Shoulder apprehension is related to changes in functional cerebral networks induced by dislocations, peripheral neuromuscular lesions and persistent mechanical glenohumeral instability consisting of micro-motion.

All the damage to the osseous and soft-tissue stabilizers of the shoulder, as well as neurologic impairment persisting even after stabilization, must be properly identified in order to offer the best possible treatment to the patient.

There is growing evidence supporting the use of a global multimodal approach, involving, on the one hand, shoulder ‘reafferentation’, including proprioception, mirror therapy and even cognitive behavioural approaches, and, on the other hand, surgical stabilization techniques and traditional physical therapy in order to minimize persistent micro-motion, which may help brain healing. This combined management could improve return to sport and avoid dislocation arthropathy in the long term.

Keywords: shoulder instability; anteroinferior glenohumeral dislocation; apprehension

Introduction

The glenohumeral joint has six degrees of freedom,1 three translational and three rotational, with minimal bony constraints that provide a large functional range of motion, making this joint vulnerable to instability. The latter can lead to increased pain, decreased level of activity, prolonged absence from work and sport, and a general decrease in quality of life.2,3 Apprehension can be difficult to diagnose pre- or post-operatively, as it seems more complex than a pure mechanical problem of the shoulder. Although clinical definition seems to be well established, its underlying pathologic mechanism remains unclear. This may explain the wide reported range (3% to 51%) of patients with ongoing apprehension or who will avoid any shoulder movement after an open or arthroscopic stabilization, despite a clinically stable joint.4-6 Failure to recognize and adequately address this issue may result in poor outcome and lead to unnecessary surgery or even revision. Furthermore, identifying this condition may allow us to establish adequately targeted rehabilitation programs.

The purpose of this article is to review the current state of knowledge about shoulder apprehension and its aetiologic factors. Finally, this work presents practical guidelines and promising future perspectives.

Definition

An important aspect to incorporate in dislocation management is apprehension, defined as anxiety and motor resistance in patients with a history of anterior glenohumeral instability. Clinically, the apprehension sign is defined as fear of imminent dislocation when placing the arm in abduction and external rotation, and should be distinct from mere pain which can be related to inflammation, stiffness and other shoulder pathologies.7,8 Proprioception, as defined by Charles Scott Sherrington, is the sense of the relative position of neighbouring parts of the body and strength of effort being employed during movement.9 It is distinct from exteroception, by which one perceives the outside world, and interoception, by which one perceives pain, hunger or the movement of internal organs. The brain integrates information from proprioception and from the vestibular system into its overall
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sensory of body position, movement and acceleration. Kinesthesia refers either to the brain’s integration of proprioceptive or vestibular inputs.

**Localization of the lesion**

The pathogenesis of apprehension is not fully understood. Theoretically, apprehension could be related to: 1) brain changes induced by dislocations;10-13 2) peripheral neuromuscular lesions consecutive to dislocation affecting proprioception;14 or 3) persistent mechanical instability consisting in micro-motion (Fig. 1).15

**Brain**

Fear, anxiety and anticipation of situations that could lead to a dislocation are essential cognitive processes in shoulder apprehension. Functional magnetic resonance imaging (fMRI) measures brain activity by detecting changes associated with blood flow.16 This technique relies on the fact that cerebral blood flow and neuronal activation are coupled. When an area of the brain is in use, blood flow to that region also increases. Recently, our group used fMRI with visual apprehension stimulation to explore neuronal connections and cerebral changes induced by shoulder dislocation.11 Several cerebral areas were modified during those analyses, representing the different aspects of shoulder apprehension. Specific reorganization was found in apprehension-related functional connectivity of the primary sensorimotor areas (motor resistance), dorsolateral prefrontal cortex (cognitive control of motor behaviour), and the dorsal anterior cingulate cortex/dorsomedial prefrontal cortex and anterior insula (anxiety and emotional regulation) (Fig. 2).

Those regions are involved in the cognitive control of motor behaviour.17 Hence, there is motor control anticipation and muscular resistance (protective reflex mechanism) in order to avoid shoulder movement that could lead to dislocation.5,6 Another recent study published by Shitara et al analysed cerebral changes induced by shoulder dislocation in 14 patients.18 Although results were similar to our study, they observed a larger and less specific spectrum of activated cerebral areas, which may be explained by the fact that they projected static and abstract
images during fMRI acquisition that may be prone to vaguer and subjective interpretation, and that, moreover, did not convey the dynamic component inherent to apprehension.

In a subsequent study, our group extended these findings by investigating further structural alterations in patients with shoulder apprehension. We found that fractional anisotropy, representing white matter integrity, was increased in the left internal capsule and partially in the thalamus in patients compared with healthy controls. Fractional anisotropy correlated positively with pain visual analogue scale (VAS) scores ($p < 0.05$) and negatively with simple shoulder test (SST) scores ($p < 0.05$). This suggests an abnormal increased axonal integrity and therefore pathological structural plasticity due to the overconnection of white matter fibres in the motor pathway. These structural alterations affect several dimensions of shoulder apprehension as pain perception and performance in daily life.

