

The Aetiology of Subacromial Impingement Syndrome

Summary Subacromial impingement syndrome has been described as the most common form of shoulder pathology (Jobe and Jobe, 1983; Kessel and Watson, 1977). Neer (1972, 1983) argued that 100% of impingement lesions and 95% of rotator cuff pathology are caused by friction between the acromion and surrounding tissues within the subacromial space. This concept has been challenged and the literature suggests that the aetiology of subacromial impingement is multifactorial. These causes include anatomical and mechanical factors, rotator cuff pathology, glenohumeral instability, restrictive processes of the glenohumeral joint, imbalance of the muscles controlling the scapula, and postural considerations. The purpose of this paper is to explore the potential factors contributing to pathology.

Introduction

Subacromial impingement syndrome (SIS) is a general term used to describe a variety of conditions that may act independently or in combination, and manifest as anterior or anterior-lateral-superior shoulder pain. SIS occurs as a result of pathology of one or more of the structures located within the subacromial space. The pain is associated with a loss of shoulder function, especially during overhead activities, occurring during vocational, sporting or the normal activities associated with daily living.

The subacromial space has also been termed the acromio-humeral joint or the bursal joint (Wiles, 1955), the supraspinatus outlet (Neer and Poppen, 1987), and the suprahumeral space (Calliet, 1991). These names reflect the borders and contents of this region. The superior border is the coracoacromial arch, comprising the inferior surface of the acromion, the coracoacromial ligament and the coracoid process. The inferior border comprises the greater tuberosity and superior aspect of the head of the humerus. Petersson and Redlund-

Key Words

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Johnell (1984) in a series of 175 radiographs reported that the mean distance between the inferior border of the acromion and the superior border of the humerus in an anteroposterior projection was between 9 to 10 millimetres (mm).

Located within the subacromial space are the tendons of the rotator cuff and the long head of biceps, the subacromial/subdeltoid bursa and the superior capsule of the glenohumeral joint. During elevation of the arm the greater tuberosity moves closer to the acromion, narrowing the space. SIS results from pathology of any of these structures. The inferior aspect of the acromioclavicular joint has also been implicated in the aetiology and pathogenesis of SIS (Petersson and Gentz, 1983).

Historical Background

Numerous early references to subacromial pathology have appeared in the literature (Adams, 1852; Bosworth, 1940; Codman, 1934; Diamond, 1964; McLaughlin and Asherman, 1951; Meyer, 1931). Neer (1972) argued that the anterior one-third of the acromion, the coracoacromial ligament and, at times, the acromioclavicular joint impinged upon the rotator cuff, primarily in the region of the insertion of the supraspinatus into the greater tuberosity. He stated that this impingement occurred mainly when the arm was in a position of forward elevation. He labelled the site of compression as the impingement zone, and hypothesised that spurs located at the acromial end of the coracoacromial ligament led to wear and tear to the rotator cuff. He proposed that SIS occurred in three stages and this could be viewed as a pathological process in continuum.

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Neer (1972, 1983) attributed 95% of all rotator cuff lesions and 100% of impingement pathology to the acromion. He also described the 'impingement sign', a clinical procedure used to reproduce symptoms, and the 'impingement test', a 10 cc injection of 1.0% xylocaine into the subacromial region to reduce symptoms. The combination of both procedures would confirm a diagnosis of SIS.

However, almost 30 years after this publication, the aetiology and pathogenesis of SIS still remain unclear and numerous authors (Jobe, 1997; Kibler, 1998; Ogata and Uthoff, 1990; Riand *et al*, 1998) have challenged Neer's original claim. It appears that SIS is a multifactorial condition whose symptoms may be attributed to a large number of causes. The potential etiological causes of SIS are summarised in the table (right), and the purpose of this paper is to review each of these factors.

Mechanical and Anatomical Causes of SIS

Primary Mechanical Impingement

SIS as described by Neer (1972, 1983) has been termed primary mechanical impingement. Neer (1983) argued that the reason rotator cuff tears and SIS develop in some people and not in others is best explained by the shape of the acromion. In support, Bigliani *et al* (1986) examined the shape of the acromion of 140 shoulders in 71 cadavers and concluded that there were three distinct shapes, type 1 flat, 2 curved and 3 hooked, arguing that these differences were due to variations in morphology (fig 1). Bigliani *et al* (1986) concluded

Categories of subacromial impingement

Category	Sub-category
Mechanical/anatomical	Primary mechanical impingement (Neer, 1972, 1983) Acromial bone spurs (Neer, 1972) Os acromiale (Neer, 1972, 1983) Coracoacromial ligament (Soslowky <i>et al</i> , 1994) Posterosuperior glenoid impingement (Jobe, 1997; Riand <i>et al</i> , 1998)
Rotator cuff	Rotator cuff overuse (Jobe and Jobe, 1983; Meister and Andrews, 1993) Degenerative tendinopathy (Ozaki <i>et al</i> , 1988)
Instability/hypermobility	Secondary tensile disease (Meister and Andrews, 1993) Secondary compressive impingement (Warner <i>et al</i> , 1990)
Restrictive processes	Restricted glenohumeral capsule (Harryman <i>et al</i> , 1990, Matsen and Arntz, 1990)
Functional scapular instability	(Kibler 1991, 1998; Warner <i>et al</i> , 1992)
Posture	(Ayub, 1991; Bowling <i>et al</i> , 1986; Calliet 1991; Solem-Bertoft <i>et al</i> , 1993)

that there was a higher incidence of rotator cuff tears in the type 3 (hooked) acromions and that this morphological variation predisposed mechanical impingement.

