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Does muscle guarding play a role in range of motion loss in patients with frozen shoulder?

L. Hollmann[a], M. Halaki[b], S.J. Kamper[c,d], M. Haber[e], K.A. Ginn[f,*]

[a] University of Canberra, Faculty of Health, Australia
[b] The University of Sydney, Faculty of Health Sciences, Discipline of Exercise & Sport Science, Australia
[c] The University of Sydney, School of Public Health, Australia
[d] Centre for Pain, Health and Lifestyle, Australia
[e] University of Wollongong, Faculty of Medicine, Australia
[f] The University of Sydney, Faculty of Medicine & Health, Discipline of Anatomy & Histology, Australia

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ABSTRACT

Study Design: Observational: cross-sectional study.
Background: Idiopathic frozen shoulder is a common cause of severe and prolonged disability characterised by spontaneous onset of pain with progressive shoulder movement restriction. Although spontaneous recovery can be expected the average length of symptoms is 30 months. Chronic inflammation and various patterns of fibrosis and contracture of capsuloligamentous structures around the glenohumeral joint are considered to be responsible for the signs and symptoms associated with frozen shoulder, however, the pathoanatomy of this debilitating condition is not fully understood.
Objectives: To investigate the feasibility of a muscle guarding component to movement restriction in patients with idiopathic frozen shoulder.
Methods: Passive shoulder abduction and external rotation range of motion (ROM) were measured in patients scheduled for capsular release surgery for frozen shoulder before and after the administration of general anaesthesia.
Results: Five patients with painful, global restriction of passive shoulder movement volunteered for this study. Passive abduction ROM increased following anaesthesia in all participants, with increases ranging from approximately 55°–110° of pre-anaesthetic ROM. Three of these participants also demonstrated substantial increases in passive external rotation ROM following anaesthesia ranging from approximately 15°–40° of pre-anaesthetic ROM.
Conclusion: This case series of five patients with frozen shoulder demonstrates that active muscle guarding, and not capsular contracture, may be a major contributing factor to movement restriction in some patients who exhibit the classical clinical features of idiopathic frozen shoulder. These findings highlight the need to reconsider our understanding of the pathoanatomy of frozen shoulder.
Level of evidence: Level 4.

1. Introduction

Idiopathic frozen shoulder has puzzled the medical community since it was first described in the late 19th century. It occurs in approximately 8%–10% of the general population and up to 29% of the diabetic population (Walker-Bone et al., 2004; Balci et al., 1999). It is characterised by spontaneous onset of pain with progressive, marked active and passive stiffness at the glenohumeral joint (Lundberg, 1969; Nash and Hazelman, 1989) usually resulting in gross loss of function (Jones et al., 2013). The condition is described as self-limiting with gradual return of painfree shoulder function after 1–3 years in most patients (Hand et al., 2008).

There is no definitive diagnostic test for frozen shoulder and diagnosis is based on physical examination following exclusion of osteoarthritis, significant rotator cuff disease, locked dislocations, fractures or avascular necrosis as the cause of symptoms (Lewis, 2015). Clinical diagnosis of frozen shoulder is made if the patient has painful restriction of active and passive motion in at least two planes of movement, of

* Corresponding author. Discipline of Anatomy & Histology, Faculty of Medicine & Health, Sydney Medical School, The University of Sydney, S213, Cumberland Campus C42, PO Box 170, Lidcombe, NSW, 1825, Australia.
E-mail address: karen.ginn@sydney.edu.au (K.A. Ginn).

