ON THE PATHOGENESIS OF SHOULDER IMPINGEMENT SYNDROME

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Abstract

The pathomechanism of the shoulder impingement syndrome has been under debate. Two main theories of the pathogenesis of the disease exist; mechanical (extrinsic) and degenerative (intrinsic) theory.

The purpose of this work was to evaluate the pathogenesis of impingement syndrome with five studies that concentrate to aspects related to etiopathology as outcome and recovery after surgery, radiological diagnosis, immunohisto- and histopathology of subacromial bursa, and subacromial mechanical pressures.

The good results of 14 shoulders of 96 operated with an open acromioplasty turned painful after an average of 5 (2 - 10) years postoperatively and had developed 6 full-thickness and 4 partial rotator cuff tears. Initially good result is not permanent in all cases, suggesting that a degenerative process is involved in the pathogenesis of impingement syndrome.

Shoulder muscle strengths of 48 patients, who had undergone an open acromioplasty, restored to near normal within one year after open acromioplasty, suggesting that mechanical compression plays a role in the pathogenesis of impingement syndrome.

Variation in the shape of the acromion, evaluated in 111 patients and their matched controls by a routine supraspinatus outlet view, is associated with impingement syndrome, but this association is weak. Validity of this radiograph in the diagnosis of impingement syndrome is therefore a minor adjunct to the other diagnostic methods.

The role of subacromial bursa in impingement syndrome was studied in 62 patients (33 tendinitis, 11 partial and 18 full-thickness RC tear) suffering from a unilateral impingement syndrome and 24 controls. Tenascin-C proved to be a more general indicator of bursal reaction compared to the conventional histological markers, being especially pronounced at the more advanced stages of impingement.

The local subacromial contact pressures measured in 14 patients and 8 controls with a piezoelectric probe were elevated in the impingement syndrome, supporting the mechanical theory.

On the basis of this study, both mechanical and degenerative factors are involved in the pathogenesis of impingement syndrome.

Keywords: etiology, pathology, shoulder impingement syndrome, shoulder pain
To my loving wife and wonderful children
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Pekka Hyvönen
**Abbreviations**

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<thead>
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<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>CG</td>
<td>control group</td>
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<tr>
<td>EGF</td>
<td>epidermal growth factor</td>
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<td>FVIII-RAG</td>
<td>factor VIII related antigen</td>
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<td>Fi</td>
<td>Fischers exact test</td>
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<td>Fr</td>
<td>Friedmans test</td>
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<td>FTG</td>
<td>full-thickness tear group</td>
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<td>HE</td>
<td>hematoxylin eosinofil</td>
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<td>MRI</td>
<td>magnetic resonance imaging</td>
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<td>MW-U</td>
<td>Mann-Whitney-U test</td>
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<td>PTG</td>
<td>partial tear group</td>
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<td>RC</td>
<td>rotator cuff</td>
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<td>SOV</td>
<td>supraspinatus outlet view</td>
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<td>TG</td>
<td>tendinitis group</td>
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List of original publications

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Over the past few decades, shoulder impingement syndrome has become an increasingly common diagnosis (Uhthoff & Sarkar 1991). However, the syndrome was first described in the early 20th century. In 1931, Meyer (Meyer 1931) proposed that tears of the rotator cuff occurred secondary to attrition due to friction against the undersurface of the acromion and described corresponding lesions on the undersurface of the acromion and the greater tuberosity. However, he did not implicate the acromion directly. Codman, in 1934, defined the critical zone where most degenerative changes occur as the portion of the rotator cuff located one centimetre medial to the insertion of the supraspinatus on the greater tuberosity (Codman 1990). Armstrong introduced the term ‘supraspinatus syndrome’ (Armstrong 1949).

Neer described subacromial impingement syndrome as a distinct clinical entity and hypothesised that the rotator cuff is impinged upon by the anterior one third of the acromion, the coracoacromial ligament and the acromioclavicular joint rather than by merely the lateral aspect of the acromion. He also suggested that the part of the rotator cuff that is impinged upon is at the insertion of the supraspinatus tendon on the greater tuberosity (the impingement zone). The clinical diagnosis of impingement syndrome is commonly based on findings called the impingement sign and the impingement test (Neer & Welsh 1977). The patient’s history typically includes pain at night and positional discomfort called ‘painful arc’ (Calvert 1997). The clinical presentation may be confusing, and it is important to differentiate subacromial impingement syndrome from other conditions that may cause symptoms in the shoulder. Especially in young patients and athletes who perform repeated overhead motions, the diagnosis of impingement should be made with caution. In many cases, the primary diagnosis is subtle glenohumeral instability, even though impingement and subacromial bursitis may be evident (Uhthoff & Sarkar 1991).

Armstrong and Diamond (Diamond 1964) proposed that the condition should be treated with total acromionectomy as described by Watson-Jones (Watson-Jones 1960). McLaughlin and Asherman (McLaughlin 1994) developed lateral acromionectomy. However, this procedure does not involve removal of the anterior portion of the acromion, which is nowadays considered to be responsible for impingement, and it necessitates detachment of a substantial portion of the deltoide origin (Bigliani & Levine

The fact that acromioplasty relieves the impingement pain suggests the importance of the acromion in the aetiology of this disease. The shape and certain morphological angles of the acromion have been presented to be associated with the pathogenesis of impingement syndrome (Neer 1983, Aoki M et al. 1986, Bigliani et al. 1986, Toivonen et al. 1995, Tuite et al. 1995). On the other hand, the mechanical aetiology of impingement might be related to several other factors (Gruber 1863, Neer 1972, Kessel & Watson 1977, Gerber et al. 1985, Uhthoff et al. 1988, Jobe et al. 1989, Walch et al. 1992). These numerous aspects are attributed to the extrinsic theory of impingement aetiology, according to which the lesion appears purely mechanically.

The alternative for the mechanical theory of the aetiology of impingement syndrome is called the intrinsic theory. Its central idea is that impingement syndrome occurs due to the degeneration of the rotator cuff tendons (Ozaki et al. 1988, Ogata & Uhthoff 1990, Uhthoff 1996). Shoulder muscle dysfunction (Nirschl 1989) and overuse of the shoulder, which causes microtrauma of the rotator cuff tendons (Uhthoff et al. 1987), are also factors included in the intrinsic theory.

The relief of pain after subacromial decompression and the weakness of the shoulder muscle suggest that mechanical pressure is an important factor in the pathogenesis of impingement syndrome. Consequently, if impingement syndrome is due to increased subacromial pressure (extrinsic force), the good outcome of subacromial decompression should be permanent.

Shoulder muscle weakness is one of the signs associated with impingement syndrome. It has been suggested that shoulder muscle strength is restored gradually after subacromial decompression (Leroux et al. 1995), which could support the mechanical aetiology of this disease. The recovery process has not been completely clear.

It is not known whether the primary cause of symptoms is associated with a lesion in the tendon or a reaction in the bursa. At the microscopic level, increased cellularity and vascularity in the bursa near the rotator cuff tear and increased fibrosis and presence of inflammatory cells in the bursa in supraspinatus tendinitis have been reported (Uhthoff & Sarkar 1991, Santavirta et al. 1992, Rahme et al. 1993, Kronberg & Saric 1997). Thus, inflammation of the bursa has been suggested to be of importance as a source of pain in this clinical entity (Thornhill 1985, Santavirta et al. 1992). However, the role of the subacromial bursa in the pathology of impingement syndrome is not totally clear.
Widening of the subacromial space by acromioplasty (Neer 1972) relieves the impingement pain, suggesting that increased subacromial pressure is involved in the pathogenesis of impingement syndrome. Some studies have revealed high pressures in the subacromial space (Sigholm et al. 1988, Regan & Richards 1990, Wuelker et al. 1995). Acromioplasty has been found to decrease the pressure (Nordt, III et al. 1999). However, it has not been clear where exactly in the subacromial space the maximal pressure occurs.

The purpose of our studies was to find out, firstly, how permanent the effect of operative treatment for impingement syndrome is. Secondly, we wanted to discover how the shoulder muscles recover during the first year after subacromial decompression. The third matter of interest was the validity of the supraspinatus outlet view in the evaluation of the acromial shape. The role of the subacromial bursa in the pathology of impingement syndrome was studied in the fourth substudy, and in the last study, we tested the assumption that subacromial pressure is elevated under the anterolateral acromion in patients with impingement syndrome.
2 Review of the literature

2.1 Anatomy of the shoulder

The shoulder consists of three real joints. Most of the range of motion of the shoulder occurs at the glenohumeral joint. The acromioclavicular and sternoclavicular joints connect the shoulder to the trunk. In addition to these, the scapulothoracic space forms an articular-like posterior connection to the trunk and the subacromial space, which consist of the subacromial and subdeltoid bursa, acts like a joint (Fig.1.). The clavicle and the scapula together suspend the upper limb from the trunk (Carr & Wallace 1997).

The three most visible structures of the scapula are the acromion, the coracoid process and the spine. They are insertion areas for certain muscles of the shoulder. The trapezius and deltoid muscles insert to the scapular spine and the acromion. Pectoralis minor, coracobrachialis and the short head of biceps brachii insert to the coracoid process. Supraspinatus, infraspinatus and subscapularis fossae are the scapular insertion areas of muscles with corresponding names. Together with the teres minor muscle, their tendons form the rotator cuff, which covers the humeral head. The medial margin of the scapula includes the insertions of the levator scapulae and rhomboid muscles. The inferior angle and lateral margin of the scapula serve as insertions of the latissimus dorsi, teres major and minor and the long head of the triceps brachii muscle. The insertion of the long head of the biceps brachii muscle is located intra-articularly in the superior part of glenoid rim. The lateral third of the clavicle includes the insertions of the trapezium and deltoid muscles and the trapezoid and coronal ligaments, which together form the coracoclavicular ligament.

In addition to the articular surface, the humeral head has insertions of the rotator cuff tendons, the greater tubercle for the supraspinatus tendon and the lesser tubercle for the subscapularis tendon. The infraspinatus and teres minor muscles insert to the posterior surface of the humeral head, and the tendon of the long head of the biceps brachii muscle passes through the intertubercular groove over the humeral head.
2.2 Impingement syndrome

Impingement arises from mechanical compression of the rotator cuff centred primarily on the supraspinatus tendinous insertion onto the greater tuberosity against the undersurface of the anterior one third of the acromion (Neer 1983, Speer et al. 1991).

2.2.1 Anatomy of impingement syndrome

To understand the etiopathology of subacromial impingement, it is necessary to be familiar with the anatomical characteristics of the subacromial space. Within this space, a number of soft-tissue structures are situated between two rigid structures, of which the inferior structures glide relative to the superior structures. The superior border (the roof) of the space is the coracoacromial arch, which consists of the acromion, the coracoacromial ligament and the coracoid process. The acromioclavicular joint is directly superior and posterior to the coracoacromial ligament. The inferior border (the floor) consists of the greater tuberosity of the humerus and the superior aspect of the humeral head (Fig.1.).
Fig. 1. Coronal section of shoulder anatomy.