However, to implicate the acromion as a causative factor, these variations in shape need to be proven to be anatomical variations and a cause-and-effect relationship between shape and pathology needs to be established. Both of these

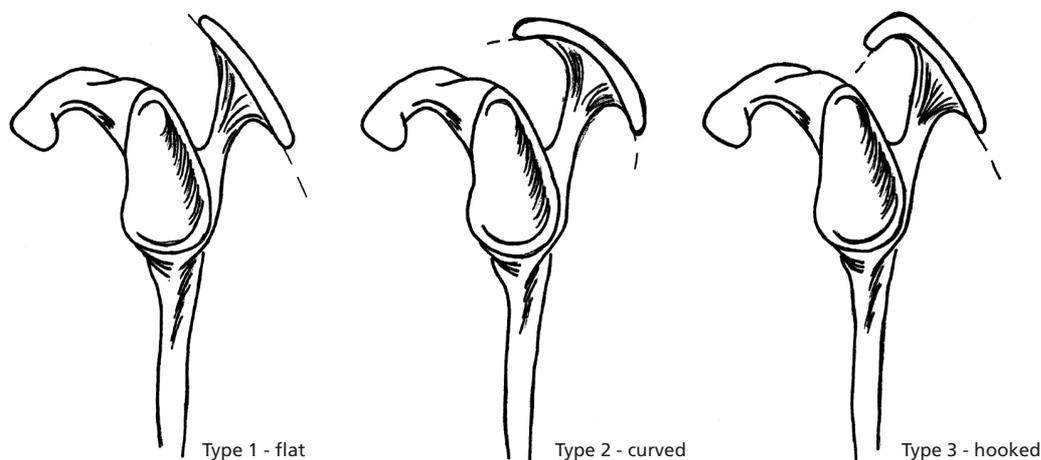


Fig 1: Os acromiale. The three defined shapes of the acromion from the lateral view

requirements have been challenged. Edelson and Taitz (1992) argued that the hook on the acromion was not an anatomical variation but represented bone spur formation due to a degenerative process. Morrison and Bigliani (1987) investigated the shape of the acromion and rotator cuff pathology in a clinical and arthrographic study of 200 patients. Although 80% of rotator cuff tears were found to occur in patients with type 3 acromions it is not possible to conclude from their findings that the hooked-shaped acromion was the cause of the rotator cuff tear.

To decompress the impinged structures Neer (1972) proposed an anterior acromioplasty. This represented a marked departure from previous surgical approaches that had advocated removing different aspects and amounts of the acromion (Armstrong, 1949; Diamond, 1964; McLaughlin and Asherman, 1951). The purpose of this procedure was to relieve the mechanical compression on the subacromial space.

Several studies have reported satisfactory results using the Neer acromioplasty procedure (Bigliani *et al*, 1989; Ellman *et al*, 1986; Ha'eri and Wiley, 1982; Hawkins *et al*, 1988; Thorling *et al*, 1985) as well as others using arthroscopic surgery to decompress the subacromial structures (Altchek *et al*, 1990; Burns and Turba, 1992; Ellman and Kay, 1991; Lazarus *et al*, 1994; Lindh and Norlin, 1993). In a prospective randomised placebo-controlled study of 125 patients with a 2^{1/2} - year follow-up, Brox *et al* (1999) reported that the success rate was higher ($P < 0.01$) for patients randomised to surgery. However this study, as with others investigating the effect of surgery for SIS, did not control the period of post-surgical relative rest with the same period and type of rest in the non-surgical interventions.

Further, according to the Neer model, the pathogenesis of impingement is due to mechanical compression by the acromion and coracoacromial arch. The disease process should therefore come to a halt as a result of decompressive acromioplasty. Hyvönen *et al* (1998) in a nine-year follow up of 96 cases of SIS found that 19 patients (20%) had developed rotator cuff tears following the surgery. Twelve of these tears were full thickness, five were joint-side and two

were bursal-side tears. These findings suggest that mechanisms other than the acromion are involved in the aetiology of SIS.

Bone Spurs on the Acromion

Edelson and Taitz (1992) examined 280 anatomical scapulae specimens. In contrast to Bigliani *et al* (1986) they concluded that acromial spurring occurring as a result of degeneration may be misdiagnosed as a type 3 acromion on radiograph. The development of an anterior spur on the acromion, a process termed enthesopathy, takes place within the substance of the coracoacromial ligament. The spur formation most probably was a secondary phenomenon resulting from the transmission of tensile forces through the ligament due to increased pressure or volume in the subacromial region (Ogata and Uhthoff, 1990). Edelson and Taitz (1992) did not find evidence of degenerative changes on the coracoid pillar of the coracoacromial ligament. They proposed that as the ligament is triangular and has a large base on insertion into the coracoid, the tensile forces would be distributed over a larger area and enthesopathic changes would not occur.

Os Acromiale

The acromion usually ossifies by the 22nd to 25th year. It is formed from three separate centres termed the pre-

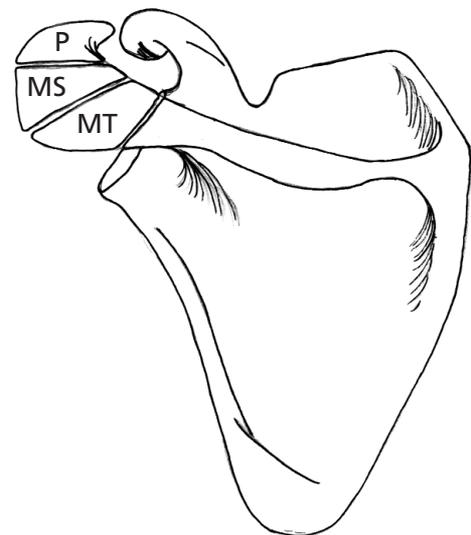


Fig 2: Os acromiale. The three ossification centres of the acromion. P = pre-acromion, MS = meso-acromion, and MT = meta-acromion. In the majority of cases, os acromiale occurs between the meso- and meta-acromion

acromion, meso-acromion and meta-acromion (fig 2). If these centres fail to unite, the ununited portion is called an os acromiale. Edelson *et al* (1993) reported an 8% incidence in an osteological examination of 270 scapulae with the length of the unfused segment varying from 1.4 cm to 2.6 cm.

It has been proposed that the loose portion of the acromion is pulled inferiorly during contraction of the deltoid, applying direct pressure and causing subsequent damage to the rotator cuff leading to impingement symptoms (Mudge *et al*, 1984). However, no study has demonstrated that os acromiale is associated with a higher incidence of SIS or is the cause of SIS in subjects presenting with both the symptoms and the condition.

