Frozen shoulder is commonly encountered in general orthopaedic practice. It may arise spontaneously without an obvious predisposing cause, or be associated with a variety of local or systemic disorders. Diagnosis is based upon the recognition of the characteristic features of the pain, and selective limitation of passive external rotation. The macroscopic and histological features of the capsular contracture are well-defined, but the underlying pathological processes remain poorly understood. It may cause protracted disability, and imposes a considerable burden on health service resources. Most patients are still managed by physiotherapy in primary care, and only the more refractory cases are referred for specialist intervention. Targeted therapy is not possible and treatment remains predominantly symptomatic. However, over the last ten years, more active interventions that may shorten the clinical course, such as capsular distension arthrography and arthroscopic capsular release, have become more popular.

This review describes the clinical and pathological features of frozen shoulder. We also outline the current treatment options, review the published results and present our own treatment algorithm.

Definitions and terminology
Many patients present with painful shoulder conditions that produce apparent ‘stiffness’ by restricting active movement, either through pain inhibition (antalgic shoulder) or muscle weakness (such as a rotator cuff tear or deltoid paresis). In contrast, patients who have a frozen shoulder have characteristic features of the painful selective restriction of certain active and passive movements of the shoulder, in the presence of normal radiographs. Unfortunately, patients in each of these two categories tend to be considered together under an inclusive clinical umbrella, leading to misdiagnosis and inappropriate treatment. The reasons for this are twofold: first, the terms in common usage, such as frozen shoulder, adhesive capsulitis and pericapsulitis, are either non-specific or inaccurate, and therefore can be misused by clinicians and patients. Secondly, although identification of the syndrome rests on the recognition of characteristic clinical features, clearly defined diagnostic criteria are lacking. The need for standardisation in diagnosis has recently been emphasised and a system of terminology and classification based on consensus would be advantageous.1 In this review, we use the term frozen shoulder, as it is the most commonly used and avoids the ambiguities associated with terms that attempt to describe the underlying pathology.

Many conditions are associated with frozen shoulder, but these should be classified into sub-groups only where there is a known difference in prognosis, or modification of treatment is required (Fig. 1). The largest single group of patients have no detectable underlying cause for their symptoms (primary idiopathic frozen shoulder). The substantial group of patients with diabetes mellitus are considered as a separate group (secondary diabetic frozen shoulder). The substantial group of patients with diabetes mellitus are considered as a separate group (secondary diabetic frozen shoulder), since their disease course is usually more severe and protracted. A wide variety of other systemic conditions are associated with frozen shoulder, but most are uncommon and do not affect prognosis or treatment.3,4 They are therefore not considered under separate categories. Patients who have a clear primary pathology in the shoulder with secondary pain and stiffness (secondary frozen shoulder) are considered as a separate group, since they often have a poorer prognosis, and may require treatment of their underlying primary shoulder condition. This heterogeneous group of patients cause the greatest difficulty in diagnosis, since it is increasingly recognised that they may have pain and stiffness from both the primary condition and the secondary capsular...
contracture. A final complex group of patients have frozen shoulder associated with stroke and other neuromuscular disorders affecting the upper limb. These patients have pain and stiffness that is multifactorial and associated with muscle spasticity as well as contracture. They are usually managed with supportive physiotherapy and are not considered in detail in this review.

**Epidemiology**

Despite the lack of strict diagnostic criteria and hence the plausible over-diagnosis, frozen shoulder is estimated to affect 2% of the general population, with a cumulative incidence of 2.4 per 1000 person-years. It is rare before the age of 40, with a peak incidence between 40 and 60 and is unusual in patients over 70 years (except secondary traumatic frozen shoulder) and in manual workers. It affects women slightly more often than men. The economic impact of the condition is underscored by its predilection for adults of working age. Recurrence in the same shoulder is rare, but up to 20% of patients develop similar problems in the other shoulder. Bilateral simultaneous involvement occurs in 14% of patients, and 80% will experience a recurrence of symptoms within five years. There is a two- to threefold increased risk in twins, suggesting a genetic component, but there is no known racial predisposition.