The mean height of the space between the acromion and the humeral head is 1.1 centimetres at 0 degree as seen on radiographs (Ellman 1990, Flatow et al. 1994). Interposed between the two osseous structures are the rotator cuff (mostly the supraspinatus tendon), the long head of the biceps tendon, the bursa and the coracoacromial ligament. Therefore, the true height of this space is considerably less than that seen on radiographs. Normally, the bursa facilitates the motion of the rotator cuff beneath the arch.
2.2.2 Stages of impingement syndrome

Neer described the classical three stages of impingement (Neer 1983). Stage I with oedema and haemorrhage of the bursa and cuff is typical in persons under twenty-five years old. Stage II involves irreversible changes, such as fibrosis and tendinitis of the rotator cuff, and typically occurs in patients who are twenty-five to forty years old. Stage III is marked by partial or complete tears of the rotator cuff and usually is seen in patients over forty years of age. Later, Neer divided impingement into outlet and non-outlet lesions (Neer 1990). Outlet impingement occurs when the coracoacromial arch encroaches on the supraspinatus outlet and non-outlet secondarily to thickening or hypertrophy of the bursa or the rotator cuff tendons. Subsequently, Ellman (Ellman 1990 72 /id) described a new classification based on the depth of the lesion in the rotator cuff tendons. A modification of Neer’s staging, presented by some other authors {Fukuda, Mikasa, et al. 1983 224 /id} {Fukuda, Craig, et al. 1987 225 /id} {Olsewski & Depew 1994 68 /id} {Wright & Cofield 1996 227 /id}, correlates more with the treatment options. This system classifies tendinitis and fibrosis with oedema and haemorrhage as stage I, partial tears as stage II and full-thickness tears as stage III.

2.2.3 Symptoms of impingement syndrome

Most symptoms of impingement begin insidiously and have a chronic component that progresses gradually during a period of several months. However, acute traumatic bursitis may not completely resolve and may develop into an impingement lesion (Bigliani & Levine 1997). Pain, muscle weakness, restricted ranges of motion and soft tissue crepitus are generally present (Neer 1983).

2.2.3.1 Pain

Pain is the most common symptom of the shoulder impingement syndrome (Neer 1983, Rockwood & Lyons 1993, McLaughlin 1994, Bigliani & Levine 1997). Night pain is typical, and daytime pain is related to overhead activities (Calvert 1997). Pain that originates from pathology in the subacromial region tends to be difficult to localise, is usually felt in the deltoid region and often radiates to the arm as far as the elbow (Calvert 1997). It is usually elicited between 70 and 120 degrees of abduction (Bigliani & Levine 1997). This sector is called the ’painful arc’ (Calvert 1997).
2.2.3.2 Weakness and stiffness of the shoulder

Weakness and stiffness of the shoulder may also be present, but these symptoms are usually secondary to pain (Bigliani & Levine 1997, Calvert 1997). Pain caused by the impingement may also propagate weakness by reflex inhibition of the muscles and wasting in the same fashion as the quadriceps becomes weak and wasted as the result of a painful knee (Duke & Wallace 1997). However, it has been verified by isokinetic strength measurements that prolonged impingement syndrome leads to a real decrease in shoulder muscle strength (Leroux et al. 1994, Leroux et al. 1995).

2.2.4 Signs

2.2.4.1 Impingement sign

The impingement sign, as described by Neer (Neer 1983), is elicited by performing passive shoulder flexion while preventing scapular rotation by pressing with a hand on the acromion. This causes pain, as the greater tuberosity of the humerus impinges against the acromion. Hawkins and Abrams modified this manoeuvre by rotating the humeral head at 90° of anterolateral elevation to produce a similar effect (Hawkins & Abrams 1987).

2.2.4.2 Impingement test

In the impingement test, which is a continuation to the impingement sign, 5–10 millilitres of local anaesthetic (Xylocain) is injected into the subacromial bursa. This causes relief of the pain when the impingement sign is repeated (Neer 1983).

2.3 Etiopathology of impingement syndrome

Many causes have been proposed for subacromial impingement syndrome (Aoki M et al. 1986, Bigliani et al. 1986, Codman 1990, Bigliani et al. 1991, Edelson & Taitz 1992, Burns & Whipple 1993, Hutchinson & Veenstra 1993, Davidson et al. 1995). These factors can be broadly classified as intrinsic or intratendinous factors, which are related to the intrinsic theory on the origin of impingement, and extrinsic or extratendinous factors, which are related to the mechanical theory. They can be further characterised as primary or secondary. A primary aetiology — either intrinsic or extrinsic — causes the impingement process by decreasing the subacromial space or by causing a degenerative
2.3.1 Biomechanical studies of impingement

Anatomical specimens (Nasca et al. 1984) and cadaveric models (Jerosch, Castro, et al. 1989) have been used to investigate the contact areas of the subacromial space. However, the use of anatomical specimens by Nasca et al did not allow direct clinical correlation. Wuelker et al (Wuelker et al. 1995) found that the peak forces under the acromion occurred between 85 and 136 degrees of elevation, which corresponds to the ‘painful arc’ sign. Equal results was detected in a stereo-photogrammetric analysis of cadaveric shoulders by Flatow et al (Flatow et al. 1994). They demonstrated that the acromial undersurface and the rotator cuff tendons are in closest proximity between 60 degrees and 120 degrees of elevation at the anteroinferior part of the acromion. With three-dimensional computer modelling, Zuckerman et al (Zuckerman et al. 1992) showed that the volume of the subacromial space decreased when the anterior part of the acromion was more prominent.

2.3.2 Factors of the intrinsic theory

2.3.2.1 Muscle dysfunction

It has been suggested that an intrinsic contractile tension overload on the muscle rather than primary impingement is the major factor in the aetiology of rotator cuff tendinitis (Nirschl 1989). When the arm is in the overhead position, eccentric contraction of the supraspinatus decelerates internal rotation and adduction of the arm, causing an overload (Bigliani & Levine 1997). This phenomenon is the most dramatic in persons who go in for overhead sports, and it may also occur in manual labourers who use overhead motions in their work (Bigliani & Levine 1997). The proximal migration of the humeral head has also been associated with muscle fatigue, injury and degenerative changes in the rotator cuff tendons (Jerosch et al. 1989, Leroux et al. 1994). Bigliani et al (Bigliani & Levine 1997) point out that resection of the coracocromial ligament should be avoided in this
situation because it may not relieve the impingement, but may allow for additional proximal migration of the humeral head.

Decrease in proprioceptive sense with muscle fatigue may play a role in decreasing athletic performance and in fatigue-related shoulder dysfunction (Carpenter et al. 1998). Some functional analysis of rotator cuff muscles has shown disturbances in strength in different pathological conditions, including impingement syndrome (Nirschl 1989, Warner et al. 1990, Leroux et al. 1994). Imbalance of the rotator cuff muscles in athletes, who have developed it as a result of training or sport activities, has generally been found to be a predisposing factor or a consequence of impingement syndrome (McMaster et al. 1991, Burnham et al. 1993, Ticker et al. 1995). Brox et al (Brox et al. 1993) reported that surgery and supervised exercise improved equally and significantly rotator cuff disease compared with placebo, suggesting the importance of considering this factor.

2.3.2.2 Overuse of the shoulder

The diagnosis of overuse syndrome can be made after possible extrinsic factors related to the coracoacromial arch that may contribute to the process has been ruled out (Bigliani & Levine 1997). This syndrome also occurs commonly in young competitive athletes and manual labourers who use overhead motions in their work (Bigliani & Levine 1997). Inflammation resulting from repetitive microtrauma increases the area occupied by soft tissues in the subacromial space and leads to friction and wear against the coracoacromial arch (Uhthoff et al. 1988, Jobe et al. 1989, Ark et al. 1992, McCann & Bigliani 1994). However, inflammation of the subacromial bursa may also result from a systemic disease, such as rheumatoid arthritis (Steinfeld et al. 1994, Reveille 1997). The findings of Soslowsky et al (Soslowsky et al. 2000) described in animal tendons changes that result from overuse activity, and they are believed to occur in rotator cuff tendons, too.

2.3.2.3 Degenerative tendinopathy

Ozaki et al (Ozaki et al. 1988) studied the pathological changes on the undersurface of the acromion as associated with tears of the rotator cuff in 200 cadaveric shoulders. After radiographic and histological analysis, they found that, in the specimens with a partial tear of the cuff, the undersurface of the acromion was almost intact. Although a lesion in the anterior one third of the undersurface of the acromion was always associated with a tear of the cuff, the reverse was not true. They concluded that the pathogenesis of most tears is probably a degenerative process. Ogata and Uhthoff (Ogata & Uhthoff 1990) suggested that tendon degeneration is the primary etiology of partial tears of the rotator cuff, and that they might allow proximal migration of the humeral head, which could result in impingement and lead to complete tears of the rotator cuff.
2.3.3 Factors of the extrinsic (mechanical) theory

2.3.3.1 Shape of the acromion

Acromial morphology and differences in the shape and slope of the acromion as a potential source of symptoms in the shoulder has been observed in early history (Hamilton 1875, Goldthwait 1909). Neer (Neer 1972) focused on the cause-and-effect relationship between acromial morphology and subacromial impingement. He proposed that variations in the shape and slope of the anterior aspect of the acromion were responsible for subacromial impingement and associated tears of the rotator cuff. A spur that apparently had been caused by tensile forces on the coracoacromial ligament was also found to be protruding into the subacromial space (Bigliani & Levine 1997). Bigliani and Morrison (Bigliani et al. 1986) studied 139 shoulders from seventy-one cadavers and, on the basis of direct observations and lateral radiographs, identified three types of acromial morphology: I = flat, II = curved and III = hooked. A higher prevalence of full-thickness tears of the rotator cuff was noted in association with type III acromions. In another study, they (Morrison D.S. & Bigliani L.U. 1987) evaluated supraspinatus outlet radiographs and found that 80 per cent of the eighty-two patients who had a tear of the rotator cuff visible an arthrogram had a type III acromion.

In a study of 420 cadaveric scapulae, Nicholson et al (Nicholson et al. 1996) found acromial morphology to be a primary anatomical characteristic that does not change with age. However, the prevalence of spur formation significantly increased after fifty years of age.

The classification system described by Bigliani et al (Bigliani et al. 1986) has been cited widely in the literature, but investigators have recently questioned its reliability. Zuckerman et al (Zuckerman et al. 1997) reported low interobserver reliability during the evaluation of 110 anatomic specimens to determine acromial shape according to the classification of Bigliani et al (Bigliani et al. 1986). Jacobson et al. (Jacobson et al. 1995) also reported low interobserver reliability when the system was used to evaluate acromial morphology as seen on supraspinatus outlet radiographs. They also questioned the correlation between acromial morphology and tears of the rotator cuff. The classification of acromial morphology on the basis of a subacromial outlet radiograph has been said to be difficult because of individual differences in the supraspinatus outlet angle (Duralde & Gauntt 1999). Some investigators have stated that fluoroscopic control is necessary for a proper supraspinatus outlet view (Kitay et al. 1995, Liotard et al. 1998).