Coracoacromial Ligament

Based on cadaver and surgical observations, Neer (1972, 1983) implicated the coracoacromial ligament as the cause of SIS in the absence of bony or anatomical abnormality. Soslowky *et al* (1994) examined the coracoacromial ligament of 20 cadavers, 10 with rotator cuff tears and 10 with a normal rotator cuff. They concluded that those with rotator cuff tears demonstrated a significantly shorter lateral band of the coracoacromial ligament with a larger cross-sectional area. It was hypothesised that this finding suggested the possibility of increased compression on the supraspinatus due to decreased subacromial height.

It is unknown whether this indicates that the ligament is involved in the pathogenesis of SIS, especially in the light of an earlier histological study of the coracoacromial ligament in subjects with SIS (Sarkar *et al*, 1990), which concluded that the ligament demonstrated degenerative changes suggesting chronic strain. The findings of Sarkar and his colleagues did not suggest that the coracoacromial ligament was responsible for the impingement but indicated that changes in the ligament were probably secondary, being caused by changes in the soft tissues and pathology in the subacromial space, producing strain on the ligament.

Burns and Whipple (1993) examined the shoulders of five fresh-frozen cadavers to determine the dynamic relationships between the subacromial contents and

the acromion and coracoacromial ligament. Glenohumeral movement was replicated using nylon sutures attached to the rotator cuff and deltoid and attached to a traction device. Their findings suggested that although other structures were involved, the primary location of impingement was the coracoacromial ligament. However, the design of the study involved bolting the scapula to a plywood frame and passive elevation of the humerus, and therefore did not allow for normal scapulohumeral movement or for the dynamic interplay of shoulder muscles.

In an attempt to reproduce the physiological action of muscles, Payne *et al* (1997) examined ten human cadavers to compare patterns of impingement under the acromion. Fishing line was sutured into the rotator cuff and deltoid and connected to pneumatic cylinders. Subacromial pressure was measured with miniature force transducers. Forces were applied to the rotator cuff and deltoid based on EMG data reported by Inman *et al* (1944). Their findings differed from those of Burns and Whipple (1993) with the largest pressures being recorded under the anterolateral aspect of the acromion, with only a few shoulders demonstrating pressure under the coracoacromial ligament. The failure to reproduce dynamic muscle forces may have accounted for the differences reported in these studies. Neither study reproduced the normal scapular rotation accompanying arm elevation, which is likely to have had an effect on subacromial pressures and areas of contact. Other factors, such as the effect of the pectoralis major, teres major and latissimus dorsi, which also act on the humerus and may influence contact patterns and pressures, were also not taken into account.

Posterosuperior Glenoid Impingement

Walch *et al* (1992), Jobe and Sidles (1993), Jobe (1997) and Riand *et al* (1998) proposed an alternative mechanical mechanism for the pain and dysfunction in SIS. In contrast to Neer (1972, 1983) the acromion was not implicated and the site of impingement of the supraspinatus was considered to occur between the greater tuberosity and the superior or posterosuperior aspect of the glenoid rim. This form of impingement

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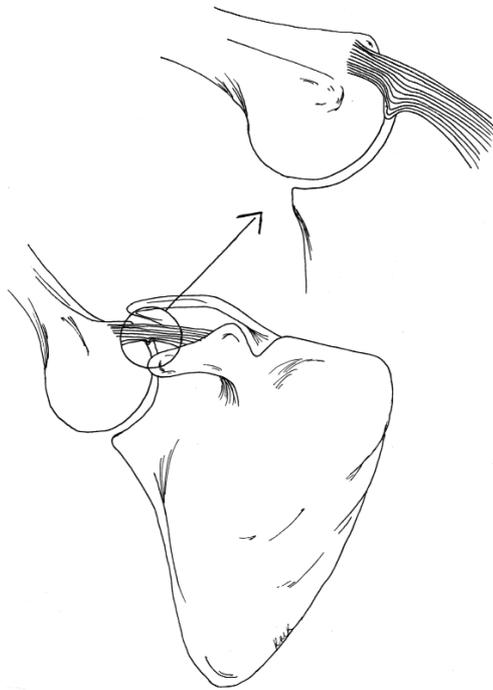


Fig 3: Superior or posterosuperior impingement. During elevation the greater tuberosity may impinge the articular-side of the rotator cuff against the superior aspect of the glenoid rim

has been termed posterosuperior glenoid impingement or internal impingement. In the impingement position (arm elevation) the articular-side fibres (those facing the humeral head) of the rotator cuff can be compressed by these two structures (Riand *et al*, 1998) (fig 3). Jobe (1997) identified that the superior aspect of the glenoid labrum as well and the origin of the long head of biceps tendon may also be compressed in this position. These structures can be impinged in abduction-external rotation (Jobe, 1997; Riand *et al*, 1998) and abduction-internal rotation (Jobe 1997).

Neer (1972) suggested that impingement caused by the acromion could be reproduced using the 'Neer sign'. In this test the clinician stabilises the scapula and passively flexes the humerus. Based on the findings of a cadaver study, Jobe (1997) argued that during the Neer sign the supraspinatus tendon has already passed under the acromion and therefore could not be compressed by it in this position. However, it could be compressed between the greater tuberosity and the superior aspect of the glenoid fossa.

Other than cadaver and surgical observations there is little evidence to support this mechanism of impingement. Dynamic observation in a functional

position in subjects with SIS would be required to confirm posterosuperior impingement as an etiological factor.

Rotator Cuff

The musculotendinous cuff formed by the subscapularis, supraspinatus, infraspinatus and teres minor cover the anterior, superior and posterior aspects of the glenohumeral joint providing static and dynamic stability. SIS may occur as a consequence of pathology of the rotator cuff (RC), which may involve over-use and degenerative changes associated with ageing (Meister and Andrews, 1993; Ogata and Uthoff, 1990).

Degenerative Tendinopathy

De Palma (1950) reported pathological findings in a series of 96 joints and found that the frequency of cuff tears, biceps degeneration and labral detachment increased with age. The most pronounced lesions were found in the seventh, eighth and ninth decades. Although this finding was supported by Ozaki *et al* (1988) it was challenged by Neer (1972), who argued that RC pathology and impingement occurred on the bursal (uppermost) side of the cuff as a result of wear and tear under the acromion.