The neuronal changes previously mentioned and presented in shoulder apprehension can also be assessed in daily clinical practice. Indeed, Cunningham et al correlated clinical scores and tests (Rowe, pain VAS, SST, simple shoulder value (SSV), WOSI) with functional cerebral imaging in patients with shoulder apprehension. Their hypothesis was that it might be possible to simplify shoulder instability scores as it has been previously possible with rotator cuff and SLAP lesions and that at least one score could encompass the spectrum of these cerebral alterations. They found that the Rowe score integrated several aspects of apprehension, notably the motor and sensory functions, as well as pain anticipation and attention. This could be explained by the fact that the Rowe score is the only tested score integrating range of motion. This also provides the ability to evaluate the motor component (stability and motion) and cognitive component (perceived pain) of shoulder apprehension. Pain VAS and WOSI seemed to correlate with fewer brain networks compared with the Rowe. This could be explained by the fact that their assessment is focused only on cognitive aspects (pain for pain VAS, shoulder function in everyday life activities for WOSI) and that they do not integrate pure shoulder motion. SST and SSV were not found to be associated with brain network alterations, which is corroborated by the fact that they are general shoulder scores and were not specifically validated for instability.

Recently, a similar study demonstrated that shoulder stabilization could allow the brain to partially ‘recover’. Patients with shoulder apprehension underwent clinical and fMRI examination before and one year after shoulder stabilization surgery. Clinical examination showed a significant improvement in post-operative shoulder function compared with pre-operative. Coherently, results showed decreased activation in the left pre-motor cortex post-operatively, demonstrating that stabilization surgery induced improvements both at the physical and at the brain level, one year post-operatively (Fig. 3). Most interestingly, right–frontal pole and right-occipital cortex activity was associated with good outcome in shoulder performance.

**Peripheral neuromuscular lesion**

During a traumatic dislocation, there is a disruption of the shoulder tendinomuscular (in 10% of cases) and peripheral nerve lesions (in 14% of cases). However, this does not account for subclinical neurologic damage that may be much more preponderant. Capsuligamentous structures surrounding the glenohumeral joint are richly innervated with proprioceptors and therefore play an important sensorimotor role in addition to their primary mechanical role.
stabilizing function. Thus, when considering the extensive and frequent damage to these structures after shoulder dislocation (Fig. 4), there is bound to be an important loss in glenohumeral proprioception. The latter plays a significant role in the stabilization of a normal healthy shoulder and after any shoulder injury by contributing to motor control. Surgical stabilization has been shown to help proper healing of these structures and thus restore proprioception of the glenohumeral joint.

**Glenohumeral joint**

The third aetiologic factor for apprehension is persistent micro-motion in the glenohumeral joint despite a clinically stable shoulder, satisfactory radiographic results and no new episode of subluxation or dislocation. As stated above, shoulder dislocation causes damage to the capsuloligamentous complex in 52% of cases, and the glenoid labrum in 73% of cases. The plastic deformation of these structures becomes progressively worse with subsequent episodes. In addition to progressive soft-tissue injury, recurrent dislocations induce bony lesions, which may involve the glenoid (bony Bankart), the posterolateral humeral head (Malgaine or Hill-Sachs lesion), or both. Severity of apprehension, quantified as the moment at which it appears during the course of abduction and external rotation, seems to be correlated to the extent of bone loss. Capsular redundancy has also been recognized as a risk factor for ongoing apprehension after surgical stabilization, and Ropars et al found a significantly decreased apprehension in patients with associated capsulorrhaphy to Latarjet procedures, compared with patients with Latarjet and no capsular reconstruction. However, these changes may be very subtle and therefore not detectable on standard clinical MRI in neutral position. This has been described by Patte et al in non-operated patients and popularized under the name of ‘unstable painful shoulder’. This micro-motion may yet still be present after surgical stabilization.

Shoulder stabilization may thus only prevent new episodes of dislocation, rather than truly stabilizing the shoulder. Studies have been able to report translation values at the glenohumeral joint using external measurement systems such as optical motion capture combined with CT or MRI. Based on this technology, a recent study described glenohumeral translation in patients with traumatic anteroinferior instability and subsequently analysed the effect of glenohumeral stabilization on this translation. For all movements, the authors recorded humeral head position of the contralateral and ipsilateral shoulders in relation to the glenoid centre pre-operatively and one year post-operatively. They observed an anterior translation of the humeral head, especially during flexion and abduction movements (p < 0.05 and p < 0.05, respectively). One year after surgery, all patients had a clinically stable shoulder; none presented with a new episode of dislocation or subluxation. However, anterior translation of the humeral head was not significantly reduced and remained close to pre-operative values, confirming that shoulder stabilization does not stabilize the shoulder but uniquely prevents further dislocation. These findings have several important implications. First, it may explain residual pain, apprehension and impossibility of return to sport at the same level as reported in other studies. Second, persistent abnormal motion between the glenoid and the humeral head might be the underlying cause of dislocation arthropathy that is observed with a prevalence of 36%. Indeed, Hovelius et al demonstrated that arthritis development was related to the instability phenomenon itself rather than to surgery, when properly carried out. Repeated sliding of the humeral head against the glenoid associated with degenerative changes of cartilage properties and decreased biological healing potential related to aging could lead to a vicious circle of extensive cartilage damage.