Wuh and Snyder (Wuh & Snyder 1992) modified the classification system of Bigliani et al (Bigliani et al. 1986) by addressing the thickness as well as the shape of the acromion. Three types of acromion were identified: type A ( < 8 mm), type B (8–12 mm) and type C ( > 12 mm).

Toivonen et al (Toivonen et al. 1995) presented the measurement of acromial angle (Fig.5.), which is in accordance with the hypothesis proposed by Morrison and Bigliani (Morrison D.S. & Bigliani L.U. 1987) that there is an association between type III acromions and tears of the rotator cuff. Aoki et al (Aoki M et al. 1986) studied 130 cadaveric shoulders and found that acromions with spur formation had a flatter slope and
were associated with increased pitting on the surface of the greater tuberosity. They also showed that the prevalence of spurs in the subacromial space increased with advancing age and noted a decreased alpha angle (= acromial tilt)(Fig. 6.) in the patients who had impingement.

Acromial slope (Fig. 4.) and length (Fig. 7) have been studied by Edelson and Taitz (Edelson & Taitz 1992), who found that the more horizontal the acromion is, the greater are the degenerative changes. They also noted that increased degenerative changes were associated with increased length of the acromion.

Rockwood and Lyons (Rockwood & Lyons 1993) pointed out the importance of the extended anterior part of the acromion in impingement syndrome. The authors developed a modified acromioplasty that includes resection of the anterior prominence of the acromion at the level of the clavicle and removal of bone from the antero-inferior surface of the acromion. The findings of Zuckerman et al (Zuckerman et al. 1992) also support the theory that the anterior projection of the acromion is an important factor in the development of tears in the rotator cuff.

2.3.3.2 Glenohumeral instability

Especially in young competitive athletes with symptoms of impingement, it is necessary to consider underlying glenohumeral instability as the primary source of the problem (Jobe et al. 1989). Glenohumeral subluxation may cause disturbances in the mechanics of overhead motion, which may lead to secondary impingement (Glousman 1993). This concept may explain why certain throwing athletes do not show improvement after anterior acromioplasty (Jobe et al. 1989, Fu et al. 1991, Glousman 1993). The underlying instability needs to be treated either with an exercise program designed to strengthen the dynamic stabilisers or with operative intervention if the exercise program fails (Bigliani & Levine 1997). The rotator cuff muscles are important dynamic stabilisers of the glenohumeral joint. Electromyographic analysis shows that they are all active throughout the act of elevation (Matsen & Arntz 1990).

2.3.3.3 Disturbed scapulothoracic rhythm

Sportsmen, typically throwing athletes and swimmers, who suffer from impingement syndrome have been demonstrated to have dysfunction of the scapulothoracic muscles (Fu et al. 1991, McMaster et al. 1991, Warner et al. 1992, Kamkar et al. 1993, Kibler 2000). The dynamic effect of weakness on the scapular muscles is best seen when the serratus anterior muscle is involved (Duke et al. 1997). The inability to protract the scapula gives rise to winging of the scapula when the arm is raised (Weiser et al. 1999). Weak or unbalanced scapular muscles alter the scapulothoracic rhythm and place a greater strain on glenohumeral articulation, which results in secondary extrinsic impingement (Duke & Wallace 1997).
2.3.3.4 Degeneration of the acromioclavicular joint

Neer proposed that degeneration of the acromioclavicular joint may contribute to subacromial impingement (Neer 1972, Neer 1983), and a number of other authors have supported this hypothesis (Kessel & Watson 1977, Watson 1978, Petersson & Gentz 1983). Osteophytes that protrude inferiorly from the undersurface of a degenerative acromioclavicular joint can contribute to impingement when the cuff passes beneath the joint (Petersson & Gentz 1983). Kessel and Watson (Kessel & Watson 1977) found that one third of the patients in their study had lesions of the supraspinatus tendon, usually associated with degeneration of the acromioclavicular joint. Penny and Welsh (Penny & Welsh 1981) subsequently found that osteoarthritis of the acromioclavicular joint may lead to failure after operative treatment of subacromial impingement. However, resection of the acromioclavicular joint should be performed only if the patient has symptoms in the joint region and if osteophytes contribute to the impingement (Bigliani & Levine 1997).

2.3.3.5 Impingement by the coracoacromial ligament

A number of investigators (Neer 1972, Neer 1983, Uhthoff et al. 1988, Ogata & Uhthoff 1990, Burns & Whipple 1993, McLaughlin 1994) have implicated also the coracoacromial ligament as a source of impingement. McLaughlin and Asherman (McLaughlin 1994) observed the condition called “snapping shoulder” and concluded that the coracoacromial ligament was an offending structure in painful shoulders. Neer (Neer 1972, Neer 1983) included resection of the ligament as an integral part of the anterior acromioplasty procedure. Some other authors (Hawkins & Kennedy 1980, Penny & Welsh 1981, Ha'eri & Wiley 1982, Burns & Turba 1992) have reported that the coracoacromial ligament is a major component in the painful arc syndrome and have also recommended resection of the ligament. Burns and Whipple (Burns & Whipple 1993) studied five cadavers and saw that impingement occurred predominantly against the lateral free edge of the coracoacromial ligament. In a study comparing rotator cuff tear with normal specimens, Soslowsky et al (Soslowsky et al. 1996) found statistically significant changes in the geometric dimensions of the lateral band of the coracoacromial ligament, which is the region most likely to impinge on the rotator cuff. In another study, they found significant changes in the material properties (Soslowsky et al. 1994) of the ligament. Sarkar et al (Sarkar et al. 1990) and Uhthoff et al (Uhthoff et al. 1988) reported that histological studies of specimens of the coracoacromial ligament from patients who had impingement syndrome revealed only degenerative changes without thickening. They proposed that the stiffness of the coracoacromial ligament might contribute to impingement in patients who have swelling of the subacromial soft tissues.
2.3.3.6 Coracoid impingement

Coracoid impingement along the more medial aspect of the coracoacromial arch is less common, but it has been reported (Gerber et al. 1985, Dines et al. 1990, Friedman et al. 1998). In patients with coracoid impingement, the pain is usually located on the anteromedial aspect of the shoulder and is referred to the arm and the forearm. Forward elevation and internal rotation may elicit pain (Bigliani & Levine 1997). Friedman et al (Friedman et al. 1998) used cine magnetic resonance imaging to measure the interval between the coracoid process and the lesser tuberosity. In a symptomless control group, the average interval between the coracoid process and the lesser tuberosity was eleven millimetres, while in symptomatic patients, the interval was found to be six millimetres. Gerber et al (Gerber et al. 1985) reported that coracoid impingement can be idiopathic, iatrogenic or traumatic. As a choice for operative treatment, Dines et al (Dines et al. 1990) recommended coracohumeral decompression by excision of the lateral 1.5 cm of the coracoid with re-attachment of the conjoined tendon.

2.3.3.7 Os acromiale

Os acromiale is an unfused distal acromial epiphysis, and it was first described in 1863 by Gruber (Gruber 1863). Folliaison (Folliaison 1933) classified the lesion into four distinct types on the basis of anatomical location, with mesoacromion being the most common type. The prevalence of os acromiale, as reported in both radiographic and anatomical studies (Mudge et al. 1984, Edelson et al. 1993), has varied a great deal, with a range of 1 to 15 per cent. It is difficult to detect an os acromiale on a routine anteroposterior radiograph, and an axillary radiograph may thus be needed (Bigliani & Levine 1997). An association between os acromiale and impingement syndrome (Bigliani et al. 1983, Hutchinson & Veenstra 1993) and rotator cuff tears (Mudge et al. 1984) has been reported. Impingement may occur because the unfused epiphysis on the anterior aspect of the acromion may be hypermobile and may tilt anteriorly as a result of its attachment to the coracoacromial ligament (Mudge et al. 1984). Hertel et al (Hertel et al. 1998) recommended stable fusion of a sizeable and hypermobile os acromiale.

2.3.3.8 Impingement on the posterosuperior aspect of the glenoid

During the past decade, another form of impingement seen in athletes who engage in overhead activities has been reported (Walch et al. 1992, Davidson et al. 1995, Jobe 1995). Especially when the arm is placed in the throwing position (extension, abduction, and external rotation), the rotator cuff is impinged on the posterosuperior edge of the glenoid. Although this impingement is probably physiological, it becomes pathological in these athletes because of the repetitive nature of the overhead activities and the potential for increased contact secondary to fatigue of the muscles of the rotator cuff (Bigliani &
Levine 1997). The abnormal finding at arthroscopy is impingement on the posterosuperior aspect of the glenoid (Davidson et al. 1995). Jobe (Jobe 1995) suggested that anterior instability may contribute to posterosuperior impingement syndrome and that this situation may injure one or more of the following: (1) superior labrum, (2) rotator cuff tendon, (3) greater tuberosity, (4) inferior glenohumeral ligament or labrum and (5) superior glenoid bone. Recently, Riand et al (Riand et al. 2002) noticed that professional athletes, or ones competing at the international level, were not very satisfied with the outcome of arthroscopic debridement.

### 2.3.4 Role of subacromial bursa in impingement syndrome

The subacromial bursa is commonly thought of as the culprit in impingement pain (Duke & Wallace 1997). Codman stated at the beginning of the 19th century that ‘bursa like peritoneum is secondarily involved’ (Codman 1990). The pain caused by intractable impingement syndrome is often alleviated by a local cortisone injection into the subacromial bursa, suggesting that inflammation of this tissue could be a source of the impingement pain (Blair et al. 1996). Gotoh et al (Gotoh et al. 1998) found that an increased amount of substance P in the subacromial bursa appears to correlate with the pain caused by rotator cuff disease. At surgery, there are occasional signs of thickening, inflammation, fibrosis or oedema in the subacromial bursa (Duke & Wallace 1997). At the microscopic level, increased cellularity and vascularity in the bursa near the rotator cuff tear and increased fibrosis and presence of inflammatory cells in the bursa in supraspinatus tendinitis have been reported (Ulthoff & Sarkar 1991, Santavirta et al. 1992, Rahme et al. 1993, Kronberg & Saric 1997). Ide et al (Ide et al. 1996) stated that the subacromial bursa is the major component of the subacromial gliding mechanism, and they also concluded that the subacromial bursa receives nociceptive stimuli and proprioception and seems to regulate appropriate shoulder movement. However, it is not fully known whether the primary cause of impingement symptoms is associated with a lesion in the tendon or a reaction in the bursa.

#### 2.3.4.1 Fibrosis

The presence of increased subacromial bursal fibrosis has been found to correlate with impingement syndrome and its removal has also predicted a better outcome after open acromioplasty (Rahme et al. 1993, Kronberg & Saric 1997).
2.3.4.2 Inflammation

Inflammation of the bursa has been suggested to be of importance as a source of pain in impingement syndrome (Thornhill 1985, Santavirta et al. 1992, Fukuda et al. 1994). However, Uhthoff and Sarkar (Uhthoff & Sarkar 1991) showed no true acute inflammatory changes in microscopical samples of bursa from patients with impingement.