Neer's hypothesis (1972, 1983) has not been substantiated in a number of histological studies (Ogata and Uthoff, 1990; Ozaki *et al*, 1988; Yamanaka and Matsumoto, 1994). Ozaki *et al* (1988) histologically and radiologically examined 200 shoulders taken from 65 male and 35 female cadaver specimens with a mean age of 72.3 years. They reported finding a normal RC in 104 specimens, of which 82 demonstrated a normal acromial process and 22 demonstrated spurs or osteophytes on the anterior aspect of the acromion. This finding suggested that pathological alterations in acromial shape do not necessarily lead to RC pathology.

Incomplete tears were found in 69 specimens with only 19 involving the bursal (uppermost) surface of the RC. Each of these specimens also demonstrated a lesion to the anterior one-third of the under-surface of the acromion. Twenty-three specimens were found to have incomplete tears on the deep articular side of the RC (the side facing the humeral head). In all of these specimens the under-surface of the acromion was intact. Another 27

specimens were found to have incomplete tears involving the deep and superficial sides and a further 27 were reported as having complete tears of the RC. Ozaki *et al* (1988) concluded that the majority of tears were degenerative in nature and associated with ageing.

They further argued that bursal side tears would lead to secondary acromial changes. A vicious cycle would then develop, whereby irregularities on the under-surface of the acromion could abrade the RC. However, the mechanism by which bursal side RC damage would lead to an acromial lesion was not elucidated.

Rotator Cuff Over-use

The potential for RC fatigue and failure becomes apparent when analysing the demands placed on the shoulder during certain sporting and vocational activities (Bowen and Warren, 1991; Jobe and Moynes, 1982). During these activities the shoulder may be subject to high forces, angular accelerations and angular velocities. The peak internal rotation velocity of the shoulder during baseball pitching has been reported to reach 6100°/second (Feltner, 1989; Feltner and Dapena, 1986, 1989). Dillman *et al* (1993) reported that from a position of 175° glenohumeral external rotation, the pitching arm internally rotated 80° at which point the ball was released. This movement occurred in approximately 30 milliseconds, with an average peak angular velocity of 6940°/second ($\pm 1080^\circ/\text{second}$) and clearly indicates the demands placed on the active and static structures required to stabilise and move the glenohumeral joint.

It has been argued that SIS may develop from RC overuse (Jobe and Jobe, 1983). Performing a repetitive task at near-maximal or maximal tolerance, in a sporting or vocational environment, rotator cuff fatigue may occur, surpassing the physiological limit of these structures. If the activity persists, pockets of microtrauma may develop. The soft tissue damage is thought to be associated with an inflammatory reaction in the subacromial space which may lead to pain (Calliet, 1991), and forms the basis of prescribing anti-inflammatory medications as well as corticosteroid injections to treat the condition (Hazleman, 1994; Williams, 1980).

Despite the acceptance of overuse and inflammation as being part of the pathogenesis of SIS there are few data to support this model. Histopathological findings of surgical specimens from subjects with RC tendinopathy suggest an absence of inflammatory cells (Khan *et al*, 1999). In contrast to an inflammatory model, Khan *et al* (2000) have proposed that biochemical irritants associated with tendon tissue damage may be the cause of pain.

The tendon angle of insertion of the infraspinatus, teres minor and subscapularis contributes to an inferiorly directed force on the humeral head during glenohumeral elevation (Basmajian, 1975). The contribution of the action of these fibres to glenohumeral stability is to balance the superior translation of the humeral head on the glenoid fossa caused by the deltoid during the movement of elevation. Deficiency of these oblique fibres due to overuse, trauma, pain inhibition or weakness may lead to superior translation of the humeral head due to the unopposed action of deltoid. This may increase impingement under the acromion (Lippitt and Matsen, 1993). Superior translation of the humeral head has been reported with a deficient rotator cuff (Neer, 1983; Weiner and Macnab, 1970). Massive tears, involving the inferior aspect of the rotator cuff, led to an average of approximately 8 mm superior translation of the humeral head at the initiation of abduction. This superior translation resulted in impingement of the humeral head under the acromion and an average maximum of only 18° abduction was possible (Thompson, 1996).

Instability/Hypermobility

The glenohumeral joint is a minimally constrained articulation that provides the greatest range of movement of any joint, requiring a functional balance between stability and mobility. Only 25% to 30% of the humeral head is in contact with the glenoid fossa in any position (Inman *et al*, 1944). Stability is dependent upon osseous morphology, the capsulo-ligamentous mechanism and muscular balance. Stability is provided by the interaction of static and dynamic factors whose influence varies during different positions of the glenohumeral joint. Despite its great range of movement and

limited inherent stability there are normally only a few millimetres of humeral head translation on the glenoid fossa during physiological movement (Harryman *et al*, 1990; Kaltas, 1983; Lippitt and Matsen, 1993; Post *et al*, 1979).

When glenohumeral hypermobility or instability is deemed to be the cause of the impingement symptoms it is generally referred to as secondary impingement. Secondary impingement may occur as a result of overuse and subsequent loss of the dynamic stabilisation provided by the RC (secondary tensile disease), or as a result of failure of the static stabilisers (secondary compressive impingement) or a combination of the two (Meister and Andrews, 1993).

Secondary Tensile Disease

Failure of the RC to dynamically stabilise the glenohumeral joint may lead to instability of the shoulder. Loehr *et al* (1994) examined 22 shoulders in 11 cadaver specimens (7 female and 4 male) with an average age of 58 years (range 22 to 83 years). Three potentiometers were attached to the humerus, and at right angles to each other, to measure humeral head translational movement. They reported that a single lesion to the supraspinatus did not increase glenohumeral translation. However, a large lesion also involving the infraspinatus did lead to instability.

Superior migration, as well as anterior and posterior translation of the humeral head was reported to increase. Superior translation was most noticeable at 30° of abduction. Increased glenohumeral external rotation was observed with the two-tendon lesion, indicating that supraspinatus may have a role in preventing posterior translation of the humeral head.

The results suggested that lesions of the RC increase humeral head translation, and the loss of stability may then cause mechanical impingement in a number of directions.

However, confounding factors such as a fixed scapula and the removal of other humeral head depressors, such as latissimus dorsi, may limit the ability to generalise the findings of this study.