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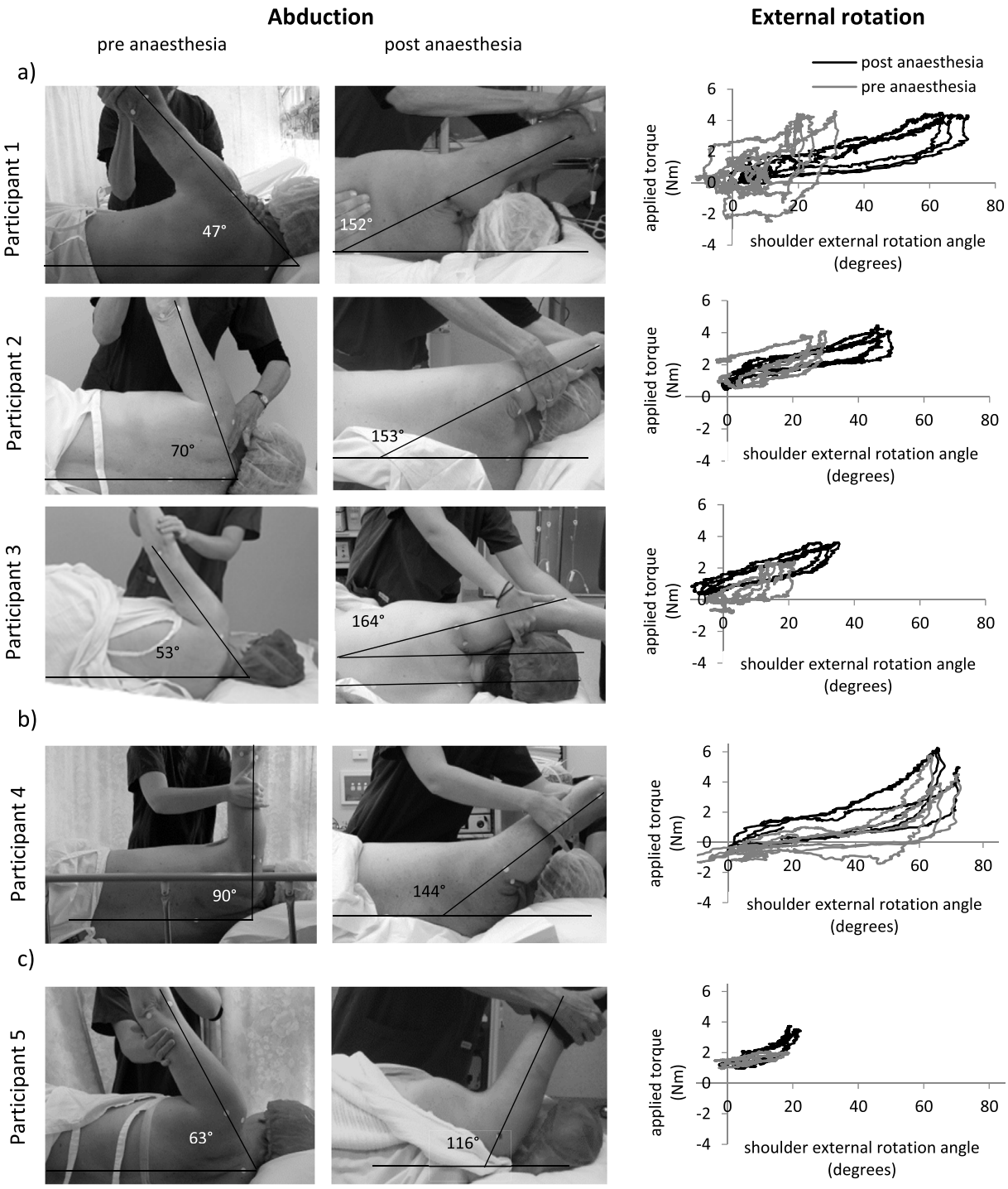

Fig. 1. Passive abduction and external rotation range of motion pre and post general anaesthetic for the a) subjects that exhibited significantly greater passive abduction and external rotation ROM under general anaesthesia compared to awake. b) subject that exhibited significantly greater passive abduction ROM under general anaesthesia compared to awake but demonstrated normal external rotation ROM under both conditions. c) subject that exhibited significant glenohumeral stiffness awake and under general anaesthesia.

which one is external rotation (Buchbinder et al., 2004).

The pathoanatomy of frozen shoulder is not fully understood. Histological and arthroscopic studies of frozen shoulder suggest chronic inflammation, fibrosis and glenohumeral joint capsule contracture is responsible for the pain and restricted range of movement (ROM) (Ryan et al., 2016). Consequently, treatment is most commonly aimed at lengthening glenohumeral joint structures to restore shoulder ROM while managing pain.

