^{o} of abduction after which the deltoid muscle plays a more important role.

The infraspinatus muscle arises from the infraspinatus fossa on the posterior aspect of the scapula and inserts into the postero-superior aspect of the greater tuberosity. It acts as an important lateral rotator of the humerus.

Teres minor arises from the axillary border of the scapula and inserts into the postero-inferior aspect of the greater tuberosity. It strengthens the capsule at this site. Its functions are similar to infraspinatus with which it is intimately structurally associated.

The subscapularis muscle arises from the subscapular fossa and inserts into the lesser tuberosity of the humerus just lateral to the glenoid rim. It is separated from the rest of the cuff by the bicipital groove containing the tendon from the long head of biceps which inserts into the supraglenoid tubercle. A transverse ligament often covers the gap between the two tuberosities and when present also gives attachment to the rotator cuff. The subscapularis plays an important role in medial rotation of the humerus.

The tendinous portion of the rotator cuff and the interrelated capsule is attached to all except the inferior aspect of the anatomical neck of the humerus. In this area where the cuff is deficient, only the capsule gains attachment.

VASCULAR SUPPLY TO THE ROTATOR CUFF: Hitzrot (1901) (119) and other early

anatomists described the blood supply to the shoulder region, but as they regarded the tendons to be avascular, they made no attempt to study their vasculature. Mayer (1916) (174) showed that the rotator cuff does have a vascular supply and Lindblom (1939) (160) studying postmortem shoulder specimens described a hypovascular area ('the critical zone') in the supraspinatus tendon, close to its insertion. With aging, he noted progressive degeneration and fibrosis in this area. Olsson (1953) (208) also noted a hypovascular area which he attributed to pressure from the humeral head on the rotator cuff. He felt this pressure caused local tendon ischaemia with resultant degeneration. Laing (1956) (150) used a dye injection technique to study the arterial supply to the humeral head and demonstrated that some osseous vessels did enter the rotator cuff through its attachment. Moseley and Goldie (1963) (187) used a similar method and showed that the rotator cuff received vessels from osseous, tendinous and muscular sources. They found no reduction in the rich blood supply with increasing age and regarded the 'critical zone' as an area of muscular and osseous vessel anastomoses and not a hypovascular area. Rothman and Parke (1965) (237) further defined the macrocirculation to the shoulder, demonstrating that 6 vessels contributed blood to the rotator cuff. They noted profuse anastamoses between the different vessels, but also reported a hypovascular zone in 63% of supraspinatus tendons and a similar area in 37% of infraspinatus and 7% of subscapularis tendons. They felt that tendon degeneration began at this site and increased with age. They used an animal model to demonstrate the progressive reduction in the capillary bed of tendons with age. Reeves (1968) (227) showed a similar reduction in the vascular bed of the rotator cuff in men of increasing age. Rathbun and Macnab (1970) (225) studied the microcirculation of the rotator cuff and demonstrated a marked difference in the vascular pattern of the supraspinatus when compared to the other rotator cuff tendons. Irrespective of age, a hypovascular zone was noted in most supraspinatus and some infraspinatus tendons close to the insertion. Like Olsson (208) they felt that pressure from the humeral head caused "wringing out" of vessels in this region. Macnab (1973) (170) extended this work by experimentally demonstrating that pressure such as that applied by the humeral head on the rotator cuff, could cause a partial interruption of the blood supply to the tendon. The resulting ischaemia caused degenerative changes in the centre of the tendon and not at the site of pressure.

GLENOHUMERAL JOINT CAPSULE: The capsule of the glenohumeral joint is a loose redundant synovial-lined fibrous structure with an intra-articular volume of 20-50 ml. It attaches proximally to the labrum and surrounding

bone and distally to the anatomical neck and shaft of the humerus. It is strengthened both by the rotator cuff tendons and by variable thickened areas which form superior, middle and inferior glenohumeral ligaments. Inferiorly there is no additional support and the capsule forms a lax redundant inferior recess. A number of other synovial-lined recesses may be present. The most constant are the bicipital tendon sheath recess and a recess at the upper end of the subscapularis tendon. In the region of the rotator cuff, the capsule fuses with the tendons and the two structures cannot be separated. Lundberg (1970) (167) analysed the glycosaminoglycan characteristics of the capsule in both normal and frozen shoulders.

NERVE SUPPLY TO THE SHOULDER: The two sensory nerves to the shoulder region, are the axillary (or circumflex) nerve which supplies the anterior aspect of the capsule and joint and the suprascapular nerve to the superior and posterior parts of the joint and capsule, most of the tendon sheath and the acromioclavicular joint (ACJ). The long thoracic nerve also sends a branch to the coracoid process and ACJ.

With the exception of the ACJ (derived from C4 sclerotome), all structures of the shoulder are derived entirely or partially from the C5 sclerotome and thus shoulder pain from whatever deep structure is perceived in the distribution of the C5 dermatome. Pain is therefore felt at the point of insertion of deltoid or down the outer aspect of the arm but not involving the hand or the superior aspect of shoulder. Pain from the ACJ is felt on the superior aspect of the shoulder and cannot radiate to the arm.

TENSILE STRENGTH OF TENDONS (WITH EMPHASIS ON THE ROTATOR CUFF) AND CHANGE WITH AGE:

The remarkable tensile strength of normal adult tendon has been recognised for many decades. McMaster (1933) (177) used an animal model to demonstrate that normal tendon could resist rupture even when severed halfway through its bulk. However, if the blood supply to the tendon was partially interrupted, the tensile strength decreased in 3 - 5 weeks. He also noted that if a normal tendon was stressed, the muscle would tear or the tendon be avulsed from the bone at a lower level of strain than that required to rupture the tendon. Cronkite (1936) (56) confirmed the remarkable tensile strength of human tendons.

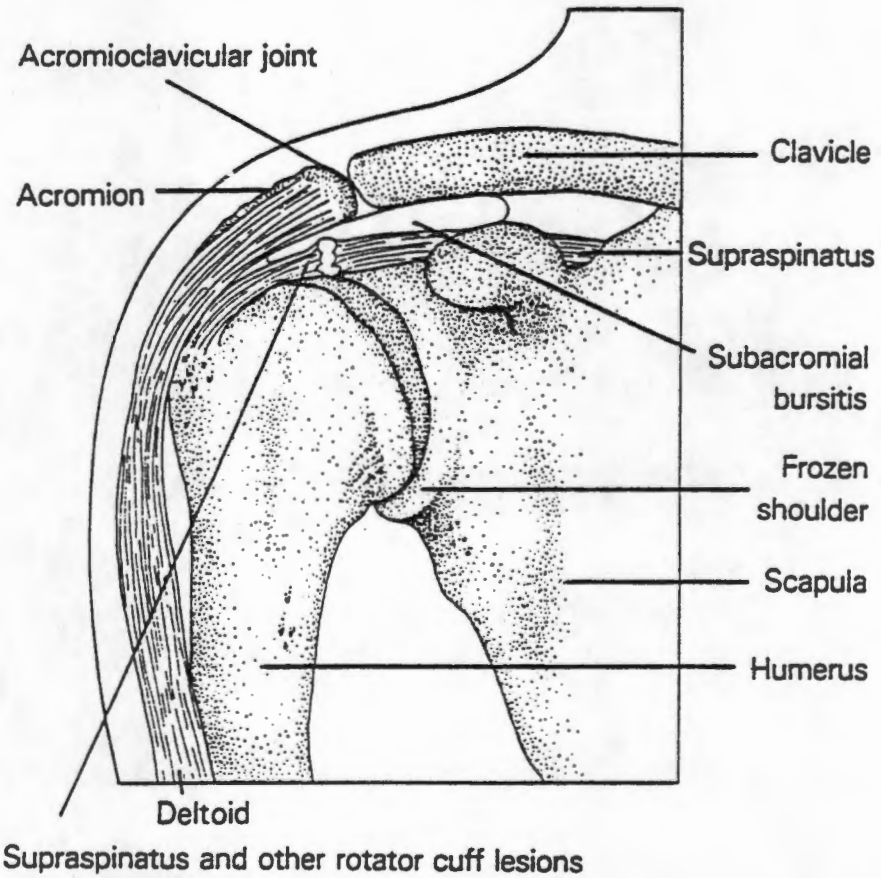
Codman and Akerson (1931) (44) dissected postmortem shoulders and recognised that the supraspinatus tendon was the site of primary pathology with

subacromial bursitis merely being a secondary phenomenon. They noted that rupture of the tendon was common and attributed this to a traumatic episode. King and Holmes (1927) (142) accepted the tendon as the site of pathology, but regarded wear and tear (attrition) as the probable cause. Meyer (1924, 1931) (180, 181) believed that the tendon to the long head of biceps and to a greater or lesser extent all other tendons were subject to increasing attrition with age. Fowler (1933) (85) studied surgical and postmortem specimens and noted a high incidence of unsuspected rupture of the supraspinatus tendon. He regarded this as the major predisposing factor to shoulder pain. Keyes (1935) (141) examined the autopsy specimens of shoulders from an elderly population and found that 20% had unsuspected rupture of the supraspinatus tendon. Skinner (1937) (251) who extended autopsy study of the supraspinatus tendon to include all age groups, regarded degenerative changes in the tendons as a normal occurrence dependent on aging, occupation and attrition. Lindblom (1939) (160), Simmonds (1949) (248) and Wilson and Duff (1943) (282) also reported progressive degeneration in the supraspinatus tendon associated with age and use. Olsson (1953) (208) in an extensive histological study of shoulder tendons found an increasing incidence of degenerative changes with age, but no clear association between tendon degeneration and shoulder pain. He observed that the degeneration first affected the centre of the tendon and was therefore unlikely to have resulted simply from attrition. He postulated that pressure from the humeral head on the supraspinatus tendon resulted in partial obstruction of the vascular supply to the tendon and eventually central tendon degeneration. This stimulated interest in the vascular supply to the rotator cuff, which led Macnab (1973) (170) to construct an animal model to show that pressure such as that applied by the humeral head on the rotator cuff, could cause partial interruption of the blood supply and central tendon degeneration, which rendered the tendon liable to tear when subjected to trivial often unrecognised trauma. This concept remains the basis for current beliefs with regard to the pathology of many of the soft tissue lesions of the shoulder.

FUNCTIONAL ANATOMY OF THE SHOULDER:

The complex and intricate activities of the upper limbs in man require a high degree of precision of muscle synergism and an extraordinary range of shoulder movement. This versatility comes at the cost of potential instability. Unlike the hip joint which has exact congruity of joint surfaces, the shoulder is characterised by a complex system of soft tissue structures (muscles, tendons and ligaments) which connect the bony

components of the shoulder girdle to each other. This permits smooth rhythmic and coordinated shoulder movement in almost every direction. The glenohumeral joint is the main articulation of the shoulder, but movement also occurs at 4 other sites. The acromioclavicular and sternoclavicular joints are true plane joints. Their anatomy has been described by De Palma (1963) (64). Movement also occurs between the scapula and the thoracic wall and in the subacromial region where a large bursa separates the acromioclavicular arch from the head of the humerus and its tuberosities and facilitates smooth movement. The subacromial bursa is intimately associated with the tendons of the rotator cuff, the acromioclavicular joint and other neighbouring structures (Figure 2:1) and often becomes secondarily involved by pathological processes of any of these structures.



F I G U R E 2 : I. Diagram of the shoulder showing the relationship between the shoulder joint, rotator cuff, subacromial bursa and the acromioclavicular joint. Common sites of pathology and common lesions are indicated.

CHAPTER 3

THE PAINFUL STIFF SHOULDER : A REVIEW OF THE LITERATURE

Duplay (1872) (73) described the clinical features of 'scapulo-humeral periarthritis' which included pain and limitation of shoulder range, but its significance was not recognised until Codman (1934) (45) published his authoritative work which attempted to differentiate various clinical shoulder disorders.

Neviaser (1945) (199) surgically explored the shoulders of ten patients with painful stiff shoulder and reported capsular thickening and chronic inflammatory changes with adhesive bands which caused the rotator cuff and capsule to adhere firmly to the humeral head. He suggested 'adhesive capsulitis' as a more suitable name. Simmonds (1949) (248) also operated on painful stiff shoulders but saw no adhesions. He felt that a degenerative lesion in the supraspinatus tendon set up a foreign body reaction with a spread of the inflammatory process to contiguous structures leading to a 'frozen shoulder'. McLaughlin (1961) (176) found true inflammatory changes in the capsule and rotator cuff of only 10% of frozen shoulders explored from 1938 to 1961. The rest showed a marked reduction in joint volume and a shrunken rotator cuff with no inflammatory changes or adhesions. He felt that frozen shoulder constituted a primary disorder of collagen. Bloch and Fisher (1961) (18) analysed over 2000 patients with frozen shoulder and concluded that the condition constituted the final stage of inflammation of the shoulder capsule and periarticular connective tissues. Macnab (1973) (170) postulated a type IV auto-immune reaction to degenerate collagen as the mechanism of development of a frozen shoulder from a localised tendinitis lesion. Lundberg (1969) (168) found marked periarticular inflammation and capsular thickening in 14 surgical specimens from patients with frozen shoulder. Electron microscopy confirmed the increase in fibroblasts and fibrous tissue. He also examined capsular material from the frozen shoulder specimens and found considerable differences in the glycosaminoglycan content when compared to normal capsular material. There was an increase in heparin, chondroitin and dermatan sulphate and a reduction in hyaluronic acid, reflecting the transformation from normal capsule to fibrosis and granulation tissue. He also documented a larger than 50% reduction in the mineralisation of the humeral head and arthrographic evidence of a shrunken capsule; the volume of the joint correlating with the degree of restriction in range. He concluded

that the main abnormality in the frozen shoulder was capsular retraction.

Dickson and Crosby (1932) (66), Meulengracht and Schwartz (1952) (179) and Wright and Haq (1976) (291) noted an association between painful stiff shoulders (PSS) and the occurrence of diabetes mellitus and thyroid disease. Bridgman (1972) (24), Gray and Gottlieb (1976) (97) and Lequesne et al (1977) (157) also found an association with diabetes. Simmonds (1949) (248) recognised the importance of disuse and immobilisation in the precipitation of PSS and also considered cardiovascular disease to be important. De Palma (1952) (62), Quigley (1956) (221) and Haggart et al (1956) (105) stressed disuse and immobilisation in the aetiology of the PSS. Bruckner and Nye (1981) (26) also showed a significant increase in the risk of frozen shoulder in patients with hemiplegia or following periods of unconsciousness.

Franklin and Nemeik (1954) (86), Johnson (1959) (133) and Fisher et al (1963) (81) noted an increased frequency of PSS lesions in patients suffering from pulmonary tuberculosis. They considered immobility and debility to be the important factors. Good et al (1965) (96) felt antituberculous therapy was the more likely cause. However, Saha (1966) (240) also documented a high incidence of PSS in patients with chronic obstructive airways disease.

Lorenz and Musser (1952) (165) felt that personality disorder and an abnormal emotional state were essential elements in the development of a PSS. Coventry (1953) (53) stressed both the 'periarthritic personality' and disuse as the triggering factors for patients with shoulder pain to develop a frozen shoulder. Fleming et al (1976) (84) and Wright and Haq (1976) (291) reviewed the concept of a 'periarthritis personality' and found increased anxiety and neurotic symptoms but no obvious personality disorder.

The association between the PSS and other soft tissue lesions (22, 61, 205, 216), cervical spine disease (195, 247) and enthesopathies (203) has been mentioned. Wright and Haq (1976) (292) noted the frequent occurrence of cervical spondylosis in PSS patients, but felt this was only a reflection of age and not of any other significance. Kopell and Thompson (1959) (145) postulated suprascapular nerve entrapment as an explanation for the severity of pain in the PSS, but no supporting evidence for this concept has been forthcoming.

A review of radiology, blood tests, other investigations and a consideration

of conservative therapy and the natural history of PSS will be given in the relevant chapters.

PART II

THE SHOULDER CLINIC AND SHOULDER CONTROLS

CHAPTER 4

THE SHOULDER CLINIC

Shoulder problems are common in the general rheumatology clinics in Cambridge, England. To facilitate study of such patients a special clinic was set up and local practitioners were asked to refer patients with shoulder pain to the clinic as soon as possible after their presentation and before instituting therapy. All the patients referred to the clinic were examined by the same physician.

CLASSIFICATION

Patients with shoulder pain were included in 3 groups (Table 4:1).

SHOULDER GROUP: Pain modified by shoulder movement and not felt proximal to the shoulder unless the acromioclavicular joint (ACJ) was affected. Neurological deficit in the upper limbs excluded patients from this group.

REFERRED PAIN GROUP: Pain referred from the cervical spine or other structures outside the shoulder with the primary source of the symptoms being identified. The pain was not markedly exacerbated by shoulder movement and was usually felt proximal to the shoulder.

UNDETERMINED GROUP: Patients in whom the site of pathology could not be accurately determined or where the findings were suggestive of a combination of lesions.

CLINICAL ASSESSMENT

All the patients seen at the shoulder clinic were assessed according to the method suggested by Cyriax (58).

General history: Patients were asked about the duration of symptoms, whether they arose spontaneously or followed trauma and if the onset was sudden or gradual. Previous or concomitant pain in the other shoulder, other illnesses and therapy was recorded.

Pain: The exact location of initial pain, its behaviour from onset, the relationship to time of day or night and the effect of active and passive

movements and other aggravating and relieving factors on the pain were documented.

Limitation of range: If the patients had noted any restriction in the range of movement, its timing in relation to the pain, its course and severity was noted.

Cervical spine: Specific enquiries were made with regard to pain and limitation of range of the cervical spine. Neurological symptoms in the upper limbs were also sought.

General examination: In addition to a full general systematic and musculoskeletal examination, particular emphasis was placed on a detailed assessment of the cervical spine and neurological system of the upper limbs. Attempts were made to reproduce the presenting pain by movement of the cervical spine to the extremes of its range.

Shoulder examination: Both shoulders were assessed in an identical manner so the unaffected shoulder acted as a control. The shoulders were inspected for muscle wasting, swelling or deformity and palpated for tenderness around the shoulder or in the region of the ACJ. Patients were instructed to move the shoulder to the extremes of range (active range) and the movement was then continued by the observer (passive range). Four movements were routinely assessed - total forward flexion, total abduction, internal and external rotation. If the passive range was markedly reduced, the glenohumeral component of abduction and flexion was also recorded. The character and rhythm of shoulder movement and presence of a painful arc on active abduction was noted. Exacerbation of the pain on resisted abduction, external and internal rotation was assessed to determine involvement of the supraspinatus, infraspinatus and subscapularis tendons respectively. Shoulder pain on resisted forearm flexion was accepted as an indication of bicipital tendon involvement. Local anaesthetic infiltration at different sites around the shoulder was used to confirm or exclude suspected rotator cuff lesions (58).

All the patients had rheumatoid factor and erythrocyte sedimentation rates estimated and most had X-rays of the shoulders and where indicated the ACJ's.

SHOULDER CLINIC PATIENTS

During a 2 year period, 438 patients were referred to the shoulder clinic. Three hundred and twenty-eight had primary shoulder disease and 64 pain referred to the shoulder. In 46 patients the cause of the symptoms could not be clearly determined (Table 4:2). Only 118 of the patients (27%) had the diagnosis at referral confirmed at the clinic.

SHOULDER GROUP

Rotator cuff lesions: Rotator cuff tendinitis was the commonest shoulder condition referred to the clinic, constituting 41% of the 'Shoulder Group'. The supraspinatus tendon was most frequently affected, although this was sometimes in association with infraspinatus involvement. All these lesions caused a painful arc on abduction. Subscapularis tendinitis was less common and only rarely presented with a painful arc on abduction. Thirty patients had mild symptoms which resolved without treatment and 6 patients refused local corticosteroid injections due to the pain produced by previous injections. The other 98 patients were treated with one or more local steroid injections to the subacromial bursa. Detailed follow-up was not carried out on all these patients, but they were offered the opportunity of returning to the clinic if symptoms recurred. Twenty-three patients with symptoms which persisted for 3 months despite therapy were included in a prospective therapeutic study which is discussed in detail below.

Four patients who presented with pain and severe restriction of range of sudden onset were found on X-ray of the shoulder, to have extensive calcification of the supraspinatus tendon. A diagnosis of acute calcific tendinitis was made. A further 10% of patients with rotator cuff tendinitis had some calcification in the rotator cuff on the affected and 6% on the unaffected side. Complete rupture of the rotator cuff (10 patients) was diagnosed in those patients who were unable to initiate active abduction, but had a full range of passive movement. Six of the 10 patients had arthrography of the shoulder which confirmed the diagnosis in all cases. It was not possible to differentiate partial tears of the cuff from tendinitis and all the patients were included in the tendinitis group. Complete rupture of the cuff failed to respond to local steroid injections and 1 patient required a surgical repair. The others learnt to live with the disability.

Bicipital tendinitis: This lesion was difficult to diagnose clinically.

Pain induced by flexion of the forearm against resistance was considered to be the only reliable sign. Tenderness over the bicipital groove was not a consistent finding. Rupture of the long head of the biceps tendon occurred spontaneously in 2 patient and followed steroid injection in a further 4 cases.

Frozen shoulder: Of the 78 patients (24% of the 'Shoulder Group') with a painful stiff shoulder (PSS), only 10 were related to trivial traumatic incidents. The PSS which followed severe trauma was not included in this group. Six PSS patients had diabetes mellitus, 3 thyroid disease, 6 angina pectoris and 12 systemic hypertension. In 1 patient the PSS followed 6 weeks after a cerebrovascular accident and in another case it followed herpes zoster involvement of the T8 dermatome on the affected side. Many of the frozen shoulder patients were included in the detailed studies of this condition and will be considered below.

Hand-shoulder syndrome: A PSS in association with pain, stiffness and swelling of the hand was found in 4 patients. Three followed acute myocardial infarction and 1 arose spontaneously.

Arthritis: The acromioclavicular joint (ACJ) was a common site of pathology. Twenty lesions followed trauma and 30 arose spontaneously - 18 being in manual workers. The clinical diagnosis of this lesion was usually easy and X-ray confirmation of ACJ arthritis was obtained in 87% of cases. Local steroid injections into the ACJ improved 30 of he 41 patients who received this therapy, but symptoms recurred within 2 months in at least 16 cases (11 manual workers), who had severe and prolonged disability.

Glenohumeral arthritis was an uncommon cause of localised shoulder pain. Osteoarthritis was noted on X-ray in 6 patients, 4 having Heberden's nodes or involvement of other peripheral joints. Only 3 patients had an inflammatory polyarthropathy, 2 with a positive rheumatoid factor. Two patients had a rapidly progressive destructive arthritis of both glenohumeral joints fitting the syndrome of 'Milwaukee Shoulder' (175). All these joints had tense blood-stained effusions containing hydroxyapatite crystals.

Traumatic lesions: Patients with serious traumatic lesions usually present to casualty or orthopaedic departments. Only 4 patients with chronic pain and restriction of range following severe trauma presented to the shoulder clinic. Fracture through the humeral head or neck was noted in all 4 cases.

It was not possible to identify any specific lesion in three other patients who complained of pain following less severe trauma and symptoms resolved without treatment.

Periscapular myalgia: A constant ache in the periscapular muscles associated with mild kyphosis of the thoracic spine and forward drooping of the shoulders was noted in 15 patients. Tenderness could be elicited at the lateral edge of the scapula with exacerbation of the pain by abduction of the shoulder. Local corticosteroid injection under the scapula usually resolved the lesion, but postural retraining was important in preventing recurrence of symptoms.

Miscellaneous: Uncommon causes of shoulder pain seen at the clinic included subscapularis nerve palsy, infection, humeral cyst, tumour and avascular necrosis. X-ray of the shoulder was diagnostic in most cases.

REFERRED PAIN GROUP

Sixty-four patients (15% of all the cases) had shoulder pain of a referred nature, 53 (83%) being due to cervical spondylosis with or without neurological abnormalities in the upper limb. Pain could usually be induced or exacerbated by moving the neck to the limits of its range. Nearly 50% of these patients had failed to recognise any pain or limitation of neck movement. X-ray of the cervical spine was sometimes helpful in confirming the diagnosis.

Secondary deposits of tumour in the cervical spine and visceral malignancy with shoulder tip pain was diagnosed in 4 patients. Polymyalgia rheumatica and the fibrositis syndrome also occasionally presented at the clinic.

UNDETERMINED GROUP

In 46 patients it was impossible to determine the source of the pain. Eight were obese females with pain in the line of the brassiere straps. Weight reduction, postural retraining and alteration in the brassiere strap tension resulted in improvement in the symptoms. In 32 cases the lesion was complex with both features of rotator cuff and cervical spine disease being present. The exact contribution of each lesion could not be determined. In the other 6 patients no signs of pathology were detected and no explanation for the shoulder pain was uncovered.

DISCUSSION

This brief survey of patients seen at the shoulder clinic is not intended to be an exhaustive study of shoulder lesions and their therapy and may not reflect the incidence of different shoulder conditions in the orthopaedic or general rheumatology clinics. The value of the clinic was to confirm (58) that most shoulder lesions can be diagnosed by careful clinical assessment.

Many shoulder lesions resolved spontaneously or with local steroid injections and did not pose a major clinical problem. However, lesions which persisted for at least 3 months despite therapy resulted in serious disability and were therefore selected for detailed study.

TABLE 4:1

CAUSES OF SHOULDER PAIN

A. SHOULDER GROUP

INTRACAPSULAR	1. Arthritis 2. Frozen shoulder 3. Trauma
EXTRACAPSULAR	1. Rotator cuff tendinitis 2. Rotator cuff rupture 3. Acromioclavicular arthritis 4. Periscapular myalgia 5. Other miscellaneous causes

B. REFERRED PAIN GROUP

CERVICAL SPINE	1. Cervical spondylosis 2. Spinal or vertebral tumours
VISCERAL MALIGNANCY	1. Lung 2. Liver
FIBROSITIS	
POLYMYALGIA RHEUMATICA	

C. UNDETERMINED

TABLE 4:2

SHOULDER CLINIC PATIENTS

A. SHOULDER GROUP (N = 328)	No. of Patients	% of Shoulder Group
ROTATOR CUFF LESIONS		
Supraspinatus tendinitis	96	29%
Infraspinatus tendinitis	20	6%
Supra and infraspinatus tendinitis	11	3%
Subscapularis tendinitis	7	2%
Acute calcific tendinitis	4	1%
Rupture of rotator cuff	10	3%
BICIPITAL TENDINITIS	6	2%
FROZEN SHOULDER		
Spontaneous	68	21%
Traumatic	10	3%
HAND-SHOULDER SYNDROME	4	1%
ACROMIOCLAVICULAR ARTHRITIS	50	15%
GLENOHUMERAL ARTHRITIS		
Osteoarthritis	6	2%
Rheumatoid arthritis	3	1%
Milwaukee shoulder	2	1%
TRAUMATIC LESIONS		
Severe trauma	4	1%
Mild sprain	3	1%
PERISCAPULAR PAIN	15	5%
SUBSCAPULARIS NERVE PALSY	2	1%
MISCELLANEOUS	7	2%

B. REFERRED PAIN GROUP (N = 64)	No. of Patients	% of Referred Pain Group
CERVICAL SPINE		
Cervical spondylosis	53	83%
Tumour	2	3%
VISCERAL MALIGNANCY		
Lung	1	2%
Liver	1	2%
POLYMYALGIA RHEUMATICA	4	6%
FIBROSITIS	3	5%

C. UNDETERMINED (N = 48)

CHAPTER 5

SHOULDER CONTROLS

The complexity of shoulder movement and the difficulty in the measurement of its range has resulted in a paucity of knowledge with regard to the normal range and factors that influence this in the general population. The American Academy of Orthopaedic Surgeons (6) described an approach for the documentation of shoulder movement and the average range in the general population. They recognised the importance of differentiating glenohumeral from total range but did not base their findings on population studies or describe a method for the accurate recording of each movement parameter. Freedman and Munro (87) used a radiological and Doody et al (71) a goniometric method for the measurement of shoulder elevation, but neither proved suitable for routine use. Lee et al (152) and Allander et al (2) described methods for recording shoulder rotation, but both were cumbersome and failed to give reproducible results. Nevertheless Allender's population survey did suggest a decrease in passive rotation with age and a lesser range in males when compared to females and the left when compared to the right shoulder. Beighton et al (14) also found a decrease in range with age and a difference between the dominant and non-dominant shoulders.

Clarke et al (41) used a spirit goniometer to measure the passive range of movement in normal controls. Their method was reproducible and showed a definite decrease in range with age, females having a superior range to males for any given age group. They found no difference between the dominant and non-dominant sides. However, they only measured glenohumeral abduction, external and total rotation and failed to report their findings in sufficient detail to determine the variation in range in the normal population. The passive range was therefore assessed in a group of healthy people of similar age to the shoulder patients using a spirit goniometer modified from that used by Clarke, but considering 6 movements - total and glenohumeral abduction and flexion, external and total rotation.

THE CONTROLS

Seventy-six normal people aged from 42 - 75 years (Figure 5:1) had the passive range of movement of both shoulders measured with a spirit goniometer using an identical method to that of the frozen shoulder patients

(see Chapter 6). Forty-two of the controls were carefully age (within 2 years) and sex-matched to the Major Frozen Shoulder Study patients (see below) to provide a specific control group for clinical and laboratory aspects of the study. The controls were drawn from hospital staff and their relatives and other hospital visitors who volunteered to help in the study. All the controls were in good health with no shoulder pain and no past history of shoulder pain or injury or other joint disease.

Age and Sex: The mean and the maximum and minimum limits of the range for each individual movement parameter (both shoulders) in the female and male controls is shown (Table 5:1). The females had a better range in all age groups, the difference increasing with age.

Ignoring age, a comparison between the females and males (Table 5:2), using unpaired t-tests and 150^{o} of freedom, showed a significant advantage ($p<0.001$) for the females for all movements, glenohumeral and total. Comparison of the mean range of each movement parameter in male and female controls in the different age groups showed the greatest difference between the sexes in patients over 60 years, although young males (under 50) had a poor range of rotation (Table 5:3).

Performing a linear regression against age for males and females, significant decreases ($p<0.001$) in the range of total flexion (Figure 5:2), total abduction (Figure 5:3), total rotation (Figure 5:4) and external rotation (Figure 5:5) occurred with increasing age. The decrease in range in the males was greater than in the females for total flexion and abduction but not rotation.

Arm Dominance: The range of movement in the controls was compared using unpaired t-tests for dominant and non-dominant (Table 5:4) and also for right and left shoulders (Table 5:5). No significant differences were noted between the groups. Rotation and forward flexion was slightly better in the left or non-dominant shoulder and abduction was better in the right or dominant side. Twelve of the controls were left dominant.

The Major Frozen Shoulder Study Control Group: The mean and maximum and minimum range of movement in the right shoulder of the 42 patients who were age and sex-matched to the Major Frozen Shoulder Study patients was very similar to the range in the right shoulders of the entire control group (Table 5:6).

DISCUSSION

Detailed analysis of the movement in the normal controls has confirmed (2, 14, 41) that females of all ages have a superior range to males and that the decrease with age is more marked in the males. The younger males (less than 50 years) had a particularly poor range of rotation which was thought to be due to their increased muscle bulk, as many of these controls were manual workers. The dominance of the arm was not found to influence the range of movement in the normal population in either sex.

TABLE 5:1

RANGE OF SHOULDER MOVEMENT IN THE NORMAL CONTROLS OF DIFFERENT AGE AND SEX (BOTH SHOULDERS)

		40 - 49 YEARS		50 - 59 YEARS		60 - 74 YEARS	
		Female	Male	Female	Male	Female	Male
Number of Shoulders		16	18	38	20	32	28
TF	Mean	176°	171°	170°	166°	164°	158°
	Range	170-180	165-180	150-180	155-175	150-180	140-170
GF	Mean	90°	88°	95°	90°	86°	76°
	Range	80-100	80-100	80-100	80- 95	65-100	60- 90
TA	Mean	172°	165°	166°	161°	161°	153°
	Range	160-180	155-170	140-180	150-175	150-180	130-140
GA	Mean	85°	85°	83°	82°	78°	66°
	Range	80- 90	80- 95	70-100	65- 90	65-100	50- 90
ER	Mean	74°	54°	69°	63°	61°	49°
	Range	60- 90	45- 70	50- 90	50- 75	45- 80	40- 60
TR	Mean	173°	158°	170°	165°	158°	147°
	Range	160-180	150-170	140-185	150-180	135-180	135-165

TABLE 5:2

COMPARISON OF RANGE IN FEMALE (F) AND MALE (M) CONTROLS

	Mean Difference in Range (F - M)	Unpaired T-test T_{150}	p<
TF	5.5°	3.340	0.001
GF	7.6°	5.075	0.001
TA	7.1°	4.730	0.001
GA	5.7°	3.826	0.001
ER	12.5°	7.399	0.001
TR	10.9°	5.895	0.001

TABLE 5:3

COMPARISON OF RANGE IN FEMALE (F) AND MALE (M) CONTROLS ACCORDING TO AGE

	Mean Difference in Range (F - M)	DF	Unpaired T-test T	p<
A. 40-49 YEARS		32		
TF	4.3°		2.886	0.01
TA	7.5°		3.942	0.001
ER	20.8°		7.400	0.001
TR	15.9°		7.017	0.001
B. 50-59 YEARS		56		
TF	3.9°		1.624	N/S
TA	5.4°		2.317	0.05
ER	6.2°		2.357	0.05
TR	2.5°		0.548	N/S
C. 60+ YEARS		58		
TF	6.2°		3.185	0.001
TA	8.2°		3.610	0.001
ER	11.9°		5.264	0.001
TR	11.5°		4.757	0.001

N/S = Not Significant

TABLE 5:4

COMPARISON OF NON-DOMINANT (N-D) AND DOMINANT (D) SHOULDERS IN CONTROLS

	Mean Difference in Range (N-D - D)	Unpaired T-test T_{150}	p<
TF	0.5^{o}	0.295	N/S
TA	-0.4^{o}	-0.288	N/S
ER	1.7^{o}	0.886	N/S
TR	1.8^{o}	0.825	N/S

N/S = Not Significant

TABLE 5:5

COMPARISON OF LEFT (L) AND RIGHT (R) SHOULDERS IN CONTROLS

	Mean Difference in Range (L - R)	Unpaired T-test T_{150}	p<
TF	0.6^{o}	0.399	N/S
TA	-0.5^{o}	-0.290	N/S
ER	1.6^{o}	0.806	N/S
TR	1.8^{o}	0.904	N/S

N/S = Not Significant

TABLE 5:6

MEAN (AND MAXIMUM AND MINIMUM) RANGE IN THE RIGHT SHOULDER OF ALL CONTROLS AND OF THE CONTROLS WHO WERE AGE AND SEX-MATCHED TO THE MAJOR FROZEN SHOULDER STUDY PATIENTS

	Major Frozen Shoulder Study Controls (N = 42)	All Controls (N = 76)
	Mean (Range)	Mean (Range)
TF	167° (145 - 180)	166° (140 - 180)
GF	90° (65 - 105)	87° (65 - 105)
TA	163° (140 - 180)	163° (140 - 180)
GA	83° (60 - 100)	79° (60 - 100)
ER	65° (40 - 90)	61° (40 - 90)
TR	164° (140 - 180)	162° (135 - 180)

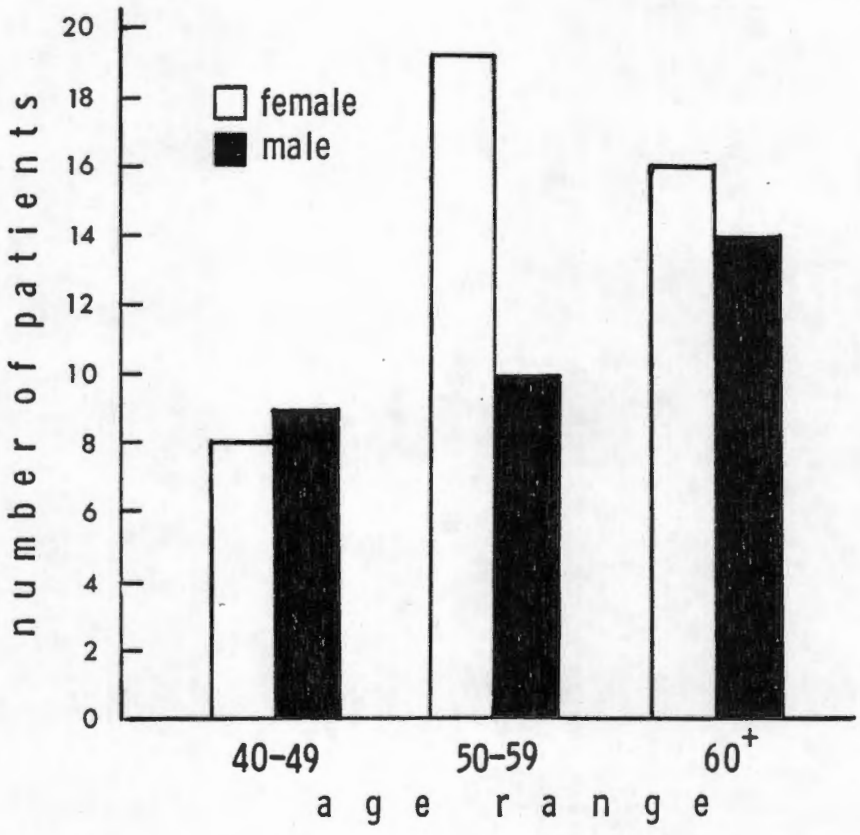
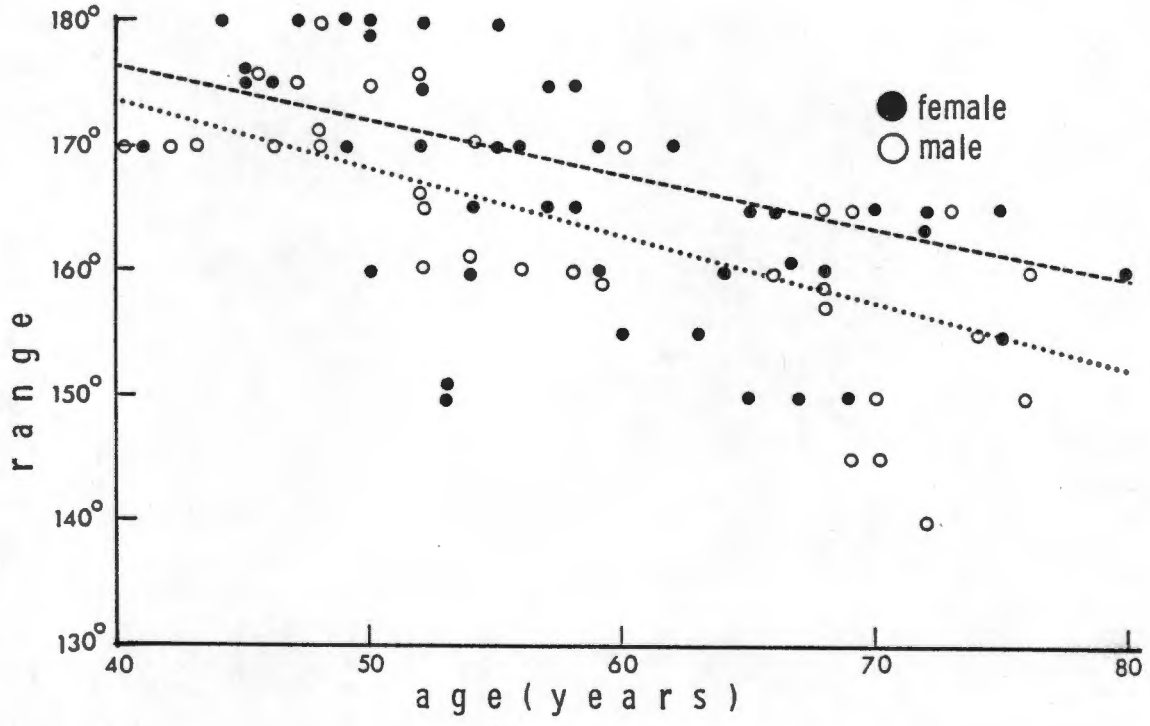
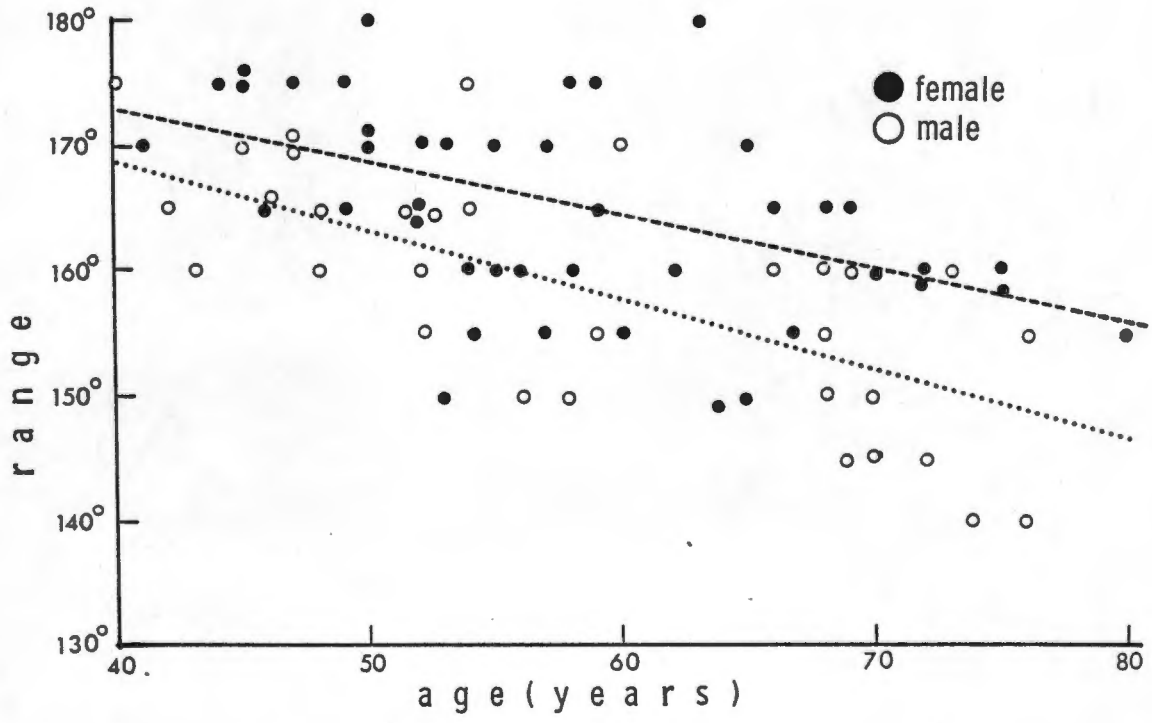


FIGURE 5 : I. Shoulder controls according to sex and age.

F I G U R E S 5 : 2 - 5. THE RANGE OF MOVEMENT IN THE CONTROLS (RIGHT SHOULDER) SHOWING REGRESSION WITH AGE IN FEMALES (----) AND MALES (·····).



F I G U R E 5 : 2. Total flexion.



F I G U R E 5 : 3. Total abduction.

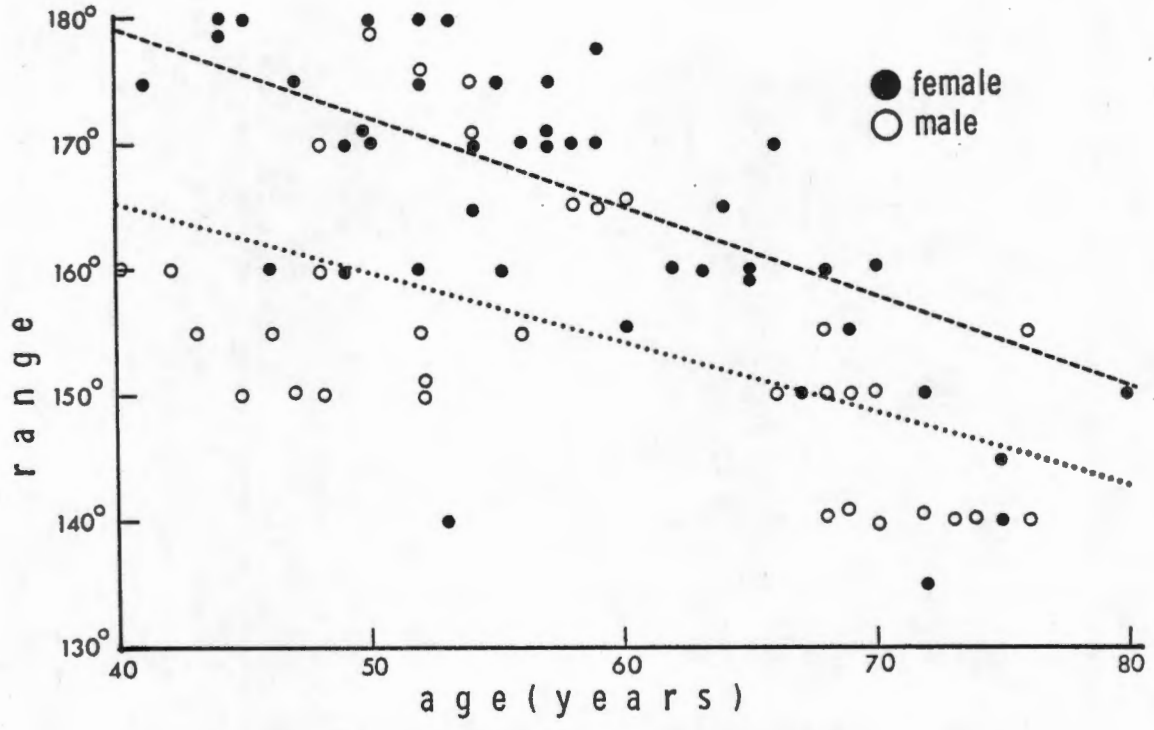


FIGURE 5 : 4. Total rotation.

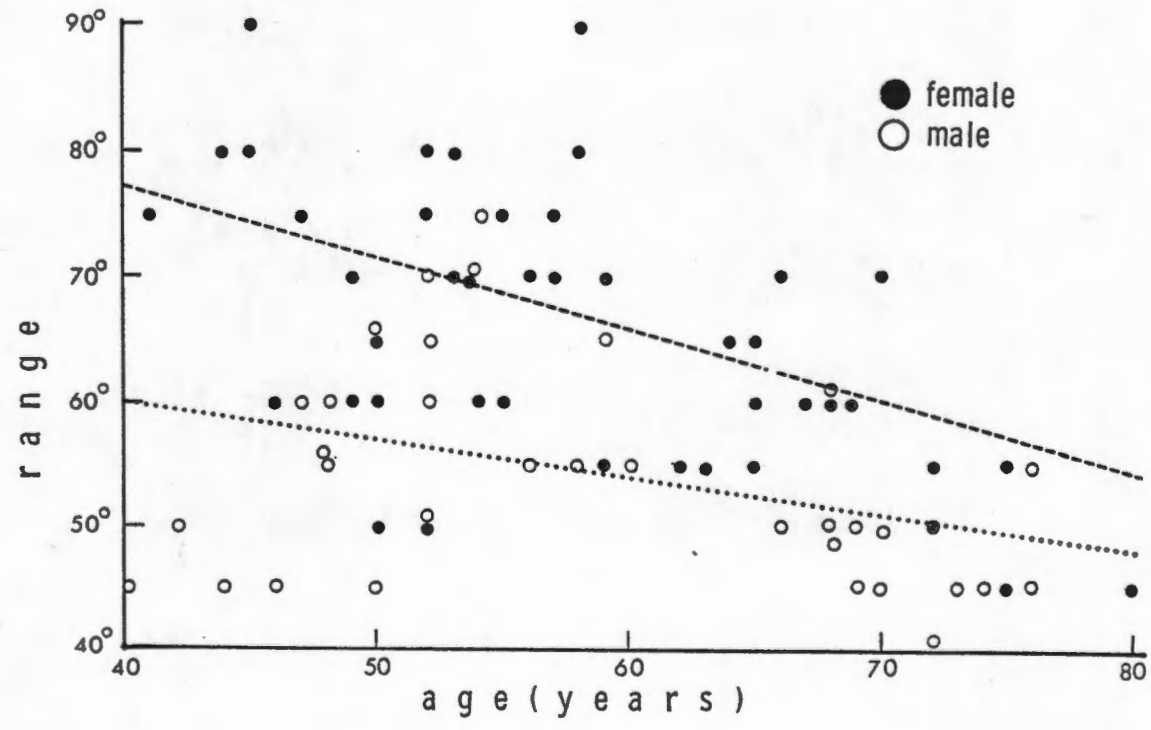


FIGURE 5 : 5. External rotation.

PART III

THE PAINFUL STIFF (FROZEN) SHOULDER

CHAPTER 6

FROZEN SHOULDER : CLINICAL ASSESSMENT AND FROZEN SHOULDER STUDY GROUPS

Periarthritis humero-scapularis (73), checkrein shoulder (221), adhesive capsulitis (199) and frozen shoulder (45) are some of the terms applied to describe the painful stiff shoulder. Whether all refer to the same condition is uncertain, as few of the reports have defined the patient populations studied. This has resulted in problems in comparative assessment of the different studies. The intention of this present work was to study patients at the more severe end of the spectrum of the painful stiff shoulder. Frozen shoulder (F/S) and painful stiff shoulder (PSS) will be used synonymously.

INCLUSION CRITERIA:

1. Shoulder pain for at least one month with night pain resulting in sleep disturbance and an inability to lie on the affected shoulder.
2. All active and passive movements restricted with passive external rotation reduced by at least 50% when compared to the opposite side or age and sex-matched controls. In the 'Major Frozen Shoulder Study' (see below) total abduction also had to be 100^{o} or less.
3. Onset of symptoms spontaneous or following trivial trauma.

EXCLUSION CRITERIA:

1. Cervical spondylosis, neurological abnormality in the upper limbs or radiation of pain up to the neck or below the elbow.
2. Clinical or radiological evidence of a generalised arthropathy, glenohumeral or acromioclavicular arthritis.
3. Severe injury to soft tissue, fracture of the humerus or dislocation of the shoulder joint.
4. PSS precipitated by cerebrovascular accident, myocardial infarction, loss of consciousness or other diseases.
5. Severe systemic illness such as malignancy, chronic infection or connective tissue disease.
6. Symptoms for more than 12 months or where therapy such as local steroid injections or physiotherapy had already begun.
7. Positive rheumatoid factor.

CLINICAL ASSESSMENT:

The patients were considered with regard to their eligibility for inclusion in the studies. The protocol for clinical assessment is shown in Appendix I. The clinical features at presentation with possible relevance to the rate and extent of recovery were age, sex and dominance of the affected shoulder. The occupation, hobbies, type of onset (traumatic or spontaneous) and duration of symptoms at presentation was also noted.

The clinical features for serial assessment were:-

Pain: Pain was particularly severe at night and on movement but an ache often persisted even when sitting at rest during the day. Ten centimetre horizontal visual analogue scales were used to quantify the severity of pain 'at night', 'on movement' and 'at rest'. The duration before patients were able to lie on the affected shoulder and the analgesic consumption from the previous visit was also recorded.

Pain on 'resisted' abduction, external and internal rotation: This was assessed on examination.

Passive range: A spirit goniometer similar to that described by Clarke et al (41) was strengthened and fitted with a curved perspex base and velcro straps and was used to measure the range of movement in the frozen shoulder studies. Although both the active and passive range was recorded, only the passive range was used for serial assessment. Six passive movements were measured. Abduction and forward flexion were recorded with the patient seated in an upright position and with the goniometer attached to the upper arm. For flexion it faced forward and for abduction laterally. For total flexion (TF) and total abduction (TA) the shoulder was moved as far as possible without restraint. Measurement of glenohumeral flexion (GF) and abduction (GA) was similar but with the observer exerting firm pressure over the acromion to prevent scapula movement. Passive external rotation (ER) and total rotation (TR) were recorded with the patient lying supine with the goniometer attached to the dorsal aspect of the forearm, ER being measured with the upper arm held against the side of the body and the elbow flexed to 90^{o}. For TR the shoulder was abducted to 90^{o} or as far as possible and rotated from full internal to full external rotation. Internal rotation (IR) was recorded using the highest vertebral level reached by the extended thumb in the "Major Frozen Shoulder Study" and by the distance (in cm) from the extended thumb to the spine of the 7th cervical vertebra in the

"Supplementary Frozen Shoulder Study Group". All measurements were performed twice, and if they differed by more than 5^{o} they were repeated once more and the mean of the closest 2 observations was taken. The passive range of the opposite shoulder was recorded on each occasion to provide a within-patient control to detect change in the goniometer function.