Secondary Compressive Impingement

Failure of the static stabilisers of the shoulder may occur as a result of trauma,

over-use or an underlying hyperlaxity of the glenohumeral joint. Patients presenting with SIS secondary to hypermobility or traumatic instability have been classified as having secondary compressive (or secondary mechanical) impingement (Jobe and Moynes, 1982; Meister and Andrews, 1993). In support of this Warner *et al* (1990) reported that in their study of 53 subjects, 28 were diagnosed as having glenohumeral instability and of those, 68% (19 subjects) were found to have concurrent signs of impingement.

Restrictive Processes

Two radiographic studies (Howell *et al*, 1988; Poppen and Walker, 1976) have demonstrated that the glenohumeral articulation behaves similarly to a constrained ball-and-socket joint during movement, with the humeral head remaining within 1 mm from the centre of the glenoid fossa during movement, except at the extremes of range of motion. Gliding is an important movement as it allows the head of the humerus to be centred on the glenoid during movement to maximise stability of the glenohumeral joint (Howell *et al*, 1988).

If the glenohumeral capsule tightens then the relationship between the humeral head and the face of the glenoid will change. This may occur due to pathological processes such as adhesive capsulitis, or following surgical procedures. Operative tightening of the posterior portion of the glenohumeral capsule was performed on eight cadaver specimens (Harryman *et al*, 1990). This simulated posterior capsule contracture was found to increase the anterior and superior translation of the humeral head on the glenoid during glenohumeral flexion.

This procedure was also found to increase anterior translation during horizontal adduction. Although the magnitude of the translation varied, the direction was consistent in each of the specimens. The inference from this study was that a tight posterior capsule would push the humeral head anteriorly and superiorly during flexion and increase the likelihood of compression against the coracoacromial arch.

Shortening and adhesions of the capsule and periarticular soft tissues of the shoulder may occur as a result of trauma and immobilisation (Akeson *et al*,

1987). Experimental studies have shown that immobilisation of dense connective tissue leads to compositional and structural changes. These include a loss of extracellular water, glycoaminoglycan depletion, fibrofatty deposition and excessive collagen cross-linking (Akeson *et al*, 1977, 1987; Akeson *et al*, 1980; Baker *et al*, 1969; Enneking and Horowitz, 1972).

Cofield and Simonet (1984) reported that impingement syndrome might be confused with a 'slightly frozen shoulder'. In support of this Matsen and Arntz (1990) found impingement signs in patients with shoulder stiffness. In particular they described tightness in the posterior capsule of the glenohumeral joint. They described how this would force the humeral head anteriorly and superiorly, causing impingement against the antero-inferior aspect of the acromion during flexion. These comments were made from clinical observations and are yet to be substantiated.

Functional Scapular Instability

Lack of adequate scapular stabilisation against the thoracic cage has also been associated with SIS, as one of the primary functions of the scapula is to provide a dynamic base for the humeral head during upper limb movements (Davies and Dickoff-Hoffman, 1993; Kibler, 1991, 1998). Abnormal scapular position and movement have been termed 'lateral scapula slide' (Kibler, 1991), 'scapulo-thoracic dyskinesis' (Warner *et al* 1992) and 'floating scapula' (Kibler, 1998). Kibler (1991, 1998) has theorised that normal scapular kinematics will be compromised if the muscles controlling its position become inappropriately shortened or lengthened, or lack their normal endurance or strength requirements.

Activities such as serving in tennis, throwing a javelin, pitching a baseball and bowling in cricket require the scapula to accelerate into protraction from a fully retracted position. A lack of the normal retraction or protraction range may alter the scapular position on the thorax during these movements, which may change the desired humeral head to glenoid fossa relationship. Inadequate scapular placement may lead to increased demands on the RC, potentially contributing to over-use and degeneration

(DiGiovine *et al*, 1992; Kibler, 1998; Warner *et al*, 1992). The scapular retractors are required to slow the scapula eccentrically following protraction and if weak may be unable to do so effectively. This may place excessive demands on the static stabilisers such as the glenoid labrum, glenohumeral ligaments and capsular structures, possibly leading to glenohumeral hyper-mobility (Kibler, 1998).

Kibler (1998) has described that a scapula positioned in excessive downward rotation due to poor muscular control would reduce acromial elevation, which may lead to impingement. This might occur if the serratus anterior and the lower trapezius were weak or if the pectoralis minor and levator scapula were short. The effect of this may be to compress the structures within the subacromial space and possible symptoms of SIS (Ayub, 1991; Bowling *et al*, 1986; Calliet, 1991; Solem-Bertoft *et al*, 1993).

In addition, Kibler (1998) has reported that during the tennis serve, 51% of total kinetic energy and 54% of total force available at the shoulder are generated in the lower legs and trunk. A loss of scapular control would suggest a reduction in energy transfer and the potential for compensatory movements and pathology.

Posture

Poor cervico-thoracic posture is often associated with a forward head posture (FHP) and an increased thoracic kyphosis. FHP is defined as an anterior translation of the head in relationship with the trunk (Kendall *et al*, 1993). FHP has been implicated in the aetiology of SIS as it has been associated with muscle imbalance around the scapula, together with the added complication of the loss of thoracic extension (Ayub, 1991; Bowling *et al*, 1986; Calliet, 1991; Nicholson, 1989; Solem-Bertoft *et al*, 1993). Thoracic extension has been shown to contribute to approximately 15° of bilateral shoulder elevation (O'Gorman and Jull, 1987).

Ayub (1991) argued that FHP and an increased thoracic kyphosis will cause the scapulae to abduct (protract) resulting in a lengthening of the rhomboids and inferior trapezius, while shortening the serratus anterior, latissimus dorsi, subscapularis and teres major muscles. The protracted position of the scapulae

will further be associated with a shortening of the pectoralis major and minor. As pectoralis minor attaches to the coracoid process it will tend to pull the acromion over the head of the humerus when it shortens, causing a mechanical block to elevation of the arm. If short, the levator scapula would elevate as well as downwardly rotate the scapula, the result of which might be to apply the acromion more closely over the head of the humerus.

The belief that scapular instability and poor posture are associated with SIS has permeated into clinical thought and practice, and these theories have underpinned many of the rehabilitation programmes designed to treat SIS (Ayub, 1991; Kibler, 1991, 1998). Although many clinicians believe these factors are indispensable for normal shoulder function, considerable research is required to confirm or refute these beliefs.

