

## **JELENA SOKK**

Shoulder function in patients with frozen shoulder syndrome: the effect of conservative treatment and manipulation under general anaesthesia







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manipulation under general anaesthesia



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## LIST OF ORIGINAL PUBLICATIONS

The thesis is based on the following original papers, which are referred to in the text by their Roman numerals:

- I. **Jürgel J., Rannama L., Gapeyeva H., Ereline J., Kolts I., Pääsuke M.** Shoulder function in patients with frozen shoulder before and after 4-week rehabilitation. *Medicina (Kaunas)* 2005; 41: 30–38.
  - II. **Sokk J., Gapeyeva H., Ereline J., Kolts I., Pääsuke M.** Shoulder muscle strength and fatigability in patients with frozen shoulder syndrome: the effect of 4-week individualized rehabilitation. *Electromyography and Clinical Neurophysiology* 2007; 47(4–5): 205–213.
  - III. **Sokk J., Gapeyeva H., Ereline J., Merila M., Pääsuke M.** Shoulder muscle isometric strength and active range of motion in patients with frozen shoulder syndrome after manipulation under anesthesia. *Medicina (Kaunas)* 2012; 48: 331–337.
  - IV. **Sokk J., Gapeyeva H., Ereline J., Merila M., Pääsuke M.** Recovery of shoulder muscle function characteristics and active range of motion in patients with frozen shoulder after manipulation under anesthesia. *European Orthopaedics and Traumatology* 2012; 3: 169–176.
- Until 2007 – surname was Jürgel

The contribution of the dissertant to the completion of the doctoral thesis: Papers I–IV.

The dissertant had primary responsibility for protocol development, subject screening, performing measurements, preliminary and final data analysis, and writing of the manuscripts.



## ABBREVIATIONS

ABD	abduction
ADD	adduction
ADL	activities of daily living
aROM	active range of motion
BM	body mass
BMI	body mass index
CS	Constant Score
CT	conservative treatment
EMG	electromyography, electromyogram
EXT	extension
EXR	external rotation
FL	flexion
FSS	frozen shoulder syndrome
INR	internal rotation
MF	median frequency
MFslope	median frequency slope
MRI	magnetic resonance imaging
MUA	manipulation under general anaesthesia
NI	net impulse
NSAID	non-steroidal anti-inflammatory drugs
pROM	passive range of motion
ROM	range of motion
SE	standard error
SRQ	Shoulder Rating Questionnaire
TENS	transcutaneous electrical nerve stimulation
US	ultrasound
VAS	visual analogue scale

# I. INTRODUCTION

Frozen shoulder syndrome (FSS), also known as “adhesive capsulitis”, “scapulohumeral periarthritis”, “stiff and painful shoulder”, “periarticular adhesion”, “adherent subacromial bursitis”, “hypomobile syndrome” involves the shoulder joint and surrounding tissues (Neviaser, 1945; Neviaser and Hannafin, 2010; Cinar et al., 2010; Manske and Prohaska, 2010; Lorbach et al., 2010; Hsu et al., 2011). FSS occurs in 2-5% of the adult population (Leung and Cheing, 2008; Lorbach et al., 2010; Cinar et al., 2010; Favejee et al., 2011), and in up to 20% of patients with diabetes (Mavrikakis et al., 1989; Balci et al., 1999). However, 70% of patients are women (Sheridan and Hannafin, 2006), and there has been a secular trend of increasing among women more recently (White et al., 2011). FSS usually presents in the sixth decade of life (Dias et al., 2005; Jewell et al., 2009; Gaspar and Willis, 2009), and onset before the age of 40 is very uncommon (Dias et al., 2005).

FSS may develop after concurrence with shoulder trauma, cardiovascular and chronic lung diseases, pulmonary tuberculosis, diabetes, stroke or tumour, thyroid disease (Milgrom et al., 2008), prolonged immobilization, and when it concurs with an autoimmune disease in the patient (Hannafin and Chiaia, 2000; Garmland et al., 2000; De Ponti et al., 2006). FSS commonly begins gradually (61%) but in many cases FSS develops rapidly over a 24-48 hour period (39%). The condition has not been reported to have a disposition for race (Hsu et al., 2011) and recurrence of the FSS is rare (Cameron et al., 2000).

FSS is a disease with unclear etiology whereas it is a long-lasting disease and needs patience from the patients as well as doctors and physiotherapists. The FSS patients had shoulder pain and localized discomfort near the insertion of the deltoid muscle, limitation and pain during shoulder active (aROM) and passive range of motion (pROM), especially during flexion (FL), external (EXR) and internal rotation (INR) and with diminishing shoulder function (Neviaser and Neviaser, 1987). Patients with FSS have difficulties in everyday activities (dressing, grooming, and performing overhead reaching activities and so on for a period of several months to several years) and shoulder pain disturbing sleep at night on the affected side, which is a key diagnostic sign (Bunker and Anthony, 1995; Watson et al., 2000; Boyles et al., 2005; Jewell, 2009; Blanchard et al., 2010), waking up at night secondary to shoulder pain (Miller et al., 1996) and muscle spasms are common in patients with FSS (Wadsworth 1986).

The characteristic “shrug sign” develops in patients with FSS during glenohumeral joint elevation, where the scapula migrates upward prior to 60 deg of abduction (ABD). This indicates compensation due to the lack of capsular flexibility as well as a change in the central nervous system’s motor pattern due to inadequate movement in the involved shoulder joint. Adaptive posture may develop in patients with FSS, such as anterior shoulders or increased thoracic kyphosis (Page and Labbe, 2010).

Usually in the studies in patients with FSS, shoulder joint recovery is assessed by shoulder aROM or pROM, pain and different shoulder and health questionnaires. In our study, we also paid attention to the recovery of shoulder region muscle isometric strength, isometric working capacity, fatigability and endurance.

The main goal of this study was to follow shoulder aROM, shoulder pain and shoulder muscle function recovery in patients with FSS, who were treated conservatively or with manipulation under general anaesthesia (MUA) and compare the involved extremity with the uninvolved extremity.

## **2. REVIEW OF LITERATURE**

### **2.1. Definition and classification of frozen shoulder syndrome**

The definition for adhesive capsulitis, which was published by the association of American Shoulder and Elbow Surgeons is: “a condition of uncertain etiology characterized by significant restriction of both active and passive shoulder motion that occurs in the absence of a known intrinsic shoulder disorder” (Zuckerman et al., 1994).

Zuckerman et al. (2011) recommended a completed frozen shoulder syndrome (FSS) definition: “Frozen shoulder syndrome is a condition characterized by functional restriction of both active and passive shoulder motion for which radiographs of the glenohumeral joint are essentially unremarkable except for the possible presence of osteopenia or calcific tendonitis”.

Thus, the proposed classification model of FSS (Zuckerman et al., 2011):

- A. Primary: a diagnosis for all cases for which an underlying etiology or associated condition cannot be identified.
- B. Secondary: includes all cases of FSS in which an underlying etiology or associated condition can be identified.
  - 1. Intrinsic: limitation during shoulder aROM and pROM that occurs in association with rotator cuff disorders, biceps tendonitis, or calcific tendonitis, acromioclavicular arthritis.
  - 2. Extrinsic: an association with an identifiable abnormality remote to the shoulder itself (shoulder aROM and pROM found in association with previous ipsilateral breast surgery, cervical radiculopathy, chest tumour, previous cerebrovascular accident, or previous humeral shaft fracture, scapulothoracic abnormalities, acromioclavicular arthritis, or clavicle fracture, cardiopulmonary disease, Parkinson’s disease).
  - 3. Systemic: systemic disorders include but are not limited to diabetes, hyperthyroidism, hypothyroidism, hypoadrenalism, or any other condition that has been documented to have an association with the development of FSS.

Kelley et al. (2009) proposed the classification system, which is based on the FSS patients’ irritability level (low, moderate and high). In this classification system, irritability is determined based on shoulder pain, range of motion (ROM), and extent of disability. Patients with low irritability have little or no shoulder pain; therefore aROM and pROM are equal and disability is lower. These patients typically report stiffness rather than shoulder pain as the chief complaint. Patients with high irritability have significant shoulder pain resulting in the limited pROM (due to muscle guarding) and greater disability. These patients typically report shoulder pain rather than stiffness as the chief complaint (Kelley et al., 2009).

In conclusion, the doctors must take into account patients’ symptoms and problems before they diagnose FSS.

## **2.2. Frozen shoulder syndrome stages**

Using arthroscopic criteria, Neviaser and Neviaser (1987) modified four stages of FSS. Hannafin and Chiaia (2000), Sheridan and Hannafin (2006), Neviaser and Hannafin (2010) FSS stages criteria are presented in Table 1.

In conclusion, in the different stages patients with FSS had different problems and symptoms in the shoulder joint and surrounding tissues and felt discomfort for a long period (Wolf and Green, 2002). However, the diagnosis and treatment depend on the patient's complaints and medical assessment findings.

## **2.3. Etiology and pathology of frozen shoulder syndrome**

The etiology of FSS is still unknown. Cases may be related to immunologic, biochemical, or hormonal reasons. Wolf and Green (2002) concluded that the idiopathic FSS is an exhausting condition. DePalma (1963) wrote that aging is an important etiological factor of FSS. With aging, changes take place in the shoulder joint's connective tissue elements of the musculotendinous cuff, which cause it to lose its elasticity and undergo regressive changes.

Studies indicate that in patients with FSS, inflammatory factor immunoglobulin A was decreased and remained so after clinical recovery (Bulgen et al., 1978; Bulgen et al., 1984). However, it has been found in studies that several cytokines: interleukin 1 $\beta$ , tumour necrosis factor-alpha (Ko and Wang, 2011), interleukin 6, interleukin 8 were elevated in patients with FSS (Kabbabe et al., 2010). The levels of the fibrinogenic cytokine matrix metalloproteinase 3 (Hutchinson et al., 1998), disintegrin and metalloproteinase with thrombospondin motifs 4 were elevated in patients with FSS too (Kabbabe et al., 2010). In this patient group, there has been an increased level of growth factors such as transforming growth factor beta, platelet-derived growth factor, and fibroblast growth factor (Bunker et al., 2000; Mullett et al., 2007) which lead to the development of capsular fibrosis (Ozaki et al., 1989; Border and Noble, 1994; Rodeo et al., 1997; Mullett et al., 2007) and the clinical symptoms of FSS (Rodeo et al., 1997; Mullett et al., 2007). The imbalance between aggressive healing, scarring, contracture and a failure to remodel may lead to protracted stiffening of the capsule (Bunker et al., 2000). Bunker and Anthony (1995) described that the pathological process was active in the fibroblastic proliferation and the result of this process was that the fibroblasts lay down collagen, which appears as a thick nodular band or fleshy mass.

**Table 1.** Frozen shoulder syndrome stages (by Hannafin and Chiaia, 2000; Sheridan and Hannafin, 2006; Neviasser and Hannafin, 2010).

Stage	Duration of symptoms	Examination under anaesthesia	Arthroscopy	Pathology	Biopsy	Patients
I	0–3 months, pain with aROM and pROM, limitation. Pain referred to deltoid insertion, pain at night. FSS patients feel capsular pain on deep palpation, empty feel at extreme of motion	Normal or minimal loss of ROM	Diffuse hypervascular glenohumeral synovitis, often most pronounced in the anterosuperior capsule. Fibrinous synovial inflammatory reaction	Hypertrophic, hypervascular synovitis, rare inflammatory cell infiltrates, normal capsule	Rare inflammatory cell infiltrate, hypervascular, hypertrophic synovitis, normal capsular tissue	The pain is described as achy at rest and sharp with motion. Patients describe night pain and rest pain
II	3–9 months, severe night pain, and stiffness. Chronic pain feels with active and passive ROM, significant ROM limitation	Some motion loss under anaesthesia. The motion loss in stage II reflects a loss of capsular volume and response to painful synovitis	Christmas tree synovitis, some loss of axillary fold, tight capsule with rubbery or dense feel on the insertion of the arthroscope	Hypertrophic, hypervascular synovitis with perivascular and subsynovial scar formation, fibroplasias. No inflammatory infiltrates have been reported in stage II		Rest pain and night pain, significant sleep disturbance may exist
III	9–15 months, profound stiffness, pain only at the end ROM, significant limitation of ROM with rigid “end feel”, tethering at ends of motion	No improvement in the ROM feel under anaesthesia, fibrosis of the glenohumeral joint capsule	No hypervascularity, seen remnants of fibrotic synovium that is not hypervascular, diminished capsular volume, complete loss of axillary fold, minimal synovitis	"Burned out" synovitis without significant hypertrophy or hypervascularity, dense scar formation of the capsule	Capsular biopsy – hypercellular, collagenous tissue with a thin synovial layer, similar features to other fibrosing conditions	Minimal pain at night or rest (except at the ROM), significant shoulder stiffness
IV	15–24 months, profound stiffness, minimal pain, significant motion loss, progressive improvement in ROM		Fully mature adhesions, identification of intraarticular structures difficult			Minimal pain and progressive improvement in ROM resulting from capsular remodeling

In the immunological pathogenesis of this condition, reduction in the lymphocyte transformation to phytohaemagglutinin was found (Bulgen et al., 1978). On the other hand, C-reactive protein and concanavalin increased (Bulgen et al., 1982; Bulgen et al., 1984). Hand et al. (2007) discovered immunocyto-chemical evidence of both chronic inflammation and proliferative fibrosis. High vascularity (Nago et al., 2010) and nerve tissue, the inflammation and fibrosis (Nago et al., 2010) explain why FSS is such a painful and stiff condition. The presence of T and B cells suggest that the pathology may be immunomodulated. Mast cells may be the cellular mediator between chronic inflammation and fibrosis (Hand et al., 2007). Harzy et al. (2004) suggest there could be an immunological or genetic basis for primary hypoparathyroidism and FSS. Alternatively, FSS may be manifestation of hypoparathyroidism. The most well-known strong association is between diabetes and FSS (Smith et al., 2003). Bridgman (1972) found that in 800 diabetic patients, 86 had FSS (32 male with mean age of 58.3 yrs, 54 female with mean age of 59.8 yrs), however, in 600 nondiabetic group 14 have had FSS (10 male with mean age of 57.8 yrs and 4 female with mean age of 62.7 yrs). Moren-Hybbinette et al. (1987) found that in 90% of diabetic patients with painful and restricted shoulder mobility are affected in the activities of daily living (ADL) in the acute phase, whereas 17% of patients had restricted mobility at the end of the study.

Patten and Hillel (1993) proposed that FSS is a principal component of the cranial nerve XI syndrome that can significantly compound the morbidity of a neck dissection even when the accessory nerve recovers. Cakir et al. (2003) found in their study that the prevalence of FSS was highest in patients with subclinical thyrotoxicosis (17%).

Intraoperative findings in patients with FSS are extensive scarring of the inferior glenohumeral ligament, axillary recess, degenerative tearing of the superior labrum, where areas of the biceps and rotator cuff appear intact (Roubal and Placzek, 2008).

The histology showed fibrosis, hyalinization, and fibrinoid degeneration in the contracted connective tissues, fibrosis of the subsynovial tissue and an absence of the synovial cell layer on the joint side of the rotator interval (Ozaki et al., 1989). However, synovial proliferation and hypervascular changes are typical of the initial phases, while fibrosis is typical in later phases of FSS (Neviaser, 1987; Neviaser and Neviaser, 1987; Neviaser and Hannafin, 2010).

It has been found in studies that in patients with FSS significant bone loss in the humerus of the affected extremity occurs, but in the long term, induced bone loss shows good recovery in patients with FSS (Leppälä et al., 1998).

Uhthoff and Boileau (2007) concluded that in patients with FSS, fibroplasia and contracture are two distinct processes. Hagiwara et al. (2012) analyzed changes in the capsule and found that in patients with FSS the number of cells was higher and capsular tissue was stiffer. They concluded that chondrogenesis plays a critical role in the pathogenesis of idiopathic FSS.