RELIABILITY OF CLINICAL PARAMETERS

Pain scores: Many of the patients reported difficulty in transcribing their verbal descriptions of pain to the visual analogue scales, so that these could not be used in isolation in the assessment of recovery.

Passive range: Twenty patients had the passive range of movement recorded by 2 independent observers (A & B) using the method described above. 'Sign' test comparison of the measurements by the 2 observers (Table 6:1) showed no significant differences at the 5% level for any movement parameter except internal rotation (measured in cms below C7 vertebra). A paired t-test analysis (Table 6:2) produced similar results. As internal rotation did not give reproducible results, it was not used in the routine assessment.

FROZEN SHOULDER STUDY GROUPS

A. The Major Frozen Shoulder Study: Forty-five patients were recruited into the study but 3 withdrew within 6 weeks and were excluded from the analysis. The reasons for withdrawal were pain induced by the arthrogram (1 patient), desire for physiotherapy elsewhere and 1 patient moved away from the area. The patients took 18 months to recruit, emphasising that the frozen shoulder using the strict criteria for diagnosis, is not all that common. The Major Frozen Shoulder Study patients were subjected to detailed investigations at presentation and were followed up weekly for 6 weeks and then monthly for a further 6 months. Many of the investigations (see below) were then repeated at the end of this time. Forty of the patients were also re-examined 40-48 months (mean 44 months) after their initial presentation.

The investigations performed at presentation (** repeated after 8 months) were:

1. Haematology ** - haemoglobin; white cell count; erythrocyte sedimentation rate.
2. Biochemistry ** - blood glucose; thyroid function tests.
3. Immunology ** - immunoglobulin classes; immune complex levels;

C-reactive protein levels; lymphocyte numbers, sub-populations and transformation studies; complement levels; tests for anti-nuclear antibodies and rheumatoid factor.

4. HLA Typing for A and B loci antigens.

5. Radiology - plain X-rays of both shoulders and acromioclavicular joints; technetium diphosphonate scans; arthrography of the affected shoulder.

Patients were randomly allocated to 1 of 4 treatment groups according to a pre-arranged randomisation schedule. Eleven patients had steroid injection therapy, 11 mobilisation physiotherapy and 12 ice-pack treatment. Eight patients were given no specific therapy. All the patients were encouraged to perform home pendular exercise for 2-3 minutes each hour during waking hours.

B. The Supplementary Frozen Shoulder Study: A further 40 patients with frozen shoulder were included in the Supplementary Study comparing oral prednisolone therapy to no specific treatment. Hourly home pendular exercise was again encouraged. Patients were randomly allocated to one or other treatment group and were followed up fortnightly for 6 weeks and then monthly for a further 6 months. Investigations at presentation were erythrocyte sedimentation rate, rheumatoid factor and plain X-ray of the shoulders.

TABLE 6:1

COMPARISON OF INDIVIDUAL SHOULDER RANGE PARAMETERS BY TWO INDEPENDENT OBSERVERS USING THE 'SIGN' TEST

Movement	+ve's	Zero	-ve's	+ve's + -ve's	+ve's	Probability (2-sided)
TF	8	8	4	12	8	0.40*
GF	6	8	6	12	6	1.00*
TA	6	9	5	11	6	1.00*
GA	8	8	4	12	8	0.40*
ER	4	7	9	13	4	0.08*
TR	3	8	9	12	3	0.15*
IR	11	7	2	13	11	0.05

* = Not Significant

TABLE 6:2

PAIRED T-TEST COMPARISION OF INDIVIDUAL SHOULDER RANGE PARAMETERS BY TWO INDEPENDENT OBSERVERS

Movement	Observer Mean Range		Paired T-test	
	'A'	'B'	T_{20}	p<
TF	100.8°	98.8°	1.773	N/S
GF	57.3°	57.0°	0.463	N/S
TA	82.8°	82.6°	0.002	N/S
GA	47.3°	46.5°	1.165	N/S
ER	15.5°	18.5°	-1.738	N/S
TR	61.5°	63.8°	-1.584	N/S
IR (cm)	20.7	11.8	3.593	0.05

N/S = Not Significant

CHAPTER 7

FROZEN SHOULDER AT PRESENTATION

The 82 patients included in the Major and Supplementary Frozen Shoulder Study groups will be considered together, but important differences between the groups will be noted. Patient details are shown in Table 7:1.

GENERAL FEATURES

Age and Sex: 52 patients (63%) were female and 30 (37%) male. Patients were aged from 44 - 76 years (mean 55.5 years). The mean age in females was 54.8 years and in males 57.5 years. The age distribution in both sexes (Figure 7:1) was similar with 28 females (54%) and 17 males (57%) presenting between the ages of 50 - 60 years.

Arm Dominance: 47 patients (57%) had non-dominant and 35 (43%) dominant arm involvement. 23 females (44%) and 12 males (40%) had dominant and 29 females (56%) and 18 males (60%) non-dominant lesions. The non-dominant arm was therefore more common in both sexes.

Occupation: At the onset of symptoms, 17 patients were performing heavy manual labour, 7 involving toolwork, bricklaying or painting and decorating (Table 7:2). A further 16 patients did only light manual labour such as driving a lorry, nursing or engineering. The other 49 were housewives, sedentary workers or were unemployed. There was no association between occupation and the arm dominance of the lesion (Table 7:3). 12 patients who performed light or heavy manual labour stopped work before presentation because of shoulder pain. Most patients also reported an inability to perform housework, carry heavy loads and perform many other normal daily activities.

Hobbies: Gardening, home improvements, knitting and sport were the most common hobbies undertaken by the patients (Table 7:4). While over 40% of patients were able to continue gardening and knitting, few were able to continue with home improvement or sporting activities.

Unassociated Disease and its Therapy:

(a) Active Disease: At presentation 5 patients were being treated for angina pectoris or systemic hypertension. 3 patients had thyroid disease requiring treatment and 1 diabetes mellitus (Table 7:5).

(b) Inactive Disease: Cervical spine lesions, low back pain and lateral humeral epicondylitis were the most common conditions in the past medical history (Table 7:6). Other miscellaneous conditions included duodenal ulceration, subarachnoid haemorrage, pulmonary tuberculosis and sarcoidosis of the lung.

(c) Symptoms in the Opposite Shoulder: 11 patients (13%) gave a past history of pain or pain and stiffness in the opposite shoulder before inclusion in the study (Table 7:7). 7 other patients had symptoms in the opposite shoulder at presentation, 2 being frozen shoulders, 3 rotator cuff tendinitis and 1 acromioclavicular arthritis.

Onset of Symptoms: 16 patients (11 Major and 5 Supplementary Study patients) reported that symptoms followed minor trauma, usually a fall onto the shoulder or the outstretched arm. More severe trauma which induced bruising, fractures or dislocations around the shoulder resulted in patient exclusion. Of the other 66 patients in whom the condition arose spontaneously, 55 could not attribute the symptoms to any cause, 3 followed stretching awkwardly, 2 each driving, painting and decorating or lifting a heavy load and 1 hedge clipping. In one patient symptoms followed 2 months after a herpes zoster infection of the eighth thoracic dermatome on the same side as the frozen shoulder. Of the 66 patients with a spontaneous onset of symptoms, 11 lesions began from January - March, 18 from April - June, 22 from July - September and 15 from October - December.

Only 7 of the 82 patients (9%) admitted to any shoulder pain before the onset of the frozen shoulder. The duration of this pain varied from 2 weeks to 3 years (mean 6.6 months), but only 2 patients sought medical aid for these symptoms.

Duration of Symptoms: All the patients in the Major and Supplementary Frozen Shoulder groups were seen from 1 - 12 months after the onset of symptoms (Figure 7:1). 31 patients were seen between 1 - 3 months, 35 from 4 - 6 months and the other 16 from 6 - 12 months after the onset. The general practitioners were circulated before the Major Study, thus more

patients in this group were seen within 3 months (Table 7:8). Patients with a traumatic onset (mean 3.3 months) presented earlier than those with a spontaneous onset (mean 5.9 months).

Treatment for Shoulder Symptoms Before Referral: Non-steroidal anti-inflammatory agents (NSAIDS) were prescribed to 51 of the 82 (62%) frozen shoulder patients, but only 5 of these patients (10%) found them beneficial (Figure 7:2). In contrast, 17 of 33 patients (52%) who received simple analgesic agents found them helpful - particularly in the reduction of night pain. Topical (counter-irritation) creams, infra-red lamps and slings were occasionally prescribed. 9 patients were still taking NSAIDS and 24 analgesic tablets at the time of presentation, although many patients did not regard the tablets as effective (Figure 7:3).

16 patients (20%) had no therapy before referral and 46 (56%) were taking no therapy at presentation. The mean duration of symptoms at presentation was 3.8 months in the untreated patients.

Of the 82 patients studied, 10 had been advised to exercise the shoulder despite the pain, 12 to use the shoulder normally but avoid undue strain and 5 to rest the shoulder. 55 patients had been given no advise regarding use of the shoulder.

SYMPTOMS AT REFERRAL

A. Pain Score: The severity of pain at rest, on movement and at night varied greatly from patient to patient, but there were no significant differences between the study groups (Table 7:9). The mean score for all 82 patients was therefore calculated (Table 7:10). The severity of pain was not dependent on the duration of symptoms at presentation (Table 7:11) or whether the symptoms arose spontaneously or following trauma (Table 7:12). Dominant or non-dominant arm involvement also did not influence the initial severity of the pain.

B. An Inability to Sleep on the Affected Shoulder: This was found at presentation in 78 of the 82 (95%) patients.

FEATURES ON EXAMINATION

A. Tenderness: The presence and severity of tenderness on palpation of the shoulder was an inconsistent and unreliable finding. 17 patients (21%)

had slight tenderness over the greater tuberosity of the humerus and 6 (7%) over the lesser tuberosity, but these patients did not differ in severity from the other 72 patients with no localised tenderness.

B. Pain on Resisted Movement: 77 of the 82 patients (94%) had pain exacerbated by resisted abduction; 75 (92%) pain on resisted external rotation and 72 (88%) pain on resisted internal rotation.

C. Passive Range: As the entry criteria for the Major and Supplementary Frozen Shoulder Study groups differed with regard to the severity of the restriction of abduction, the 2 groups will be considered separately (Table 7:13). The mean range for all the frozen shoulder patients at presentation is also shown. The duration of symptoms at presentation (Table 7:14) and type of onset (Table 7:15) did not affect the passive range of movement at the initial visit.

DISCUSSION

This study has confirmed (113, 139, 168) that the painful stiff shoulder (PSS) occurs over a narrow age range (50 - 70 years), with symptoms very rarely beginning below the age of 40 years (291). While a slight female preponderance was noted in agreement with the general literature, this study did not confirm Lundberg's (168) observation that females develop symptoms at a younger age. In keeping with Lundberg (168), Kessel et al (139), and others (46, 162, 200), but in contrast to Wright et al (291) and Sheldon (246), non-dominant arm involvement was more common.

The PSS has been associated with manual labour (53, 291), but like Kozin (148), De Palma (63) and Herberts et al (116), there was no relationship of symptoms to any specific occupation or hobby. There was also no confirmation (168) of a seasonal variation in the onset of symptoms.

Unlike several other reported series (179, 246, 263, 291) and possibly as a reflection of the stringent entrance criteria used, the patients were in good general health. However, this study did confirm (63, 168, 228, 233, 246) that symptoms were common in the opposite shoulder, but were rarely evident on the affected side (233) in the weeks or months preceding the onset of the frozen shoulder.

Minor trauma was an infrequent precipitating factor for the PSS (233, 291) and the only difference at presentation between the traumatic and

spontaneous lesions, was that those following trauma presented earlier in the course of the disease.

Non-steroidal anti-inflammatory agents are still the mainstay of general practitioner therapy of the PSS, but were ineffective in most the trial patients. Reported studies (72, 130, 229) have compared two or more of these agents to each other, but several of these authors have doubted their value in this condition. A higher proportion of the patients who used simple analgesic agents found them of value, particularly in the amelioration of pain at night. Few of the patients had been given any advice regarding the need to gently move the shoulder and several had been told to avoid any movement of the affected limb.

The clinical details at presentation of a group of patients with the PSS has been documented to permit their careful prospective study.

TABLE 7:1

CLINICAL DETAILS OF FROZEN SHOULDER PATIENTS AT PRESENTATION

	Major F/S Study (N = 42)	Supplementary F/S Study (N = 40)
Sex - F : M	28 : 14	24 : 16
Age range in years (Mean)	44 - 74 (55.5)	45 - 76 (54.8)
Onset - spontaneous:traumatic	28 : 14	36 : 4
Arm - dominant:non-dominant	22 : 20	13 : 27
Duration of symptoms in months (Mean)	1 - 12 (4.8)	1 - 12 (5.5)

TABLE 7:2

OCCUPATION OF FROZEN SHOULDER PATIENTS AT THE ONSET OF SYMPTOMS (N = 82)

Occupation	No. of Patients	% of N
Housewife	25	30%
Non-manual	24	29%
Light manual	16	20%
Heavy manual	17	21%

TABLE 7:3

ASSOCIATION BETWEEN PATIENT OCCUPATION AND ARM DOMINANCE

	Dominant Arm (N = 35)	Non-dominant Arm (N = 47)
Manual	15	18
Sedentary	20	29

X_2^2 With Yates Correction = 0.0356 (Not Significant)

TABLE 7:4

MORE COMMON HOBBIES UNDERTAKEN BY FROZEN SHOULDER PATIENTS BEFORE THE ONSET OF SYMPTOMS (N = 82)

Hobbies	No. of Patients	No. Who Stopped Hobby Because of Frozen Shoulder.
Gardening	19	11
Home Improvement	16	15
Knitting	10	6
Darts	6	4
Squash	2	2
Other Sport	4	3

TABLE 7:5

ACTIVE MEDICAL CONDITIONS AND THERAPY IN FROZEN SHOULDER PATIENTS AT PRESENTATION (N = 82)

Condition	No. of Patients	Therapy
Angina pectoris	2	B-Blockers, trinitrate
Systemic hypertension	3	B-Blockers, diuretics
Diabetes mellitus	1	Insulin
Thyroid disease	3	Thyroxine
Asthma	1	Bronchodilator inhaler
Duodenal ulcer	1	Cimetadine
Alcoholic hepatitis	1	-

TABLE 7:6

MEDICAL CONDITIONS IN THE PAST IN FROZEN SHOULDER PATIENTS

Condition	No. of Patients
Chronic low backache	7
Lateral epicondylitis	7
Cervical spondylosis	4
Duodenal ulcer	2

TABLE 7:7

SYMPTOMS IN THE OPPOSITE SHOULDER BEFORE OR AT THE TIME OF PRESENTATION (N = 82)

	Symptoms at Presentation	Symptoms in Past
Shoulder Pain	5	7
Shoulder Pain and Stiffness	2	4

TABLE 7:8

DURATION OF SYMPTOMS AT PRESENTATION IN THE MAJOR AND SUPPLEMENTARY FROZEN SHOULDER GROUPS

Duration at Presentation	Major Frozen Shoulder Study (N = 42)	Supplementary Frozen Shoulder Study (N = 40)
	No. %	No. %
1 - 3 months	20 (48%)	11 (27%)
4 - 6 months	14 (33%)	21 (53%)
6 - 12 months	8 (19%)	8 (20%)

TABLE 7:9

MEAN PAIN SCORES FOR PAIN AT REST, ON MOVEMENT AND AT NIGHT IN MAJOR AND SUPPLEMENTARY FROZEN SHOULDER STUDY PATIENTS (N = 82)

	Mean Score in Major F/S Study	Mean Score in Supplementary F/S Study	Unpaired T-test T_{80}	p<
Pain at rest	2.83	3.40	1.540	N/S
Pain on movement	6.33	6.19	-0.278	N/S
Pain at night	5.74	6.06	0.615	N/S

N/S = Not Significant

TABLE 7:10

MEAN PAIN SCORE AT PRESENTATION FOR PAIN AT NIGHT, AT REST AND ON MOVEMENT IN ALL THE FROZEN SHOULDER PATIENTS (N = 82)

	Mean Score	Standard Error of Mean (SEM)
Pain at rest	3.15	0.19
Pain on movement	6.27	0.25
Pain at night	5.93	0.27

TABLE 7:11

ASSOCIATION BETWEEN THE DURATION OF SYMPTOMS AND SEVERITY OF PAIN AT PRESENTATION

	1-3 Months (N=31) Mean (SEM)	4-6 Months (N=34) Mean (SEM)	7-12 Months (N=17) Mean (SEM)
Pain at rest	3.26 (0.23)	2.97 (0.24)	3.12 (0.46)
Pain on movement	6.29 (0.43)	5.99 (0.45)	6.76 (1.64)
Pain at night	6.21 (0.47)	5.71 (0.38)	5.71 (0.56)

TABLE 7:12

ASSOCIATION BETWEEN THE TYPE OF ONSET (TRAUMATIC OR SPONTANEOUS) AND SEVERITY OF PAIN AT PRESENTATION

	Spontaneous Onset (N=66) Mean (SEM)	Traumatic Onset (N=16) Mean (SEM)
Pain at rest	3.08 (0.20)	3.25 (0.48)
Pain on movement	6.40 (0.28)	5.69 (0.68)
Pain at night	5.94 (0.30)	5.72 (0.63)

TABLE 7:13

PASSIVE RANGE OF SHOULDER MOVEMENT IN THE MAJOR AND SUPPLEMENTARY FROZEN SHOULDER GROUPS AT PRESENTATION

	Major F/S Group (N = 42) Mean (Range)	Supplementary F/S Group (N = 40) Mean (Range)	All F/S Patients (N = 82) Mean
TF	99° (50-135)	106° (60-145)	103°
GF	51° (40- 70)	68° (40- 80)	60°
TA	69° (20-110)	79° (40-130)	74°
GA	40° (0- 70)	46° (20- 75)	43°
ER	13° (0- 35)	21° (0- 50)	17°
TR	41° (0-100)	58° (10-130)	50°

TABLE 7:14

PASSIVE RANGE OF MOVEMENT IN FROZEN SHOULDER PATIENTS OF VARYING DURATIONS OF SYMPTOMS AT PRESENTATION

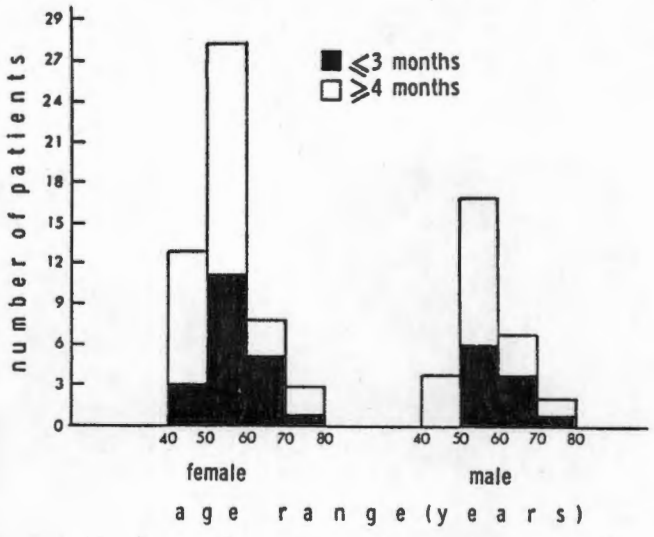
	1-3 Months (N = 31) Mean (SEM)	4-6 Months (N = 35) Mean (SEM)	7-12 Months (N = 16) Mean (SEM)
TF	99.7° (3.8)	105.6° (3.3)	105.9° (6.9)
TA	71.8° (5.0)	79.3° (3.0)	77.2° (8.8)
ER	20.3° (2.4)	16.0° (2.1)	12.8° (2.1)
TR	46.6° (3.5)	51.2° (3.3)	57.5° (6.4)

TABLE 7:15

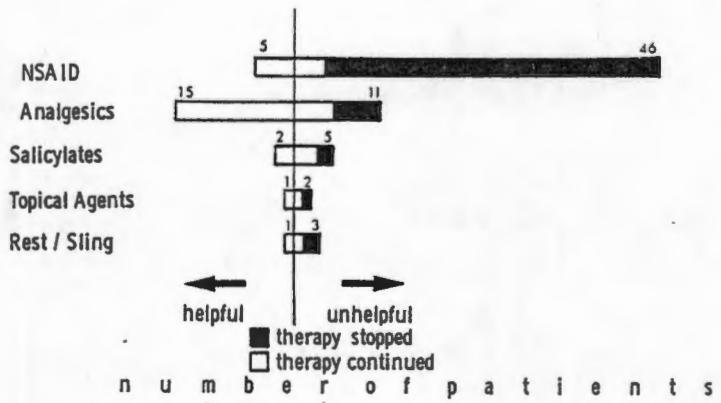
PASSIVE RANGE OF MOVEMENT AT PRESENTATION IN PATIENTS WITH A SPONTANEOUS AND TRAUMATIC ONSET OF SYMPTOMS

	Spontaneous Onset (N = 66) Mean (SEM)	Traumatic Onset (N = 16) Mean (SEM)	Unpaired T-test T_{80}	p<
TF	105.4° (2.7)	95.3° (4.5)	1.665	N/S
TA	77.9° (3.0)	68.4° (7.6)	1.323	N/S
ER	16.7° (1.5)	18.1° (2.7)	-0.401	N/S
TR	48.9° (2.8)	57.8° (6.5)	-1.349	N/S

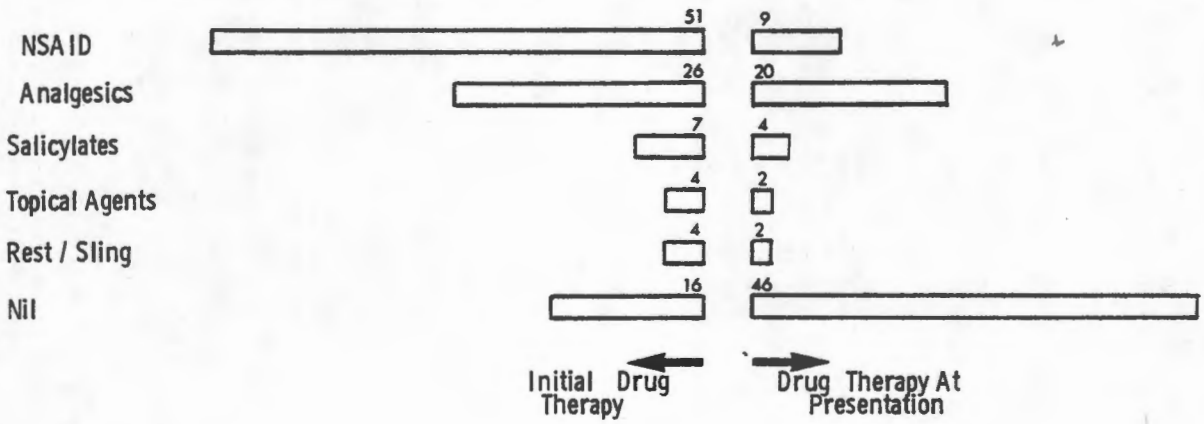
N/S = Not Significant



F I G U R E 7 : I. The distribution of frozen shoulder patients according to age, sex and duration of symptoms at presentation.



F I G U R E 7 : 2. Initial treatment prescribed by general practitioners. (NSAID = non-steroidal anti-inflammatory drug).



F I G U R E 7 : 3. Treatment being taken at the first out-patient visit. (NSAID = non-steroidal anti-inflammatory drug).

CHAPTER 8

FROZEN SHOULDER DURING THE FIRST 8 MONTHS FOLLOW-UP

Few studies have considered the rate of recovery of the frozen shoulder and the factors which may affect this. Lee et al (153) reported the use of multivariate analysis to simplify analysis of the recovery of the range of movement in different treatment groups. As the patients in the Major and Supplementary Frozen Shoulder Groups differed slightly with regard to entry criteria and timing of follow-up visits they will be considered separately. Therapy will be ignored but where a parameter was unevenly distributed amongst the treatment groups, it will not be considered.

ASSESSMENT OF RECOVERY The two main symptoms in the frozen shoulder are pain and restriction of range.

Pain: The severity of pain at night, at rest and on movement was assessed using 10 cm horizontal visual analogue scales (VAS) as previously described. To simplify the analysis of improvement in pain, a 'pain score' was formulated from the sum of these 3 VAS's. The mean difference between consecutive visits was then used to plot recovery curves.

In addition to the VAS's - the other parameters to assess pain were the duration before (a) patients felt they were much improved
(b) able to sleep on the affected shoulder
(c) lost pain on resisted movements
(d) stopped using analgesics.

Range of Movement: The recovery in the passive range of movement between consecutive visits was analysed using multivariate analysis. The principal component (C) was based on the range of movement at the initial visit. As this range was different in the Major and Supplementary Frozen Shoulder groups, different formulae were calculated for the two studies. In the Major Frozen Shoulder Study, the principal component (C) = (0.536 x total flexion) + (0.201 x glenohumeral flexion) + (0.679 x total abduction) + (0.263 x glenohumeral abduction) + (0.079 x external rotation) + (0.369 x total rotation) - 137.7. C accounted for 58.6% of the total variation in range between the patients at the initial visit.

In the Supplementary Frozen Shoulder Study group, C = (0.506 x TF) + (0.215 x GF) + (0.583 x TA) + (0.253 x GA) + (0.124 x ER) + (0.528 x TR) - 163.388. In this study, C accounted for 57.25% of the variation between patients at the initial visit. The three major components - TF, TA and TR were therefore very comparable in the 2 studies. Using the respective formulae, the principal component was calculated for each patient at each visit. From this data the difference between consecutive visits was found. Recovery curves were then plotted from the mean cumulative change, so a higher value of C signified an improvement and a lower value of C, a deterioration in the passive range of movement.

Follow-up: The Major Frozen Shoulder patients were seen weekly for 6 weeks and then monthly for a further 6 months. The Supplementary Frozen Shoulder patients were seen fortnightly for the first 6 weeks and then monthly for a further 6 months.

RESULTS

A. The Improvement in Pain: The mean reduction in pain at night, at rest and on movement was similar in the Major (Figure 8:1) and Supplementary (Figure 8:2) Frozen Shoulder Study patients with the maximum improvement occurring within 6 weeks of inclusion in the studies. Further improvement continued until the end of the follow-up period.

52 of the 82 patients (63%) reported the pain as 'much improved' by 3 months and 69 (85%) by 6 months (Figure 8:3). 8 patients (10%) still regarded the pain as severe even after 8 months follow-up. With an end point of 8 months (the end of the study), the mean duration (± SEM) before marked improvement was reported was 3.3 (± 0.4) months in the Major and 3.1 (± 0.4) months in the Supplementary Frozen Shoulder Study groups. Although the duration before patients were able to lie on the affected shoulder also varied greatly from patient to patient (Figure 8:4), this was very similar to when the patients reported the pain as 'much improved'. The mean duration (± SEM) before patients were able to sleep on the affected shoulder was 2.7 (± 0.4) months in the Major and 3.0 (± 0.3) months in the Supplementary groups. The loss of pain on resisted movement mirrored exactly the time before patients were able to sleep on the affected shoulder.

32 patients (39%) did not use any paracetamol during the study. The other 50 patients (61%) used the analgesic tablets for variable periods but mainly during the first 6 weeks of follow-up (Figure 8:5). 30 of these patients

only used the paracetamol before retiring to facilitate sleep and the other 20 also used analgesics during the day. 14 patients (17%) also requested diazepam at night to aid sleep. 2 patients regarded their NSAIDS tablets as effective in reducing pain and restarted this therapy. The use of analgesics and diazepam showed no association with the severity of pain and probably reflected the patients attitudes to this therapy.

B. The Recovery of Range: The mean recovery in the range of the individual movement parameters was similar in the Major (Figure 8:6) and Supplementary Study (Figure 8:7) patients, with the most rapid improvement in the first 6 weeks, but further recovery continuing throughout the entire period of follow-up. The improvement in the glenohumeral movements and external rotation showed greater variability between the 2 studies.

CLINICAL FACTORS WHICH POSSIBLY INFLUENCE THE RATE OF RECOVERY: The importance of age, sex, arm dominance, occupation and duration of symptoms at presentation on the rate of recovery was considered. Whether patients with a traumatic onset behaved differently from those arising spontaneously was also assessed. Each parameter was considered both with regard to the reduction in pain and improvement in range between consecutive visits in the both studies. To simplify these analyses, pain was assessed using the pain score and range using the cumulative principal component (ΔC). Wilcoxon rank sum tests were used for comparisons of the rate of recovery.

Age: Patients aged 55 or less at presentation were compared to those aged 56 or more. The older patients showed a small advantage in reduction of pain (Figure 8:8 and 8:9) and improvement in range (Figure 8:10 and 8:11) within the first 6 weeks, but no difference thereafter.

Sex: The reduction in pain (Figure 8:12 and 8:13) and improvement in range (Figure 8:14 and 8:15) showed no consistent advantage for the male or female patients in either study.

Duration at Presentation: Patients who presented within 3 months of the onset of symptoms showed a better initial reduction in the pain score when compared to patients who presented later in both the Major (Figure 8:16) and Supplementary (Figure 8:17) studies. The initial improvement in range was also better in patients who presented early in the Major (Figure 8:18) but not the Supplementary (Figure 8:19) study. Wilcoxon rank sum tests did not show the difference in the rate of recovery of pain or range to be significant.

Arm Dominance: There was no significant difference in the rate of recovery in patients with dominant or non-dominant arm involvement (Figure 8:20-23), although the initial improvement in range was more rapid in patients with dominant arm involvement in the Major Frozen Shoulder Study.

Occupation: The patients who resumed manual labour in the convalescent phase recovered as rapidly as the sedentary workers with regard to reduction in pain (Figure 8:24), but improvement in range (Figure 8:25) was slower after 3 months in the manual labourers in the Major Study. As the manual workers were unevenly distributed amongst the treatment groups in the Supplementary Study (only 2 being in the treated group), this parameter was not considered in this study.

Type of Onset: Major Frozen Shoulder patients with a traumatic onset showed some initial advantage in the reduction in pain (Figure 8:26) and the recovery of range (Figure 8:27). However, by 3 months no difference was found between the groups. Patients with a traumatic onset presented earlier (mean 3.3 months) than those with a spontaneous onset (mean 5.9 months) and this may have accounted for at least some of the initial advantage of the traumatic group. As only 4 patients in the Supplementary Study had a traumatic onset, this parameter was not considered in this study.

DISCUSSION

The rate of recovery of the pain score and the range of movement within the first 8 months was similar in the Major and Supplementary Study patients despite differences in the entrance criteria and therapy. Both parameters showed the most rapid improvement within the first 6 weeks after inclusion in the studies, with continuing recovery until the end of the follow-up period. This supports Sheldon's (246) observation of a striking consistency in the time of recovery from presentation, irrespective of the duration of symptoms at that time.

Because of the difficulty many patients experienced in transcribing the verbal descriptions of pain to the visual analogue scales, it was not felt reasonable to use this parameter in isolation to determine the improvement in pain. The duration before the pain was reported to be 'much improved', the ability to lie on the affected shoulder was regained and pain on resisted movements had been lost, showed remarkable consistency in most cases. Thirty-eight patients (46% of all cases) were 'much improved' and 44 (54%) were able to sleep on the affected side and had lost the pain on

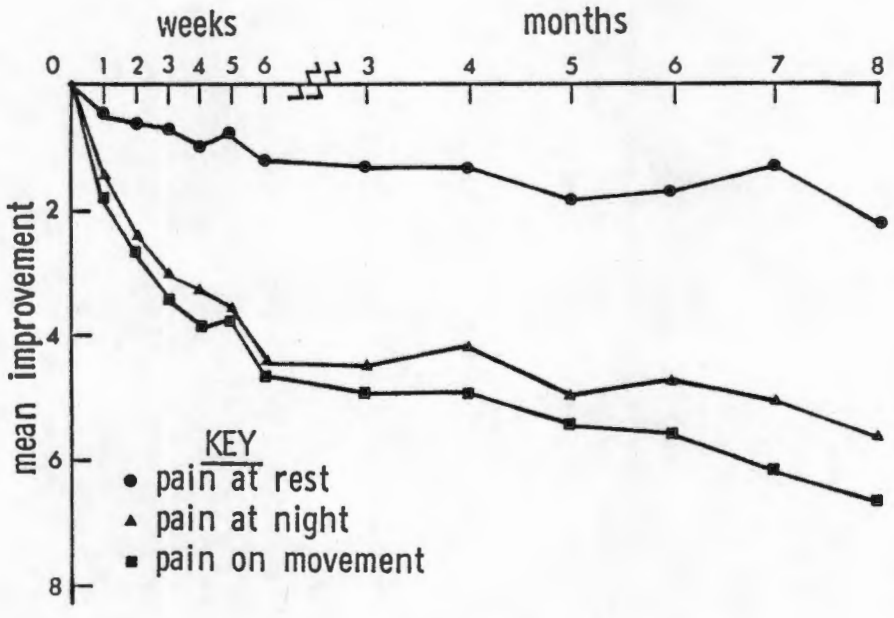
resisted movements by 6 weeks. However, 15 patients (18%) still had severe pain at 6 months and this persisted in 8 case (10%) until the end of the follow-up period. The analgesic consumption did not reflect the severity of the pain, but more often the personality and individual preference of the patient.

Multivariate analysis simplified the assessment of the recovery of the range of movement and accurately reflected the rate of improvement of the individual movement parameters. This supports the observation by Lee et al (153) and Clarke et al (42) that there is a constant relationship between all movements in the PSS. The continuing improvement in the range of movement after 8 months suggests that longer follow-up is necessary to determine the eventual outcome.

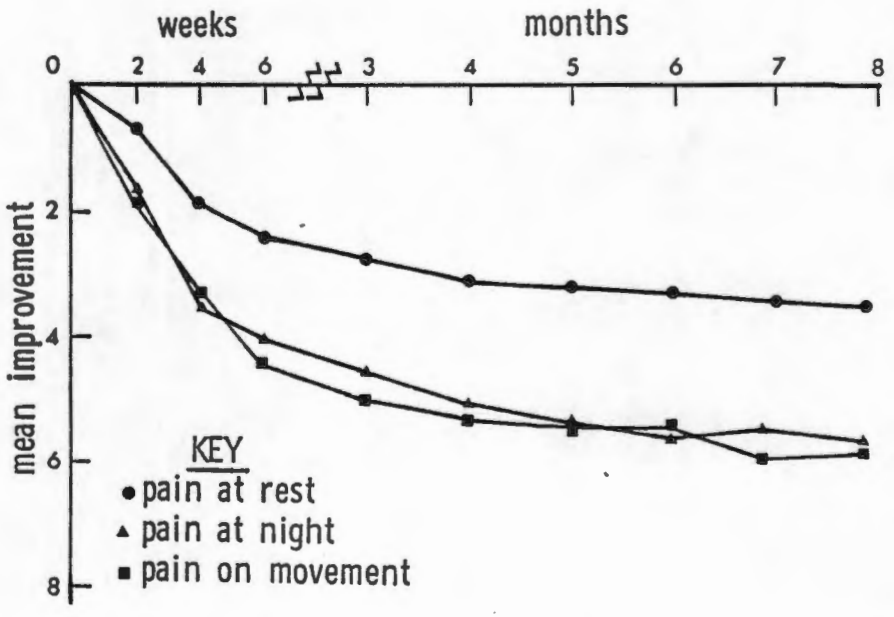
There is a paucity of data on the factors which influence the rate of recovery of the PSS. Patients with a traumatic onset of symptoms showed a slight initial advantage in the rate of recovery when compared to those with a spontaneous onset. This may simply reflect the earlier presentation of these patients, for like Hazleman (113) and Roy and Oldham (238), early presentation was associated with a slightly better initial rate of recovery. However, this study has not confirmed (226) that the post-traumatic lesions have a radically different outcome. Clarke et al (42) reported that the dominant arm improved more slowly, but like Lundberg (168) this was not found. The patients aged 56 or more showed a slight advantage in the initial period, but this difference was not sustained after 3 months.

Pain and loss of range of movement are the two cardinal features of the PSS and the rate of improvement of these during the first 8 months of follow-up has been carefully documented with a consideration of the factors which may influence this recovery.

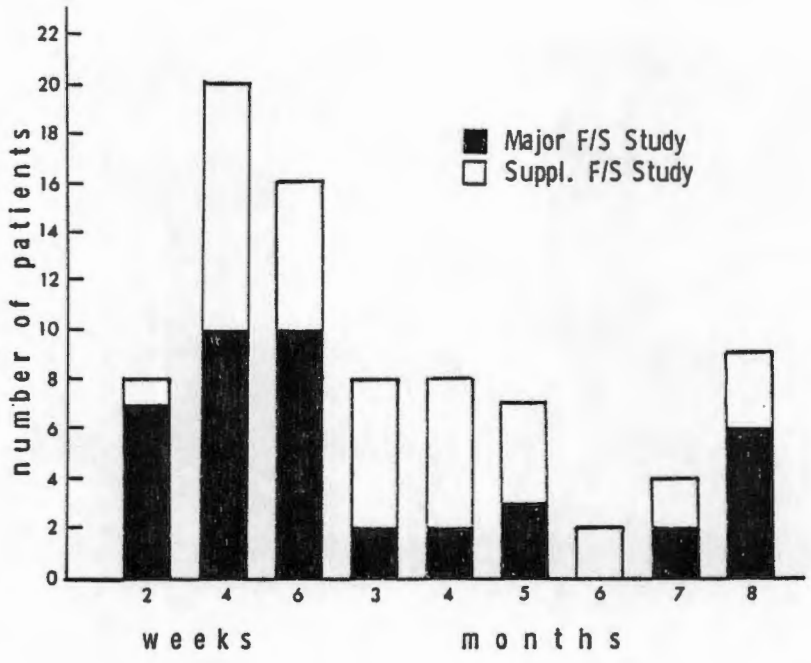
F I G U R E S 8 : I - 2. THE IMPROVEMENT IN THE PAIN PARAMETERS IN THE FIRST 8 MONTHS IN THE MAJOR AND SUPPLEMENTARY FROZEN SHOULDER STUDIES.



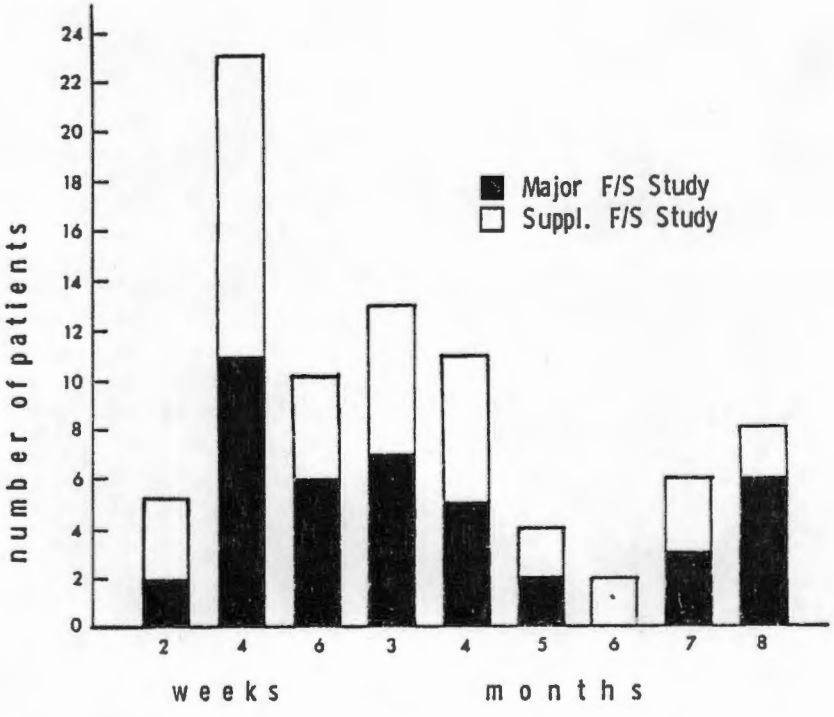
F I G U R E 8 : I. The Major Frozen Shoulder Study.



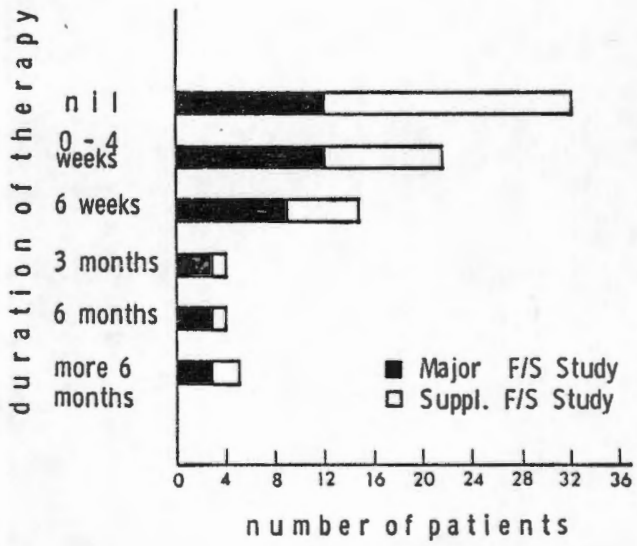
F I G U R E 8 : 2. The Supplementary Frozen Shoulder Study.



F I G U R E 8 : 3. Duration before patients considered the shoulder pain to be 'Much Improved'.

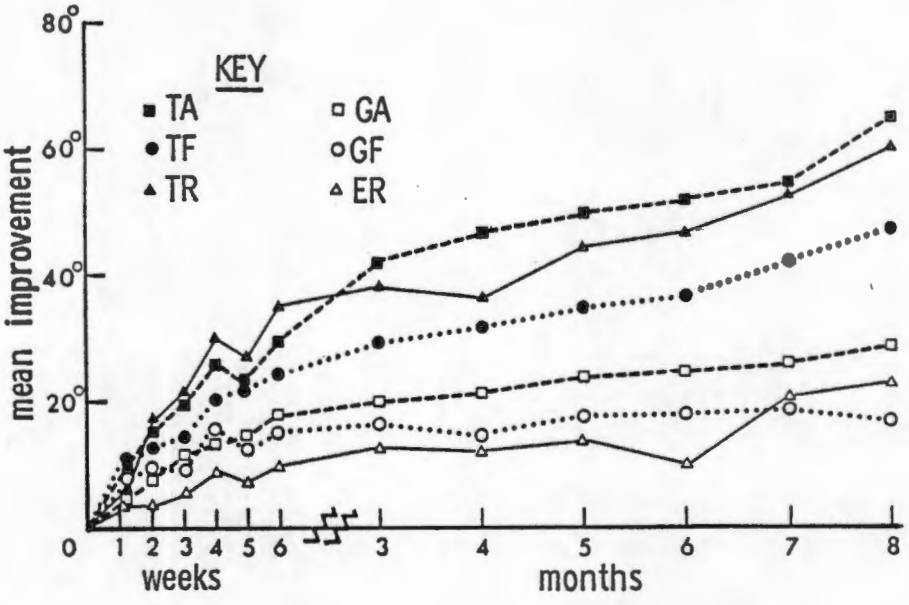


F I G U R E 8 : 4. Duration before patients were able to sleep on the affected shoulder.

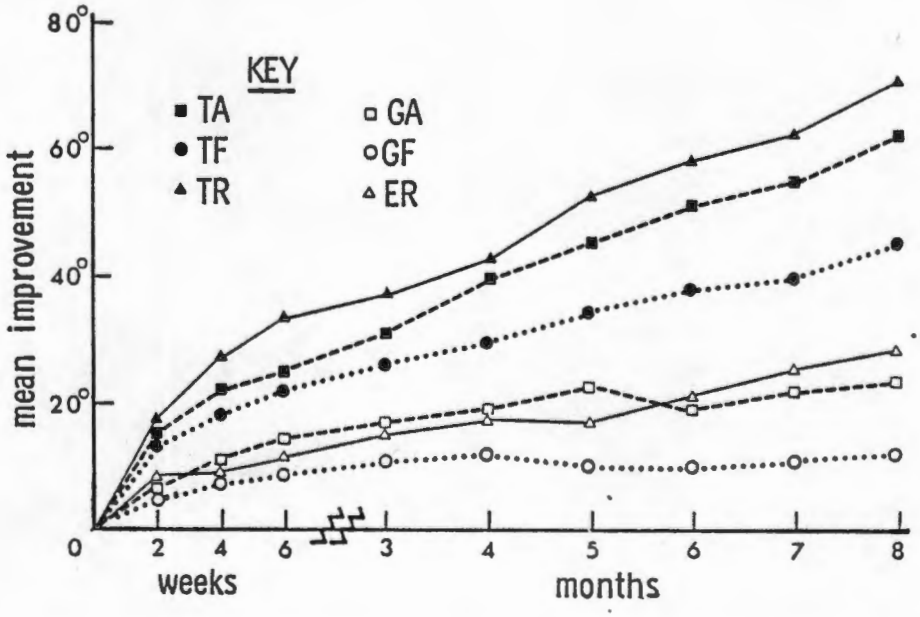


F I G U R E 8 : 5. Duration of analgesic use following inclusion in the Major and Supplementary Frozen Shoulder Studies.

F I G U R E S 8 : 6 - 7. THE MEAN RECOVERY IN INDIVIDUAL MOVEMENT PARAMETERS IN THE MAJOR AND SUPPLEMENTARY FROZEN SHOULDER STUDIES.



F I G U R E 8 : 6. The Major Frozen Shoulder Study.



F I G U R E 8 : 7. The Supplementary Frozen Shoulder Study.

F I G U R E S 8 : 8 - 27. THE INFLUENCE OF CLINICAL FACTORS AT PRESENTATION ON THE RATE OF RECOVERY OF THE PAIN SCORE AND RANGE OF MOVEMENT (ΔC) IN THE FROZEN SHOULDER STUDIES.

A) PATIENT AGE : IMPROVEMENT IN PAIN SCORE.

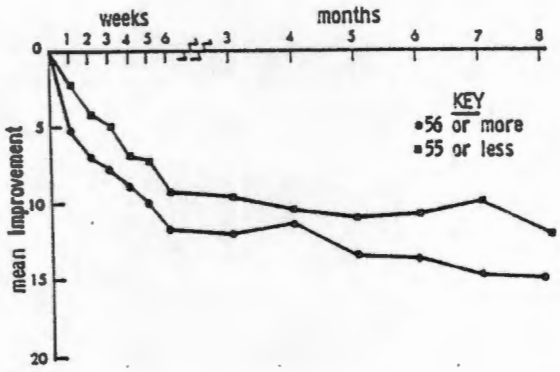


FIGURE 8 : 8. Major Frozen Shoulder Study.

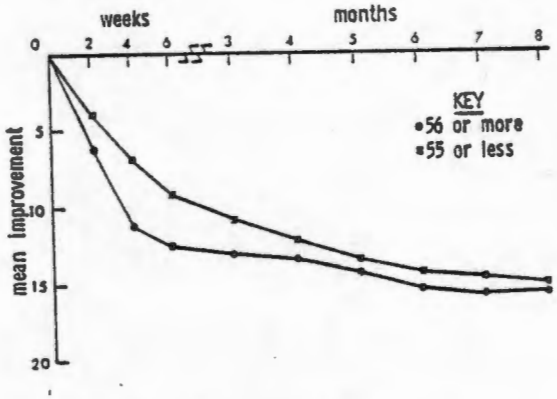


FIGURE 8 : 9. Supplementary Frozen Shoulder Study.

PATIENT AGE : IMPROVEMENT IN RANGE OF MOVEMENT (ΔC).

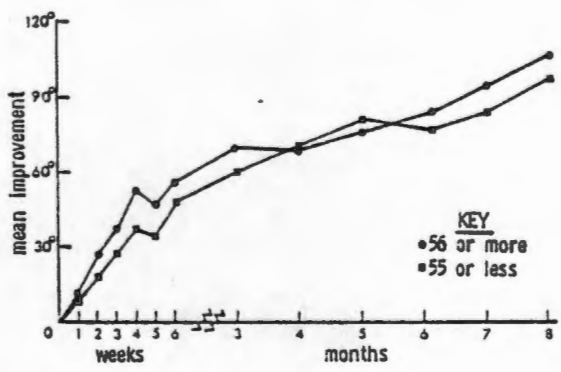


FIGURE 8 : 10. Major Frozen Shoulder Study.

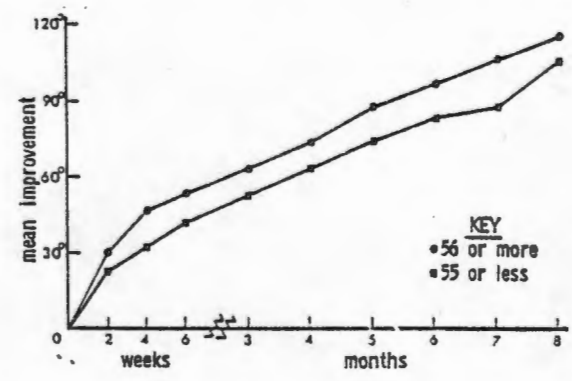


FIGURE 8 : 11. Supplementary Frozen Shoulder Study.

B) PATIENT SEX : IMPROVEMENT IN PAIN SCORE.

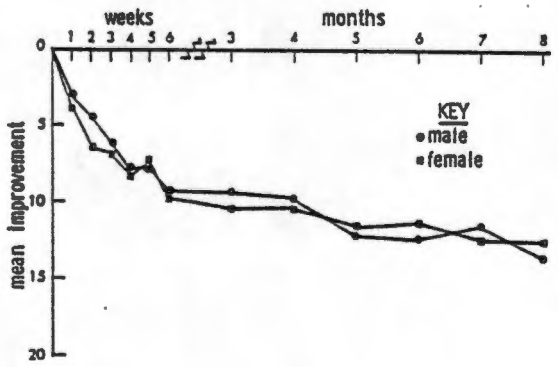


FIGURE 8 : I2. Major Frozen Shoulder Study.

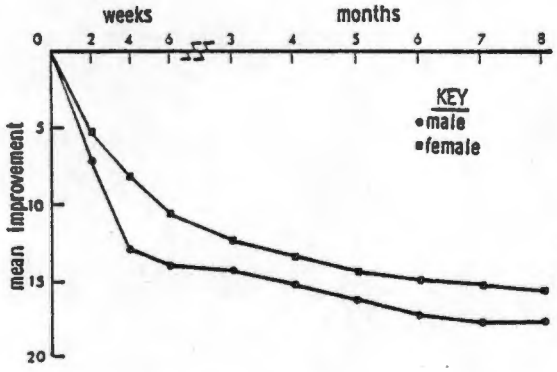


FIGURE 8 : I3. Supplementary Frozen Shoulder Study.

PATIENT SEX : IMPROVEMENT IN RANGE OF MOVEMENT (ΔC).

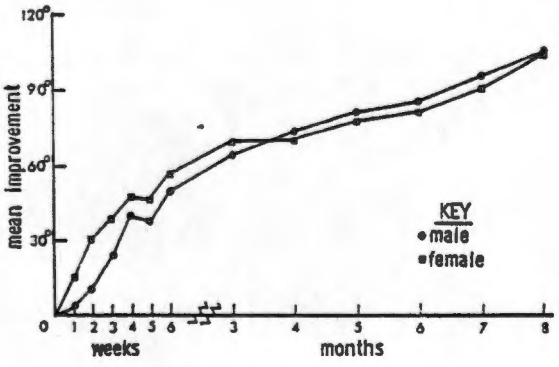


FIGURE 8 : I4. Major Frozen Shoulder Study.

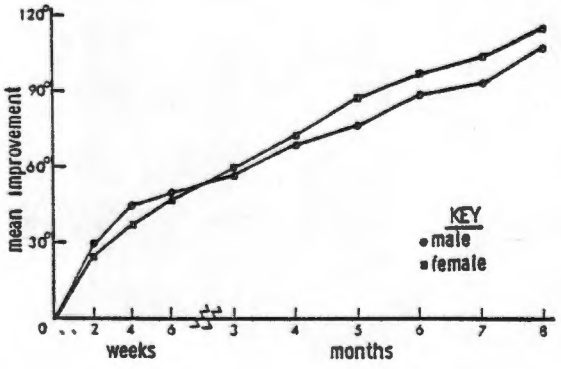


FIGURE 8 : I5. Supplementary Frozen Shoulder Study.