Diabetes mellitus is the condition most commonly associated with frozen shoulder. The combined prevalence of a diabetic predisposition and frozen shoulder is estimated to be as high as 71.5%. Approximately half of these patients have been diagnosed previously with either Type I or II diabetes, and the remainder have pre-diabetes with an abnormal fasting blood glucose or glucose tolerance test. Diabetics have a 10% to 20% lifetime risk of developing a frozen shoulder, with a 4% point prevalence, and a two to four times greater risk than the general population.

**Pathoanatomy**

The shoulder is the most unconstrained and mobile articulation of the appendicular skeleton. The balance between instability and stiffness is therefore largely maintained by the static and dynamic soft-tissue stabilisers. Loss of both
active and passive movement can be produced by loss of the normal congruity of the articulation (such as in a chronic dislocation), increased constraint from bony overgrowth (such as that produced by osteophytes), or contracture of the dynamic muscular stabilisers. However, the most common cause of painful restriction of movement is an idiopathic frozen shoulder, which is characterised by an inflammatory contracture of the capsule and ligaments, which reduces the available intra-articular volume, limiting glenohumeral movement. Macroscopically, the capsule has a glassy appearance with acute vasculitis, inflammation and thickening, progressing to a more indolent fibrotic appearance with time (Fig. 2).

Cadaveric studies have demonstrated predictable patterns of limited movement produced by plication of different parts of the capsule. The selective restriction of external rotation characteristic of frozen shoulder syndrome is produced by anterosuperior capsular tightening, which particularly affects external rotation of the adducted arm, and anteroinferior tightening, which reduces external rotation in abduction. Clinical and MRI studies corroborate these findings, confirming that the structures primarily involved are the rotator interval (which comprises the superior glenohumeral ligament), the rotator interval capsule, and the coracohumeral ligament, and the anterior capsule and the inferior glenohumeral ligament. Posterosuperior capsular tightening limits internal rotation, and may be present in more severe forms. In frozen shoulder secondary to trauma or previous surgery for instability, there may also be stiffness due to extrinsic contracture of the rotator cuff (especially subscapularis) and obliteration of the normal subdeltoid tissue planes. This may be due to soft-tissue trauma, or post-operative scarring.

**Histological and biochemical pathways**

A variety of immunological, biomechanical, inflammatory and endocrine abnormalities have been described in patients with idiopathic frozen shoulder, but the basic pathogenesis remains poorly understood. Much work has been done to characterise the microscopic pathology and there is evidence of both inflammatory and fibrotic processes. However, no model completely explains why the stiffness tends to resolve, or why a disparate group of other co-morbidities predispose to its development.

Characteristically, pain precedes stiffness, which suggests an evolution from inflammation to fibrosis. Histological evidence of both chronic inflammatory cell infiltrate and fibrosis have been found in rotator interval biopsies of patients undergoing arthroscopic release. However, these findings have been observed in more severe forms of the condition and may not apply to the early phases. The pathological appearances have been likened to those of Dupuytren's disease, with a fibrous contracture of the coracohumeral ligament and the surrounding tissues of the rotator interval, which is composed of a dense matrix of mature type-III collagen containing fibroblasts and myofibroblasts. The compact arrangement of capsular collagen fibres causes the limitation of movement. A proposed mechanism highlights the role of cytokines and growth factors, leading to the accumulation and propagation of fibroblasts, which produce excess Type III collagen. The cytokine response may also initiate angiogenesis within the capsule, producing the typical arthroscopic appearances of new blood vessels on the capsular surface.

Collagen remodelling is currently being studied in order to elucidate a possible treatment. Local metalloproteinases degrade the connective tissue matrix and can be inhibited by specific tissue metalloproteinase inhibitors, other cytokines and growth factors. Frozen shoulder can be induced by administering a synthetic metalloproteinase inhibitor, suggesting that a decrease in metalloproteinases: metalloproteinase inhibitors ratio affects collagen turnover. Capsular tissues from patients with frozen shoulder have also been found to have increased mRNA for metalloproteinases, as well as a natural metalloproteinase inhibitor.

