Physiotherapy assessment of shoulder stiffness and how it influences management

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Abstract

Common causes of shoulder stiffness include osteoarthritis, trauma, rheumatological conditions and stiffness secondary to soft tissue adaptation. Physiotherapy assessment of the stiff shoulder aims to ascertain the key causative factors of stiffness to inform effective management planning. Identification of whether a patient presents with pain or stiffness as their predominant symptom further guides treatment selection. The current evidence base underpins a management algorithm which has been developed to guide the assessment and management of patients presenting with shoulder stiffness.

Keywords

Bony anatomy, physiotherapy assessment; shoulder stiffness

Introduction

Shoulder stiffness at the glenohumeral joint can occur as a result of bony anatomy, the adaptation of soft tissues around the joint or a combination of both. The physiotherapy assessment of the stiff shoulder aims to determine the key drivers of stiffness in an effort to decide the most appropriate management strategies.

The most common cause of bony shoulder stiffness is osteoarthritis (OA) of the glenohumeral joint. On examination, this characteristically presents as stiffness with a hard end feel capsular pattern, although this is less marked in the early stages of the pathology. It is commonly described as a disease process of wear and tear but is better described as a dynamic process of changes in a joint followed by repair and remodelling.¹

OA involves an imbalance of normal and degenerative cartilage resulting in the net loss of cartilage and therefore joint space. Associated with these changes are hypertrophic structural changes leading to the formation of bone growths called osteophytes.² OA is confirmed radiologically by the four features: reduced joint space, osteophytes, subchondral bone sclerosis and subchondral bone cysts (Fig. 1).

Another cause of articular stiffness can be following trauma. This can result in damage and malalignment of the articular surfaces of the joint. Any articular causes of glenohumeral joint stiffness must be confirmed radiologically (Fig. 2).

Less commonly, shoulder stiffness can be also be caused by rheumatological conditions, such as rheumatoid arthritis or any of the spondyloarthropathies.

Shoulder stiffness secondary to soft tissue adaptation can be thought of in three types of conditions: adhesions secondary to trauma (including postoperative) resulting in contracture formation, poor muscle control and idiopathic frozen shoulder.

Soft tissue trauma to the shoulder region results in the formation of irregular and uneven collagen fibres causing fibrotic adhesions. Shoulder stiffness can be

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Figure 1. X-ray osteoarthritis shoulder.

Figure 2. X-ray glenohumeral joint with malunion proximal humerus.

caused by immobilization following an insult such as fracture. Stiffness can also result not only from pain associated with other shoulder structure pathology, but also in the presence of thyroid, cardiac or pulmonary disease, thoracic surgery, diabetes, neurological conditions and postmenopausal changes. Inactivity associated with psychological factors such as emotional stress and depression could also have an effect on shoulder stiffness.³

Soft tissue adaptation can occur with habitual poor posture and use. However, muscle tightness as a result of overactivity, rather than adaptive shortening, can also result in a perceived contracture.

Frozen shoulder can be a primary or idiopathic problem or may be secondary and associated with another systemic illness. A primary or 'true' frozen shoulder occurs where there is no exogenous cause or pre-existing condition. It presents an idiopathic decreased range of motion (ROM) in which no systemic diagnosis, precipitating shoulder condition or radiographic explanation can be found.⁴⁻⁶ Secondary frozen shoulders are defined as those with a known intrinsic or extrinsic precursor, typically causative of shoulder pain

and dysfunction that ultimately leads to global stiffness (e.g. diabetes, stroke, hypo- or hyperthyroidism, rotator cuff disease, cardiac disease) and in association with prolonged immobilization or trauma.⁷⁻⁹

Although the aetiology of frozen shoulder remains elusive, the understanding of its pathogenesis is increasing. Generally, three schools of thought have emerged. These are:

1. An inflammatory process¹⁰⁻¹²
2. A fibrotic process¹³⁻¹⁵
3. An inflammatory process with subsequent reactive capsular fibrosis¹⁶

Reeves¹⁷ first divided the clinical presentation of frozen shoulder into three stages in 1975, when he studied the natural history of 49 cases over 10 years (Table 1).

There is general agreement that the condition will pass through three stages¹⁷

- Stage I: involving pain
- Stage II: pain and restricted movement and finally
- Stage III: painless restriction

Neviaser and Neviaser⁴ have described an arthroscopic four stage classification (Table 2) and stressed the importance of an individual treatment plan based on an understanding of the clinical stages of the disease. More recently, Hannafin et al.¹² have described a correlation between the arthroscopic stage, the clinical examination and the histological appearance of the tissues.