C) DURATION AT PRESENTATION : IMPROVEMENT IN PAIN SCORE.

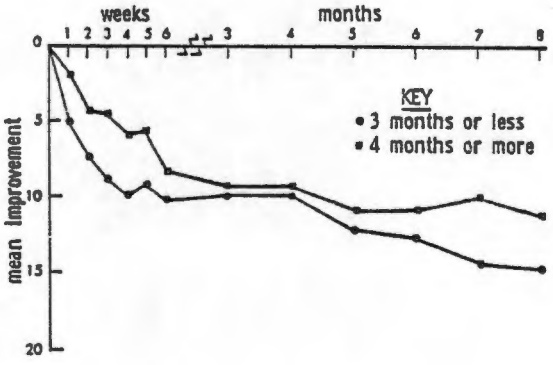


FIGURE 8 : I6. Major Frozen Shoulder Study.

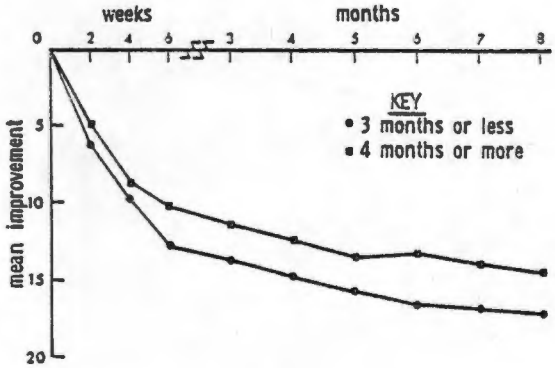


FIGURE 8 : I7. Supplementary Frozen Shoulder Study.

DURATION AT PRESENTATION : IMPROVEMENT IN RANGE OF MOVEMENT (ΔC).

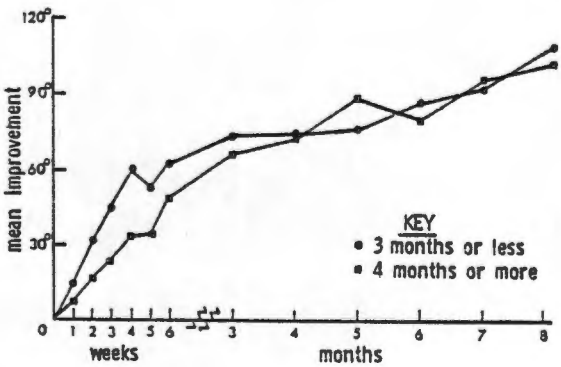


FIGURE 8 : I8. Major Frozen Shoulder Study.

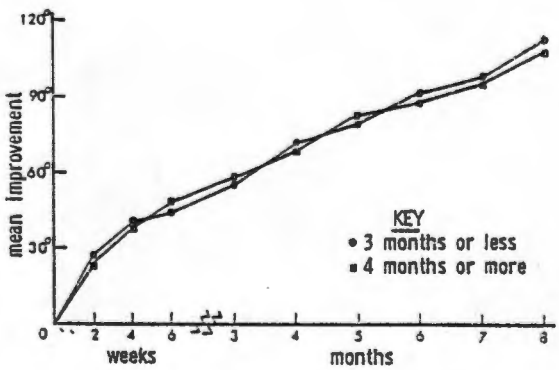


FIGURE 8 : I9. Supplementary Frozen Shoulder Study.

D) ARM DOMINANCE : IMPROVEMENT IN PAIN SCORE.

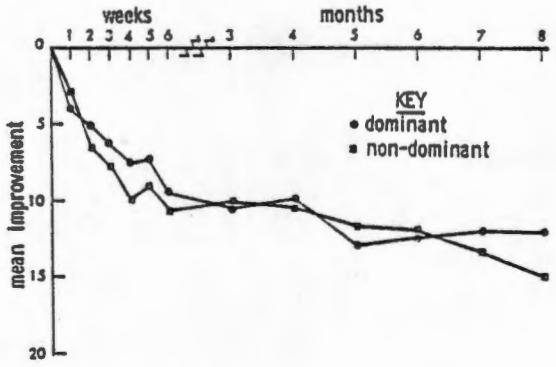


FIGURE 8 : 20. Major Frozen Shoulder Study.

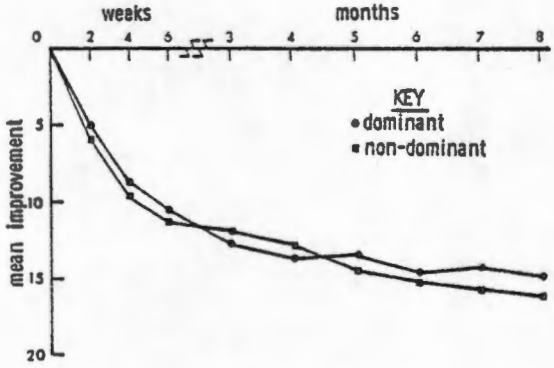


FIGURE 8 : 2I. Supplementary Frozen Shoulder Study.

ARM DOMINANCE : IMPROVEMENT IN RANGE OF MOVEMENT (ΔC).

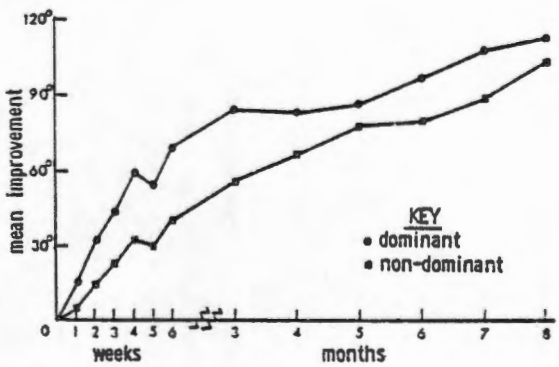


FIGURE 8 : 22. Major Frozen Shoulder Study.

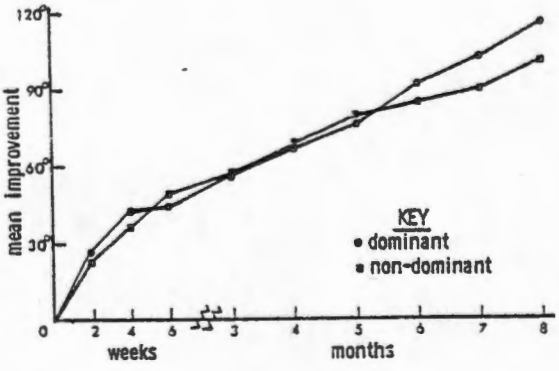


FIGURE 8 : 23. Supplementary Frozen Shoulder Study.

E) PATIENT OCCUPATION :

IMPROVEMENT IN PAIN SCORE.

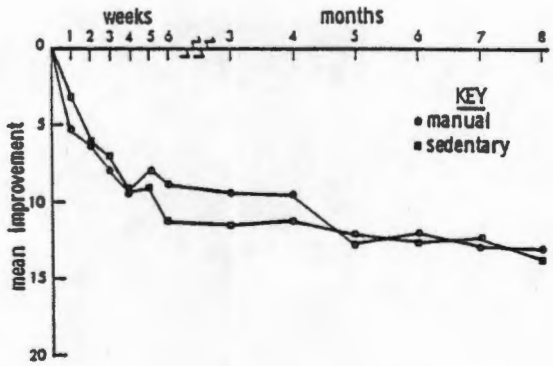


FIGURE 8 : 24. Major Frozen Shoulder Study.

IMPROVEMENT IN RANGE OF MOVEMENT (ΔC).

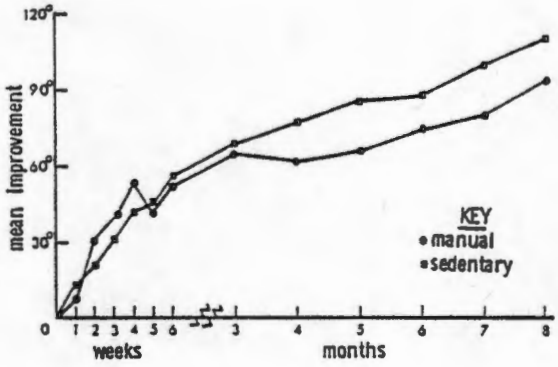


FIGURE 8 : 25. Major Frozen Shoulder Study.

F) MODE OF ONSET OF SYMPTOMS :

IMPROVEMENT IN PAIN SCORE.

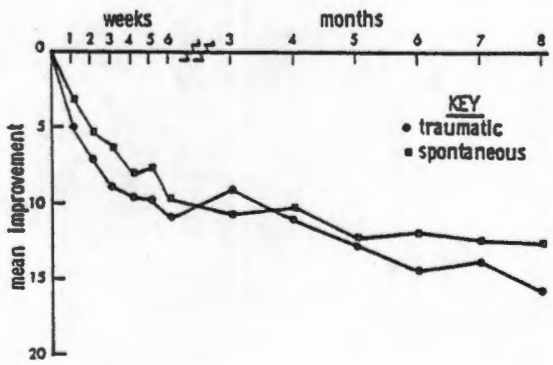


FIGURE 8 : 26. Major Frozen Shoulder Study.

IMPROVEMENT IN RANGE OF MOVEMENT (ΔC).

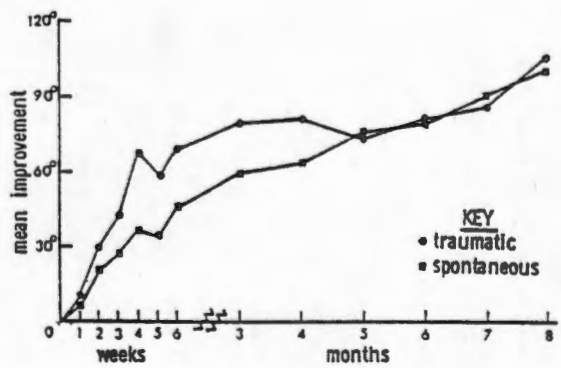


FIGURE 8 : 27. Major Frozen Shoulder Study.

CHAPTER 9

FROZEN SHOULDER AT 8 MONTHS

The natural history of the frozen shoulder is poorly documented. While Watson-Jones (272) reported that less than 5% of his 226 patients with frozen shoulder had any disability at 6 months, Simmonds (248), Clarke et al (42) and Reeves (228) found severe and persistent disability many years later.

PATIENTS

The 82 frozen shoulder patients in the Major and Supplementary Studies remained under regular follow-up for 8 months and the extent of their disability when compared to the normal controls (right shoulder) was determined at the end of this period. To permit further detailed comparative analysis of the Major Study patients who remained under review, the 2 study groups will be considered separately, again ignoring therapy.

RESULTS

Pain: Thirty-three of the 82 patients (40%) still had some residual pain at 8 months, although this was worse than at presentation in only 1 patient. Of these 33 patients, 17 were in the Major and 16 in the Supplementary group. The number of patients with residual pain at night, at rest and on movement was similar in the 2 studies (Table 9:1). Only considering the 33 patients with residual pain at 8 months, the severity of pain at night and on movement was similar in both studies (Table 9:2). However, pain at rest was significantly worse (unpaired t-tests, $p<0.05$) in the Major Frozen Shoulder group. 8 patients (6 Major, 2 Supplementary) still considered the pain severe (Figure 8:3) and 9 (6 Major, 3 Supplementary) were not yet able to sleep on the affected shoulder (Figure 8:4). All had pain at night and 7 also pain on movement. 5 were taking regular analgesics and 3 nocturnal diazepam.

Range: The mean and the maximum and minimum ranges of each movement parameter in the normal controls and the Major and Supplementary Frozen Shoulder groups at 8 months is shown (Table 9:3). A comparison of the range of movement in the controls with the Major Frozen Shoulder Group (Table 9:4) and the Supplementary Frozen Shoulder Group (Table 9:5), using unpaired

t-tests, showed a highly significant advantage ($p<0.0001$) for the controls for all the movement parameters in each study.

When the range in the patients at 8 months was compared to the controls matched for age (in decades) and sex, over 50% of the patients had not attained a range equal to the minimum of the (age, sex) matched controls in any movement parameter except glenohumeral flexion and abduction (Table 9:6). Using the cumulative principal component at 8 months, 73 of the 82 patients (89%) had not attained a range equal to the minimum of the matched controls. However, only 45 of the 82 patients (55%) were aware of this persisting restriction in range.

The range of movement at 8 months was better in the Supplementary than the Major Study (Table 9:7), but the difference between the groups was only significant (unpaired t-tests) for total rotation ($p<0.05$). The recovery in range from presentation to 8 months (Table 9:8) showed a slight advantage for the Major Study, although only with glenohumeral flexion did the difference reach significance ($p<0.05$).

Clinical Factors Which Possibly Influence The Outcome: To simplify the assessment of a possible influence of clinical factors on the outcome, the pain score (sum of pain at rest, at night and on movement) and the principal component (ΔC) was used. There was no association between the age, sex, arm dominance, type of onset (traumatic or spontaneous) or the duration of symptoms at presentation and either the pain score or range (ΔC) at 8 months in either study (Table 9:9). However, the Major Frozen Shoulder Study patients who resumed manual labour in the convalescent phase had a significantly lower principal component at 8 months than the sedentary group. Significant restriction in all movements except total rotation was also evident at that time (Table 9:10). The pain score was also higher in the manual workers, but the difference did not reach significance. The Supplementary Study showed a similar result, but detailed analysis was not carried out as the manual and sedentary workers were not evenly distributed in the treatment groups, and a possible bias from this source could not be excluded.

DISCUSSION

Thirty-three of the patients (40%) still had some pain at 8 months, but in only 8 (10%) was this severe. Restriction of the range of movement on the other hand was present in 73 patients (89%), when compared to the minimum

range of the controls matched for age and sex. Objective measurements of range showed a highly significant ($p<0.0001$) restriction in all the movement parameters when compared to the controls, refuting Watson-Jones's (272) contention that 95% of frozen shoulder patients regain a normal range by 6 months. Like Allander et al (2), Clarke et al (42) and Hazleman (113), many of the patients who claimed they have regained full movement were found to have marked restriction on objective testing.

The initial advantage in the rate of recovery within the first 6 weeks found in association with older patients, early presentation and lesions following trauma conveyed no long-term benefits to these patients. The only factor associated with a significantly worse outcome at 8 months was the resumption of manual labour in the convalescent phase. While these patients were no worse at presentation or at 6 weeks, they showed a slower rate of recovery from three months until the end of the study (Figure 8:25). This suggests that excessive use of the shoulder in the convalescent phase may be harmful.

TABLE 9:1

NUMBER OF FROZEN SHOULDER PATIENTS WITH RESIDUAL PAIN AT 8 MONTHS

	Patients with Residual Pain	
	Major F/S Study (N = 17)	Supplementary F/S Study (N = 16)
Pain only at night	2	2
Pain only on movement	5	6
Pain at night and on movement	2	5
Pain at rest, at night and on movement	8*	3

* = 1 patient worse than at presentation

TABLE 9:2

COMPARISON OF THE MEAN SEVERITY OF PAIN AT 8 MONTHS IN THE 33 PATIENTS WITH RESIDUAL SYMPTOMS IN THE MAJOR AND SUPPLEMENTARY FROZEN SHOULDER GROUPS

	Mean Severity of Pain		Unpaired T-test	
	Major F/S Study (N = 17) Mean (SEM)	Supplementary F/S Study (N = 16) Mean (SEM)	T_{31}	p<
Pain at rest	1.06 (0.3)	0.25 (0.15)	2.157	0.05
Pain at night	1.29 (0.3)	1.25 (0.3)	0.101	N/S
Pain on movement	2.41 (0.5)	1.5 (0.25)	1.693	N/S

N/S = Not Significant

TABLE 9:3

MEAN, MAXIMUM AND MINIMUM RANGES OF MOVEMENT IN CONTROLS AND PATIENTS IN THE MAJOR AND SUPPLEMENTARY FROZEN SHOULDER STUDIES AT 8 MONTHS

	Major F/S Study at 8 Months (N = 42)		Control Group (N = 76)		Supplementary F/S Group at 8 Months (N = 40)	
	Mean	(Range)	Mean	(Range)	Mean	(Range)
TF	144°	(80-160)	167°	(140-180)	150°	(110-175)
GF	74°	(50- 90)	87°	(60-100)	77°	(70- 85)
TA	135°	(60-175)	163°	(140-180)	140°	(90-170)
GA	69°	(30- 85)	79°	(50-100)	72°	(40- 85)
ER	41°	(0- 70)	62°	(40- 90)	43°	(25- 80)
TR	100°	(50-160)	162°	(135-185)	116°	(65-165)

TABLE 9:4

COMPARISON OF RANGE IN THE MAJOR FROZEN SHOULDER STUDY PATIENTS AT 8 MONTHS (M) AND THE CONTROLS (C).

	Mean Difference in Range (C - M)	Unpaired T-test T_{116}	$p<$
TF	22°	11.984	.0001
GF	13°	7.474	.0001
TA	28°	10.982	.0001
GA	10°	6.895	.0001
ER	21°	9.142	.0001
TR	61°	21.746	.0001

TABLE 9:5

COMPARISON OF RANGE IN THE SUPPLEMENTARY FROZEN SHOULDER STUDY PATIENTS AT 8 MONTHS (S) AND THE CONTROLS (C)

	Mean Difference in Range (C - S)	Unpaired T-test T_{114}	$p<$
TF	17^{o}	8.848	.0001
GF	10^{o}	6.840	.0001
TA	22^{o}	10.947	.0001
GA	7^{o}	4.562	.001
ER	9^{o}	7.926	.0001
TR	46^{o}	17.473	.0001

TABLE 9:6

PERCENTAGE OF PATIENTS IN THE MAJOR AND SUPPLEMENTARY FROZEN SHOULDER STUDIES WITH AN 8 MONTH RANGE LESS THAN THE MINIMUM FOR AGE (IN DECADES) AND SEX-MATCHED CONTROLS

	Major F/S Study (N = 42)		Supplementary F/S Study (N = 40)	
	N	%	N	%
TF	27	64%	22	55%
GF	18	43%	9	23%
TA	24	57%	27	68%
GA	12	29%	7	18%
ER	24	57%	20	57%
TR	39	93%	34	85%

TABLE 9:7

COMPARISON OF THE RANGE OF MOVEMENT AT 8 MONTHS IN PATIENTS IN THE SUPPLEMENTARY (S) AND MAJOR (M) FROZEN SHOULDER GROUPS

	Mean Difference in Range (S minus M)	Unpaired T-test T_{80}	p<
TF	6^{o}	1.680	N/S
GF	3^{o}	1.397	N/S
TA	6^{o}	1.174	N/S
GA	3^{o}	1.423	N/S
ER	2^{o}	1.354	N/S
TR	16^{o}	2.263	.05

N/S = Not Significant

TABLE 9:8

COMPARISON OF MEAN RECOVERY IN THE RANGE OF MOVEMENT FROM PRESENTATION TO 8 MONTHS IN THE SUPPLEMENTARY (S) AND MAJOR (M) FROZEN SHOULDER STUDIES

	Mean Difference in Recovery (M - S)	Unpaired T-test T_{80}	p<
TF	4^{o}	0.935	N/S
GF	8^{o}	2.362	.05
TA	9^{o}	1.491	N/S
GA	2^{o}	0.657	N/S
ER	6^{o}	1.260	N/S
TR	1^{o}	0.374	N/S

N/S = Not Significant

TABLE 9:9

ASSESSMENT OF THE EFFECT OF CLINICAL PARAMETERS ON THE PAIN SCORE AND PRINCIPAL COMPONENT AT 8 MONTHS IN THE 2 FROZEN SHOULDER STUDIES (UNPAIRED T-TEST COMPARISONS)

MAJOR FROZEN SHOULDER STUDY

CLINICAL PARAMETER	N	PAIN SCORE Mean	PAIN SCORE SD	PRINCIPAL COMPONENT Mean	PRINCIPAL COMPONENT SD
A. AGE:					
55 years or less	21	1.3	3.5	107.8	30.5
56 years or more	21	2.6	3.2	100.4	38.6
		$T = 1.254^{+}$		$T = 0.685^{+}$	
B. SEX:					
Male	14	1.8	2.7	101.0	27.8
Female	28	2.0	3.7	105.6	37.9
		$T = 0.193^{+}$		$T = 0.387^{+}$	
C. ARM:					
Dominant	22	2.2	3.5	109.0	35.4
Non-dominant	20	1.6	3.3	98.8	33.6
		$T = 0.602^{+}$		$T = 0.956^{+}$	
D. DURATION AT PRESENTATION:					
3 months or less	20	1.2	1.9	105.2	39.7
4 months or more	22	2.6	4.2	103.1	30.1
		$T = 1.360^{+}$		$T = 0.198^{+}$	
E. OCCUPATION:					
Manual	15	2.7	4.0	86.1	42.7
Sedentary	27	1.2	2.6	114.1	24.7
		$T = 1.518^{+}$		$T = 2.697^{*}$	
F. CAUSE:					
Traumatic	11	1.2	2.1	111.9	25.0
Spontaneous	31	2.2	3.7	101.3	37.3
		$T = 0.859^{+}$		$T = 0.869^{+}$	

* = $p<0.05$

+ = Not Significant

TABLE 9:9 (CONTINUED)

SUPPLEMENTARY FROZEN SHOULDER STUDY

	N	PAIN SCORE Mean	SD	PRINCIPAL COMPONENT Mean	SD
A. AGE:					
55 years or less	18	0.9	1.7	101.0	27.7
56 years or more	22	1.4	1.9	104.0	31.9
		$T = 0.835^{+}$		$T = 0.318^{+}$	
B. SEX:					
Male	16	1.0	1.8	103.1	24.6
Female	24	1.2	1.9	101.8	32.5
		$T = 0.354^{+}$		$T = 0.140^{+}$	
C. ARM:					
Dominant	13	0.9	1.8	104.0	21.0
Non-dominant	27	1.1	1.7	101.5	32.9
		$T = 0.260^{+}$		$T = 0.250^{+}$	
D. DURATION AT PRESENTATION:					
3 months or less	11	1.3	1.6	102.2	24.9
4 months or more	29	1.1	1.9	102.4	31.2
		$T = 0.387^{+}$		$T = 0.020^{+}$	
E. OCCUPATION:					
Manual	13	1.5	2.2	88.8	29.6
Sedentary	27	0.9	1.6	108.8	27.4
		$T = 1.008^{+}$		$T = 2.126^{*}$	

* = $p<0.05$

+ = Not Significant

TABLE 9:10

RANGE OF MOVEMENT AT 8 MONTHS IN MANUAL AND SEDENTARY WORKERS IN THE MAJOR FROZEN SHOULDER STUDY

	Mean Range Sedentary Workers (S=27)	Mean Range Manual Workers (M=15)	Mean Difference in Range (M - S)	Unpaired T-test T_{40}	p<
TF	148°	138°	10°	2.193	0.05
GF	76°	68°	8°	2.920	0.01
TA	142°	122°	20°	2.667	0.02
GA	72°	63°	9°	2.585	0.02
ER	45°	33°	11°	2.188	0.05
TR	100°	94°	7°	0.725	N/S

N/S = Not Significant

CHAPTER 10

FROZEN SHOULDER AFTER 3 - 4 YEARS

Hazleman (113) in a retrospective survey of 130 patients with painful stiff shoulder found 15% to have persistent disability. Simmonds (248) reported 15 of his 21 patients (71%) to be symptomatic after 3 years. Clarke et al (42) retrospectively reviewed 48 patients after a period of 6 years and found 20 (42%) to have some range deficit. A prospective 5 - 10 years study of 49 patients by Reeves (228) showed 3 patients to have severe and 22 mild persistent disability. Dickson and Crosby (66) and Meulengracht and Schwartz (179) also reported prolonged disability in many patients. On the other hand, Watson-Jones (272) reported that less than 5% of his 226 patients with frozen shoulder had any disability by 6 months, although simple pendular exercise was the only therapy given. Grey (99) also found recovery of 24 of 25 patients with "untreated" frozen shoulder followed up for 2 years. Withers (286), Haggart et al (105) and Lundberg (168) also reported a favourable outcome. These conflicting reports probably reflect on both patient selection and the criteria used for diagnosis and recovery. As recovery was not complete at 8 months, patients in the Major Frozen Shoulder Study were recalled after 3 -4 years to record the long-term outcome of the frozen shoulder and also to try to assess whether any clinical features at presentation influence this outcome.

PATIENTS

40 of the 42 Major Frozen Shoulder Study patients attended for an interview and re-examination 40-48 (mean 44) months after initial presentation. 1 patient could not be traced and the other was excluded as she had undergone mastectomy on the affected side 2 months before recall.

METHOD

At review, persistent or recurrent pain and/or restriction of movement was recorded. Symptoms in the opposite shoulder since discharge were also documented. The passive range of movement was measured using a spirit goniometer as previously described.

CONTROLS

The 76 normal people of similar age also provided a control group for passive range in each decade (40-70) in this study. Forty of the controls had been carefully matched for age and sex with the patients. Unpaired t-tests were used for statistical analysis unless otherwise stated.

RESULTS

Residual Symptoms: Although 18 patients (45%) had residual symptoms at review, these were only severe in 1 case (Table 10:1). The pain was most severe on shoulder movement - especially performing manual tasks, but 4 patients also had pain at night. Only the one patient with severe symptoms still had residual pain at rest and also inability to lie on the affected shoulder and was using regular analgesics. 7 patients (18%) had also developed pain in the opposite shoulder, 4 having a frozen shoulder.

Passive Range: Comparison of the mean range of passive movement at 'discharge' (8 months follow-up) with the mean range at 'review' (mean follow-up 44 months), using paired t-tests (Table 10:2) showed significant improvements in all movements, although 5 patients had shown some deterioration of range during this time. However, when the range at review was compared to the matched controls (Table 10:3), significant restriction in range of all movements except glenohumeral abduction and flexion was still present.

Of the 11 patients (28%) who considered their range restricted at review, only five were found to have this on objective testing (Table 10:1). However, many other patients who regarded their range as normal were found to have significant restriction. Sixteen of the 40 patients (40%) had still not attained a range of total flexion, abduction and rotation comparable with the minimum range of the controls of similar age (in decades from 40 - 70) and sex. In five of these patients the reduction in total range was still severe (more than 25%). Using the principal component as previously described, 18 patients (45%) still had a range below the minimum for the matched controls.

Age and Sex: The range in the male patients was less than in the females (Table 10:4) and showed a proportionately larger decrease with age, but the difference did not reach statistical significance and was similar to the differences reported in the Control Group. The older patients (56 or older)

had a better range of external and total rotation, but not total flexion or abduction when compared to the younger (55 or less) patients, but these differences were not significant (Table 10:5).

Arm Dominance: At review, the patients with non-dominant arm involvement had a better range than those with the dominant arm involved - the difference being significant (at the 5% level) for total abduction, total flexion and total rotation but not external rotation (Table 10:6). During the first 8 months, the dominant arm had consistently shown a better (but not significant) range. Comparison of the recovery from 8 months to review, showed that the accelerated recovery of the non-dominantly affected group was a later development (Table 10:7).

Manual Work: The patients who resumed manual labour or strenuous activity in the convalescent phase also had a more restricted range at review (Table 10:8). However, significant restriction was already evident at 8 months follow-up and there was no further differences in the recovery from 8 months to review (Table 10:9), so this constituted an earlier development.

Duration of Symptoms at Presentation: The 18 patients who presented early - within 3 months of the onset of the symptoms - showed no advantage over the rest who presented between 4 and 12 months, with regard to the rate or extent of recovery (Table 10:10).

Mode of Onset: The 11 patients who reported trivial trauma as a precipitating factor also showed no difference from those lesions which arose spontaneously (Table 10:11).

DISCUSSION

This study has concentrated on both the subjective and objective outcome after at least 3 years, as the long term prognosis in frozen shoulder remains uncertain.

The marked discrepancy between the patients subjective awareness of residual range deficit and measurable (objective) restriction when compared with a normal control group of similar age and sex has again been high-lighted. Only half of the patients (5/11) who regarded their range as abnormal were found to have any reduction in total range. However, 40% of the entire patient group (16/40) had some range deficit on objective measurement. While 1 patient complained of severe pain and restriction, 5 were considered

to have severe (greater than 25%) residual restriction. That so many patients with range deficit considered their recovery to be complete shows the excellent adaptation to this minor disability achieved by a majority of patients. This difference in subjective and objective assessment of recovery (2, 42, 113) and the variation in the diagnostic criteria for frozen shoulder and length of follow-up probably account for the conflicting reports with regard to prognosis.

This study has shown that although significant improvement continued after 8 months, the mean range after 3-4 years was still markedly less than matched controls.

The observations of Clarke et al (42) that age and sex affect the range of movement in both patients and controls has been confirmed. Dominant arm involvement was associated with a less satisfactory recovery, although interestingly this phenomenon was a later development. The resumption of manual labour in the convalescent phase may also retard the recovery. This study has not confirmed that the duration of symptoms before therapy (113) or the mode of onset (226) affect the long-term prognosis.

5 patients (13%) still had severe and 11 (28%) mild shoulder restriction on objective testing after 3 years. However, only 1 patient regarded symptoms as severe. This accords with the experience of Clarke et al (42) who retrospectively assessed their patients. The patients in this study were followed prospectively and subjected to close and detailed examination.

It was concluded that although the range of movement remains objectively restricted there is little functional impairment in the late stage of frozen shoulder.

TABLE 10:1

SYMPTOMS AT REVIEW IN AFFECTED SHOULDER (N = 40)

	No.	% of Total N.
Mild ache	7	18%
Mild restriction	4	10%
Mild ache and restriction	6	15%
Severe pain and restriction	1	3%

TABLE 10:2

IMPROVEMENT IN RANGE OF MOVEMENT FROM 8 MONTHS (DISCHARGE) TO REVIEW

Movement	At 8 Months Mean (Range)	At Review Mean (Range)	Mean Improvement From Discharge To Review	Paired T-test T_{38}	p<
TF	144° (80-160)	156° (90-175)	12°	4.525	0.0001
GF	69° (50- 80)	89° (60-100)	20°	6.254	0.0001
TA	134° (60-175)	146° (70-175)	12°	3.581	0.0009
GA	66° (30- 85)	80° (55- 90)	14°	5.363	0.0001
ER	40° (0- 70)	52° (15- 85)	12°	4.581	0.0001
TR	99° (50-140)	147° (60-180)	48°	11.353	0.0001

TABLE 10:3

COMPARISON OF RANGE IN PATIENTS AT REVIEW WITH MATCHED CONTROLS

Movement	At Review (R) Mean (Range)	Controls (C) Mean (Range)	Mean Difference (C - R)	Paired T-test T_{78}	p<
TF	156° (90-175)	167° (145-180)	11°	4.062	0.0002
GF	89° (60-100)	90° (65-105)	1°	0.901	N/S
TA	146° (70-175)	163° (140-180)	17°	5.291	0.0001
GA	80° (55- 90)	83° (60-100)	3°	1.132	N/S
ER	52° (15- 85)	65° (40- 90)	13°	3.671	0.0007
TR	147° (60-180)	164° (140-180)	17°	3.983	0.0003

N/S = Not Significant

TABLE 10:4

COMPARISON OF RANGE IN FEMALE AND MALE PATIENTS AT REVIEW

	Mean Range in Females (F=26)	Mean Range in Males (M=14)	Mean Difference (F - M)	Unpaired T-test T_{38}	p<
TF	157°	156°	0.8°	0.161	N/S
TA	148°	144°	4°	0.696	N/S
ER	54°	49°	5°	0.947	N/S
TR	151°	140°	11°	1.308	N/S

N/S = Not Significant

TABLE 10:5

COMPARISON OF THE RANGE AT REVIEW IN PATIENTS IN DIFFERENCE AGE GROUPS

	Mean Range in Patients Aged 55 or Less (Y = 20)	Mean Range in Patients Aged 56 or More (O = 20)	Mean Difference (Y - O)	Unpaired T-test T_{38}	p<
TF	159°	154°	6°	1.147	N/S
TA	150°	143°	7°	1.154	N/S
ER	49°	56°	-8°	1.482	N/S
TR	145°	150°	-6°	0.700	N/S

N/S = Not Significant

TABLE 10:6

COMPARISON OF THE RANGE AT REVIEW IN PATIENTS WITH DOMINANT AND NON-DOMINANT ARM INVOLVEMENT

	Mean (Range) in Non-Dominant Group (N-D=27)	Mean (Range) in Dominant Group (D=13)	Mean Difference (N-D - D)	Unpaired T-test T_{38}	p<
TF	161° (150-175)	152° (90-170)	10°	2.096	0.050
TA	153° (130-175)	140° (70-160)	13°	2.084	0.044
ER	56° (30- 85)	49° (15- 75)	7°	1.323	0.194*
TR	157° (140-180)	139° (60-175)	18°	2.043	0.050

* = Not Significant

TABLE 10:7

COMPARISON OF MEAN RECOVERY IN RANGE FROM DISCHARGE (8 MONTHS) TO REVIEW (44 MONTHS) IN PATIENTS WITH NON-DOMINANT AND DOMINANT ARM INVOLVEMENT

	Mean Recovery		Unpaired T-test	
	Non-Dominant	Dominant	T_{38}	p<
TF	19°	5°	2.743	0.009
TA	25°	0.7°	2.740	0.009
ER	17°	7°	1.944	0.073*
TR	57°	40°	2.421	0.05

* = Not Significant

TABLE 10:8

COMPARISON OF RANGE AT REVIEW IN MANUAL AND SEDENTARY WORKERS

	Mean (Range) in Sedentary Workers (S=15)	Mean (Range) in Manual Workers (M=25)	Mean Difference (S - M)	Unpaired T-test	
				T_{38}	p<
TF	164° (135-170)	149° (90-175)	15°	2.035	0.05
TA	152° (130-165)	138° (70-175)	14°	2.439	0.02
ER	57° (40- 85)	44° (15- 70)	13°	2.687	0.01
TR	156° (125-180)	132° (60-165)	24°	2.877	0.01

TABLE 10:9

MEAN RECOVERY IN RANGE FROM DISCHARGE (8 MONTHS) TO REVIEW (44 MONTHS) IN MANUAL AND SEDENTARY WORKERS

	Mean Recovery		Unpaired T-test	
	Sedentary Workers (N = 15)	Manual Workers (N = 25)	T_{38}	p<
TF	16°	11°	0.270	N/S
TA	10°	16°	-0.658	N/S
ER	12°	11°	0.323	N/S
TR	55°	38°	1.855	N/S

N/S = Not Significant

TABLE 10:10

COMPARISON OF RANGE AT REVIEW IN PATIENTS PRESENTING EARLY (1-3 MONTHS) OR LATE (4-12 MONTHS)

	Mean Range		Unpaired T-test	
	Early Presentation (N = 18)	Late Presentation (N = 22)	T_{38}	p<
TF	155°	157°	0.361	N/S
TA	145°	147°	0.331	N/S
ER	57°	49°	1.585	N/S
TR	152°	143°	1.158	N/S

N/S = Not Significant

TABLE 10:11

COMPARISON OF RANGE AT REVIEW IN PATIENTS WITH A SPONTANEOUS (S) OR TRAUMATIC (T) ONSET

	Mean Range		Unpaired T-test	
	Spontaneous Onset (N = 29)	Traumatic Onset (N = 11)	T_{38}	$p<$
TF	157°	156°	0.178	N/S
TA	146°	144°	0.316	N/S
ER	58°	50°	1.304	N/S
TR	150°	140°	1.152	N/S

N/S = Not Significant

CHAPTER 11

IMMUNOLOGY AND THE FROZEN SHOULDER

Codman (45) first proposed that the initial lesion in frozen shoulder was a supraspinatus tendinitis. Simmonds (248) histological studies supported this view and suggested that it was due to chronic inflammation secondary to impairment of blood supply to the tendons. Rathbun and Macnab (225) demonstrated a constant hypovascular area in the supraspinatus tendon, close to its insertion, which they felt was due to the "milking" action of the head of the humerus on the tendon during activity. However, Macnab (70) later produced identical central degenerative changes in the tendon by partial interruption to its blood supply. He felt that the frozen shoulder was due to a type IV immunological reaction to the damaged collagen in the supraspinatus tendon. Bulgen et al (31) reported evidence of deficiency of the cellular immune system in patients with frozen shoulder which could predispose them to autoimmune disease (5). In a study of 25 patients with frozen shoulder, they (31) found reduced lymphocyte transformation to phytohaemagglutinin and low serum IgA levels. A possible genetic element was also suggested by Bulgen et al (30) who reported an increased incidence of HLA-B27 in a study of 38 frozen shoulder patients. Kessel et al (139), in a recent study of 50 patients with a spontaneous onset of frozen shoulder, found no such abnormalities. The aim of the immunological study was to confirm and extend the work of Bulgen et al (30, 31) which suggested immunological abnormality and to try to link the immunological abnormalities to the clinical pattern.

PATIENTS

The 42 patients in the Major Frozen Shoulder Study had blood samples taken at the first visit (pre Rx) and repeated after 8 months (post Rx).

CONTROLS

The 42 controls matched for age (within 2 years) and sex to the Major Frozen Shoulder Study group also had blood samples taken on one occasion to provide control data for many of the blood tests. All these patients were free of systemic illness and had no history of joint symptoms.

IMMUNOLOGICAL TESTS

a. Immunoglobulin classes IgG, IgM and IgA.
b. C-reactive protein (CRP).
c. Total haemolytic complement (CH50) and the third component of complement (C3).
d. Immune complex (IC) levels.
e. Lymphocyte count.
f. Lymphocyte transformation studies with 3 mitogens - phytohaemagglutinin (PHA), concanavalin A (Con A) and pokeweed mitogen (PWM).
g. Lymphocyte sub-populations - T cell, Fc and C3 rosettes.
h. Erythrocyte sedimentation rate (ESR, Westergren).
i. Auto-antibody screen including anti-thyroid and anti-nuclear antibodies.
j. Rose-Waaler test for rheumatoid factor.

OTHER BLOOD TESTS

a. Haemoglobin and full blood count.
b. Blood glucose.
c. Thyroxine, tri-iodothyronine and thyroid stimulating hormone levels.
d. HLA tissue typing for A + B loci antigens.

METHODS

THE IMMUNOGLOBULIN (IgG, IgA, IgM) AND C-REACTIVE PROTEIN (CRP) LEVELS were measured by a single radial immunodiffusion technique. The antigen was loaded into wells bored into agar plates containing a fixed concentration of antibody. By diffusion the antigen combined with antibody in the gel until equivalence was reached. At this point an immunoprecipitin "halo" formed around the well. The diameter of the halo at equivalence reflected the antigen concentration. Standard commercial plates were used for the immunoglobulin assays. Commercial anti-CRP and CRP standard sera (Behringwerke) were used for the CRP estimations. The normal range for IgG was 7-13 g/l; IgA 1-3.5 g/l and IgM 0.5-2 g/l. The normal range for CRP was 0-60 mg/dl.

TOTAL HAEMOLYTIC COMPLEMENT (CH50) was estimated using the kinetic turbimetric method (50% haemolytic time assay) (149). 0.5 ml of complement fixing diluent was mixed with 12.5 ml of serum at 37^{o}C. 0.5 ml of 0.1%

rabbit anti-sheep red cells were also warmed to 37°C and both were rapidly mixed in a 4 mm x 1 cm cuvette and incubated at 37°C in a Unicam SP 500 spectrophotometer with a thermostatically controlled 4-cuvette changer and a plotter. A reaction followed at 600 nm where haemolysis was seen as a reduction in turbidity. The time taken to achieve a 50% lysis of the sensitised red cells was measured. Using normal serum, a standard line was plotted on log-log paper of the 50% haemolysis time against CH50 units. From this line the amount of normal complement activity could be obtained from a single determination. Using this technique the normal range (mean ±3 SD) was 900 - 1800 units/ml.

THIRD COMPONENT OF COMPLEMENT (C3). This was measured using a rocket immunoelectrophoretic technique. Antigen was loaded into wells bored into antibody containing agar and was driven into the gel by electrophoresis. At equivalence, the resultant immunoprecipitation gave a rocket-shaped line, the height of the rocket being proportional to the antigen concentration. The normal range was 60 - 180 mg/dl.

IMMUNE COMPLEX (IC) LEVELS. A polyethylene glycol precipitation - complement consumption assay (PEG-CC) (110) was used to measure the IC levels. The complexes were first isolated from the serum by precipitation in 2.5% polyethylene glycol and then concentrated. Functional assay was achieved by measurement of their ability to fix complement using the kinetic turbimetric assay for CH50 as described above. Normal range was 0-24%.

LYMPHOCYTE TRANSFORMATION STUDIES. Lymphocyte culture was performed by a method described by Jones et al (135). 20 ml of heparinised blood was incubated at 37°C for 30 minutes, rotating the blood with 200 mg of carbonyl iron which was then removed (twice) using a magnet. 2 ml of dextran 240 was added to the blood which was allowed to stand for 30 minutes at 37°C. The white cell rich supernatant was then layered onto 5 ml of Ficoll Hypaque and centrifuged at 400 g for 20 minutes. The lymphocytes were washed twice with Eagles Medium MEM (pH 7.2) supplemented by non-essential amino acids 1%, glutamine 200 mM, foetal calf serum 10%, penicillin 100 units/ml, streptomycin 0.01%, sodium bicarbonate and HEPES buffer 25 mM. Each microtiter well in a microtiter plate was filled with 100 ul of medium containing 10^5 lymphocytes. The lymphocytes were cultured for 72 hours at 37°C with 50 ul of medium containing either -

a. Purified phytohaemagglutinin (PHA, Wellcome) in 9 dilutions, 2.5 - 0.01 ug,

b. Concanavalin A (Con A, Sigma Type IV) in 7 dilutions, 50 - 0.79 ug, or

c. Pokeweed Mitogen (PWM, Gibco) in 8 dilutions $^{1}/3$ - $^{1}/384$.

1 uCi of tritiated thymidine was added four hours before harvesting. The stimulation index (SI) was calculated for each mitogen concentration. SI = counts with medium plus mitogen / counts with medium alone.

LYMPHOCYTE POPULATION STUDIES. Lymphocytes were prepared with an identical method described for the transformation studies. T cells were identified by their ability to rosette with washed sheep red cells (T rosettes). B cells were recognised by rosette formation with ox cells sensitised with IgM anti-ox and mouse complement (C3 rosettes) and ox cells sensitised with IgG anti-ox (Fc rosettes). The percentage of cells which showed rosette formation with each method was calculated.

LYMPHOCYTE COUNTS. Both automated (Coulter-counter) and manual methods were used to count the number of lymphocytes in each specimen.

AUTO-ANTIBODY SCREENING. Antibody to cell nuclei (ANF), mitochondria, smooth muscle, parietal cell and thyroid microsomes were detected by direct immunoelectrophoresis. The Rose-Waaler test was used to detect the presence of rheumatoid factor.

HAEMOGLOBIN was measured by an automated Coulter-counter. The ERYTHROCYTE SEDIMENTATION RATE (ESR) employed the Westergren method with 0-20 mm/hr as the accepted normal range.

THYROXINE (T4), TRI-IODOTHYRONINE (T3), AND THYROID STIMULATING HORMONE (TSH) levels were measured by the hospital biochemistry department. The normal range for T4 = 65 - 145 nmol/L; T3 = 1 - 2.8 nmol/L and TSH = less than 4.0 mU/L.

BLOOD GLUCOSE was estimated by automatic analyser.

HLA TYPING was performed using the standard NIH microcytotoxicity method and national plate sera. Normal frequencies were taken to be those reported by Nelson et al (197) in a study of normal blood donors. The control group also had HLA typing performed.

RESULTS

Immune complexes (IC) were raised above the accepted normal range of 0 - 24% complement consumption in 17 of 39 (44%) pretreatment patients (pre Rx) and 2 of 40 (5%) controls (Figure 11:1). This rise was significant at the 5% level (Wilcoxon rank sum test). Eleven of 40 (28%) posttreatment patients (post Rx) also had raised IC levels (3 being normal preRx), but these values were not significantly different from those of the preRx patients or the controls. Nine of the 39 (23%) preRx patients had CH50 levels below the normal range (5 less than 700 U/ml) and of these, 5 also showed raised IC levels. All except 1 of these CH50 levels returned to normal postRx and a further 7 (18%) fell to below normal postRx. One preRx and one postRx serum showed a low C3 but neither was associated with a reduction in CH50.

CRP levels (Figure 11:2) were raised above the accepted normal range of 0 - 60 mg/dl in 10 of 40 (25%) preRx patients and 1 of 40 (3%) controls. This difference was significant at the 5% level (X^2 test). Only 3 of the 39 (8%) postRx group had raised levels. The fall in CRP measured in these patients postRx was significant at the 5% level (McNemar's test). An ESR above 20 mm/hr was noted in 8 patients preRx (5 were > 30 mm/hr) - 6 of the 8 also showed an elevated CRP level. The abnormal ESR persisted in 3 patients postRx and became abnormal in a further 3 (with no other obvious cause manifesting). Only 1 control had an abnormal ESR (31 mm/hr).

Lymphocyte counts were all within the normal laboratory range. Lymphocyte transformation in the preRx group showed a significant reduction, $p<0.01$, using Wilcoxon rank sum test, in the maximum SI irrespective of mitogen concentration, to both PHA and Con A compared to the controls (Figure 11:3). The postRx levels tended towards the control levels. Lymphocyte response to PWM was similar in all groups. When comparing the SI at each dilution for the 3 mitogens, significant differences were noted between preRx and control groups at 5 concentrations of PHA (Figure 11:4), 1 of these also being significant when comparing the postRx and control groups. Neither Con A (Figure 11:5) nor PWM (Figure 11:6), showed sequential significant differences along the dilution curves, although with Con A (preRx compared to controls) the difference was significant ($p<0.05$) at the concentration giving the maximum SI (1.57 ug) and at one other concentration. Lymphocyte populations were similar in all groups (Table 11:1) and although immunoglobulin levels tended to be slightly higher in the preRx patients (Table 11:2), this was not significantly different from the other groups.

The results of the immunological tests are summarized in Table 11:3.

To see if abnormal immunological tests at presentation were associated with differences in the severity of the pain, range limitation or their rate of recovery, patients with abnormal preRx parameters were compared to those with normal levels. To simplify these comparisons the 'pain score' (sum of pain at night, at rest and on movement) and the principal component was used. Five immunological parameters had sufficient patients with abnormal pre-treatment levels to permit comparative study. These were immune complexes (IC - raised in 17 patients), C-reactive protein (CRP raised in 10 patients), sedimentation rate (ESR raised in 8 patients) and total complement (CH50 reduced in 9 patients). Patients with an initial (pre Rx) lymphocyte stimulation index below the lowest level of the controls for phytohaemagglutinin (PHA abnormal in 16 patients) and concanavalin A (Con A abnormal in 18 patients) were also considered.

Using unpaired t-test, there were no significant differences (at the 5% level) between patients with normal and abnormal pre-treatment parameters with regard to the pain score (Table 11:4) or principal component (Table 11:5) at presentation or subsequent follow-up.

The mean recovery of range (principal component) from 0 to 8 months and from 8 to 44 months (Table 11:6) was also similar, whether the initial parameter was normal or not.

The rate of recovery of the pain score and principal component in the first 8 months (Figure 11:7 - 18) was also similar whether the initial immunological tests were normal or not.

Other Blood Tests:

5 patients (13%) had elevated thyroid function tests - thyroxine in 3, tri-iodothyronine in 1 and both in a further patient. 4 of these were associated with elevated thyroid microsomal antibody titres and a further 3 patients had positive thyroid microsomal antibodies but normal thyroid function. 1 patient had a positive anti-nuclear antibody (1:400) at presentation which was negative at follow-up. Rose-Waaler test was negative and the blood glucose normal in all patients at presentation and when repeated after 8 months.

HLA tissue typing for common A and B loci antigens showed no preponderance

for any histocompatibility antigens (Table 11:7). HLA-B27 was found in only 5 of the 42 (12%) frozen shoulder patients and 4 of the 42 (10%) controls, compared with 7.4% in the normal British population (197).

DISCUSSION

This study has found a range of immunological abnormalities in patients with frozen shoulder. IC, as measured by the PEG-CC test and the CRP levels were significantly raised in the preRx patients. These findings would support the concept of an inflammatory response in the shoulder. However, they do not form a consistent pattern and no relationship could be identified between them.

The reported observation (31) of reduced lymphocyte responsiveness to stimulation with the mitogen PHA in patients with frozen shoulder has been confirmed. This fact was further strengthened by a similar suppression noted with Con A and the improvement in lymphocyte reactivity in the postRx group. The response to PWM was the same in all 3 groups which is not unexpected as unlike PHA and Con A, it stimulates B as well as T cells (69, 131). The finding of reduced IgA concentrations (31) has not been confirmed despite using a similar method in both studies. An increased incidence of HLA-B27 (30) was also not found in this as in several recent studies (139, 206, 234, 243). Kessel et al (139) did not detect any immunological abnormalities in 50 patients with spontaneous onset of frozen shoulder. They measured immunoglobulin levels, IC, complement, ESR, full blood count and auto-antibodies and also performed tissue typing. The findings are similar apart from the IC levels. Kessel does not disclose which method was used to detect them nor their experimental error. The assays measure different components of the IC; such as size, immunoglobulin class and complement fixing ability, and thus results will vary (158). Kessel also used patients with osteoarthritis (OA) as controls, but Cooke et al (47, 48) and Pringle (220) have described the deposition of immunoglobulin and complement in the cartilage and synovial membrane in OA, making this a poor control group. Significantly raised CRP and IC levels and evidence of impaired cell-mediated immunity which show improvement in time, has been found in patients with frozen shoulder. However, these abnormalities are random and form no consistent pattern. Their presence lends some support to Macnab's hypothesis (170) of an autoimmune reaction accounting, in part, for the clinical syndrome of frozen shoulder.