In patients with diabetes mellitus and other systemic disorders, microvascular disease may cause abnormal collagen repair, which could then predispose to frozen shoulder. The pain may also be neurogenic, as there are similarities with Type 1 complex regional pain syndrome. In both conditions, there is sympathetic autonomic hyperactivity, peripheral alpha-adrenoreceptor hyper-responsiveness, abnormal dorsal root reflexes, central nervous hypersensitivity, and sensory nerve degeneration. In addition, both conditions are more common after trauma to the shoulder, and are
associated with diabetes, thyroid disease and dyslipidaemia. However, it is over-simplistic to consider frozen shoulder as a form of complex regional pain syndrome. Other neuropathic mechanisms, including suprascapular nerve compression, have been considered. Although evidence suggests an association between these neuropathic and vascular conditions, there is no convincing single pathophysiological pathway that fully explains the pathology of frozen shoulder.

**Natural history**

Many studies suggest that frozen shoulder is a benign condition, with most cases recovering within two years. However, it is now accepted that up to 40% of patients may experience persistent symptoms. In the most comprehensive study of the natural history, 50% of patients still had mild pain after seven years, and 60% had persistent stiffness. It is estimated that approximately 7% to 15% have some degree of permanent loss of movement, though few have persistent functional disability.

**Clinical assessment**

Frozen shoulder is a clinical diagnosis; the history and physical examination are therefore crucial. It is essential to confirm the characteristic features of the condition, grade its severity and exclude other contributory systemic or local causes which may require laboratory investigations, radiographs and specialist imaging.

**History.** The pain is characteristically severe, felt diffusely around the shoulder girdle, with a deep-seated burning quality. It should have been present for more than one month to exclude other transient causes of shoulder pain. Intense night pain disturbing sleep is invariably present and is a key diagnostic feature (Fig. 1), as is impairment of most normal daily activities. Pain from the acromio-clavicular joint is common, presumably because the restricted glenohumeral movement increases the stress on this joint. Primary frozen shoulder is traditionally divided into three consecutive stages: freezing with acute pain and stiffness, frozen with established stiffness and reduced pain and thawing with the return of movement. However, clear stages of development are often difficult to define, or may be absent.

**Clinical examination.** The clinical diagnosis rests on confirming the characteristic pattern of limitation of both active and passive ranges of glenohumeral movement. Typically, there is pronounced and selective loss of passive external rotation, both with the arm in neutral and in abduction, which may not reach 90°. There is also some limitation of flexion and internal rotation. Since the assessment of pain is subjective and the limitation of external rotation is an objective and constant feature, we grade the clinical severity on external rotation (Fig. 1).

The pattern of stiffness helps to determine the anatomical structures most involved. Contracture of the rotator interval produces anterosuperior tightness and limits external rotation with the arm adducted, whereas anteroinferior contracture of the capsule and inferior glenohumeral ligament limits external rotation in abduction. Posterior capsular contracture limits adduction and internal rotation, and extra-articular contractures produce global stiffness. Intra-articular local anaesthetic injection may improve pain, but loss of passive movement remains. Scapulothoracic movement may also be limited, with secondary pseudowinging.

Shoulder girdle wasting may be present in protracted cases. The presence of swelling or erythema should suggest the possibility of infection or tumour. Specific clinical tests of the biceps tendon and tests for rotator cuff and acromio-clavicular joint dysfunction and instability should be performed to exclude secondary causes.

**Investigation.** There are no laboratory tests or radiological markers for frozen shoulder. Conventional radiographs are usually performed to assess for causes of secondary frozen shoulder syndrome, such as a bone tumour, acute calcific deposits or fracture (if there is a recent history of trauma). Specialist imaging (ultrasound or magnetic resonance arthrography) may be useful if rotator cuff pathology, or a radiologically occult fracture of the greater tuberosity is suspected. Blood glucose levels should be checked to exclude diabetes. Other haematological or biochemical tests may be ordered, if other endocrine or rheumatological abnormalities are suspected.

**Differential diagnosis.** Selective loss of external rotation is associated with only two other commonly encountered shoulder pathologies: locked posterior dislocation and osteoarthritis. Both of these can usually be excluded with conventional radiographs. Features of frozen shoulder are commonly present in patients with calcific tendonitis, fractures of the proximal humerus (especially those of the greater tuberosity), tears of the rotator cuff and early osteoarthritis of the glenohumeral joint (Table I). These pathologies can usually be detected using conventional radiographs and ultrasound examination. It may be difficult to determine whether the clinical features are due to the primary condition, or a secondary frozen shoulder. Other secondary causes are rare, but indicators such as weight loss, systemic upset, shoulder swelling and bony tenderness should raise the possibility of infection or tumour, and prompt urgent investigation.