Summary

Subacromial impingement syndrome is one of the most common causes of shoulder pathology (Jobe and Jobe, 1983; Kessel and Watson, 1977) with reference to the condition appearing in the

literature approximately 150 years ago (Adams, 1852). The pain and dysfunction associated with SIS are generally considered to occur when the shoulder is placed in positions of elevation, an activity commonplace during many sporting and vocational pursuits, as well as during the activities involved in daily living.

Although it has been popular to assign blame for this condition on the acromion, considerable evidence suggests that SIS is of multifactorial aetiology. Other factors include rotator cuff over-use and degeneration, glenohumeral hypermobility and instability, restrictive processes of the shoulder, functional scapular instability as well as poor posture. Many of these models of impingement still remain hypothetical and require confirmation.

Further, many other structures may refer symptoms to the shoulder and may mimic the symptoms of SIS. Each category of subacromial impingement may require its own specific treatment and rehabilitation programme. At present there is not enough evidence in the literature to determine the most appropriate assessment methods or treatment strategies for each category of impingement.

References

- Adams, R (1852).** 'Shoulder joint' in: Tobb, R B (ed) *Cyclopaedia of Anatomy and Physiology*, Longman, London, vol 4.
- Akeson, W H, Amiel, D, and Woo, S L-Y (1977).** 'Collagen cross-linking alterations in joint contractures: Changes in reducible cross-links in periarticular connective tissue collagen after nine weeks of immobilisation', *Connective Tissue Research*, **5**, 15-19.
- Akeson, W H, Amiel, D and Woo, S L-Y (1980).** 'Immobility effects on synovial joints: The pathomechanics of joint contracture', *Biorheology*, **17**, 95-110.
- Akeson, W H, Amiel, D, Abel, M F, Garfin, S R and Woo, S L-Y (1987).** 'Effects of immobilisation on joints', *Clinical Orthopaedics and Related Research*, **219**, 28-37.
- Altchek, D W, Warren, R F, Wickiewicz, T L, Skyhar, M J, Ortiz, G and Schwartz, E (1990).** 'Arthroscopic acromioplasty: Technique and results', *Journal of Bone and Joint Surgery*, **72-A**, 1198-1207.
- Armstrong, J R (1949).** 'Excision of the acromion in treatment of the supraspinatus syndrome', *Journal of Bone and Joint Surgery*, **31-B**, 3, 36-442.
- Ayub, E (1991).** 'Posture and the upper quarter' in: Donatelli, R A (ed) *Physical Therapy of the Shoulder*, Churchill Livingstone, New York, 2nd edn.
- Baker, W C, Thomas, T G and Kirkaldy-Wallis, W H (1969).** 'Changes in the cartilage of the intervertebral joints after anterior fusion', *Journal of Bone and Joint Surgery*, **51-B**, 737-746.
- Basmajian, J V (1975).** *Grant's Method of Anatomy*, Williams and Wilkins, Baltimore, 9th edn.
- Bigliani, L, Morrison, D and April, E (1986).** 'The morphology of the acromion and its relationship to rotator cuff tears', *Orthopaedic Transactions*, **10**, 228.
- Bigliani, L U, D'Alessandro, D F, Duralde, X A and McIlveen, S J (1989).** 'Anterior acromioplasty for subacromial impingement in patients younger than 40 years of age', *Clinical Orthopaedics and Related Research*, **246**, 111-116.