Neviaser and Neviaser⁴ stressed that it is critical to remember that the stages referred to by the authors actually represent a continuum of disease rather than discrete well-defined stages.

More recently, Hanchard et al.¹⁸ recommend a simple 'pain-predominant' or 'stiffness-predominant' classification as a basis for decision-making in treatment selection. This is also supported by Cyriax,¹⁹ who suggests that there is a phase of increasing pain and stiffness, whereby pain is the predominant complaint until the pain abates leaving stiffness as the predominant complaint. Physiotherapy can be useful throughout all stages of a frozen shoulder; however, it has been found that intensive stretching into the painful ROM could aggravate the symptoms.²⁰ This may prolong the course of the disease and increase pain levels. Therefore, in the pain-predominant phase, physiotherapy treatment should focus on ROM exercises within comfort and pain relief modalities. Russell et al.²¹ identified the importance of the psychosocial support provided by physiotherapy intervention analysing the Hospital Anxiety and Depression Scale.

Table 1. Three stages of classification¹⁷

Stage I: Freezing stage
2.5 months to 3 months
The patient reports an insidious onset of diffuse shoulder pain, difficulty lying on the affected side and progressive loss of shoulder motion. Patients often identify pain at night. Ache is unrelated to activity and may be worse at rest
Stage II: Frozen stage
4 months to 12 months (approximation)
The pain gradually subsides and is described as a dull aching type of sensation and occurs at the extreme of their available movement. Loss of motion plateaus with passive motion equal to active motion or gross reduction of glenohumeral movements, with near total obliteration of external rotation (capsular pattern)
Stage III: Thawing stage
12 months to 42 months (approximation)
This is characterized by the gradual improvement of shoulder motion and the reduction of pain symptoms, mean duration from onset of frozen shoulder to resolution is over 30 months

Table 2. Four stages of classification⁴

Stage I 0 months to 3 months	Pre-adhesive stage. A fibrous synovial inflammatory reaction is detectable only by arthroscopy. The patients usually present with signs and symptoms of impingement syndrome. The main complaint is pain and minimum deficit in range of motion is detected
Stage II 4 months to 9 months	Adhesive stage. The acute synovial inflammation is apparent on physical inflammation. Arthroscopic findings demonstrated that the normal spacing between capsular fold, humeral head and biceps tendon, glenoid and humeral head diminish significantly. The patient experiences severe pain and loss of motion. (Neviaser and Neviaser ⁴ do not give this stage a name but compared to other classifications, it appears to be equivalent to the Adhesive stage)
Stage III 10 months to 15 months	Maturation stage. This stage is evident by the maturation of the inflammatory process. The dependant fold is only half its original size and adherences between various structures are formed
Stage IV 16 months to 24 months	Chronic stage. Capsular adhesions are fully mature and markedly restricted. Clinically, the shoulder is 'frozen'

Physiotherapy assessment

Subjective assessment

The aim of the subjective examination is to gather sufficient information about the patient's symptoms. This will allow a safe, effective and efficient objective examination. It also establishes whether the patient is within the pain or stiffness-predominant phase of a frozen shoulder.

Present condition/history of present condition

When assessing a stiff shoulder, it is imperative to establish the history of the onset, as well as the current symptoms and their duration, aiming to determine

the possible classification and diagnosis of frozen shoulder.

Subjective questioning should also clarify whether there is a known cause to the complaint and any associated factors that have contributed to the patient's condition. If precipitated by trauma, the incident may be minor and the initial pain usually settles; therefore, the patient may have difficulty recalling a traumatic incident or associating it with the onset of the shoulder pain.

Past medical history

It is essential to check for a history of trauma or episodes of ill health that the patient may have associated

Table 3. Specific shoulder masqueraders and red flags

Masqueraders	Red flags
Lungs	Neoplasm
Heart	Infective arthritis
Diaphragm	Osteoporosis
Gallbladder	Inflammatory joint disease
Spleen	Anticoagulant therapy
Cervical/upper thoracic spine	Steroid use
Thoracic outlet	Diabetes
	Trauma/fracture
	Autoimmune disease

with their shoulder. Awareness of other associated conditions is not only important for the safe management of the patient, but also may contribute to their symptoms for example diabetes. The physiotherapist ascertains the state of the patient's general health identifying possible 'masqueraders' (i.e. cardiac visceral pathologies) (Table 3) and possible red flags.

Special questions

The patient may identify precautions or contraindications to the objective examination and/or treatment.²² The importance of this information will establish limitations to treatment identifying benign musculoskeletal conditions that are suitable for manual therapy and differentiating from systemic, neoplastic or other non-muscular conditions, which are not suitable for physiotherapy treatment and require referral on for further investigation (Table 4).