Frozen shoulder is considered notoriously difficult to treat and there is no consensus regarding optimal management (Lewis, 2015). Evidence suggests that corticosteroid injection confers significant short term benefit (Buchbinder et al., 2003), however, there is little evidence to support the effectiveness of treatments aimed at lengthening the glenohumeral joint capsule. Physiotherapy aimed at increasing ROM is only slightly more effective than placebo injection in the short term (Carette et al., 2003); efficacy of arthroscopic capsular release is not supported by evidence from randomised control trials (Lewis, 2015); and capsular hydrodilation or distension is no more effective than

corticosteroid injection alone (Tveita et al., 2008).

Perhaps current treatment approaches are ineffective because they target the wrong structures? In other painful musculoskeletal conditions such as back and neck pain ROM is often restricted by muscle guarding (spasm). It may be that restricted shoulder ROM in people with frozen shoulder is due to muscle contraction in response to underlying pathophysiology or to cognitive or emotional factors such as fear of pain or anxiety. The fact that the glenohumeral joint capsule is strongly reinforced by the rotator cuff tendons makes this hypothesis plausible from an anatomical perspective. Contraction of the anterior rotator cuff (subscapularis), an internal rotator of the shoulder, could feasibly restrict shoulder external rotation range of motion, one of the crucial clinical findings required to make a diagnosis of frozen shoulder. Given that the humerus must externally rotate during shoulder elevation muscle guarding to prevent this movement could also be associated with restricted shoulder abduction & flexion range. This could also explain why shoulder ROM improves spontaneously in patients suffering from frozen shoulder as pain and/or anxiety subsides.

The aim of this case series, was to investigate a muscle guarding component to movement restriction in patients with frozen shoulder. To do so, we compared passive shoulder ROM before and after the administration of general anaesthesia.

2. Methods

2.1. Participants

Patients scheduled to undergo capsular release surgery for frozen shoulder were informed about the study. The indication for surgery was a clinical impression of capsular tightness by the participating orthopaedic surgeon. Surgery was only offered if osteoarthritis, significant rotator cuff disease and shoulder region fractures or dislocations had been ruled out by X-ray and MRI, and if physical examination performed in standing indicated global restriction of all active and passive shoulder movements greater than 50% normal ROM. Patients were excluded if they had a history of trauma to the affected shoulder within the four weeks prior to surgery, previous surgery on the affected shoulder, shoulder pain referred from the vertebral column (defined as shoulder pain exacerbated during neck movement and/or palpation of the cervicothoracic vertebral column) or concurrent inflammatory or neurological disease involving the affected shoulder.

2.2. Outcome measurements

To minimise the time participants spent under general anaesthetic prior to surgery, only two passive shoulder ROM outcome measurements were compared before and after general anaesthesia: external rotation ROM because passive restriction in this direction is a diagnostic criterion for frozen shoulder (Bunker, 1997); and abduction ROM because it could readily be measured in the side-lying position required by the orthopaedic surgeon. A portable custom-built arm frame instrumented with a potentiometer (Vishay Model 357, Germany) and a force transducer (XTran, Model S1W 250N, Applied Measurement PTY. LTD. Australia) was constructed to record the torque applied to each participant's arm to achieve maximum passive external rotation ROM. The participant's arm was strapped into the arm frame which maintained the elbow in 90 degrees of flexion and shoulder in 45° abduction. Both shoulder external rotation angle and torque applied were recorded at a sample rate of 100 Hz using a 32-bit analogue to digital converter (cDAC 9171, National Instruments, TX, USA) and LABVIEW software. Abduction ROM was measured by passively moving the participant's arm through range. Maximal achieved abduction ROM was measured from a digital photograph by measuring the angle at the intersection of a horizontal reference line and a line between the acromion process and the lateral epicondyle. (Fig. 1).

2.3. Procedure

On the day of the scheduled surgery, researchers met potentially eligible patients and described the aim and design of the study. For consenting patients, active and passive shoulder flexion, abduction, external rotation with arm by the side and hand-behind-back were assessed on the affected shoulder to confirm eligibility. As is common clinical practice, these movements were performed with the patient standing and measured with a goniometer (adduction, flexion, external rotation) or tape measure (hand-behind-back). Following confirmation that inclusion and exclusion criteria were met, participants signed a consent form and demographic data were collected, including age, handedness and duration of symptoms. To determine the impact of frozen shoulder on their quality of life, participants completed the Shoulder Pain and Disability Index (SPADI).