**Treatment**

The goal of treatment is to relieve pain, restore movement and regain function of the shoulder. Treatment should be individualised and based on the severity and chronicity of symptoms. A multidisciplinary approach is preferable, and most patients can be managed non-operatively in a primary care setting with the expectation of a good outcome. There are many alternative forms of treatment for this condition, but evidence of their efficacy is not well-established from clinical trials, and it is unclear if several interventions used in combination are better.
Physiotherapy. An initial six- to 12-week therapeutic trial of physiotherapy is typically prescribed early, to try to prevent further limitation of movement, and later to re-establish movement. Passive mobilisation and capsular stretching is commonly used. However, this may be counter-productive in the painful acute inflammatory phase, where analgesia and activity modification may be more appropriate. Use of oral non-steroidal anti-inflammatory drugs and local heat/cold treatment may help to increase movement and reduce inflammation. The physiotherapist plays a major role in supervising the exercise programme, explaining the time course to resolution of symptoms, and encouraging a programme of home exercises.

Despite its ubiquity, the use of physiotherapy alone is not supported by higher level studies, although several lower level studies report a benefit. A recent Cochrane review concluded that the existing literature was insufficient to prove that physiotherapy alone was beneficial, with two small clinical trials concluding that physiotherapy alone did not offer any benefit when compared with no-treatment controls. A further study found little difference in outcome, regardless of the physiotherapy technique used.

Other interventions have included ultrasound, acupuncture, bipolar interferential current, transcutaneous electromagnetic stimulation, laser and pulsed electromagnetic field therapy. None of these have been subjected to prospective comparative studies and their value remains uncertain. The physiotherapist plays a major role in supervising the exercise programme, explaining the time course to resolution of symptoms, and encouraging a programme of home exercises.

Although some studies have shown improvement with intra-articular steroid injection, others have found that this produces little benefit. A recent meta-analysis showed little evidence of benefit from steroid injection. Like oral steroids, they appear to provide earlier relief from pain, when compared with placebo, but whether this is sustained in the long term is unknown.

Distension arthrography. This local anaesthetic has the advantage of producing rapid improvement in movement, without recourse to a more interventional surgical procedure. Under fluoroscopic control, an arthrogram is initially performed to exclude a rotator cuff tear. The diagnosis of frozen shoulder is supported by the characteristic arthrographic appearance of a contracted capsule (Fig. 3). Sterile water is then injected under pressure sufficient to cause capsular rupture (evidenced by a decrease in the pressure necessary to continue injection). An intra-articular injection of steroid is usually given at the end of the procedure, but rupture of the contracted capsule is the probable cause of symptomatic relief. Intensive physiotherapy should begin immediately after the procedure to retain the improved range of movement achieved.

It is not possible to draw firm conclusions about the efficacy of the technique, due to the small number of studies available. It is the authors’ experience that the technique is very successful in the treatment of primary frozen shoulder, but less effective in secondary frozen shoulder after fracture, either treated non-operatively, or by open reduction and internal fixation. This is probably because the limitation of movement in these patients is produced by a combination of capsular contractures and extra-articular adhesions.

Data from a small placebo-controlled trial suggested that arthrographic distension provides significant short-term benefit, which is maintained in the medium term. A second trial showed similar improvements when distension was compared with physiotherapy alone. Further comparative studies are required to evaluate the efficacy of this technique.

Manipulation under anaesthesia. Manipulation under anaesthesia (MUA) has been used extensively if physiotherapy fails. It has been successfully used alone or combined with a steroid injection or with an arthroscopic capsular release, and usually results in a rapid return of movement of the shoulder.