2.3.4.3 Nerves and pain mediators

Substance P is contained in primary afferent nerves, and its quantity increases during chronic pain (Lembeck et al. 1981). Gotoh et al (Gotoh et al. 1998) noticed that patients with an intact rotator cuff had more severe impingement pain than those with a torn rotator cuff, and they also had increased amounts of substance P in the subacromial bursa. Using special immunohistochemical stains and electron microscopy, Soifer et al (Soifer et al. 1996) identified neural elements within the subacromial bursa, rotator cuff tendon, biceps tendon and tendon sheath and transverse humeral ligament. There was a significantly richer supply of free nerve fibres in the bursa compared with the other tissues. Scattered free nerve endings were found throughout the subacromial bursae by Vangsness et al (Vangsness, Jr. et al. 1995). They suggested that removal of symptomatic, inflamed bursae might decrease pain signals from this part of the shoulder.

2.3.5 Surgery of the shoulder at stage II impingement syndrome

From the early 20th century until the 7th decade, the surgical procedure included quite radical resection of the acromion. (Armstrong 1949, Watson-Jones 1960, Diamond 1964, McLaughlin 1994) In the recent years, however, excessive removal of acromial bone has been associated with complications and unsatisfactory clinical results (Neer & Marberry 1981, Bigliani et al. 1992). The development of surgical techniques has led to open (Neer 1972) or arthroscopic (Ellman 1987) anterolateral acromioplasty. If non-operative treatment fails to reduce symptoms within six months, operative intervention may be indicated. Anterior acromioplasty with resection of the coracoacromial ligament is the preferred treatment (Neer 1972, Bigliani & Levine 1997).

2.3.5.1 Operative technique of open acromioplasty

Two types of incisions are recommended: bra-strap (or sabre) incision or coronal plane incision along the deltoid muscle fibres (Duke et al. 1997). The first of these is more cosmetic, while the second provides better access to the acromioclavicular joint and the
acromion. Deltoid split can be simple or more radical (Rockwood & Lyons 1993) if better exposure of the acromion or concomitant acromioclavicular resection is needed. The important thing is to leave enough good tissue to reattach the deltoid insertion (Bigliani & Levine 1997, Duke et al. 1997). The coracoacromial ligament is most commonly divided and partly resected, but because it has been thought to be an important superior support structure, some recommend reconstruction or minor detachment during acromioplasty (Duke et al. 1997). Neer proposed that the inferior part of the anterolateral acromion should be resected (Neer 1972). It can be done with either a saw or an osteotome. Rockwood modified acromial resection and recommends that the anterior projection of the acromion should also be removed from the level of the anterior border of the clavicle (Rockwood & Lyons 1993). It has been recommended (Kronberg & Saric 1997) that the fibrotic, thickened bursa, which is the probable origin of the impingement pain (Gotoh et al. 1998), should be removed (Rahme et al. 1993). However, the bursa may have a role in regenerative processes, and it has been suggested therefore to be preserved (Codman 1990). Resection of the acromioclavicular joint is not routinely performed as part of subacromial decompression and is indicated only when the joint is tender or when inferiorly protruding excrescences or osteophytes contribute to the impingement (Bigliani & Levine 1997). At the end of the operation, before the wound is closed, it is vital to obtain good reattachment of the deltoid to the acromion (Duke et al. 1997).

2.4 Outcome after open acromioplasty

The results of open acromioplasty are difficult to interpret, partly because the criteria for publication had not been carefully delineated at the time that many of the earlier studies were conducted (Bigliani & Levine 1997). In the basic study of Neer (Neer 1972), the outcome was considered satisfactory if the patient was satisfied with the operation, had no pain and had less than 20 degrees of limitation of overhead elevation and at least 75 per cent of normal strength. A number of other investigators have also reported high percentages of satisfactory results ranging from 85% to 95% in association with anterior acromioplasty (Ha'eri & Wiley 1982) (90%) (Post & Cohen 1986) (89%) (Hawkins et al. 1988) (87%) (Daluga & Dobozi 1989) (94%) (Frieman & Fenlin, Jr. 1995) (97%) (Rockwood & Lyons 1993) (87%). Some reports suggest that the outcome may be related to the worker’s compensation status or pending litigation (Post & Cohen 1986, Hawkins et al. 1988, Ogilvie-Harris et al. 1990). The same factor has also been found to be predictive in cases with rotator cuff repair (Vastamaki 1986).

A higher percentage of unsatisfactory results has been reported in some other studies (Thorling et al. 1985, Sahlstrand 1989, Bjorkenheim et al. 1990). Thorling et al (Thorling et al. 1985), after an average follow-up of twenty months (range six to forty-two months), reported that thirty-nine patients (76%) were satisfied with the outcome. The authors also concluded that the prognosis is worse for patients who undergo concomitant resection of the acromioclavicular joint. This tendency was not reported in another study (Daluga & Dobozi 1989), in which resection of the acromioclavicular joint had also been performed in every fourth of the cases. Sahlstrand (Sahlstrand 1989) reported, using another rating
scale after an average follow-up of only eleven months, that 77% of 52 cases had an excellent or good outcome. Bjorkenheim et al (Bjorkenheim et al. 1990) used the functional assessment of Neer (Neer 1972) and reported excellent or satisfactory results in 73% of the cases after an average follow-up of forty-eight months. Tibone et al (Tibone et al. 1985) reported, in a study of thirty-three athletes (thirty-five shoulders) who were less than forty years old, an excellent or good outcome for only fourteen patients (42%). The outcome was even worse for the athletes who were involved in pitching or throwing, which may be due to primary instability of the shoulder (Jobe et al. 1989, Glousman 1993). However, Bigliani et al (Bigliani et al. 1989) reviewed retrospectively twenty-six patients who were less than forty years old with an average of 33 months of follow-up after anterior acromioplasty for the treatment of subacromial impingement syndrome. 96% of them reported subjective improvement after the procedure. Seven of the ten recreational athletes had a satisfactory outcome.

2.5 Recovery of shoulder muscle strength after subacromial decompression

Recovery of strength in the shoulder muscles after operative treatment of rotator cuff tears has been found to be a slow process lasting for up to one year according to isokinetic studies (Walker et al. 1987, Rabin & Post 1990, Rokito et al. 1996). Rabin and Post (Rabin & Post 1990), who evaluated both rotator cuff tear patients (52) and subjects with an intact rotator cuff (21), showed that muscle recovery does not correlate fully with the clinical assessment, which shows early improvement. Endurance of the muscles may decrease for a longer period. The results of Leroux et al (Leroux et al. 1995) indicate that surgery restores the normal muscular balance between shoulder rotator muscles affected by impingement syndrome.

2.5.1 Measurement of shoulder muscle strengths

Most studies have involved measurement of the strength of the rotator cuff muscles isokinetically with slow (60°/sec) and fast (180°/sec) torque arm speed (Ivey, Jr. et al. 1985, Walker et al. 1987, Leroux et al. 1994, Holm et al. 1996). Leroux et al (Leroux et al. 1995) made their measurements of postoperative scapular muscle functions after a long (mean 44.5 months) period of follow-up following surgery for stage II and III impingement syndrome, to avoid the possible effect of pain. Pain inhibition has turned out to be significant in the isokinetic shoulder muscle testing (Ben Yishay et al. 1994). Hand dominance does not have any significant effect on shoulder muscle strengths in isometric measurements (Ivey, Jr. et al. 1985). Isometric measurement has been considered an equally valid method of measuring the strength of the shoulder muscles as isokinetic measurements (Gore et al. 1986, Kuhlman et al. 1992). Gore et al (Gore et al.
Kuhlman et al. (1992) recommended that the isometric strength of external rotation should be measured at 45° of abduction and 45° of internal rotation, and the strength of abduction in the scapular plane with the shoulder at 45° of abduction.

### 2.6 Plain radiography in the evaluation of acromial shape and subacromial space

Anteroposterior radiographs may help in identifying abnormalities, such as osteoarthritis of the acromioclavicular joint, calcific tendinitis, evidence of glenohumeral instability (osseous Bankart lesion or Hill-Sachs lesion), tumours and osteoarthritis of the glenohumeral joint (Bigliani & Levine 1997). In making the diagnosis of subacromial impingement, anteroposterior radiographs may show subchondral cysts or sclerosis of the greater tuberosity with corresponding areas of sclerosis or spur formation on the anterior edge of the acromion (Cone, III et al. 1984, Gold et al. 1993). An axillary radiograph may be needed to diagnose an unfused acromial epiphysis (Os acromiale) (Edelson et al. 1993). Neer and Poppen (Neer & Poppen 1987) described the supraspinatus outlet view (SOV), which is a lateral radiograph taken in the plane of the scapula with the x-ray beam directed 10 degrees caudal. The supraspinatus outlet radiograph has been widely used in the diagnosis of subacromial impingement. However, the findings may be difficult to reproduce consistently because of thoracic kyphosis or superimposition of adjacent osseous structures, such as the clavicle, ribs or scapular body (Bigliani & Levine 1997). Variations of the supraspinatus outlet angle (Duralde & Gauntt 1999) and a high rate of interobserver error (Jacobson et al. 1995) have been reported, suggesting inaccuracy of SOV. Ono et al (Ono et al. 1992) described a 30-degree caudal tilt anteroposterior radiograph that demonstrates the anteroinferior projection of the acromion. Andrews et al (Andrews et al. 1991) suggested the use of a profile radiograph to facilitate evaluation of the lateral aspect of the acromion.

### 2.7 Tenascin-C as an indicator of tissue reactions

Tenascin-C is a disulfide-bonded hexamer composed of subunits with molecular weights in the range of 120–300 kD, depending on the expression of different isoforms in different species. The subunits contain epidermal growth factor (EGF)-like and fibronectin-like repeats commensurate with the growth-promoting properties of tenascin-C (Engel 1989, Swindle et al. 2001).
2.7.1 Tenascin-C expression in normal tissue

Tenascin-C, the proteotypical tenasin, was originally isolated from embryonic tissues (Chiquet-Ehrismann et al. 1986, Erickson & Bourdon 1989). It is the most widely distributed tenasin and is typically seen only at sites of epithelial-mesenchymal interaction in mature tissues. It is involved in various cellular functions, such as growth promotion, hemagglutination, immunosuppression of T-cells, promotion of angiogenesis and chondrogenesis. It also has an anti-adhesive effect on many cell types (Fischer et al. 1997).

