



Movement and pain patterns in early stage primary/idiopathic adhesive capsulitis: a factor analysis

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Abstract

Objectives To evaluate patients clinically diagnosed with early stage primary/idiopathic adhesive capsulitis to determine the existence of any pattern of movement loss and associated pain that may facilitate early recognition.

Design Cross-sectional study.

Setting Private upper limb specialty clinic, Newcastle, Australia.

Participants Fifty-two patients clinically diagnosed with early stage adhesive capsulitis by a medical practitioner or physiotherapist.

Main outcome measures Percentage loss of active and passive ranges of eight shoulder movements and the pain level at the end of each movement. The reason for limitation of movement was also recorded.

Results Factor analysis clearly identified two groups for percentage loss of active range of movement. Notably external rotation movements grouped separately from other movements. A single group emerged for percentage loss of passive range of movement suggesting a non-specific global loss. For both pain at the end of active range of movement and passive range of movement two groups emerged, however the delineation between the groups was less clear than for percentage loss of active range of movement suggesting a pattern of end range pain may be less useful in identifying patients in this stage.

Conclusions External rotation movements in neutral and abduction generally group together and behave differently to other shoulder movements in patients clinically diagnosed with early stage primary/idiopathic adhesive capsulitis. In particular external rotation in abduction has emerged as the most painfully limited movement in this sample. This study provides preliminary evidence of patterns of range of movement and end range pain that require testing in a population of mixed shoulder diagnoses to determine their diagnostic utility for early stage adhesive capsulitis.

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Keywords: Shoulder patterns; Shoulder pain; Adhesive capsulitis; Range of motion; Factor analysis

Introduction

Adhesive capsulitis is a shoulder disorder that is recognised as consisting of three stages and reported to last from one to three years [1]. The disorder is described as either primary or idiopathic when the onset is insidious, and secondary when a known event precedes the onset [2]. Adhesive

capsulitis has a number of reported associations that include, but are not limited to, diabetes [3], Dupuytren's disease [4] and thyroid dysfunction [5], as well as a reported higher incidence in females [6]. The first or early stage is generally agreed to last up to nine months [7] and is typically characterised by pain rather than marked loss of movement [2]. Whilst adhesive capsulitis is usually recognisable in the later stages due to distinct restriction of both active and passive ranges of movement [8], it is considered difficult to identify and differentiate from other shoulder disorders in its early stage [9].

Routine assessment of patients with musculoskeletal disorders generally includes measurement of both active and

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passive ranges of movement, as well as any pain associated with each movement. Patterns of movement deficit and the behaviour of pain often assist in diagnosis [10]. As a means of differentiating joint capsular pathology from other causes of symptoms, James Cyriax described what is called the ‘capsular pattern’ [11]. This capsular pattern suggests a fixed proportion of movement loss is present and that each joint has a characteristic pattern [11]. The pattern for the glenohumeral joint proposed by Cyriax is that the proportional passive loss of external rotation will be greater than the proportional loss of abduction, which will be greater than the proportional loss of internal rotation. Although the literature on adhesive capsulitis frequently acknowledges this ‘capsular pattern’ [1,12], recent studies have demonstrated that it may not be consistently present [13–15]. Notably, however, these studies have involved populations in the latter stages of the disorder. No studies have examined the presence of the ‘capsular pattern’, nor any other recognisable pattern of movement loss in the early stage of adhesive capsulitis.

Recent research into the pathology of adhesive capsulitis has identified that initial inflammation of the glenohumeral joint capsule is followed by fibrosis and contracture [16]. This understanding of the pathology provides an explanation for the temporal behaviour of the symptoms, which are reported to initially manifest with pain followed by subsequent progressive movement restriction [17]. Surgical and radiological investigations have identified that anterior structures of the glenohumeral joint are predominantly affected [18,19], which may help explain the observed pattern of movement loss or pain reported in adhesive capsulitis, notably in external rotation [20]. However, the contribution of other active and passive shoulder movements to diagnosis have not been similarly considered.

As well as the lack of investigation of any pattern of either active or passive movement loss in early stage adhesive capsulitis, any associated pain pattern has also not been described to date. As pain is reported to be a key component of the early stage, it would therefore be potentially valuable to evaluate any contribution it may make to the clinical presentation of this disorder.