Table I. Differential diagnoses of primary frozen shoulder

<table>
<thead>
<tr>
<th>Differential diagnosis</th>
<th>Signs and symptoms</th>
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<tbody>
<tr>
<td>Calcific tendonitis*</td>
<td>Painful arc; characteristic radiological appearances</td>
</tr>
<tr>
<td>Rotator cuff tears*</td>
<td>Selective pattern of rotator cuff weakness on testing</td>
</tr>
<tr>
<td>Rotator cuff impingement</td>
<td>Positive impingement signs on testing</td>
</tr>
<tr>
<td>Glenohumeral arthritis*</td>
<td>Generalised pain and crepitus on movement of glenohumeral joint</td>
</tr>
<tr>
<td>Acromioclavicular arthritis*</td>
<td>Localised tenderness and restricted cross-body adduction</td>
</tr>
<tr>
<td>Cervical spine pathology</td>
<td>Neck stiffness; upper limb neurology</td>
</tr>
<tr>
<td>Glenohumeral instability</td>
<td>Recurrent dislocation; apprehension</td>
</tr>
<tr>
<td>Proximal humeral fracture*</td>
<td>History of trauma; localised tenderness and bruising</td>
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<tr>
<td>Tumour/infection</td>
<td>Red flags of fever, weight loss, other systemic signs</td>
</tr>
<tr>
<td>Parsonage-Turner syndrome</td>
<td>Sudden pain; marked muscle weakness and wasting</td>
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* indicates conditions that often have a concomitant element of secondary frozen shoulder.
However, there is disagreement as to whether it shortens the natural history of the disease. Manipulation is performed in a controlled manner, and in order to decrease the risk of a humeral fracture, the arm is grasped close to the axilla. Forward flexion is initially undertaken, rupturing the inferior capsule. This is followed by external rotation, first with the arm close to the body and then in abduction. Internal rotation is the final manoeuvre performed with the arm in abduction. This procedure should be avoided in the elderly, osteoporotic patient because of the risk of fracture. It is a wise precaution to take pre and post-manipulation radiographs and the risk of complications can be minimised by attention to technique. Post-manipulation physiotherapy is important to maintain the movement that has been gained.

Several case-series have found good to excellent results in the long-term following MUA, but there are few high level studies. These suggest that MUA is more beneficial in patients with refractory severe stiffness, which is unresponsive to physiotherapy, when compared with those with less severe disease.

Arthroscopic capsular release. This technique is generally performed if MUA fails to restore movement, although some surgeons now favour arthroscopic release without MUA. The procedure comprises three basic components: anaesthesia (usually with interscalene block), manipulation of the shoulder, and arthroscopic release.

The order of arthroscopic release and manipulation is controversial. Some suggest MUA in the first instance, with arthroscopic capsular release performed only if adequate movement is not restored. Without prior manipulation, placement of the arthroscope into the joint can be more difficult due to the capsular contracture. However, some authors advocate arthroscopy before manipulation, since prior manipulation causes haemarthrosis and may make subsequent capsular release more difficult.

The surgery is performed in either the ‘beach-chair’ or the lateral decubitus position. Care is taken to avoid articular cartilage injury during insertion of the arthroscope through the tight capsule. A systematic inspection is performed to identify areas of synovitis and contracted tissue and any coincident pathology. The initial release consists of resection of all contractures within the rotator interval triangle bounded by the biceps tendon superiorly, the subscapularis inferiorly and the superior glenoid medially. Structures released include the coracohumeral ligament, anterior capsule, superior and middle glenohumeral ligaments, and the subscapularis bursa. Adhesions between the conjoint tendon and the subscapularis are then released if present. The identification of the coracoid process as a landmark is a key early step, to avoid inadvertent dissection proceeding too far medially and injuring the brachial plexus. Most commonly an ‘inside-out’ release from within the glenohumeral joint is performed using an anterior rotator interval portal, although an ‘outside-in’ technique with access through the subacromial bursa is gaining in popularity.

The release then continues antero-inferiorly freeing the anterior capsule and the anterior band of the inferior gleno-humeral ligament. The subscapularis should then be seen to track normally underneath the coracoid on passive rotation of the shoulder. Lengthening of the subscapularis is occasionally required, if this is extensively contracted. In the few cases where there is persistent loss of internal rotation or flexion after the anterior release, division of the posterior capsule, posterior band of the inferior glenohumeral ligament and axillary pouch is required, switching the viewing portal anteriorly and performing the release though the posterior portal. Care is required with this more aggressive form of release to avoid injuring the axillary nerve, at risk during the inferior release, by ensuring that the electrocautery device is directed towards the glenoid when dividing the posterior capsule.