TABLE 11:1

T AND B CELL LYMPHOCYTE POPULATIONS IN FROZEN SHOULDER PATIENTS AND CONTROLS : MEAN AND RANGE (% ROSETTES FORMED)

Lymphocyte Populations	PRE-RX (N = 38)	POST-RX (N = 31)	CONTROLS (N = 40)
T Cells	61.4 (45.0 - 89.7)	69.4 (27.0 - 86.6)	72.9 (53.0 - 89.0)
Fc Rosettes	30.6 (6.3 - 74.0)	24.8 (9.0 - 37.0)	23.0 (9.5 - 56.0)
C3 Rosettes	11.5 (0 - 26.7)	10.0 (2.7 - 37.0)	10.3 (2.0 - 30.0)

TABLE 11:2

IMMUNOGLOBULIN LEVELS IN FROZEN SHOULDER PATIENTS AND CONTROLS : MEAN AND RANGE (G/L)

Immunoglobulin	PRE-RX (N = 38)	POST-RX (N = 38)	CONTROLS (N = 40)
IgG	11.3 (3.5 - 17.5)	10.3 (4.1 - 27.6)	9.7 (5.9 - 13.2)
IgA	3.0 (1.1 - 7.4)	2.5 (0.7 - 3.3)	2.6 (0.9 - 5.5)
IgM	1.2 (0.3 - 4.0)	1.0 (0.4 - 2.8)	1.0 (0.4 - 2.1)

TABLE 11:3

SUMMARY OF RESULTS OF IMMUNOLOGICAL TESTS IN FROZEN SHOULDER PATIENTS AND CONTROLS

TEST	NORMAL RANGE	SAMPLE SIZE	OUTSIDE NORMAL RANGE N (%)
A. PRE-RX			
IC	0-24%cc	39	17 (44)
CH50	900-1800 u/ml	39	9 (23)
C_3	60-180 mg/dl	39	1 (3)
CRP	0-60 mg/dl	40	10 (25)
ESR	0-20 mm/hr	40	8 (20)
Lymphocyte count	$1.5 \bar{9} 4.0$ x 10^9/l	40	0 (0)
B. POST-RX			
IC	0-24% cc	40	11 (28)
CH50	900-1800 u/ml	40	8 (20)
C_3	60-180 mg/dl	38	1 (3)
CRP	0-60 mg/dl	39	3 (7)
ESR	0-20 mm/hr	40	6 (15)
Lymphocyte count	$1.5 \bar{9} 4.0$ x 10^9/l	40	0 (0)
C. CONTROLS			
IC	0-24% cc	40	2 (5)
CH50	900-1800 u/ml	-	- -
C_3	60-180 mg/dl	-	- -
CRP	0-60 mg/dl	40	1 (3)
ESR	0-20 mm/hr	40	1 (3)
Lymphocyte count	$1.5 \bar{9} 4.0$ x 10^9/l	40	0 (0)

TABLE 11:3 (CONTINUED)

SUMMARY OF RESULTS OF IMMUNOLOGICAL TESTS IN FROZEN SHOULDER PATIENTS AND CONTROLS

	ACCEPTED NORMAL RANGE (ANR)	PRE-RX SAMPLE SIZE	PRE-RX OUTSIDE ANR N (%)	POST-RX SAMPLE SIZE	POST-RX OUTSIDE ANR N (%)	CONTROL SAMPLE SIZE
PHA	Above lower decile of controls	40	16 (40)	40	9 (23)	39
Con A		38	18 (47)	40	6 (15)	39
PWM		38	4 (11)	39	2 (15)	37
T Rosettes	Control Mean±2SD	38	0 (0)	31	0 (0)	40
C_3 Rosettes		38	2 (5)	31	1 (3)	40
Fc Rosettes		38	1 (3)	31	0 (0)	40
IgG	Control Mean±2SD	38	6 (16)	38	4 (11)	40
IgA		38	2 (5)	38	1 (3)	40
IgM		38	5 (13)	38	1 (3)	40

TABLE 11:4

COMPARISON OF MEAN PAIN SCORE IN PATIENTS WITH NORMAL AND ABNORMAL PRE-RX IMMUNOLOGICAL PARAMETERS

PARAMETER	MEAN PAIN SCORE		Unpaired T-test	
	Normal Group	Abnormal Group	T_{38}	p<
A. AT PRESENTATION				
IC	14.2	15.7	0.889	N/S
CRP	14.6	15.9	0.637	N/S
ESR	14.3	16.5	1.227	N/S
CH50	15.1	14.5	-0.316	N/S
PHA	15.0	14.7	-0.158	N/S
CON A	15.3	14.5	-0.425	N/S
B. AT 8 MONTHS				
IC	2.4	1.4	-1.022	N/S
CRP	2.2	1.0	0.911	N/S
ESR	1.7	2.4	0.523	N/S
CH50	2.2	1.1	-1.034	N/S
PHA	1.6	2.5	0.903	N/S
CON A	1.1	3.1	1.877	N/S

N/S = Not Significant

TABLE 11:5

COMPARISON OF MEAN PRINCIPAL COMPONENT IN PATIENTS WITH NORMAL AND ABNORMAL PRE-RX IMMUNOLOGICAL PARAMETERS

PARAMETER	MEAN PRINCIPAL COMPONENT		Unpaired T-test	
	Normal Group	Abnormal Group	T_{38}	p<
A. AT PRESENTATION				
IC	2.0	- 2.1	0.368	N/S
CRP	4.5	-16.4	-1.576	N/S
ESR	2.7	- 7.3	-0.784	N/S
CH50	-0.8	2.2	0.242	N/S
PHA	1.1	- 1.9	-0.264	N/S
CON A	5.3	-11.9	1.526	N/S
B. AT 8 MONTHS				
IC	101.3	107.2	0.545	N/S
CRP	104.8	101.6	-0.243	N/S
ESR	105.5	100.2	0.435	N/S
CH50	102.9	107.1	0.353	N/S
PHA	101.2	109.2	0.713	N/S
CON A	103.7	102.4	0.117	N/S
C. AT 44 MONTHS				
IC	135.6	150.2	1.617	N/S
CRP	142.4	144.7	0.205	N/S
ESR	139.4	152.1	1.235	N/S
CH50	142.6	143.5	0.090	N/S
PHA	142.7	143.3	0.058	N/S
CON A	147.8	139.3	-0.878	N/S

N/S = Not Significant

TABLE 11:6

MEAN RECOVERY IN PRINCIPAL COMPONENT IN PATIENTS WITH NORMAL AND ABNORMAL IMMUNOLOGICAL TESTS

PARAMETER	MEAN RECOVERY IN PRINCIPAL COMPONENT		Unpaired T-test	
	Normal Group	Abnormal Group	T_{40}	p<
A. 0 - 8 MONTHS				
IC	99.3	109.3	0.826	N/S
CRP	100.3	118.0	1.217	N/S
ESR	102.9	107.5	0.334	N/S
CH50	103.7	104.9	0.088	N/S
PHA	100.1	111.2	0.879	N/S
CON A	94.7	114.3	1.597	N/S
B. 8 - 44 MONTHS				
IC	35.8	43.0	0.619	N/S
CRP	38.4	43.1	0.336	N/S
ESR	34.7	51.9	1.345	N/S
CH50	41.7	35.3	-0.525	N/S
PHA	44.3	38.4	-0.514	N/S
CON A	40.7	36.4	-0.337	N/S

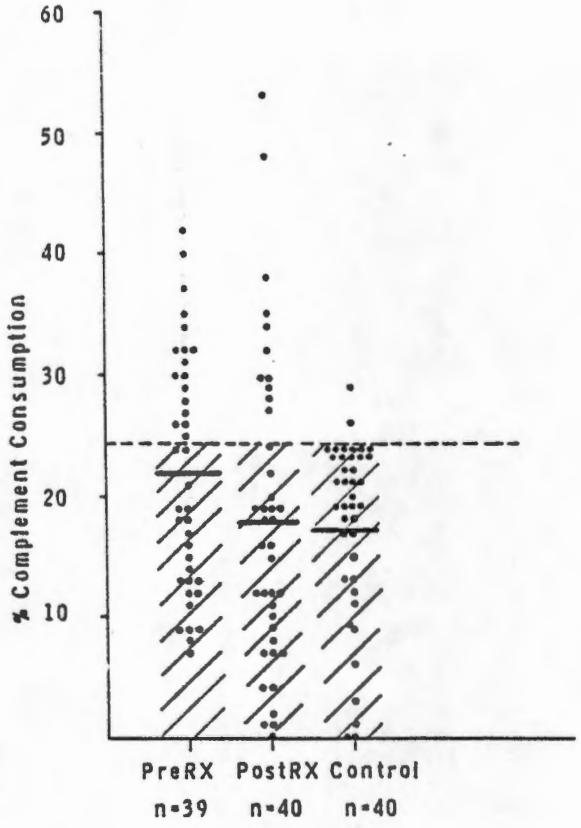
N/S = Not Significant

TABLE 11:7

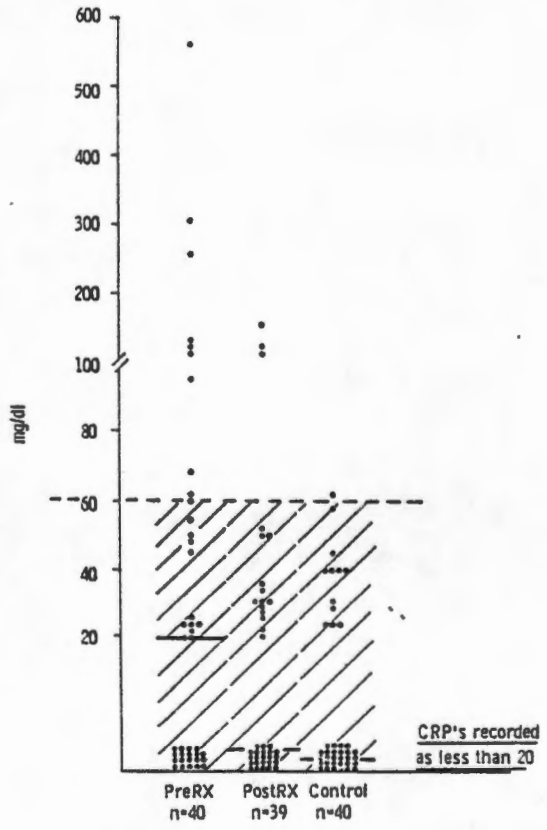
FREQUENCY OF COMMON HLA A & B LOCI ANTIGENS IN FROZEN SHOULDER PATIENTS COMPARED WITH FREQUENCY IN CONTROLS AND BRITISH BLOOD DONORS *

HLA-Antigen	Frozen Shoulder Patients (N = 42)		Frozen Shoulder Controls (N = 40)		British Blood Donors (N = 1036)	
	no.	%	no.	%	no.	%
A1	15	(36%)	16	(40%)	348	(34%)
A2	15	(36%)	18	(45%)	536	(52%)
A3	10	(24%)	9	(23%)	265	(26%)
A9	4	(10%)	6	(15%)	190	(18%)
A10	7	(17%)	5	(13%)	87	(8%)
A11	6	(14%)	6	(15%)	106	(10%)
B5	7	(17%)	4	(10%)	114	(11%)
B14	3	(7%)	3	(8%)	72	(7%)
B15	2	(5%)	3	(7%)	30	(3%)
B17	2	(5%)	2	(5%)	79	(8%)
B18	2	(5%)	1	(3%)	35	(3%)
B27	5	(12%)	4	(10%)	77	(7%)

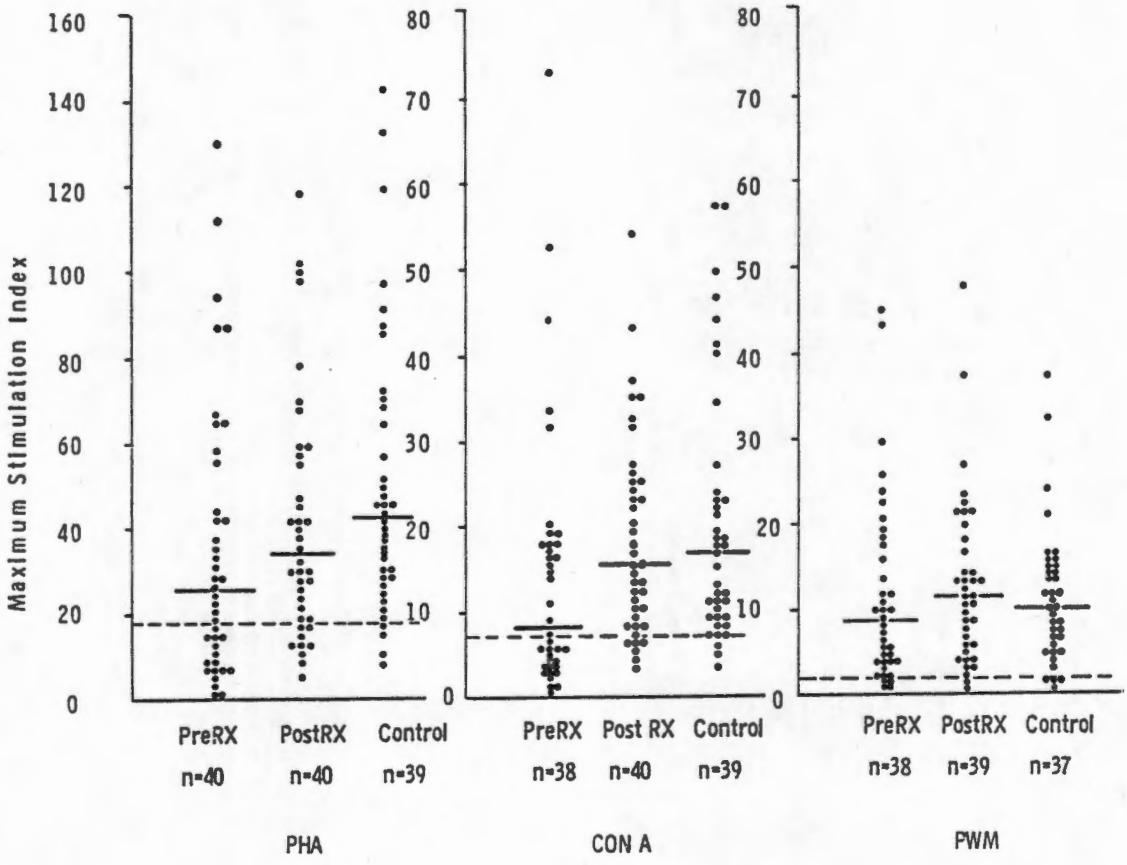
* Reference 197



F I G U R E II : I. IC levels in frozen shoulder patients and controls showing means and the accepted normal range (0 - 24%).

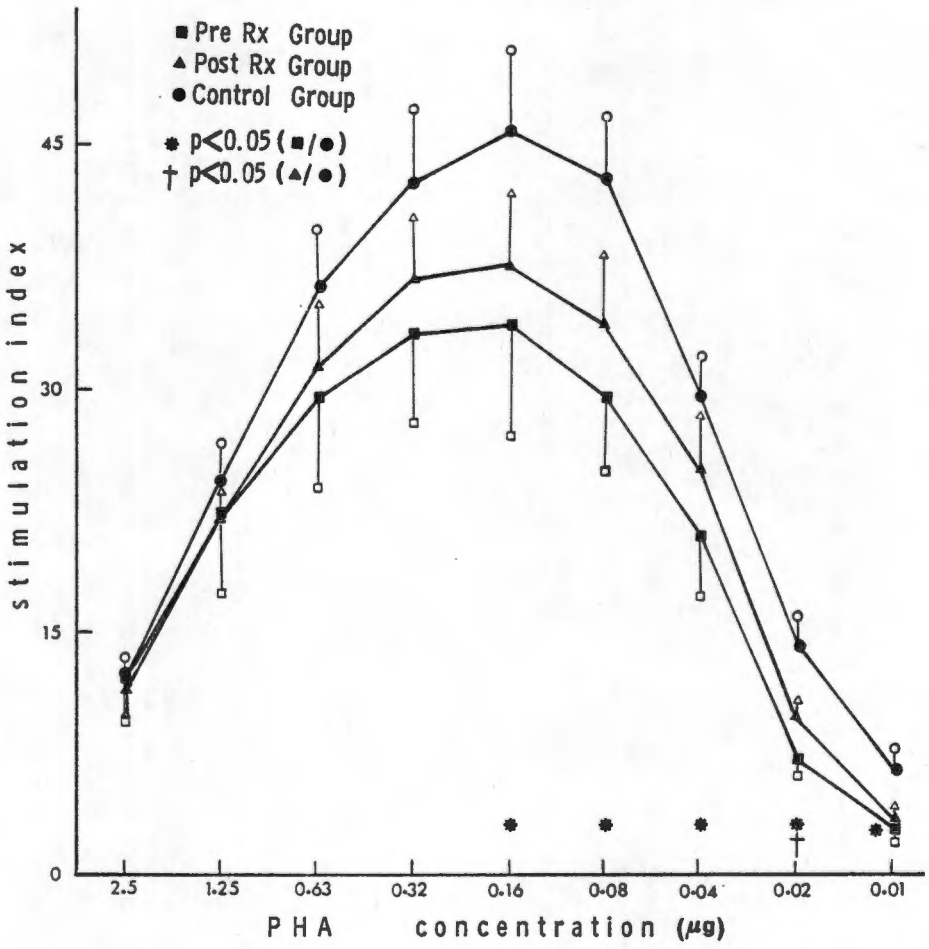


F I G U R E II : 2. CRP levels in frozen shoulder patients and controls showing medians and the accepted normal range (0 - 60 mg/dl).



F I G U R E II : 3. Lymphocyte transformation maximum S. I. for PHA, Con A and PWM in frozen shoulder patients and controls showing medians (——) and lower decile of the controls (----).

F I G U R E S II : 4 - 6. THE S.I. FOR LYMPHOCYTES FROM FROZEN SHOULDER PATIENTS AND CONTROLS AT DIFFERENT CONCENTRATIONS OF MITOGEN SHOWING THE SEM'S AND A COMPARISON (WILCOXON RANK SUM TESTS) BETWEEN THE GROUPS.



F I G U R E II : 4. PHA.

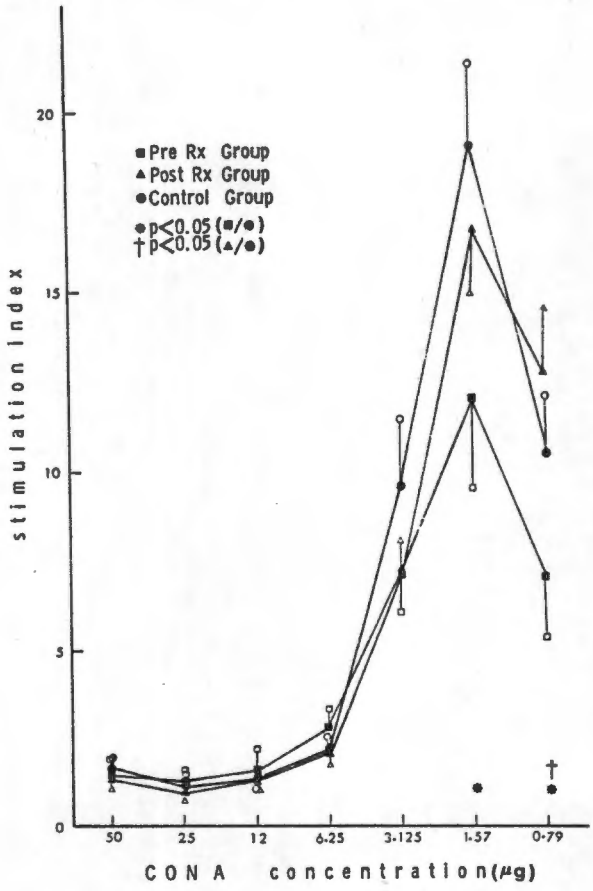


FIGURE II : 5. Con A.

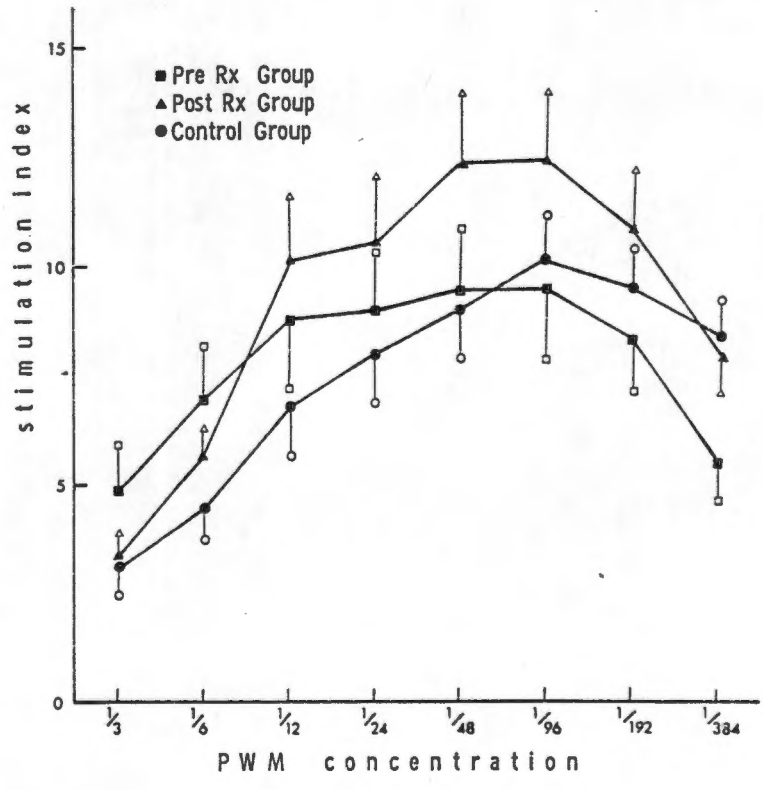


FIGURE II : 6. PWM.

F I G U R E S II : 7 - I8. COMPARISON OF THE RATE OF RECOVERY OF THE PAIN SCORE AND RANGE OF MOVEMENT (ΔC) IN PATIENTS WITH NORMAL AND ABNORMAL IMMUNOLOGICAL PARAMETERS AT PRESENTATION.

A) IMMUNE COMPLEX LEVELS.

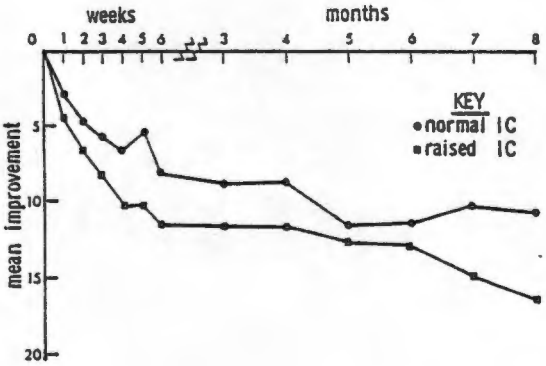


FIGURE II : 7. Improvement in pain score.

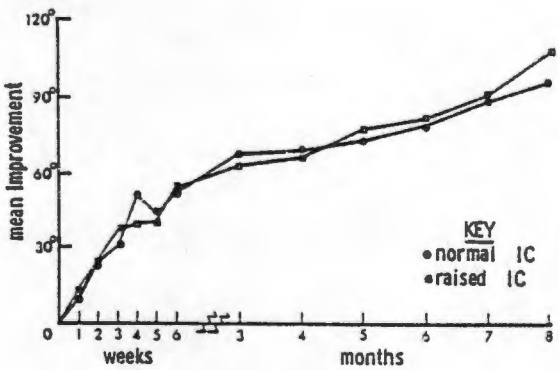


FIGURE II : 8. Improvement in range of movement (ΔC).

B) C REACTIVE PROTEIN LEVELS.

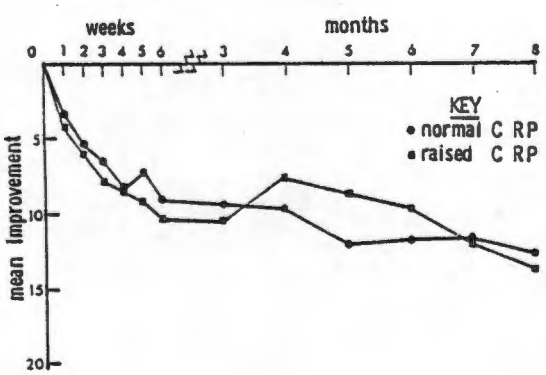


FIGURE II : 9. Improvement in pain score.

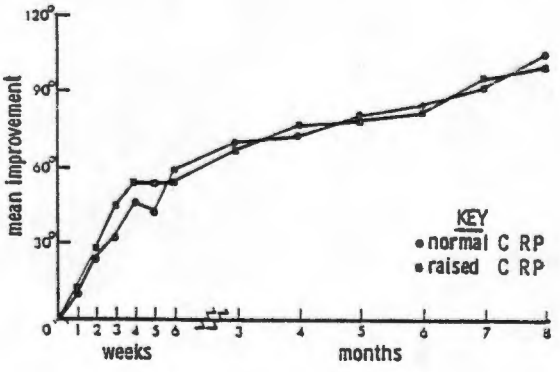


FIGURE II : IO. Improvement in range of movement (ΔC)

C) ERYTHROCYTE SEDIMENTATION RATE.

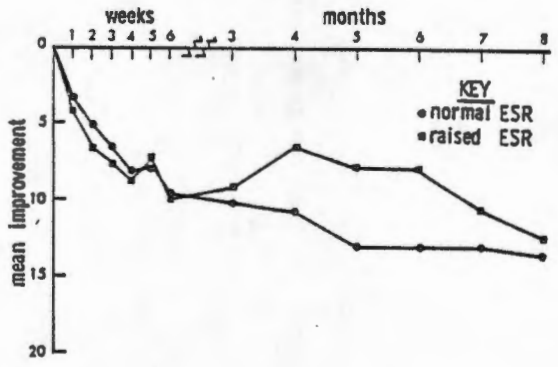


FIGURE II : II. Improvement in pain score.

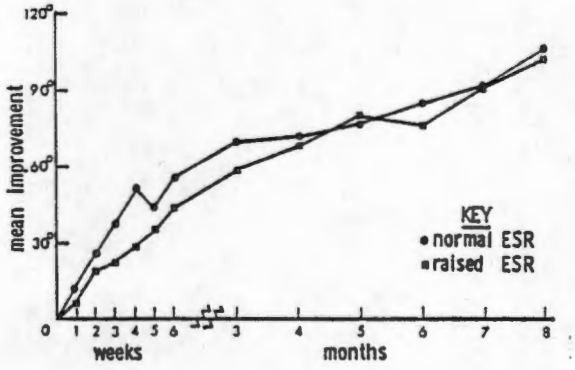


FIGURE II : I2. Improvement in range of movement (ΔC).

D) TOTAL COMPLEMENT (CH50) LEVELS.

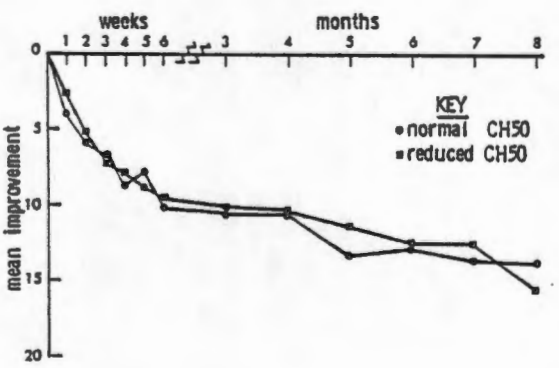


FIGURE II : I3. Improvement in pain score.

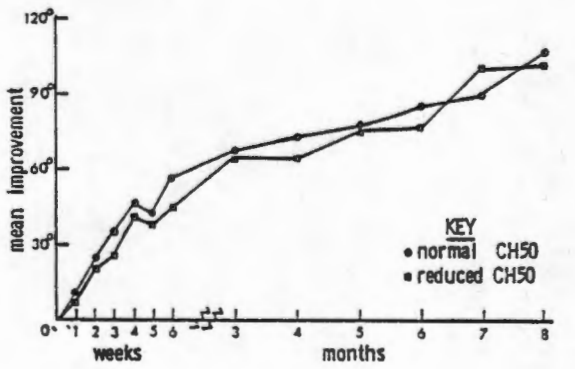


FIGURE II : I4. Improvement in range of movement (ΔC).

E) PHA STIMULATION INDEX.

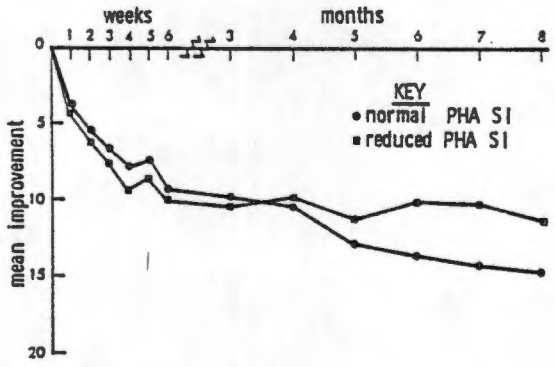


FIGURE II : I5. Improvement in pain score.

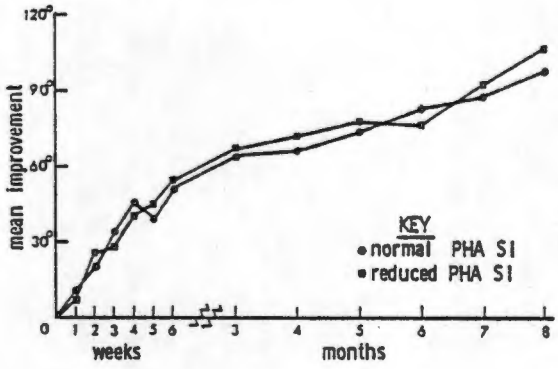


FIGURE II : I6. Improvement in range of movement (ΔC).

F) CON A STIMULATION INDEX.

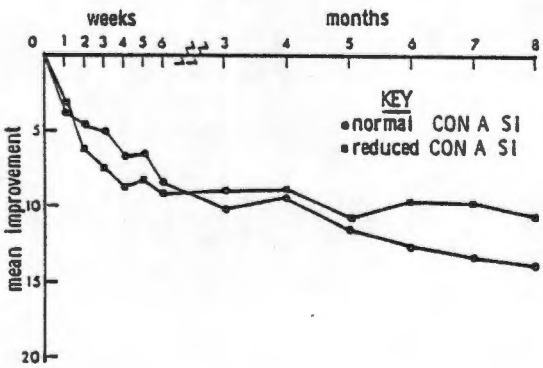


FIGURE II : I7. Improvement in pain score.

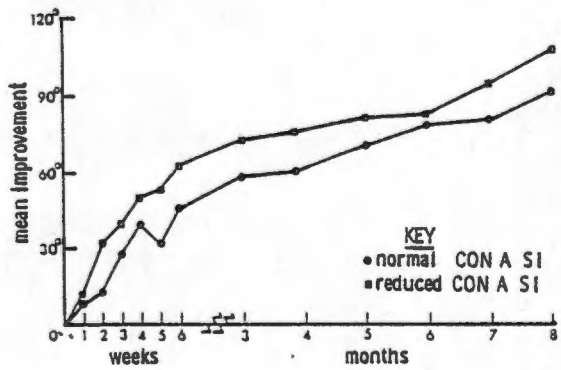


FIGURE II : I8. Improvement in range of movement (ΔC).

CHAPTER 12

RADIOLOGICAL ASPECTS OF THE FROZEN SHOULDER

Frozen shoulder is used to describe the clinical condition of a painful stiff shoulder. Neviaser (199) suggested that adhesive capsulitis was a more suitable name, as his pathological studies revealed chronic inflammation and fibrosis of the joint capsule which became closely adherent to the humeral head. Neviaser (201) later described the arthrographic features in patients with painful stiff shoulder. Most showed capsulitis with reduced joint volume and obliteration of the inferior and subscapularis recesses. However, 11 of his 53 patient (21%) showed no arthrographic abnormality. He considered the restriction in range in these latter patients was due to muscle spasm and that a good response to treatment and a good prognosis for early recovery was likely. Reeves (226) reported arthrography to be useful in differentiating post-traumatic stiff shoulders with a small distensible shoulder joint from frozen shoulder where distensibility was also lost. Treatment and prognosis were considered to be different. Lundberg (168) found a correlation between the severity of range restriction and reduction in joint volume, but recognised that 9% of patients had a normal arthrogram. Wright and Haq (292) found variable features at arthrography of their patients with clinical frozen shoulder and regarded the investigation of little use. Other authors have considered arthrography to be of value (196, 219, 233, 273) or even essential (200, 274) to make a correct diagnosis and decide on appropriate therapy and likely prognosis in patients with painful stiff shoulder. It has also been suggested to have therapeutic value (7, 49,207). Minor degenerative changes on X-ray in both affected and unaffected shoulder (52, 95, 208, 292) has been reported. An increased incidence of acromioclavicular arthritis has also been mentioned (292) in frozen shoulder patients.

The aim of this study was to assess the plain X-ray and arthrographic features at presentation in patients with clinical frozen shoulder and examine the relevance to the rate and extent of recovery. The importance of a traumatic onset was also assessed.

PATIENTS AND METHODS

The 42 patients included in the Major Frozen Shoulder Study had plain X-rays taken of both shoulders with special views to demonstrate the

acromioclavicular joints (ACJ). Thirty-six of the patients also had arthrography of the affected shoulder performed, although this was only successful in 31 cases. The arthrography was performed via the anterior route with a maximum of 20 ml of Conray 280 being introduced through a 22 gauge needle under fluoroscopic control. This procedure was performed within 1 week of presentation and before beginning treatment.

RESULTS

A. PLAIN X-RAY OF SHOULDERS: The X-ray of the affected shoulder (Table 12:1) was normal in 30 of the 42 patients (71%). The other 12 patients showed a variety of minor abnormalities. The most common abnormality was minor degenerative changes in the humeral head. These changes were most commonly seen in the greater tuberosity but also affected the lesser tuberosity. Degenerative change involving the glenohumeral joint was not accepted as part of the frozen shoulder syndrome and resulted in patient exclusion from the study. 5 patients showed reduction in the distance between the acromion and the humeral head. Only 6 patients (14%) had any radiological abnormality in the unaffected shoulder. 15 patients were manual labourers (Table 12:2) and 11 had a traumatic onset (Table 12:3), but neither factor was associated with an increased incidence of degenerative change on plain X-ray.

The mean pain score at presentation was higher in the patients with X-ray abnormality (Table 12:4), but unpaired t-tests did not show the difference to be significant. The pain score at 8 months also showed a slight advantage for patients with normal shoulder X-rays. There was no difference between the groups in the rate (Figure 12:1) or extent of recovery of the pain score. Of the 14 patients with mild persistent pain at review (mean follow up 44 months), 6 had abnormal and 8 normal X-rays. The 2 patients with the highest pain scores at review both had ACJ degeneration on X-ray. The abnormal X-ray group also had a more restricted range, that is a lower principal component, at presentation (Table 12:5) than the group with normal X-rays. This difference increased during the first 3 months of follow-up (Figure 12:2), although it never reached significance.

B. ARTHROGRAPHY: Although this was attempted in 36 patients, 4 were excluded due to difficulty in entering the joint. One further patient withdrew from the study due to the severity of the pain following arthrography. Five other patients reported the injection to be unacceptably painful and 27 (87%) noted a definite exacerbation of the shoulder pain

starting 4 - 6 hours after injection and lasting 24 - 48 hours thereafter. In 3 patients the pain lasted 2 - 4 weeks after the procedure. Five patients felt the shoulder was improved by the arthrography.

Of the 31 patients who had successful arthrography, three different subgroups emerged:

1. Normal Group (5 patients). The arthrogram was accepted as normal if it revealed a distensible shoulder joint with a smooth regular outline and a volume of at least 15 ml. An inferior and subscapularis recess was usually noted and contrast entered the bicipital tendon sheath but not the subacromial bursa.

2. Capsulitis Group (15 patients). A marked reduction in joint volume (often under 5 ml) with loss of distensibility of the shoulder joint was found. Marked irregularity of joint outline (Figure 12:3) and early lymphatic filling (Figure 12:4) was sometimes seen.

3. Rupture Group (11 patients). Rupture of the rotator cuff was considered to be present if an immediate flow of contrast into the subacromial/subdeltoid bursa occurred (Figure 12:5). With contrast medium present in the shoulder joint and subacromial bursa, the rotator cuff was clearly visible as a crescent above the humeral head. In 3 patients the rupture was small and revealed a normal looking shoulder joint. In the other 8 patients it was not possible to outline the joint sufficiently to assess detail. All 5 patients with a decrease in the distance between the acromion and the humeral head showed rupture of the rotator cuff.

The mean pain score at presentation (Table 12:6) was greater in the patients with normal arthrography, although analysis of variance did not show this difference to be significant. The rate of recovery of the pain score within the first 8 months (Figure 12:6) was similar in the 3 groups, but the capsulitis group (Table 12:6) had a lower mean pain score at 8 months. Of the 14 patients with persistent ache at review, 6 were in the capsulitis group, 3 in the rupture and 2 in the normal group.

The capsulitis group had the best range (that is the highest principal component, C) at presentation (Table 12:7), but analysis of variance did not show the difference to be significant. Contrasting C in the capsulitis to that in the other two groups at this time just reached significance at the 5% level. Assessment of the individual movements parameters at presentation

(Table 12:8) confirmed that the capsulitis group had a better range for all movements - glenohumeral and total. However, the rate of recovery of range (Figure 12:7) was slower in the capsulitis group in the first 6 weeks, allowing the other two groups to 'catch up'. After 6 weeks there was no further difference between the groups. The mean range of total flexion, abduction and rotation during the follow-up period (Table 12:9) revealed that the initial advantage for the capsulitis group disappeared by 3 months. The range was similar at 8 months and the normal group was slightly worse when patients were reviewed between 40 - 48 months after their initial presentation. The principal component (Table 12:7) at 8 and 44 months reflected these slight differences. The mean duration of symptoms at presentation was 9.0 months (range 3 - 12) in the normal group and 3.8 months (range 1 - 12) in the other two groups. Analysis of variance showed this difference to be significant, $p<0.0092$. However, the normal group could not be distinguished from the other 2 groups by the severity of the initial pain or range limitation or an improved rate or extent of recovery.

Patients with a traumatic onset were similar to those which arose spontaneously, both with regard to the distribution of patients in the 3 arthrographic groups (Table 12:10), initial severity and the rate and extent of recovery.

There was no association between the arthrographic features and immunological abnormalities at presentation, although raised CRP levels were more common in the capsulitis group (Table 12:11). IC, ESR, Con A and PHA levels were abnormal in a higher percentage of patient with a normal arthrogram but this group was too small to study in detail.

DISCUSSION

This study has attempted to evaluate the initial and long-term significance of radiological investigations performed at presentation in a carefully defined group of patients with clinical frozen shoulder. The high incidence of minor degenerative changes on plain X-ray of the affected shoulder (52, 95, 208, 292) was confirmed, but a high incidence of abnormality in the acromioclavicular joint (292) or opposite shoulder was not found. The only X-ray finding of practical value was the reduction in distance between the acromion and humeral head, which reliably predicted rupture of the rotator cuff.

The shoulder arthrography results differ markedly from many other reported

series where the features of capsulitis are said to always be present. Kernwein et al (138), Samilson et al (241) and Preston and Jackson (219) found capsulitis in all their patients but provided little clinical detail. Neviaser (200) regarded arthrography as essential for the diagnosis of adhesive capsulitis but his initial clinical study (201) did not confirm that the patients with normal arthrography had a better prognosis than the clinically identical patients showing capsulitis. Reeves (226) showed that arthrography was normal when performed in the convalescent phase after the recovery of range. The normal group in the present study did have a significantly longer mean duration at presentation, but their range was just as restricted as the other arthrographic groups. The rate and extent of recovery was also no different in patients with normal arthrography, so the lack of arthrographic abnormality in these patients cannot be attributed solely to the duration of symptoms at presentation. It is unclear if the patients with a traumatic onset in this study are similar to those described by Reeves (226), as he has given little clinical detail. However, no difference in prognosis was found in patients with a traumatic or spontaneous onset. There was also no association between the type of onset (traumatic or spontaneous) and the features at arthrography. As only half the patients showed evidence of capsulitis at arthrography, no attempt was made to confirm the reported correlation (168) between range restriction and joint volume.

The variability in the features at arthrography may reflect differences in the underlying pathology, but were not useful in predicting outcome. Arthrography is an invasive procedure which produced increased pain in most patients. This study has not suggested that arthrography is of value in the routine assessment of the frozen shoulder.

TABLE 12:1

PLAIN X-RAY FEATURES IN THE AFFECTED AND UNAFFECTED SHOULDERS IN THE MAJOR FROZEN SHOULDER STUDY (N = 42)

	Affected Shoulder	Unaffected Shoulder
Normal X-ray	30	36
Degenerative Changes of the Humeral Head	8	2
Supraspinatus Tendon Calcification	2	3
Degenerative Change of Acromioclavicular Joint	4	1
Reduction in the Acromiohumeral Distance	5	1

TABLE 12:2

PLAIN X-RAY FEATURES IN THE AFFECTED SHOULDER OF MANUAL AND SEDENTARY WORKERS IN THE MAJOR FROZEN SHOULDER STUDY

	Sedentary Workers (N = 27)		Manual Workers (N = 15)	
	no.	%	no.	%
Normal X-ray	20	(74%)	10	(67%)
Degenerative Changes of the Humeral Head	4	(15%)	4	(27%)
Supraspinatus Tendon Calcification	1	(4%)	1	(7%)
Degenerative Changes of Acromioclavicular Joint	3	(11%)	1	(7%)
Reduction in the Acromiohumeral Distance	3	(11%)	1	(7%)

TABLE 12:3

PLAIN X-RAY FEATURES IN THE AFFECTED SHOULDER OF MAJOR FROZEN SHOULDER STUDY PATIENTS WITH A SPONTANEOUS OR TRAUMATIC ONSET

	Spontaneous Onset (N = 31)		Traumatic Onset (N = 11)	
	no.	%	no.	%
Normal X-ray	23	(74%)	7	(64%)
Degenerative Changes of the Humeral Head	5	(16%)	3	(27%)
Supraspinatus Tendon Calcification	-		2	(18%)
Degenerative Changes of Acromioclavicular Joint	3	(10%)	1	(9%)
Reduction in the Acromiohumeral Distance	3	(10%)	2	(18%)

TABLE 12:4

COMPARISON OF PAIN SCORE AT PRESENTATION AND AT 8 MONTHS IN PATIENTS WITH NORMAL AND ABNORMAL SHOULDER X-RAYS

	N	Pain Score at Presentation Mean	(SEM)	Pain Score at 8 Months Mean	(SEM)
Normal X-ray	30	14.3	(1.0)	1.5	(0.5)
Abnormal X-ray	12	16.2	(1.3)	3.1	(1.2)
Unpaired T-test	T =	1.161*		1.396*	

* = Not Significant

TABLE 12:5

COMPARISON OF PRINCIPAL COMPONENT AT PRESENTATION, 8 AND 44 MONTHS IN PATIENTS WITH NORMAL AND ABNORMAL SHOULDER X-RAYS

	Principal Component at Presentation		Principal Component at 8 Months		Principal Component at 44 Months	
	Mean	(SEM)	Mean	(SEM)	Mean	(SEM)
Normal X-ray	6.3	(7.8)	108.6	(6.1)	145.4	(4.3)
Abnormal X-ray	-12.6	(4.4)	95.1	(10.3)	138.2	(10.7)
Unpaired T-test						
	T = 1.636*		1.207*		0.745*	

* = Not Significant

TABLE 12:6

COMPARISON OF PAIN SCORE AT PRESENTATION AND 8 MONTHS IN PATIENTS IN THE 3 ARTHROGRAPHIC GROUPS

	Pain Score at Presentation		Pain Score at 8 Months	
	Mean	(SEM)	Mean	(SEM)
Rupture Group	14.5	(1.9)	3.7	(1.5)
Capsulitis Group	14.5	(1.4)	1.1	(0.5)
Normal Group	16.2	(1.0)	3.0	(1.8)

TABLE 12:7

COMPARISON OF THE PRINCIPAL COMPONENT AT PRESENTATION, 8 AND 44 MONTHS IN THE 3 ARTHROGRAPHIC GROUPS

	Principal Component at Presentation		Principal Component at 8 Months		Principal Component at 44 Months	
	Mean	(SEM)	Mean	(SEM)	Mean	(SEM)
Rupture Group	-16.6	(10.9)	98.3	(14.7)	145.5	(8.3)
Capsulitis Group	15.6	(8.5)	105.2	(6.9)	145.6	(6.8)
Normal Group	-23.2	(12.7)	99.1	(15.6)	126.3	(27.7)

TABLE 12:8

MEAN, MINIMUM AND MAXIMUM RANGE AT PRESENTATION IN THE 3 ARTHROGRAPHIC GROUPS

	Capsulitis Group (N=15)		Rupture Group (N=11)		Normal Group (N=5)	
	Mean	Range	Mean	Range	Mean	Range
TF	111°	(70-135)	90°	(50-120)	86°	(50-120)
GF	52°	(35-70)	50°	(20-65)	46°	(30-70)
TA	80°	(30-110)	53°	(20-110)	54°	(35-90)
GA	41°	(20-70)	34°	(0-60)	35°	(30-45)
ER	17°	(0-35)	16°	(0-30)	5°	(0-10)
TR	47°	(20-80)	43°	(0-80)	35°	(20-60)

TABLE 12:9

MEAN RANGE OF THE MAIN MOVEMENT PARAMETERS AT DIFFERENT TIMES IN THE 3 ARTHROGRAPHIC GROUPS

A: TOTAL FLEXION

	Capsulitis Group	Rupture Group	Normal Group
Presentation	111°	90°	86°
At 6 Weeks	132°	113°	118°
At 3 Months	133°	123°	128°
At 8 Months	148°	137°	144°
At 44 Months	160°	155°	147°

B: TOTAL ABDUCTION

	Capsulitis Group	Rupture Group	Normal Group
Presentation	80°	53°	54°
At 6 Weeks	108°	98°	88°
At 3 Months	118°	106°	108°
At 8 Months	134°	128°	138°
At 44 Months	148°	149°	138°

C: TOTAL ROTATION

	Capsulitis Group	Rupture Group	Normal Group
Presentation	47°	43°	35°
At 6 Weeks	71°	92°	66°
At 3 Months	81°	87°	58°
At 8 Months	95°	113°	85°
At 44 Months	147°	149°	138°

TABLE 12:10

ARTHROGRAPHIC FEATURES IN PATIENTS WITH A TRAUMATIC OR SPONTANEOUS ONSET

	N	Spontaneous Onset	Traumatic Onset
Capsulitis Group	(15)	12	3
Rupture Group	(11)	7	4
Normal Group	(5)	4	1

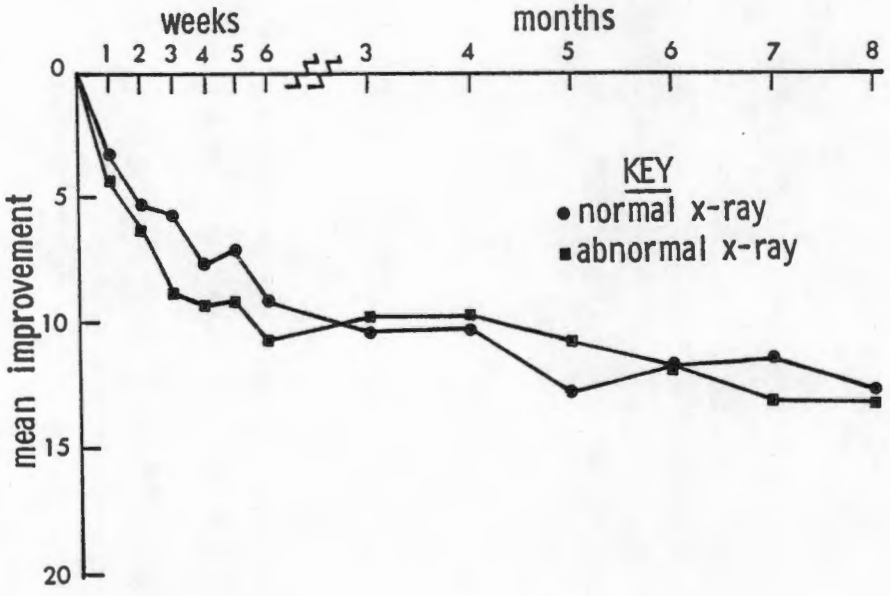
X^2 with Yates Correction = 0.98, p Not Significant

TABLE 12:11

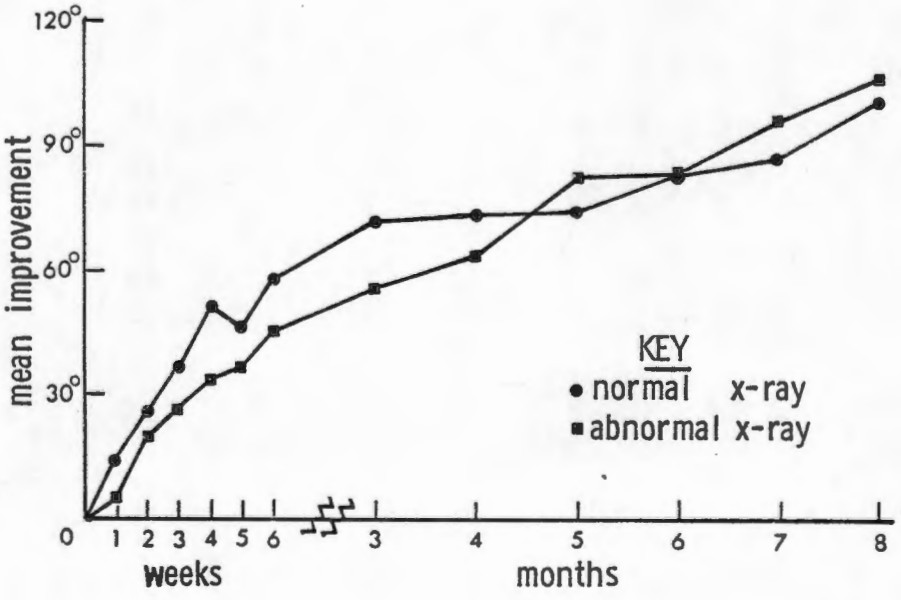
ASSOCIATION BETWEEN THE ARTHROGRAPHIC FEATURES AND ABNORMALITY OF IMMUNOLOGICAL PARAMETERS AT PRESENTATION

	Capsulitis Group (N=15)		Rupture Group (N=11)		Normal Group (N=5)	
	N	%	N	%	N	%
Raised IC Levels	5	(33%)	5	(45%)	3	(60%)
Raised CRP Levels	4	(27%)	1	(9%)	0	(0%)
Raised ESR Levels	4	(27%)	1	(9%)	1	(20%)
Reduced Con A Transformation	5	(33%)	5	(45%)	3	(60%)
Reduced PHA Transformation	3	(20%)	4	(36%)	2	(40%)

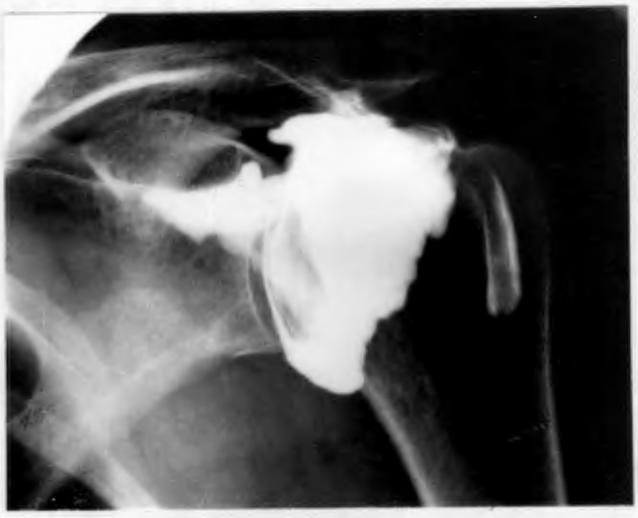
F I G U R E S I2 : I - 2. COMPARISON OF THE RATE OF RECOVERY OF THE PAIN SCORE AND RANGE OF MOVEMENT (ΔC) IN PATIENTS WITH NORMAL AND ABNORMAL X-RAYS OF THE SHOULDER AT PRESENTATION.



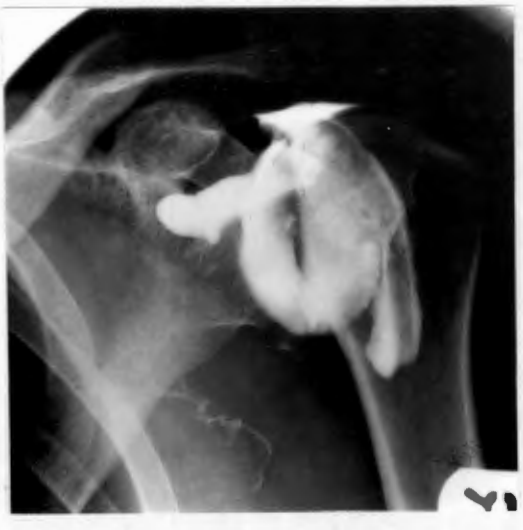
F I G U R E I2 : I. Improvement in pain score.



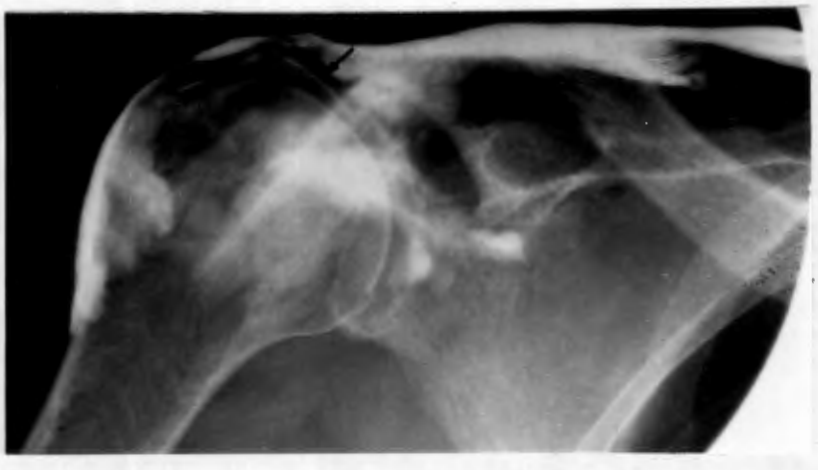
F I G U R E I2 : 2. Improvement in range of movement (ΔC).



F I G U R E I2 : 3. Arthrography showing marked irregularity of outline due to 'capsulitis'.