2.7.2 Tenascin-C expression in different pathological situations

A high level of expression of tenascin-C in acute injury is well established, and it may possibly play an important role in ensuring both inflammatory and reparative processes at the site of injury (Mackie et al. 1988, Dalkowski et al. 1999). Prominent induction of tenascin-C expression is seen in diverse reactive conditions, such as inflammation and wound healing (Koukoulis et al. 1991), and in the stroma of various carcinomas (Mighell et al. 1996, Riley et al. 1996, Kostianovsky et al. 1997, Lohi et al. 1998). The expression of tenascin-C in different tissues varies, depending on the developmental stage of the organism analyzed. It changes dramatically under various pathological conditions, such as tumours, tendon degeneration, synovitis, colitis, colon adenoma and colorectal carcinoma, pathological bone marrow and interstitial pneumonia (Erickson & Bourdon 1989, McCachren & Lightner 1992, Riedl et al. 1992, Soini et al. 1993, Riley et al. 1996, Kaarteenaho-Wiik et al. 1996). Elevated expression of tenascin-C is seen in active reparative processes, such as in wound healing, inflammatory lung diseases and keloids (Mackie et al. 1988, Dalkowski et al. 1999, Kaarteenaho-Wiik et al. 2000).

2.8 Subacromial pressure

Subacromial contact pressure has been studied in cadaveric experiments (Jerosch, Castro, et al. 1989 120 /id\{Regan & Richards 1990\}(Wuelker et al. 1995). They found increased pressures under the acromion and coracoacromial arch upon forward elevation, but few clinical correlations emerged.

Sigholm et al (Sigholm et al. 1988) evaluated with the microcapillary infusion (MCI) technique the pressure in the subacromial bursa in 30 shoulders in healthy volunteers. Elevated bursal pressure was detected upon lifting up the arm, and it was more prominent when a 1kg weight was held up. They found the MCI method also suitable for recording pressure in the subacromial bursa during exercise. This method has not got much support, because the subacromial space is not enclosed and the fluid pressure does not necessarily
reflect contact pressure (Nordt, III et al. 1999). Further, no local pressures can be measured with this method.

The study of Nordt et al (Nordt, III et al. 1999) presents that subacromial pressures were highest in the patients who had type III acromial morphology. They also pointed out that fully abducted and cross-reach positions generate the highest impingement pressures. Acromioplasty decreased significantly the anterolateral edge subacromial pressures. There were technical difficulties with the catheter placement, and no measurement was possible in active movements.
3 Aims of the study

The studies of the thesis focused on the pathogenesis of impingement syndrome. The aim was:

1. to assess whether the outcome of open acromioplasty is permanent in the long run, supporting the mechanical theory, or whether the outcome deteriorates over time, indicating a degenerative pathomechanism.

2. to evaluate if the strength of the shoulder muscles is restored soon after open acromioplasty, indicating the relief of mechanical or pain inhibition, or whether the muscles remain deteriorated, indicating a more permanent lesion.

3. to study if the shape of the acromion evaluated by plain radiography is related to the impingement syndrome and its stages, which would be in line with the mechanical theory.

4. to investigate the role of the subacromial bursa in the pathogenesis of impingement syndrome using tenascin-C expression and histological findings as parameters of tissue reaction.

5. to study if the pressure conditions and distribution in subacromial space are altered in shoulders with impingement in accordance with the mechanical theory.
4 Material and methods

4.1 Patients

4.1.1 Late results of open acromioplasty

The study population consisted of the 102 patients who had had open acromioplasty during 1977–1986 for chronic (6–180 months, mean 39 months) impingement syndrome (Neer 1983, Hawkins et al. 1988, Jalovaara et al. 1989). Two of them subsequently died, and 7 patients could not be reached for the study. Thus, 96 shoulders (36 female, 60 male; 62 right, 34 left; 67 dominant, 23 non-dominant, 6 ambidextrous, 3 bilateral) of 93 patients with a mean age at operation of 45 years (26–69) were available for the study.

4.1.2 Recovery of shoulder muscle strengths

Recovery of the strength of the shoulder muscles was studied in 48 patients (21 female, 27 male). Their mean age was 44 years at operation (range 18–58), and they had undergone open acromioplasty because of stage II impingement syndrome between 1989 and 1994. No prior surgery or pain in the opposite shoulder was reported. All patients (45 right-handed, 3 ambidextrous, 31 dominant, 17 non-dominant) had a positive impingement test (Neer 1983) and an aggravated impingement sign (Neer 1983, Hawkins et al. 1988) preoperatively. There were no signs of rotator cuff tear in ultrasonographic examination (35 cases), arthrography (17 cases), MRI (3 cases) and open surgery.
4.1.3 Acromial morphology as analysed by supraspinatus outlet view

Supraspinatus outlet view (Neer & Poppen 1987) and standard shoulder roentgenograms were taken before surgery from 137 shoulders (133 patients) with stage II or stage III impingement syndrome. None of the patients had had prior subacromial surgery. Staging of the impingement syndrome was based on findings in open (111 cases) or arthroscopic surgery (28 cases). Ninety cases presented with tendinitis-stage impingement syndrome (stage II) and 47 with a full-thickness rotator cuff (RC) tear (stage III). Twenty-six SOVs (16 in the tendinitis group and 10 in the RC tear group) were excluded because of inadequate quality. Thus, 111 shoulders (74 stage II and 37 stage III) were available for study. For controls, similar roentgenograms were obtained from both shoulders of 75 voluntary patients and 84 members of hospital staff who had had no shoulder problems or shoulder surgery. After excluding 5 control SOVs because of inadequate quality, the SOVs of 313 shoulders were available for matching.

4.1.4 Bursal reaction in different stages of impingement syndrome evaluated by tenascin-C expression and histology

Tissue samples were taken from the subacromial bursa during open subacromial surgery of 62 patients (39 males, mean age 47, range 26 to 70 years, and 23 females, mean age 50, range 37 to 70 years) suffering from unilateral impingement syndrome. Thirty-three of these patients had tendinitis (11 females and 22 males, mean age 43, range 26 to 54 years, tendinitis group = TG). Eleven had a partial (2 joint side, 7 bursal side and 2 intratendinous) tear (3 females and 9 males, mean age 50, range 35 to 70 years, partial tear group = PTG). Eighteen had a full-thickness rotator cuff tear (9 females and 9 males, mean age 55, range 45 to 67 years, full-thickness tear group = FTG). The preoperative duration of symptoms of positional discomfort and night pain was more than six months in all cases.

Tissue samples were also taken from 20 shoulders of 12 cadavers (10 males and 2 females, mean age 49, range 35 to 76 years). The samples from three shoulders were excluded due to a partial joint side tear and one due to a shoulder prosthesis. In addition, bursal biopsies were taken from the shoulders of four males operated on for traumatic acromioclavicular joint dislocation (mean age 36, range 26 to 44 years) who had not had any previous shoulder problems. The samples of these two groups were combined and served as a control group = CG (N = 24, mean age 46, range 26 to 76 years).

4.1.5 Measurement of the subacromial pressure

Measurement of local subacromial contact pressures was done on 14 patients (7 male, 7 female; mean age 45 years; range: 33 to 55 years) who underwent acromioplasty for stage
II impingement syndrome (Neer 1983). Eight patients (7 male, 1 female; mean age 36, range 27 to 45 years) undergoing surgery for complete acromioclavicular dislocation (Jalovaara, Paivansalo, et al. 1991 195 /id), of which 7 were acute and one chronic, served as controls.

4.2 Methods

4.2.1 Clinical follow-up and radiological examination of the rotator cuff

At follow-up a mean of nine (6–15) years after open acromioplasty, a single investigator performed the clinical examinations and interviewed the patients. The subjective outcome was evaluated by using the method described by Thorling et al (Thorling et al. 1985). Ultrasound examination and routine x-rays were performed on all shoulders. Magnetic resonance imaging (MRI) and/or single contrast arthrography were performed if there were any signs of RC pathology in the ultrasound examination or if the shoulder was painful (subjective outcome poor or fair).

4.2.2 Measurement of shoulder muscle strengths

Isometric strengths of flexion (Fig. 2a), abduction (Fig. 2b) and external rotation (Fig. 2c) were measured by a single physiotherapist at less painful 0° of flexion and abduction of the shoulder and at 90° flexion of the elbow preoperatively (n = 25) and three months (n = 33), six months (n = 13) and one year (n = 43) postoperatively. After warming up by performing the maximal range of motions, the patient was familiarised with the equipment and three attempts at maximal isometric contraction were made for each muscle group, with a brief rest between the attempts. The means of these values were used for analysis. In order to prevent associated movement of the trunk during strength testing in an upright position, the patients were stabilised with a strap around the chest. An isometric force meter was used (Newtest Force™, Newtest Co. Oulu, Finland) (Fig. 2a–c).
4.2.3 Technique of supraspinatus outlet view and true AP view

The supraspinatus outlet view (Neer & Poppen 1987) was obtained in a standing position, with the patient facing the film and the coronal shoulder line turned outwards 45 degrees. The arm of the involved side was in a neutral position. The x-ray beam was pointed along
the scapular axis with a 15° caudal tilt (Rockwood et al. 1990). The tube-screen distance was 100 cm, and the x-ray voltage was usually 60–65 kV.

The true anteroposterior radiographs were obtained by angling the x-ray beam 45° from medial to lateral (Rockwood et al. 1990).

4.2.4 Analysis of roentgenograms

Supraspinatus outlet views were first classified for acromion morphology as type I (flat), II (curved) or II (hooked) solely on the basis of the criteria established by Bigliani et al (Bigliani et al. 1986) (Fig. 3).

Fig. 3. Three types of acromion. (From Bigliani L U.: Impingement syndrome, Aetiology and overview. In: Surgical disorders of the shoulder, p.237. Edited by M.S. Watson. Churchill Livingstone, New York 1991.) A) Flat = Type I. B) Curved = Type II. C) Hooked = Type III.
The acromial shape (curvature) was also evaluated by measuring the acromial slope as described by Bigliani et al (Bigliani et al. 1986) and Kitay et al (Kitay et al. 1995) (Fig. 4).

**Fig. 4.** Measurement of acromial slope ($\delta$). A = anteroinferior point of the acromion. B = posteroinferior point of the acromion. D = middle point of the inferior surface of the acromion. $\delta$ = slope angle.

In addition as a method of quantitating the shape of the anteroinferior acromion, the acromial angle ($\alpha$) was measured as described by Toivonen et al (Toivonen et al. 1995)

**Fig. 5.** Measurement of the acromial angle ($\alpha$). A and B as in fig. 3. C = turning point (junction) of the inferior surface of the hooked acromion. $\alpha$ = acromial angle.
Measurement of acromial posture in relation to the scapula was based on the method described by Aoki et al (Aoki M et al. 1986) and Kitay et al (Kitay et al. 1995) (Fig. 6).

![Diagram of acromial tilt angles](image1)

**Fig. 6.** Measurement of acromial tilt angles (β and γ). A and B as in Figs. 3 and 4. E = inferior coracoid curve. F = inferior tip of the coracoid process. β and γ = tilt angles.

The length of acromion was measured between the most anterior and posterior points of the acromion, and the thickness of the acromion was measured from two points: the anterior part and the middle acromial body (Fig. 7).

![Diagram of acromial dimensions](image2)

**Fig. 7.** Measurements of acromial dimensions. Length: Distance between the points A (most anterior) and B (most posterior) of the acromion. Thickness of the anterior (line 1) and middle (line 2) parts of the acromion.