It has been suggested that treatment in the early stage of adhesive capsulitis may reduce the overall morbidity of the disorder [17]. The mixed results of treatment reported however, have been suggested to be at least partially as a result of the inability to define or classify sub-groups of patients likely to respond to physiotherapy and other interventions [21]. Although a set of clinical identifiers that may assist diagnosis in the early stage have been proposed, including global loss of active and passive ranges of movement and pain at the end-range in all directions, they have yet to be validated [22]. The recognition of any pattern of movement restriction or pain that may assist early diagnosis or identify sub-groups of patients would therefore be valuable. The overall aim of this study was to evaluate patients with a clinical diagnosis of early stage adhesive capsulitis to determine if it was possible to identify a pattern of movement loss and/or associated end

range pain that may facilitate recognition of this diagnostically challenging stage of the disorder. The findings of this preliminary study will enable future studies of mixed diagnosis populations to determine whether any patterns that may emerge are unique to the early stage of primary/idiopathic adhesive capsulitis.

Materials and methods

Participants

Fifty-two participants attending an upper limb specialty clinic diagnosed with early stage adhesive capsulitis on the basis of clinical presentation by various health care practitioners, including orthopaedic surgeons, a shoulder physician, general practitioners and physiotherapists were included in the study. In the absence of any validated criteria for the diagnosis of early stage primary/idiopathic adhesive capsulitis the clinical decision of the referring practitioner was considered pragmatically appropriate. Participants were required to have had symptoms for less than nine months, consistent with the reported duration of the early stage of the disorder [7]. As primary/idiopathic adhesive capsulitis was being investigated, patients with a history of major trauma or surgery of the shoulder were excluded. Potential participants were also required to have had recent shoulder X-rays and ultrasound examinations which did not demonstrate potential alternate diagnoses. Further exclusion criteria included a diagnosis of any systemic inflammatory joint disease, as well as neurological or current cervical spine disorders. Glenohumeral joint injection in the preceding six weeks was also an exclusion criterion.

Procedure

Each participant underwent routine clinical examination including measurement of active and passive shoulder ranges of movement. These included total shoulder flexion (TSF) and abduction (TSA), glenohumeral joint flexion (GHF) and abduction (GHA), and external rotation in neutral (ERN), together with external and internal rotation in 90° abduction (ERA and IRA respectively). Hand behind back (HBB) range was also measured. Measurement was performed by one of the researchers, an experienced musculoskeletal physiotherapist, using a Baseline digital inclinometer (Fabrication Enterprises Incorporated, Irvington, NY, USA) for all movements with the exception of HBB which was measured with a tape measure. Digital inclinometry has been demonstrated to have a measurement error of $\pm 1^\circ$ [23]. The range of movement was recorded in degrees for all movements other than HBB which was recorded in millimetres.

Measurement of shoulder ranges of movement was based on the method described by Green *et al.* [24]. The following movements were performed in sitting: TSF, GHF, TSA, and GHA. The starting position for these movements was with

the palm of the hand facing medially. The inclinometer was held on the mid shaft of the humerus by the researcher and the participant maintained an extended elbow [24]. In order to stabilise the scapula and isolate the glenohumeral joint for GHF and GHA, a device was developed that provided an arm that rested on the acromion, preventing upward rotation of the scapula (Supplementary Fig. 1).

The following movements were performed in the supine lying position:

- ERN: The shaft of the humerus was placed beside the participant's trunk in 0° of abduction and rotation. A towel was placed under the humerus to ensure it rested parallel to the plinth. The elbow was flexed to 90° and the forearm was in neutral rotation. The inclinometer was placed on the dorsal surface of the participant's forearm.
- ERA: The arm was abducted to 90° where possible, or if not possible due to either movement restriction or pain, abduction was taken to the limit of movement. The position of the humerus and placement of the inclinometer was the same as measurement of ERN.
- IRA: The arm was placed as described for ERA and internally rotated until either the posterolateral acromion was visualised to rise off the plinth [25], or the movement was limited by pain.

HBB was measured in standing as the distance between the spinous process of T1 and the radial styloid process. This has been demonstrated to have excellent intrarater reliability [26].

In order not to aggravate the participant's pain, each movement was performed only once. All active movements were performed prior to passive movements and in the same sequence for each participant. The order of measurement was: TSF, GHF, TSA, GHA, ERN, ERA, IRA, HBB. Active range of movement was performed by asking the participant to move their arm in the required direction until it was not possible to move any further or the pain became intolerable. Similarly, passive range of movement was performed by the researcher to the point of resistance limitation or when the participant reported the pain was intolerable. The limiting factor to movement was recorded simply as pain or inability to move for active movements and resistance or pain for passive movements. Regardless of the cause of limitation, each participant scored their level of pain at the end of each movement on a 100 mm visual analogue scale.