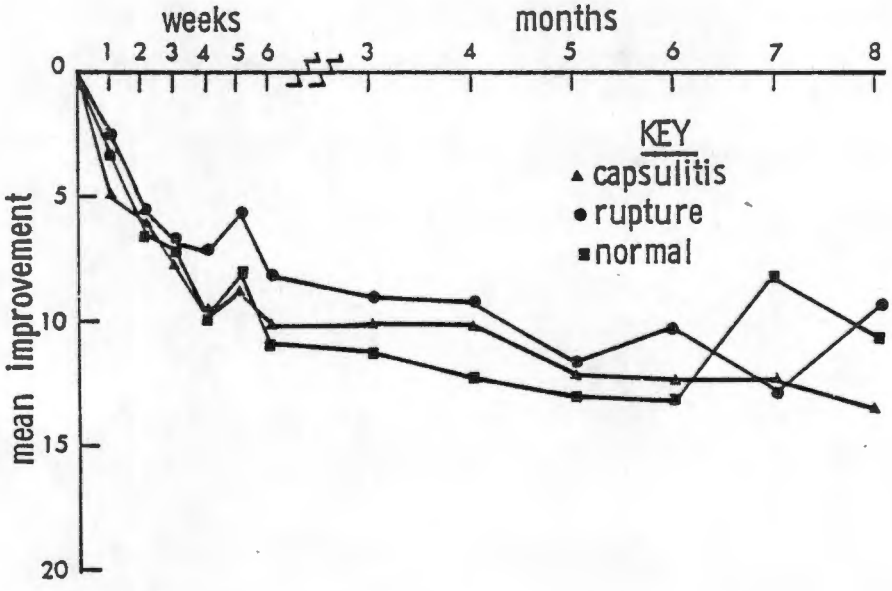


F I G U R E I2 : 4. Arthrography showing early lymphatic filling associated with 'capsulitis'.

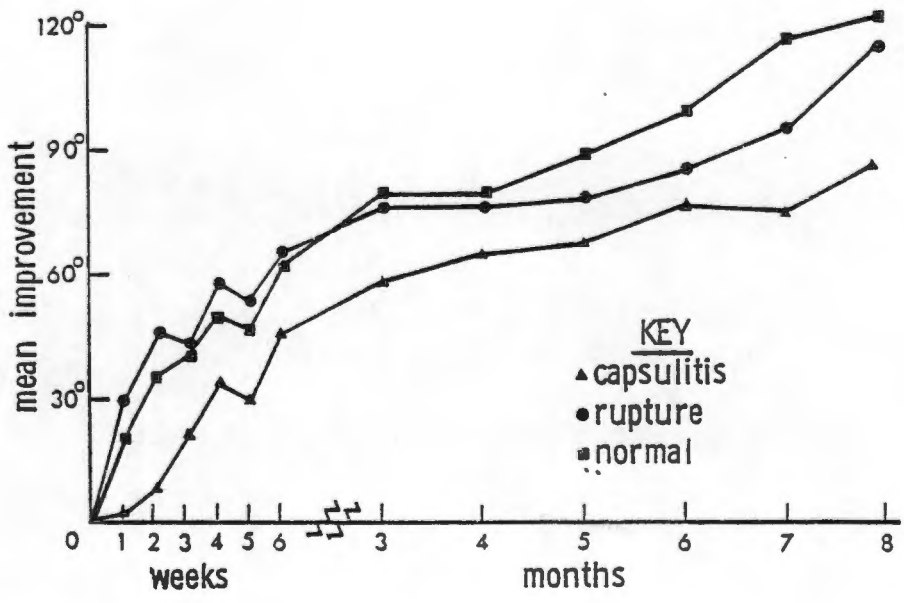


F I G U R E I2 : 5. Arthrography showing rupture of the rotator cuff with contrast medium present in the shoulder joint and sub-acromial bursa. The rotator cuff is indicated above the humerus.

F I G U R E S I2 : 6 - 7. COMPARISON OF THE RATE OF RECOVERY OF THE PAIN SCORE AND RANGE OF MOVEMENT (ΔC) IN THE 3 ARTHROGRAPHIC GROUPS.



F I G U R E I2 : 6. Improvement in pain score.



F I G U R E I2 : 7. Improvement in range of movement (ΔC).

CHAPTER 13

RADIO-NUCLEAR SCANNING AND THE FROZEN SHOULDER

Wright et al (290) reported an increased uptake of 99-m technetium pertechnetate in 4 of 10 patients with capsulitis of the shoulder thus suggesting an inflammatory component. All 4 responded to intra-articular steroid as did one of the negative patients and it was felt that this might be a way of identifying steroid responsiveness. Stodell et al (258) confirmed the increased uptake of pertechnetate, but showed a greater uptake if the diphosphonate isotope was used.

The aim of this study was to assess the technetium diphosphonate uptake at presentation and to examine its relevance to the initial symptoms and the subsequent rate and extent of recovery.

PATIENTS AND METHOD

Thirty-eight of the 42 Major Frozen Shoulder Study patients had 99-m technetium methylene diphosphonate scans performed before any treatment was given and usually on the same day and immediately before the arthrogram. Three hours after the injection of 10 - 15 mCi of 99-m technetium diphosphonate, an Elscint wholebody scanner was used to search for the hottest spot over each shoulder. The count was read with a 5 inch (12.7 cm) crystal and the ratio of the counts at the affected/unaffected shoulder (the uptake ratio) was calculated.

Forty patients of similar age and sex with no shoulder complaints but having diphosphonate scans for unrelated reasons also had counts taken over both shoulder to provide a control group. The count showed little variation between shoulders whether dominant or non-dominant, so control ratios were obtained from uptake in the right/left shoulders.

RESULTS

Thirty-five of the 38 patients had an increased technetium diphosphonate uptake (Figure 13:1-2) in the affected shoulder when compared to the opposite side (uptake ratio greater than 1), 11 of these scans showing more than a 50% increase in uptake. Comparison of the 2 shoulders (unpaired

t-test) showed the increase in uptake by the affected shoulder to be significant ($p<0.0001$). The distribution of the uptake ratios (Figure 13:3) showed a mean of 1.31 in the patients and 1.04 in the controls, the difference again being significant ($p<0.001$, unpaired t-test).

The 11 patients with an uptake ratio of 1.5 or more (High Scan Uptake Group) were compared to the 27 patients with a lower uptake ratio (Low Scan Uptake Group) to assess its relevance to initial severity of symptoms and the rate and extent of recovery.

The High Scan Uptake Group had a higher mean pain score at presentation and also at 8 months follow-up when compared to the Low Scan Uptake Group, but the difference between the groups was not significant (Table 13:1). The former group also showed a slower rate of recovery of the pain score within the first 6 weeks, but no difference thereafter (Figure 13:4). When the patients were reviewed from 40 - 48 months after presentation, 4 of 10 (40%) in the High and 6 of 27 (22%) in the Low Scan Uptake Groups still had some shoulder pain.

The passive range of movement as assessed by the principal component also showed greater restriction (Table 13:2) in the High Scan Uptake Group at presentation and also at 8 and 44 months follow-up. The difference at 8 months just failed to reach significance. However, the rate of recovery of the principal component was similar in both groups (Figure 13:5).

The incidence of abnormally raised immune complex, CRP and ESR levels and reduced stimulation to PHA and Con A at presentation was independent of the technetium uptake (Table 13:3). There was also no association between the arthrographic features and technetium uptake (Figure 13:6).

An uptake ratio >1.5 was found in 4 of 19 patients (21%) who presented within 3 months of the onset of symptoms and 7 of 12 (37%) who presented from 4 - 12 months. Three of 10 (30%) with a traumatic and 8 of 28 (29%) with a spontaneous onset of symptoms had a high uptake ratio, so that the duration of symptoms and type of onset did not influence the technetium uptake.

DISCUSSION

This study confirmed (258) the increased uptake of technetium diphosphonate by the painful stiff shoulder when compared to the opposite side or a

control group, but has not shown any significant correlation with the duration of symptoms, type of onset (traumatic or spontaneous), initial severity or recovery. Patients with a marked increase in technetium uptake showed no better recovery than those with little or no increase. The explanation for the increased uptake of the diphosphonate ('bone-seeking') isotope remains unclear. Only 2 of the patients had macroscopic calcification of the supraspinatus tendon on plain X-ray. Microscopic calcium hydroxyapatite crystal deposition, increased vascularity (101) and interaction between collagenous structures and microcrystalline complexes containing calcium (290) have been suggested.

An increased uptake of technetium diphosphonate in frozen shoulder lesions has been confirmed but this has not been found useful in the assessment of the painful stiff shoulder.

TABLE 13:1

COMPARISON OF PAIN SCORES AT PRESENTATION AND 8 MONTHS FOLLOW-UP IN PATIENTS WITH HIGH OR LOW SCAN UPTAKE RATIOS

	N	Pain Score At Presentation Mean	(SEM)	Pain Score At 8 Months Mean	(SEM)
Low Scan Uptake Group	27	14.8	(0.9)	1.5	(0.6)
High Scan Uptake Group	11	15.5	(1.6)	3.6	(1.1)
Unpaired T-test		T = 0.403*		1.787*	

* = Not Significant

TABLE 13:2

COMPARISON OF PRINCIPAL COMPONENT AT PRESENTATION, 8 AND 44 MONTHS FOLLOW-UP IN PATIENTS WITH HIGH OR LOW SCAN UPTAKE RATIOS

	Principal Component at Presentation Mean	(SEM)	Principal Component at 8 Months Mean	(SEM)	Principal Component at 44 Months Mean	(SEM)
Low Scan Uptake Group	3.3	(7.0)	108.4	(6.1)	149.0	(4.1)
High Scan Uptake Group	-11.1	(11.0)	84.9	(11.8)	131.1	(13.4)
Unpaired T-test T =	1.107*		1.940*		1.663*	

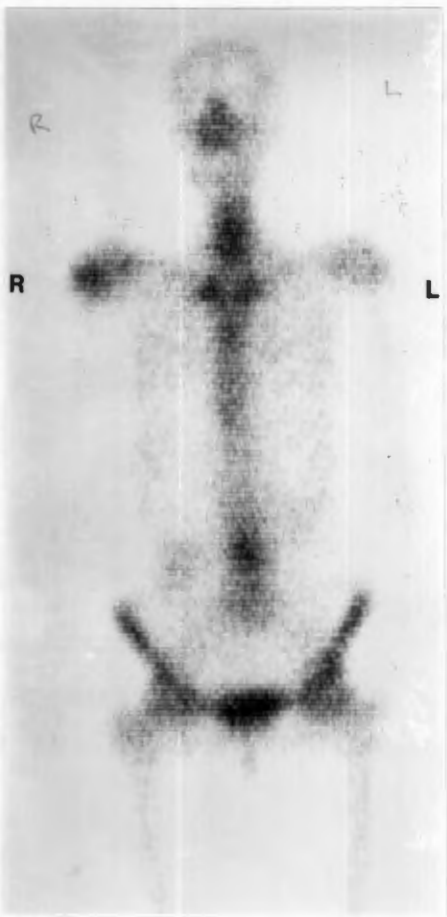
* = Not Significant

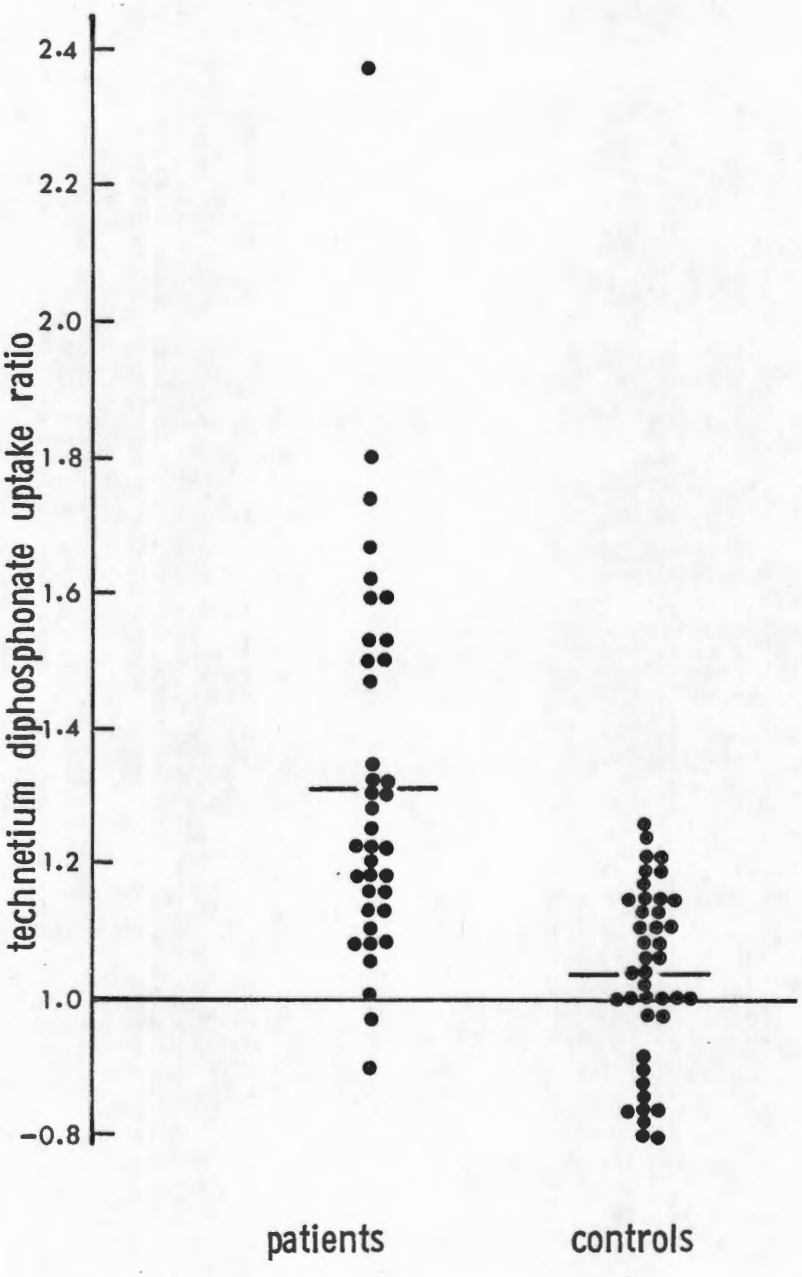
TABLE 13:3

INCIDENCE OF ABNORMAL IMMUNOLOGICAL PARAMETERS IN PATIENTS WITH HIGH AND LOW SCAN UPTAKE RATIOS

	Low Scan Uptake Group (N = 27)		High Scan Uptake Group (N = 11)	
	no.	%	no.	%
Raised IC Levels	16	(59%)	4	(36%)
Raised CRP Levels	5	(19%)	2	(18%)
Raised ESR Levels	6	(22%)	3	(27%)
Reduced Con A Transformation	11	(41%)	4	(36%)
Reduced PHA Transformation	11	(41%)	3	(27%)

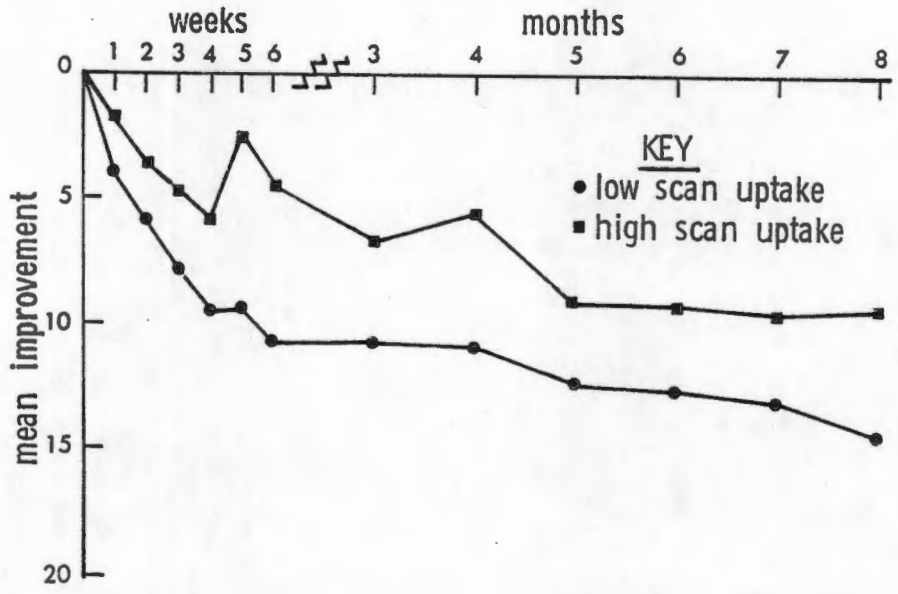
F I G U R E S I3 : I - 2. TECHNETIUM DIPHOSPHONATE SCANS FROM TWO FROZEN SHOULDER PATIENTS SHOWING THE INCREASED UPTAKE OF ISOTOPE BY THE AFFECTED SHOULDER (RIGHT) WHEN COMPARED TO THE OPPOSITE SIDE.



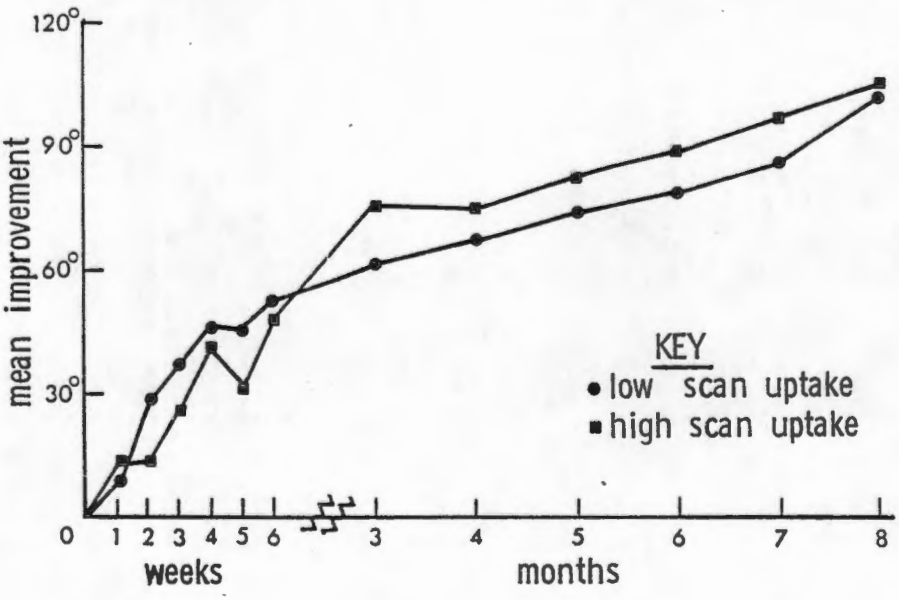


F I G U R E I3 : 3. Technetium uptake ratios in frozen shoulder (affected/unaffected) patients and controls (right/left) showing the mean value in each group.

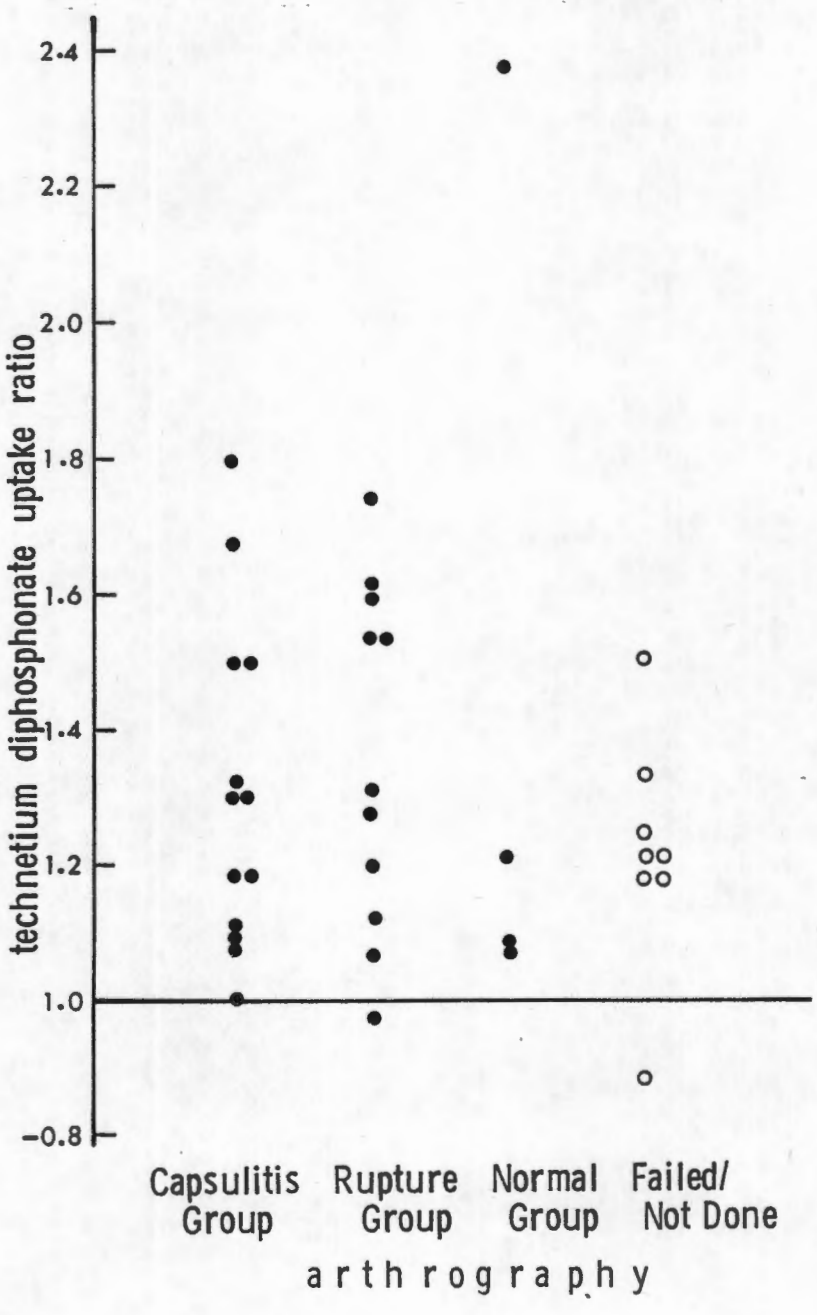
F I G U R E S I3 : 4 - 5. COMPARISON OF THE RATE OF RECOVERY OF THE PAIN SCORE AND RANGE OF MOVEMENT (ΔC) IN PATIENTS WITH A HIGH AND LOW TECHNETIUM UPTAKE RATIO AT PRESENTATION.



F I G U R E I3 : 4. Improvement in pain score.



F I G U R E I3 : 5. Improvement in range of movement (ΔC).



F I G U R E I3 : 6. The technetium uptake ratios in patients in the different arthrographic groups.

CHAPTER 14

THERAPY AND THE FROZEN SHOULDER

A wide range of therapeutic regimes have been suggested to treat painful stiff shoulders. These include locally injected and oral steroids, physiotherapy, manipulation under anaesthesia (MUA), infiltration brisement (249), radiotherapy, stellate ganglion block (280) and traction associated with transcutaneous nerve stimulation (232). There is no general agreement in favour of one form of treatment and the response to a particular treatment varies in different series. Hazleman (113) in a retrospective survey of 130 patients, could not find any difference in outcome in patients treated with local steroid injections, physiotherapy or MUA.

Disuse and immobility have for a long time been considered important in the aetiology of the frozen shoulder and prophylactic and therapeutic exercise has been advocated alone or in combination with other measures (53, 159, 248). Lee et al (153) showed that an exercise programme produced equally beneficial effects if it was combined with local steroid injections at different sites or heat. All these groups had a better outcome than was obtained using simple analgesics but without exercise. Quin (224) also showed that exercise alone was as effective as a similar regimen combined with ultrasound. Watson-Jones (272) and more recently Neviaser (200), Jayson (132) and Bruckner (25) have considered simple pendular exercise as effective as any other therapeutic measure. Heat and massage with or without an exercise programme (32, 159, 249), ultrasound (28, 154, 192) and shortwave diathermy (66) have all produced conflicting results. When the studies of ultrasound included a control group (15, 83, 189), the beneficial effects were much less evident. Ice packs are widely employed in physiotherapy departments for the treatment of the frozen shoulder especially in the acute phase, but little literature exists to support their use.

Local steroid injections have been used in this condition since Hollander's report (124) of encouraging results. Lloyd-Roberts and French (164) reported that steroid injections and MUA were superior to oral steroids. Roy and Oldham (238), Crisp and Kendall (55) and Steinbrocker and Argyros (256) claimed much better results if multiple sites of injection were used. Quin (223) found that local steroid injections reduced the pain but did not

accelerate the recovery of the range of movement. Murnaghan and McIntosh (194) were unable to show that local steroid injections were any more effective than local anaesthetic agents injected in a similar way. Cyriax and Trosier (59), Glyn and Newton (93), Fearnley and Vadasz (79) and Hollingsworth et al (125) failed to demonstrate any beneficial effects and Neviaser (201) even suggested that a marked response to this therapy cast a doubt on the accuracy of the original diagnosis.

The use of oral (systemic) steroid therapy has been subjected to even less controlled study. Ehlich et al (78), Coventry (53), Bateman (13) and Bland et al (17) have advocated its use without study. Blockey et al (19) compared oral steroids to placebo in 31 cases of frozen shoulder and reported significant improvement in pain but not range of movement in the treated group. Kessel et al (139) also found oral steroids and MUA more effective than MUA alone. However, Lloyd-Roberts and French (164) reported systemic steroids to be of little benefit.

Manipulation under anaesthesia has been variously accepted as the treatment of choice (105, 106, 118, 286) or outrightly condemned (62, 241) because of unacceptable side effects. Bloch and Fisher (18) reported an 80% success rate for MUA in the treatment of over 2000 frozen shoulder lesions. Thomas et al (263) also found MUA and steroid injections superior to injection alone. Kessel et al (139) felt oral steroids and MUA were most effective. Charnley (39), Reeves (226) and Lundberg (168) found MUA useful in reducing pain but less effective in improving range. Many reports (62, 176, 231, 241) considered MUA too hazardous for routine use, as it sometimes resulted in tears of the capsule and rotator cuff, fracture or dislocation of the humerus, nerve injury or haemorrhage around the shoulder. Poor efficacy (32, 231) has also been reported in many studies.

Non-steroidal anti-inflammatory agents (NSAIDS) and simple analgesics are still the mainstay of general practitioner treatment of the PSS but have not been subjected to controlled study. Lee et al (153) found analgesics much less effective than any other treatment regimes providing the latter included an exercise regime. Duke et al (72), Rhind et al (229) and Huskisson and Bryans (130) compared different NSAIDS to each other and were not convinced that this therapy influenced the natural history of the condition.

Based on the physiotherapeutic policy in Cambridge at that time, the Major Frozen Shoulder Study sought to assess the value of mobilisation exercise

and ice therapy compared to steroid injection in treating the frozen shoulder. Home pendular exercise was considered as the minimum therapy possible and was given to all the patients, but was the only treatment given to the control (non-treatment) group. The value of oral prednisolone was then assessed in the Supplementary Frozen Shoulder Study again comparing the response to home pendular exercise (non-treatment), which was performed by all the patients. MUA is still advocated by some people as the treatment of choice, but was not in use in Cambridge in view of the potentially serious risks. It was therefore not included in the studies.

PATIENTS

A. The Major Frozen Shoulder Study: The 42 patients were randomly allocated to one of four treatment groups using a predetermined randomisation schedule:-

1. Steroid Injection Group (11 patients). 20 mg of methylprednisolone acetate and 0.5 ml of 1% lignocaine hydrochloride was injected into the subacromial bursa and a similar amount into the shoulder joint by the anterior route. Both injections were given weekly for 3 weeks.

2. Mobilisation Group (11 patients). Maitlands mobilisations (171) were given to these patients by a research physiotherapist. Therapy was given for 30 minutes, three times a week for 6 weeks.

3. Ice Therapy Group (12 patients). Ice packs followed by proprioceptive neuromuscular facilitation (PNF), was supervised by the same research physiotherapist with a similar time schedule to the Mobilisation Group.

4. Non-treatment Group (8 patients).

All the patients were taught pendular exercise and advised to do them for 2 - 3 minutes every hour. Non-salicylate analgesics and diazepam 5 mgs at night were available as required.

B. The Supplementary Frozen Shoulder Study: The 40 patients in this study were randomly allocated to one of two groups:-

1. Oral Steroid Group (20 patients). 10 mg of enteric coated prednisolone was given to these patients as a morning-only dose for a period of 4 weeks. The dose was then reduced to 5 mg for a further 2 weeks before therapy was

stopped.

2. Non-treatment Group (20 patients).

All the patients in this study were also taught pendular exercise using an identical regime to the Major Study. Analgesics and nocturnal diazepam was also permitted if required.

The Major Study patients were assessed weekly for 6 weeks and monthly for a further 6 months, while the Supplementary patients were seen fortnightly for 6 weeks and then monthly till the 8 months follow-up period was complete.

RESULTS

A. The Major Frozen Shoulder Study: While the steroid injection group showed an initial advantage and the ice therapy group a disadvantage in the rate of improvement in 'pain at night' (Figure 14:1), one-way analysis of variance did not show the difference between the groups to be significant. The rate of recovery of 'pain on movement' (Figure 14:2) was also similar in all 4 groups. However, the steroid injection and mobilisation groups had a better (not significant) initial improvement in 'pain at rest' (Figure 14:3). The (summated) pain score (Figure 14:4) showed an overall initial advantage for the steroid injection group, although analysis of variance still failed to show the difference between the groups to be significant. A comparison of the mean pain score in the treatment groups at presentation and at 8 months follow-up revealed no significant differences at either time (Table 14:1A). At 8 months, severe and disabling pain was still present in 3 patients treated with ice therapy; 2 with mobilisation physiotherapy; 1 with steroid injection and 2 with no therapy. When 40 of the patients were reviewed at a mean follow-up of 44 months, mild residual pain was present in 3 of the mobilisation group (30%), 4 of the ice therapy group (33%), 3 of the steroid injection (30%) and 2 of the non-treatment (25%) patients. The one patient with severe pain and restriction at this time had received mobilisation physiotherapy.

From the graphs (Figure 14:5-9), it can be seen that the initial rate of improvement in the individual movement parameters and the principal component was more rapid in the steroid injection group, although this advantage was not maintained after 3 months. This difference between the groups was confirmed by performing a one-way analysis of variance on the mean principal component in the four treatment groups at different times

during the study (Table 14:2). At presentation, the non-treatment group had a better range - that is a higher principal component - although there was no signficant differences between the groups. At the end of therapy (at 6 weeks), the steroid injection group had the best range - that is the highest principal component, and the mobilisation group the worst range. Analysis of variance showed that the groups differed significantly at the 2% level. The mobilisation group continued to recover more slowly, but at the end of the study (8 months) there was no significant difference between the groups. However, this trend for a slower recovery by the mobilisation group continued even after 8 months. While analysis of variance did not show a significant difference between the groups at review (44 months), contrasting the principal component in the mobilisation group with that in the other 3 groups (general linear models procedure) showed a significant difference at the 5% level. The mean and the maximum and minimum ranges of individual movement parameters in the treatment groups at presentation (Table 14:3A) showed no significant differences between the groups, although the non-treatment group had a slightly better and mobilisation group a slightly worse range. The range at 8 months showed greater restriction of all movement parameters in the mobilisation group and no difference between the other groups (Table 14:4A). This poor outcome for the mobilisation group was even more marked at review (Table 14:5). While analysis of variance still failed to show a significant difference between the groups, contrasting the range of the mobilisation group with the other 3 groups (general linear models procedure) showed a significantly worse range (at the 5% level) for the mobilisation group for all individual movements except external rotation.

B. <u>The Supplementary Frozen Shoulder Study</u>: The oral prednisolone group showed a significantly more rapid initial improvement ($p<0.05$, Wilcoxon rank sum tests) in 'pain at rest' (Figure 14:10) and 'pain at night' (Figure 14:11) when compared to the non-treatment group. The level of significance is indicated on the graphs. 'Pain on movement' (Figure 14:12) also showed an advantage for the treated group, but the difference was not significant. However, the recovery of the (summated) pain score was significance (Figure 14:13). After the prednisolone was tailed off, two prednisolone treated patients had a recurrence of severe pain and four mild pain which settled spontaneously. After 6 weeks, there was no difference in the level of pain in the 2 groups. The mean pain score in the treatment groups was similar at presentation and at the end of the study (Table 14:1B). At 8 months, 3 patients (2 in non-treatment group) still complained of severe pain and one other could not sleep on the affected side. There was no advantage for the

treated group in recovery of individual movement parameters including total flexion (Figure 14:14), total abduction (Figure 14:15), total rotation (Figure 14:16) and external rotation (Figure 14:17). A cumulative recovery curve using the principal component also failed to show a significant difference between the groups (Figure 14:18). The range of movement at presentation (Table 14:3B) and at 8 months follow-up (Table 14:4B) and the principal components at these times (Table 14:6) showed no significant differences between the groups.

SIDE-EFFECTS OF THERAPY

Three of the patients treated with local steroid injections complained of exacerbation of the shoulder pain lasting for 12-24 hours after the injections. Five of the mobilisation group also noted an increased ache especially at rest following the physiotherapy sessions. Two patients developed mild indigestion on full dosage oral prednisolone which settled on reducing the dose. No other untoward reactions were reported.

DISCUSSION

Therapy for the frozen shoulder remains empirical as few long-term prospective studies have been performed. The therapeutic measures most widely used in Cambridge to treat the frozen shoulder were subjected to careful study. Both pain and passive range of movement were recorded at each visit to determine if any of the therapeutic measures could influence the rate or extent of recovery. Recovery curves in both frozen shoulder studies showed that the most marked improvement in pain and range occurred within 4 - 6 weeks of initiating therapy, suggesting that a course of therapy should be limited to this period. Manipulation under anaesthesia was not included in the studies as the potential side-effects of this therapy were considered to be unacceptable.

Roy and Oldham (238) reported an excellent response to paired local steroid injections. A similar steroid dosage, frequency and method of injection was used in the Major Study but with less satisfactory results. While 95% of their 55 patients were pain-free 8 weeks after being seen, only 36% of patients in this study and treated with steroid injections were pain-free at the same stage. Similarly less improvement in the range of movement was also achieved. These differences may reflect patient selection - our patients were more severe - and differences in measurement techniques. Nevertheless, steroid injection therapy was the most effective treatment in

the Major Study with regard to a reduction in pain and to a lesser extent improvement in passive range. However, this benefit was only short-lived.

The report by Blockey et al (19) that oral steroids are effective at decreasing pain, but have no effect in hastening the recovery of range of movement was confirmed. Only two patients had recurrence of symptoms after stopping this therapy and few side-effects were encountered. Non-steroidal anti-inflammatory agents are ineffective in reducing pain in this condition so the anti-inflammatory actions of steroid may not alone explain their efficacy whether given by local injection or systemic routes.

The patients who received Maitland's mobilisations (171) as part of the physiotherapy treatment in the first 6 weeks, had the least satisfactory outcome at both 8 and 44 months. This group had slightly greater restriction at presentation and may not be strictly comparable to the other groups, but the differences were not significant at that time and only became so during the follow-up period. This suggests that active physiotherapy in the acute stage may aggravate the condition and should probably be avoided. Lee et al (152) reported very good results with physiotherapy, but they studied a slightly different group of patients in that 16/25 of their patients had suffered symptoms for more than one year. Their placebo group was measured after 6 weeks, while the active treatment groups were measured weekly and this may have influenced their results. The ice packs proved to be ineffective in terms of pain relief and improvement in range and like mobilisation exercises did not justify the time and expense for patient or physiotherapist.

Home pendular exercise alone produced a less satisfactory initial reduction in pain than steroid given by injection or orally. Nevertheless these patients did improve rapidly within the first 4 weeks after inclusion in the study, following a similar recovery curve to the other treatment groups. There was also no difference in the rate of recovery of range in these patients when compared to the other groups. Indeed the long-term results are better than the mobilisation group in the Major Study. Home pendular exercise was chosen for the control (non-treatment) groups as it was considered impossible and unethical to offer these patients no treatment during regular follow-up over a prolonged period. These patients received no hospital treatment and permitted study of the natural history of the frozen shoulder subjected to the minimum therapy. A comparison of the improvement in the non-treatment group before with that after entering the study is difficult, but supports the belief (25, 132, 272) that home

pendular exercise may be important in initiating recovery in this condition. The role of pendular exercise requires further study.

In summary, paired local steroid injection therapy was most effective in accelerating recovery, but these benefits were only short-lived. Oral steroids also reduced shoulder pain but did not enhance the recovery of the passive range of movement. Physiotherapy especially assisted mobilisations were possibly detrimental to the recovery of range and should probably be avoided. The improvement in pain and passive range in the patients who only performed home pendular exercises was much better after than before inclusion in the studies and suggests that this therapy may be important in promoting recovery in this condition.

TABLE 14:1

COMPARISON OF THE (SUMMATED) PAIN SCORE AT PRESENTATION AND 8 MONTHS FOLLOW-UP IN THE TREATMENT GROUPS OF (A) MAJOR FROZEN SHOULDER AND (B) SUPPLEMENTARY FROZEN SHOULDER STUDIES

	N	Pain Score at Presentation Mean	(SEM)	Pain Score at 8 months Mean	(SEM)	Mean Recovery 0 - 8 months
A. MAJOR FROZEN SHOULDER STUDY						
Mobilisation Group	11	17.2	(1.2)	1.3	(0.6)	15.9
Steroid Injection Group	11	15.0	(1.1)	1.5	(0.8)	13.5
Ice Therapy Group	12	14.6	(1.6)	3.8	(1.4)	10.8
Non-Treatment Group (I)	8	11.8	(1.4)	0.4	(0.4)	11.4
B. SUPPLEMENTARY FROZEN SHOULDER STUDY						
Oral Steroid Group	20	16.9	(0.7)	1.1	(0.4)	15.8
Non-Treatment Group (II)	20	14.5	(1.2)	1.2	(0.4)	13.3

TABLE 14:2

COMPARISON OF MEAN PRINCIPAL COMPONENTS IN THE 4 MAJOR FROZEN SHOULDER TREATMENT GROUPS AT DIFFERENT TIMES DURING THE STUDY

	MEAN PRINCIPAL COMPONENT			
	At Presentation (Time 0)	End of Therapy (6 Weeks) *	End of Study (8 Months)	At Review (44 Months) +
Mobilisation Group	-7.1	27	85.5	124.9
Steroid Injection Group	-7.7	73	110.6	150.0
Ice Therapy Group	-1.3	50	109.0	148.4
Non-Treatment Group (I)	16	62	113.2	151.2

* Analysis of Variance $p<0.02$

\+ Contrasting Mobiliation Group to the Rest $p<0.05$

TABLE 14:3

RANGE OF MOVEMENT IN THE THERAPY GROUPS AT PRESENTATION

	N	Total Flexion Mean (Range)	Total Abduction Mean (Range)	Total Rotation Mean (Range)	External Rotation Mean (Range)
A. MAJOR FROZEN SHOULDER STUDY					
Mobilisation Group	11	94° (50-120)	71° (20-100)	33° (0-70)	5° (0-30)
Steroid Injection Group	11	95° (60-130)	61° (20-110)	45° (20-80)	19° (0-35)
Ice Therapy Group	12	100° (70-135)	65° (40-135)	47° (20-80)	14° (0-30)
Non-treatment Group (I)	8	106° (80-130)	84° (40-120)	53° (20-100)	15° (0-50)
B. SUPPLEMENTARY FROZEN SHOULDER STUDY					
Oral Steroid Group	20	106° (60-135)	79° (45-120)	54° (10-80)	22° (5-40)
Non-treatment Group (II)	20	111° (70-140)	87° (40-120)	61° (20-130)	20° (0-50)

TABLE 14:4

RANGE OF MOVEMENT IN THE THERAPY GROUPS AT 8 MONTHS FOLLOW-UP

	N	Total Flexion Mean (Range)	Total Abduction Mean (Range)	Total Rotation Mean (Range)	External Rotation Mean (Range)
A. MAJOR FROZEN SHOULDER STUDY					
Mobilisation Group	11	138° (80-160)	123° (60-160)	84° (50-115)	33° (0-60)
Steroid Injection Group	11	147° (140-160)	137° (85-175)	107° (80-125)	47° (15-70)
Ice Therapy Group	12	145° (120-160)	140° (90-160)	104° (60-140)	41° (15-70)
Non-treatment Group (I)	8	148° (130-160)	139° (100-160)	108° (65-160)	41° (25-65)
B. SUPPLEMENTARY FROZEN SHOULDER STUDY					
Oral Steroid Group	20	147° (110-175)	138° (90-170)	116° (80-160)	40° (25-80)
Non-treatment Group (II)	20	153° (130-170)	142° (100-165)	116° (65-165)	41° (25-70)

TABLE 14:5

COMPARISON OF MEAN RANGE OF MOVEMENT AT REVIEW IN THE 4 MAJOR FROZEN SHOULDER TREATMENT GROUPS

	Mobilisation Group (N=11)		Steroid Injection Group (N=10)		Ice Therapy Group (N=11)		Non-treatment Group (N=8)	
	Mean	(Range)	Mean	(Range)	Mean	(Range)	Mean	(Range)
TF	147°	(90-170)	161°	(140-175)	156°	(140-175)	163°	(160-170)
TA	135°	(70-175)	150°	(110-165)	152°	(130-175)	151°	(140-165)
TR	132°	(70-180)	153°	(120-175)	152°	(139-170)	154°	(125-180)
ER	46°	(15- 80)	52°	(40- 75)	55°	(30- 70)	57°	(40- 80)

TABLE 14:6

COMPARISON OF MEAN PRINCIPAL COMPONENT AT PRESENTATION AND 8 MONTHS FOLLOW-UP IN THE SUPPLEMENTARY FROZEN SHOULDER TREATMENT GROUPS

	Oral Steroid Group (N = 20)	Non-treatment Group (N = 20)	Unpaired T-test T_{38}	$p<$
Presentation	-6.7	99.5	1.096	N/S
8 Months Follow-up	4.9	105.2	0.610	N/S

N/S = Not Significant

F I G U R E S I4 : I - 4. MEAN IMPROVEMENT IN PAIN PARAMETERS AND PAIN SCORE IN THE MAJOR FROZEN SHOULDER STUDY TREATMENT GROUPS.

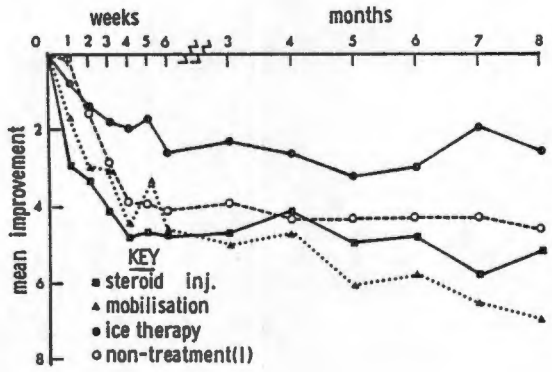


FIGURE I4 : I. Pain at night.

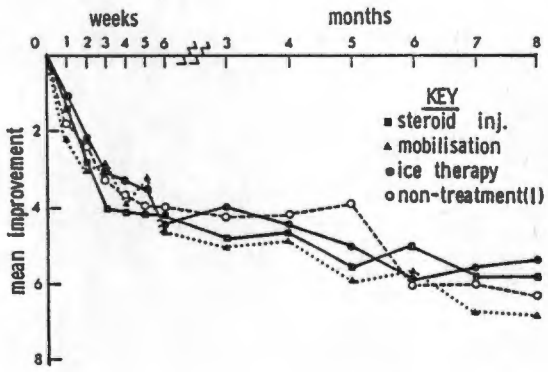


FIGURE I4 : 2. Pain on movement.

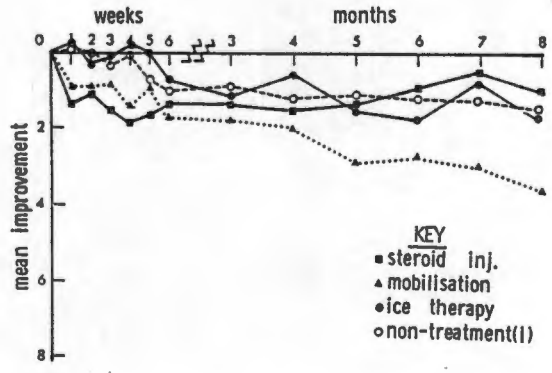


FIGURE I4 : 3. Pain at rest.

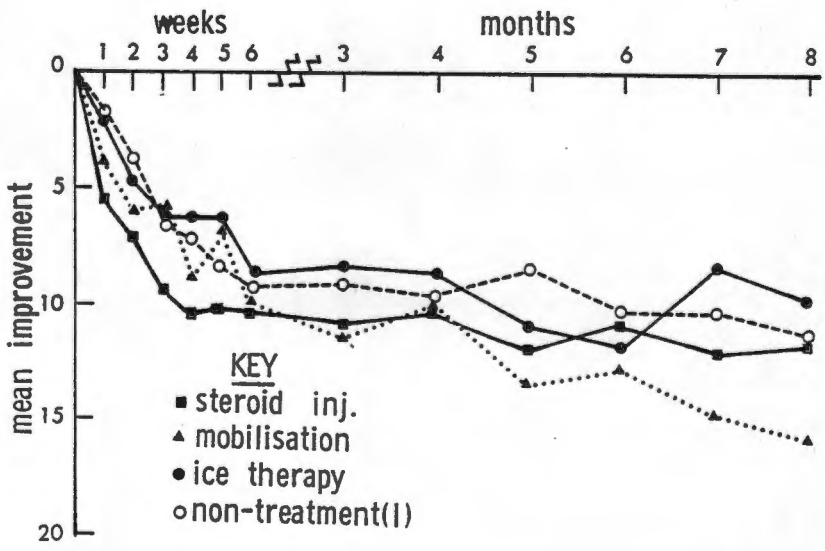


FIGURE I4 : 4. Pain score.

F I G U R E S I4 : 5 - 8. MEAN IMPROVEMENT IN INDIVIDUAL MOVEMENT PARAMETERS IN THE MAJOR FROZEN SHOULDER STUDY TREATMENT GROUPS.

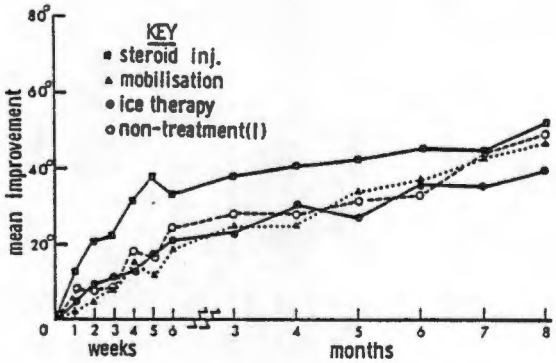


FIGURE I4 : 5. Total flexion.

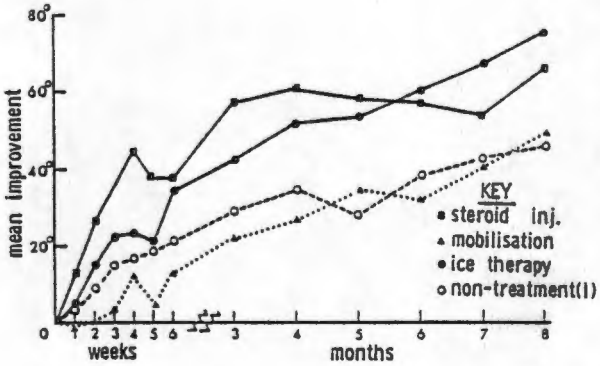


FIGURE I4 : 6. Total abduction.

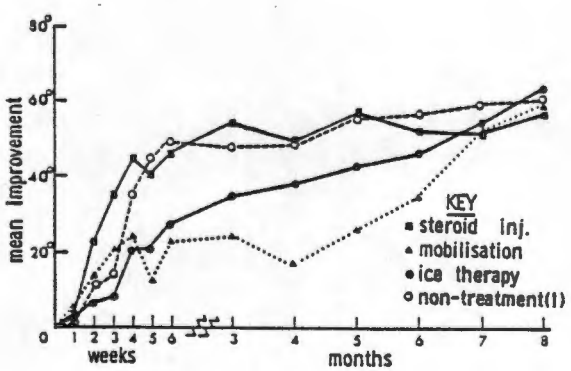


FIGURE I4 : 7. Total rotation.

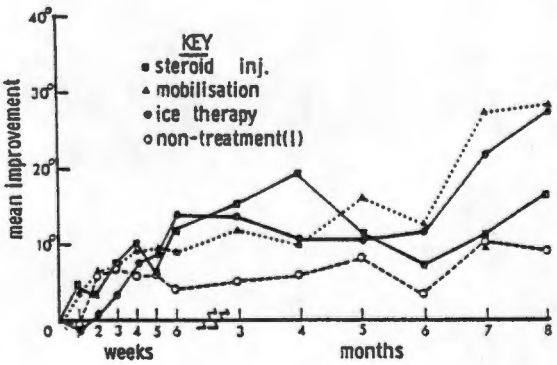
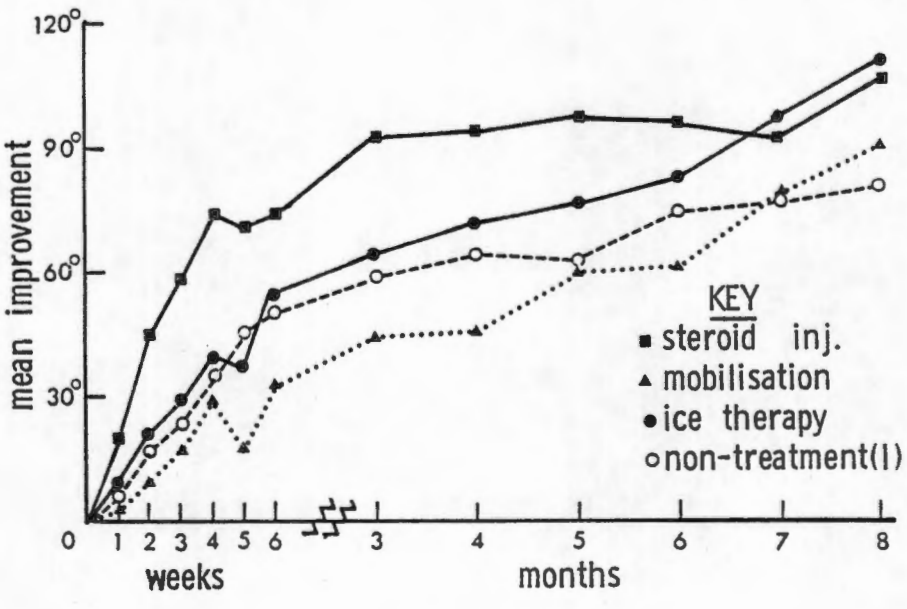


FIGURE I4 : 8. External rotation.



F I G U R E I4 : 9. Comparison of the recovery in the cummulative principal component (ΔC) in the Major Frozen Shoulder Study treatment groups.

F I G U R E S I4 : I0 - I3. MEAN IMPROVEMENT IN PAIN PARAMETERS AND PAIN SCORE IN THE SUPPLEMENTARY FROZEN SHOULDER STUDY TREATMENT GROUPS.

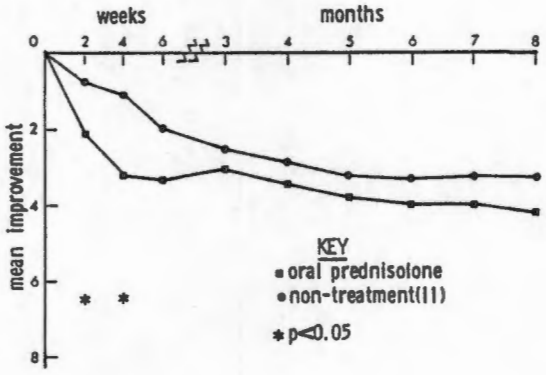


FIGURE I4 : I0. Pain at rest.

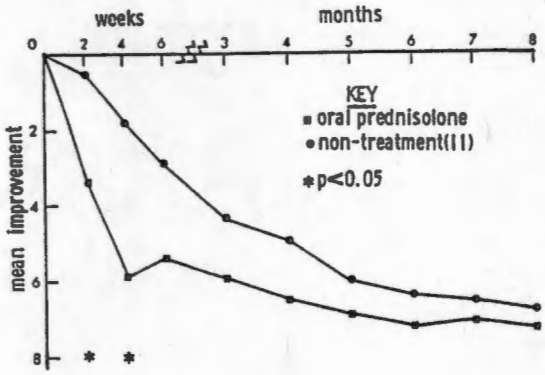


FIGURE I4 : II. Pain at night.