Further investigations

It is important to establish whether the patient has undergone any recent investigation to complement the assessment assisting with the diagnosis. Routine radiographs are typically normal in primary frozen shoulder pathology but, following trauma, they provide an evaluation of the articular glenohumeral joint anatomy and potential malalignment.²³ Radiographs are important to rule out serious pathology, as well as abnormalities in the bone, joint or in the local soft tissues (e.g. calcific deposit), and are a prerequisite to a definitive diagnosis of frozen shoulder.²⁴ Magnetic resonance imaging reveals a decreased rotator interval and arthrography demonstrates a marked reduction in joint volume capacity in frozen shoulder pathology (Fig. 3).

Social history

Social history is relevant to the onset and progression of the patient's problem. This includes the patient's

Table 4. Special questions specifically related to the shoulder²²

Previous shoulder injury Care should be taken to avoid dislocation (e.g. for a history of previous anterior dislocation avoiding combined lateral rotation and abduction)
Neurological symptoms Spinal cord compression symptoms? Bilateral tingling in feet or hands? And/or disturbances of gait? Weakness or altered sensation in the arm?
Vascular symptoms Coldness or change in colour or loss of sensation in the arm or hands? Increase in symptoms when arms in an overhead position? All these symptoms may indicate vascular problems and require further investigation
Vertebrobasilar insufficiency If shoulder symptoms are considered to originate from the cervical spine, further assessment will require questioning and testing for vertebrobasilar insufficiency

Figure 3. An arthrogram of a frozen shoulder during hydrodilatation.

perspectives, experience and expectation, age, employment, home situation and details of their leisure and sporting activities.²⁵ To treat a frozen shoulder effectively, it is important that this condition is managed within the context of the patient's social and work environment, and that the patient's expectations and agreed realistic goals are established.

Drug history

Knowledge of the patient's frequency and dose of analgesia requirements can help the physiotherapist

establish the patient's grasp of shoulder pain and can be a good indicator of the level of severity. An awareness of other medications such as steroids and anticoagulants is essential for safe management. The use of anti-inflammatory medication identifies an inflammatory component to the patient's symptoms. This information regarding medication can also help aid the classification of the shoulder into the pain- or stiffness-predominant phase, and therefore guides treatment options.

Symptom behaviour

The use of a body chart is helpful to ensure a thorough and methodical assessment of the patient's symptoms. Symptoms from the glenohumeral joint are commonly felt at the insertion of deltoid but may be referred proximally to the lower cervical spine and/or distally to the forearm and hand.²⁶ It is also essential to establish whether symptoms are of a radicular nature from the cervical spine or purely shoulder-related. Quality, intensity and behaviour of symptoms should be explored. Common aggravating factors for the shoulder are hand behind back, above-head activities and lying on the affected shoulder. Detailed information on each of the aggravating and easing activities is useful for determining the structure at fault and identifying functional restrictions.

24-hour behaviour of symptoms

The presence of night pain leads to disturbance of sleep and the patient often describes difficulty lying on the affected shoulder.^{6,27} This could also be indicative of a shoulder presenting in the early stages of the pain-predominant phase of a frozen shoulder. This is supported by the process of pathogenesis in the early inflammatory process described by Hannafin et al.¹² The physiotherapist needs to determine the pattern of the symptoms first thing in the morning, throughout the day and at the end of the day. Morning stiffness that lasts more than 2 hours is suggestive of an inflammatory condition such as rheumatoid arthritis. Stiffness lasting only 30 minutes or less is likely to be mechanical or degenerative in nature. As shoulder movement increases, more difficulties are encountered with activities of daily living.¹⁵

A thorough and effective subjective assessment should enable the physiotherapist to have a clear idea of the severity and irritability of the presenting condition. It should provide a subjective hypothesis of the structures that are potentially contributing to the signs and symptoms. The objective assessment then confirms or negates this hypothesis.

Objective assessment

The objective examination aims to collect evidence and investigate the validity of the physiotherapist's initial hypothesis and rule out other possible diagnoses. All associated joints of the shoulder girdle should be included in an examination of a stiff shoulder. The following need to be conducted to provide a comprehensive physical assessment:

Observation

This element of the assessment commences from the moment the therapist meets the patient and continues throughout the assessment. Asymmetry, wasting, swelling and redness should be noted. The patient's posture should be noted because soft tissue adaptation can occur with habitual poor posture and use. However, muscle tightness as a result of overactivity, rather than adaptive shortening, can also result in a perceived contracture. A common adaptation at the shoulder is overactivity and/or tightness in the pectoralis minor in a protracted shoulder girdle posture. This will adversely affect an individual's ability to improve their scapulo-thoracic posture and shoulder biomechanics. The patient's symptoms of tightness can be assessed by the physiotherapist when examining the joint's ROM.