With the participant in side-lying, passive shoulder abduction ROM was measured. This was followed by measurement of passive external rotation; three repetitions of passive external rotation ROM were averaged for analysis. Each participant was asked to relax their affected shoulder while the investigator slowly moved the arm through available range. Movement was stopped when resistance to the movement was felt or severe pain prevented further movement.

Each participant then entered the operating theatre and general anaesthesia was administered. The researchers then entered the operating theatre and repeated the passive shoulder ROM measures. For external rotation ROM measurements which were not limited by severe pain pre-anaesthesia, a torque similar to that achieved during the pre-anaesthetic condition was applied. For participants who experienced severe pain which limited external rotation ROM pre-anaesthesia, torque was applied gradually until resistance to movement was felt. Following completion of passive external rotation and abduction ROM measurements, the participant was left under the care of the treating shoulder surgeon.

3. Results

Three females and two males were included. Participants ranged from 51 to 64 years of age, with symptoms ranging from 6 to 30 months. None of the participants were diabetic and the non-dominant shoulder was affected in three participants. SPADI scores ranged from 67 to 87, indicating moderate to high levels of pain and disability.

Passive abduction ROM increased following anaesthesia in all participants, with increases ranging from 53° to 111° (Table 1, Fig. 1). Four participants demonstrated passive external rotation ROM restriction pre-anaesthesia when measured in the arm frame in the side-lying position at 45° abduction. This ROM increased in three participants by 15°–41° (Table 1, Fig. 1a) and was substantially unchanged in one participant (Table 1, Fig. 1c) following anaesthesia under similar force levels. One participant, who satisfied the inclusion criterion of severely restricted passive external rotation ROM while standing with the arm by the side, demonstrated passive external rotation ROM within normal limits (68°) when measured in side-lying pre-anaesthesia. This ROM was very similar (69°) following general anaesthesia when the same force was applied (Table 1, Fig. 1b).

4. Discussion

This is the first study to report passive shoulder ROM in patients diagnosed with frozen shoulder measured without the confounding variables of pain and muscle contraction. The five participants demonstrated increases of 53°–111° in passive shoulder abduction ROM following anaesthesia representing increases of between 60% and 223% (Table 1; Fig. 1). These large increases in abduction ROM suggest that muscle guarding is likely a significant contributor to abduction ROM restriction in some patients with frozen shoulder.

Passive shoulder external rotation ROM improvement in this study

Table 1
Demographic and outcome measurement data for all participants.

	Participant 1	Participant 2	Participant 3	Participant 4	Participant 5
age	64	53	51	59	57
gender	male	female	female	male	female
side affected	dominant	non-dominant	non-dominant	non-dominant	dominant
duration of symptoms (months)	9	18	6	6	30
pain & functional limitation (SPADI score)	67	87	79	67	76
passive abduction ROM					
pre anaesthesia	47°	70°	53°	90°	63°
post anaesthesia	152°	153°	164°	144°	116°
ROM increase (%)	105° (223%)	83° (119%)	111° (209%)	54° (60%)	53° (84%)
passive external rotation ROM					
pre anaesthesia	26.3°	28.3°	17.3°	68.0°	17.0°
post anaesthesia	67.7°	48.3°	32.7°	69.0°	20.0°
ROM increase (%)	41.4° (156%)	20° (71%)	15.4° (89%)	1.0° (< 1%)	3.0° (18%)

also points to a role for muscle contraction in movement restriction in some patients diagnosed with frozen shoulder. Three of the five participants demonstrated large increases in ROM under anaesthesia (Table 1, Fig. 1a). For participants 1 and 2 passive external rotation ROM increased by 41° and 20° respectively at approximately the same external rotation torque applied pre-anaesthesia. For participant 3, whose pre-anaesthesia passive external rotation ROM was limited by severe pain, external rotation ROM increased by 15° when torque was applied at similar levels as for participants 1 and 2 (Table 1, Fig. 1b).

For the remaining two participants, passive shoulder external ROM did not increase substantially under anaesthesia. Participant 4 was included in the study because he satisfied the inclusion criterion of significantly restricted passive external rotation ROM when measured in standing with the arm by the side. However, when measured in the side-lying position using the arm frame prior to anaesthesia, passive external rotation range was 68° which is within normal range. This range was unchanged (69°) under general anaesthesia (Table 1, Fig. 1b). Reduced muscle guarding when the participant's arm was supported and relaxed in the arm frame in the side-lying position could explain this result.