In conclusion, the etiology of FSS is multifaceted, damage has been found in different shoulder joint structures and different processes have taken place. This

may explain why FSS is long-lasting, goes through many stages and is so displeasing for the patients.

## **2.4. Assessment of patients with frozen shoulder syndrome**

The doctors and physiotherapists have a lot of different methods for assessing the patients with FSS before, during and after therapy. FSS diagnosis is based on medical history, physical examination and clinical symptoms presentation. The examination of the patients with FSS includes observation, cervical examination, assessment of shoulder joint ROM, assessment of the shoulder region muscle strength and provocative testing (Pearsall and Speer, 1998). After that, the diagnosis FSS is commonly given when other causes can be excluded, such as major trauma, rotator cuff contusion, labral tear, bone contusion, subacromial bursitis, cervical or peripheral neuropathy, or history of a previous surgical procedure that may have led to shoulder stiffness. If there is no indication of the above pathologies and radiographs do not demonstrate osteoarthritis, the diagnosis of FSS can be given (Manske and Prohaska, 2010; Ahn et al., 2011). Binder et al. (1984) found on plain x-rays that in patients with FSS in the involved shoulder, the shoulder was normal in 30 of the 42 patients. Changes in the shoulder joint in patients with FSS were: decreased distance between the acromion and the humeral head, degenerative changes in the humeral head which involved greater tuberosity.

### **2.4.1. Arthrography and magnetic resonance imaging**

In patients with FSS, the arthrography shows a decrease in the joint capacity with obliteration of the reflected axillary fold, the subscapularis bursa is decreased in size or not visualized at all and, the bicipital sheath is well outlined (Neviaser, 1962; Siegel et al., 1999; Yilmaz et al., 2007). Binder et al. (1984) divided patients with FSS into three subgroups: (1) normal group – shoulder joint volume at least 15 ml, (2) capsulitis group – marked reduction in joint volume, loss of distensibility of the shoulder joint, marked irregularity of joint outline and early lymphatic filling, (3) rupture group – rupture of the rotator cuff.

Magnetic resonance (MR) arthrographic findings in patients with FSS included the thickening of the coracohumeral ligament (correlates with ROM limitations in INR and EXR (Lee et al., 2012) and thickening the joint capsule in the rotator cuff interval, and subcoracoid triangle sign (Ozaki et al., 1989, Lee et al., 2003; Mengiardi et al., 2004; Song et al., 2011; Li et al., 2011; Teixeira et al., 2012). In the magnetic resonance imaging (MRI), Ahn et al. (2012) found that the thickening and gadolinium improvement of the shoulder joint capsule in the axillary recess are associated with shoulder ROM limitation and shoulder pain in patients with FSS. It has been found in studies that rotator



interval dimensions in shoulders with FSS were significantly different compared with normal controls (Kim et al., 2009). It was concluded that the absence of a full-thickness rotator cuff tear, the thickness of a capsule and synovium greater than 3 mm at the level of the axillary recess is a practical MR criterion for diagnosing FSS on oblique coronal T2-weighted MR arthrography images without fat suppression (Jung et al., 2006). Sofka et al. (2008) evaluated noncontrast MRI findings in patients with FSS and correlated them with clinical stages. They concluded that specific MRI criteria correlated with the clinical stage of FSS, including the thickness and signal intensity of the shoulder joint capsule and synovium as well as the presence and severity of scarring in the rotator interval. In cases of patients suspected of having FSS, contrast-enhanced magnetic resonance imaging should be obtained (Gokalp et al., 2011).

In conclusion, understanding the anatomy and pathology of the shoulder joint, especially of the rotator interval, may be helpful for successful diagnosis and treatment of patients with FSS.

#### **2.4.2. Active and passive range of motion**

FSS is believed to be a self-limiting condition, lasting 18–30 months period (Wies, 2005; Baums et al., 2007; Tasto and Elias, 2007; Brue et al., 2007). On the other hand, studies report that 20–50% of patients suffer a long-term motion deficit that can at times last up to 10 years (Binder et al., 1984; Bulgen et al., 1984; Shaffer et al., 1992). Shaffer et al. (1992) evaluated sixty-two patients with FSS who had been treated nonoperatively. Subjective and objective follow-up lasts an average of seven years. 50% of these patients had either mild pain or stiffness of the shoulder, or both. 60% of patients demonstrated some restriction of shoulder ROM, and 11% reported mild functional limitation. However, Vastamäki et al. (2012) argued that 94% of patients with spontaneous FSS improved to normal shoulder function and ROM without treatment.

The shoulder aROM and pROM in patients with FSS is usually measured with a goniometer. The main treatment goal in patients with FSS is decreasing shoulder pain and increasing shoulder ROM. Different treatment modalities in patients with FSS are: nonoperative treatment (Lorbach et al., 2010), interventional microadhesiolysis (Ahn et al., 2008), MUA (Ng et al., 2009; Rill et al., 2011), arthroscopic capsular release (Cinar et al., 2010). With treatment, the fastest shoulder ROM recovery was usually achieved during the first 8 weeks. However, the recovery continued until the final follow-up. It was concluded that in the late stage of FSS, there was little functional impairment (Binder et al., 1984).

In FSS patients, using the assessment with the three-dimensional electromagnetic motion capture system, a significant difference was found in shoulder motion loss patterns between the involved and non-involved shoulder. The INR less than ABD less than EXR pattern was demonstrated in 56% involved shoulders. No pattern was presented in 67% non-involved shoulders. With the

arm ABD, INR was the most limited motion in 92% involved shoulders (Rundquist and Ludewig, 2004).

In conclusion, usually after treatment the patients with FSS showed improvement in the involved extremity pROM and aROM. The fastest improvement usually happened during the first eight weeks of therapy, after which the involved shoulder ROM continued to improve, but it was not so fast and depended on how much the patient was exercising.

### **2.4.3. Isometric and isokinetic strength**

Muscle atrophy in the rotator cuff and in the deltoid, biceps brachii, and triceps brachii muscles may occur in patients with FSS (Wadsworth, 1986). The muscle strength and endurance examination in patients with FSS may provide additional information about the patient's shoulder function. It has been found in studies that the high-speed shoulder muscle isometric strength during EXR and INR of the affected shoulder decreased significantly in patients with FSS (Lin et al., 2009). It was concluded that isokinetic dynamometry may provide additional information as compared with the usual outcome measures of shoulder pain and function level in patients with FSS (van Meetern et al., 2006). Liem et al. (2008) measured isometric and isokinetic shoulder muscle strength during INR and EXR in FSS patients who were treated by arthroscopic capsular release. They found that 53 months (range 12–106 months) after surgery, the isometric and isokinetic shoulder muscle strength in the standard ABD position showed no significant side-to-side difference during the INR and EXR.

Literature describes that the hand-held dynamometer has been the most reliable tool for assessing shoulder muscle strength of the rotator cuff in symptomatic subjects (Hayes et al., 2002; Bohannon, 2009). It was found that 13 weeks after MUA, the patients with FSS experienced mild weakness with manual muscle testing in 5.3% of patients during EXR and 10.5% during INR (Reichmister and Friedman, 1999). In patients with FSS, increasing muscle activity levels of the upper and lower trapezius in different testing positions occurred (120 deg FL, ABD in the frontal plane, and scapular plane, 60 deg FL, ABD in the frontal plane, and in the scapular plane). These indicate that patients with FSS compensated impaired glenohumeral motion using accessory musculature (Lin et al., 2005).

In conclusion, the condition of shoulder region muscles, especially rotator cuff muscles, is important in patients with FSS. The assessment of shoulder muscle strength and activation provide additional information about the patient's shoulder function before and after treatment.

### **2.4.4. Shoulder pain**

The shoulder pain associated with FSS is not only related to capsular and ligamentous tightness, but also to fascial restrictions, muscular tightness, and

trigger points within the muscles (Page and Labbe, 2010). In their long-term outcome study (4.4 yrs), Hand et al. (2008) demonstrated that 59% of patients had normal or near normal shoulders and 41% reported some ongoing symptoms, however shoulder pain being the most common complaint. FSS patients with severe symptoms at condition onset had the worst long-term prognosis. The patients with FSS usually report shoulder pain on palpation of the anterior and posterior capsule and pain that is experienced during aROM of the arm (Marx et al., 2007), and pain in the acromioclavicular joint (Anakwenze et al., 2011). It is very important to assess shoulder pain in patients with FSS before therapy, during the therapy process and after therapy.

Shoulder pain is usually assessed with a 10 or 100 point visual analog scale (VAS). When using the 10 point VAS scale for assessing shoulder pain, the average score before therapy reported in the studies has been 6–8 points; however, after treatment shoulder pain decreased to 0.9–3 points for average 13–16 weeks (Reichmister and Friedman, 1999; Watson et al., 2000; Griggs et al., 2000; Lee et al., 2009; Huang et al., 2010; De Carli et al., 2012). Gleyze et al. (2011) showed in their study that in patients with stiff shoulder in the conventional sub-threshold rehabilitation group and capsulotomy group, the shoulder pain decreased progressively during the first eight weeks.

Carbone et al. (2010) evaluated the coracoid pain test in patients with FSS and concluded that the digital pressure over the coracoideus area elicits pain in the vast majority of patients with FSS and, it can be considered an easy reliable clinical test for identifying patients with or without this condition.

#### **2.4.5. Self-administered questionnaires**

In the literature self-administered questionnaires for assessing patients with FSS are widely used, which include the global assessment, shoulder pain, ADL, recreational activities, athletic activities, work satisfaction and areas for improvement. For shoulder pathologies, more than 40 assessment tools are available (Fayad et al., 2005). To assess FSS patients' recovery, researches used consecutive questionnaires: Shoulder Rating Questionnaire (SRQ) (L'Insalata 1997; Vad et al., 2003; Diwan and Murrell, 2005; Vermeulen et al., 2006; Marx et al., 2007); Shoulder Disability Questionnaire (Ryans et al., 2005; Vermeulen et al., 2006; Kivimäki et al., 2007); Shoulder Pain and Disability Index (Boyles et al., 2005). Williams et al. (1995) found that Shoulder Pain and Disability Index is responsive to change and accurately discriminates between patients whose condition improves or worsenes; Oxford shoulder score (Hand et al., 2008); Disabilities of the Arm, Shoulder, and Hand Questionnaire (Griggs et al., 2000; Wolf and Green, 2002; Bron et al., 2007); General health status was measured by the 36-Item Short-Form Health Survey (Griggs et al., 2000; Wolf and Green, 2002; Vermeulen et al., 2006; Baums et al., 2007; Levine et al., 2007; Jacobs et al., 2009); American Shoulder and Elbow Surgeons score (Omari and Bunker, 2001; Nicholson, 2003; Farrell et al., 2005; Baums et al., 2007; Levine et al., 2007; Leung and Cheing, 2008; Rill et al., 2011; De Carli et al., 2012); Simple

Shoulder Test (Wolf and Green, 2002; Nicholson, 2003; Castellarin et al., 2004; Farrell et al., 2005; Baums et al., 2007; Levine et al., 2007; Rill et al., 2011; De Carli et al., 2012); Constant score (CS) (Andresen et al., 1998; Dodenhoff et al., 2000; Othman and Taylor, 2002; Jerosch, 2001; Massoud et al., 2002; Diercks and Stevens, 2004; Quraishi et al., 2007; Wang et al., 2007; Jacobs et al., 2009; Wang et al., 2010; Celik 2010); Japanese Orthopaedic Association assessment (Ide and Takagi, 2004); Constant-Murley scale (Castellarin et al., 2004; Cheing et al., 2008; De Carli et al., 2012). University of Los Angeles shoulder scores (De Carli et al., 2012).

Dupeyron et al. (2010) investigated the validity, reliability, and responsiveness to change of The Standardized Index of Shoulder Function and found that it is a proper assessment tool for pain, mobility, strength and function in shoulder disorders, easy to administer and of good metric value. However, Buchbinder et al. (2007) concluded that of the general questionnaires on the quality of life, the 36-Item Short-Form Health Survey and Assessment of Quality of Life is not useful for measuring in clinical trials of interventions for FSS.

In conclusion, the point score of the evaluation scales significantly improved after the therapy sessions in patients with FSS. However, there is a difference between subjective (questionnaires) and objective (measured by a physiotherapist) assessment results of recovery. For the treatment process evaluation it is important to objectively and subjectively assess patients with FSS.

## **2.5. Treatment of patients with frozen shoulder syndrome**

FSS affects the patient's shoulder, which is treated by different medical specialists including general practitioners, orthopaedic surgeons, rheumatologists, and physiatrists and physiotherapists (Zuckerman et al., 2011). The primary goal of therapeutic intervention is to restore shoulder pain-free functional ROM. The treatment modalities are divided into conservative, nonsurgical and surgical interventions (Chamblor and Carr, 2003). Surgical release should only be considered after failed nonoperative treatment.

### **2.5.1. Three-step algorithm for managing the stiff shoulder**

Three-step stiff shoulder management algorithm proposed by Gleyze et al. (2011) after a complete clinical and radiological work-up:

– First step (3 months of intensive self-rehabilitation):

Checked and supervised treatment with a physiotherapist that stimulates and relieves the patient and progressively introduces conventional rehabilitation exercises.

– Second step (3 months to 6 months):

As progression is favourable, supervised self-rehabilitation should be continued. If progression is unfavourable and it is certain that the patient is doing his best, performing distension or locoregional anaesthesia with intensified conventional rehabilitation may be warranted. When progression is unfavourable and there is doubt as to the patient's willingness to work, rehabilitation should continue, but without proposing additional intervention.

– Third step (6 months):

When progression is favourable, the shoulder is considered to be normal and care is terminated. If progression is unfavourable and there is certainty that the patient is doing his best, a capsulotomy can be proposed. When progression is unfavourable and there is doubt concerning the patient's work or willingness, a clinical and radiological work-up must be done to look for a hidden problem (a usual patient profile, a physiotherapist "limiting" recovery but also an undetected organic disorder, etc.).

### **2.5.2. Conservative treatment methods**

After the diagnosis, the FSS patients started with intensive conservative treatment (CT), which consists of many different strategies. Conservative treatment methods are: different drugs (oral or injection in the shoulder joint) used for shoulder pain and inflammation relief; patient education, physiotherapy, different manipulation techniques, massage, and electrotherapy for shoulder pain relief and shoulder function improvement (Kelley et al., 2009).

Levine et al. (2007) followed the treatment and recovery of 105 FSS patients. 89.5% of the patients resolved symptoms with nonoperative treatment, including 17 of 19 diabetic shoulders. All patients received nonsteroidal anti-inflammatory medication, 52.4% received a standardized physiotherapy program without cortisone injection, and 37.1% received therapy with at least 1 corticosteroid injection. The duration of treatment in successfully nonoperatively treated patients averaged 4 months. Patients who needed surgery were treated with an average of 12 months of nonoperative treatment.

#### *Drugs*

There are several studies assessing the efficacy of different drugs (Bulgen et al., 1984; Ryans et al., 2005; Bal et al., 2008; Lorbach et al., 2010; Blanchard et al., 2010; Griesser et al., 2011) in the treatment of patients with FSS. Binder et al. (1986) followed patients with FSS for eight months: one group used oral prednisolone and home pendular exercises, while the other group performed home pendular exercises. The improvement in the treated group occurred more rapidly, however at the end of the study there was no difference between groups and the patients experienced significant restriction during shoulder ROM for the involved extremity as compared with normal controls. Blokcey and Wright (1954), and Tashijan (2012) found that the use of cortisone in the treatment of

patients with FSS leads to fast shoulder pain relief and improvement of shoulder ROM. The same recovery tendencies were found by Roh et al. (2011) in diabetic patients with FSS. The diclofenac epolamine (2-hydroxyethyl-pyrrolidine DHEP) lecithin gel is a topically effective analgesic product in the treatment of patients with FSS (Spacca et al., 2005). Rhind et al. (1982) concluded that naproxen and indomethacin were equally effective in the treatment of shoulder pain in patients with FSS but did little to change the loss of shoulder movement associated with this disorder. Buchbinder et al. (2004) have demonstrated that a short course of prednisolone for the treatment of patients with FSS is highly effective in the short term. Takase (2010) investigated the possible early symptom relief with oral steroids in patients with FSS and found that the treatment was effective.