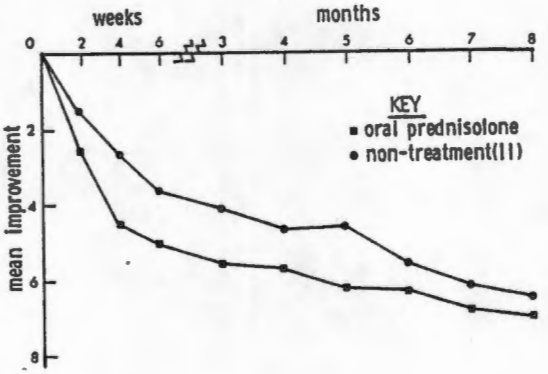


FIGURE I4 : I2. Pain on movement.

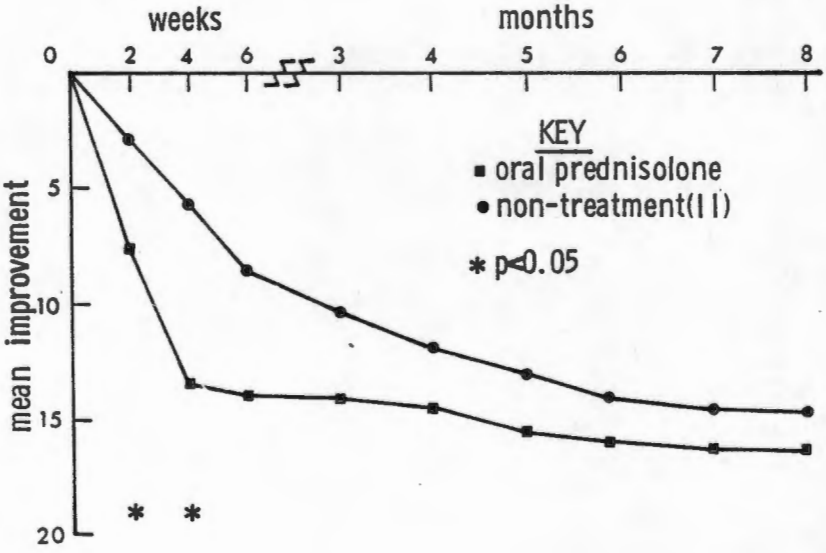


FIGURE I4 : I3. Pain score.

F I G U R E S I4 : I4 - I7. MEAN IMPROVEMENT IN INDIVIDUAL MOVEMENT PARAMETERS IN THE SUPPLEMENTARY FROZEN SHOULDER STUDY TREATMENT GROUPS.

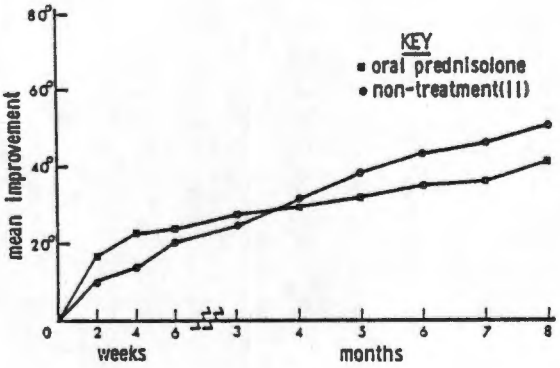


FIGURE I4 : I4. Total flexion.

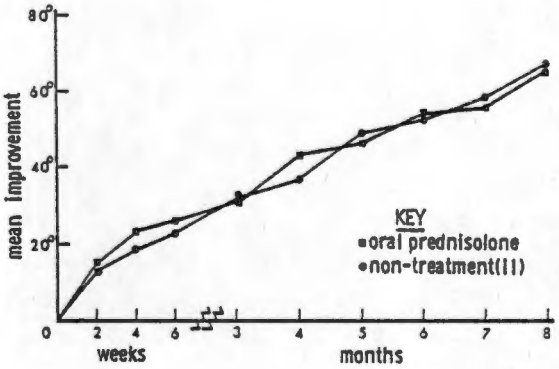


FIGURE I4 : I5. Total abduction.

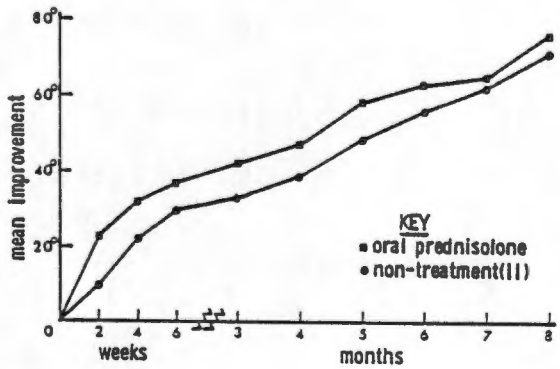


FIGURE I4 : I6. Total rotation.

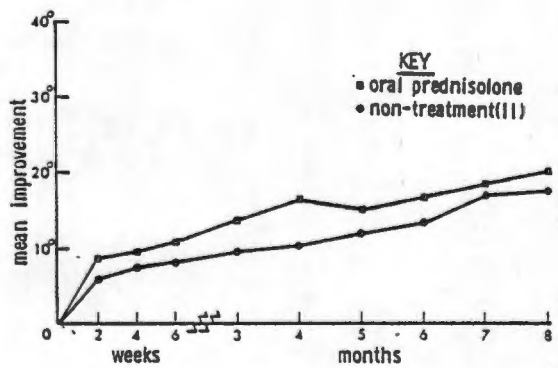
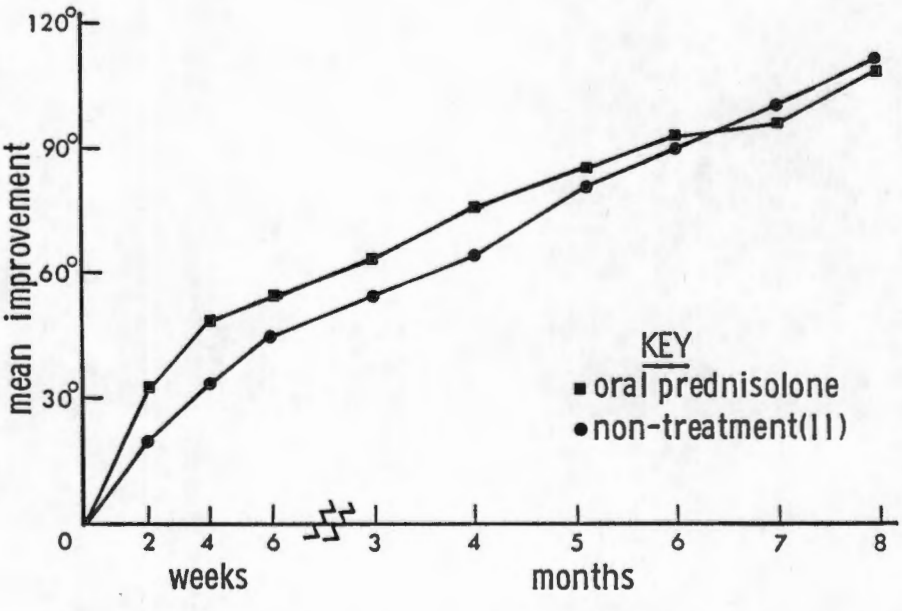


FIGURE I4 : I7. External rotation.



F I G U R E I4 : I8. Comparison of the recovery of the cummulative principal component (ΔC) in the Supplementary Frozen Shoulder Study treatment groups.

PART IV

PERSISTENT ROTATOR CUFF TENDINITIS AND PULSED ELECTRO-MAGNETIC FIELD THERAPY

CHAPTER 15

PERSISTENT ROTATOR CUFF TENDINITIS AND STUDIES OF PULSED ELECTRO-MAGNETIC FIELD (PEMF) THERAPY

Rotator cuff tendinitis whether symptomatic or not, is believed to be the initiating lesion for the painful stiff (frozen) shoulder (45), although documentation of this progression is incomplete (233). Patient with persistent rotator cuff tendinitis lesions were therefore studied to determine if this progression was usual. As the lesion is common, it also permitted study of the earlier tendon lesions. Rotator cuff tendinitis is particularly disabling in patients who do not respond, or show no more than a transient response, to local corticosteroid injections (79), as all other forms of conventional conservative therapy are of limited or unproven benefit (15, 140). Of three hundred and twenty-eight patients with primary shoulder pathology referred to the shoulder clinic over a period of two years (see Chapter 4), 138 (42%) were diagnosed as having rotator cuff tendinitis. Twenty-three percent of this group failed to respond or derived no more than temporary benefit from local corticosteroid injections. This latter group formed the nucleus for the clinical studies. The possible relevance of partial interruption of the blood supply to the rotator cuff tendons by impingement between the humeral head and the acromion (170, 208) to the initiation of tendon degeneration has previously been considered. The conflicting views regarding the importance of trauma (45) and attrition with age (142, 180, 181) has also been presented. Irrespective of the cause of tendon degeneration, once present, this renders the rotator cuff liable to damage by trivial, often unrecognised trauma. As the vascular supply to adult tendon is normally poor, healing of these lesions is slow and often incomplete.

Pulsed electro-magnetic fields (PEMF) have been reported to influence bone repair (10, 11, 191, 245, 262). Similar stimulatory effects have also been observed in nerve regeneration (147, 283), skin ulcer healing (287) and recovery from both soft tissue injuries of the ankle (284) and avascular necrosis of the femoral head (12). In addition, the formation of collagen has been promoted in various model systems (23, 82, 166). These findings prompted study of this therapy in rotator cuff tendinitis lesions which, despite the use of conventional therapy had persisted for over three months. Information on whether these patients with severe and long-standing rotator

cuff tendinitis lesions did show progression to a painful stiff (frozen) shoulder was also sought.

DIAGNOSTIC CRITERIA

The diagnosis of rotator cuff tendinitis (RCT) was based on the criteria of Cyriax (58) - that is shoulder pain being exacerbated by movement against resistance in one or more of the following:-
abduction (supraspinatus tendinitis); external rotation (infraspinatus tendinitis); internal rotation (subscapularis tendinitis). Although the active range of shoulder movement was usually limited by pain, the passive range was approximately normal when compared to the opposite side or (age/sex) matched controls. A 'painful arc' on abduction was often but not invariably present.

Patients were accepted if symptoms had persisted for at least 3 months with no more than transient benefit from previous conservative therapy and if lesions arose spontaneously or followed minor trauma. Patients were excluded if they had severe neck pain, neurological changes in the upper limbs, clinical or radiological evidence of glenohumeral, acromioclavicular or generalised arthritis, radiological calcification of the soft tissues, or a clinical diagnosis of rupture of the rotator cuff or a painful and restricted (frozen) shoulder. Subjects also needed a normal erythrocyte sedimentation rate (Westergren) and Rose-Waaler test for rheumatoid factor.

15 patients with rotator cuff tendinitis of at least 3 months duration were included in a pilot study of PEMF therapy. As the results were promising, a further 29 patients were included in a double-blind controlled study. The data from both these studies will be presented separately.

METHODS

Shoulder Assessment: At each visit the following clinical parameters were recorded:

1. Pain score. This consisted of the sum of the severity of pain at night, on movement and at rest (to the nearest 0.5 cm) as estimated by the patient on visual analogue scales as previously described.

2. Pain on resisted movement. The pain induced by resisted abduction, external and internal rotation was assessed on a 4-point scale and the

results summated (0 = no pain; 1 = slight pain but full power; 2 = moderate pain and reduced power; 3 = severe pain with absent power against even minimal resistance).

3. Painful arc on active abduction was also assessed on a 4-point scale (0 = no pain; 1 = catching only at 1 point; 2 = painful arc; 3 = unable actively to overcome the painful arc).

4. The total range of active movement was calculated as the sum of the range of total abduction, forward flexion and rotation. Measurements were made with a spirit goniometer (41) in an identical manner to the frozen shoulder studies but using active rather than passive movements. The passive range of movement was also recorded to detect progression to a painful stiff (frozen) shoulder, but these measurements were not used for serial assessment.

5. Paracetamol tablets were permitted during both studies but non-steroidal anti-inflammatory agents were stopped. The number of analgesic tablets used each week was recorded and used as a parameter for serial assessment in the pilot but not the controlled study.

Patients were reviewed fortnightly while undergoing therapy and monthly thereafter.

The time at which the patient and the observer considered the shoulder much improved was also recorded.

PEMF Regimen: A single ovoid coil ($12.2\pm1.2 \times 13.2\pm0.7$ cm^2) consisting of 50 turns of copper wire (1.4 mm diameter) covered by insulating tape was fitted over padding to the outer aspect of the affected shoulder so that it protruded through the centre. Two velcro straps held it in place. Patients were instructed to use the coil for 5 - 9 hours each day. Treatment sessions had to last at least 1 hour at any time. The coils were supplied with pulse generators set at 73 ± 2Hz; waveform parameters (Figure 15:1) for each active coil were regularly checked so they did not vary by more than 7%. For the controlled study, some patients initially used dummy coils which were identical to the active coils but provided no electro-magnetic stimulation.

PILOT STUDY

PATIENTS: Clinical details of the 15 patients included in the pilot study are shown (Table 15:1). Previous therapy given to these patients will be considered together with the controlled study. All the patients used active coils for 4 - 8 weeks but remained under follow-up for at least 20 weeks. One patient failed to complete 4 weeks therapy and was withdrawn from the study.

RESULTS: The improvement in the mean score for pain, painful arc on abduction, pain on resisted movement, active range and the decrease in analgesic consumption is summarised in Table 15:2. The maximum improvement in all clinical parameters was noted within 4 weeks of initiating therapy. By 16 weeks, 7 patients (50%) were asymptomatic; 5 (35%) patients had only minor residual disability; 2 were unchanged and 1 had developed a painful stiff shoulder.

SIDE-EFFECTS: One patient with a past history of cervical spondylosis and a cervical laminectomy had recurrence of neck pain and neurological abnormality in both upper limbs following successful treatment of the shoulder. Therapy was otherwise well tolerated.

DOUBLE-BLIND STUDY

STUDY DESIGN: Patients fulfilling the entrance criteria were randomly allocated to the treated group (A), or the control group (B). In the first 4 weeks (phase I), group A received active coils and group B received dummy coils. Neither patient nor medical assessor was aware of the treatment group. At the end of the 4 weeks and without breaking the code, both groups were given active coils and therapy was continued for another 4 weeks (phase II). Treatment was then stopped but patients continued to be reviewed for another 8 weeks (phase III), at the end of which the grouping was revealed to patient, medical assessor and others involved in the study. After the code was broken patients were either discharged or offered alternative therapy.

STATISTICAL ANALYSIS: The rate of recovery of each clinical parameter in the two groups from time 0 to each follow up visit was compared by the use of Wilcoxon rank sum tests.

RESULTS:

Patients: 29 fulfilled the criteria for the study. 15 entered the treated group (A) and 14 the control group (B). There were no significant differences between the groups (Table 15:3). The supraspinatus tendon was most commonly affected, although this was often in association with infraspinatus involvement. All the lesions arose spontaneously.

Twenty-eight of the 29 patients had received non-steroidal anti-inflammatory agents (NSAIDS) before inclusion in the study, but only 3 patients (12%) found them beneficial. Analgesics on the other hand were considered to be of some value in 8 of the 18 patients (44%) who had received this therapy. A particular benefit was reduction of night pain facilitating sleep. Twenty-seven of the 29 patients had also received local steroid injection (mean 2.2 injections) therapy, but not within 1 month of inclusion in the study. Two patients (1 in each group) had refused injection therapy because of the pain induced by this therapy during treatment of lesions in the opposite shoulder. In the pilot study, 2 of 13 (15%) patients found NSAIDS helpful and 5 of 12 (42%) found analgesic tablets of value. All these patients had prior local corticosteroid injection (mean 3.5 injections) therapy.

Phase I (Week 0 - 4): The improvement in the mean pain score (Figure 15:2) was greater in the treated than the control group at both 2 ($p<0.05$) and 4 ($p<0.02$) weeks. The reduction in the scores for the painful arc on abduction (Figure 15:3) and for pain on resisted movement (Figure 15:4) and the improvement in range (Figure 15:5) showed an even greater advantage ($p<0.005$) for group A, at both 2 and 4 weeks.

Phase II and III (Weeks 4 - 16+): With the rapid improvement in group B after the change to active coils (Figures 15:2-5), there was no significant difference between the groups in any of the clinical parameters at week 6. Although 7 patients (25%) had a minor flare-up of symptoms after stopping therapy (phase III), this resolved without further treatment.

Outcome: Nineteen of 29 patients (66%) were symptom-free at week 16 (Table 15:4) and were discharged. Of the 10 who remained under follow-up, 2 became symptom-free; 3 were discharged with only minor residual disability; 2 had pain and tenderness at the acromioclavicular joint and received local steroid injections; and 3 had no improvement despite further steroid injections into the subacromial bursa. No untoward reactions occurred

during the study, although many patients found the coils cumbersome especially at night.

Timing of Maximum Improvement: If the 4 weeks of placebo therapy in group B were disregarded, maximum subjective pain relief occurred at a mean of 5.7 weeks (range 2 weeks to 6 months). Full recovery on examination was, however, delayed until 13.2 weeks (4 weeks to > 6 months) from initiating therapy.

Factors of Possible Relevance to Outcome: Nineteen patients were able to sleep with the coils in situ and achieved treatment times of 6 - 10 (mean 8.2) hours per day. The other 10 achieved treatment times of only 3 - 8 (mean 4.7) hours per day during waking hours. Four patients (all group A) refused therapy after four weeks since symptoms has resolved. Thus 11 patients had active therapy over 8 weeks and 18 patients over only 4 weeks. However, the duration of therapy did not affect the outcome. The duration of symptoms before the start of active therapy was also not important; but outcome was influenced by the severity of the lesion and in particular, involvement of more than one tendon which was present in 8 of the 10 controlled study (and all 3 pilot study) patients who were still symptomatic at 4 months. The development of acromioclavicular tenderness (2 patients) was also associated with a poor result. Three of the 5 patients with recurrence of severe pain during the extended follow-up period were manual labourers and this activity was considered responsible for several of the minor and temporary flares following cessation of therapy.

DISCUSSION

Rotator cuff tendinitis is extremely common and is usually responsive to local corticosteroid injections, but 23% of these patients at the shoulder clinic responded poorly or only transiently to this treatment. The response to non-steroidal anti-inflammatory agents is usually unsatisfactory, but analgesics may be of some value, especially in patients in whom night pain is a prominent feature. The design of the controlled study was chosen because many patients in the pilot study improved rapidly with PEMP therapy. In the first phase, the treated group had a significant advantage over the placebo-treated control group, but the latter group soon improved once they were put on active therapy and no significant differences were noted thereafter. Patient and observer bias was minimised by maintaining the code relating to treatment groups until all the patients had completed 4 months of follow-up.

Rotator cuff tendinitis, like many soft tissue lesions, offers few objective clinical parameters for use in serial follow-up. All 4 parameters chosen for this study reflect pain and therefore presumably severity of the underlying condition. All improved more with treatment than placebo. The pain score, based on visual analogue scales, was the most subjective parameter and showed a greater placebo effect and lesser (but still significant) advantage for the treated group than did the other 3 parameters, which were based on observer assessment. Subjective improvement was noted by many of the patients before objective evidence of healing was recorded by the medical assessor. This difference suggests that there may be other local effects which induce pain relief.

Treatment was generally well tolerated, although many patients found the treatment coils cumbersome and uncomfortable. The duration of therapy varied from 3 - 10 h per day over 4 - 8 weeks, but there was no evidence that either longer daily treatment sessions or longer overall treatment times conveyed an advantage. The stimulation signal pulse used was empirically based on other clinical (11) and experimental (82) data.

More than 70% of patients improved on PEMF therapy. All the patients were followed up for at least 4 months, with few showing evidence of recurrence during this time. This study suggests that PEMF therapy is safe and may be effective in the treatment of persistent soft tissue lesions such as rotator cuff tendinitis which have been resistant to conventional conservative measures.

Two of the 44 patients with persistent rotator cuff tendinitis lesions developed some restriction in the passive range of movement while under review. However, the loss of range was not severe enough to fulfill the criteria for a frozen shoulder. All the patients in the PEMF studies were encouraged to keep their shoulders moving and this may have influenced the clinical features.

FURTHER CLINICAL STUDIES OF PEMF

A pilot study of PEMF therapy was also carried out in 15 other patients with tendon lesions which had persisted despite therapy for at least 4 months.

PATIENTS

1. The Frozen Shoulder Group: 6 patients with frozen shoulder according to the previously described criteria were studied. All these patients were symptomatic despite prior local corticosteroid injection (mean 1.5 injections) therapy. All except 1 patient had received NSAIDS with no benefit.

2. The Tennis Elbow Group: 9 patients with tennis elbow were studied. All had received local corticosteroid (mean 5.3 injections) therapy and had used NSAIDS with no more than transient benefit.

Details of patients included in the 2 groups is summaried in Table 15:5. Patients with neck pain, acromioclavicular joint tenderness or motor or sensory abnormality in the affected limb were excluded. All the patients had a normal sedimentation rate (Westergren), Rose-Waaler test for rheumatoid factor and normal plain X-ray of the affected region (shoulder or elbow).

METHODS

Clinical Assessment: Serial assessment of frozen shoulder patients included pain score (sum of pain at night, at rest and on movement), total passive range (sum of total flexion, abduction and rotation) and mean weekly paracetamol consumption. Tennis elbow patient assessment was with a pain score (10 cm horizontal visual analogue scales) and grip strength with the elbow extended and forearm pronated. The response to therapy in both frozen shoulder and tennis elbow groups on discharge was assessed as much improved, slightly improved or unchanged.

PEMF Regimen: a single coil identical to that used in the tendinitis studies was fitted to the affected shoulder or elbow and treatment given as previously described using a similar regimen. All the coils were active and therapy was given over an 8 week period. Shoulder patients were asked to perform pendular exercise for 2 - 3 minutes each hour as previously described.

RESULTS

1. The Frozen Shoulder Group: The improvement in the clinical parameters

with time (Table 15:6) suggested possible benefits for this therapy. All 6 patients considered themselves much improved and this was substantiated on examination.

2. The Tennis Elbow Group: The improvement in the clinical parameters is shown (Table 15:7). 2 patients withdrew before completing therapy due to lack of improvement and the discomfort induced by prolonged immobilisation. A third patients withdrew from follow-up after 8 weeks of therapy with no benefit. The response to therapy (Table 15:8) showed marked improvement in 5 patients (56%). The patient who was slightly improved by the therapy relapsed within 8 weeks of completing therapy.

DISCUSSION

Soft tissue lesions such as frozen shoulder and tennis elbow which becomes chronic or recurrent can considerably disrupt work or hobbies but are only rarely considered severe enough to justify surgery, usually only after prolonged attempts at conservative treatment. Only steroid injection has been shown to enhance resolution, but not necessarily prevent recurrence. All 15 patients with frozen shoulder or tennis elbow had received local corticosteroid injections with no more than transient benefit. The 6 patients with frozen shoulder showed a satisfactory rate of recovery, but as these patients also performed home pendular exercise, the true value of PEMF requires further controlled study. Only five of the nine patients with lateral epicondylitis were improved, but all these patients had severe and chronic lesions. Some modification in the coil design may improve patient compliance and possible efficacy.

The studies of PEMF have suggested a possible use in the treatment of tendon lesions which show no more than a transient benefit to conventional conservative therapy.

TABLE 15:1

CLINICAL DETAILS OF ROTATOR CUFF TENDINITIS PATIENTS IN THE PILOT STUDY (N=15)

Age (Years)	Mean (Range)	57.2 (30-80)
Sex	Male : Female	8 : 7
Arm	Dominant : Non-dominant	11 : 4
Onset	Spontaneous : Traumatic	13 : 4
Duration at Presentation (months)	Mean (Range)	8.3 (3 - 18)
Tendon Lesion(s)	Supraspinatus	9
	Supraspinatus and Infraspinatus	6

TABLE 15:2

MEAN SCORE OF CLINICAL PARAMETERS DURING FOLLOW-UP OF THE PILOT STUDY (N = 14*)

		Mean Score					
Parameter	Week	0	2	4	6	12	20
Pain Score		14.5	6.6	3.1	4.5	2.3	1.3
Painful Arc of Abduction		2.5	1.1	0.6	0.5	0.3	0.1
Pain on Resisted Movement		3.6	2.3	1.9	2.0	1.2	0.1
Active Range of Movement		184^{o}	233^{o}	237^{o}	248^{o}	244^{o}	247^{o}
Analgesic Use/Week		20.8	12.3	6.0	4.4	2.5	0.5

* = 1 Patient Withdrawn

TABLE 15:3

CLINICAL DETAILS OF ROTATOR CUFF TENDINITIS PATIENTS IN THE CONTROLLED STUDY

	CONTROLLED STUDY	
	Treated Group (N = 15)	Control Group (N = 14)
Mean Age (Years)	54.4	53.2
Sex Male : Female	10:5	11:3
Duration at Presentation (Months)		
Mean	9.2	9.5
Range	3-24	3-24
Arm - Dominant : Non-dominant	9:6	10:4
Tendon Lesions		
Supraspinatus	8	6
Supraspinatus & Infraspinatus	5	5
Infraspinatus	2	1
Subscapularis	-	2

TABLE 15:4

STATUS OF PATIENTS 16 WEEKS AFTER ENTRY INTO THE CONTROLLED STUDY

Status at 16 Weeks	Treated Group (N = 15)	Control Group (N = 14)
Asymptomatic	9	10
Minor Residual Symptoms	5	2
Severe Disability	1	2

TABLE 15:5

CLINICAL DETAILS OF PATIENTS IN THE PILOT FROZEN SHOULDER AND TENNIS ELBOW PEMF STUDIES

		Frozen Shoulder (N = 6)	Lateral Epicondylitis (N = 9)
Mean Age (Years)		58.8	40.5
Sex - Male : Female		5:1	4:5
Duration at Presentation (Months)			
	Mean	10	24
	Range	5-18	7-84

TABLE 15:6

IMPROVEMENT IN MEAN CLINICAL PARAMETERS WITH TIME IN FROZEN SHOULDER PATIENTS TREATED WITH PEMF (N = 6)

Week	0	2	4	6	12	20
Analgesics/Week	12.2	5.1	3.2	2.5	0	0
Pain Score	15.4	10.0	5.0	2.4	0.2	0.5
Total Passive Range	235^{o}	283^{o}	329^{o}	367^{o}	398^{o}	433^{o}

TABLE 15:7

IMPROVEMENT IN MEAN CLINICAL PARAMETERS WITH TIME IN TENNIS ELBOW PATIENTS TREATED WITH PEMF (N = 9)

Week	0	2	4*	6**	8***	12
Pain Score	3.5	2.4	2.6	1.4	0.2	0.2
Grip Strength (mm Hg)	138	176	198	260	293	293

* 1 Patient Withdrawn

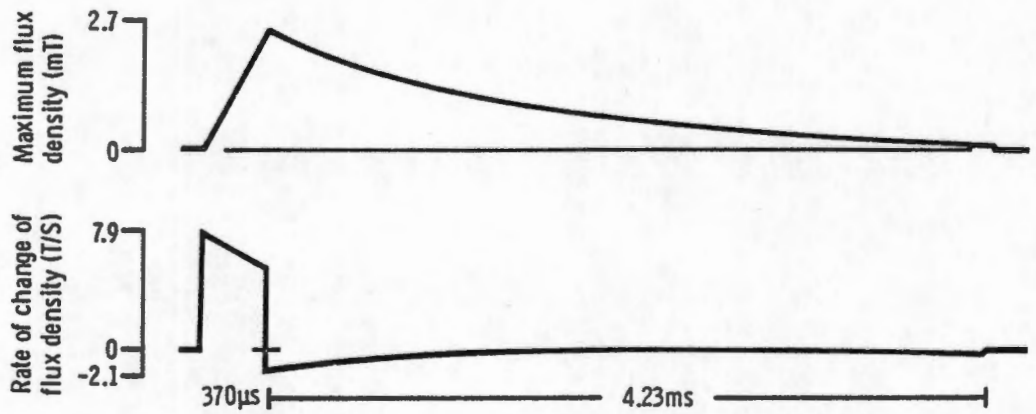
** 2 Patients Withdrawn

*** 3 Patients Withdrawn

TABLE 15:8

RESPONSE OF TENNIS ELBOW PATIENTS TO PEMF THERAPY

	N
Greatly Improved	5
Slightly Improved	1
Unchanged	3



F I G U R E I5 : I. Wave forms showing the magnetic flux density (TOP) and rate of change of magnetic flux density (BOTTOM) as measured by a Hall probe and search coil (7mm diameter), respectively.

F I G U R E S I5 : 2 - 5. IMPROVEMENT WITH TIME IN THE CLINICAL PARAMETERS IN THE TREATED AND CONTROL ROTATOR CUFF TENDINITIS GROUPS.

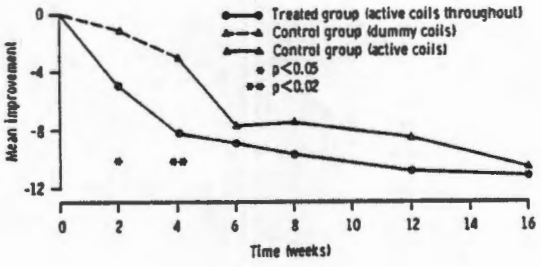


FIGURE I5 : 2. Pain score.

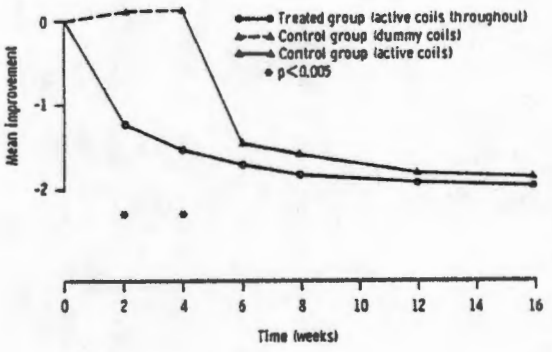


FIGURE I5 : 3. Painful arc on abduction.

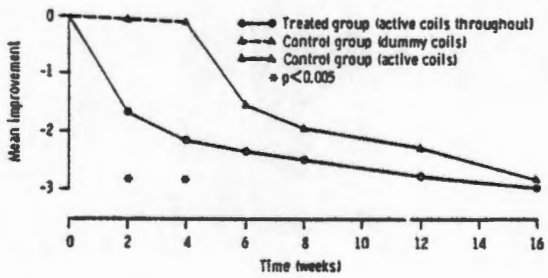


FIGURE I5 : 4. Pain on resisted movement.

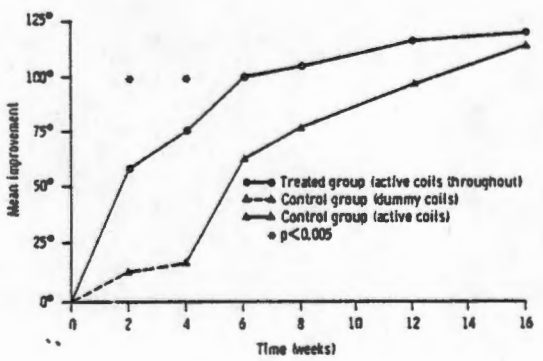


FIGURE I5 : 5. Active range.

PART V

LATERAL HUMERAL EPICONDYLITIS - TENNIS ELBOW

INTRODUCTION

As the anatomy and function of the shoulder is so complex and the evaluation of conditions in this region so difficult, lateral epicondylitis which is discrete superficial and easy to diagnose clinically was chosen for study, despite some disagreement on its pathology and pathogenesis.

Runge (1873) (239) first described the clinical features under the title "writers cramp". Morris (1882) (186) called it the "lawn tennis elbow", a sporting connotation which has persisted despite the recognition that this is an uncommon cause of the condition. Lateral humeral epicondylitis was proposed as a more accurate term, but general agreement on nomenclature has not been reached and tennis elbow and lateral (humeral) epicondylitis are used synonymously.

CHAPTER 16

REVIEW OF THE LITERATURE RELATING TO PATHOLOGY AND PATHOGENESIS OF LATERAL EPICONDYLITIS

PATHOLOGY

Degeneration and tears of the common extensor tendon (CET) close to its origin is the most widely accepted pathological lesion in tennis elbow, but many other local lesions have been proposed, on the basis of the few patients treated surgically after prolonged conservative therapy. It is uncertain if the abnormalities described are pathological or reflect the damage caused by local steroid injections and other therapy.

The lesion was initially thought to be a 'traumatic periostitis' (123, 239, 266, 276), but Carter (1925) (38) could not verify this and Hansson and Horwich (1930) (109) demonstrated experimentally that periosteal avulsion was more likely. Extra-articular bursitis was described by Osgood (1922) (209) and others (67, 213, 255) sometimes with calcification (35), but Goldie (1964) (94) in an extensive surgical study found no evidence of a bursa or inflammation or soft tissue calcification at this site. He found granulation tissue both deep to and involving the CET. Hyaline degeneration (20) or tears (182) of the orbicular ligament and chondromalacia (21) or fibrillation (202) of the radial head have been reported. Radio-humeral synovitis with pinching of synovial fringes (3, 184, 265) and radio-ulnar arthritis (80, 236) have also been thought important in some cases.

Cyriax (1936) (57) reviewed over 30 suggested sites of pathology and favoured degeneration in the CET close to its origin. Meherin and Cooper (1950) (178) and Sinclair (1965) (250) found definate evidence of tendinitis at this site and Spencer and Herndon (1953) (254) declared the tendon to be the only site of pathology in 93 operations over 10 years. Tears of the lower end of the CET have often been noted (50, 91, 121, 122) and Sarkar and Uhthoff (1980) (242) reported ultra-structural evidence of degeneration, tears and healing of the tendon at this site. Calcification has also been reported at this site (128, 173, 178, 268), but Cowan and Stone (1952) (54) found no relationship between the extent of calcification in the tendon and the severity of symptoms. Niepal and Sitaj (1979) (203) proposed enthesopathy as the underlying pathology. Lesions affecting the

extensor carpi radialis brevis (36, 57, 90, 204, 260), pronator teres (185) and supinator tendons (236) have been suggested and surgical treatment directed at these sites.

Nerve entrapment has been considered as a common cause of resistant tennis elbow (33, 65, 92, 146, 210, 235, 253, 285), with the posterior interosseous branch of the radial nerve being singled out for particular mention. Carpener (1966) (34) reported good results in 8 out of 10 chronic tennis elbow lesions treated by nerve decompression. Werner (1979) (275) in an extensive study concluded that 5% of resistant lesions could be attributed to nerve entrapment. Van Rossum et al (1978) (269) could not substantiate entrapment as a cause and Hagert et al (1977) (104) considered the 2 diseases as separate entities. Case reports of idiopathic posterior interosseous nerve compression (27, 120, 190) have stressed weakness and not pain. Kaplan (1959) (137) proposed radial neuritis as a cause, but this has not been substantiated. The association between tennis elbow and cervical spondylosis with radiculopathy (102, 134, 195, 247, 259) has been mentioned previously.

PATHOGENESIS

The excessive use of specific muscles was proposed by Runge (1873) (239). Edgar (1929) (76) felt this diverted blood away from the tendon to the active muscle predisposing to tendon degeneration. Forced gripping (76, 183, 250) and repeated pronation and supination (57, 91, 184, 186, 271) have been considered important. Trauma has been considered as the main cause by some early workers (20, 38, 163, 236), but is not a common cause (89, 90, 121, 193) in most series. Hohl (1961) (121) felt that repeated minor trauma led to attrition of the tendon. Uhthoff and Sarkar (1980) (242) considered the lesion to be multifactorial with age-related degeneration and repetitive movements being important.

While overuse or repetitive activities during manual labour, housework and hobbies are often mentioned as precipitating factors, many cases remain unexplained.

DISCUSSION

A historical review of the pathology and pathogenesis of lateral epicondylitis seems to high-light the marked disagreement expressed by different workers. While many of these hypotheses have not been subjected

to critical analysis, the predominant view at the present time is that tennis elbow constitutes a degenerative process affecting the common extensor tendon at or close to its insertion. Niepal and Sitaj (203) considered the lesion to be an enthesopathy which was very similar to the rotator cuff lesions of the shoulder. The pathogenesis of the lesion is probably multifactorial with age-dependant tendon degeneration and repetitive rotatory movements playing an important role.

CHAPTER 17

CLINICAL FEATURES OF LATERAL EPICONDYLITIS ; PATIENTS INCLUDED IN THE PROSPECTIVE AND RETROSPECTIVE STUDIES

Runge (239) considered the disease to be occupational and Hohl (121), Goldie (94), Sinclair (250) and Kivi (143) documented its preponderance in some occupational groups. Morris (186), Nirschl (205), Gruchow and Pelletier (100) and Carroll (37) assessed the incidence of the lesion in tennis players and found it to be common. However, most series assessing the lesion in the general population show tennis to be an uncommon cause of symptoms (90). While toolwork, sport, hobbies or trauma often (94) precipitate symptoms, nearly half the patients in most series are not aware of any causative factors (121, 250).

A review of the literature suggests that females are affected as frequently as males (50), with onset occurring from 30 - 65 years with a mean of 42 - 44 years in most series (50, 90, 94, 121, 235). Onset is rare before 20 or after 70 years of age. The marked dominant arm preponderance is documented in all reports (50, 94, 121, 235), although the condition can be bilateral (50, 205). Symptoms usually arise insidiously but can begin suddenly.

SYMPTOMS

Patients with tennis elbow experience pain around the lateral aspect of the elbow which is worse when the forearm extensors contract against resistance. Weakness of grip strength is often noted (94). The pain may radiate down the forearm and night pain and morning stiffness of the elbow sometimes occurs.

SIGNS

Severe local tenderness near the lateral epicondyle is the most striking feature. While this tenderness is usually anterior to the lateral epicondyle, the site of maximum tenderness can vary (61, 90, 94). The elbow pain is exacerbated by forced wrist dorsiflexion and sometimes by supination and pronation against resistance. Grip strength is always reduced due to pain. The active and passive range of elbow movement is usually normal, but is severe cases a few degree of elbow extension can be lost.

PATIENT GROUPS

141 patients with lateral epicondylitis were studied:-

A. The 'Prospective Group'

Seventy-six patients suffering from lateral epicondylitis (Table 17:1) of at least one month's duration were included in the study. Criteria for inclusion were pain and localised tenderness near the lateral epicondyle of the humerus with exacerbation of pain on resisted wrist dorsiflexion and reduction in grip strength. Local arthritis of the elbow (clinical or radiological), generalised polyarthritis or neurological abnormality in the upper limbs resulted in patient exclusion. Patients who had a single steroid injection were included if symptoms persisted for at least one month. However, more than one injection resulted in patient exclusion.

B. The 'Retrospective Group'

Seventy-seven patients treated in the rheumatology outpatient department in Cambridge from 1 - 5 years previously were sent questionnaires with regard to therapy, side-effects, initial outcome and subsequent course. Where possible the information was verified from the case records. Sixty-five of the 77 patients completed the questionnaires - 3 had died and 9 could not be traced. Mean follow-up was 39.5 (range 12 - 60) months.

PATIENT DETAILS

Patients: Of 141 patients included in the 2 groups, 79 were female and 62 male. Age range at presentation was 29 - 65 (mean 43.5) years. Treatment was to the dominant arm in 106, non-dominant in 31 and both arms in 4 cases. The duration of symptoms at presentation varied from 1 - 12 (mean 4.5) months in the prospective group. This information was not available from the retrospective group. The clinical details are summarised in Table 17:1.

Aetiology: Seventy-four patients (52%) attributed symptoms to specific activities especially housework, toolwork, lifting or sports (Table 17:2). Trauma to the elbow was an uncommon cause. The other 67 patients (48%) could not recall any causative factors.

Occupation: The occupations of the patients at the time of presentation

are shown (Table 17:3). The majority were housewives, domestic workers, sedentary workers or were unemployed and only 30 (21%) did manual labour.

Hobbies: Gardening, home decoration (do it yourself) and racket sports were the most common hobbies undertaken by the tennis elbow patients before the onset of symptoms (Table 17:4).

Therapy Before Presentation: This information was only recorded for the prospective group (Table 17:5). Non-steroidal anti-inflammatory agents and simple analgesics were frequently prescribed, but were rarely considered beneficial by the patients.

CLINICAL PARAMETERS

No accepted clinical parameters have been described for the routine prospective study of tennis elbow. Tests used for serial clinical assessment of the prospective group were:-

1. A pain score using 10 cm horizontal visual analogue scales.
2. Pain (0-3) induced by resisted wrist dorsiflexion (0 = no pain; 1 = mild pain but normal power; 2 = moderate pain and reduced power; 3 = severe pain and absent power).
3. A weight test (250) (0-3) which assessed the ability to lift weights of 4, 2 and 1 pounds (1.8, 0.90 and 0.45 kg) with the elbow extended and forearm pronated.
4. Grip strength (204) was performed using a 300 mm Hg spring-coil gauge attached to a rubber bag pre-set to 30 mm Hg. An average of 3 estimations was taken and recordings taken both with the elbow (a) in a position of flexion (and supination) and (b) extension (and pronation).

Inter-observer Assessment of Clinical Parameters

Twenty-five patients with tennis elbow lesions were examined by 2 independent observers (A & B), at the same time and using an identical examination technique. As the position of the elbow seemed to influence the result, it was carefully standardised for the assessments. Pain on resisted wrist dorsiflexion and the weight test were performed with the forearm pronated and the elbow extended as far as possible. The severity of local tenderness was assessed with the elbow flexed and forearm pronated. A 4-point scale of severity was used (0 = no tenderness; 1 = slight/diffuse tenderness; 2 = moderate tenderness; 4 = severe tenderness).

Grip strength was assessed both in flexion (with the forearm supinated) and extension (with the forearm pronated).

As the inter-observer differences for pain on resisted wrist dorsiflexion, weight test and tenderness take in very limited values - the maximum scale being -2, -1, 0, 1 and 2, the paired t-test was not applicable. Comparision of the results of the individual parameters using the 'sign test', showed no difference between observers A and B for pain on wrist dorsiflexion or the weight test, but did show a significant difference ($p<0.05$) for tenderness. The latter parameter was therefore not used in serial assessments. Comparison of grip strength using paired t-tests showed no difference between the observers when the arm was assessed in a similar position (Table 17:6). When assessments were compared by the same observer in flexion and extension, using paired t-tests, a significant difference ($p<0.001$) was found (Table 17:7). Although the effect of the position of the arm was not studied in a similar way with the other clinical parameters, care was taken to carefully standardise this factor in the routine assessments.

The site of maximum tenderness was assessed in the Prospective patients and was not always the same. In 58 (76%) it was anterior to the lateral epicondyle and in 18 cases (24%) posterior. This feature was documented at each follow-up visit to determine its prognostic significance.

DISCUSSION

The clinical features in the patients were similar to those in the medical literature in all aspects except the sex of the patients, with females outnumbering males in this study. Previous studies have shown no difference or a male preponderance (121, 143, 193), possibly due to patient selection on the basis of occupation (143), sport (37, 100, 205) or a need for surgery (94, 267).

The clinical parameters chosen for serial assessment were based on tests used in previous studies, but the inter-observer error was also evaluated for each parameter.

While non-steroidal anti-inflammatory agents were commonly prescribed to patients with tennis elbow, they were of little value. Simple analgesics were helpful in some patients.

TABLE 17:1

CLINICAL DETAILS OF TENNIS ELBOW PATIENTS

		Prospective Group (N = 76)	Retrospective Group (N = 65)
Sex -	Female : Male	48 : 28	31 : 34
Age (Years)	Mean	43.3	45.1
	Range	29 - 65	29 - 62
Arm -	Dominant : Non-Dominant	56 : 20	50 : 11
Duration at Presentation (Months)			
	Mean	4.5	-
	Range	1-12	-

TABLE 17:2

AETIOLOGY OF TENNIS ELBOW LESIONS IN THE PROSPECTIVE AND RETROSPECTIVE STUDY GROUPS

	Prospective Group (N = 76)	Retrospective Group (N = 65)
Housework / Domestic Work	11	16
Toolwork	9	8
Lifting/Carrying	8	4
Sport/Hobbies	7	5
Trauma	4	2
No Cause	37	27
Can't Remember	-	3

TABLE 17:3

OCCUPATIONS OF TENNIS ELBOW PATIENTS AT THE TIME OF PRESENTATION

Occupation	Prospective Group (N = 76)	Retrospective Group (N = 65)
Sedentary/Unemployed	25	25
Housework/Domestic	30	18
Manual/Toolwork	13	17
Secretarial	8	5

TABLE 17:4

HOBBIES UNDERTAKEN BY THE TENNIS ELBOW PATIENTS AT THE TIME OF PRESENTATION

	Prospective Group (N=76)	Retrospective Group (N=65)
Gardening	47	38
Do It Yourself	20	23
Racket Sports	12	10
Golf	6	6
Darts	4	6
Knitting	6	9
No Hobbies	19	12

TABLE 17:5

THERAPY (AND ITS EFFICACY) GIVEN TO THE PROSPECTIVE TENNIS ELBOW PATIENTS BEFORE PRESENTATION (N = 76)

	N	Effective	Ineffective
Non-steroidal Anti-Inflammatory Agents	60	4	56
Analgesic Tablets	38	6	32
Immobilisation	4	0	4
Single Steroid Injection	10	6*	4

* Symptoms Recurred in 6 - 12 Weeks

TABLE 17:6

COMPARISON OF GRIP STRENGTH BY TWO INDEPENDENT OBSERVERS

	Observers		Paired T-test	
	'A'	'B'	T_{24}	p<
Grip Strength in Flexion (mmHg)	192.6	191.9	0.424	N/S
Grip Strength in Extension (mmHg)	133.1	131.3	0.748	N/S

N/S = Not Significant

TABLE 17:7

COMPARISON OF GRIP STRENGTH IN FLEXION AND EXTENSION BY THE SAME OBSERVER

	Grip Strength		Paired T-test	
	Flexion	Extension	T_{24}	p<
Observer 'A'	192.6	133.1	5.078	0.001
Observer 'B'	191.9	131.3	5.238	0.001

CHAPTER 18

A CLINICAL AND THERMOGRAPHIC STUDY OF LATERAL EPICONDYLITIS

The investigation of most soft tissue lesions has been hampered by a lack of objective reproducible parameters which permit serial assessment. As a pilot study of tennis elbow suggested that thermography may produce a consistent abnormality, this lesion was chosen for further study. Whilst the clincal diagnosis is usually simple, some differences in the site of localized tenderness have been noted (57, 61).

The aim of this study was to examine the clinical and thermographic features of tennis elbow with regard to their diagnostic and prognostic significance and the possible application of thermography to the investigation of other soft tissue lesions.

PATIENTS

Fifty-six of the patients from the prospective tennis elbow study were studied. 39 were female and 17 male. The dominant arm was involved in 50 and the non-dominant arm in 6 cases. Mean age was 43 years and mean duration at presentation was 4.5 months (range 1-12 months). Trauma initiated symptoms in three cases. Eight were attributed to sport, seven to manual work and the other 38 had a spontaneous onset.

METHODS

Clinical: The clinical assessment of severity was based on the 5 parameters used in the prospective study (see Chapter 17), each judged from 0-3. The pain score, pain induced by resisted wrist dorsiflexion and the weight test were judged as previously described. Grip strength in flexion and extension was measured in mmHg and the range divided to fit the 4-point scale shown (0 = >280; 1 = 205-275; 2 = 105-200; 3 = <100 mmHg).

Summation of the score for the individual parameters gave an overall 'clinical score'. The exact site of tenderness and presence or absence of pain on resisted pronation and supination were recorded.

Infrared Thermography: AGA 680M thermographs were taken of the lateral aspect of both elbows after 15 minutes stabilization at 20°C. All

thermographs were taken at 1 meter using a 0.5°C sensitivity setting. Data was then analysed via an OSCAR interface and recorded on tape, allowing numerical analysis of the grey areas in an area of interest (of constant size) using an Apple II microcomputer. The box centred over the abnormal area (position A) permitted mean and maximum temperature and peak frequency of isotherms to be measured. Moving the box medially (position B) permitted the slope of the thermal gradient with distance to be calculated (Figure 18:1). Patients were reviewed fortnightly until asymptomatic for at least 1 month, but thermographic analysis is based on the initial visit and the visit at 6 weeks (or on discharge if earlier). Ten patients had thermography performed in the morning and afternoon to assess diurnal variation and 60 age and sex-matched normal controls (120 elbows) were also studied. All 56 patients were also reviewed clinically and thermographically one year after their initial presentation.

RESULTS

A. Clinical: Tenderness was anterior to the lateral epicondyle in 39 patients (70%), posterior in 11 (19%) and variable in six (11%), but this did not correlate with pain on pronation or supination or other clinical or thermographic features and did not influence the response to local therapy.

B. Thermographic: Thermography of the affected elbow showed a discrete localized area of increased heat near the lateral epicondyle ('hot spot') in 53 of the 56 affected elbows, the centre being 1-3°C above the background isotherm (Figure 18:2). A similar abnormality was found in only three of the 120 control elbows, the lesion never being more than 1°C above the background. Contingency table analysis showed the difference to be significant (X^2 with Yates correction = 145.0, $p<0.0001$). The normal thermographic pattern showed no specific features near the lateral epicondyle, although a negative thermal gradient was often present from the elbow joint towards the olecranon (Figure 18:3).

A clear diurnal variation was detected in all 10 patients - the temperature tending to rise through the day (Table 18:1). Both mean temperature and peak frequency of isotherms showed a significant increase in the afternoon compared with the morning temperature. Maximum temperature also increased through the day, but this increase did not reach significance ($p<0.1$). Gradient slope analysis, however, showed minimal diurnal change ($p>0.5$) and also correlated well with the severity of the condition and reflected recovery (Figure 18:4, $r=0.49$, $p<0.02$). Mean and maximum temperature and

peak frequency of isotherms did not show this correlation.

Sixteen patients (39%) with unilateral tennis elbow also had a hot spot near the lateral epicondyle on the unaffected side, two later developing pain. The morphology of the hot spot was always similar to the unaffected side, but the peak temperature and gradient slopes were less.

C. Clinical/Thermographic Outcome: Mild ache and/or tenderness often persisted for weeks or months after full functional recovery (that is normal grip strength, weight test and painless resisted wrist dorsiflexion). Thermography reflected the clinical improvement with a fall in the peak temperature; the localized lesion often increasing in size before merging with muscle heat. The thermographic lesion persisted on discharge in 18 patients - nine still having clinical tennis elbow and the others having continuing ache and/or tenderness (Table 18:2). Seven contralateral elbows were also still abnormal on discharge. The three patients with consistently negative thermography failed to respond to local therapy - two later complaining of neck pain.

When the patients were reviewed 1 year after initial presentation, 9 were still symptomatic and 7 had a persistent thermographic abnormality. Two patients who never showed a local thermographic change were still symptomatic but with normal thermography of the lateral elbow. All the other patients had normal elbow thermography even when an intermittent ache in the elbow persisted following use. The 5 patients with tennis elbow in the opposite side also developed hot spots at the lateral elbow and 3 with medial epicondylitis, hot spots at the medial aspect of the elbow.

DISCUSSION

Lateral epicondylitis produces some variability in the site of local tenderness and severity of pain on resisted supination and pronation. Whilst these differences may reflect different underlying pathology, they did not seem to influence the response to therapy nor affect the prognosis. The thermographic abnormality described was present in over 98% of painful elbows (irrespective of the site of tenderness), and analysis of the gradient across the abnormal area showed good correlation with clinical severity. The gradient slope showed far less diurnal fluctuation than the temperature-based parameters.

Similar thermographic changes were found in over 30% of the asymptomatic

contralateral elbows and only 3% of the normal controls and may indicate an underlying susceptibility in these patients, or more likely reflect the stress placed on the asymptomatic elbow in order to protect the painful arm.

Cervical spine radiographs were no different from age and sex-matched controls, but a persistently normal thermograph in patients with clinical tennis elbow may reflect an undetected cervical root lesion. While the pathology of tennis elbow remains controversial, thermography mirrors clinical severity and offers definite possibilities for prospective study of this and other soft tissue lesions and their noted associations.