Participant 5 had limited passive external shoulder rotation pre-anaesthesia which showed little change when measured under anaesthesia. This participant, who had the longest duration of symptoms (30 months), recorded a passive external rotation ROM pre-anaesthesia of 17° which was limited by severe pain. During anaesthesia and under applied torques similar to those applied to participants 1, 2 and 3 external rotation ROM was substantially unchanged at 20° This finding suggests that shortened connective tissue structures may be the cause of the movement restriction in this patient. It is possible that tight connective tissues in this patient are a function of prolonged restriction of movement, although further evidence is needed to confirm this hypothesis.

While this study was not designed to answer the question of why this group of patients might have muscle guarding that results in such severe functional limitations, it is possible to speculate. Muscle guarding in patients suffering from frozen shoulder could be a protective mechanism or in response to underlying pathophysiology. Altered muscle activity has been observed in other painful musculoskeletal conditions, such as back pain, although physiological mechanisms are not clear (van Dieën et al., 2003). There may also be cognitive or emotional factors that result in limitations to movement such as fear (of movement or pain), anxiety or catastrophic appraisal of the condition. These psychologically-mediated factors are thought to influence behaviour in other painful conditions. Studies involving much larger groups of frozen shoulder patients are needed to confirm the veracity of these findings.

Accurate assessment of passive shoulder ROM is essential to determine the contribution of tight connective tissue structures to shoulder ROM deficits, as it is the critical criterion for frozen shoulder diagnosis and guides treatment decisions. If we accept that ROM measurements under anaesthesia reflect true passive ROM, this study suggests that the accuracy of assessment of passive shoulder ROM in the presence of pain is poor. Importantly, this draws into question the validity of physical assessment procedures that define the diagnosis of frozen shoulder. It is noted that this study was not designed to robustly test the validity of passive ROM assessment, but the findings demonstrate the critical importance of making a distinction between active and passive ROM restrictions.

Conclusions from this study need to be considered in light of several limitations. Most obviously, results from the five participants undergoing surgery for the treatment of their frozen shoulder may not generalise to the general frozen shoulder population. In addition, the shoulder abduction ROM measurements were performed without stabilising the scapula and so may not accurately reflect glenohumeral joint movement. It is possible that abduction ROM increases following anaesthesia could have been due to increased scapular movement, although this is unlikely to explain the one to twofold increases to virtually full range demonstrated by some participants.

This study has demonstrated that muscle guarding may be a significant contributing factor to shoulder movement deficits in some patients suffering from frozen shoulder. Given that many contemporary treatment options rest on the assumption that restriction is due to tightening of passive structures, this finding is worthy of further investigation.

Key points

Findings: Immediately following anaesthesia to eliminate the influence of pain and muscle contraction on shoulder function, passive shoulder abduction and external rotation ROM increased by approximately 150%–300% in some patients with the classical clinical features of idiopathic frozen shoulder.

Implications: In patients diagnosed with frozen shoulder large percentage increases in shoulder ROM following anaesthesia indicate that muscle guarding, and not capsular contracture, may be the major contributing factor to movement restriction in some of these patients.

Caution: Increases in passive shoulder ROM following anaesthesia have only been demonstrated in a very small number of frozen shoulder patients. Further studies concentrating on glenohumeral joint ROM in a larger group of frozen shoulder patients is required to confirm the veracity of these findings.

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Ethical approval

Ethical approval was obtained from the University of Wollongong and Illawarra Shoalhaven Local Health District Health and Medical Human Research Ethics Committee (Ethics No: HE12/434) and The University of Sydney Human Research Ethics Committee (Project No. 2014/754).

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Contributors

L Hollmann, M Halaki, M Haber & KA Ginn contributed to the design of the study and data collection and all authors provided critical input during manuscript drafting and approved the final version of the manuscript for publication.

Transparency declaration

The corresponding author (KAG) affirms that this manuscript is an honest, accurate, and transparent account of the study being reported, that no important aspects of the study have been omitted.

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