Statistical analysis

The data were analysed initially using descriptive statistics. The affected shoulder's percentage of movement of the unaffected shoulder was calculated for each of the eight active and eight passive movements.

For all movements with the exception of HBB:

$$\frac{\text{unaffected shoulder range of movement} - \text{affected shoulder range of movement}}{\text{unaffected shoulder range of movement}}$$

Table 1
Demographic characteristics of the participants (n = 52).

Characteristic	
Age (yrs), mean (SD)	55.2 (6.9)
Duration of symptoms (months), mean (SD)	5.5 (1.9)
Gender (% female)	51.9
Dominance (% right)	84.6
History of diabetes (%)	9.6
History of Dupuytren's disease (%)	13.5

For HBB:

$$\frac{d1 \text{ affected shoulder} - d1 \text{ unaffected shoulder}}{d1 \text{ unaffected shoulder}}$$

(d1 = distance between T1 spinous process and radial styloid process).

Factor analysis was then used to determine if it was possible to identify any relationships between the ranges of movement loss and similarly the pain behaviour at the end of each of the ranges of movement. Any such relationships, or movements grouping together, may denote the formation of patterns. Exploratory factor analysis was performed using the principal components method for extraction of factors followed by Varimax rotation. A combination of an eigenvalue of >1.00 and inspection of the scree plot was used to determine the optimum number of factors within each range of movement or pain score. Item loadings of ≥ 0.60 were considered to contribute strongly to that factor. Factors with four or more variables ≥ 0.60 were considered strong factors. All statistical analyses were performed using JMP 9.0 (SAS Institute Inc., Cary, NC, USA).

Results

Demographic characteristics of the participants are presented in Table 1. The mean (SD) shoulder ranges of active and passive movement (affected and unaffected), percentage loss of range of movement and pain scores at the end of range of movement are reported in Tables 2A and 2B.

Percentage loss of movement

Active range of movement

The mean percentage loss of active range of movement ranged between 68% (HBB) and 26% (GHF).

Two factors were extracted which accounted for 68% of the variance of the eight measured ranges of active movement (Table 3). These two factors represented a pattern comprising two groups of movements. The first group of movements (movement group 1), accounting for 52% of the variance

Table 2A

Mean (SD) shoulder ranges of active movement (unaffected and affected), percentage loss of active ranges of movement and pain scores at the end of range of each movement.

Movement	Unaffected shoulder ROM (°), Mean (SD)	Affected shoulder ROM (°), Mean (SD)	% loss ROM, Mean (SD)	Pain score end of range (mm), Mean (SD)
Total shoulder flexion	161.9 (12.8)	116.4 (22.8)	28 (13)	62 (25)
Glenohumeral joint flexion	126.8 (12.8)	93.6 (18.2)	26 (14)	50 (28)
Total shoulder abduction	146.0 (16.4)	81.4 (28.3)	46 (18)	69 (25)
Glenohumeral joint abduction	114.9 (21.0)	55.6 (23.2)	52 (18)	59 (28)
External rotation in neutral	67.3 (9.9)	38.5 (14.6)	42 (21)	57 (30)
External rotation in abduction	83.2 (12.9)	36.0 (17.6)	57 (20)	71 (22)
Internal rotation in abduction	77.1 (9.1)	51.7 (14.6)	33 (19)	45 (29)
Hand behind back (mm)	28.3 (5.3)	46.4 (9.4)	68 (43)	6 (28)

Table 2B

Mean (SD) shoulder ranges of passive movement (unaffected and affected), percentage loss of passive ranges of movement and pain scores at the end of range of each movement.

Movement	Unaffected shoulder ROM (°), Mean (SD)	Affected shoulder ROM (°), Mean (SD)	% loss ROM, Mean (SD)	Pain score end of range (mm), Mean (SD)
Total shoulder flexion	170.4 (9.4)	129.7 (21.1)	24 (11)	63 (25)
Glenohumeral joint flexion	132.3 (11.1)	105.7 (18.4)	20 (12)	48 (31)
Total shoulder abduction	153.9 (14.4)	97.0 (25.0)	37 (16)	63 (29)
Glenohumeral joint abduction	118.8 (14.0)	72.8 (19.8)	39 (16)	64 (23)
External rotation in neutral	73.2 (9.6)	42.3 (16.8)	42 (21)	68 (24)
External rotation in abduction	92.4 (12.8)	38.9 (16.0)	58(17)	77 (18)
Internal rotation in abduction	84.1 (8.8)	55.8 (15.7)	34 (18)	45 (29)
Hand behind back (mm)	24.7 (4.3)	42.2 (9.0)	72 (36)	71 (22)

ROM, range of movement.