- Bosworth, D M (1940).** 'Analysis of 28 consecutive cases of incapacitating shoulder lesions radically explored and repaired', *Journal of Bone and Joint Surgery*, **22**, 369.
- Bowen, M and Warren, R (1991).** 'Ligamentous control of shoulder stability based on selective cutting and static translation experiments', *Clinics in Sports Medicine*, **10**, 4, 757-782.
- Bowling, R W, Rochar, P A, and Erhard, R (1986).** 'Examination of the shoulder complex', *Physical Therapy*, **66**, 1866-77.
- Brox, J I, Gjengedal, E, Uppheim, G, Bolimer, A S, Brevick, J I, Ljunggren, A E, and Staff, P H (1999).** 'Arthroscopic surgery versus supervised exercises in patients with rotator cuff disease (stage II impingement syndrome): A prospective, randomised, controlled study in 125 patients with a 2¹/₂ - year follow-up', *Journal of Shoulder and Elbow Surgery*, **8**, 2, 102-111.
- Burns, T P and Turba, J E (1992).** 'Arthroscopic treatment of shoulder impingement in athletes', *American Journal of Sports Medicine*, **20**, 13-16.
- Burns, W C and Whipple, T L (1993).** 'Anatomic relationships in the shoulder impingement syndrome', *Clinical Orthopaedics and Related Research*, **294**, 96-102.
- Calliet, R (1991).** *Shoulder Pain*, F A Davis, Philadelphia, 3rd edn.
- Codman, E A (1934).** *The Shoulder: Rupture of the supraspinatus tendon and other lesions in or about the subacromial bursa*, Thomas Todd Company, Boston.
- Cofield, R H and Simonet, W T (1984).** 'Symposium on sports medicine. Part 2: The shoulder in sports', *Mayo Clinic Proceedings*, **59**, 157-164.
- Davies, G J and Dickoff-Hoffman, S (1993).** 'Neuromuscular testing and rehabilitation of the shoulder complex', *Journal of Sports Physical Therapy*, **18**, 2, 445-458.
- De Palma, A F (1950).** *Surgery of the Shoulder*, Lippincott, Philadelphia.
- Di Giovine, N M, Jobe, F W, Pink, M and Perry, J (1992).** 'An electromyographic analysis of the upper extremity in pitching', *Journal of Shoulder and Elbow Surgery*, **1**, 15-25.
- Diamond, B (1964).** *The Obstructing Acromion: Underlying diseases, clinical development, and surgery*, Charles C Thomas, Springfield.
- Dillman, C J, Fleisig, G S and Andrews, J R (1993).** 'Biomechanics of pitching with emphasis upon shoulder kinematics', *Journal of Sports Physical Therapy*, **18**, 2, 402-408.
- Edelson, J and Taitz, C (1992).** 'Anatomy of the coraco-acromial arch: Relation to degeneration of the acromion', *Journal of Bone and Joint Surgery*, **74-B**, 4, 589-594.
- Edelson, J, Zuckerman, J and Hershkovitz, I (1993).** 'Os acromiale: Anatomy and surgical implications', *Journal of Bone and Joint Surgery*, **75-B**, 4, 551-555.
- Ellman, H, Hanker, G and Bayer, M (1986).** 'Repair of the rotator cuff: End-result study of factors influencing reconstruction', *Journal of Bone and Joint Surgery*, **68-A**, 8, 1136-44.
- Ellman, H and Kay, S P (1991).** 'Arthroscopic subacromial decompression for chronic impingement: Two- to five-year results', *Journal of Bone and Joint Surgery*, **73-B**, 3, 395-398.
- Enneking, W F and Horowitz, M (1972).** 'The intra-articular effects of immobilisation on the human knee', *Journal of Bone and Joint Surgery*, **54-A**, 973.
- Feltner, M (1989).** 'Three-dimensional interactions in a two-segment kinetic chain. Part II: Application to the throwing arm in baseball pitching', *International Journal of Sports Biomechanics*, **5**, 420-450.
- Feltner, M E and Dapena, J (1986).** 'Dynamics of the shoulder and elbow joints of the throwing arm during the baseball pitch', *International Journal of Sports Biomechanics*, **2**, 235-259.
- Feltner, M and Dapena, J (1989).** 'Three-dimensional interactions in a two-segment kinetic chain. Part I: General model', *International Journal of Sports Biomechanics*, **5**, 403-419.
- Ha'eri, G B and Wiley, A M (1982).** 'Shoulder impingement syndrome: Results of operative release', *Clinical Orthopaedics and Related Research*, **168**, 128-132.
- Harryman, D, Sidles, J A, Clark, J M, McQuade, K J, Gibb, T D, and Matsen, F A (1990).** 'Translation of the humeral head on the glenoid with passive glenohumeral motion', *Journal of Bone and Joint Surgery*, **72A**, 9, 1334-43.
- Hawkins, R J, Brock, R M, Abrams, J S and Hobeika, P (1988).** 'Acromioplasty for impingement with an intact rotator cuff', *Journal of Bone and Joint Surgery*, **70-B**, 5, 795-797.
- Hazleman, B (1994).** 'Musculoskeletal and connective tissue disease' in: Souhami, R L and Moxham, J (eds) *Textbook of Medicine*, Churchill Livingstone, Melbourne, 2nd edn, chapter 23.
- Howell, S M, Galiant, B J, Renzi, A J and Marone, P J (1988).** 'Normal and abnormal mechanics of the glenohumeral joint in the horizontal motion plane', *Journal of Bone and Joint Surgery*, **70-A**, 2, 227-232.
- Hyvönen, P, Lohi, S and Jalovaara, P (1998).** 'Open acromioplasty does not prevent the progression of an impingement syndrome to a tear: Nine-year follow-up of 96 cases', *Journal of Bone and Joint Surgery*, **80-B**, 5, 813-816.

- Inman, V T, Saunders, J B, Dec, M and Abbott, L C (1944).** 'Observations of the function of the shoulder joint', *Journal of Bone and Joint Surgery*, **26**, 1, 1-30.
- Jobe, C M (1997).** 'Superior glenoid impingement', *Orthopedic Clinics of North America*, **28**, 2, 137-143.
- Jobe, F W and Jobe, C M (1983).** 'Painful athletic injuries of the shoulder', *Clinical Orthopaedics and Related Research*, **173**, 117-124.
- Jobe, F W and Moynes, D R (1982).** 'Delineation of diagnostic criteria and a rehabilitation program for rotator cuff injuries', *American Journal of Sports Medicine*, **10**, 6, 336-339.
- Jobe, C and Sidles, J (1993).** 'Evidence for a superior glenoid impingement upon the rotator cuff' (abstract) *Journal of Shoulder and Elbow Surgery*, **3**, 2, S19.
- Kendall, F P, McCreary, E K and Provance, P G (1993).** *Muscles Testing and Function*, Williams and Wilkins, Baltimore, 4th edn.
- Kessel, L and Watson, M (1977).** 'The painful arc syndrome', *Journal of Bone and Joint Surgery*, **59B**, 166-172.
- Khan, K M, Cook, J L and Bonar, F (1999).** 'Histopathology of common overuse tendon conditions: Update and implications for clinical management', *Sports Medicine*, **27**, 393-408.
- Khan, K M, Cook, J L, Maffulli, N and Kannus, P (2000).** 'Where is the pain coming from in tendinopathy? It may be biochemical, not only structural, in origin', *British Journal of Sports Medicine*, **34**, 81-83.
- Kibler, W B (1991).** 'Role of the scapula in the overhead throwing motion', *Contemporary Orthopaedics*, **22**, 5, 525-533.
- Kibler, W B (1998).** 'The role of the scapula in athletic shoulder function', *American Journal of Sports Medicine*, **26**, 2, 325-337.
- Lazarus, M D, Chansky, H A, Misra, S, Williams, G R and Iannotti, J P (1994).** 'Comparison of open and arthroscopic subacromial decompression', *Journal of Shoulder and Elbow Surgery*, **3**, 1-11.
- Lindh, M and Norlin, R (1993).** 'Arthroscopic subacromial decompression versus open acromioplasty: A two-year follow-up study', *Clinical Orthopaedics and Related Research*, **290**, 174-176.
- Lippitt, S and Matsen, F (1993).** 'Mechanisms of glenohumeral joint stability', *Clinical Orthopaedics and Related Research*, **291**, 20-28.
- Loehr, J F, Helmig, P, Sjøbjerg, J-O and Jung, A (1994).** 'Shoulder instability caused by rotator cuff lesions: An *in vitro* study', *Clinical Orthopaedics and Related Research*, **304**, 84-90.
- Matsen, F A and Arntz, C T (1990).** 'Subacromial impingement' in: Rockwood, C A and Matsen, F A (eds) *The Shoulder*, W B Saunders, Philadelphia, vol 2, pages 623-646.
- McLaughlin, H L and Asherman, E G (1951).** 'Lesions of the musculotendinous cuff of the shoulder. IV. Some observations based upon the results of surgical repair', *Journal of Bone and Joint Surgery*, **33A**, 1, 76-86.
- Meister, K and Andrews, J (1993).** 'Classification and treatment of rotator cuff injuries in the overhand athlete', *Journal of Sports Physical Therapy*, **18**, 2, 413-421.
- Meyer, A W (1931).** 'The minuter anatomy of attrition lesions', *Journal of Bone and Joint Surgery*, **13**, 341.
- Morrison, D S and Bigliani, L U (1987).** 'The clinical significance of variations in acromial morphology', *Orthopaedic Transactions*, **11**, 234.
- Mudge, M, Wood, V and Frykman, G (1984).** 'Rotator cuff tears associated with os acromiale', *Journal of Bone and Joint Surgery*, **66-A**, 3, 427-429.
- Neer, C S (1972).** 'Anterior acromioplasty for the chronic impingement syndrome in the shoulder', *Journal of Bone and Joint Surgery*, **54A**, 41-50.
- Neer, C S (1983).** 'Impingement lesions', *Clinical Orthopaedics and Related Research*, **173**, 70-77.
- Neer, C H and Poppen, N K (1987).** 'Supraspinatus outlet', *Orthopaedic Transactions*, **11**, 234.
- Nicholson, G G (1989).** 'Rehabilitation of common shoulder injuries', *Clinics in Sports Medicine*, **8**, 633.
- Ogata, S and Uhthoff, H K (1990).** 'Acromial enthesopathy and rotator cuff tear: A radiological and histological post-mortem investigation of the coracoacromial arch', *Clinical Orthopaedics and Related Research*, **254**, 5, 39-48.
- O'Gorman, H J and Jull, G A (1987).** 'Thoracic kyphosis and mobility: The effect of age', *Physiotherapy Theory and Practice*, **3**, 154-162.
- Ozaki, J, Fujimoto, S, Nakagawa, Y, Masuhara, K and Tamai, S (1988).** 'Tears of the rotator cuff of the shoulder associated with pathological changes in the acromion', *Journal of Bone and Joint Surgery*, **70-A**, 8, 1224-30.
- Payne, L Z, Deng, X-H, Craig, E V, Torzilli, P A and Warren, R F (1997).** 'The combined dynamic and static contributions to subacromial impingement', *American Journal of Sports Medicine*, **25**, 6, 801-808.
- Petersson, C J and Gentz, C F (1983).** 'Ruptures of the supraspinatus tendon: The significance of distally pointing acromioclavicular osteophytes', *Clinical*