### *Injection*

Corticosteroid injections of glenohumeral and subacromial space are the usual care for patients with FSS. Jacobs et al. (2009) recommended the use of corticosteroid injections, rather than MUA and physiotherapy, as a first-line treatment for patients in the “freezing” phase. An intra-articular glucocorticoid injection showed better results in objective shoulder scores, during shoulder ROM, and in patients’ satisfaction compared with a short course of oral corticosteroids (Lorbach et al., 2010). Intra-articular corticosteroid injection (only one) in FSS stage I recovered the involved shoulder more rapidly (2 weeks to 3 months) than in those who were in stage II (2 week to 2 years). However, early injection of corticosteroid and local anaesthesia are diagnostic and therapeutic (Marx et al., 2007) and the combination of the corticosteroid injection and therapeutic exercises is effective (Bulgen et al., 1984; Carette et al., 2003; Bal et al., 2008). There is no difference between ultrasound (US) guided subacromial injection with high-dose triamcinolone acetonide (40 mg) and low-dose (20 mg), whereas in the initial stage a low dose is recommended (Hong et al., 2011). However, de Jong et al. (1998) showed that greater symptom relief was achieved with a 40 mg dose of intra-articular triamcinolone acetonide injection than with 10 mg, the effect on shoulder pain and sleep disturbance was more evident than during shoulder ROM. Oh et al. (2011) found that steroid injection into the glenohumeral joint or subacromial space, followed by stretching exercises and non-steroidal anti-inflammatory drugs (NSAID) supplementation is an alternative modality in the treatment of patients with FSS. Roh et al. (2011) compared corticosteroid injections and home stretching exercises with home exercises in diabetic patients with FSS. They found that shoulder pain relief and shoulder function recovery was better and faster in the corticosteroid injection group, while in the long-term there were no differences between groups.

Fluoroscopic-guided intra-articular injection series is an effective and safe treatment option in the treatment of patients with FSS and leads to fast shoulder pain reduction and an increase in shoulder ROM (Richardson, 1975; Tveita et al., 2008; Lee et al., 2009; Lorbach et al., 2010). Arslan and Celicer (2001) investigated (a) local steroid injection of 40 mg methylprednisolone acetate

1 ml with 1 ml of 2% lidocaine group and (b) physiotherapy and NSAID (ace-methazine 120 mg/day) group. Physiotherapy consisted of hot pack application for 20 min, US therapy at 3.5 W/cm<sup>2</sup> for 5 min and passive glenohumeral joint stretching exercises to the patients' tolerance, followed by Codman's exercises and wall climbing. The study results showed that intra-articular corticosteroid injection was as effective as combined physiotherapy and NSAID. Jancovic and van Zundert (2006) demonstrated in five patients who did not respond to routine therapy that the selective subscapularis fossa nerve block combined with subscapularis trigger point infiltration have had diagnostic and therapeutic value in the treatment of patients with FSS. There are several possible pathways, involving anti-inflammatory and anti-fibrotic mechanisms. The analgesic effect can be expected to last longer than the effect of steroid injections. Chen et al. (2011) hypothesized that intra-articular injection of botulinum toxin can be an effective alternative for the treatment of FSS. They concluded that botulinum toxin intra-articular injection in the shoulder joint is a potential treatment for patients with FSS. Harris et al. (2011) showed in their systematic review that sodium hyaluronate injection into the glenohumeral joint significantly improves shoulder ROM, CS, and shoulder pain at short-term follow-up following the treatment of patients with FSS. On the other hand, Hsieh et al. (2012) found that intra-articular hyaluronic acid injections did not produce added benefits in patients with FSS who were already receiving physiotherapy.

Schydrowsky et al. (2012) found that tumour necrosis factor-alpha blockade with subcutaneous adalimumab compared with intraarticular steroid injections had no effect in patients with FSS. Ahn et al. (2008) developed a new effective non-surgical intervention for FSS treatment: they used three specially made needles in interventional microadhesiolysis.

### *Physiotherapy*

The physiotherapist plays an important role in: composing and supervising the exercise program, explaining the time course of the disappearance of symptoms, patient education and encouraging a program of home exercises. Winters et al. (1997) found that in patients with shoulder girdle disorders, who were treated with manipulation or physiotherapy, the duration of symptoms was shorter after manipulation. Rizk et al. (1983) followed two different treatment programs. The first group was treated with therapeutic heating modalities followed by therapeutic exercises and gentle rhythmic stabilization manipulation, while the second group was treated with prolonged pulley traction and transcutaneous electrical nerve stimulation (TENS). The improvement was greater in the second group. Griggs et al. (2000) found that in stage II, patients with FSS should be treated with a four-direction shoulder-stretching exercise program that includes passive forward elevation, passive EXR, passive INR and passive horizontal ADD. This treatment should be continued for at least three months before more aggressive or invasive management is considered.

Gaspar and Willis (2009) achieved the best treatment results with shoulder Dynaslant and standardized physiotherapy. It was concluded that end range

mobilization treatment (three weeks) significantly improved shoulder aROM during ABD (Sarkari et al., 2006; Dempsey et al., 2011). In combination with a simple home exercise program, adding supervised physiotherapy, the improvement of shoulder ROM was faster, while supervised physiotherapy alone had limited efficacy (Carette et al., 2003). In a single-case study, Maricar et al. (2009) found that exercises only and exercises with Maitland mobilization were beneficial in the treatment of the patient with FSS in stage III.

It was suggested that a multimodal nonoperative treatment program (supervised physiotherapy, home exercises, oral NSAID or corticosteroids, corticosteroid injections) is effective for most patients with FSS (Rill et al., 2011). Yang et al. (2011) examined the effectiveness of the end-range mobilization/scapular mobilization treatment approach in FSS patients. They found that the end-range mobilization/scapular mobilization treatment approach was more effective than a standardized physiotherapy program. In a study by Boyles et al. (2005) the physiotherapist performed manipulation with low-amplitude procedures after a regional interscalene block was performed by an anaesthesiologist. Patients were instructed to perform shoulder aROM exercises every two hours when awake at home, for the next 24 hours and apply ice packs on the shoulder for 20 min. The patients had received physiotherapy every day for one week, after that three times per week, and after three weeks, the patients continued with the home program. The patients showed rapid improvement during shoulder pROM and an improved level of disability measured by the Shoulder Pain and Disability Index. Johanson et al. (2007) compared the effectiveness of anterior versus posterior glide mobilization techniques for improving shoulder ROM during EXR in patients with FSS, and found that the posteriorly directed joint mobilization technique was more effective.

It has been found that high-grade mobilization techniques were more effective in improving glenohumeral joint mobility and reducing disability than low-grade mobilization techniques (van den Hout 2005; Vermeulen et al., 2006). O’Kane et al. (1999) followed patients with FSS who performed a simple home program. They concluded that this program can lead to improvement in self-assessed shoulder function health status, 56% of patients were able to place a 3.6 kg weight on a shelf, and 66% were able to carry 9.1 kg at their side. Diercks and Stevens (2004) found that “supervised neglect” of idiopathic FSS is superior to passive stretching and mobilization with regard to the functional end results and the speed of recovery. In the van der Windt et al. (1998) study, patients with FSS received no more than three intraarticular injections of 40 mg triamcinolone acetonide (injection group), during six weeks. There were twelve sessions of physiotherapy with 30 min duration, during which all patients received passive joint mobilization and exercise treatment. Ice, hot packs, or electrotherapy were used to reduce shoulder pain. The study results showed that the corticosteroid injections group experienced quicker symptom relief as compared to the physiotherapy group. The study results of Ryans et al. (2005) were similar. However, they found that physiotherapy is effective in improving shoulder ROM during EXR 6 weeks after treatment. Bulgen et al. (1984)



followed forty-two patients with FSS for eight months. The treatment methods in this study were: (a) intraarticular steroids, (b) mobilization, (c) ice therapy and (d) no treatment group. They found that all treatments had a positive effect but the steroid injections may benefit shoulder pain and shoulder ROM in the early stages of the disease. FSS patients treated with physiotherapy (mobilization), steroid injection, or with a combination had less shoulder pain and showed improvement at six weeks and six months (Dacre et al., 1989). Ulusoy et al. (2011) found that supervised physiotherapy supplemented with NSAIDs improved shoulder ROM values in most FSS patients.

In conclusion, there are many different treatment techniques used in the treatment of patients with FSS. However, in the opinion of the author of this thesis, the combination of orthopaedic massage (manipulation) and shoulder ROM exercises is more effective than shoulder ROM exercises alone.

### *Therapeutic modalities*

In FSS patients, the treatment used following therapeutic modalities: different electrotherapy currents, cold and hot packs, US, and magnetotherapy. Leclaire and Bourgouin (1991) treated patients with FSS three times a week for a maximum of three months with: (a) hot pack application and passive manual stretching and pulley exercises, (b) hot pack application and passive manual stretching and pulley exercises and magnetotherapy. They concluded that magnetotherapy had no benefit in the treatment of patients with FSS. It was concluded that electroacupuncture or interferential electrotherapy in combination with shoulder exercises is effective in treating FSS patients (Leung and Cheing, 2008). Deep heating and stretching exercises produced greater improvement in shoulder pain relief, and resulted in better performance in ADL and shoulder ROM (Leung and Cheing, 2008). Hamer and Kirk (1976) found that ice and US with specific exercises shortened the painful stage and fastened the recovery of FSS. It has been found in studies that TENS, cold packs, NSAID and scapulothoracic exercises decreased shoulder pain and increased shoulder ROM more than glenohumeral ROM exercises alone (Celic, 2010). Carette et al. (2003) used different treatment programs. In the acute stage, the physiotherapy program consisted of TENS, mobilization techniques, shoulder aROM exercises, ice application. In the chronic stage, they used US, mobilization techniques, active and auto-assisted shoulder ROM exercises, shoulder muscle isometric strengthening exercises, ice application. There were four different groups: (a) corticosteroid injection and 12 sessions of physiotherapy, (b) corticosteroid injection, (c) saline injection, followed by supervised physiotherapy and (d) saline injection (placebo). The fastest symptom relief and recovery was attained in the corticosteroid injection and physiotherapy group. In long-term follow-up the differences between groups disappeared. In their case study, Gulick et al. (2007) reported the positive effect of combined therapy that consisted of twelve treatments of moist heat, analgesic nerve block electrical stimulation; contract/relax exercises for shoulder INR and EXR, and Codman's Pendulum exercises.

In conclusion, the therapeutic modalities fasten shoulder pain relief and patients with FSS are able use their involved extremity in ADL and exercise performance without shoulder pain.

### **2.5.3. Treatment possibilities after failed conservative treatment**

When conservative treatment in FSS patients does not had produce a positive effect after two or three months of treatment, the next choice is non-surgical and surgical intervention (Chambler and Carr, 2003; Kelley et al., 2009). The choices are: MUA (Jenkins et al., 2012), arthroscopic release (Kelley et al., 2009), open capsular release (Omari and Bunker, 2001), pulsed mode radio-frequency lesioning of the suprascapular nerve (Huang et al., 2010), sonoguided capsular distension (Park et al., 2012), arthrographic distension (Quaraisi et al., 2007). The surgical release is contraindicated if the patient is in clinically significant depression or suffers from autonomic dystrophy. Arthroscopic capsular release is absolutely contraindicated in patients who cannot tolerate the surgical stress of fluid challenge (e.g. renal or cardiac failure) (Ko et al., 2011).

#### *Manipulation under general anaesthesia*

MUA is performed either under general or local brachial plexus block, which completely relaxes the shoulder muscles, ensuring that the force applied by the surgeon reaches the capsuloligamentous structures (Kelley et al., 2009; Wang et al., 2010; Jenkins et al., 2012).

In their study, Jenkins et al. (2012) described MUA as follows: “General anesthetic was administered with the patient supine. Once the patient was fully anesthetized, the patient’s scapula was fixed by gripping across the top of the shoulder, and the pROM of the glenohumeral joint was assessed and recorded in comparison with the anatomic position. INR and EXR were assessed and recorded by flexing the elbow to 90 deg before using the forearm as a pointer. Manipulation was then initiated by holding the patient’s arm between the shoulder and elbow and manipulating it initially into ABD, then FL, EXR, cross-body ADD, and finally, INR. This sequence was repeated until the maximum possible shoulder ROM was achieved and again recorded”.

MUA is a simple, safe and noninvasive procedure to improve symptoms in a short period of time with early physiotherapy and pool exercises (Ng et al., 2009; Jenkins et al., 2012) even in diabetes patients (Placzek et al., 1998; Dodenhoff et al., 2000; Kivimäki and Pohjolainen, 2001; Othman and Taylor, 2002; Massoud et al., 2002; Farrell et al., 2005; Wang et al., 2007; Wang et al., 2010; Thomas et al., 2011; Jenkins et al., 2012). Early MUA (less than 9 months from the onset of symptoms) had better recovery results (Flannery et al., 2007). In the study with 246 FSS patients treated with MUA, 47 shoulders required a further MUA and three required a third manipulation (Thomas et al., 2011). Reichmister and Friedman (1999) found that 8% of patients needed the second MUA to achieve good results, while Jenkins et al. (2012) reported that

36% diabetic and 15% nondiabetic patients with FSS needed repeated MUA during a 10-year study. Manipulation followed by arthroscopic release and rehabilitative treatment is an effective way of shortening the course of FSS (Andresen et al., 1998; Castellarin et al., 2004). Roubal et al. (1996) found that MUA following the interscalene brachial plexus blocks improved shoulder ROM during FL, ABD, EXR and INR. It increased function in patients, such as overhead activities, dressing activities, and hair combing. Several serious complications during the MUA procedure have been reported in studies: fracture of the humerus, dislocation of the glenohumeral joint, isolated paralysis of infraspinatus muscle and other brachial plexus traction injuries, rotator cuff tear, hemorrhagic effusions and hematomas (Takagishi et al., 1994; Dodenhoff et al., 2000). Reichmister and Frideman (1999) followed 38 patients for an average time of 58 months after MUA; they had no evidence of biceps tendon rupture or rotator cuff insufficiency, fractures, dislocations or nerve palsies in their study. On the other hand, it was found that MUA does not add effectiveness to an exercise program carried out by the patient after instruction (Kivimäki et al., 2007). It was demonstrated that translational manipulation following an interscalene block, caused rapid improvement in shoulder pROM and improved levels of disability as measured with the Shoulder Pain and Disability Index (Boyles et al., 2005).

#### *Other choices*

Arthroscopic capsular release is an effective and safe alternative to manipulation in patients with FSS (Pearsall et al., 1999; Watson et al., 2000; Jerosch, 2001; Diwan and Murrell, 2005; Baums et al., 2007; Cinar et al., 2010). It has been shown in studies that arthroscopic release is effective after failed conservative treatment, which lasted at least six weeks without progress with symptoms for at least three months (Ide and Takagi, 2004). Baums et al. (2007) demonstrated that arthroscopic release in patients with FSS combined with gentle manipulation provides reliable expectations for improvement in both clinical and general health status for most patients. De Carli et al. (2012) compared the use of manipulation and arthroscopic arthrolysis with glenohumeral steroid injections in patients with FSS and found that both of these treatments seemed to be effective in restoring a satisfactory shoulder ROM and decreasing painful symptomatology in the shoulder. Ogilvie-Harris et al. (1995) followed patients with FSS for 2–5 years. One year after failed conservative treatment, 40 patients were divided into two groups: one group of patients was treated with manipulation and arthroscopy and the other group with arthroscopy. They found that 15 of 20 patients treated with arthroscopy had excellent results compared with 7 of 18 patients treated with arthroscopy and manipulation. Selective arthroscopic capsular release was suggested to patients with FSS who did not respond positively to CT, and patients with diabetes may benefit from early intervention (Pollock et al., 1994; Ogilvie-Harris et al., 1995; Segmüller et al., 1995; Ogilvie-Harris and Myerthall, 1997; Pearsall and Speer, 1998) and postoperative exercises and physiotherapy are also important for excellent

recovery (Sabat and Kumar, 2008). Huang et al. (2010) treated two patients with FSS by using real-time, high resolution US guidance to facilitate pulsed mode radiofrequency lesioning of the suprascapular nerve. Both patients experienced shoulder pain relief and increased shoulder flexibility for 5–6 months.