Table 3

Factor loadings for the factor models for the percentage loss of active and passive ranges of movement.

Movement	Active		Passive
	Factor 1: movement group 1 (eigenvalue = 4.13)	Factor 2: movement group 2 (eigenvalue = 1.31)	Factor 1: global loss of movement (eigenvalue = 4.76)
Total shoulder flexion	0.90 ^a	0.08	0.85 ^a
Glenohumeral joint flexion	0.83 ^a	0.15	0.83 ^a
Total shoulder abduction	0.73 ^a	0.17	0.87 ^a
Glenohumeral joint abduction	0.75 ^a	0.35	0.84 ^a
External rotation in neutral	0.15	0.66 ^a	0.51
External rotation in abduction	0.25	0.97 ^a	0.58
Internal rotation in abduction	0.48	0.18	0.62 ^a
Hand behind back	0.55	0.22	0.68 ^a

^a Loadings ≥ 0.60 .

included TSF, GHF, TSA and GHA. The second group of movements (movement group 2), accounting for 16% of the variance included ERN and ERA. The loadings of the eight movements on the two factors are shown in Table 3.

Passive range of movement

The mean percentage loss of passive range of movement ranged between 72% (HBB) and 20% (GHF).

A single factor with an eigenvalue of 4.76 was extracted for the measured ranges of passive movement which accounted for 60% of the variance suggesting a global loss of passive range of movement rather than an identifiable pattern. Six of the eight loadings (TSF, GHF, TSA, GHA, IRA, HBB) were >0.6 (range 0.62 to 0.87). The loadings of the eight movements are shown in Table 3.

Pain at the end of range of movement

Active range of movement

The active range of movement scoring the highest mean (SD) pain score for all participants was ERA (71 mm (22)).

A two factor structure accounted for 66% of the variance of the pain scores at the end of active range of movement. These two factors represented a pattern of two groups of movements. The relative weights of the eight movements are shown in Table 4, which provides factor loadings for each of the ranges of active movement in the two-factor solution. The first group of movements (movement group 1), accounting for 53% of the variance included TSF, TSA and GHA. The second group of movements (movement group 2), accounting for 13% of the variance included ERA and IRA.

Table 4

Factor loadings for two factor models for pain at the end of active and passive ranges of movement.

Movement	Active		Passive	
	Factor 1: movement group 1 (eigenvalue = 4.20)	Factor 2: movement group 2 (eigenvalue = 1.06)	Factor 1: movement group 1 (eigenvalue = 4.60)	Factor 2: movement group 2 (eigenvalue = 1.01)
Total shoulder flexion	0.71 ^a	0.23	0.76 ^a	0.21
Glenohumeral joint flexion	0.50	0.33	0.51	0.24
Total shoulder abduction	0.86 ^a	0.22	0.78 ^a	0.26
Glenohumeral joint abduction	0.70 ^a	0.39	0.72 ^a	0.46
External rotation in neutral	0.47	0.54	0.22	0.98 ^a
External rotation in abduction	0.22	0.73 ^a	0.41	0.72 ^a
Internal rotation in abduction	0.21	0.67 ^a	0.32	0.53
Hand behind back	0.36	0.58	0.60 ^a	0.44

^a Loadings ≥ 0.60 .

Table 5

Reason for limitation of movement.

Movement	Active		Passive	
	Pain limited, <i>N</i> (mean % loss ROM)	Movement limited, <i>N</i> (mean % loss ROM)	Pain limited, <i>N</i> (mean % loss ROM)	Resistance limited, <i>N</i> (mean % loss ROM)
Total shoulder flexion	26 (28)	26 (28)	45 (23)	7 (28)
Glenohumeral joint flexion	18 (25)	34 (26)	29 (22)	23 (18)
Total shoulder abduction	30 (49)	22 (38)	37 (40)	15 (29)
Glenohumeral joint abduction	26 (55)	26 (48)	42 (39)	10 (37)
External rotation in neutral	30 (42)	22 (42)	44 (45)	8 (30)
External rotation in abduction	37 (55)	15 (62)	49 (58)	3 (50)
Internal rotation in abduction	19 (33)	33 (32)	24 (31)	28 (36)
Hand behind back	34 (60)	18 (84)	48 (74)	4 (53)

Passive range of movement

The passive range of movement scoring the highest mean (SD) pain score for all participants was ERA (77 mm (18)).