Palpation

Palpation of the shoulder is essential for evaluating shoulder girdle tenderness, temperature changes, presence of oedema or effusion mobility and the feel of superficial tissues and muscle tone.

Active ROM

Observations should be made of the range and quality of the movement, behaviour of pain and resistance throughout the ROM and provocation of any muscle spasm.

Passive ROM

Examining the shoulder joint passively allows the clinician to explore the available range eliminating the muscle's power limitations. The glenohumeral joint capsular pattern is described in Table 5.²⁸

This form of examination allows comparison of the response of symptoms between the active and

Table 5. Glenohumeral joint capsular pattern²⁸

Greatest limitation into external rotation
Less limitation into abduction
Least limitation internal rotation

Figure 4. Which soft tissue structures cause restriction in the glenohumeral joint?

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Table 6. Normal end feels^{19,29}

Cyriax	Kaltenborn	Description
Soft tissue approximation	Soft tissue approximation or soft tissue stretch	Soft end feel
Capsular feel	Firm soft tissue stretch	Fairly hard halt to movement due to capsular/ligamentous restriction
Bone to bone	Hard	Abrupt halt to movement (e.g. elbow extension)

Table 7. Abnormal end feels^{19,29}

Cyriax	Kaltenborn	Description	Pathology
Empty feel	Empty	No resistance offered due to severe pain	Infection, active inflammation, fractures or tumour
Springy block		Rebound feel at end range	Loose body
Spasm		Sudden hard end feel	Muscle spasm

passive movement that can help determine whether the structure at fault is contractile or noncontractile (Figure 4).¹⁹ Passive ROM testing also allows the physiotherapist to evaluate the associated muscle tone and lengths. The strongest evidence that a joint is the source of the patient's symptoms comes from an assessment of whether the patient's symptoms are reproduced during active, passive physiological movement, passive accessory movement and palpation of joint.

Abnormality is also recognized if a joint does not have its characteristic end feel or its resistance is felt too early or too late in what is considered their normal range²⁹ (Tables 6 and 7).

Accessory joint movement

Accessory movements are defined as those movements that a person cannot perform actively but which can be

Figure 5. Management algorithm for shoulder stiffness.

performed on that person by an external force.³⁰ They allow the physiotherapist to assess the range and quality of movement throughout the glenohumeral joint and identify any resistance indicating the location of the joint capsule abnormality. These manual techniques allow for assessment of changes in pain behaviour, both locally and referred, throughout the glenohumeral joint's ROM. Accessory movements also explore the soft tissue pliability and any possible associated muscle spasm.

Physiological movement restriction may be the result of a limitation of the accessory ROM at the joint; for example, during shoulder elevation through abduction the head of humerus rolls upwards and translates inferiorly on the glenoid cavity. Therefore, positive findings when assessing physiological or accessory movement can be used to formulate a treatment plan.

Muscle testing

It is important to assess specific muscle lengths of the shoulder girdle. A poor relationship resulting in inappropriate co-contraction between agonist and antagonist muscle force-couples may present as a reduction in ROM, often displaying characteristics of joint restriction and muscle stiffness.³¹ This may suggest muscle control can be altered when pathology is present and these changes may alter movement patterns at the shoulder.^{32–34} This can be assessed by supporting and decreasing the load of the arm and/or altering patient position to differentiate between soft tissue tightness and poor motor control. Poor proprioception and an inability to control movement into these ranges will restrict the glenohumeral joint's ROM. This myofascial stiffness can also be a protective response to pain or apprehension presenting as spasm or muscle guarding.²² During shoulder assessment, physiotherapists should be aware that optimum shoulder function is dependent not only on normal shoulder muscle activity, but also on normal muscle activity further down the kinetic chain, through the trunk and lower limbs.³⁵

Spinal

Cervical spine pathology is most easily confused with (and commonly coexists with) shoulder girdle pathology. Hence, a screening movement of the cervical spine should be performed to determine whether it reproduces the patient's shoulder pain. This should include cervical ROM with over pressure at the end of the physiological range to clear cervical pain as a referral source. If the subjective assessment suggests any neurological or radicular pathology, then a neurological assessment should be conducted.

Mobility of the thoracic spine should not be overlooked. Clinically, thoracic stiffness can contribute to shoulder stiffness.³⁶

Consideration of all these issues and a thorough examination ensures the most appropriate management strategy for the stiff shoulder (Figure 5).

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