The extent of capsular release depends on clinical judgement: some authors have advocated routinely performing a ‘360-degree’ release, whilst others have adopted a more cautious approach. Although one study supported a more extensive release, two more recent comparative studies suggested that improved movement did not follow an extensive compared with a more limited approach. Further investigation is needed to determine the optimal extent of release.

Subacromial bursectomy, acromioplasty and the release of sub-deltoid adhesions are sometimes performed if there is adhesion of the rotator cuff to the acromion and deltoid. This is most commonly encountered in patients with secondary frozen shoulder after fracture, especially if they have been treated operatively. If the fracture has been treated surgically and has united, retained implants should be removed if they are causing impingement. This will usually require a supplementary open surgical approach and release of sub-deltoid extra-articular contractures at the

Fig. 3

The characteristic arthrographic appearances of frozen shoulder. The joint volume is reduced and there is no filling of the inferior capsular recess.
same time. If the frozen shoulder is secondary to calcific tendonitis, the deposit is usually excised from within the subacromial bursa at the time of release. If there is an associated rotator cuff tear, it would be typical practice to perform a release to re-establish movement before proceeding to rotator cuff repair.

Intensive rehabilitation should begin immediately postoperatively with daily stretching exercises. Continuous passive motion machines may also be useful to maintain movement. Adequate analgesia in the early post-operative period is therefore important and this may be administered via an indwelling interscalene catheter for several days post-operatively. Patients are made aware that pain may not subside for up to six weeks and some residual loss of movement is to be expected.

High-level comparative studies of arthroscopic release are lacking, though in a quasi-randomised study arthroscopic release was compared with manipulation in patients with refractory frozen shoulder. Similar movement was achieved, but there was greater pain relief and functional improvement in the arthroscopic group.

Open surgical release. Open release of contracture was originally described by Codman and other authors later recommended and described various open techniques. Current indications include previous failed arthroscopic release, no improvement after six months of home exercise, and frozen shoulder secondary to fracture or previous surgery. Dense adhesions and scarring following soft-tissue injury and haemorrhage can make closed manipulation or attempted arthroscopic release particularly difficult and the risk of injury is high. An open procedure therefore allows direct visualisation of structures undergoing release and lengthening, removal of retained implants and excision of heterotopic ossification or bony spurs. Disadvantages include the risk of disruption of the lengthened subscapularis tendon and its repair, and the greater post-operative pain
and limitation of movement, compared with arthroscopic release. A deltopectoral approach is used and subdeltoid adhesions are released after careful identification and protection of the axillary nerve. The rotator interval is next identified and opened along the line of the superior aspect of the subscapularis tendon. The coracohumeral ligament and rotator interval capsule are excised, and the subscapularis tendon can be z-lengthened if contracted. Further release of the inferior and posterior capsule can be performed if abduction and internal rotation are still limited. Secondary frozen shoulder after non-operative treatment of a proximal humeral fracture may be due to both capsular contracture and malunion or nonunion of the fracture. In this situation, capsular release may need to be combined with correction of any deformity of the humeral head.

Summary of treatment
Most patients with primary frozen shoulder are best initially treated symptomatically with pain modification and supportive physiotherapy. Steroid injection is a useful adjunctive primary care treatment. Increasingly distension arthrography is being advocated at an earlier stage in those patients who fail to respond to this ‘first-line’ treatment. Manipulation under anaesthesia supplemented with open or arthroscopic capsular release is useful in the minority of patients with frozen shoulder syndrome who are refractory to these measures. Those with frozen shoulder secondary to structural abnormalities within the shoulder usually require additional treatment of the underlying pathology, in addition to treatment of the frozen shoulder. Further comparative clinical studies are required to evaluate the efficacy of the various methods of treatment. However, a suggested algorithm for treatment currently used by the authors is shown in Figure 4.

Supplementary material
Tables detailing studies investigating treatments for frozen shoulder: i) physical therapies, ii) steroid treatment, iii) arthrographic distension, iv) manipulation under anaesthesia, v) arthroscopic release, and vi) open surgical release, are available with the electronic version of this paper on our website www.jbjs.org.uk

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