TABLE 18:1

DIURNAL VARIATION IN THERMOGRAPHIC PARAMETERS OF TEN TENNIS ELBOW LESIONS

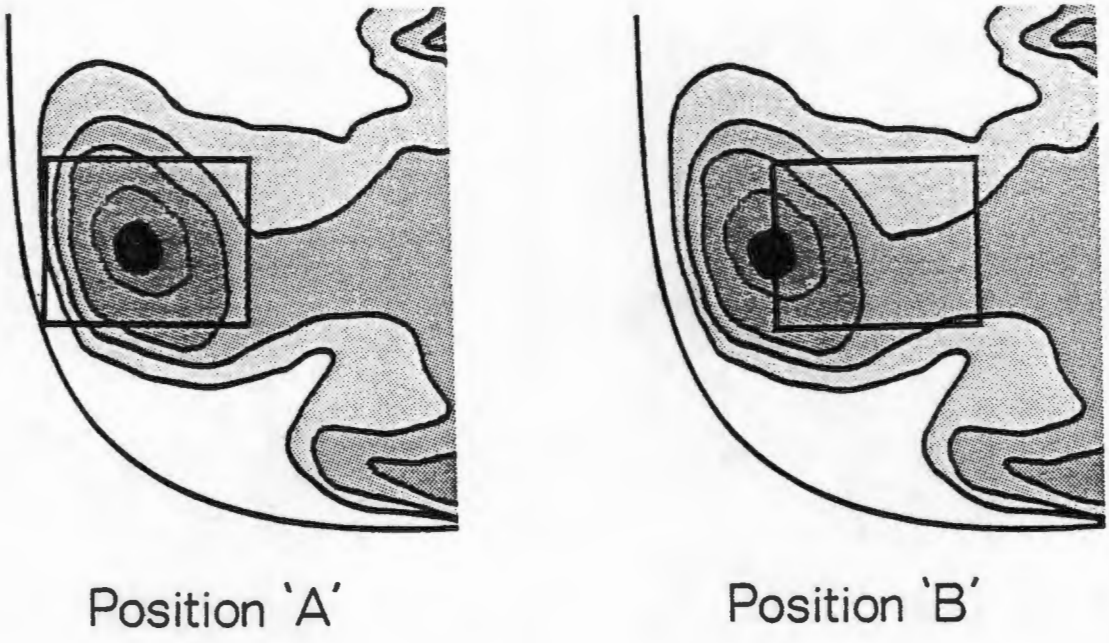
	A.M.		P.M.		Paired T-test
	Mean	SD	Mean	SD	p<
Box Position A					
Mean Temperature	30.84	0.94	31.88	0.69	0.05
Maximum Temperature	31.29	0.93	31.80	0.72	0.1
Peak Frequency	30.83	0.92	31.49	0.83	0.02
Box Position B					
Gradient Slope	0.41	0.14	0.46	0.14	>0.5

TABLE 18:2

CLINICAL AND THERMOGRAPHIC FEATURES ON DISCHARGE

		Thermography	
Clinical Features	No.	Abnormal	Normal
Persistent Tennis Elbow	12	9	3*
Ache After Use	17	2	15
Localised Tenderness	7	5	2
Asymptomatic	20	2	18

* All Three Remained Persistently Normal.



F I G U R E I8 : I. Position of the 'Regions of Interest' for analysis of the thermographic data in the lateral epicondylitis study.

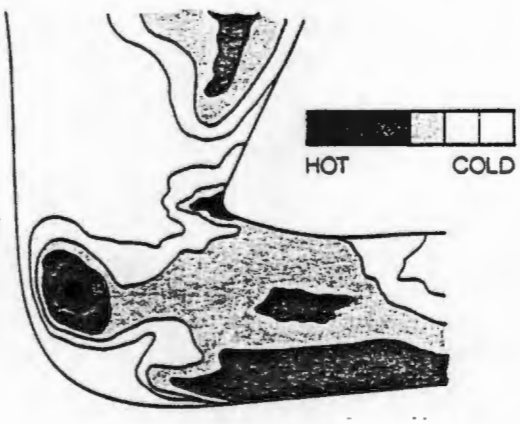


FIGURE I8 : 2. Thermographic abnormality in lateral numeral epicondylitis.

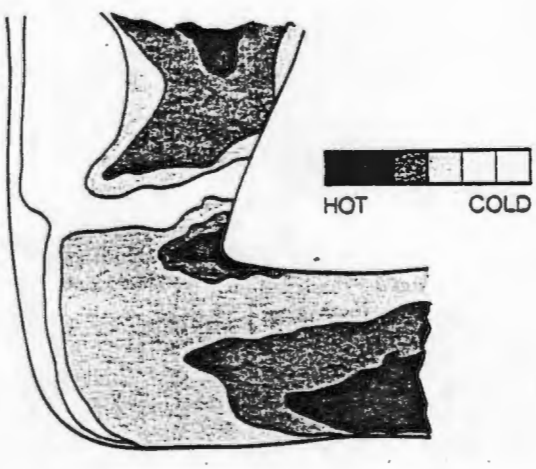
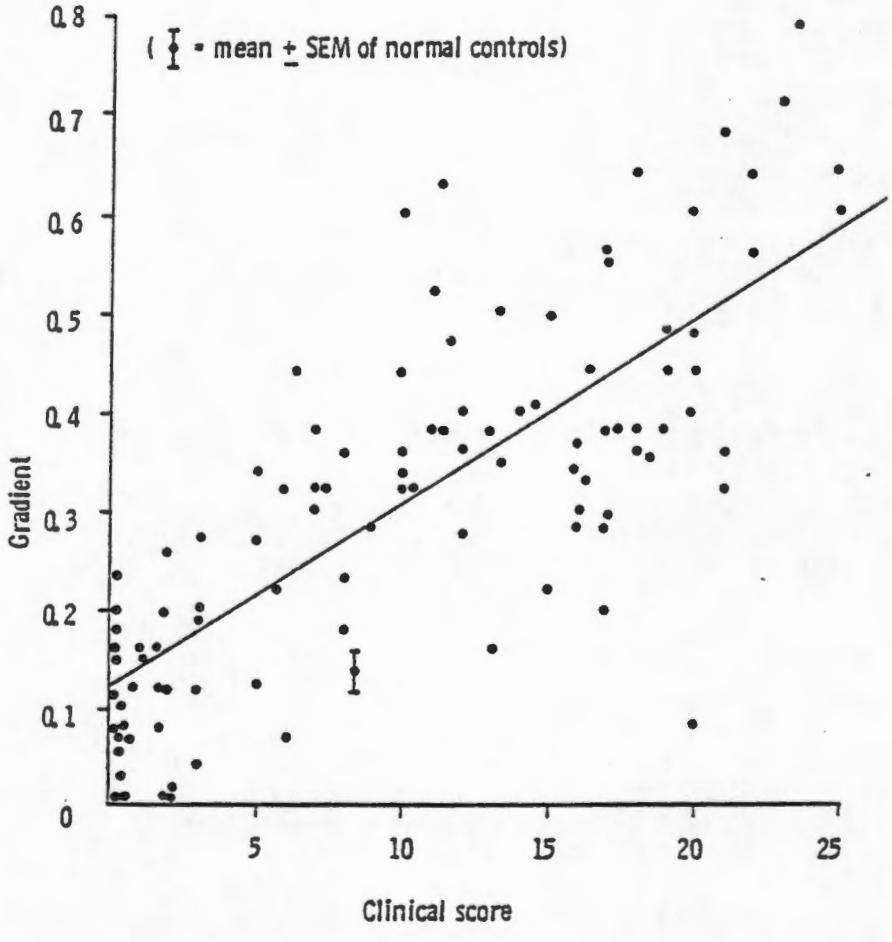


FIGURE I8 : 3. Thermography of the normal elbow.



F I G U R E I8 : 4. Correlation of the thermographic gradient slope and clinical score in lateral epicondylitis.

CHAPTER 19

LATERAL EPICONDYLITIS : AN EVALUATION OF THERAPEUTIC ULTRASOUND

Since Wood and Loomis (289) first investigated the interaction between ultrasound and living tissue, biological effects of the radiation have been recognised. These include enhanced blood flow, increased membrane permeability and altered connective tissue extensibility and nerve conduction (156). Stimulation of protein synthesis with fibroblast activation has also been reported (111). All these sequelae were initially attributed to the deep thermal effects, but introduction of pulsed ultrasound largely eliminated the rise in temperature within tissues, showing non-thermal effects also to be present (75).

The effectiveness of ultrasound remains unproven. Reports have claimed its value in treating tennis elbow (1), painful shoulder (155, 192) and other conditions (156) but the failure to randomise treatment and lack of controls has cast doubt on their conclusions. In the few studies to include a control group (83, 189), beneficial results were less pronounced. However, Dyson et al using control groups showed enhanced tissue regeneration in an animal model (75), and improved varicose ulcer healing in man (74).

Tennis elbow (lateral epicondylitis) was chosen as an ideal soft tissue lesion for study as it is common, clinical diagnosis simple, and the lesion discrete, localised and superficial. Ultrasound by its ability to cross myofascial planes and concentrate near bone (154), offers further theoretical advantages.

PATIENTS

The seventy-six patients in the Prospective Group suffering from lateral epicondylitis of at least one month's duration were included in the study (Table 19:1).

METHODS

The Electro-medical Supplies Therasonic ultrasound machine which was employed in the study continued to be used for routine physiotherapy. The

machine was standardised initially and then every month thereafter on a master balance. Output was also checked before each treatment session on a simple underwater radiation balance (Figure 19:1) constructed (169) for this purpose. An on/off key introduced into the transducer circuit permitted mock-insonation to be given to a placebo group, without affecting the normal ultrasonic output when the key was turned to on.

Therapy Regime: Therapy was 'pulsed' with an on to off ratio of 1 to 4 and a frequency of 1.0 megahertz. Treatment was given in contact, using Electro-medical Supplies ultrasonic coupling medium. The space-averaged intensity was increased from 1 to 2 watts per cm^2 and therapy time from 5 to 10 minutes during the course of therapy. Twelve treatments were given (2-3 per week) over a 4-6 week period.

A therapist not taking part in giving the therapy, randomly allocated patients to the ultrasound or placebo groups according to a prearranged schedule. She also set the transducer switch accordingly before each treatment session, so the patient, medical assessor and primary therapist were unaware of the treatment groups.

Patients were reviewed fortnightly while receiving therapy. Follow-up continued for at least one further month before patients were discharged or if symptomatic, offered alternative therapy.

At each visit, clinical assessment included the pain score, pain on resisted wrist dorsiflexion, weight test and grip strength in flexion and extension as previously described. The patient's assessment protocol is shown in Appendix II.

After completing therapy and during further follow-up, patients were asked to assess the results of treatment. The medical assessor also judged the outcome. A satisfactory outcome on examination was judged as full functional recovery with no more than a minor ache and/or slight tenderness remaining. The 'end point' of the study was discharge, withdrawal, or alternative therapy. All patients were, however, re-examined or completed a postal questionnaire at 1 year after inclusion in the study as part of a larger assessment of natural history of lateral epicondylitis (see Chapter 20).

RESULTS

Objective Outcome: Twenty-four patients treated with ultrasound (63%) and 11 who had received placebo (29%) showed a satisfactory outcome on objective testing both at the termination of therapy and during further follow-up. Contingency table analysis (Table 19:2) showed that the difference between the groups was significant (X^2 with Yates' correction = 7.63, $p<0.01$).

Subjective Outcome: Six patients (2 given ultrasound treatment and 4 given placebo) reported the outcome immediately after completing therapy, to be satisfactory despite persistent disability on objective assessment. Review one month later confirmed an unsatisfactory result in 5 of the 6 patients.

Parameter Analysis: The two treatment groups showed no significant differences in the mean severity of any of the clinical parameters at presentation. Comparisons of the rate of recovery from time 0 to each follow-up visit (Wilcoxon rank sum tests) however, confirmed a significant advantage for the ultrasound over the placebo group. The reduction in the pain score, pain on resisted wrist dorsiflexion, pain on weight test and the improvement in grip strength in flexion and extension in the 2 groups is shown (Figure 19:2).

The mean grip strength in all the patients at presentation was 58 mmHg better with the elbow flexed (GF) than with it extended (GE). By the end of the controlled study the advantage for GF was still 28 mmHg in the ultrasound and 39 mmHg in the placebo groups.

The duration of symptoms at presentation, dominance of the affected arm and therapy given before referral did not influence the outcome, but patients who responded to mock-insonation had less severe symptoms at presentation than those responding to ultrasound.

Reliability of the Ultrasound Machine: Fluctuation in the ultrasonic output was detected in the study machine when subjected to checks on the underwater radiation balance prior to treatment. Therapy had to be delayed for repairs to the ultrasound machine on 2 occasions when there was no output and on a further 4 occasions when the radiation was considerably reduced.

Further Therapy Given: Forty-one patients (14 given ultrasound and 27

given placebo) still had an unsatisfactory outcome at the end of the controlled study and were offered ultrasound (some placebo group patients) and/or steroid injections (Table 19:3). Eight patients either withdrew still complaining of severe disability or required surgery to the elbow; Of these, 4 were housewives, 2 domestic workers and 2 manual labourers.

Review at 1 Year: All the patients were re-examined or completed a postal questionnaire at 1 year. A much lower incidence of recurrence of severe pain (Table 19:4) was noted in the patients who responded to ultrasound than those who required steroid injections. Minor or intermittent pain in the elbow was still present in over 50% of patients.

DISCUSSION

This study has shown that ultrasound enhances recovery in patients with lateral epicondylitis (1), but only in 63% of cases. Serial assessment of clinical parameters has confirmed that the treated patients had a significantly better rate of recovery than the placebo group, and later review suggested a lower incidence of recurrence in the patients who responded to ultrasound. Eleven of the patients (29%) in the placebo group recovered without any other therapy. This may reflect the benefits of careful supervision, increased rest and natural remission, but may also result from the massage effect of the transducer head over the affected area during mock-insonation.

Subjective patient assessment immediately after the completion of therapy has usually been used to determine the efficacy of treatment. However, a definite discrepancy between the subjective and objective assessments was found. Review 4-6 weeks later showed better agreement. Repetitive activity especially housework often precipitated the onset of symptoms, and failure to rest from this activity resulted in a poor response. Although Dyson et al using an animal model (75) suggested that maximum benefit could be obtained from starting therapy as early as possible, no advantage was detected in patients who did present early.

Clarke and Stenner (43) and Allen and Battye (4) noted considerable alterations of ultrasonic output with time. Using a simple underwater balance this was confirmed, emphasising the need for frequent assessment. Ultrasound is time consuming to patient and therapist and further controlled studies in other conditions are necessary.

TABLE 19:1

CLINICAL DETAILS OF TENNIS ELBOW PATIENTS

		Ultrasound Group (N = 38)	Placebo Group (N = 38)
Sex -	Female : Male	25 : 13	23 : 15
Age (Years)	Mean	44.1	42.4
	Range	29 - 59	29 - 65
Arm -	Dominant : Non-Dominant	29 : 9	27 : 11
Duration of symptoms (Months)			
	Mean	4.8	4.3
	Range	1 - 12	1 - 12

TABLE 19:2

OBJECTIVE OUTCOME (END OF THERAPY AND 1 MONTH LATER)

Outcome	Ultrasound Group	Placebo Group
Satisfactory	24	11
Unsatisfactory	14	27

X^2 with Yates Correction = 7.63, $p<0.01$

TABLE 19:3

OUTCOME IN PATIENTS WITH AN UNSATISFACTORY INITIAL RESULT

Outcome	Placebo Group (N = 27)	Ultrasound Group (N = 14)
Healed with Ultrasound	8	-
Healed with Injections	7	11
Healed with U/S and Injection	7	-
Withdrew while Symptomatic	3	1
Referred for Surgery to Elbow	2	2

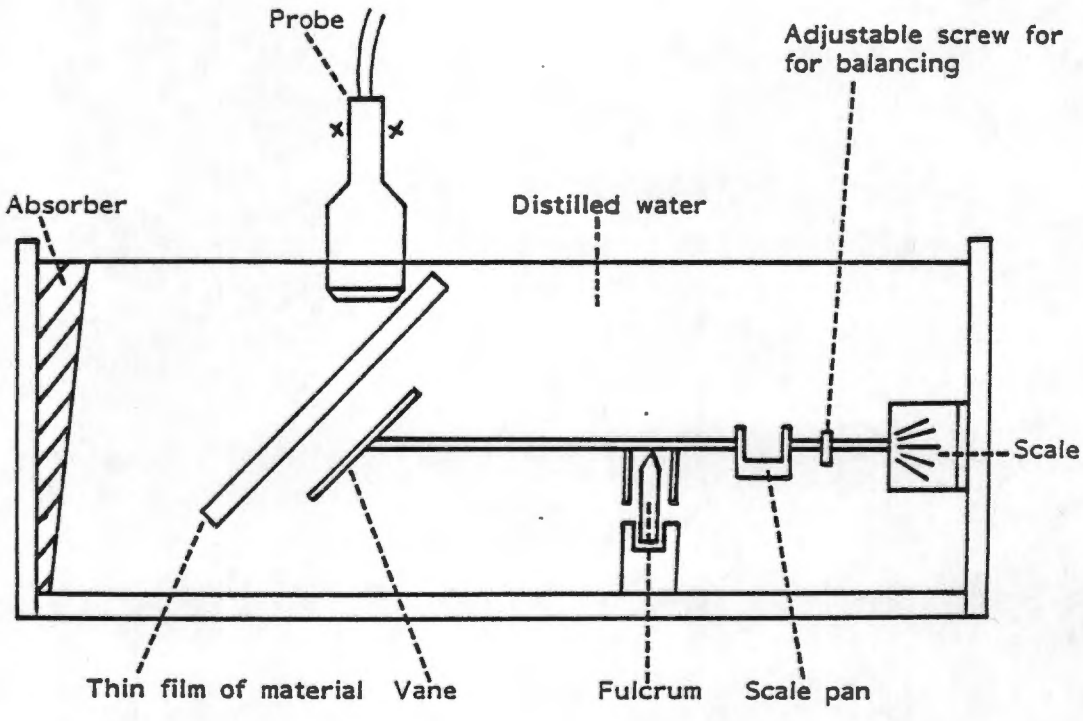
TABLE 19:4

OVERALL OUTCOME AT DISCHARGE AND AT REVIEW ONE YEAR LATER

At Discharge		Review at One Year	
		Satisfactory	Unsatisfactory
Satisfactory after:			
Mock-insonation (placebo)	11	6	5
Ultrasound (± placebo)	32	29	3
Steroid injection (± U/S ± placebo)	25	13	12
Unresolved/refused further therapy	8	2*	6+

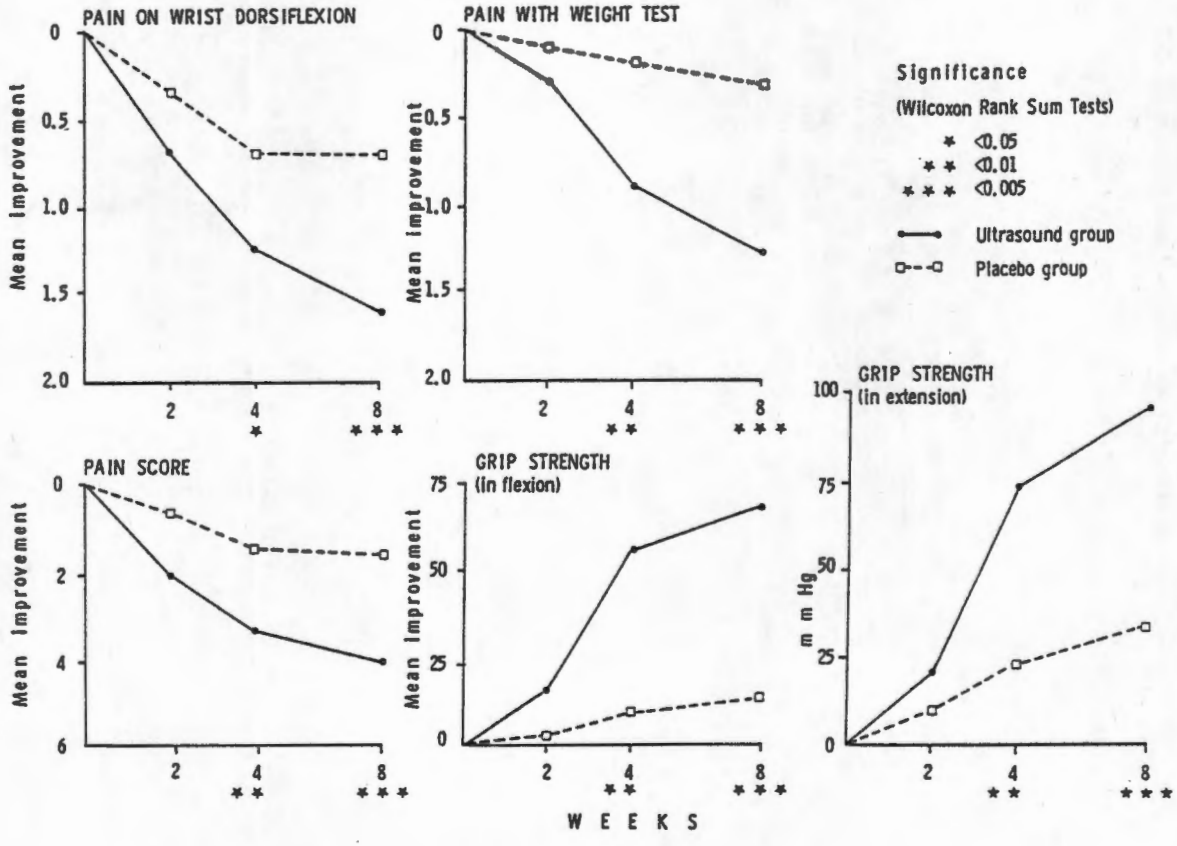
* 1 Patient after Surgery

\+ 2 Patients after Surgery



F I G U R E I9 : I. Simple underwater radiation balance.

F I G U R E I9 : 2. MEAN IMPROVEMENT IN THE CLINICAL PARAMETERS FROM TIME ZERO IN THE LATERAL EPICONDYLITIS TREATMENT GROUPS.



CHAPTER 20

LATERAL EPICONDYLITIS : AN ASSESSMENT OF NATURAL HISTORY AND THE EFFECT OF CONSERVATIVE THERAPY

Many of the conservative therapeutic measures advocated for the treatment of tennis elbow have fallen into disuse. Marlin (172) and Mills (183) considered manipulation therapy to be the treatment of choice, but this was never widely accepted. Stoddard (257) and Wadsworth (270) have more recently advocated it for persistent lesions which have not responded to repeated local steroid injections. Immobilisation (36, 50, 109, 213), shortwave diathermy (57, 109, 250), deep massage (236, 266) and deep X-ray therapy (54, 117, 178) have been used but not subjected to controlled study. Therapeutic ultrasound has been discussed in detail (see Chapter 19).

Local steroid injection therapy has been shown to enhance recovery (8, 40, 59, 61, 121, 129) and remains the treatment of first choice. However, even after repeated injections, some patients remain symptomatic or show a rapid recurrence of symptoms (193, 222, 250, 293). Cyriax (57) regarded tennis elbow as a self-limiting condition, improving with or without treatment within 8 - 12 months. He felt recurrence was unusual. However, symptoms do persist in some patients. Clarke and Woodland (40) reviewed patients after 6 months and found recurrence in 54% of cases, although few were receiving further treatment. Nevelos (198) reported recurrence in 18% of his patients after a similar period. During the therapeutic study of ultrasound (see Chapter 19), persistent or recurrent symptoms were common. As no long-term studies of conservative therapy in tennis elbow have been reported, follow-up of the prospective group was extended and the outcome in a retrospective group of patients treated in the out-patients department in the previous 5 years was assessed.

PATIENTS AND METHODS

Clinical details of the patients have been given in Chapter 17 and 19 and will only be briefly summarised.

The Prospective Group consisted of the 76 patients included in the study of therapeutic ultrasound (Chapter 19). 'Discharge' was at 3-4 months and

irrespective of the outcome all the patients were recalled for 'review' one year later.

The Retrospective Group. 77 patients treated 1-5 years previously in the out-patients department for tennis elbow were sent questionnaires with regard to therapy and outcome (see Appendix III). Where possible information was verified from the case records. 65 patients completed the questionnaires. 3 had died and 9 could not be traced. Mean follow-up was 39.5 (range 12-60) months. 12 of the 65 patients (18%), had defaulted from the clinic - 4 because acute symptoms had resolved; 6 because treatment was ineffective and 2 because the steroid injections had been unacceptably painful. 'Discharge' refers to symptoms at the time of discharge or default.

RESULTS

Clinical Data: The details relating to the 141 patients in the 2 groups is shown in Table 17:1.

Hospital Treatment: Therapy given in the two groups (Table 20:1) differed in that most prospective patients were initially treated with ultrasound and were only given steroid injections if the former was ineffective. Steroid injection was the preferred treatment in the retrospective group. 90% of patients (both groups) were improved by the injections, but some patients in the retrospective group had up to 8 injections. 70% of the injected patients reported marked exacerbation of pain lasting 24-72 hours after the injections. 8 prospective and 12 retrospective patients defaulted or refused further injections. Ultrasound produced improvement in 60% of cases without any initial exacerbation of symptoms. Only 4 of 34 patients who received both ultrasound and injections considered ultrasound more effective.

Prospective Group: The results of therapy (Table 20:2) at 3-4 months ('discharge') showed 10% of patients still to be symptomatic. On review after one year, 22% had severe pain. Steroid injection improved 90% of patients even when the symptoms persisted after ultrasound. Recurrence of severe symptoms (Table 20:3) occurred almost exclusively in the patients treated with injections. Only 3 patients who improved with ultrasound had a recurrence within 4 months. More than 50% of all patients had persisting or intermittent discomfort (Table 20:2) which was never considered severe enough to warrant further treatment, but did cause them to stop or limit

some activities. 5 patients (3 domestic workers and 2 manual labourers) had changed occupations; 10 stopped or had not fully resumed sport and 15 other hobbies, because of the elbow pain. Few patients sought further medical aid even when they had severe persisting or recurrent symptoms (Table 20:4). 4 patients attended 'fringe' medical practitioners without benefit and 4 after further medical treatment, were referred for surgery to the elbow. Most resigned themselves to prolonged disability.

Retrospective Group: Results of therapy at 'discharge' and on review (Table 20:2) and interim recurrences (Table 20:3) are shown. With the longer follow-up in this group, some increase in late recurrence (after 6 months) was noted, but fewer patients at review had severe disability. Despite the longer follow-up - 42% of patients still had intermittent symptoms which prevented full resumption of normal activities. 6 patients had changed occupations (4 manual and 2 domestic workers) and 20 permanently stopped a hobby or sport because of persisting pain or fear of further recurrence. 10 of the patients had been symptomatic for over 2 years - 5 for longer than 5 years. Only 7 patients (Table 20:4) had sought further medical care, 6 turning unsuccessfully to 'fringe' medicine to obtain relief. 2 patients were awaiting surgery to the elbow.

Other Soft Tissue Lesions: Two patients in the prospective group had bilateral tennis elbow lesions at presentation and 10 others developed bilateral symptoms during the follow-up period. 5 patients also developed medial epicondylitis (2 bilateral) and 6 tendinitis of the shoulder. In addition to the 3 retrospective patients with bilateral tennis elbow at presentation - 12 others developed bilateral elbow pain and 15 shoulder pain during the follow-up period.

DISCUSSION

Tennis elbow is of common occurrence and is generally regarded as a trivial condition which is self-limiting or responsive to steroid injection. Most studies have evaluated the efficacy of different surgical procedures in the small proportion of chronic lesions coming to surgery (22, 50, 88, 94, 204). Studies of conservative therapy especially steroid injection have concentrated on the immediate results. Clarke and Woodland (40) and Nevelos (198) extended follow-up to 6 months and noted a high recurrence of symptoms, but did not define the severity of these symptoms. This study has confirmed the large number of patients who do not respond to treatment or who show recurrence of severe symptoms sometimes associated with

resumption of the activity which induced the initial pain. Manual workers, especially mechanics and builders and domestic workers were most susceptible to recurrence and tended to seek medical or fringe medical care to permit continuation of their occupations. More surprising was the large number of patients who had persisting or recurrent discomfort following use which induced some modification in their normal activities. This minor disability was not considered severe enough to require therapy, but lasted for far longer than is generally recognised. The absence of suitable controls and the use of retrospective patients makes comparison of steroid injection and ultrasound therapy difficult. The lower recurrence rate noted in ultrasound-responsive patients may reflect the milder condition in these patients or a true enhancement of healing, but as only 60% the patients responded, this therapy cannot be regarded as the treatment of choice. Steroid injections though more effective (90% improved) were often followed by recurrence. The pain induced by the injections can also be considerable and was the reason a number of patients did not seek further treatment.

The development of bilateral tennis elbow may be due to increased stress placed on the unaffected arm but the high incidence of medial epicondylitis, shoulder tendinitis and other soft tissue lesions observed in this and other studies (22, 61, 204) remains as yet unexplained.

Tennis elbow has been shown to be a common cause of prolonged disability and even when responsive to conservative therapy, mild or even severe disability may persist.

TABLE 20:1

HOSPITAL THERAPY

Therapy	Prospective Group (N = 76)	Retrospective Group (N = 65)
Steroid Injection	12	40
Ultrasound	32	8
Ultrasound and Injection	21	13
Other	11*	4+

* = Mock-insonation (Placebo)

+ = Shortwave Diathermy, Ice, Sling

TABLE 20:2

CLINICAL STATUS AT DISCHARGE AND ON REVIEW

Clinical Status	Prospective Group				Retrospective Group			
	At Discharge		On Review		At Discharge		On Review	
	N	%	N	%	N	%	N	%
No Symptoms	25	(33%)	21	(28%)	19	(29%)	30	(46%)
Intermittent Discomfort	43	(57%)	38	(50%)	39	(60%)	27	(42%)
Severe Pain	8	(10%)	17	(22%)	7	(11%)	8	(12%)

TABLE 20:3

RECURRENCE OF SEVERE PAIN AND DISABILITY

Recurrence	Prospective Group		Retrospective Group	
	N	%	N	%
Within 3 Months	9	(12%)	7	(11%)
3 - 6 Months	6	(8%)	4	(6%)
After 6 months	2	(3%)	8	(12%)

TABLE 20:4

TREATMENT AFTER DISCHARGE

Treatment	Prospective Group	Retrospective Group
General Practitioner	2	5
Hospital	5	2
Acupuncture/Osteopath	4	6

PART VI

DISCUSSION OF SOFT TISSUE RHEUMATISM IN THE CONTEXT OF THE PRESENT STUDIES

The lack of definition, understanding and research into soft tissue rheumatism is widely recognised (68, 151). The investigation into these conditions has been hampered by a paucity of clinical features or abnormalities in blood tests or other special investigations which permit classification of these lesions. This lack of an accepted classification has also resulted in differences in terminology and interpretation of data from existing studies. Soft tissue lesions never cause death and rarely come to surgery, so there is little material for study at the tissue level. Even those patients who come to surgery have usually received repeated steroid injections and other conservative measures over a prolonged period, making it difficult to determine if the histological abnormalities reflect the primary pathology or are secondary to healing and the therapeutic endeavours.

The special "shoulder clinic" set up in Cambridge fulfilled an important role in the study of conditions causing pain in the shoulder region. The initial response by local general practitioners to a request for patients with shoulder symptoms suggested that many lesions are inadequately diagnosed and treated. The frequency and severity of the different shoulder syndromes was assessed and the lesions causing the greatest difficulty in clinical practice were chosen for further study.

The complexity of normal shoulder function has presented particular problems in the study of conditions in this region and most methods advocated for the measurement of the range of movement have been cumbersome and unreliable (2,71,87,152). Information relating to the range of movement in health and disease and the factors which influence this have therefore been lacking. Clarke et al (41) used a hydro-goniometer in

healthy controls and patients with painful stiff shoulders and found the instrument simple to use with satisfactory reproducibility of results. However, they only measured a few variables and published limited data. A modification of this goniometer was therefore used to define the range of movement in the normal population and the factors which might influence this range. In the Painful Stiff Shoulder (PSS), the passive range was reproducible and was used for serial study. Like Lee et al (153), a consistent relationship was found between individual movement variables in the PSS, permitting simplification of the analysis of the recovery in range by the use of multivariate or principal component analysis. As a consistent reduction in the passive range was not found in the Rotator Cuff Tendinitis (RCT) syndrome, this was not useful for serial study. The active range of movement was usually reduced due to pain and was used instead. Pain on resisted movement (58) and the severity of the painful arc on abduction could also be quantified for serial study of the RCT lesions. A reduction in the range of elbow extension was not a consistent finding in the Lateral Epicondylitis (L/E) lesions and could not be used to follow the progress of these lesions. Pain on resisted wrist dorsiflexion, grip strength and the weight test reflected the clinical severity of these lesions, were reproducible and were used for serial study. The importance of careful standardisation of the position of the elbow when performing the clinical assessment of L/E was indicated by the significant difference between the grip strength when performed with the elbow held in flexion and extension.

Many patients reported difficulty in transcribing the severity of pain to visual analogue scales (VAS), but no more satisfactory method of quantifying pain was found. The ability to sleep on the affected shoulder and the duration before pain was much improved provided an end-point for determination of recovery in the shoulder conditions studied, but could not be used to assess serial change in the severity of the pain. The use of simple analgesic agents was also thought to be dependent on the personality of the patient rather than the severity of the pain.

Thermography produced a consistent abnormality in L/E lesions which correlated with clinical severity and altered with clinical improvement. Similar changes were not evident in the shoulder lesions as the rich vasculature in the region of the shoulder distorted the thermographic image. Nevertheless, thermography is an objective and non-invasive technique which may be of value in the study of other soft tissue lesions.

Arthroscopy of the shoulder (103, 279) has been reported in the PSS and other shoulder syndromes and may be a useful method of obtaining tissue in these and other lesions close to large joints at a much earlier stage in their natural history and before local steroid injections and other therapy has distorted the histological picture.

As the pathogenesis and pathology of the soft tissue lesions remains poorly understood, treatment is largely empirical. Local steroid injections are the treatment of choice in many of these conditions, but some patients fail to respond or show no more than a transient response. Most other therapies advocated to treat these lesions have not been subjected to controlled study. To assess efficacy, the condition chosen for study needs to be clearly defined with suitable parameters for serial analysis. As many of the lesions are self-limiting and the natural history uncertain, large groups of patients, a suitable control group and randomisation procedures are essential. The study of therapeutic ultrasound in L/E was carried out along these lines; 63% of L/E patients were improved by ultrasound and 29% by placebo. The subjective outcome at the end of treatment was an unreliable guide to the eventual outcome and should not be used in isolation to determine efficacy. The high recurrence rate of tennis elbow lesions following steroid injection therapy also suggests that the follow-up period needs to be prolonged to determine the true value of treatment. Many patients also fail to return for further treatment if symptoms recur. The ultrasound study also indicated that the ultrasound machines are often unreliable and the reduced radiation output can be missed by the physiotherapist in her routine pre-treatment checks. This has important implications both for the routine use and study of this and other therapeutic modalities. An improvement in the quality of pre-treatment checks and increase in the frequency of professional equipment maintenance may reduce problems from this source with a more consistent therapeutic response.

Pulsed electro-magnetic field (PEMF) therapy has not previously been used to treat soft tissue lesions. The study was based on the clinical and experimental evidence that it stimulates growth and regeneration of connective tissue structures and bone. This therapy was safe and useful in patients with severe persistent RCT lesions and warrants study in other chronic tendon and soft tissue lesions.

Treatment of the PSS remains unsatisfactory. These studies have suggested that steroids given by mouth or by local injection do decrease pain, but

have little effect on the recovery in the range of movement. The maximum improvement in pain occurred within 4 - 6 weeks of presentation and active treatment is most effective during this period. Ice packs were of no value and mobilisation physiotherapy possibly harmful if given in the acute stage. Little justification for the referral of PSS patients to the physiotherapist was found. The role of home pendular exercise in the prevention and treatment of this condition is still unclear, as all the patients were encouraged to perform this activity. The recovery in the patients who only performed home pendular exercise was similar to those who also recevied active treatment, suggesting either that therapy does not influence the rate of recovery or that the pendular exercise was the important factor in their recovery. A comparison of the progress before and after the inclusion in the studies suggests that the latter explanation may be correct.

Non-steroidal anti-inflammatory agents are the mainstay of general practitioner treatment of most soft tissue lesions, but was reported to be of little value by a majority of the patients who received this therapy. Simple analgesics were of some value in the shoulder patients as they facilitated sleep. Few patients received advice from their general practitioners on the need to rest or exercise the affected limb. The retrospective L/E study suggested that patients do not regard the therapy given by their general practitioner or rheumatologist as being of prolonged benefit, so many defaulted or failed to return when symptoms recurred.

The natural history of these conditions has not been documented previously; 50% of the L/E patients had a persistent or recurrent ache in the elbow which limited some activities. This lasted for 6 months or longer usually without severe relapse. 10% of patients had severe and prolonged disability which markedly affected work and other activities. The PSS also produced prolonged reduction in range, but as this was usually without pain, most patients adapted to this minor disability and were unaware that it was present.

The patients included in all the groups studied had an increased incidence of other soft tissue lesions and the presenting complaint was often bilateral. While this supports the hypothesis of individual susceptibility to these lesions, no explanation for this was found. HLA-B27 was no more common in the patients than the controls and no other antigen associations were evident. However, only the A and B loci antigens were analysed. Whether the lesions arise from pathology at the enthesis (enthesopathies)

was not assessed, but thermography of the elbow did suggest a localised lesion at this site in most L/E patients. Why the enthesis should be unusually susceptible to injury is unclear. Clinical cervical spondylosis was not common, but 3 of the patients with symptoms of L/E but with persistently normal thermography developed neck pain. It was not possible to ascertain if these factors were causally related (195). Painful 'trigger' points in the classical areas described by Smythe et al (252) were not demonstrated and there was no evidence of the 'fibrositis' syndrome in these patients.

Whether the RCT lesions precede the PSS remains uncertain. RCT which persisted for at least 3 months was studied, but only 2 patients developed a loss in the passive range during the follow-up period. Neither was severe enough to fulfill the criteria of the PSS. This lack of progression may have been due to therapy, pendular exercise or a lack of relationship between the 2 conditions. Few of the PSS patients could recall symptoms of a RCT lesion in the months before the onset of the PSS. The importance of immobilisation in the prevention of the PSS needs further study. The PSS was more common in females aged from 40 - 75 years (mean 54.4), and involved the non-dominant arm. RCT was slightly more common in males with a similar mean age but a much wider age range (20 - 80 years). The dominant arm was also more often involved. This does not support a causal relationship, but as tendon degeneration is usually bilateral the differences may be due to chance. There was also no strong association between manual labour and any of the lesions studied. The immunological studies showed some abnormalities in the PSS and with the increased uptake of technetium would support an inflammatory reaction, but were not specific or consistent enough to prove Macnab's hypothesis of a type IV immunological reaction to damaged collagen (170). Further more specific immunological studied are needed. The concept of connective tissue failure also needs to be explored.

In summary 3 common soft tissue lesions in the upper limb have been studied in a prospective manner evaluating clinical, laboratory and other factors of possible relevance. The natural history of these lesions and the possible influence of therapy has been considered. An attempt has been made to explore the previously documented association between a variety of soft tissue lesions which may shart a common pathology and pathogenesis. Useful principles for the further study of Soft Tissue (Non-Articular) Rheumatism has also been presented.

PART VII

CONCLUSIONS

A. THE SHOULDER STUDIES

1. Many patients with shoulder pain do not receive any specific therapy. The shoulder clinic was useful for studying these patients and confirmed that most lesions can be diagnosed by careful clinical examination.

2. Non-steroidal anti-inflammatory agents are the mainstay of treatment of all shoulder conditions in the community, but were considered ineffective by most patients studied. Few patients received any advice from their general practitioners with regard to use of the shoulder after symptoms developed.

3. Detailed analysis of shoulder movement in the normal population showed that females of all ages have a better range than males and that the decrease in range with age was more marked in the males. Arm dominance did not influence the range.

4. Frozen shoulder using the strict criteria in these studies is not common, recruitment of patients took 18 months in the Major Study and 12 months in the Supplementary study.

5. A modified spirit goniometer was easy to use and gave reproducible results for the serial measurement of shoulder range. The passive range of movement was more useful than the active range in the assessment of the painful stiff shoulder (PSS) and component analysis was a convenient method of simplifying analysis of the recovery in range, accurately reflecting the change in individual movement parameters. The active range was useful in the assessment of rotator cuff tendinitis (RCT) lesions.

6. Patients experienced difficulty in transcribing the severity of pain to visual analogue scales, but no better method of serial assessment of this parameter was found.

7. The frozen shoulder patients were generally in good health and few of the associated illnesses suggested in the literature were found. This possibly reflected the strict entrance criteria used in the studies.

8. The rate at which pain and range of movement improved was similar in the two Frozen Shoulder Studies, suggesting that therapy did not markedly influence the natural history of this condition. A slight advantage in the rate of recovery was found in older patient (>56 years), early presentation

(<3 months), and dominant arm involvement, but these benefits never reached significance and were only short-lived.

9. Eight months after presentation, 40% of the PSS patients still had some pain, although it was severe in only a quarter of the cases. Whilst 89% of patients had a range less than the minimum for age and sex-matched controls, only 55% regarded their range restricted at this time. The PSS patients (both studies) had a highly significant range deficit at 8 months for all movements, glenohumeral and total, when compared to the matched controls. Only the resumption of manual labour in the convalescent phase was associated with a significantly worse outcome at this time.

10. Review of the Major Frozen Shoulder Study patients after 40-48 months also showed a marked discrepancy between the patients subjective opinion of a full recovery and the finding of objective restriction in many cases. While glenohumeral movement was not significantly different from the control group, all the other movement parameters still showed significant restriction. One patient had severe pain and restriction and 4 others severe restriction at this time. The discrepancy between the patient and observer assessement of recovery indicates either the excellent adaptation many patients make to the minor disability which often follows the frozen shoulder or the low expectations and ignorance of many of the patients.

11. Patients with PSS who resumed manual labour in the convalescent phase continued to show a worse range at review (40-48 months follow-up). Patients with dominant arm involvement also had a worse range at this time, but this was a later development.

12. Immunological abnormalities at presentation were not sufficiently consistent features to support an immunological hypothesis for the PSS.

13. The contention that all patients with painful stiff shoulders have evidence of capsulitis at arthrography was disputed. Only half the patients showed these features; the others showed rotator cuff rupture or no abnormality. The capsulitis group had the least restriction at presentation but the other groups soon caught up and there was no further difference in outcome between the groups.

14. The increase in technetium diphosphonate uptake by the PSS was confirmed, but this did not influence the outcome.

15. Paired steroid injection therapy was more effective than the other treatment regimes in terms of reduction in pain and improvement in range of the PSS, but the difference from the other groups was not significant and the benefits were only short-lived. Oral steroids were also effective at reducing pain, but did not influence the recovery in range. Steroid therapy by either route is justified only in those patients in whom pain is particularly severe. As the pain usually improved within 4 - 6 weeks irrespective of therapy, active therapy should possibly be confined to this time. Ice therapy was of no value, and mobilisation physiotherapy possibly harmful. Like dominant arm involvement and a resumption of manual labour in the convalescent phase, active physiotherapy in the acute stage was associated with a worse range at late follow-up of the PSS.

16. The progression from persistent rotator cuff tendinitis to the frozen shoulder was not shown, but this course may have been prevented by pendular exercise performed by all the patients. The assocation between RCT and frozen shoulder, the importance of immobility in triggering the frozen shoulder and the role of pendular exercise in the prevention and treatment of the latter condition requires further study.

17. Pulsed electro-magnetic field therapy, enhanced the recovery of persistent RCT lesions and may have a value in the treatment of other chronic tendon lesions.

B. THE LATERAL EPICONDYLITIS STUDIES

1. The clinical diagnosis of lateral epicondylitis is easy, but the site of local tenderness can vary. The condition usually affects the dominant arm. The lesion was more common in females and non-manual workers which is at variance with some studies in the literature based on occupation, sport or a need for surgery.

2. Non-steroidal anti-inflammatory agents are the mainstay of general practitioner treatment of tennis elbow, but few patients found them beneficial.

3. Clinical parameters which are reliable and reproducible and reflected severity have been described. The importance of standardising the position of the arm when performing these assessments has been noted.

4. Thermography demonstrates a consistent abnormality in this condition which reflects severity. This is one of the first examples of the use of an objective test in the assessment of severity of a soft tissue lesion.

5. A double-blind controlled study of therapeutic ultrasound has shown the use of lateral epicondylitis as a model for the assessment of physiotherapeutic measures. The ultrasound machine was unreliable and as this was not detected by the therapist, it may contribute to the poor results of this therapy in some patients.

6. Therapeutic ultrasound has been shown to promote recovery of tennis elbow lesions. No untoward reactions occured and the recurrence rate was low. On the other hand steroid injection therapy was more effective - 90% of lesions were improved - but recurrence of symptoms was frequent and unacceptable side-effects occurred in some patients.

7. There was a discrepancy between the subjective and objective outcome at the end of the treatment period and the subjective outcome at this time cannot be used in isolation to determine the efficacy of therapy. Studies of conservative treatment need to include more objective measurements in the assessments and also need to prolong the period of follow-up.

8. Few patients who have a recurrence of symptoms return for further treatment.

9. The natural history of tennis elbow was found to be longer than is generally recognised. 50% of the patients had minor disability which limited some activities. Over 10% of patients had severe and prolonged pain.

10. This study had confirmed the high incidence of soft tissue lesions in patients presenting with lateral epicondylitis. A few patients with persistently normal elbow thermography and a poor response to therapy to the elbow developed neck pain and this may suggest that lesions of the cervical spine can manifest themselves as soft tissue lesions of the upper limb.

ACKNOWLEDGEMENTS

I am deeply endebted to Dr Brian Hazleman, Consultant Rheumatologist and Head of the Department of Rheumatology at Addenbrookes Hospital, Cambridge for his support and enthusiasm in all aspects of the thesis. I am also grateful to Dr Dianne Bulgen, Consultant Rheumatologist, who initiated both the interest and study of the frozen shoulder in the department. Particular thanks go to Mr Graham Parr, the Clinical Metrologist, who helped with the serial clinical and thermographic assessments and provided willing assistance in numerous other ways. Other members in the Department of Rheumatology who aided in the studies were Dr Peter Page-Thomas who provided many useful ideas and much of the thermographic expertise; Ms Joy Dutton, Research Physiotherapist and Ms Jennifer Park, Research Assistant who performed some of the immunological tests in the Major Frozen Shoulder Study.

I would also like to express my gratitude to the following consultants and departments at Addenbrookes Hospital for their assistance:

Ms S. Roberts and the Department of Community Medicine for help in planning the studies and statistical analysis of the data.

Dr J. Tudor and the staff of the Radiology Department who assisted with the arthrography and its interpretion.

Dr P. Wraight and the staff of the Nuclear Medicine Department who performed the technetium scans.

Ms D. Voak and the Department of Haematology who performed the tissue typing.

The Immunology Department for carrying out some of the immunological investigations and assisting with those done in the Rheumatology Department.

Other Rheumatologists and Orthopaedic Surgeons who contributed patients for the studies.

I would also like to thank Ms G. Hodge, Superintendant Physiotherapist of Newmarket General Hospital, Ms A Greenwood, Senior Physiotherapist

at Addenbrookes Hospital and their staff for their invaluable help in the ultrasound study. Dr P Page-Thomas built the underwater radiation balance used in this study.

Further thanks are due to Dr S. Fitton-Jackson and her staff - Dr C. Murray, Mr T. Marsland and Mr A. Boutle for their technical help in the pulsed electro-magnetic field studies. The equipment used was kindly provided by Electro-biology International.

I would also like to express thanks to my sister Merle and Ms Smita Patel for typing the thesis and Dr's B. Ansell, B. Hazleman, E. Paice and A. White for their comments and suggestions in the preparation of the text.

Finally, I would like to thank the Arthritis and Rheumatism Council (ARC) for the provision of a research fellowship together with additional funds without which this work would not have been possible.

REFERENCES

1. Aldes J.H. Ultrasonic radiation in the treatment of epicondylitis. G.P. 1956; 13: 89 - 96.

2. Allander E., Bjornsson O.J., Olafsson O., Sigfusson N., Thorsteinsson J. Normal range of joint movements in shoulder,hip, wrist and thumb with special reference to side : a comparison between two populations. Internat. J. Epidemiol. 1974; 3: 253 - 61.

3. Allen J.C.B., Shearman C.H. Traumatic radio-humeral synovitis. Med. J. Australia. 1947; i: 48 - 51.

4. Allen K.G.R., Battye C.K. Performance of ultrasonic therapy instruments. Physiotherapy. 1978; 64: 174 - 9.

5. Allison A.C., Denman A.M., Barnes R.D. Co-operating and controlling functions of thymus-derived lymphocytes in relation to autoimmunity. Lancet. 1971; ii: 135 - 40.

6. American Academy of Orthopaedic Surgeons. Joint motion, method of measuring and recording. (Sixth reprint). Churchill Livingstone, Edinburgh and London, 1966.

7. Andren L, Lundberg B.J. Treatment of rigid shoulders by joint distension during arthrography. Acta Orthop. Scandinav. 1965; 36: 45 - 53.

8. Baily R.A., Brock B.H. Hydrocortisone in tennis elbow. A controlled series. Proc. Roy. Soc. Med. 1957; 50: 389 - 90.

9. Ball J. Enthesopathy of rheumatoid and ankylosing spondylitis. Ann. Rheum. Dis. 1971; 30: 213 - 23.

10. Bassett C.A.L., Mitchell S.N., Gaston S.R. Pulsing electro-magnetic field treatment in un-united fractures and failed arthrodeses. J.A.M.A. 1982; 247: 623 - 8.

11. Bassett C.A.L., Pilla A.A., Pawluk R.J. A non-operative salvage of surgically resistant pseudarthrosis and non-unions by pulsing electro-magnetic fields. Clin. Orthop. 1977; 124: 128 - 43.

12. Bassett C.A.L. Schink M.M., Mitchell S.N. Treatment of osteonecrosis of the hip with specific pulsed electro-magnetic fields (PEMF's): a preliminary report. In: Hungerford D.S. (ed). Proceedings of the 3rd International Symposium on Circulation in Bone. Williams and Wilkins, Baltimore, 1983.

13. Bateman J.E. The shoulder and neck, 2nd edition. W.A. Saunders Co., Philadelphia, 1978.

14. Beighton P., Solomon L., Soskoline C.L. Articular mobility in an African population. Ann. Rheum. Dis. 1973; 32: 413 - 18.

15. Berry H., Fernandes L., Bloom B., Clark R.J. Hamilton E.B.D. Clinical study comparing acupuncture, physiotherapy, injection and oral anti-inflammatory therapy in shoulder-cuff lesions. Curr. Med. Res. Opin. 1980; 7: 121 - 6.

16. Bjelle A., Hagberg M., Michaelsson G. Clinical and ergonomic factors in prolonged shoulder pain among industrial workers. Scand. J. Work. Environ. Health. 1979; 5: 205 - 10.

17. Bland J.H., Merritt J.A., Boushey D.R. The painful shoulder. Semin. Arth. Rheum. 1977; 7: 21 - 47.

18. Bloch J., Fisher F.K. Frozen shoulder. Acta Rheumatol. No. 15. Geigy, Basle, 1961.

19. Blockey N.J., Wright J.K., Kellgren J.H. Oral cortisone therapy in periarthritis of the shoulder. Br. Med. J. 1954; i: 1455 - 7.

20. Bosworth D.M. The role of the orbicular ligament in tennis elbow. J. Bone Jt. Surg. 1955; 37A: 527 - 33.

21. Bosworth D.M. Surgical treatment of tennis elbow : a follow-up study. J. Bone Jt. Surg. 1965; 47A: 1533 - 6.

22. Boyd H.B., McLeod A.C. Tennis elbow. J. Bone Jt. Surg. 1973; 55A: 1183 - 7.

23. Braun K.A., Lemons J.E. Effects of electro-magnetic fields on the recovery of circulation in mature rabbit femoral heads. Trans. Orthop. Res. Soc. 1982; 7: 313.

24. Bridgman J.F. Periarthritis of the shoulder and diabetes mellitus. Ann. Rheum. Dis. 1972; 31: 69 - 71.

25. Bruckner F.E. Frozen shoulder (Adhesive capsulitis). J. Roy. Soc. Med. 1982; 75: 688 - 9.

26. Bruckner F.E., Nye C.J.S. A prospective study of adhesive capsulitis of the shoulder (frozen shoulder) in a high risk population. Quart. J. Med. 1981; 198: 191 - 204.