The true AP views were examined to rule out malignancies, rotator cuff arthropathy and advanced osteoarthritis of the glenohumeral and acromioclavicular joints, but no cases had to be excluded for these.
4.2.5 Immunohistochemical and histological methods in the evaluation of subacromial bursa

4.2.5.1 Biopsies and preparation of samples

The specimens for examinations of the subacromial bursa were taken at open acromioplasty (Neer 1983) during acromioclavicular reconstruction (Jalovaara et al. 1991) or at autopsy. The deltoid muscle was detached from the anterior part of the acromion, and the samples were taken from the deltoid side of the subacromial bursa. For histological and immunohistochemical investigations, the tissue samples were fixed in 10 percent neutral formalin and embedded in paraffin. 5-µm thick sections were cut and stained with hematoxylin and eosin and Herovici stains.

4.2.5.2 Tenascin-C expression

Tenascin-C expression was determined by an immunohistochemical method. The 5-µm thick sections were deparaffinised in xylene and rehydrated in graded alcohol. The endogenous peroxidase was consumed by incubating the sections in 0.3% hydrogen peroxide in absolute methanol for 30 min. The sections were then treated with 0.4% pepsin (Merck, Darmstadt) at 37°C for 30 minutes. To visualise tenasin, a monoclonal mouse antibody to human tenasin (143DB7) was employed (Tiitta et al. 1992). In order to visualise the microvasculature, a rabbit antibody to human anti-factor VIII related antigen (FVIII-RAG), a well-established marker for endothelial cells (Miettinen et al. 1983) (Dako A/S, Glostrup, Denmark), was used. The sections were incubated with the primary antibody at 4°C overnight, followed by biotinylated rabbit anti-mouse IgG antibody (to visualise tenasin) and biotinylated goat anti-rabbit IgG (to visualise FVIII-RAG). As a further amplification step, the avidin-biotin peroxidase complex (Dako A/S, Glostrup, Denmark) was applied. The peroxidase reaction was developed with diaminobenzidine. Thereafter, the sections were counterstained with a light hematoxylin stain, mounted in an aqueous medium and overlaid with glass coverslips for viewing under a light microscope. Agfapan 400 film was used for recording the images. The amount of tenascin-C in the tissue was determined by analysing anti-tenascin-stained tissue sections. The evaluation was done on a semiquantitative scale ranging from 1 to 3, corresponding to the abundance of tenascin-C seen in the samples with a minimum amount ( = 1, < 33% of the area of the specimen), an intermediate amount ( = 2, 33–66%) and a maximal amount ( = 3, > 66%).
4.2.5.3 Thickness

The thickness of the bursal tissue was estimated visually under a microscope by using a semiquantitative scale: 1 = normal, 2 = moderately thickened, 3 = markedly thickened.

4.2.5.4 Fibrosis

A semiquantitative scale ranging from 0 = no fibrosis to 1 = moderate and 2 = extensive fibrosis was used to estimate the extent of fibrosis in the bursa. This was based on an evaluation of the histological sections in which reactive fibrosis could be identified, especially in Herovici stain.

4.2.5.5 Vascularity

The assessment of the degree of vascularity and neovascularisation was based on immunohistochemical staining with anti-FVIII-RAG antibodies. The abundance of vasculature was estimated based on the number of FVIII-RAG-positive vascular profiles projected on a semiquantitative scale of 1 to 3, as described for tenascin.

4.2.5.6 Hemorrhage

The degree of hemorrhage was estimated based on a microscopic examination of HE-stained sections on a scale of 1 to 3, as described for tenascin.

4.2.5.7 Inflammatory cells

The degree of inflammatory cell reaction was based on an examination of HE-stained sections and evaluation on a scale ranging from 1 to 3, as described for tenascin.

4.2.5.8 Evaluation of the samples

Two pathologists evaluated the immunohistochemical stainings independently, and a consensus was negotiated in the cases where their opinions differed.
4.2.6 Measurement of subacromial pressure

The pressures of the subacromial space were recorded from four locations in the subacromial space (Fig. 8.).

1) Anterolateral; tip of the acromion as close to the borders as possible, 6 mm from the lateral and anterior borders.
2) Anteromedial; 6 mm distal to the acromioclavicular joint and posterior to the anterior border of the acromion.
3) Posterolateral; 25 mm posterior to point 1.
4) Posteromedial; 25 mm posterior to point 2.

Fig. 8. Points of subacromial pressure measurements.

At each measurement point, the recordings were made with the arm abducted in the coronal plane at 0°, 30°, 60° and 90°. An assisting person achieved the positions of the arm passively without any traction.

4.2.7 Statistical analysis

The results were analysed by SPSS versions 8.0 (I–IV) or 10.0 (V) for Windows (SPSS Inc., Chicago, Illinois). Student's t-test (I–III and V) for paired samples, Fisher’s exact test (IV and V), Mann-Whitney U-test (V) and Friedman’s non-parametric test (V) were used in the statistical analysis. Non-parametric Kendall’s correlation coefficient was calculated in the bivariate correlation analysis (IV). Because the data of study V included excess zeros, the two-part model described by Lachenbruch {Lachenbruch 2002 229 /id} was applied. Fisher’s exact (Fi) test was used for dichotomous data and Student’s t-test or Mann-Whitney U-test (MW-U) for continuous data. Friedman’s non-parametric test (Fr) was used to compare the differences between different locations, where the recordings at all abduction angles were considered. A level of p < 0.05 was used to determine significant differences.
5 Results

5.1 Long-term results of open acromioplasty and rotator cuff pathology

The overall subjective outcome was excellent in 45, good in 24, fair in 18 and poor in 9 shoulders. Nineteen (20%) RC tears were found (Table 1.), twelve of which were complete. Six cases were diagnosed by using MRI + ultrasound, 2 cases by using MRI + ultrasound + arthrography, 3 cases by using ultrasound + arthrography and 1 case by using MRI + arthrography. The 5 joint side partial RC tears and the 2 bursal side partial RC tears were diagnosed by ultrasound and MRI. Rotator cuff tears were more common in men (25%) (N = 60, mean age at operation 45 (26 to 69) years) than in women (11%) (N = 36, mean age at operation 46 (27 to 61) years) (Table 1.). The tear rate was higher in the groups with fair and poor results than in those with good and excellent results, being 4% in excellent, 25% in good, 33% in fair and 55% in poor cases (Table 1.). Ultrasound showed signs of partial RC tear in 4 more shoulders with a good or excellent outcome. Three subjects refused further examination (not counted as RC tear here), and one had no pathological findings in MRI. The mean age at the time of the operation in the 12 cases with a total RC tear had been higher (mean 50, range 37–58) compared to the 7 cases with a partial RC tear (mean 45, range 37–54) (p = 0.090) or to the remaining 77 affected shoulders (mean 45, range 26–69) (p = 0.034).

Fourteen shoulders initially symptom-free after acromioplasty (mean age at operation 46 years (range 34 – 57) turned painful at an average of 5 (2 – 10) years postoperatively. The subjective outcome for 6 of these shoulders was still good, however, while 6 had a fair and 2 a poor outcome. Six shoulders (3 good, 3 fair) had a complete tear and 4 (2 good, 2 poor) a partial tear (71%) (Table 1).

In 4 patients, no rotator cuff tear could be detected in US. Two of them refused further examinations and the reason for impairment thus remained obscure. One shoulder (fair) displayed a marked spur at the inferior margin of the acromion and thinning of the supraspinatus tendon in MRI, while the other (fair) had developed marked osteoarthritis and the patient had had shoulder hemiarthroplasty 8 years after acromioplasty.
Table 1. Mean (range) Constant functional shoulder scores and ages of the patients in the different groups and respective pain-free unaffected shoulders (controls).

<table>
<thead>
<tr>
<th></th>
<th>Affected shoulders</th>
<th>Unaffected shoulders</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean Constant score (range)</td>
<td>Number of cases</td>
</tr>
<tr>
<td>All cases</td>
<td>70 (24–100)</td>
<td>96</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>74 (26–100)</td>
<td>60</td>
</tr>
<tr>
<td>Female</td>
<td>64 (24–90)</td>
<td>36</td>
</tr>
<tr>
<td>Rotator cuff tear</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>73 (24–100)</td>
<td>77</td>
</tr>
<tr>
<td>Partial</td>
<td>64 (26–91)</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>59 (33–88)</td>
<td>12</td>
</tr>
<tr>
<td>Surgical technique</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excision of distal clavicle</td>
<td>61 (32–100)</td>
<td>21</td>
</tr>
<tr>
<td>No clavicular resection</td>
<td>72 (24–98)</td>
<td>75</td>
</tr>
<tr>
<td>Initial outcome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excellent</td>
<td>82 (55–100)</td>
<td>45</td>
</tr>
<tr>
<td>Good</td>
<td>66 (33–86)</td>
<td>24</td>
</tr>
<tr>
<td>Fair</td>
<td>52 (26–71)</td>
<td>18</td>
</tr>
<tr>
<td>Poor</td>
<td>49 (24–87)</td>
<td>9</td>
</tr>
<tr>
<td>Late outcome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deteriorated</td>
<td>60 (36–84)</td>
<td>14</td>
</tr>
</tbody>
</table>

5.2 Recovery of shoulder muscle strengths after open acromioplasty

5.2.1 Flexion

The mean preoperative flexion strength of the involved shoulder was 72.6% of that of the uninvolved shoulder and increased to 77.1% by the third postoperative month, to 88.3% by the sixth and by the 12th month (Table 2.). The difference was statistically significant between the preoperative condition and that at 12 months (p = 0.04). Recovery was more distinct when the poor cases (n = 7) were not included in the analysis, the difference being significant at six (p = 0.02) and 12 months (p = 0.01). The strengths of male shoulders showed no statistically significant recovery, but in the female shoulders the difference between the preoperative condition and that at six months (p = 0.02) and 12 months (p = 0.01) was statistically significant.
5.2.2 Abduction

The recovery of abduction strength in the whole group showed a similar trend compared to flexion (Table 2.). The difference between the preoperative and 12-month results was statistically significant (p = 0.01). Here, too, recovery was more distinct and already significant after three months, when the poor cases (n = 7) were excluded from analysis. No significant recovery was observed in the male shoulders. In the female shoulders, the difference in abduction strength between the preoperative condition and that at six months (p = 0.01) and 12 months (p = 0.00) was statistically significant.