Open surgical release has provided good results in patients with primary FSS who fail to improve with either conservative treatment or MUA (Omari and Bunker, 2001). It has been concluded that sono-guided capsular distension is an advantageous technique for treating FSS from the viewpoint of radiation hazard mitigation, time, cost-effectiveness and convenience (Park et al., 2012). Literature recommends arthrographic distension (Sharma et al., 1993; van Royen and Pavlov 1996; Fareed and Gallivan, 1998; Quaraishi et al., 2007) followed by intraarticular steroid injection and high-intensity physiotherapy for improving shoulder pain and shoulder pROM in patients with FSS within the first five days, and retaining the improved results after one month (Laroche et al., 1998; Buchbinder et al., 2007). MUA is a more costly inpatient procedure, whereas arthrographic distension can be carried out as an outpatient procedure without general anaesthesia and with less attendant risks in the treatment of patients with FSS (Gavant et al., 1994; Fareed and Gallivan, 1998; Gam et al., 1998; Vad et al., 2003; Jacobs et al., 2009). On the other hand, Tveita et al. (2008) did not find important treatment effects when comparing three arthrographic distensions that included steroids with three steroid injections alone. However, after analyzing 11 systematic reviews of the evidence of the effectiveness of interventions used to manage primary FSS, Rookmoneea et al. (2010) found that there is a demand for standardization of diagnostic criteria, standardization of outcome measurement and improvement of the quality of randomized controlled trials in the studies of patients with FSS.

In conclusion, the correct diagnosis and intervention choice and active physiotherapy thereafter are important factors for achieving good and fast treatment results.

### **3. AIMS OF THE STUDY**

The general aim of the present study was to evaluate the recovery of shoulder function in patients with FSS following conservative treatment and after MUA.

The specific objectives were:

- (1) To evaluate the changes in shoulder aROM, shoulder muscle isometric strength and endurance in patients with FSS before and one month after conservative treatment (Papers I–II).
- (2) To assess the changes in shoulder aROM, shoulder muscle isometric strength and endurance in patients with FSS before, one month and six months after MUA (Papers III–IV).
- (3) To assess the pain and the functional limitations and disability of the shoulder in patients with FSS before and after conservative treatment and MUA (Papers I–IV).

## 4. MATERIALS AND METHODS

### 4.1. Subjects

#### 4.1.1. Description of the subjects

Twenty-five patients with FSS aged 18–74 years and ten age- and gender matched healthy people as controls participated in this study. Table 2 demonstrates mean age and anthropometric characteristics of FSS patients and controls in the different studies.

**Table 2.** Age and anthropometric characteristics of the subject groups (mean±SE).

Papers	N	Age (years)	Height (cm)	BM (kg)	BMI (kg·m <sup>-2</sup> )
<b>Papers I–II</b>					
Patients	10	50.2±4.6	168.7±2.8	72.7±3.8	25.6±1.0
Controls	10	49.0±4.6	167.3±2.7	74.8±3.5	25.9±0.9
<b>Papers III–IV</b>					
Patients	15	53.6±9.7	167.1±9.1	71.9±11.2	25.0±4.0

BM = body mass; BMI = body mass index.

In the first study (2002–2004), the participants were ten patients with FSS (7 women and 3 men), who were treated conservatively (CT group) (Thomas et al., 2011) and ten subjects with asymptomatic shoulders (7 women and 3 men) as controls (Papers I–II).

In patients with FSS, shoulder pain and function limitation lasted 2 to 9 months before the CT. The subjects were moderately physically active; however, no professional athletes were included. They had no orthopaedic or neurological limitations or contraindications for exercise testing or training. The final data analysis was conducted on all 20 subjects.

In the second study (2006–2008), the participants were eighteen patients with FSS (10 women and 8 men), who were treated with MUA in combination with physiotherapy (MUA group) (Papers III–IV). Subjects' inclusion criteria were: unilateral FSS defined as >50% loss of pROM of the shoulder joint relative to the non-affected side in 1 or more of 3 movement directions (i.e. ABD, FL, or EXR) (Diercks, 2004), shoulder pain at rest, inability to sleep on the affected side. Exclusion criteria were: previous MUA of the affected shoulder; other conditions involving the shoulder (rheumatoid arthritis, osteoarthritis, damage of the glenohumeral cartilage, Hill-Sachs lesion, osteoporosis or malignancies in the shoulder and chest region), traumatic bone or tendon changes in the affected shoulder; neurologic deficits affecting shoulder function in ADL; shoulder pain or disorders of the cervical spine, elbow, wrist, or hand; and an injection with corticosteroids in the affected shoulder within 4 weeks.

Patients who have had serious cardiac problems or cardiac surgery were excluded from this study (Vermeulen et al., 2006).

The average duration from the onset of the disease to MUA was 8.6 months, ranging from 3 to 12 months. The FSS stage was II or III. The dominant shoulder was involved in 7 patients and the nondominant one in 11 patients. Three subjects did not return after the first session and were not included in data analysis. One subject broke the other hand one month after MUA. Two patients did not come back after the first session. The final data analysis was therefore conducted on 15 subjects. The subjects were moderately physically active; however, no professional athletes were included. They had no orthopaedic or neurological limitations or contraindications for exercise testing or training. All patients had physiotherapy about 7 times before MUA.

The studies were conducted in the Laboratory of Kinesiology and Biomechanics at the University of Tartu. The subjects were recruited by orthopaedic surgeons in Tartu University Hospital, in the Department of Orthopaedics and Traumatology.

The study was carried out with the approval of the Ethics Committee of Human Research of the University of Tartu.

#### **4.1.2. Treatment**

*Conservative treatment* (Papers I–II). In the CT, a four-week individualized physiotherapy program was used for all FSS patients, which consisted of 10 individualized exercise therapy procedures in a gymnasium and a swimming pool with the duration of 30 min/day. Patients performed exercises for improving shoulder joint aROM and shoulder muscle strength. In the 5–10 massage procedures work was performed on the stiff and painful shoulder muscles. The duration of 5–10 electrical therapy procedures was 5–10 min/day (interferential current, TENS) for pain relief in the shoulder region.

*Manipulation under general anaesthesia* (Papers III–IV). The manipulation was done under general intravenous barbiturate anaesthesia for all patients. The following technique was used for manipulation: (a) gradual FL in the sagittal plane to maximum possible extent while the surgeon's assistant fixed the scapula; (b) passive EXR was performed on 0 deg of ABD; (c) EXR in 90 deg of ABD; (d) INR at 90 deg of ABD and gross-body ADD were performed. Care was taken not to fracture the humerus during manipulation. EXR force was very carefully applied when the patient's elbow was fixed and the wrist moved at the same time by the surgeon's thumb and two opposing fingers. A full shoulder ROM was always achieved. The shoulder joint was injected with 19 ml of 1% Lidocaine and 1 ml of corticosteroid in a 20 ml syringe immediately after manipulation.

All patients received immediate passive exercise in the ward soon after MUA. They underwent gentle active-assisted motion with a physiotherapist after MUA. Motion was practiced in FL, extension (EXT), ABD, ADD, INR and EXR. Physiotherapy continued on an outpatient basis, and included super-

vised and therapeutic home exercise programs focused on shoulder muscle stretching (two times per day, five days per week). Further physiotherapy procedures included shoulder muscle isometric strengthening exercises followed by the use of elastic bands and power simulator as soon as post-MUA shoulder pain and shoulder aROM allowed. The subjects were treated by physiotherapists with at least 2 years of clinical experience. The subjects had ten physiotherapy sessions during one month, three times per week. They were advised to use the affected shoulder in ADL whenever possible.

Demographic data, including age, sex, employment status, and sports and leisure activities, were recorded at baseline. A history was taken concerning the duration of complaints (months), previous treatments (injections, physiotherapy), and current pain medication. Concomitant diseases and the use of medication were registered.

## **4.2. Study design**

The subjects were instructed and the shoulder muscle strength and isometric endurance testing procedures were demonstrated 24–48 hours before collecting the first data. This was followed by a practical session to familiarize the subjects with procedures. Before testing, each subject underwent a 10 min warm-up consisting of gymnastics and stretching exercises. Both extremities were tested, whereas the uninvolved extremity was tested first. The data collection was performed in the first study before and one month after individualized CT and in the second study one day before MUA, and one and six months after MUA.

In paper I, changes in shoulder joint aROM, shoulder muscle isometric strength, shoulder muscle isometric endurance, and shoulder pain in patients with FSS and controls were assessed before and one month after individualized CT.

In paper II, changes in shoulder muscle isometric working capacity and fatigability of the deltoideus, infraspinatus and trapezius muscles in patients with FSS and controls were evaluated before and one month after individualized CT.

Paper III followed changes in shoulder muscle isometric strength, shoulder aROM, shoulder muscle isometric endurance, and self-administered SRQ in patients with frozen shoulder before, one and six months after MUA.

Paper IV described changes in shoulder aROM, shoulder muscle isometric strength, and shoulder pain at day and by night in patients with FSS before, one and six months after MUA.



## **4.3. Methods**

### **4.3.1. Measurement of shoulder active range of motion**

The shoulder aROM during FL, EXT, ABD and ADD was measured by gravitational goniometer Bubble Inclinometer (Fabrication Enterprises Inc., USA) and shoulder aROM during INR and EXR was measured by gravitational goniometer Myrin (Follo A/S, Norway) (Papers I–IV). The subjects were positioned standing for all shoulder ROM tests according to standard guidelines (Clarkson, 2005). All measurements were rounded as is common in research practice off to the nearest 5 degrees (Vermeulen et al., 2000). All assessments were performed by the same physiotherapist.

### **4.3.2. Shoulder muscle isometric strength testing**

Shoulder muscle maximal isometric strength during FL, EXT, ABD, ADD, INR and EXR was measured by a hand-held dynamometer (Lafayette Manual Muscle Test System, Lafayette Instrument Company, USA) (Papers I–IV). Hand-held dynamometry is considered an objective method of measuring muscle strength (Bohannon and Andrews, 1987; Hayes et al., 2001; 2002). It has been indicated that the intraclass correlation coefficients were high ranging from 0.971 to 0.972 for the test-retest trials, while a hand-held dynamometer with a stabilization device was used for testing shoulder muscle isometric strength (Kolber et al., 2007).

During shoulder muscle strength testing, the subject was in a seated position on a standard chair. Shoulder muscle strength assessment during FL, EXT, ABD and ADD was performed with the full ended upper extremity positioned with the shoulder abducted to 45 deg. A hand-held dynamometer was placed laterally on the distal end of the humerus approximately 5 cm superior to the elbow joint. Shoulder muscle isometric strength assessment during INR and EXR was performed with the shoulder in a vertical position and the elbow flexed to 90 deg. The hand-held dynamometer was placed laterally (external) or medially (internal) on the distal part of the elbow approximately 5 cm superior to the wrist. The forearm was pronated during all strength tests. The position was carefully supervised by an experimenter and the subjects were encouraged to act in the required way. The subjects were asked to exert maximum voluntary isometric contractions pushing against the dynamometer for approximately 3 s. Before each contraction, the subjects were instructed to “push as hard as possible”. The best results from 3 attempts were recorded as isometric maximal voluntary strength. A rest period of 1 min was allowed between attempts. All shoulder muscle strength assessments were performed by the same physiotherapist.

### 4.3.3. Shoulder muscle endurance testing and electromyography

Shoulder girdle muscle isometric endurance was evaluated by weight holding time. During endurance testing, the subject was in a seated position on a standard chair. The full extended upper extremity was positioned with the shoulder flexed 45 deg and abducted 45 deg. In this position, the subject held weight in hand (30% of shoulder muscle maximal isometric strength assessed by a hand-held dynamometer – Papers I–II and a weight of 5 kg for men and 3 kg for women, – Paper IV) as long as possible (Fig. 1).

Isometric working capacity of shoulder muscles was characterized by net impulse (NI), which was calculated by formula (Papers I–II):

$$NI = P \cdot t \text{ (N}\cdot\text{s)},$$

where P is hand-held weight x 9.81, and t is endurance time.

During the isometric endurance test, electromyographic (EMG) activity of the deltoideus, infraspinatus and trapezius muscles (Paper II) was continuously recorded using the standard electromyograph Medicor MG 440 (Hungary). Paired bipolar surface electrodes (Ag-AgCl, 8 mm diameter, 20 mm inter-electrode distance) were used. The skin under the electrode was shaved, abraded and cleaned with alcohol, and a conducting gel was applied to obtain a good signal transfer from the skin to the electrodes. The electrodes over the infraspinatus muscle were placed 3 to 4 cm below the spine of scapula and parallel to the fibres of the muscle in the middle of its belly, and over the middle deltoid muscle halfway between the insertion and the acromion. The electrodes over the upper trapezius muscle were placed one third lateral and 2 to 4 cm above the line from processus C7 to acromion. The ground electrode was placed at the radial styloid process of the non-tested arm. Correct electrode placement was confirmed by observing the appropriate EMG activity while performing a manual muscle test.

The output signals from EMG preamplifiers were digitized one-line (sampling frequency 1 kHz) by an analogue-to-digital converter installed in a personal computer. The digitized signals were stored on a hard disk for further analysis, the EMG power spectrum MF was calculated by using the Fast Fourier Transform Algorithms, whereas a 1024 data point window (1 s) slides over the whole recorded signal area with a 512 point shift (50% overlap). During the shoulder muscle isometric endurance test, MF was determined and averaged over each period of 5 s, whereas the following characteristics were calculated: initial MF (MF<sub>i</sub>) as mean of the first 10 s and mean of the last 10 s (MF<sub>e</sub>).

Additionally, MFslope per kilogram of weight held during the endurance test was calculated by formula:

$$\text{MF slope} = \frac{(\text{MFi} - \text{MFe}) \cdot t}{\text{MFi} \cdot P \cdot 60} \cdot 100 \text{ (\%} \cdot \text{min/kg)},$$

where t is endurance time, and P is weight, which was held by a subject in hand during the endurance test. MFslope was used for the assessment of shoulder muscle fatigability.



**Figure 1.** Shoulder girdle muscle endurance testing.

#### **4.3.4. Shoulder pain assessment**

Patients reported their shoulder pain level on a self-assessment 10-point visual analogue scale (VAS) with endpoints of no pain (0) and the worst possible pain (10) (Kivimäki et al., 2007). In the CT group, shoulder pain was assessed before and one month after treatment at day (Papers I–II). In the MUA group, shoulder pain was reported at day and by night before MUA, one and six months after MUA (Paper III).

#### **4.3.5. Shoulder Rating Questionnaire**

The functional limitation and disability of the shoulder was scored by the SRQ (Paper IV) before MUA and one and six months after MUA. The SRQ is a self-administered questionnaire including global assessment shoulder pain, daily activities, areas of improvement and satisfaction, recreational and athletic activities, and work. The total score ranges from a minimum of 17 points (worst functional status) to a maximum of 100 points (best functional status) (L'Insalata 1997).