27. Bryan F.S., Miller L.S., Panijayanond P. Spontaneous paralysis of posterior interosseous nerve. A case report and review of the literature. Clin. Orthop. 1971; 80: 9 - 12.

28. Buchan J.F. The use of ultrasonics in physical medicine. Practitioner. 1970; 205: 319 - 26.

29. Buck R.C. Regeneration of tendon. J. Path. Bact. 1953; 116: 1 - 18.

30. Bulgen D.Y., Hazleman B.L., Voak D. HLA-B27 and frozen shoulder. Lancet. 1976; i: 1042 - 4.

31. Bulgen D., Hazleman B., Ward M., McCullum M. Immunological studies in frozen shoulder. Ann. Rheum. Dis. 1978; 37: 135 - 8.

32. Caldwell G.A. Ukauf B.M. Results of treatment of subacromial bursitis in 340 cases.
Ann. Surg. 1950; 132: 432 - 42.

33. Capener N. Bio-mechanical studies of sport.
Br. Med. J. 1960; ii: 130.

34. Capener N. The vulnerability of the posterior interosseous nerve of the forearm.
J. Bone Jt. Surg. 1966; 48B: 770 - 3.

35. Carp L. Tennis elbow (epicondylitis) caused by radio-humeral bursitis. Anatomical, clinical, roentgenologic and pathological aspects with a suggestion as to treatment.
Arch. Surg. 1932; 24: 905 - 22.

36. Carroll R.E., Jorgensen E.C. Evaluation of the Garden proceedure for lateral epicondylitis.
Clin. Orthop. 1968; 60: 201 - 4.

37. Carroll R. Tennis elbow : incidence in local league players.
Brit. J. Sports Med. 1981; 15: 250 - 6.

38. Carter R.M. Epicondylitis.
J. Bone Jt. Surg. 1925; 7: 553 - 62.

39. Charnley J. Periarthritis of the shoulder.
Postgrad. Med. J. 1959; 35: 384 - 8.

40. Clarke A.K., Woodland J. Comparison of two steroid preparations used to treat tennis elbow, using the hypospray.
Rheumatol. Rehabil. 1975; 14: 47 - 9.

41. Clarke G.R., Willis L.A., Fish W.W., Nichols P.J.R. Assessment of movement at the glenohumeral joint.
Orthopaedics, Oxford. 1974; 7: 55 - 71.

42. Clarke G.R., Willis L.A., Fish W.W., Nichols P.J.R. Preliminary studies in measuring range of motion in normal and painful stiff shoulders.
Rheumatol. Rehabil. 1975; 14: 39 - 46.

43. Clarke G.R., Stenner L. Use of therapeutic ultrasound.
Physiotherapy. 1976; 62: 185 - 90.

44. Codman E.A., Akerson I.B. The pathology associated with rupture of the supraspinatus tendon.
Ann. Surg. 1931; 93: 348 - 59.

45. Codman E.A. The shoulder.
Todd Co., Boston, 1934.

46. Connolly J., Regen E., Evans O.A. The management of the painful stiff shoulder.
Clin. Orthop. 1972; 84: 97 - 103.

47. Cooke T.D.V., Bennett E.L., Ohno O. Identification of immunoglobulins and compliement components in articular collagenous tissues of patients with idiopathic osteoarthrosis.
In : Nuki G. (ed). The aetiopathogenesis of osteoarthrosis. Pitman medical, London, 1980: 144 - 54.

48. Cooke T.D.V., Richer S., Hurd E., Jasin H.E. Localisation of antigen antibody complexes in intra-articular collagenous tissues.
Annals New York Acad. Sci. 1975; 256: 10 - 24.

49. Coombes W.N. Frozen shoulder.
J. Roy. Soc. Med. 1983; 76: 711.

50. Coonrad R.W., Hooper W.R. Tennis elbow : it's course, natural history, conservative and surgical management.
J. Bone J. Surg. 1973; 55A: 1177 - 82.

51. Cooper R.R., Misol S. Tendon and ligament insertion : A light and electron microscopic study.
J. Bone Jt. Surg. 1970; 52A: 1 - 20.

52. Cotton R.E., Rideout D.F. Tears of the humeral rotator cuff. A radiological and pathological necropsy survey.
J. Bone Jt. Surg. 1964; 46B: 314 - 28.

53. Coventry M.B. Problem of painful shoulder.
J.A.M.A. 1953; 151: 177 - 85.

54. Cowan I.I., Stone J.R. Painful periarticular calcification at the wrist and elbow : diagnosis and treatment.
J.A.M.A. 1952; 149: 530 - 3.

55. Crisp E.J., Kendall P.H. Treatment of periarthritis of the shoulder with hydrocortisone.
Br. Med. J. 1955; i: 1500 - 1.

56. Cronkite A.E. The tensile strength of human tendons.
Anat. Rec. 1936; 64: 173 - 86.

57. Cyriax J.H. The pathology and treatment of tennis elbow.
J. Bone Jt. Surg. 1936; 18: 921 - 40.

58. Cyriax J. Diagnosis of soft tissue lesions.
In : Textbook of orthopaedic medicine. Bailliere Tindall, London, 1971.

59. Cyriax J., Trosier O. Hydrocortisone and soft tissue lesions.
Br. Med. J. 1953; ii: 966 - 8.

60. Davies D.V., Young L. The distribution of radio-active sulphur (^{35}S) in the fibrous tissues, cartilage and bones of the rat following it's administration in the form of inorganic sulphate.
J. Anat. (London). 1954; 88: 174 - 83.

61. Day B.H., Govindasamy N., Patnaik R. Corticosteroid injections in the treatment of tennis elbow.
Practitioner. 1978; 220: 459 - 62.

62. De Palma A.F. Loss of scapulohumeral motion (frozen shoulder).
Ann. Surg. 1952; 135: 193 - 204.

63. De Palma A.F. Surgery of the shoulder.
J.B. Lippincott, Philadelphia, 1973.

64. De Palma A.F. Surgical anatomy of acromioclavicular and sternoclavicular joints.
Surg. Clin. N. America. 1963; 43: 1541 - 50.

65. Dewey P. The posterior interosseous nerve and resistant tennis elbow.
J. Bone Jt. Surg. 1973; 55B: 435.

66. Dickson J.A., Crosby E.H. Periarthritis of the shoulder.
J.A.M.A. 1932; 99: 2252 - 7.

67. Dittrich R.J. Radiohumeral bursitis (tennis elbow). Report of two cases.
Am. J. Surg. 1929; 7: 411 - 4.

68. Dixon A. St-J. Soft tissue rheumatism : concept and classification.
Clin. Rheum. Dis. 1979; 5: 139 - 42.

69. Doenhoff M.J., Janossy G., Greaves M.F., Gomer K.J., Snajdr J. Lymphocyte activation. 6. A re-evaluation of factors affecting selectivity of polyclonal mitogens for mouse T & B cells.
Clin. Exp. Immunol. 1974; 17: 475 - 90.

70. Dolgu-Saburoff B. Uber ursprung und insertion der skelettmuskeln.
Anat. Anz. 1929; 68: 30 - 87.

71. Doody S.G., Waterland J.C., Freedman L. Scapulohumeral goniometer.
Arch. Phys. Med. Rehabil. 1970; 51: 711 - 3.

72. Duke O., Zecler E., Grahame R. Anti-inflammatory drugs in periarthritis of the shoulder : a double-blind between-patient study of naproxen versus indomethacin.
Rheumatol. Rehabil. 1981; 20: 54 - 9.

73. Duplay S. De la peri-arthrite scapulo-humerale et des raideurs de l'epaule qui en sont la consequence.
Arch. Gen. Med. 1872; 20: 513 - 42.

74. Dyson M., Franks C., Suckling J. Stimulation of healing of varicose ulcers by ultrasound.
Ultrasonics. 1976; 14: 232 - 6.

75. Dyson M., Pond J.B., Joseph J., Warwick R. The stimulation of tissue regeneration by means of ultrasound.
Clin. Sci. 1968; 35: 273 - 85.

76. Edgar W.H. Tennis elbow.
Br. Med. J. 1929; ii: 1219 - 20.

77. Edwards D.A.W. The blood supply and lymphatic drainage of tendons.
J. Anat. 1946; 8: 147 - 52.

78. Ehlich M., Carp S., Berkowitz S., Spitzer N., Silver M., Steinbrocker O. ACTH and cortisone in periarthritis of the shoulder.
Ann. Rheum. Dis. 1951; 10: 485 - 6.

79. Fearnley M.E., Vadasz I. Factors influencing the response of lesions of the rotator cuff of the shoulder to local steroid injections.
Ann. Phys. Med. 1969; 10: 53 - 63.

80. Fisher A.G.T. Treatment by manipulation. 2nd Edition.
Lewis & Co., London, 1928.

81. Fisher H., Mankin H., Mooney V. Painful shoulders in patients with pulmonary tuberculosis.
Am. Rev. Respir. Dis. 1963; 87: 88 - 92.

82. Fitton-Jackson S., Bassett C.A.L. The response of skeletal tissues to pulsed magnetic fields.
In : Richards R.J., Rajan K.T. (eds.). Tissue culture in medical research (II). Pergamon Press, Oxford, 1980 : 21 - 8.

83. Flax H.J. Ultrasound treatment of peritendinitis calcarea of the shoulder.
Am. J. Phys. Med. 1964; 43: 117 - 24.

84. Fleming A., Dodman S., Beer T.C., Crown S. Personality in frozen shoulder.
Ann. Rheum. Dis. 1976; 35: 456 - 7.

85. Fowler E.B. Stiff painful shoulders exclusive of TB and other infections.
J.A.M.A. 1933; 101: 2106 - 8.

86. Franklin E.C., Nemeik F.J. Shoulder dysfunction in pulmonary tuberculosis.
Am. J. Med. Sci. 1954; 227: 601 - 8.

87. Freedman L., Munro R. Abduction of the arm in the scapular plane: scapular and glenohumeral movement. A roentgenographic study.
J. Bone Jt. Surg. 1966; 48A: 1503 - 8.

88. Friedlander H.L., Reid R.L., Cape R.F. Tennis elbow.
Clin. Orthop. 1967; 51: 109 - 16.

89. Furlong R. Tennis elbow.
Practitioner. 1953; 171: 320 - 2.

90. Garden R.S. Tennis elbow.
J. Bone Jt. Surg. 1961; 43B: 100 - 6.

91. Gardener R.C. Tennis elbow : diagnosis, pathology and treatment. 9 severe cases treated by a new reconstructive operation.
Clin. Orthop. 1970; 72: 248 - 53.

92. Gianetti J.F. The anatomy of the radial nerve at the elbow and it's relationship of tennis elbow.
J. Bone Jt. Surg. 1968; 50A: 843.

93. Glyn J.H., Newton D.R.L. Steroid therapy.
Ann. Phys. Med. 1958; 4: 176 - 92.

94. Goldie I. Epicondylitis lateralis humeri, epicondylalgia or tennis elbow.
Acta. Chir. Scand. 1964: Suppl. 339.

95. Golding F.C. The shoulder - the forgotten joint.
Br. J. Radiol. 1962; 35: 149 - 58.

96. Good A.E., Green R.A., Zarafonetis C.J.D. Rheumatic symptoms during tuberculosis therapy.
Ann. Intern. Med. 1965; 63: 800 - 7.

97. Gray R.G., Gottlieb N.L. Rheumatic disorders associated with diabetes mellitus : literature review.
Semin. Arth. Rheum. 1976; 6: 19 - 34.

98. Gray's Anatomy, 36th Edition.
Williams P.L., Warwick R (ed's). Churchill Livingstone, London, 1980.

99. Grey R.G. The natural history of 'idiopathic' frozen shoulder.
J. Bone Jt. Surg. 1978; 60A: 564.

100. Gruchow H.W., Pelletier D. An epidemiological study of tennis elbow.
Amer. J. Sports Med. 1979; 7: 234 - 8.

101. Guillemart A., Besnard J.C., Le Pape A., Gally G., Fetiffoff F.
Skeletal uptake of pyrophosphate-labelled with technetium-95m and technetium-96 as evaluated by autoradiography.
J. Nucl. Med. Allied Sci. 1978; 19: 895 - 9.

102. Gunn C.C., Milbrandt W.E. Tennis elbow and the cervical spine.
Can. Med. Assoc. J. 1976; 114: 803 - 9.

103. Ha'eri G.B., Maitland A. Arthroscopic findings in the frozen shoulder.
J. Rheumatol. 1981; 8: 149 - 52.

104. Hagert C.G., Lundberg G., Hansen T. Entrapment of the posterior interosseous nerve.
Scand. J. Plast. Reconstr. Surg. 1977; 11: 205 - 12.

105. Haggart G.E., Dignam R.J., Sullivan T.S. Management of frozen shoulder.
J.A.M.A. 1956; 161: 1219 - 22.

106. Haines J.F., Hargadon E.J. Manipulation as the primary treatment of the frozen shoulder.
J. Roy. Coll. Surg. Edinb. 1982; 27: 271 - 5.

107. Ham A.W. Histology
J.P. Lippincott & Co, London, 1974: 363 - 77.

108. Hamilton, Boyd & Mossman's Human Embryology, 4th Edition.
In : Hamilton W.J., Mossman H.W. (ed's). Williams's & Wilkins Co, Baltmore, 1972 : 548 - 65.

109. Hansson K.G., Horwich I.D. Epicondylitis humeri.
J.A.M.A. 1930; 94: 1557 - 61.

110. Harkiss G.D., Brown D.L. Detection of immune complexes by a new assay, the polyethylene glycol precipitation-complement consumption test (PEG-CC).
Clin. Exp. Immunol. 1979; 36: 117 - 29.

111. Harvey W., Dyson M., Pond J.B., Grahame R. The stimulation of protein synthesis in human fibroblasts by therapeutic ultrasound.
Rheumatol. Rehabil. 1975; 14: 273.

112. Havers C. Osteologia Nova.
London, Smith 1691.

113. Hazleman B.L. The painful stiff shoulder.
Rheum. Phys. Med. 1972; 11: 413 - 21.

114. Heikkinen E., Suominen H., Vibersari M., Vuori I., Kiiskinen A. Effect of physiological training in enzyme activities of bones, tendons and skeletal muscle in mice.
In : Howard H., Poortmans J.R. (ed's). Metabolic adaptation to prolonged physical exercise. Birkhauser, Basel, 1975: 262 - 7.

115. Heiple K.G., Nash C.L., Klein I. A study of ^{14}C-labelled collagen of rat homograft tendon.
J. Bone Jt. Surg. 1967; 49A: 1109 - 14.

116. Herberts P., Kadefors R., Andersson G., Petersen I. Shoulder pain in industry : an epidemiological study on welders.
Acta Orthop. Scand. 1981; 52: 299 - 306.

117. Hess P., Bonmann K.H. Roentgentherapy in Epicondylitis.
Strahlentherapie. 1955; 96: 75 - 81.

118. Hitchcock H.H., Bechtol C.O. Painful shoulder : observation on the role of the tendon of the long head of biceps brachii in it's causation.
J. Bone Jt. Surg. 1948; 30A: 263 - 73.

119. Hitzrot J.M. A composite study of the axillary artery in man.
Bull. John Hopkins Hosp. 1901; 121: 136 - 59.

120. Hobhouse N., Heald C.B. A case of posterior interosseous paralysis.
Br. Med. J. 1936; i: 841.

121. Hohl M., Epicondylitis - tennis elbow.
Clin. Orthop. 1961; 19: 232 - 8.

122. Hohmann G. Das wesen und die behandlung des sogenannten Tennisellenbogens.
Munchener Med. Wochenscrift. 1933; 80: 250 - 2.

123. Hohmann G. Tennisellenbogen Verhandl.
Deutsch Orthop. Gesellsch. 1927; 21: 349 - 54.

124. Hollander J.L. Intra-articular hydrocortisone in arthritis and allied conditions.
J. Bone Jt. Surg. 1953; 35A: 983 - 90.

125. Hollingsworth G.R., Ellis R.M., Hattersley T.S. Comparison of injection techniques for shoulder pain : results of a double-blind randomised study.
Br. Med. J. 1983; 287: 1339 - 41.

126. Holloszy J.O. Biochemical adaptations to endurance exercise in muscle.
Ann. Rev. Physiol. 1971; 38: 273 - 91.

127. Hombal J.W.R., Owen R. Carpal tunnel decompression and trigger digits.
The Hand. 1970; 2: 192 - 6.

128. Hughes E.S.R. Acute deposition of calcium near the elbow.
J. Bone Jt. Surg. 1950; 32B: 30 - 4.

129. Hughes G.R.V., Currey H.L.F. Hypospray treatment of tennis elbow.
Ann. Rheum. Dis. 1969; 28: 58 - 62.

130. Huskisson E.C., Bryans R. Diclofenac sodium in the treatment of the painful stiff shoulder.
Curr. Med. Res. Opin. 1983; 8: 350 - 3.

131. Janossy G., Greaves M.F. Lymphocyte activation. 1. Response of T and B lymphocytes to phytomitogens.
Clin. Exp. Immunol. 1971; 9: 483 - 98.

132. Jayson M. Frozen shoulder : adhesive capsulitis.
Br. Med. J. 1981; 283: 1005 - 6.

133. Johnson J.T.H. Frozen shoulder syndrome in patients with pulmonary tuberculosis.
J. Bone Jt. Surg. 1959; 41A: 877 - 82.

134. Jones D.M. Tennis elbow.
J. Bone Jt. Surg. 1958; 40B: 825.

135. Jones J.G., Park J.R., Hazleman B.L., Ward M., Bulgen D.Y. Lack of consistent peripheral blood lymphocyte transformation responses in polymyalgia rheumatica and giant cell arteritis.
J. Rheumatol. 1980; 7: 891 - 4.

136. Jozsa L., Balint J.B., Reffy A., Demel Z. Histochemical and ultrastructural study of adult human tendon.
Acta. Histochem. 1979; 65: 250 - 7.

137. Kaplan E.B. Treatment of tennis elbow (epicondylitis) by denervation.
J. Bone Jt. Surg. 1959; 41A: 147 - 51.

138. Kernwein G.A., Rosenberg B., Sneed W.R. Arthrographic studies of the shoulder joint.
J. Bone Jt. Surg. 1957; 39A: 1267 - 79.

139. Kessel L., Bayley I., Young A. The frozen shoulder.
Br. J. Hosp. Med. 1981; 25: 334 - 8.

140. Kessel L., Watson M. The painful arc syndrome : clinical classification as a guide to management.
J. Bone Jt. Surg. 1977; 58B: 166 - 72.

141. Keyes E.L. Anatomical observations on senile changes in the shoulder.
J. Bone Jt. Surg. 1935; 17A: 953 - 60.

142. King J.M., Holmes G.W. The diagnosis and treatment of 450 painful shoulder.
J.A.M.A. 1927; 89: 1956 - 81.

143. Kivi P. The etiology and conservative treatment of humeral epicondylitis.
Scand. J. Rehab. Med. 1982; 15: 37 - 41.

144. Kolliker A. Manual of Human Histology.
Adlard, London, 1853.

145. Kopell H.P., Thompson W.A.L. Pain and the frozen shoulder.
Surg. Gyn. Obstet. 1959; 109: 92 - 6.

146. Kopell H.P., Thompson W.A.L. Peripheral entrapment neuropathies.
Williams and Wilkins Co., Baltimore, 1963.

147. Kort J., Ito H., Bassett C.A.L. Effects of pulsing electromagnetic fields on peripheral nerve regeneration.
J. Bone Jt. Surg. Orthop. Trans. 1980; 4: 238.

148. Kozin F. Painful shoulder and the reflex sympathetic dystrophy syndrome
In : McCarty D.J. (ed). Arthritis and Allied Conditions, 9th Edition. Lea & Febiger, Philadelphia, 1979: 1091 - 1120.

149. Lachmann P.J., Hobart M.J., Aston W.P. Complement technology.
In : Weir D.M. (ed). Handbook of Experimental Immunology (Immunochemistry), 2nd Edition. Blackwell Scientific Publications. Oxford, 1973: 5.1 - 5.17.

150. Laing P.G. The arterial supply of the adult humerus.
J. Bone Jt. Surg. 1956; 38A: 1105 - 16.

151. Lancet Editorial Soft-tissue rheumatism.
Lancet. 1972; ii: 624.

152. Lee M., Haq A.M.M.M., Wright V., Longton E.B. Periarthritis of the shoulder : a controlled trial of physiotherapy.
Physiotherapy. 1973; 59: 312 - 5.

153. Lee P.N., Lee M., Haq A.M.M.M., Longton E.B., Wright V. Periarthritis of the shoulder - trial of treatments investigated by multivariate analysis.
Ann. Rheum. Dis. 1974; 33: 116 - 9.

154. Lehmann J.F., De Lateur B.J., Warren C.G., Stonebridge J.B. Healing produced by ultrasound in bone and soft tissue.
Arch. Phys. Med. 1967; 48: 397 - 401.

155. Lehmann J.F., Erickson D.J., Martin G.M., Krusen F.H. Comparison of ultrasonic and microwave diathermy in the physical treatment of periarthritis of the shoulder.
Arch. Phys. Med. 1954; 35: 627 - 38.

156. Lehmann J. F., Warren C.G., Scham S.M. Therapeutic heat and cold.
Clin. Orthop. 1974; 99: 207 - 45.

157. Lequesne M., Dang N., Bensasson M., Mery C. Increased association of diabetes mellitus with capsulitis of the shoulder and shoulder-hand syndrome.
Scand. J. Rheumatol. 1977; 6: 53 - 6.

158. Levinsky R.J., Soothill J.S. The heterogeneity of immune complexes in disease.
In : Peters H. (ed). Protides of the biological fluids. Proceedings of the 26th colloquium. Pergamon Press, Oxford, 1978: 243 - 46.

159. Liebolt F.L. Frozen shoulder. Passive exercise for treatment
N.Y. State J. Med. 1970; 70: 2085.

160. Lindblom K. On pathogenesis of rupture of the tendon aponeurosis of the shoulder joint.
Acta. Radiol. 1939; 20: 563 - 77.

161. Lindblom K., Palmer I. Ruptures of the tendon aponeurosis of the shoulder joint - the so-called supraspinatus rupture.
Acta. Chir. Scand. 1939; 82: 133 - 42.

162. Lippmann R.K. Frozen shoulder, periarthritis, bicipital tenosynovitis.
Arch. Surg. 1943; 47: 283 - 96.

163. Little E.M. Tennis elbow.
Br. Med. J. 1920; ii: 190.

164. Lloyd-Roberts G.C., French P.R. Periarthritis of the shoulder. A study of the disease and its treatment.
Br. Med. J. 1959; i: 1569 - 71.

165. Lorenz T.H., Musser M.J. Life stress, emotions and painful stiff shoulders.
Ann. Intern. Med. 1952; 37: 1232 - 44.

166. Luben R.A., Chin C.D., Chen M.C.Y., Rosen D.M., Adey W.R. Effects of electro-magnetic stimuli on bone cells in vitro, inhibition of responses to parathyroid hormone by low energy low frequency waves.
Proc. Natl. Acad. Sci. USA. 1982; 79: 4180 - 4.

167. Lundberg B.J. Glycosaminoglycans of the normal and frozen shoulder joint capsule.
Clin. Orthop. 1970; 69: 279 - 84.

168. Lundberg B.J. The frozen shoulder.
Acta. Orthop. Scand. 1969: Suppl. 119.

169. Lunt M.J., Ashley B. A simple radiation balance for measuring ultrasonic power.
J. Med. Engin. & Technol. 1979; 3: 194 - 7.

170. Macnab I. Rotator cuff tendinitis.
Ann. Roy. Coll. Surg. Engl. 1973; 53: 271 - 87.

171. Maitland G.D. Peripheral manipulations.
Butterworth, London, 1970: 25 - 51.

172. Marlin T. Treatment of tennis elbow with some observations on joint manipulation.
Lancet. 1930; i: 509 - 11.

173. Matthews P., Leyshon R. Acute calcification in tennis and golfer's elbow.
Rheumatol. Rehabil. 1980; 19: 151 - 3.

174. Mayer L. The physiological method of tendon transplantation.
Surg. Gynae. Obstet. 1916; 22: 182 - 97.

175. McCarty D.J., Halverson P.B., Carrera G.F., Brewer B.J., Kozin F. 'Milwaukee Shoulder' - association of microspheroids containing hydroxy-apatite crystals, active collagenase and neutral protease with rotator cuff defects.
Arth. Rheum. 1981; 24: 464 - 73.

176. McLaughlin H.L. Frozen shoulder.
Clin. Orthop. 1961; 20: 126 - 31.

177. McMaster P.E. Tendon and muscle ruptures : clinical and experimental studies of the course and location of subcutaneous ruptures.
J. Bone Jt. Surg. 1933; 15A: 705 - 22.

178. Meherin J.M., Cooper C.E. Tennis elbow.
Am. J. Surg. 1950; 80: 622 - 5.

179. Meulengracht E., Schwartz M. The course and prognosis of periarthrosis humeroscapularis with special regard to cases with general symptoms.
Acta. Med. Scand. 1952; 143: 350 - 60.

180. Meyer A.W. Further evidence of attrition in the human body.
Am. J. Anat. 1924; 34: 241 - 67.

181. Meyer A.W. The minute anatomy of attrition lesions.
J. Bone Jt. Surg. 1931; 13A: 341.

182. Mills G.P. Treatment of tennis elbow.
Br. Med. J. 1928; i: 12 - 3.

183. Mills G.P. Treatment of tennis elbow.
Br. Med. J. 1937; ii: 212 - 3.

184. Moore M. Radiohumeral synovitis. A cause of persistent elbow pain.
Surg. Clin. N. Amer. 1953; 33: 1363 - 71.

185. Morris H. Lawn tennis elbow.
Br. Med. J. 1883; ii: 557.

186. Morris H. The rider's sprain.
Lancet. 1882; ii: 133 - 4.

187. Moseley H.F., Goldie I. The arterial pattern of the rotator cuff of the shoulder.
J. Bone Jt. Surg. 1963; 45B: 780 - 9.

188. Moseley H.F. Shoulder lesions, 3rd Edition.
Charles C. Thomas, Springfield, Ill., 1947.

189. Mueller E.E., Mead S., Schultz B.F., Vaden M.R. A placebo-controlled study of ultrasound treatment for periarthritis.
Amer. J. Phys. Therapy. 1954; 33: 31 - 5.

190. Mulholland R.C. Non-traumatic progressive paralysis of the posterior interosseous nerve.
J. Bone Jt. Surg. 1966; 48A: 781 - 5.

191. Mulier J.C., Spaas F. Outpatient treatment of surgically resistant non-unions by induced pulsing current - clinical results.
Arch. Orthop. Traumat. Surg. 1980; 97: 293 - 7.

192. Munting E. Ultrasonic therapy for painful shoulders.
Physiotherapy. 1978; 64: 180 - 1.

193. Murley A.H.G. Tennis elbow treated with hydrocortisone acetate.
Lancet. 1954; ii: 223 - 5.

194. Murnaghan G.F., McIntosh D. Hydrocortisone in painful shoulder. A controlled study.
Lancet. 1955; ii: 798 - 800.

195. Murray-Leslie C.F., Wright V. Carpal tunnel syndrome, humeral epicondylitis and the cervical spine : a study of clinical and dimentional relations.
Br. Med. J. 1976; i: 1439 - 42.

196. Nelson C.L., Burton R.I. Upper extremity arthrography.
Clin. Orthop. 1975; 107: 62 - 72.

197. Nelson S.D., Darke C., Tovey G.H. HL-A antigen frequencies in normal blood donors, kidney donors and prospective kidney recipients.
Tissue Antigens. 1974; 4: 361 - 5.

198. Nevelos A.B. The treatment of tennis elbow with triamcinolone acetonide.
Curr. Med. Res. Opin. 1980; 6: 507 - 9.

199. Neviaser J.S. Adhesive capsulitis of the shoulder : a study of the pathological findings in periarthritis of the shoulder.
J. Bone Jt. Surg. 1945; 27A: 211 - 22.

200. Neviaser J.S. Adhesive capsulitis and the stiff and painful shoulder.
Orthop. Clin. N. Amer. 1980; 11: 327 - 31.

201. Neviaser J.S. Arthrography of the shoulder joint : study of the findings in adhesive capsulitis of the shoulder.
J. Bone Jt. Surg. 1962; 44A: 1321 - 30.

202. Newman J.H., Goodfellow J.W. Fibrillation of head of radius as one cause of the tennis elbow.
Br. Med. J. 1975; ii: 328 - 30.

203. Niepel G.A., Sit'aj S. Enthesopathy.
Clin. Rheum. Dis. 1979; 5: 857 - 72.

204. Nirschl R.P., Pettrone F.A. Tennis elbow : the surgical treatment of lateral epicondylitis.
J. Bone Jt. Surg. 1979; 61A: 832 - 9.

205. Nirschl R.P. Tennis elbow.
Orthop. Clin. N. Amer. 1973; 4: 787 - 800.

206. Noy S., Dekel S., Orgad S., Efter T., Mizrachi Y., Gazit E.
HL-A B27 and frozen shoulder.
Tissue Antigens. 1981; 18: 364.

207. Older M.W.J., McIntyre J.L., Lloyd G.J. Distension arthrography of the shoulder joint.
Canad. J. Surg. 1976; 19: 203 - 7.

208. Olsson O. Degenerative changes in the shoulder joint and their connection with shoulder pain.
Acta. Chir. Scand. 1953: Suppl. 181.

209. Osgood R.B. Radiohumeral bursitis, epicondylitis and epicondylalgia (tennis elbow). A personal experience.
Arch. Surg. 1922; 4: 420 - 33.

210. O'Sullivan S. Tennis elbow.
Br. Med. J. 1883; ii: 1168.

211. Palmer I. Remarks on the so-called supraspinatus ruptures in the shoulder joint, their diagnosis and treatment.
Acta Orthop. Scand. 1939; 10: 323 - 30.

212. Partridge R.E.M., Duthie J.J.R. Rheumatism in dockers and civil servants. A comparison of heavy manual workers and sedentary workers. Ann. Rheum. Dis. 1968; 27: 559 - 67.

213. Paul N.W. Radiohumeral bursitis - is it traumatic? Analysis and report of 314 cases. Industr. Med. Surg. 1957; 26: 383 - 90.

214. Peacock E.E. A study of the circulation in normal tendons and healing grafts. Ann. Surg. 1959; 149: 415 - 28.

215. Pettersson G. Ruptures of the tendon aponeurosis of the shoulder joint in anterior inferior dislocation. Acta Chir. Scand. 1942: Suppl 77.

216. Phalen G.S. The carpal tunnel syndrome. J. Bone Jt. Surg. 1966; 48A: 211 - 28.

217. Piaggi V., Mingione A. A study of tendon blood flow using ^{133}Xenon. The Hand. 1981; 13: 48 - 50.

218. Potenza A.D. Critical evaluation of flexor-tendon healing and adhesion formation within artificial digital sheaths. J. Bone Jt. Surg. 1963; 45A: 1217 - 33.

219. Preston B.J., Jackson J.P. Shoulder arthrography. Br. J. Radiol. 1976; 49: 266.

220. Pringle J.A. Byers P.D., Brown M.E.A. Immunofluorescence in osteoarthritis. Nature. 1978; 274: 94.

221. Quigley T.B. Treatment of checkrein shoulder by use of manipulation and cortisone. J.A.M.A. 1956; 161: 850 - 4.

222. Quin C.E., Binks F.A. Tennis elbow (epicondylalgia externa) treated with hydrocortisone. Lancet. 1954; ii: 221 - 3.

223. Quin C.E. 'Frozen' shoulder : evaluation of treatment with hydrocortisone injections and exercise. Ann. Phys. Med. 1965; 8: 22 - 9.

224. Quin C.E. Humeroscapular periarthritis : observations on the effects of X-ray therapy and ultrasonic therapy in cases of frozen shoulder. Ann. Phys. Med. 1969; 10: 64 - 9.

225. Rathbun J.B., Macnab I. The microvascular pattern of the rotator cuff. J. Bone Jt. Surg. 1970; 52A: 540 - 53.

226. Reeves B. Arthrographic changes in frozen and post-traumatic stiff shoulders. Proc. Roy. Soc. Med. 1966; 59: 827 - 30.

227. Reeves B. Experiments on the tensile strength of the anterior capsular structures of the shoulder in man.
J. Bone Jt. Surg. 1968; 50B: 858 - 65.

228. Reeves B. The natural history of the frozen shoulder syndrome.
Scand. J. Rheumatol. 1975; 4: 193 - 6.

229. Rhind V., Downie W.W., Bird H.A., Wright V., Engler C. Naproxen and indomethacin in periarthritis of the shoulder.
Rheumatol. Rehabil. 1982; 21: 51 - 3.

230. Rhodin J.A. Histology. A text and atlas.
Oxford University Press. New York, 1974: 139 - 71.

231. Richardson A.T. The painful shoulder.
Practitioner. 1975; 215: 27 - 35.

232. Rizk T.E., Christopher R.P., Pinals R.S., Higgins A.C., Frix R. Adhesive capsulitis (frozen shoulder) : a new approach to it's management.
Arch. Phys. Med. Rehabil. 1983; 64: 29 - 33.

233. Rizk T.E., Pinals R.S. Frozen shoulder.
Semin. Arth. Rheum. 1982; 11: 440 - 52.

234. Rizk T.E., Pinals R.S. Histocompatibility type and racial incidence in frozen shoulder.
Arch. Phys. Med. Rehabil. 1984; 65: 33 - 4.

235. Roles N.C., Maudsley R.H. Radial tunnel syndome : resistant tennis elbow as a nerve entrapment.
J. Bone Jt. Surg. 1972; 54B: 499 - 508.

236. Romer F. Some observations on 'tennis elbow'.
Lancet. 1922; ii: 67.

237. Rothman R.H., Parke W.W. The vascular anatomy of the rotator cuff.
Clin. Orthop. 1965; 41: 176 - 86.

238. Roy S., Oldham R. Management of painful shoulder.
Lancet. 1976; ii: 1322 - 4.

239. Runge F. Zur genese und Behandlung des Schreibekrampfes.
Berliner Klin. Wochenschrift. 1873; 10: 245 - 9.

240. Saha N.C. Painful shoulder in patients with chronic bronchitis and emphysema.
Am. Rev. Respir. Dis. 1966; 94: 455 - 6.

241. Samilson R.L., Raphael R.L., Post L., Noonan C., Siris E., Raney Jr. F.L. Shoulder arthrography.
J.A.M.A. 1961; 175: 773 - 9.

242. Sarkar K., Uhthoff H.K. Ultrastructure of the common extensor tendon in tennis elbow.
Virchows Arch. Path. Anat. & Histol. 1980; 386: 317 - 30.

243. Seignalet J., Sang J., Cuillens T.P., Lapinski H. Lack of association between HL-A B27 and frozen shoulder.
Tissue Antigens. 1981; 18: 364.

244. Sharpey W., Ellis G.V. Elements of anatomy 6th Edition.
Quain J. (ed). Walton & Moberly, London, 1856.

245. Sharrard W.J.W., Sutcliffe M.L., Robson M.J., MacEachern A.G. The treatment of fibrous non-unions of fractures by pulsing electro-magnetic stimulation.
J. Bone Jt. Surg. 1982; 64B: 189 - 93.

246. Sheldon P.J.H. A retrospective survey of 102 cases of shoulder pain.
Rheum. Phys. Med. 1972; 11: 422 - 7.

247. Silberstein C.E. The evolution of degenerative changes in the cervical spine and an investigation into the joints of Luschka.
Clin. Orthop. 1965; 40: 184 - 204.

248. Simmonds F.A. Shoulder pain with particular reference to the frozen shoulder.
J. Bone Jt. Surg. 1949; 31B: 426 - 32.

249. Simon W.H. Soft tissue disorders of the shoulder : frozen shoulder, calcific tendinitis and bicipital tendinitis.
Orthop. Clin. N. Amer. 1975; 6: 521 - 39.

250. Sinclair A. Tennis elbow in industry.
Br. J. Industr. Med. 1965; 22: 144 - 8.

251. Skinner H.A. Anatomical considerations relative to rupture of the supraspinatus tendon.
J. Bone Jt. Surg. 1937; 19A: 137 - 51.

252. Smythe H.A. Fibrositis and other diffuse musculoskeletal syndromes.
In : Kelly W.N., Harris E.D.M., Ruddy S., Sledge C.B. (ed's).
Textbook of Rheumatology Volume 1. W.B. Saunders & Co, Philadelphia, 1981: 485 - 93.

253. Somerville E.W. Tennis elbow.
J. Bone Jt. Surg. 1963; 45B: 621.

254. Spencer G.E., Herndon C.H. Surgical treatment of epicondylitis.
J. Bone Jt. Surg. 1953; 35A: 421 - 4.

255. Stack J.K. Acute and chronic bursitis in the region of the elbow.
Surg. Clin. N. Amer. 1949; 29: 155 - 62.

256. Steinbrocker O., Argyros T.G. Frozen shoulder treated by local injection of depot corticosteroids.
Arch. Phys. Med. Rehabil. 1974; 55: 209 - 13.

257. Stoddard A. Manipulation of the elbow joint.
Physiotherapy. 1971; 57: 259 - 60.

258. Stodell M.A., Nicholson R., Scott J., Sturrock R.D. Radio-isotope scanning in the painful shoulder.
Rheumatol. Rehabil. 1980; 19: 163 - 6.

259. Storey G.O. Cervical spondylosis : its early diagnosis and treatment.
Wilkinson M.W. (ed). Heinemann, London, 1977.

260. Stovell P.B., Beinfield M.S. Treatment of resistant lateral epicondylitis by lengthening of the extensor carpi radialis brevis tendon.
Surg. Obstet. Gynae. 1979; 149: 526 - 8.

261. Suominen H., Heikkinen E. Enzyme activities in muscle and connective tissue of musculus vastus lateralis in habitually training and sedentary 33 - 70 year old men.
Eur. J. Appl. Physiol. 1975; 34: 249 - 54.

262. Sutcliffe M.L., Goldberg A.A.J. The treatment of congenital pseudarthrosis of the tibia with pulsing electro-magnetic fields.
Clin. Orthop. 1982; 166: 44 - 57.

263. Thomas D., Williams R.A., Smith D.S. The frozen shoulder : a review of manipulative treatment.
Rheumatol. Rehabil. 1980; 19: 173 - 9.

264. Tipton C.M., Vailas A.C., Laughlin M.H., Diana J.M. Aerobic characteristics of isolated ligaments and tendons.
Physiologists. 1975; 14: 422 - 30.

265. Trethowan W.H. Tennis elbow.
Br. Med. J. 1929; ii: 1218.

266. Tucker W.E. Injuries and their treatment.
H.K. Lewis, London, 1935.

267. Uhthoff H.K., Sarkar K. A re-appraisal of tennis elbow.
Acta Orthop. Belg. 1980; 46: 74 - 82.

268. Van Demark R.E., Myrabo A.K. Calcaneous tendinitis at the elbow.
Clin. Orthop. 1956; 7: 237 - 40.

269. Van Rossum J., Buruma O.J.S., Kamphuisen H.A.C., Onvlee G.J. Tennis elbow - a radial tunnel syndrome ?
J. Bone J. Surg. 1978; 60B: 197 - 8.

270. Wadsworth T.G. Lateral epicondylitis (tennis elbow).
Lancet. 1972; ii: 959 - 60.

271. Wall J. Tennis elbow.
Industr. Med. Surg. 1960; 29: 173 - 5.

272. Watson-Jones R. Simple treatment of stiff shoulder.
J. Bone Jt. Surg. 1963; 45B: 207.

273. Weiss J.J., Thompson G.R., Doust V., Burgener F.A. Arthrography in the diagnosis of shoulder pain and immobility.
Arch. Phys. Med. Rehabil. 1974; 55: 205 - 9.

274. Weiss J.J., Ting Y.M. Arthrography-assisted intra-articular injection of steroids in the treatment of adhesive capsulitis.
Arch. Phys. Med. Rehabil. 1978; 59: 285 - 7.

275. Werner C.O. Lateral elbow pain and posterior interosseous nerve entrapment.
Acta Orthop. Scand. 1979: Suppl 174.

276. West E.F. Tennis elbow.
Med. J. Australia. 1938; i: 330 - 1.

277. WHO Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death. 1965 Revision.
WHO; Geneva, 1967.

278. WHO Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death. 1975 Revision.
WHO; Geneva, 1977.

279. Wiley A.M., Older M.W. Shoulder arthroscopy. Investigation with a fibro-optic instrument.
Am. J. Sports Med. 1980; 8: 31 - 8.

280. Williams N.E., Seifert M.H., Cuddigan J.H.P., Wise R.A. Treatment of capsulitis of the shoulder.
Rheumatol. Rehabil. 1975; 14: 236.

281. Willis R.A. The borderline of embryology and pathology.
Butterworth and Co., London, 1958.

282. Wilson C.L., Duff G.L. Pathological study of degeneration and rupture of the supraspinatus tendon.
Arch. Surg. 1943; 47: 121 - 35.

283. Wilson D.H., Jagadeeshi P., Newman P.P., Harriman D.G.F. The effects of pulsed electro-magnetic energy on peripheral nerve regeneration.
Ann. N.Y. Acad. Sci. 1974; 238: 575 - 85.

284. Wilson D.H. Treatment of soft tissue injuries by pulsed electrical energy.
Br. Med. J. 1972; i: 269 - 70.

285. Winckworth C.E. Lawn tennis elbow.
Br. Med. J. 1883; ii: 708.

286. Withers R.J.W. The painful shoulder : review of 100 personal cases with remarks on the pathology.
J. Bone. Jt. Surg. 1949; 31B: 414 - 7.

287. Wolcott L.E., Wheeler P.C., Hardwicke H.M., Rowley B.A. Accelerated healing of skin ulcers by electro-therapy : preliminary clinical results.
Southern Med. J. 1969; 62: 795 - 801.

288. Wood P.H.N., Sturrock A.W., Badley E.M. Soft tissue rheumatism in the community.
Clin. Rheum. Dis. 1979; 5: 743 - 53.

289. Wood R.W., Loomis A.L. The physical and biological effects of high-frequency sound waves of great intensity.
The London, Edinburgh and Dublin Philosophical Magazine and Journal of Science (7th series). 1927; 4: 417 - 36.

290. Wright M.G., Richards A.J., Clarke M.B. 99-M pertechnetate scanning in capsulitis.
Lancet. 1975; ii: 1265.

291. Wright V., Haq A.M.M.M. Periarthritis of the shoulder.
I Aetiological considerations with particular reference to personality factors.
Ann. Rheum. Dis. 1976; 35: 213 - 9.

292. Wright V., Haq A.M.M.M. Periarthritis of the shoulder.
II Radiological features.
Ann. Rheum. Dis. 1976; 35: 220 - 6.

293. Young H.H., Ward L.E., Henderson E.D. Use of hydrocortisone acetate in the treatment of some common orthopaedic conditions.
J. Bone Jt. Surg. 1954; 36A: 602 - 9.

APPENDIX I

PROTOCOL FOR ASSESSMENT OF PATIENTS IN THE SHOULDER STUDIES

A. ASSESSMENT ONLY AT THE INITIAL VISIT:

GENERAL	Date; Patient No. Age; Sex; Arm - affected, dominant. Cause.
PAIN INQUIRY	Duration since onset of symptoms. Distribution and radiation. Present (yes/no) at night, on movement, at rest. Can you lie on the affected shoulder?
RANGE RESTRICTION INQUIRY (if present)	Duration since onset. Severity - Mild/Moderate/Severe. Progress from onset - Inc./Same/Dec.
NECK INQUIRY	Pain; Restriction; Paraesthesia/weakness in hands.
PREVIOUS THERAPY AND EFFICACY	NSAID'S, analgesics, injections, physiotherapy, other

GP ADVICE ON USE ON THE SHOULDER.

PRESENT MEDICATION.

PAST HISTORY	Shoulder pain; Musculoskeletal disorders; Medical and surgical conditions.
SYSTEMATIC INQUIRY	Opposite shoulder; Musculoskeletal disorders; General Health.
FAMILY HISTORY	Musculoskeletal; Other.
OCCUPATION AND HOBBIES	At onset of symptoms; During convalescence; In past; Time lost from each.
EXAMINATION	General; Systematic; Musculoskeletal Neck - flexion, extension, lateral flexion and rotation. Arms - power, sensation, reflexes.

X-RAY SHOULDER ± ACROMIOCLAVICULAR JOINTS.

BLOOD TESTS - ESR (Westergren), Rheumatoid factor.

DIAGNOSIS BY GP AT REFERRAL.

DIAGNOSIS AT CLINIC.

SUITABILITY FOR STUDY.

CONSENT (if applicable).

ARRANGE SPECIAL TESTS (if applicable).

B. ASSESSMENT AT THE INITIAL AND EACH FOLLOW-UP VISIT:

GENERAL — Date; Week No., Patient No.

THERAPY SINCE LAST VISIT

Paracetamol, diazepam, other.

Change since last visit.

PATIENT ASSESSMENT

Much/Abit better/Same/Worse

PAIN (10 cm scales)	'No pain'	'Severe as possible'
At night	I ----------	I
On movement	I ----------	I
At rest	I ----------	I

Can you lie on the affected shoulder?

Distribution - Same/Changed.

TENDERNESS — ACJ, Other

PAINFUL ARC ON ABDUCTION (0-3)

PAIN (0 - 3) ON RESISTED ABDUCTION, EXTERNAL AND INTERNAL ROTATION.

RANGE (Degrees)		RIGHT		LEFT	
		Active	Passive	Active	Passive
Abd.	gl-hum.				
	total				
Flex	gl-hum.				
	total				
Ext. Rot.					
Tot. Rot.					
Int. Rot.	(cm.)				

DIAGNOSIS

CHANGE IN THERAPY

COMMENTS

DATE OF NEXT VISIT.

APPENDIX II

PROTOCOL FOR ASSESSMENT OF PATIENTS WITH LATERAL EPICONDYLITIS IN THE PROSPECTIVE STUDY

A. ASSESSMENT ONLY AT THE INITIAL VISIT.

GENERAL	Date; Patient No. Age; Sex; Arm - affected, dominant. Cause.
PAIN INQUIRY	Duration since onset of symptoms. Distribution and radiation. Present (yes/no) - night pain, sleep disturbance. Actions causing pain (yes/no) - housework; carrying; lifting; writing/typing; sport; hobbies; other. Severity - Mild/Moderate/Severe. Progress from onset - Inc./Same/Dec.
RANGE INQUIRY	Elbow extension - Normal/Reduced Supination/Pronation - Normal/Reduced.
GRIP STRENGTH	Normal/Decreased.
NECK INQUIRY	Pain; Restriction; Paraesthesia/weakness in hands.
PREVIOUS THERAPY AND EFFICACY	NSAID'S, Analgesics, Injections (and/no.), Ultrasound, Other. Date of last injection. Was it of benefit?
PRESENT MEDICATION.	
PAST HISTORY.	Shoulder pain; Musculoskeletal disorders; Medical and surgical conditions.
SYSTEMATIC INQUIRY	Opposite shoulder; Musculoskeletal disorders; General health.

FAMILY HISTORY Musculoskeletal; Other.

OCCUPATION AND HOBBIES

At onset of symptoms; Duration unable to do these.

EXAMINATION General; Systematic; Musculoskeletal.

Neck - flexion, extension, lateral flexion and rotation.

Arms - power, sensation, reflexes.

Shoulders; Other elbow.

X-RAY ELBOW

BLOOD TESTS - ESR (Westergren); Rheumatoid factor.

DIAGNOSIS BY GP AT REFERRAL

DIAGNOSIS AT CLINIC.

SUITABILITY FOR STUDY.

B. ASSESSMENT AT THE INITIAL AND EACH FOLLOW-UP VISIT:

GENERAL Date; Week No., Patient No.

THERAPY SINCE LAST VISIT

Paracetamol, Other.

Change since last visit.

PATIENT ASSESSMENT Much/Abit better/Same/Worse.

PAIN 'No pain' 'Severe as possible'
(10 cm scale) I ---------- I

Site of maximum pain; Radiation.

Distribution - Same/Changed.

TENDERNESS On L.E./Ant./Post./Diffuse/Absent/Other.

PASSIVE RANGE Elbow extension; Pronation; Supination.

PAIN (0 - 3) ON RESISTED WRIST DORSIFLEXION.
ON RESISTED MIDDLE FINGER EXTENSION.

GRIP STRENGTH (mm Hg). IN FLEXION.
IN EXTENSION.

WEIGHT TEST (0 - 3).

ACTIONS INDUCING PAIN - Housework; Carrying; Lifting; Writing/typing;
Sport; Hobbies; Other (state).

OTHER JOINTS Elbow; Shoulders; Neck.

DIAGNOSIS

CHANGE IN THERAPY

DATE OF NEXT VISIT.

APPENDIX III

QUESTIONNAIRE FOR THE RETROSPECTIVE ASSESSMENT OF LATERAL EPICONDYLITIS TREATED AT A RHEUMATOLOGY CLINIC 1 - 5 YEARS PREVIOUSLY

A. GENERAL QUESTIONS

Name; Sex; Age at onset of symptoms.
Date of tennis elbow; Arm affected; Dominant arm.
Occupation and hobbies at onset.
Cause of symptoms.

B. BEFORE HOSPITAL REFERRAL

Did the tennis elbow interfere (yes/no) with Work; Hobbies; Sport; Sleep?
What treatment did your GP give? - Tablets; Injections; Other (state).

C. HOSPITAL THERAPY

Duration before referral.
Treatment given (1) Injections (if yes) - How many? Were they painful? Did they help? Did the pain recur? If yes - after how long?

(2) Ultrasound (if yes) - Did it help? Did the pain recur? If yes - after how long?

(3) Other treatment (if yes) What? Did it help?

If more than 1 treatment - Which was first? Which was more effective?

D. AFTER DISCHARGE

Was elbow (choose one only) completely healed/need to protect arm/ache after extreme use/pain as bad as ever?
How long did symptoms persist?
Has pain recurred? How long after 'improved'?
If pain recurred - did you (choose 1) seek further help from GP or hospital/learn to live with pain/give up or limit work or hobbies/seek alternative therapy?

Is elbow still painful?
Has the other elbow been affected?
Have you ever had tennis elbow before?
Have you ever suffered from shoulder pain?

APPENDIX IV

MEDICAL PUBLICATIONS CONTAINING DATA IN THE THESIS

Binder A.I., De Silva M., Hazleman B.L. Soft tissue rheumatism (1).
Hospital Update. 1983; 9: 341 - 9.

Binder A.I., De Silva M., Hazleman B.L. Soft tissue rheumatism (2).
Hospital Update. 1983; 9: 471 - 80.

Bulgen D.Y., Binder A.I., Hazleman B.L., Park J.R. Immunological studies in frozen shoulder.
J. Rheumatol. 1983; 9: 893 - 8.

Binder A.I., Parr G., Hazleman B., Fitton-Jackson S. Pulsed electro-magnetic field therapy of persistent rotator cuff tendinitis : A double-blind controlled assessment.
Lancet. 1984; i: 695 - 8.

Bulgen D.Y., Binder A.I., Hazleman B.L., Dutton J., Roberts S. Frozen shoulder : prospective clinical study with an evaluation of three treatment regimens.
Ann. Rheum. Dis. 1984; 43: 353 - 60.

Binder A.I., Bulgen D.Y., Hazleman B.L., Roberts S. Frozen shoulder : a long-term prospective study.
Ann. Rheum. Dis. 1984; 43: 361 - 4.

Binder A.I., Bulgen D.Y., Hazleman B.L., Tudor J., Wraight P. Frozen shoulder : an arthrographic and radio-nuclear scan assessment.
Ann. Rheum. Dis. 1984; 43: 365 - 9.

Binder A.I., Hazleman B.L., Parr G., Roberts S. A controlled study of oral prednisolone in frozen shoulder.
Br. J. Rheumatol. (In press).

Binder A.I., Hazleman B.L. Lateral humeral epicondylitis - A study of natural history and the effect of conservative therapy.
Br. J. Rheum. 1983; 22: 73 - 6.

Binder A.I., Parr G., Page-Thomas P., Hazleman B. A clinical and thermographic study of lateral epicondylitis.
Br. J. Rheum. 1983; 22: 77 - 81.

Binder A.I., Hodge G., Greenwood A. , Hazleman B.L., Page-Thomas D.P. Is therapeutic ultrasound effective in treating soft tissue lesions?
Br. Med. J. 1985; 290: 512 - 4.