5.2.3 External rotation

In the whole group, the recovery of external rotation strength was statistically significant between the preoperative condition and that at six months (p = 0.04) and 12 months (p = 0.03), (Table 2.). Again, recovery was more distinct when only the cases with satisfactory outcome (n = 41) were considered. The recovery of male shoulders was not statistically significant, but female shoulder strength increased significantly between the preoperative condition and that at 12 months (p = 0.02).
Table 2. Shoulder strengths in the different groups preoperatively and at different times postoperatively. Values are percentages of the uninvolved sides. (n = number of observations)

<table>
<thead>
<tr>
<th></th>
<th>Flexion</th>
<th></th>
<th>Abduction</th>
<th></th>
<th>External rotation</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>SD</td>
<td>mean</td>
<td>SD</td>
<td>mean</td>
<td>SD</td>
</tr>
<tr>
<td><strong>Preoperative</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All cases</td>
<td>72.6%</td>
<td>31.4</td>
<td>68.4%</td>
<td>30.0</td>
<td>75.1%</td>
<td>29.9</td>
</tr>
<tr>
<td>Male</td>
<td>82.8%</td>
<td>27.9</td>
<td>78.9%</td>
<td>29.2</td>
<td>77.5%</td>
<td>25.4</td>
</tr>
<tr>
<td>Female</td>
<td>60.5%</td>
<td>32.7</td>
<td>55.0%</td>
<td>26.5</td>
<td>72.1%</td>
<td>35.8</td>
</tr>
<tr>
<td>Poor not considered</td>
<td>68.6%</td>
<td>29.3</td>
<td>63.7%</td>
<td>26.7</td>
<td>72.1%</td>
<td>30.5</td>
</tr>
<tr>
<td>Poor</td>
<td>10.7%</td>
<td>36.3</td>
<td>102.6%</td>
<td>35.9</td>
<td>97.6%</td>
<td>8.9</td>
</tr>
<tr>
<td><strong>3 month</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All cases</td>
<td>77.1%</td>
<td>26.4</td>
<td>80.4%</td>
<td>40.0</td>
<td>77.4%</td>
<td>26.7</td>
</tr>
<tr>
<td>Male</td>
<td>83.2%</td>
<td>20.1</td>
<td>86.2%</td>
<td>43.8</td>
<td>82.2%</td>
<td>18.7</td>
</tr>
<tr>
<td>Female</td>
<td>68.3%</td>
<td>31.5</td>
<td>72.9%</td>
<td>31.6</td>
<td>69.9%</td>
<td>33.9</td>
</tr>
<tr>
<td>Poor not considered</td>
<td>78.0%</td>
<td>24.3</td>
<td>84.0%</td>
<td>(p = 0.05)</td>
<td>40.0</td>
<td>76.4%</td>
</tr>
<tr>
<td>Poor</td>
<td>71.6%</td>
<td>37.9</td>
<td>61.9%</td>
<td>32.3</td>
<td>82.2%</td>
<td>30.7</td>
</tr>
<tr>
<td><strong>6 month</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All cases</td>
<td>88.3%</td>
<td>21.0</td>
<td>88.7%</td>
<td>33.4</td>
<td>95.15</td>
<td>(p = 0.04)</td>
</tr>
<tr>
<td>Male</td>
<td>80.0%</td>
<td>22.2</td>
<td>80.7%</td>
<td>41.0</td>
<td>88.4%</td>
<td>21.4</td>
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<tr>
<td>Female</td>
<td>98.0%</td>
<td>(p = 0.02)</td>
<td>16.2</td>
<td>98.0%</td>
<td>(p = 0.01)</td>
<td>21.4</td>
</tr>
<tr>
<td>Poor not considered</td>
<td>90.7%</td>
<td>(p = 0.03)</td>
<td>18.5</td>
<td>94.0%</td>
<td>(p = 0.01)</td>
<td>33.4</td>
</tr>
<tr>
<td>Poor</td>
<td>74.8%</td>
<td>38.3</td>
<td>59.1%</td>
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<td>8.4</td>
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<td><strong>12 month</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All cases</td>
<td>88.3%</td>
<td>(p = 0.04)</td>
<td>27.4</td>
<td>91.0%</td>
<td>(p = 0.01)</td>
<td>34.4</td>
</tr>
<tr>
<td>Male</td>
<td>86.6%</td>
<td>27.4</td>
<td>86.3%</td>
<td>39.4</td>
<td>87.5%</td>
<td>31.0</td>
</tr>
<tr>
<td>Female</td>
<td>90.3%</td>
<td>(p = 0.01)</td>
<td>27.8</td>
<td>96.5%</td>
<td>(p &lt; 0.01)</td>
<td>27.6</td>
</tr>
<tr>
<td>Poor not considered</td>
<td>92.7%</td>
<td>(p &lt; 0.01)</td>
<td>24.3</td>
<td>92.7%</td>
<td>(p &lt; 0.01)</td>
<td>24.3</td>
</tr>
<tr>
<td>Poor</td>
<td>45.8%</td>
<td>(p = 0.04)</td>
<td>17.7</td>
<td>40.3%</td>
<td>(p = 0.02)</td>
<td>13.9</td>
</tr>
</tbody>
</table>

p-values indicate the significance in comparison with the preoperative percentage
* = Cases with at least satisfactory outcome
5.3 Acromial morphology based on the supraspinatus outlet view

5.3.1 Length and thickness

The mean values of the length of the acromion varied from 47.1 to 50.3 mm, showing no differences between the patients and the controls (Table 3.). The mean thickness of the acromion was 7.8 mm in the patients with the tendinitis stage and 7.6 mm in their controls and 8.0 mm and 7.8 mm in the patients with the tear stage and in their controls, the differences being not significant (Table 3.).

5.3.2 Acromial slope and tilt

The mean acromial slope was 29.2° in the tendinitis stage group, 30.1° in the respective controls and 30.4° and 31.2° in the tear stage group and their controls, but the differences were not significant (Table 3.). The mean acromial tilt values varied from 31.7° to 33.5°, displaying no significant differences between the study groups and their controls (Table 3.).

5.3.3 Types and acromial angle

There were no significant differences in the distribution of different types of acromion between the groups evaluated according to Bigliani et al (Bigliani et al. 1986) (Table 4.). However, there were more type III acromions in the patient groups than in their controls. Type II acromion was the most common type in the patients and their controls, accounting for 70.2–86.5%, whereas flat acromion was a rare finding. There was no significant difference in the acromial angle between the tendinitis group and the control group, but the acromial angle was significantly greater in the rotator cuff tear group than in the control group (p = 0.03) (Table 3.).
Table 3. Parameters of acromial morphology examined in the different groups.

<table>
<thead>
<tr>
<th></th>
<th>Tendinitis stage group</th>
<th>Tear stage group</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 74)</td>
<td>(n = 37)</td>
<td>(n = 111)</td>
</tr>
<tr>
<td></td>
<td>patients</td>
<td>controls</td>
<td>patients</td>
</tr>
<tr>
<td>Length (mm)</td>
<td>mean 47.7, SD 6.9</td>
<td>mean 47.7, SD 5.9</td>
<td>mean 50.3, SD 6.6</td>
</tr>
<tr>
<td>Thickness (mm)</td>
<td>Middle: 7.8, 1.7</td>
<td>Middle: 7.6, 2.0</td>
<td>Middle: 8.0, 1.5</td>
</tr>
<tr>
<td></td>
<td>Anterior: 10.3, 2.0*</td>
<td>Anterior: 9.6, 1.6</td>
<td>Anterior: 10.6, 1.4</td>
</tr>
<tr>
<td></td>
<td>Angle (°): 33.2, 9.5</td>
<td>Angle (°): 34.7, 9.9</td>
<td>Angle (°): 38.6, 9.3**</td>
</tr>
<tr>
<td></td>
<td>Tilt (γ angle) (°): 33.5, 5.3</td>
<td>Tilt (γ angle) (°): 34.5, 5.1</td>
<td>Tilt (γ angle) (°): 33.2, 4.7</td>
</tr>
</tbody>
</table>

*p = 0.02
**p = 0.03

Table 4. Types of acromions in the different groups

<table>
<thead>
<tr>
<th></th>
<th>Tendinitis stage group</th>
<th>Tear stage group</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 74)</td>
<td>(n = 37)</td>
<td>(n = 111)</td>
</tr>
<tr>
<td></td>
<td>n patients</td>
<td>%</td>
<td>n controls</td>
</tr>
<tr>
<td>Flat</td>
<td>5</td>
<td>6.8</td>
<td>2</td>
</tr>
<tr>
<td>Curved</td>
<td>52</td>
<td>70.2</td>
<td>64</td>
</tr>
<tr>
<td>Hooked</td>
<td>17</td>
<td>23.0</td>
<td>8</td>
</tr>
</tbody>
</table>
5.4 Reactions of subacromial bursa at the different stages of impingement syndrome

5.4.1 Tenascin-C expression

In uninvolved tissue, tenascin-C was only seen around small arteries, which is its regular site of native expression. No accumulation of tenascin was seen in the acellular matrix. In tissue samples from involved bursas, on the other hand, variable degrees of tenascin were also seen in the stroma. Tenascin was usually associated with fibrous and inflammatory lesions. Its expression was significantly increased in FTG compared to CG, TG and PTG (p ≤ 0.001) and almost significantly increased in TG compared to CG (p = 0.06) (Fig. 9).

Fig. 9. Percentages of tenascin-C expression in controls and at different stages of impingement syndrome. (For explanations of p-values, see text.)
Fibrosis was a frequent finding (58%) in the tissue samples of the involved groups, but rare in CG (8%) (Fig. 10). It was significantly increased in TG (64%), PTG (45%) and FTG (56%) compared to CG (p = 0.001 vs TG, p = 0.05 vs PTG and p = 0.002 vs FTG). No significant differences were seen between TG, PTG and FTG.

Fig. 10. Percentages of fibrosis in controls and at different stages of impingement syndrome. (For explanations of p-values, see text.)
5.4.3 Thickness

The thickness of the bursa was evaluated as normal in the majority of cases in CG, but in only 39% of the involved shoulders, but the differences between the groups were not significant (Fig. 11). In general, a low degree of vascularity, mostly close to the surface, was seen in the predominantly acellular bursal tissue.

Fig. 11. Percentages of the grade of thickness in controls and at different stages of impingement syndrome. (For p-values, see text.)
5.4.4 Vascularity

A slight increase in vascularity was seen in the bursae of the involved groups (Fig. 12). The increase was most marked in FTG, but the differences compared to the other groups were not significant. Haemorrhages seen in the bursal wall were usually restricted to small focal areas and only appeared here and there in the stroma.

Fig. 12. Percentages of the grade of vascularity in controls and at different stages of impingement syndrome. (For p-values, see text.)
5.4.5 Haemorrhage

The lowest abundance of haemorrhage was a frequent finding in CG and FTG, whereas TG and PTG presented with intermediate abundance (Fig. 13). An increase of haemorrhage was a significant finding in TG compared to CG (p = 0.016). On the other hand, haemorrhages were significantly less often found in FTG compared to CG (p = 0.006) and PTG (p = 0.038).

Fig. 13. Percentages of haemorrhage in controls and at different stages of impingement syndrome. (For p-values, see text.)

5.4.6 Inflammatory cells

In uninvolved tissue, no inflammatory cells were seen. They were also rare in involved tissue. Variable amounts of lymphocytes were seen, usually close to the degenerative or fibrotic areas in one sample in TG and in two samples in PTG and FTG. Statistical comparison between the groups was not possible.
5.4.7 **Correlations between tenascin-C expression and histological findings**

There were no significant correlations between tenascin-C and the histological parameters, which suggests that tenascin-C reflects a different phase of the bursal reaction than the conventional histological parameters. The same was true of the intercorrelations between the histological parameters. Increased fibrosis tended to correlate slightly with vascularity and thickness according to the stage of the impingement syndrome in the samples taken from the patient groups (TG, PTG and FTG).