#### **4.4. Statistical evaluation of the data**

Data are presented as means and standard errors ( $\pm$ SE). One-way analysis of variance (ANOVA) followed by Bonferroni *post hoc* comparisons was used to evaluate differences between the involved and the uninvolved extremity. A paired *t*-test was used to evaluate differences between pre- and post-treatment characteristics (Papers I–IV). A level of  $p < 0.05$  was selected to indicate statistical significance. The main differences in measures of the present study (shoulder aROM and shoulder muscle isometric strength in EXR and INR) between the involved and the uninvolved extremity were tested for statistical significance ( $\alpha = 0.05$ ). Statistical power analysis demonstrated that 15 participants were sufficient to detect a significant difference in shoulder aROM ( $\beta = 0.96$  and  $\beta = 0.99$ ) and shoulder muscle isometric strength ( $\beta = 0.99$  and  $0.97$ ) in EXR and INR between the involved and the uninvolved extremity before MUA (Paper III).

## 5. RESULTS

### 5.1. Active range of motion in patients with frozen shoulder syndrome before and after conservative treatment and manipulation under general anaesthesia

#### *Conservative treatment*

Before CT, patients with FSS demonstrated a reduction ( $p<0.05$ ) in shoulder aROM during FL, EXT, ABD and ADD for the involved extremity compared with the uninvolved extremity and controls (Table 3). FSS patients also showed a reduction ( $p<0.05$ ) in shoulder aROM during INR, EXR for the involved extremity compared with controls before CT. There were no significant differences in shoulder aROM during INR and EXR for the involved and the uninvolved extremities in FSS patients before CT. After the 4-week individualized CT, the shoulder aROM during FL, EXT, ABD and ADD in FSS patients for the involved extremity increased ( $p<0.05$ ) as compared with the pre-treatment level. However, in FSS patients, shoulder aROM during FL, EXT and ABD for the involved extremity remained significantly lower ( $p<0.05$ ) compared with the uninvolved extremity and controls after CT. There were no significant differences in shoulder aROM during ADD in FSS patients for the involved extremity compared with the uninvolved extremity and controls after the 4-week CT. The shoulder aROM during INR and EXR in FSS patients for the involved extremity did not change significantly with CT.

**Table 3.** Mean ( $\pm$ SE) values of active range of motion (aROM=deg) in frozen shoulder syndrome (FSS) patients ( $n=10$ ) before and after conservative treatment (CT) comparing the involved extremity (IN) with the uninvolved extremity (UN) and control subjects ( $n=10$ ).

Direction	Before			After	
	IN	UN	IN	UN	CONTROLS
FL	116.5 $\pm$ 8.7 <sup>a,b</sup>	173.8 $\pm$ 1.9	155.0 $\pm$ 6.2 <sup>a,b,c</sup>	174.5 $\pm$ 1.5	175.5 $\pm$ 1.8
EXT	37.5 $\pm$ 2.7 <sup>a,b</sup>	65.5 $\pm$ 6.6	50.5 $\pm$ 2.5 <sup>a,b,c</sup>	68.5 $\pm$ 4.7	67.5 $\pm$ 3.4
ABD	73.5 $\pm$ 11.1 <sup>a,b</sup>	160.0 $\pm$ 6.1	141.8 $\pm$ 8.9 <sup>abc</sup>	165.0 $\pm$ 4.2	169.5 $\pm$ 2.4
ADD	32.9 $\pm$ 2.8 <sup>ab</sup>	48.4 $\pm$ 3.3	46.0 $\pm$ 2.7 <sup>c</sup>	44.0 $\pm$ 3.0	54.0 $\pm$ 2.7
INR	59.7 $\pm$ 5.0 <sup>b</sup>	70.5 $\pm$ 3.9	59.5 $\pm$ 4.8 <sup>b</sup>	62.8 $\pm$ 4.3	78.2 $\pm$ 5.6
EXR	50.5 $\pm$ 5.7 <sup>b</sup>	60.9 $\pm$ 6.5	52.4 $\pm$ 5.9 <sup>b</sup>	59.5 $\pm$ 7.5	81.7 $\pm$ 5.2

<sup>a</sup>significant difference ( $p<0.05$ ) compared with the uninvolved extremity,

<sup>b</sup>significant difference ( $p<0.05$ ) compared with controls, <sup>c</sup>significant difference ( $p<0.05$ ) compared with the pre-treatment level. FL–flexion, EXT–extension, ABD–abduction, ADD–adduction, INR–internal rotation, EXR–external rotation.

### *Manipulation under general anaesthesia*

Before MUA, patients with FSS demonstrated a reduction ( $p<0.05$ ) in shoulder aROM during FL, ABD, EXT, ADD, INR and EXR for the involved extremity as compared with the uninvolved extremity (Table 4). One and six months after MUA, the shoulder aROM in all measured directions increased ( $p<0.05$ ) in patients with FSS for the involved extremity as compared with the pre-MUA level. Six months after MUA and physiotherapy, the shoulder aROM during FL and EXR of the involved extremity in patients remained significantly lower ( $p<0.05$ ) as compared with the uninvolved extremity, whereas shoulder aROM during INR, ABD, EXT and ADD did not differ significantly as compared with the uninvolved extremity.

**Table 4.** Mean ( $\pm$ SE) values of active range of motion (aROM=deg) in frozen shoulder syndrome (FSS) patients ( $n=15$ ) before, one and six months after manipulation under general anaesthesia (MUA) comparing the involved extremity (IN) with the uninvolved extremity (UN).

Direction	Before		1 month		6 months	
	IN	UN	IN	UN	IN	UN
FL	95.5 $\pm$ 4.8 <sup>a</sup>	166.2 $\pm$ 3.6	149.8 $\pm$ 5.7 <sup>ac</sup>	167.2 $\pm$ 2.2	162.5 $\pm$ 4.2 <sup>abc</sup>	171.5 $\pm$ 1.6
EXT	33.0 $\pm$ 2.8 <sup>a</sup>	57.1 $\pm$ 2.4	47.6 $\pm$ 2.3 <sup>ac</sup>	59.2 $\pm$ 1.7	56.2 $\pm$ 3.0 <sup>bc</sup>	60.1 $\pm$ 2.5
ABD	53.0 $\pm$ 4.9 <sup>a</sup>	168.0 $\pm$ 3.8	135.5 $\pm$ 9.2 <sup>ac</sup>	168.4 $\pm$ 3.6	160.0 $\pm$ 6.7 <sup>bc</sup>	173.1 $\pm$ 2.1
ADD	32.6 $\pm$ 1.9 <sup>a</sup>	46.5 $\pm$ 1.6	44.8 $\pm$ 1.2 <sup>ac</sup>	48.4 $\pm$ 1.6	49.8 $\pm$ 1.9 <sup>bc</sup>	50.7 $\pm$ 1.7
INR	41.3 $\pm$ 4.1 <sup>a</sup>	68.2 $\pm$ 3.4	63.4 $\pm$ 1.5 <sup>ac</sup>	70.5 $\pm$ 2.6	73.9 $\pm$ 2.6 <sup>bc</sup>	74.6 $\pm$ 1.3
EXR	33.8 $\pm$ 4.4 <sup>a</sup>	62.6 $\pm$ 3.7	49.9 $\pm$ 3.9 <sup>ac</sup>	68.4 $\pm$ 2.5	63.3 $\pm$ 3.4 <sup>bc</sup>	74.9 $\pm$ 3.2

<sup>a</sup>significant difference ( $p<0.05$ ) compared with the uninvolved extremity,

<sup>b</sup>significant difference ( $p<0.05$ ) compared with data after one month of treatment,

<sup>c</sup>significant difference ( $p<0.05$ ) compared with the pre-treatment level. FL–flexion, EXT–extension, ABD–abduction, ADD–adduction, INR–internal rotation, EXR–external rotation.

## **5.2. Shoulder muscle isometric strength in patients with frozen shoulder syndrome before and after conservative treatment and manipulation under general anaesthesia**

### *Conservative treatment*

Before the CT, patients with FSS showed a reduction ( $p<0.05$ ) in shoulder muscle isometric strength during FL, ABD, ADD, INR and EXR for the involved extremity compared with the uninvolved extremity (Table 5) and a reduction ( $p<0.05$ ) in shoulder muscle isometric strength during FL, ABD, ADD, EXR and INR compared with controls. After the 4-week CT, shoulder muscle isometric strength during FL and INR in FSS patients for the involved extremity increased ( $p<0.05$ ) as compared with pre-therapy level. In patients with FSS, shoulder muscle isometric strength during EXR for the involved extremity was significantly lower ( $p<0.05$ ) compared to controls after CT.

**Table 5.** Mean ( $\pm$ SE) values of shoulder muscle maximal isometric strength (kg) in frozen shoulder syndrome (FSS) patients (n=10) before and after conservative treatment (CT) comparing the involved extremity (IN) with the uninvolved extremity (UN) and control subjects (n=10).

Joint position	Before		After		CONTROLS
	IN	UN	IN	UN	
FL	10.8 $\pm$ 1.7 <sup>ab</sup>	17.8 $\pm$ 1.2	15.7 $\pm$ 1.9 <sup>c</sup>	16.2 $\pm$ 1.3	20.2 $\pm$ 1.9
ABD	10.3 $\pm$ 2.0 <sup>b</sup>	15.7 $\pm$ 3.1	14.1 $\pm$ 2.0 <sup>c</sup>	16.7 $\pm$ 1.2	17.4 $\pm$ 1.5
ADD	15.0 $\pm$ 2.4 <sup>b</sup>	20.3 $\pm$ 3.0	18.1 $\pm$ 2.0 <sup>c</sup>	19.3 $\pm$ 1.9	23.3 $\pm$ 1.3
INR	7.5 $\pm$ 5.0 <sup>b</sup>	11.4 $\pm$ 2.1	8.9 $\pm$ 1.0 <sup>abc</sup>	14.5 $\pm$ 1.2	14.5 $\pm$ 1.5
EXR	10.1 $\pm$ 2.2 <sup>ab</sup>	14.9 $\pm$ 2.6	12.5 $\pm$ 1.9 <sup>abc</sup>	14.2 $\pm$ 1.2	16.2 $\pm$ 1.3

<sup>a</sup> significant difference (p<0.05) compared with the uninvolved extremity,

<sup>b</sup> significant difference (p<0.05) compared with controls, <sup>c</sup>significant difference (p<0.05) compared with the pre-treatment level. FL–flexion, ABD–abduction, ADD–adduction, INR–internal rotation, EXR–external rotation.

#### *Manipulation under general anaesthesia*

A significant baseline reduction (p<0.05) was noted in shoulder muscle isometric strength for the involved extremity during FL, EXT, ABD, ADD, EXR and INR as compared with the uninvolved extremity before MUA (Table 6). Shoulder muscle isometric strength for the involved extremity in all mentioned joint positions increased (p<0.05) at the one-month follow-up compared with the baseline level, whereas the increase was significant (p<0.05) during FL and EXT at the six-month follow-up. After the one- and six-month follow-ups, shoulder muscle isometric strength for the involved extremity did not differ significantly compared with the uninvolved extremity.

**Table 6.** Mean ( $\pm$ SE) values of shoulder muscle maximal isometric strength (kg) in frozen shoulder syndrome (FSS) patients (n=15) before and after manipulation under general anaesthesia (MUA) comparing the involved extremity (IN) with uninvolved extremity (UN).

Direction	Before		1 month		6 months	
	IN	UN	IN	UN	IN	UN
FL	10.2 $\pm$ 1.4 <sup>a</sup>	15.2 $\pm$ 1.2	14.5 $\pm$ 1.1 <sup>c</sup>	17.9 $\pm$ 1.1	16.3 $\pm$ 1.3 <sup>c</sup>	17.9 $\pm$ 0.9
EXT	10.8 $\pm$ 0.9 <sup>a</sup>	16.4 $\pm$ 1.6	14.4 $\pm$ 1.1 <sup>c</sup>	17.7 $\pm$ 1.2	17.2 $\pm$ 0.9 <sup>c</sup>	18.3 $\pm$ 0.9
ABD	8.9 $\pm$ 1.0 <sup>a</sup>	15.4 $\pm$ 0.9	16.0 $\pm$ 1.4 <sup>c</sup>	16.3 $\pm$ 1.1	13.6 $\pm$ 1.0	16.2 $\pm$ 0.8
ADD	13.1 $\pm$ 0.9 <sup>a</sup>	17.8 $\pm$ 1.0	18.9 $\pm$ 1.9 <sup>c</sup>	20.1 $\pm$ 2.0	18.4 $\pm$ 0.9 <sup>bc</sup>	18.9 $\pm$ 0.7
INR	6.4 $\pm$ 0.6 <sup>a</sup>	11.2 $\pm$ 1.0	10.8 $\pm$ 1.2 <sup>c</sup>	13.0 $\pm$ 0.9	12.4 $\pm$ 0.8 <sup>c</sup>	13.3 $\pm$ 0.9
EXR	5.3 $\pm$ 0.6 <sup>a</sup>	10.2 $\pm$ 0.6	7.9 $\pm$ 0.7 <sup>c</sup>	9.6 $\pm$ 0.7	9.7 $\pm$ 1.3 <sup>c</sup>	10.6 $\pm$ 0.7

<sup>a</sup>significant difference (p<0.05) compared with the uninvolved extremity,

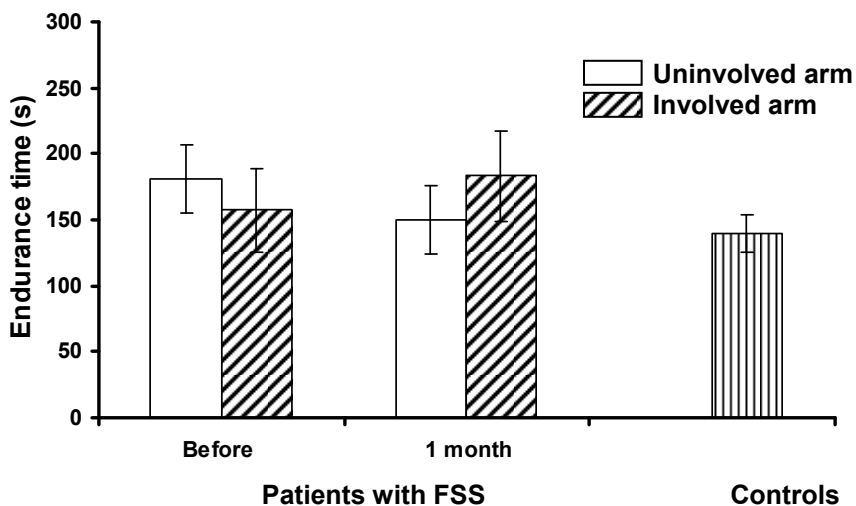
<sup>b</sup>significant difference (p<0.05) compared with data after the one-month treatment,

<sup>c</sup>significant difference (p<0.05) compared with the pre-treatment level. FL – flexion, EXT – extension, ABD – abduction, ADD – adduction, INR – internal rotation, EXR – external rotation.

### 5.3. Shoulder muscle isometric endurance test time in patients with frozen shoulder syndrome before and after conservative treatment and manipulation under general anaesthesia

#### *Conservative treatment*

Isometric endurance test time did not differ significantly in patients with FSS for the involved extremity before and after CT compared with the uninvolved extremity and control subjects (Fig. 2). No significant changes were observed in endurance time in patients with FSS for the involved and the uninvolved extremity after CT as compared with the pre-treatment level.

