Musculoskeletal Science and Practice 37 (2018) 64–68

Contents lists available at ScienceDirect

Musculoskeletal Science and Practice

journal homepage: www.elsevier.com/locate/msksp

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

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ARTICLE INFO

Keywords:
Adhesive capsulitis
Anaesthesia
Passive range of motion

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

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https://doi.org/10.1016/j.msksp.2018.07.001
Received 25 March 2018; Received in revised form 2 July 2018; Accepted 4 July 2018

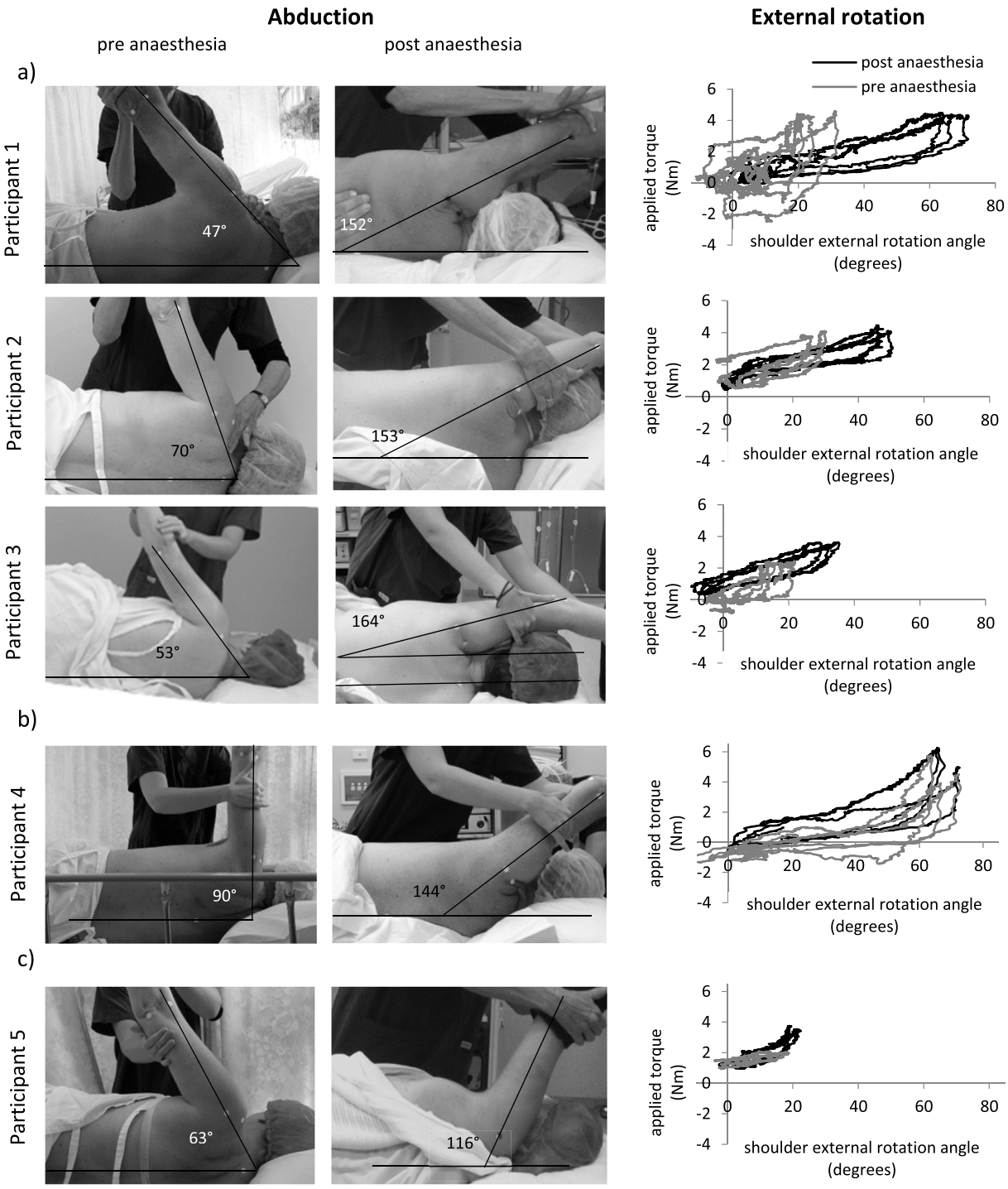

Fig. 1. Passive abduction and external rotation range of motion pre and post general anaesthetic for the a) subjects that exhibited significantly greater passive abduction and external rotation ROM under general anaesthesia compared to awake. b) subject that exhibited significantly greater passive abduction 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