A two factor structure accounted for 70% of the variance for pain scores at the end of passive range of movement. These two factors suggested a pattern of two groups of movements. The relative weights of the eight movements are shown in Table 4, which provides factor loadings for each of the ranges of passive movement in the two-factor solution. The first group of movements (movement group 1), accounting for 58% of the variance included TSF, TSA, GHA and HBB. The second group of movements (movement group 2), accounting for 13% of the variance included ERN and ERA.

The factor loading plots for percentage loss of active range of movement, and for the pain level scores at the end of each of the active and passive ranges of movement are presented in Supplementary Fig. 2. These plots demonstrate that only percentage loss of active movement resulted in a clear separation of the two groups of movements (ERN and ERA with the other group of movements comprising TSF, GHF, TSA and GHA) (Supplementary Fig. 2A). Similar separation is not observed for pain at the end of both active and passive movements (Supplementary Fig. 2B and C) suggesting a recognisable pattern for pain at the end of range did not emerge.

Limitation to movement

Descriptive statistics describing the reason for limitation to movement are presented in Table 5. The movement

most frequently limited by pain, rather than an active inability to move or passive resistance was ERA for both active (71%) and passive (94%) ranges of shoulder movement. The movement least frequently limited by pain was GHF (35%) for active movement and IRA (46%) for passive movements.

Discussion

This is the first study to investigate the presence of any recognisable pattern of movement loss that may exist in a group of participants clinically diagnosed with early stage primary/idiopathic adhesive capsulitis. Unlike earlier studies, this study has utilised factor analysis to determine relationships or patterns that may exist within the percentage loss of both active and passive ranges of movement and pain experienced at the end of each range of movement. It is also unique as it has considered the reason for limitation to movement in a larger sample than previously reported. The results of this study have demonstrated that in this group of patients diagnosed clinically with early stage primary/idiopathic adhesive capsulitis, the percentage loss of both active and passive ranges of movement does not fit the ‘capsular pattern’ previously reported by Cyriax to be characteristic of this disorder [1,12]. The selection of factor analysis has enabled the detection of groups, rather than isolated shoulder movements that may involve common anatomical,

pathological or biomechanical characteristics. In this study the movements that have grouped together as a result of the factor analysis may be reflecting the underlying pathological process in the glenohumeral joint capsule. In particular, the grouping together of the two external rotation movements may indicate an area of capsular involvement leading to restriction or pain different from the other measured shoulder movements.

The clearest pattern to emerge from this study was from the analysis of the percentage loss of active range of movement which identified a pattern with two distinct groups (Table 3 and Supplementary Fig. 2A). One group included the shoulder movements TSF, GHF, TSA and GHA, whilst the other comprised the two measured external rotation movements (ERN and ERA). The two groups of movements show a degree of correlation with each other and this is demonstrated by the acute angle between each of the groups of variables in Supplementary Fig. 2A. The two external rotation movements are not completely independent from the other group of movements suggesting there is a small amount of similarity between the two. Although perhaps not surprising, external rotation in both neutral and in abduction appeared to behave differently from the other measured shoulder movements. However the classic ‘capsular pattern’ of proportional loss of external rotation being greater than the proportional loss of abduction, which in turn is greater than the proportional loss of internal rotation, did not emerge. Although not entirely consistent with ‘the capsular’ pattern previously described for loss of passive range of movement [11], this is in accordance with the reported pathological involvement of the anterior glenohumeral structures in adhesive capsulitis and the previously recognised involvement of external rotation [27].

Percentage loss of passive range of movement grouped differently to active movement and demonstrated only one pattern of approximately equivalent loss across all movements (Table 3). Again the ‘capsular pattern’ did not emerge and in contrast to active movement, this would suggest a non-specific global loss of passive shoulder movement. Whilst not clearly emerging as a second group, ERN appeared least related to the other movements. Similarly an earlier study of passive range of movement loss in adhesive capsulitis, reported loss in all measured ranges, with no ‘capsular pattern’ evident in their sample of 30 participants [15]. That study measured abduction as well as internal and external rotation in 45° of abduction. They demonstrated that external rotation was significantly limited in comparison to abduction and internal rotation, with the latter two movements not differing from each other. Whilst direct comparison with the current study is problematic due to methodological differences the trend for global passive movement loss appears to be consistent with a greater loss in external rotation.