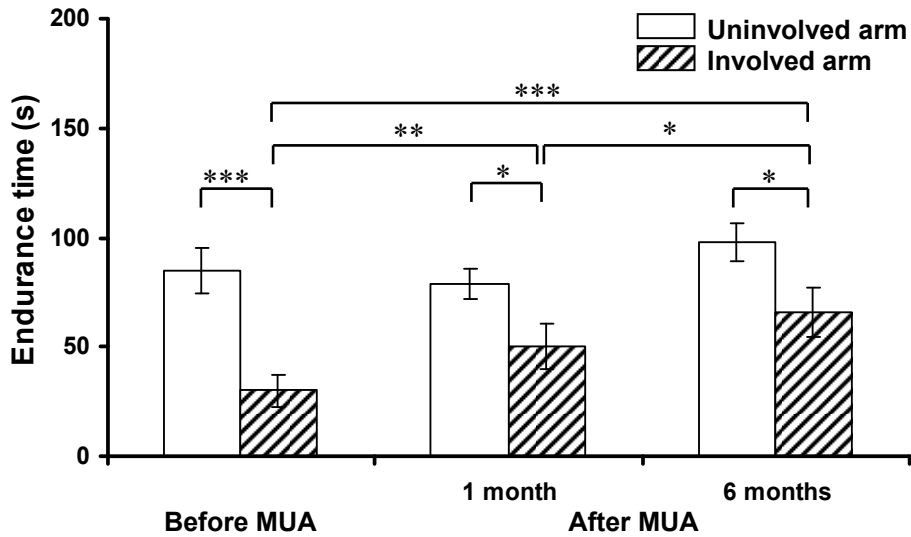


**Figure 2.** Mean ( $\pm$ SE) isometric endurance time in patients ( $n=10$ ) with frozen shoulder syndrome (FSS) before and after conservative treatment (CT) and in control subjects ( $n=10$ ).

#### *Manipulation under general anaesthesia*

Shoulder muscle isometric endurance time was significantly shorter ( $p<0.001$ ) at baseline for the involved extremity compared with the uninvolved extremity before MUA (Fig. 3). Shoulder muscle isometric endurance time was prolonged ( $p<0.01$ ) at one- and six-month follow-ups for the involved extremity compared with the baseline level. After the six-month follow-up, shoulder muscle isometric endurance time for the involved extremity was and remained significantly shorter ( $p<0.05$ ) compared with the uninvolved extremity.



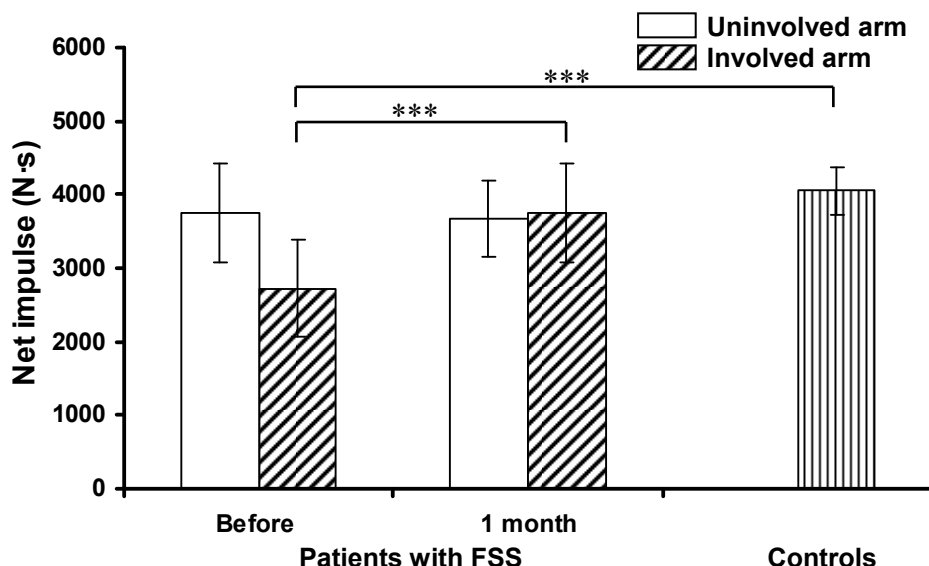


**Figure 3.** Mean ( $\pm$ SE) shoulder muscle isometric endurance time in patients (n=15) with frozen shoulder syndrome (FSS) before and after manipulation under general anaesthesia (MUA) \* $p$ <0.05, \*\* $p$ <0.01, \*\*\* $p$ <0.001.

#### **5.4. Shoulder muscle isometric working capacity in patients with frozen shoulder syndrome before and after conservative treatment**

##### *Conservative treatment*

Before the CT, patients with FSS showed lower ( $p$ <0.05) shoulder muscle isometric working capacity (NI) during the shoulder muscle isometric endurance test for the involved extremity as compared with controls (Fig. 4). There was a significant increase in NI during the shoulder muscle isometric endurance test in patients with FSS for the involved extremity after the 4-week CT as compared with the pre-treatment level. No significant differences in NI during the shoulder muscle isometric endurance test in patients with FSS for the involved extremity were observed as compared to controls and with the uninvolved extremity after CT.

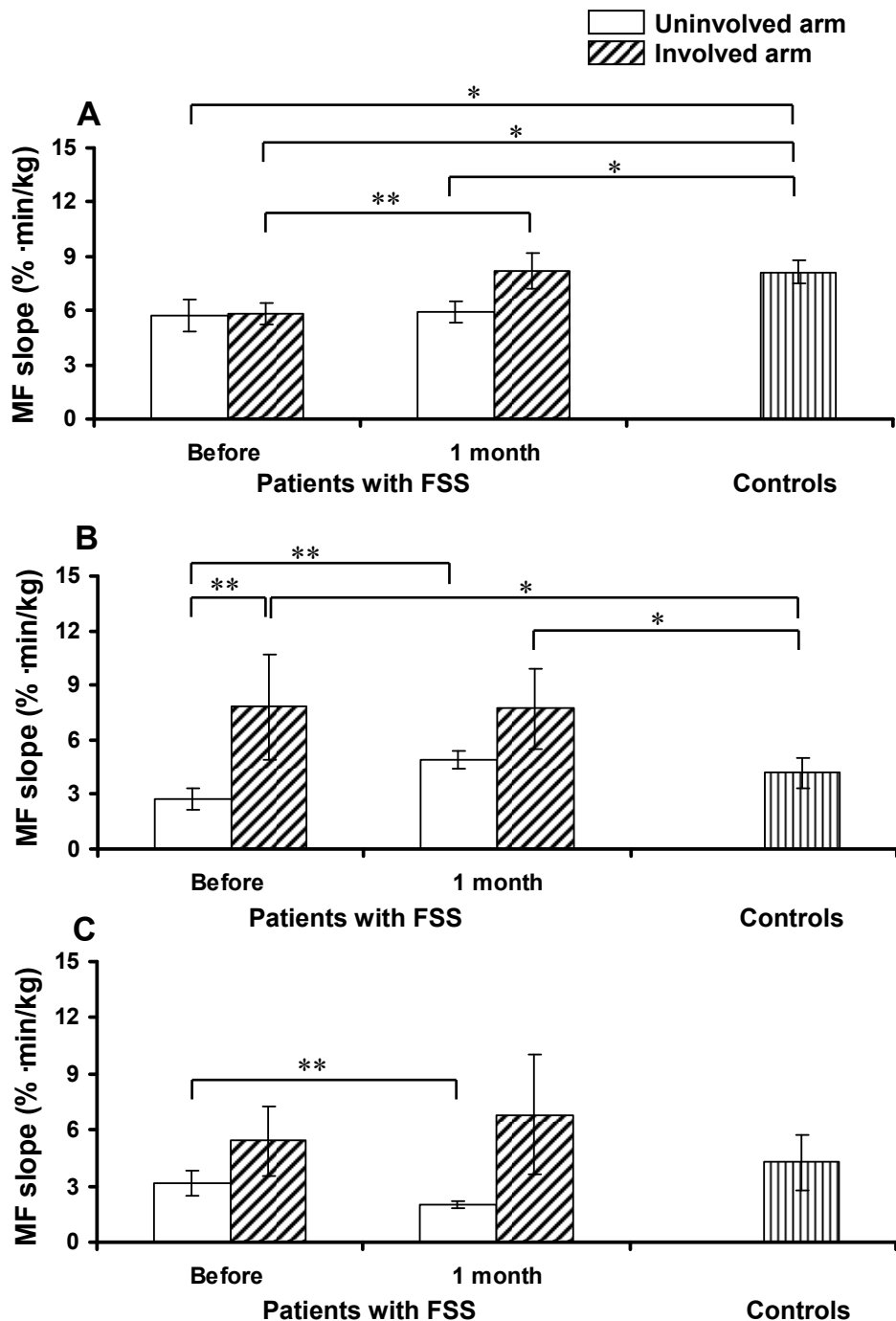


**Figure 4.** Mean ( $\pm$ SE) net impulse during shoulder muscle isometric endurance test in patients ( $n=10$ ) with frozen shoulder syndrome (FSS) before and after conservative treatment (CT) and in control subjects ( $n=10$ ), \*\*\* $p<0.001$ .

### **5.5. Electromyogram power spectrum median frequency in patients with frozen shoulder syndrome before and after conservative treatment**

Before the CT, EMG power spectrum median frequency (MF) slope of the deltoideus muscle during the isometric endurance test was lower ( $p<0.05$ ) in patients with FSS for the involved and the uninvolved extremity compared with control subjects (Fig. 5A). After CT, MF slope of the deltoideus muscle in FSS patients for the involved extremity increased ( $p<0.01$ ) compared with the pre-treatment level, whereas it did not differ significantly compared with the control subjects. MF slope of the deltoideus muscle in patients with FSS for the uninvolved extremity was lower ( $p<0.05$ ) compared to control subjects after CT.

Before the CT, patients with FSS showed higher ( $p<0.05$ ) MF slope of the infraspinatus muscle for the involved extremity during the isometric endurance test compared with the uninvolved extremity and control subjects (Fig. 5B). In patients with FSS, MF slope of the infraspinatus muscle for the involved extremity did not change significantly after the CT compared with the pre-treatment level, whereas it was lower ( $p<0.05$ ) than in control subjects. MF slope of the infraspinatus muscle in patients with FSS for the uninvolved extremity increased ( $p<0.01$ ) compared with the pre-treatment level.

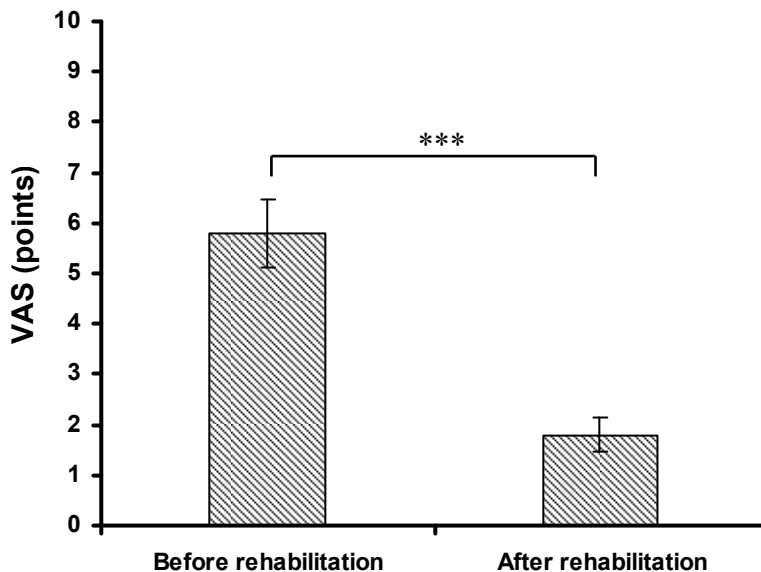


**Figure 5.** Mean ( $\pm$ SE) EMG power spectrum median frequency (MF) slope per kilogram weight, held in hand during the isometric endurance test determined from the deltoid (A), infraspinatus (B), and trapezius (C) muscles in patients ( $n=10$ ) with frozen shoulder syndrome (FSS) before and after conservative treatment (CT) and controls ( $n=10$ ), \* $p<0.05$ , \*\* $p<0.01$ .

Before and after the CT, MFslope of the trapezius muscle during the isometric endurance test in patients with FSS for the involved extremity did not differ significantly compared with the uninvolved extremity and control subjects (Fig. 5C). No significant changes were found in MFslope of the trapezius muscle in patients with FSS for the involved extremity after the CT as compared with the pre-treatment level. After the CT, MFslope of the trapezius muscle in patients with FSS for the uninvolved extremity decreased ( $p<0.05$ ) as compared with the pre-treatment level, whereas it did not differ significantly compared with the control subjects.

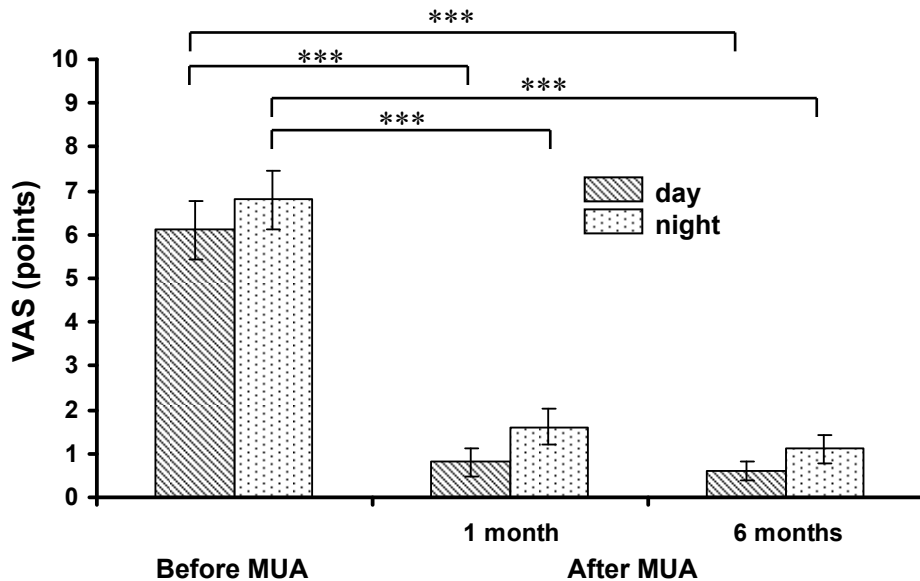
### 5.6. Shoulder pain in patients with frozen shoulder syndrome before and after conservative treatment and manipulation under general anaesthesia

In FSS patients, shoulder pain decreased ( $p<0.05$ ) after CT as compared with the pre-treatment level (Fig. 6).



**Figure 6.** Mean ( $\pm$ SE) shoulder pain in patients ( $n=10$ ) with frozen shoulder syndrome (FSS) measured by the visual analogue scale (VAS) before and after conservative treatment (CT), \*\*\* $p<0.001$ .

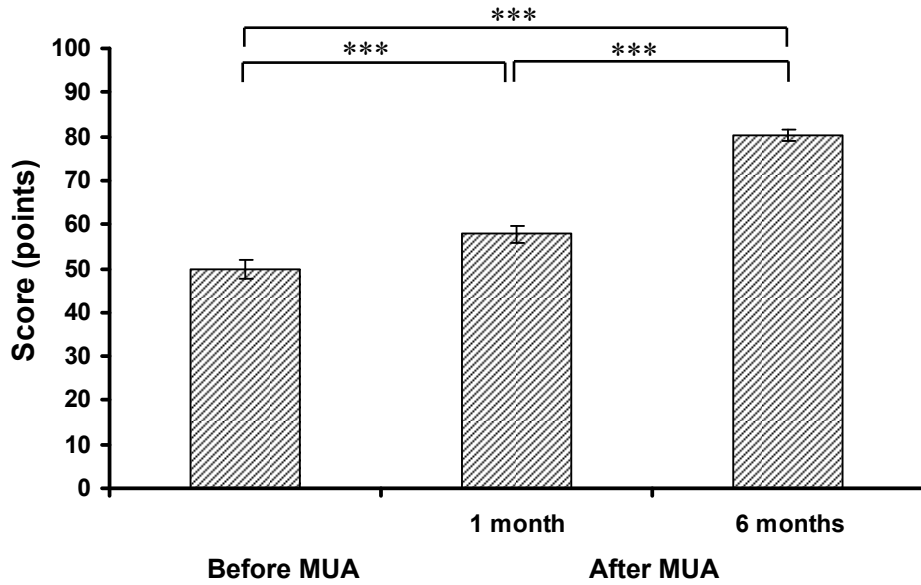
In patients with FSS, a significant decrease ( $p<0.05$ ) of pain by day and at night was noted one and six months after MUA and physiotherapy as compared with the before MUA level (Fig. 7).