**Treatments and perspectives**

The degree, nature and combination of injuries induced by traumatic glenohumeral instability are highly variable. Damage to the bony and soft-tissue stabilizers of the shoulder, as well as neurologic impairment, must be detected and analysed in order to provide the patient
with the most adequate treatment option. This new knowledge should be applied to rehabilitation therapy and surgical stabilization techniques. As the current stabilization techniques do not seem to prevent residual glenohumeral micro-motion, it remains to be determined which factors help to minimize this phenomenon, whether it is the increase in the anteroposterior diameter of the glenoid with a bone graft, the sling effect provided by the conjoined tendon or the long head of the biceps, the capsulorrhaphy, the repaired labrum or the remplissage. Interestingly, less invasive approaches do not seem to improve results regarding stability compared with open ones; more recurrences have been noted after arthroscopic Latarjet or Bankart procedures. Although the latter factor could be related to technical problems linked to the development of these new procedures, the bulk effect conferred by anterior scar tissue formation, already sought since the beginning of shoulder stabilization, may also play an important role in decreasing glenohumeral translation. New and minimally invasive techniques lead to less fibrosis, which may allow more post-operative mobility, but could, on the other hand, also reduce stability.

Heading towards a better understanding of the complex and multifactorial origins of glenohumeral instability and apprehension, post-operative management may in turn also be improved, notably in challenging cases of patients with persistent apprehension, despite a clinically stable shoulder. Knowing that shoulder apprehension could be the result of ongoing cerebral abnormalities or residual micro-motion may avoid costly series of onerous investigations, useless physiotherapy sessions or even re-operations. Furthermore, this perspective offers a new angle of a therapeutic approach that differs from conventional manual rehabilitation methods centred on the glenohumeral joint itself. If persistent apprehension or micro-motion is detected, growing research evidence supports the use of a multidisciplinary approach including: 1) a ‘reafferentation’ (reconveying and connecting the neurological peripheral input to the cortex) of the shoulder particularly focused on proprioceptive work, which has been proven to lead to better neuromuscular control than strengthening alone; 2) a biofeedback therapy where the patient directly visualizes his abnormal response to a negative stimulus on fMRI or electroencephalogram and can thereby actively correct it – this treatment modality has already been shown to improve shoulder control and performance in various settings; 3) a cognitive behavioural approach to decondition this pathological residual apprehension by making them realize residual apprehension does not necessarily lead to recurrent instability, with gradual exposition that has already shown successful results in the treatment of kinesiophobia, a condition based on a re-injury fear-avoidance model initially described in low-back pain, further popularized in sports medicine and various upper limb conditions; 4) electrical stimulation of hypoactive rotator cuff and periscapular muscles. Table 1 provides practical advice for multimodal therapy. From a surgical point of view, different techniques
of stabilization with, for example, better restoration of the glenoid concavity\textsuperscript{61} and of the anterior capsulo-ligamentous complex\textsuperscript{62} or dynamic anterior stabilization\textsuperscript{44} may open new horizons leading to improved management regimens for instability of the shoulder, but also other joints that suffer from instability (e.g. the knee).\textsuperscript{63}

**Overview**

This article provides a concise and comprehensive summary of the central and peripheral impairments observed after an anterior shoulder dislocation. Apprehension is a common problem with a complex multisystem origin that can be regrouped in three types: central neurologic; peripheral neurologic; and mechanical. Shoulder instability induces major alterations in the central nervous system, especially in the primary sensorimotor cortex, dorsolateral and dorsomedial prefrontal cortex, as well as the insula. Changes in these brain areas involve complex emotional and cognitive functions, anxiety and salience, and induce more negative anticipation and motor resistance in patients, following a cerebral conditioning process generated by dislocation episodes. Persistent peripheral neurologial impairment and articular micro-movement may also explain why some patients keep an apprehension despite a clinically stable shoulder with no further episode of instability. Unfortunately, there is no clinical score that can entirely encompass the extent of the cognitive alterations induced by apprehension. However, instability-specific scores including items about shoulder motion, such as the Rowe score, are more correlated than general scores and should preferably be utilised. The suggested range of treatments should help to streamline the clinician’s and physiotherapist’s rehabilitation strategy, avoiding stabilizations or revision surgery in some situations, as well as preventing long-term dislocation arthropathy.

| Types of therapy               | Approach                                                                 |
|-------------------------------|--------------------------------------------------------------------------|
| Reafferentation              | Proprioceptive work                                                      |
| Biofeedback therapy          | • Direct visualization of the response of a stimulus (electrical sensors, pressure) that help the patient to receive information about his/her body  |
|                              | • Postural tape and mirrors (sensory/tactile feedback) to correct muscle activation |
| Cognitive behavioural approach| Gradual exposition to shoulder position at which apprehension is experienced |
| Electric stimulation         | Stimulation of hypoactive rotator cuff and periscapular muscles, which leads to subluxation or dislocation during shoulder movement |

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