Orthopaedics and Related Research, **173**, 143-148.

Petersson, C J and Redlund-Johnell, I (1984). 'The subacromial space in normal shoulder radiographs', *Acta Orthopædica Scandinavica*, **55**, 57-58.

Poppen, N K, and Walker, P S (1976). 'Normal and abnormal motion of the shoulder', *Journal of Bone and Joint Surgery*, **58A**, 2, 195-201.

Post, M, Jablon, M, Miller, H and Singh, M (1979). 'Constrained total shoulder joint replacement: A critical review', *Clinical Orthopaedics and Related Research*, **144**, 135-150.

Riand, N, Levisne, C, Renaud, E and Walch, G (1998). 'Results of derotational humeral osteotomy in posterior glenoid impingement', *American Journal of Sports Medicine*, **26**, 3, 453-459.

Sarkar, K, Taine, W and Uhthoff, H K (1990). 'The ultrastructure of the coracoacromial ligament in patients with chronic impingement syndrome', *Clinical Orthopaedics and Related Research*, **254**, 49-54.

Solem-Bertoft, E, Thuomas, K A and Westerberg, C E (1993). 'The influence of scapular retraction on the width of the subacromial space', *Clinical Orthopaedics and Related Research*, **296**, 99-103.

Soslowsky, L, An, C, Johnston, S, and Carpenter, J (1994). 'Geometric and mechanical properties of the coracoacromial ligament and their relationship to rotator cuff disease', *Clinical Orthopaedics and Related Research*, **304**, 10-17.

Thompson, W (1996). 'A biomechanical analysis of rotator cuff deficiency in a cadaveric model', *American Journal of Sports Medicine*, **24**, 3, 286-292.

Thorling, J, Bjerneld, H, Hallin, G, Hovelius, L and Hägg, O (1985). 'Acromioplasty for impingement syndrome', *Acta Orthopædica Scandinavica*, **56**, 147-148.

Walch, G, Boileau, P, Noel, E and Donell, S T (1992). 'Impingement of the deep surface of the supraspinatus tendon on the posterosuperior glenoid rim: An arthroscopic study', *Journal of Shoulder and Elbow Surgery*, **1**, 238-245.

Warner, J J P, Micheli, L J, Arslanian, L E, Kennedy, J and Kennedy, R (1990). 'Patterns of flexibility, laxity and strength in normal shoulders and shoulders with instability and impingement', *American Journal of Sports Medicine*, **18**, 4, 366-375.

Warner, J P, Micheli, L J, Arslanian, L E, Kennedy, J and Kennedy, R (1992). 'Scapulothoracic motion in normal shoulders and shoulders with instability and impingement', *Clinical Orthopaedic and Related Research*, **285**, 191-199.

Weiner, D S and Macnab, I (1970). 'Superior migration of the humeral head: A radiological aid in the diagnosis of tears of the rotator cuff', *Journal of Bone and Joint Surgery*, **52-B**, 3, 524-527.

Wiles, P (1955). *Essentials of Orthopaedics*, Little Brown, Boston, 2nd edn.

Williams, J P G (1980). *Colour Atlas of Injury in Sport*, Wolfe Medical, Holland.

Yamanaka, K and Matsumoto, T (1994). 'The joint side tear of the rotator cuff: A follow-up study by arthrography', *Clinical Orthopaedics and Related Research*, **304**, 7, 68-73.

Key Messages

- Subacromial impingement syndrome has been described as the most common form of shoulder pathology.
- The pain of subacromial impingement is generally experienced around the anterolateral aspect of the shoulder during activities involving shoulder elevation.
- The aetiology of subacromial impingement syndrome is multifactorial. Each potential cause may act independently or in combination with one another.