**Figure 7.** Mean ( $\pm$ SE) shoulder pain in patients ( $n=15$ ) with frozen shoulder syndrome (FSS) measured by the visual analogue scale (VAS) before, one and six months after manipulation under general anaesthesia (MUA), \*\*\* $p<0.001$

### 5.7. Shoulder Rating Questionnaire in patients with frozen shoulder syndrome before and after manipulation under general anaesthesia

SRQ scores improved significantly from baseline ( $49.7\pm 8.7$  points) to the one-month follow-up ( $57.8\pm 7.6$  points,  $p<0.001$ ). Significant improvements were also recorded from baseline to the six-month follow-up ( $80.1\pm 4.8$  points,  $p<0.001$ ) (Fig. 8).



**Figure 8.** Mean ( $\pm$ SE) shoulder rating questionnaire score in patients (n=15) with frozen shoulder syndrome (FSS) before, one and six months after manipulation under general anaesthesia (MUA), \*\*\*p<0.001.

## 6. DISCUSSION

### 6.1. Active range of motion and shoulder pain

#### *Conservative treatment*

A marked shoulder aROM deficit was observed in patients with FSS before the CT, and MUA. The shoulder aROM during FL, EXT, ABD, ADD, INR and EXR in patients with FSS for the involved extremity was 25–59% lower, as compared with controls. Several previous studies demonstrated a reduced shoulder aROM in different directions in patients with FSS (Griggs et al., 2000; Vermeulen et al., 2000). The pathogenesis of primary FSS is unknown. Loss of dependent fold decreased capsular volume and capsular contractions have been demonstrated in patients with FSS (Neviasser, 1987). Additionally, contracture of the coracohumeral ligament, and capsular, and intraarticular subscapularis tendon thickening have been reported (Bunker and Anthony, 1995). Thus, the occurrence of the above-mentioned destructive changes might be the cause of shoulder aROM deficit in patients with FSS observed in this study.

In our study, patients with FSS showed substantial improvement in shoulder aROM during FL, EXT, ABD and ADD for the involved extremity after the 4-week CT coupled with non-significant changes in shoulder aROM during INR and EXR. The important factor for the rehabilitation of patients with FSS is decreasing shoulder pain using multiple therapeutic manoeuvres (massage, electrical stimulation, therapeutic exercises and analgesic) (Kordella, 2002).

In the present study, pre-treatment shoulder pain measured by VAS in patients with FSS was 5.8 points. Shoulder pain in patients with FSS decreased significantly after CT as compared with the pre-treatment level. Thus, the improvement of shoulder aROM in patients after treatment might partly be caused by reduced shoulder pain. Kibler et al. (1998) showed that after the rehabilitation program, shoulder muscles became more elastic permitting major movements in the shoulder girdle. However, the present study indicated that after CT, shoulder aROM during FL, EXT and ABD in patients with FSS in the involved extremity remained significantly lower compared with the uninvolved extremity and controls. Shaffer et al. (1992) showed that the post-rehabilitation deficit of shoulder aROM in patients with FSS was fairly long-standing. In a non-operative treatment study, Lorbach et al. (2010) found that in patients with FSS the recovery of shoulder pROM during FL, ABD, INR and EXR continued for 12 months after treatment. Binder et al. (1984) found that 48 months after rehabilitation, shoulder aROM in patients with FSS decreased significantly, compared with controls.

Shoulder pain reduction is important because this makes it possible to use the affected hand in performing therapeutic exercises and ADL. It has been suggested that shoulder pain reduction is most likely attributed to the inhibition of mechanoreceptors and free nerve endings that are distributed in the subacromial bursa and the rotator cuff (Vangsness et al., 1995).

### *Manipulation under general anaesthesia*

Shoulder aROM during FL, EXT, ABD, ADD, INR and EXR was lower in patients with FSS by 30–68% as compared with the uninvolved extremity before MUA. The mean differences in shoulder aROM for the involved compared to the uninvolved extremity one and six months after MUA were 5–20%. However, FL and EXR remained significantly lower six months after MUA as compared with the uninvolved extremity. After MUA, the patients performed supervised physiotherapy sessions three times per week in outpatient facilities and additionally performed a home exercise program. One month after MUA, the improvement of shoulder aROM in patients was good, this improvement continued for six months after the MUA survey. Most patients in our study commented that the shoulder pain was bad on the first days after MUA, but they were able to perform normal daily tasks, including personal hygiene.

DePalma (1952) identified that FSS developed only when muscular inactivity occurred in the shoulder joints of individuals past 40 years of age or in that period of life when degenerative alteration in the musculotendinous cuff, synovialis, and biceps tendons were demonstrable both macroscopically and microscopically.

Mengiadi et al. (2004) found that thickening of the coracohumeral ligament and the joint capsule in the rotator cuff interval, as well the subcoracoid triangle sign are characteristic MR arthrographic findings in FSS. Contracture of the rotator cuff interval is prevalent in patients with FSS (Ide and Takagi, 2004). Baums et al. (2007) and Wang et al. (2010) demonstrated similar shoulder pain and shoulder ROM recovery after arthroscopic release and MUA in patients with FSS during FL, INR and EXR. The limitation of shoulder aROM during INR and EXR is specific for patients with FSS (Farrell et al., 2005; Kivimäki et al., 2007). Kivimäki et al. (2007) found that shoulder aROM during EXR was 38 deg 6 weeks, 48 deg 3 months, 59 deg 6 months, and 65 deg 12 months after MUA. Farrell et al. (2005) follow-up 15 years (range 8.1 to 20.6 years) for the shoulder aROM during EXR improved from 23 deg to 67 deg. Bunker (1995) found that patients with FSS regained movement and stretching within 8 week after manipulation. Ng et al. (2009) followed patients with FSS six weeks after MUA and early physiotherapy. They found that shoulder ROM during FL improved from 104.2 to 157.6 deg, ABD from 70.5 to 150.0 deg and EXR from 13.9 to 45.6 deg, respectively. Rill et al. (2011) followed patients with FSS after MUA for a minimum of 24 months. They concluded that the fastest shoulder pROM recovery happened during the first 8 weeks, but recovery continued until the final follow-up. Six weeks after MUA patients usually continue with exercises at home but with no supervision they stop doing the exercises. Andresen (1998) suggested that at the 12-month follow-up, 79% of patients with FSS are relieved from their shoulder pain and 75% regain a near to normal shoulder ROM after MUA. Wiley (1991) had satisfactory results in 62% of 37 cases at a follow-up of 6 months. Bal (2008) concluded that therapeutic exercises are critically important in patients with FSS. They suggested that it is important to educate the patients regarding the improvement in shoulder ROM.



Stretching should be the focus of the treatment, whereas it can be taken beyond the limits of the available shoulder ROM. It is possible that a rehabilitation period of more than 4 weeks might be necessary to determine the relevant improvement in shoulder aROM in patients with FSS.

In our study, before MUA the mean shoulder pain by VAS was 6.1 points by day and 6.8 points at night. One and six months after MUA, shoulder pain significantly decreased by day and at night as compared to the pre-MUA level by 0.8 and 1.6 points one month after MUA and 0.6 and 1.1 points six months after MUA, respectively. Patients said that before MUA shoulder pain disturbed their everyday life, especially activities requiring overhead movement with the involved hand, and sleeping at night.

DePalma (1952) identified that at all times during the course of the disease, shoulder pain is the most significant clinical manifestation. A few days after MUA, patients use analgesics. One week after MUA, shoulder pain begins to decrease. Diwan et al. (2005) have shown that preoperative shoulder pain by VAS score range decreased from severe to very severe to mild-to-moderate one week postoperatively. This shoulder pain reduction was maintained at 12 weeks, and continued to reduce to mild at 6 months, and remained on this level at 2 years. Kivimäki et al. (2007) investigated patients with FSS who were divided into a MUA group, and a home exercise group. Shoulder pain before therapy was 6.6 points in the manipulation group and 6.4 points in the exercise group, respectively. Outcomes were measured during follow-up examinations at 6 weeks and 3, 6, and 12 months after manipulation. Shoulder pain in the manipulation group was 4.9, 3.9, 2.0, and 1.5 points, respectively. Roubal et al. (2008) found that traditional manipulation after failed arthroscopic capsular release for recalcitrant FSS showed the shoulder pain changing as follows: at the initial evaluation 7, one day after post-arthroscopic release 6, after post-translational manipulation 2, and at the two-year follow-up 1 points.

## **6.2. Shoulder muscle isometric strength**

### *Conservative treatment*

The present study demonstrated a significant deficit in shoulder muscle isometric strength in patients with FSS before CT, and MUA, whereas shoulder muscle isometric strength was measured by a hand-held dynamometer.

CT group results indicated that before treatment, shoulder muscle isometric strength for the involved extremity was 38–48% lower in the measured directions. FSS is accompanied by shoulder pain and the patients tried to use the hand sparingly (Kibler, 1998; Andersen et al., 1998). It has been shown that ADL was markedly decreased in patients with FSS as compared with healthy subjects (Vermeulen et al., 2002). The decreased physical activity and shoulder immobilization are important factors of shoulder muscle atrophy, decreased strength and endurance. Kitahara et al. (2003) showed that three-week hand immobilization decreased hand muscle strength by 18–45%.

The present study indicated a significant improvement in shoulder muscle isometric strength in patients with FSS in all measured directions after the 4-week CT. The observed increase in shoulder muscle isometric strength during FL, ABD, ADD, INR and EXR in FSS patients for the involved extremity was 15–31%. Thus, shoulder muscle isometric strength improvement in patients with FSS after CT was more pronounced for shoulder FL, ABD and ADD than INR and EXR. The improvement in maximal voluntary force generation capacity in patients with FSS may be partly caused by significantly reduced shoulder pain after treatment. The link that explains how voluntary muscle force production is impaired by pain is more difficult to explore.

#### *Manipulation under general anaesthesia*

Before MUA, patients with FSS showed a significant reduction in shoulder muscle isometric strength for the involved extremity as compared with the uninvolved extremity. Our results indicated that before MUA in patients with FSS, shoulder muscle isometric strength for the involved extremity was 26–48% lower in the measured directions as compared with the uninvolved extremity. One month after MUA, shoulder muscle isometric strength for the involved extremity in patients with FSS increased by 27–49% in the measured directions, as compared with the pre-MUA level. Six months after MUA, shoulder muscle isometric strength in patients for the involved extremity improved, while this was a non-significant difference as compared with the uninvolved extremity.

Physical activity level, which varies depending on the individual's job or how much they exercise, and mental factors, such as motivation or pain-related depression, affect the measurement of maximal strength (Kramer et al., 2005). A markedly reduced isometric voluntary force generation capacity of shoulder muscles in patients with FSS before MUA can be related to several factors: muscle spasm, capsular contracture and immobilization (Lin et al., 2009). It has been indicated that muscle strength, and both the fast-twitch and slow-twitch muscle fibre area significantly decreased after 5–6 weeks of immobilization (MacDougall et al., 1980). Decreased shoulder pain and increased shoulder ROM, as well as exercise therapy were important factors in improving shoulder muscle isometric strength in patients with FSS during the early rehabilitation period after MUA.

Andrews et al. (1996) composed the normative values for isometric muscle strength with a hand-held dynamometer for asymptomatic adults. In the 50–59-year-old group, the mean values of shoulder muscle isometric strength during INR and EXR in the dominant hand were 10.3 and 10.4 kg in women, and 15.9, and 19.7 kg in men, respectively.

In our study of patients with FSS, the isometric strength of shoulder muscles for the uninvolved extremity was similar while, isometric strength for the involved extremity 6 months after MUA was lower.

Bohannon (1986) measured shoulder muscle isometric strength in patients with neurological diagnoses, and found that the mean strength during ADD,

INR and EXR was 8.6, 8.2, and 9.7 kg, respectively. This indicates that shoulder muscle isometric strength decreased in different diseases. It is important to begin with strength exercises as soon as possible with FSS patients after MUA. Cadogan et al. (2011) demonstrated that the resisted muscle test for peak isometric muscle strength during resisted ABD (the affected side) with a manual muscle tester had high levels of reliability. Hirschmann et al. (2010) found a high retest reliability for the strength measurements in all evaluated positions (shoulder ABD in 30, 60, 90 deg). We believe that shoulder muscle isometric strength assessment in patients with FSS is important because in successful ADL activities we needed near normal shoulder ROM and muscle strength. Wong and Tan (2010) noted that in examining patients with FSS, mild disuse atrophy of the deltoid and supraspinatus have usually been observed in long-standing cases. DePalma (1963) noted that all shoulder motions are guarded, and painful atrophy of the deltoid and spinatus muscle can be detected.

Before MUA, the patients' movements are limited and painful and muscles are inactive. This condition is ideal for the development of muscle atrophy. Immediately after MUA, patients should begin with physiotherapy. Othman and Taylor (2002) noted that an aggressive postoperative physiotherapy regime is essential immediately after MUA in patients with FSS. It has been suggested that the recruitment of motor units is dependent on produced muscle force, which is smaller in the pain condition compared with the non-pain condition. The effect of muscle pain on the motor system can be seen as an adaptation to pain (Madeleine, 2010). The pain-adaptation model and the neural mechanisms subserving these changes in motor function during muscle pain can be related to reciprocal inhibition or excitation of motoneurons. It has been demonstrated that experimentally induced muscle pain decreases motor unit firing rate during sub-maximal isometric contractions in humans (Farina et al., 2004). Henneman et al. (1965) showed that the recruitment of motor units was dependent on the produced torque, which was smaller in the pain condition compared with the non-pain condition. Muscle pain influences motor control via numerous reflex and central mechanisms (Roubal and Placzek, 2008).

This can explain why shoulder muscle isometric strength in patients with FSS before MUA, and one month after MUA was lower as compared with the uninvolved extremity. When shoulder pain decreased, patients with FSS were able to produce more strength in shoulder muscles, and could increasingly use the involved extremity in ADL. Roe et al. (2000) explained that in patients with rotator tendinosis, the increase in muscle activation could be caused by a combination of the training effect on the muscles, and increased motor drive. It has been demonstrated that the increase in shoulder muscle strength is related to the decrease in shoulder pain perception and the psychological improvement the patients experienced during the program (Koumandakis et al., 2005) and such improvements have been positively related to the muscle strength level before rehabilitation (Mannion et al., 2001). In five chronic musculoskeletal disease conditions, Lund et al. (1991) found that pain reduces agonist and increases antagonist muscle activity. Graven-Nielsen et al. (1997) concluded that muscle

pain reduces isometric force as well as endurance during submaximal isometric contractions.

However, if patients with FSS did not feel pain in the shoulder joint and muscles, they started to use the involved extremity more in ADL, which helped them improve shoulder muscle condition.

### **6.3. Shoulder muscle isometric endurance test time**

Before treatment, the shoulder muscle isometric endurance time was significantly shorter for the involved extremity in the MUA group patients, whereas there was no difference in the shoulder muscle isometric endurance time in the CT group patients.

The shoulder muscle isometric endurance time in patients with FSS for the involved extremity before MUA was shorter by 65% as compared with the uninvolved extremity. Before MUA, six patients failed to perform the shoulder muscle isometric endurance test; they were not able to hold the weight at the target level. One month after MUA, the mean shoulder muscle isometric endurance time in patients for the involved extremity was shorter by 37% as compared with the uninvolved extremity, whereas three patients failed to perform the endurance test. Six months after MUA, shoulder muscle isometric endurance time in patients with FSS for the involved extremity was shorter by 33% as compared with the uninvolved extremity, whereas two patients failed to perform this test. This improvement in shoulder muscle function might primarily result from the neural adaptation observed, especially during the earlier weeks of exercise training (Häkkinen et al., 1998).