The early stage of adhesive capsulitis has been reported to be characterised by pain rather than movement restriction [2], and to our knowledge there are no other reported

studies that have quantified and analysed pain at the end of range of movement in this stage of the disorder. Pain at the end of active movement suggested two groups of movements (Table 4 and Supplementary Fig. 2B). The first group contained only three movements with loadings ≥ 0.60 , suggesting only a weak association. This group comprised the movements of TSF, TSA and GHA, while the second suggested a relationship between two of the rotational movements (ERA and IRA). Consideration of the descriptive data would suggest that when ERA recorded a high level of pain at the end of range, IRA conversely recorded a low level of pain. Interestingly, of the two groups that emerged in analysing pain at the end of passive range of movement (Table 4 and Supplementary Fig. 2C), the first contained HBB as well as TSF, TSA and GHA. While active HBB has been used clinically to assess shoulder internal rotation, it has been reported that it is not solely related to internal rotation at the glenohumeral joint [28]. This might help explain HBB grouping with the other movements. Notably the second factor again consisted of the two external rotation movements (ERN and ERA). Despite the presence of this grouping, inspection of the factor loading plots (Supplementary Fig. 2B and C) would suggest that a clear pattern did not emerge. This indicates that whilst pain reportedly is a feature of early adhesive capsulitis, the absence of a pattern may make this symptom less useful than percentage loss of active range of movement in identifying patients at this stage.

It would be reasonable to expect that the limitation to movement in early stage adhesive capsulitis may be more likely due to pain rather than resistance or weakness. Interestingly, for both active and passive movements, ERA and HBB were those movements most frequently limited by pain. ERA is reportedly limited by anterior capsular structures [29], which suggests those structures may be responsible for pain experienced with that movement. As pain not only from the capsule, but also from muscle spasm has been previously suggested as a reason for limitation of movement [14], it could potentially be that spasm from the scapulothoracic musculature is responsible for at least some of the pain limiting the HBB movement in these participants.

There are some limitations to this study. The sample size was modest although it compares favourably with earlier studies [13–15]. Interpretation of factor analysis with this sample has suggested findings that require confirmation with a larger sample. The participants in this sample were recruited from a limited number of practice environments and it is possible this may have led to biased estimates due to participants not being representative of other patient sources. The absence of a gold standard for diagnosis of adhesive capsulitis in its early stage remains a limitation in all related research. Heterogeneity of participants has previously been reported as a limitation of similar studies [14], however strict inclusion and exclusion criteria were applied in the current study to minimise participants with potentially alternate diagnoses. Although based on previously reported reliable measurement

methods, intrarater reliability was not specifically determined in this study due to the clinical nature of the research and the ethical requirement to minimise any worsening of each participant's pain. The order of testing was not randomised which may have resulted in greater pain scores for the later measured movements due to aggravation by earlier movements.

Conclusion

This study has specifically investigated patients clinically diagnosed with early stage primary/idiopathic adhesive capsulitis to determine whether any recognisable movement patterns may be present which could assist diagnosis. The main finding of the study was that active external rotation movements in both neutral and in abduction grouped together and behaved differently to the other measured active shoulder movements. Percentage loss of passive ranges of movement identified a non-specific global loss. Unlike the percentage loss of active range of movement, a clear pattern for pain at the end of range of movement did not emerge. Interestingly, ERA has emerged as both the most painful active and passive movement and the movement most frequently limited by pain, rather than weakness or resistance. Clinically this indicates the involvement of this movement in the early stage as has been previously recognised in the later stages, and suggests that careful assessment of movement range and pain at the end of range of external rotation in both neutral and 90° abduction should be undertaken in patients with suspected early stage adhesive capsulitis. Whilst percentage loss of active and passive ranges of movement, pain at the end of range of movement and limitation to movement have highlighted the involvement of external rotation, further studies are required to investigate the inter-relationships among these parameters. The findings of this preliminary study therefore, will direct future studies of mixed populations comprising patients with varying shoulder diagnoses, to test the patterns that have emerged, and determine if they are unique to the early stage of adhesive capsulitis.

Ethical approval: The Human Research Ethics Committee of The University of Newcastle (No. H-2009-0234).

Conflict of interest: None declared.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <http://dx.doi.org/10.1016/j.physio.2014.02.001>.

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