Pain has been shown to be an important predictor of isometric endurance of shoulder muscles (Brox et al., 1996). Brox et al. (1996) showed that increased shoulder pain, emotional stress and muscle weakness were the limiting factors for shoulder muscle isometric endurance in patients with rotator tendonitis of the shoulder. However, it has been suggested that after local shoulder pain reduction, endurance time remains unchanged indicating that pain reduction is not sufficient to improve shoulder muscle endurance (Brox et al., 1997). The musculature of the shoulder joint can be divided into intrinsic (centering and stabilizing) and extrinsic (mobilizing) muscle groups (Irlenbusch, 1999). It is known that more than in other joints, shoulder movements require a control system for constant readjustment of the intramuscular coordination of all muscles involved. It has been speculated that improved intra- and intermuscular coordination, reduced shoulder pain, shoulder muscle atrophy and increased shoulder joint mobility are possible factors for improvement of shoulder muscle function in patients with FSS after rehabilitation. It is very important to pay attention not only to the strengthening of the shoulder region muscles in the rehabilitation program after MUA in patients with FSS, but also to the improving the endurance of these muscles. It can be concluded that the recovery of shoulder muscle endurance in patients with FSS after MUA is more delayed

than the recovery of shoulder muscle isometric strength. A shoulder muscle endurance training program should be recommended for patients after MUA.

In the CT group, the recovery of shoulder muscle isometric endurance time did not differ significantly during one month of rehabilitation, whereas in the MUA group the shoulder muscle isometric endurance time recovery was lower. It can be seen that the shoulder muscle function was worse in the MUA group. It could be explained by the longer disease period.

#### **6.4. Changes in electromyogram power spectrum during shoulder muscle isometric endurance test**

We examined the EMG power spectrum MFslope over time per kilogram weight, which was held in hand during the shoulder muscle isometric endurance test, whereas MFslope recordings were obtained from the deltoideus, infraspinatus and trapezius muscles. The results indicated that MFslope of the infraspinatus and trapezius muscles was unchanged by CT for the patients' involved extremity, whereas this parameter increased by 29% for deltoideus muscle following CT. In patients with FSS, MFslope of the infraspinatus muscle for the involved extremity was significantly higher before and after the CT compared with the control subjects and the uninvolved extremity indicating higher fatigability. Thus, FSS leads to a significant increase in fatigability of the infraspinatus muscle. For deltoideus and trapezius muscles, MFslope in patients for the involved extremity did not differ significantly after CT as compared to the uninvolved extremity and healthy controls.

The EMG power spectrum MF or half-power point of the EMG measures was used as an indicator of local muscle fatigue (Merletti et al., 1984; De Luca, 1993). During sustained contraction, metabolic factors may contribute to reducing force production and this is compensated by a gradual increase in motor drive leading to an increased number of active motor units (Bigland-Ritchie et al., 1986). This increase is reflected in a progressive increase in EMG amplitude and in a shift of EMG power spectrum to the lower frequencies (spectral compression) (Merletti et al., 1984; De Luca, 1993). The initial value of MF was associated with the distribution of the muscle fibre type recruited (Mannion and Dolan, 1996), while MFslope, i.e. the rate of decline over time was associated with the fatigability properties of the active motor units (De Luca, 1993). EMG power spectrum MFslope (spectrum compression) during a fatiguing submaximal contraction has been attributed to the changes in the action potential propagation of individual muscle fibres, which are the result of the underlying accumulation of metabolites (i.e.  $H^+$ , lactate and extracellular  $K^+$ ) (Bigland-Ritchie et al., 1981; Tesch et al., 1983) during the fatiguing contraction reducing intracellular pH (Brody et al., 1991), and thus, decreasing sarcolemma excitability.

## **6.5. Shoulder Rating Questionnaire**

The present study demonstrated that the mean SRQ score in patients with FSS increased from 50 points of 100 before MUA to 58 and 80 points of 100 at one and six months after MUA, respectively. Therefore, it has to be considered that the functional limitation and disability of the involved shoulder in patients with FSS after MUA in our study markedly decreased during the 6-month post-treatment period. This means that after MUA, the patients were able to perform more active movements with the involved upper extremity. Similar SRQ score improving tendencies were reported in a study by Marx et al. (2007), where the SRQ score improved 6 months after intraarticular corticosteroid injections meanly to 90 (52–100) points. In the Vermeulen et al. (2006) study, the mean changes in the SRQ score after using the high-grade mobilization technique were 37.5 initial, 25.8 three months after treatment, 32.3 six months after treatment and 38.3 twelve months after treatment, respectively.

In conclusion, when shoulder pain was relieved in patients with FSS, shoulder aROM, shoulder muscle isometric strength and shoulder muscle isometric endurance time increased, fatigability decreased and the involved extremity function improved. Patients are more active in everyday life, whereas extreme movements are limited in a number of patients for a long time.

## CONCLUSIONS

1. In patients with FSS, a markedly reduced shoulder active range of motion and shoulder muscle isometric strength for the involved extremity was observed before conservative treatment and MUA.
2. A conservative treatment program with the duration of one month improved shoulder active range of motion during flexion, extension, abduction and adduction in patients with FSS with no effect on shoulder external and internal rotation. The improvement of shoulder muscle isometric strength after this treatment was more pronounced for shoulder flexors, abductors and adductors than in external and internal rotators.
3. In patients with FSS, isometric working capacity of shoulder flexors for the involved extremity during sustained submaximal contraction improved after one month of conservative treatment.
4. The fastest improvement of shoulder active range of motion and shoulder muscle isometric strength in patients with FSS proceeded following the first month after MUA. Shoulder active range of motion during flexion and external rotation remained reduced six months after MUA.
5. The recovery of shoulder muscle isometric endurance after MUA was more delayed than the recovery of isometric strength and shoulder active range of motion.
6. In patients with FSS, shoulder pain reduced markedly following one month of conservative treatment and MUA, and functional limitation and disability of the involved shoulder progressively decreased following six months of MUA.

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## SUMMARY IN ESTONIAN

### **Õlaliigese ja õlavöötme lihaste funktsionaalse seisundi muutused adhesiivse kapsuliidiga patsientidel konservatiivse ravi ja redressiooni mõjul**

#### **Sissejuhatus**

Adhesiivne kapsuliit on haigusseisund, mida iseloomustab tugev valu õlaliigeses eriti liigutuste sooritamisel ning õlaliigese aktiivse ja passiivse liikuvuse oluline vähenemine. Haiguse esinemissagedus populatsioonis on 2–5%, kusjuures sagedamini esineb seda naistel vanuses üle 40 eluaasta. Adhesiivne kapsuliit võib tekkida pärast õlaliigese traumat ning kaasnedä südameveresoonkonna ja kopsuhaigustega, radikulopaatiaga, diabeediga või insuldiga, põhjustades patsientidele raskusi igapäevaelu toimetustega hakkamasaamisel. Seejuures on selle haiguse etioloogia veel ebaselge. Ravi esmaseks ülesandeks on valu leevendamine õlaliigeses ning õlaliigese funktsiooni taastamine. Kasutusel on erinevaid ravimeetodeid, kuid reeglina alustatakse konservatiivse raviga, kus olulisel kohal on füsioteraapia ja liigesesisesed süstid. Kui konservatiivne ravi ei ole andnud kolme kuni kuue kuu jooksul haiguse diagnoosimisest oodatud tulemusi, siis kasutatakse kas mittekirurgilist ravi (näiteks redressiooni üldanesteesias) või kirurgilist ravi (õlaliigese artroskoopiline kapsulotoomia jne).

Läbitõõtatud kirjanduse põhjal võib väita, et adhesiivse kapsuliidiga patsientide funktsionaalse seisundi hindamiseks kasutatakse erinevaid küsimustikke, hinnatakse valu õlaliigeses ning kahjustatud õlaliigese liikuvust. Samas on vähe informatsiooni õlavõõtme lihaste jõu, vastupidavuse ja väsimuse kohta.

#### **Uurimistõõ eesmärk ja ülesanded**

Uurimistõõ eesmärgiks oli välja selgitada õlaliigese ja õlavõõtme lihaste funktsionaalse seisundi taastumise iseärasused adhesiivse kapsuliidiga patsientidel pärast konservatiivset ravi ja redressiooni üldanesteesias.

Tõõs püstitati järgmised ülesanded:

1. Määrata muutused õlaliigese aktiivses liikuvuses, õlavõõtme lihaste isomeetrilises jõus ja vastupidavuses adhesiivse kapsuliidiga patsientidel enne ja pärast ühekuulist konservatiivset ravi (I–II artikkel);
2. Määrata muutused õlavõõtme lihaste isomeetrilises jõus, vastupidavuses ja õlaliigese aktiivses liikuvuses adhesiivse kapsuliidiga patsientidel enne, üks kuu ja kuus kuud pärast redressiooni üldanesteesias (III–IV artikkel);
3. Hinnata valu õlaliigeses ja funktsionaalseid piiranguid adhesiivse kapsuliidiga patsientidel enne ja pärast konservatiivset ravi ja redressiooni üldanesteesias (I–IV artikkel).

## Uuritavad ja kasutatud meetodika

Uuringus osales 28 adhesiivse kapsuliidiga patsienti ning 10 õlaliigese- ja kaelavaevusteta inimest (kontrollrühm). Uuringud viidi läbi Tartu Ülikooli kinesioloogia ja biomehaanika laboris ja füsioteraapia Tartu Ülikooli Kliinikumi Spordimeditsiini ja Taastusravi Kliiniku ambulatoorses osakonnas.

Esimeses uuringus osales 10 adhesiivse kapsuliidiga patsienti (7 naist ja 3 meest), moodustades konservatiivse ravi grupi. Teises uuringus osales 18 adhesiivse kapsuliidiga patsienti (10 naist ja 8 meest), kellel teostati õlaliigese redressioon üldanesteesias.

Konservatiivse ravi rühma liikmed läbisid 10 individuaalse füsioteraapia seansi, mis koosnes terapeutilisest harjutusest saalis ja basseinis õlaliigese liikuvuse ning lihaskõhu taastamiseks, samuti massaažist ja elektroteraapiast õlavöötme piirkonna valu leevendamiseks.

Redressiooni üldanesteesias teostas ortopeed. Sellele järgnes patsientidel individuaalne füsioteraapia õlaliigese funktsiooni taastamiseks.

Õlaliigese aktiivne liikuvus määrati seisvas asendis õlavarre fleksioonil, ekstensioonil, abduktsioonil, aduktsioonil, sise- ja välisrotatsioonil (Mellin et al., 1994, Braddom et al., 1996; Clarkson 2005). Kasutati goniomeetreid (*Bubble Inclinator*, USA ja *Myrin*, Norra). Goniomeetri näit fikseeriti uuritava liigutuse lõppasendis.

Õlavöötme lihaste tahteline isomeetriline maksimaaljõud määrati istuvas asendis õlavarre fleksioonil, ekstensioonil, abduktsioonil, aduktsioonil, sise- ja välisrotatsioonil. Kasutati manuaalset lihastestrit (*Lafayette Manual Muscle Test System*, USA). Lihastester fikseeriti lähteasendisse, kusjuures vajalik nurk õlaliigeses määrati goniomeetriga (*Arthrodial Protractor*, USA).

Õlavöötme lihaste staatilise vastupidavuse testimisel istus vaatlusalune toolil, hoides sirget kätt ees 45° nurga all õlaliigeses (õlavars oli viidud 45° fleksioon- ja abduktsioon-asendisse). Konservatiivse ravi grupi liikmetel tuli selles asendis hoida käes raskust, mis moodustas 30% tahtelisest isomeetrisest maksimaaljõust ning redresseeritud õlaliigesega patsientidel raskust, mis oli naistel 3 kg ja meestel 5 kg. Vastupidavustest sooritati suutlikkuseni. Õlavöötme lihaste staatilist vastupidavust hinnati raskuse hoidmise aja järgi. Raskuse hoidmise käigus registreeriti delta-, trapets- ja harjaaluse lihase bioelektrilist aktiivsust elektromüograafia (*Medicor MG 440*, Ungari).

Valu õlaliigeses hinnati patsientidel 10-punktilise visuaal-analoog skaala (VAS) abil: 0 – valu ei ole, 10 – äärmuslik valu (Kivimäki et al., 2007). Spetsiaalse õlavaevuste küsimustikuga (*“Shoulder Rating Questionnaire”*) hinnati patsientidel igapäevaelu toimetustega hakkamasaamist.

## Järeldused

1. Adhesiivse kapsuliidiga patsientidel oli õlaliigese aktiivne liikuvus ja õlavöötme lihaste isomeetriline jõud haigel jäsemel oluliselt vähenenud enne konservatiivset ravi ja redressiooni üldanesteesias.
2. Ühekuulise konservatiivse ravi tulemusena paranes patsientidel õlaliigese aktiivne liikuvus fleksioonil, ekstensioonil, abduktsioonil ja aduktsioonil, kuid raviefekti ei ilmnenud õlaliigese aktiivses liikuvuses sise- ja välisrotatsioonil. Ravi järgselt toimus õlavöötme lihaste isomeetrilise jõu suurenemine, mis oli enam väljendunud õlavöötme fleksorites, abduktorites ja aduktorites võrrelduna sise- ja välisrotaatoritega.
3. Ühekuulise konservatiivse ravi tulemusena suurenes patsientidel õlavöötme lihaste staatiline töövõime, hinnatuna lihasvastupidavuse näitajate alusel.
4. Patsientidel toimus haige jäseme õlaliigese aktiivse liikuvuse kiirem taastumine esimese kuu jooksul pärast redressiooni üldanesteesias. Seejuures õlaliigese aktiivne liikuvus fleksioonil ja välisrotatsioonil jäi piiratuks kuus kuud pärast redressiooni üldanesteesias.
5. Pärast redressiooni üldanesteesias oli patsientidel õlavöötme lihaste staatilise vastupidavuse taastumine aeglasem võrreldes nende lihaste isomeetrilise jõu ja õlaliigese aktiivse liikuvusega.
6. Nii pärast konservatiivse ravi algust kui ka redressiooni üldanesteesias vähenes patsientidel märgatavalt valu õlaliigeses esimese kuu lõpuks. Kuue kuu jooksul pärast redressiooni üldanesteesias vähenes patsientidel progresseruvalt õlaliigese funktsionaalne piiratus.

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Since 2003 University of Tartu, Institute of Exercise Biology and Physiotherapy. Assistant of Physiotherapy of Internal Diseases (part time), Research fellow (part time)  
1996–2010 Tartu University Hospital, Sports Medicine and Rehabilitation Clinic, physiotherapist  
1993–2001 Sports club “Tähtvere” coach  
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Evidence based stroke rehabilitation. Eesti Taastusarstide Selts. Laulasmaa, 20.04.2012.  
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Clinical education in physiotherapy – the best way to learn professional skills. Tartu Ülikool, Tartu Tervishoiukõrgkool, Eesti Füsioterapeutide Liit, 01.–02.02.2008.  
M-Test, Basic Practical course. Estonian Physiotherapy Association. Tartu, 20.–21.09.2008.

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Low back pain, assessment and manual therapy. Tartu, 11.–12.04.2009  
Body balance and its measurement with digital biometry. Tartu Ülikool, Avatud  
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Kliiniline haridus füsioteraapias – parim võimalus erialaste pädevuste omandamiseks. Tartu Ülikool, Tartu Tervishoiukõrgkool, Eesti Füsioterapeutide Liit, 01.–02.02.2008.  
M-Test, Basic Practical course. Estonian Physiotherapy Association. Tartu, 20.–21.09.2008.

Ortopeediline massaaž. Tartu Ülikool. Tartu, 17.–18.03.2006  
Alaseljavaevused, nende hindamine ja manuaalteraapia. Tartu, 11.–12.04.2009  
Keha tasakaal ja selle mõõtmine digitaliseeritud biomeetria meetodil. Tartu  
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Muud teaduslikud artiklid – 8

Konverentside teesid – 5

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