5.5 **Pressures in different parts of the subacromial space**

The highest subacromial compressions in both groups were recorded under the anterolateral part of the acromion (Fig. 8 and 14). These values were significantly (p < 0.001, Friedman’s test) higher than those in the anteromedial, posterolateral and posteromedial parts in the impingement group. No such differences were observed in the control group.

The pressures were significantly higher in the impingement group than in the control group under the anterolateral acromion at 60° (p = 0.016, MW-U test) and 90° (p = 0.015) of abduction and under the anteromedial acromion at 90° (p = 0.046) of abduction.

The pressures under the anterolateral acromion increased during abduction in the impingement group (Fig. 14). The differences were significant between 0° and 30° (p = 0.007, Student’s t-test), 0° and 60° (p = 0.006) and 0° and 90° (p = 0.004). Similar significant increases were seen under the anteromedial acromion between 0° and 60° (p = 0.044) and 0° and 90° (p = 0.046). An increase of abduction angle had no significant effect on subacromial pressures in the control group.
Fig. 14. Relative pressures in the A) anterolateral, B) anteromedial, C) posterolateral and D) posteromedial parts of the acromion. (Median and 25% and 75% quartiles)
6 Discussion

6.1 Methods

We focused on the condition of the rotator cuff, examining all the operated shoulders by using ultrasound and, in suspicious cases and symptomatic shoulders, by using arthrography and/or MRI. These imaging methods, which have relatively high accuracy rates, are routinely used in the diagnosis of RC tears, and our results therefore appear reliable, especially in view of the fact that they were used as paired or triple. It must be emphasised that, in some cases, the patient may have had a partial joint side or intratendinous RC tear at the time of operation, which did not heal or which progressed to a full-thickness tear. Shoulder joint arthroscopy and MRI were not in routine use at the time when the operations were performed. Trial tenotomy, which is an accurate method to diagnose intratendinous partial tears (Fukuda 2003), was not done routinely, but only in suspected (by palpation of RC tendons) cases at the initial operations.

Most of the earlier studies have involved measuring the strength of the rotator cuff muscles isokinetically with slow (60°/sec) and fast (180°/sec) torque arm speed (Ivey, Jr. et al. 1985, Leroux et al. 1994, Leroux et al. 1995, Holm et al. 1996). We used only isometric measurements, which have been shown to be equally valid for the measurement of the strength of the external rotation and abduction of the shoulder as isokinetic measurements (Gore et al. 1986, Kuhlman et al. 1992). Gore et al (Gore et al. 1986), who used the isometric method, carried out measurements of internal and external rotation at abduction angles of 0° and 90° (if possible) of the shoulder, and those of abduction at angles of 45° and 90° (if possible), but did not record flexion. However, Kuhlman et al (Kuhlman et al. 1992) recommended that the isometric strength of external rotation should be measured at 45° of abduction and 45° of internal rotation, and the strength of abduction in the scapular plane with the shoulder at 45° of abduction. We carried out all our measurements at 0° of abduction and flexion, because this position is the least painful preoperatively and even in the very early postoperative period. It has been shown that the effect of pain on strength measurement is remarkable (Ben Yishay et al. 1994), and isokinetic measurements were therefore not used here.
The strength values of the involved shoulder were given as percentages of those of the uninvolved shoulder, as in some earlier studies (Walker et al. 1987, Kirschenbaum et al. 1993). This management was especially useful here due to the great number of lacking recordings, which unfortunately prevented the use of repeated measures analysis of variance (ANOVA) in statistical analysis. The variation of their group size detracts from the value of the information obtained from that study. However, we think that the statistical values of the percentages of the opposite shoulder are still comparable to each other, because the groups are homogenous for age and sex. We also believe that our measurements are valid, as they were carried out in a consistent manner by a single physiotherapist and give an accurate picture of the relative changes of shoulder muscle strength preoperatively and in the early recovery period after decompressive surgery.

Proper projection is essential for an accurate evaluation of radiological acromial morphology (Duralde & Gauntt 1999). Correct x-ray beam direction can be obtained by fluoroscopic control as described by Liotard et al (Liotard et al. 1998) and Prato et al (Prato et al. 1998). They pointed out that, for a good projection of SOV, the direction of the x-ray beam varies between 5° cranial to 25° caudal. However, a proper technique for obtaining traditional SOV can produce acceptable and reproducible SOVs, as suggested by Duralde et al (Duralde & Gauntt 1999). We did not use fluoroscopy but only used standard positioning of the patient, film and beam with 15° caudal tilt, which is the mean of the values reported by Prato et al (Prato et al. 1998). Proper SOVs were also ensured by selecting only the valid projections according to Liotard et al (Liotard et al. 1998) for the study. Using this method, we found 10% of the x-rays inadequate. This figure is clearly lower than that (23%) reported by Kitay et al (Kitay et al. 1995).

Our histological evaluation of bursal reactions in impingement syndrome was mainly based on the same criteria as in some earlier studies (Chiquet-Ehrismann et al. 1986, Uhlthoff & Sarkar 1991, Rahme et al. 1993, Kronberg & Saric 1997). Therefore, reliable and valid comparisons could be made with the previous results.

There are aspects in the study of subacromial pressures that may cause some bias. The pressures between the acromion and the rotator cuff proved to be surprisingly high. It must be considered that the sensor, although not more than 2 mm thick, interferes with the recording by stretching the subacromial space, which may increase the real pressure value. However, our sensor was markedly thinner than the balloon used in the study by Nordt et al (Nordt, III et al. 1999), which was 4 mm thick. We give the measured pressures in relative values, which are sufficient for our purposes. To obtain pressure values near to the real, a transducer as thin as possible should be developed. The measurements were performed under general anaesthesia. It is possible that, under normal conditions, the co-ordination of muscle function and arm torque would have an effect on the pressures in the subacromial space. It is, however, difficult to find a valid control group for measurements of this kind. Here, we performed the control measurements during repair of the dislocated acromioclavicular joint, which was the only frequently performed operation in our hospital during which the subacromial space can be reached without unnecessarily widening the approach. Some biomechanical error might occur in the case of scapular poise (Rasyid et al. 2000). This must be accepted because it is impossible to make the measurements used here in a completely non-affected shoulder or under local anesthesia. This was also pointed out by Nordt et al (Nordt, III et al. 1999) after some preliminary attempts at a study design similar to ours.
We believe that these sources of error are not decisive and that our results reflect the real pressure conditions in the subacromial space.

6.2 Long-term results of open acromioplasty and rotator cuff pathology

The subjective results of open acromioplasty are generally favourable, the percentage of excellent and good outcomes ranging from 43% to 94% (Neer 1972, Thorling et al. 1985, Tibone et al. 1985, Post & Cohen 1986, McShane et al. 1987, Hawkins et al. 1988, Jalovaara et al. 1989, Bjorkenheim et al. 1990, Stuart et al. 1990, Rockwood & Lyons 1993, Hartwig & Burkhard 1996). The subjective outcomes in this study were similar to those reported earlier. We observed a relatively high incidence of RC tears after open acromioplasty in long-term follow-up, which has not been reported earlier. Most of the earlier studies on the outcome of open acromioplasty have concentrated on the clinical outcome, and the mean follow-up times have been mostly too short to show these late failures (Neer 1972, Thorling et al. 1985, Tibone et al. 1985, Post & Cohen 1986, McShane et al. 1987, Hawkins et al. 1988, Jalovaara et al. 1989, Bjorkenheim et al. 1990, Stuart et al. 1990, Rockwood & Lyons 1993, Hartwig & Burkhard 1996).

In our study with a long follow-up time, 15% of the shoulders showing a favourable primary outcome deteriorated some years postoperatively, and the majority of these had developed an RC tear. This suggests that the disease process sometimes continues in the RC tendon after acromioplasty despite the disappearance of symptoms and the elimination of subacromial compression. Cuff tears were also found in shoulders which did not benefit from acromioplasty and in pain-free shoulders. Including these, nearly one fifth of the operated shoulders displayed either a partial or a complete tear. However, it has been shown in cadaveric, ultrasonic and MRI studies that partial and complete RC tears also appear in the asymptomatic population, and they are age-related (Neer 1983, Thorling et al. 1985, Sher et al. 1995).

6.3 Pathogenesis of impingement syndrome

Ozaki et al (Ozaki et al. 1988) suggested that RC tears or injuries are the result of intrinsic rather than extrinsic causes associated with impingement, as advocated by Neer (Neer 1983). They found that although a lesion in the anterior third of the undersurface of the acromion was always associated with a RC tear, the reverse was not true, and they concluded that the pathogenesis of most cuff tears was probably an intrinsic process. Our results partially support the findings of Ozaki et al. (Ozaki et al. 1988). The majority of the patients remained symptomless and showed no evidence of RC tear, suggesting that the process in the tendon did not progress in these cases.
According to the extrinsic theory of the pathogenesis of impingement, the lesion in the rotator cuff tendon is caused by mechanical compression by the coracoacromial arch. Consequently, the disease process should come to a halt after decompressive acromioplasty, and the surgical outcome should be permanent. On the other hand, if the symptoms recur and the disease progresses to the tear stage despite acromioplasty, intrinsic factors, i.e. a degenerative process in the RC tendons, might be significant.

### 6.4 Recovery of shoulder muscle strengths after open acromioplasty

It has been shown that shoulder muscle strength decreased by supraspinatus tendinitis and rotator cuff tear is restored after surgical treatment (Leroux et al. 1994, Leroux et al. 1995). The recovery of shoulder strength after reconstruction of rotator cuff tear is a slow process, lasting up to a year (Vastamaki 1986, Walker et al. 1987, Kirschenbaum et al. 1993, Rokito et al. 1996). However, strength does not always completely recover to the level of the uninvolved opposite shoulder (Gore et al. 1986, Rabin & Post 1990). In this regard, our findings are in agreement with those of earlier reports, but no report focusing on the early recovery of isometric shoulder muscle strength after surgery of stage II impingement syndrome could be found.

The recovery of strength was more marked in women, and this is at least partly due to their more pronounced loss of strength preoperatively than seen in men. The favourable outcome of surgery is reflected in the good recovery of strengths, suggesting that mechanical aspects are involved in the pathogenesis of impingement syndrome. On the other hand, in cases with a poor outcome at one year, strengths mostly decreased compared with the preoperative situation, even though this group was too small for statistical analysis. The finding that male shoulders did not show equally significant recovery as female shoulders might provide some support to the intrinsic theory (overuse syndrome). Dominance was not considered because its effect has been proved to be non-significant (Ivey, Jr. et al. 1985, Murray et al. 1985, Walker et al. 1987).

It must be emphasised that rehabilitation mostly remained the personal responsibility of the patient, based on guidance given by the physiotherapist at discharge from the hospital, with no controlled supervision afterwards. In fact, supervised exercises have been suggested to be as effective as surgery based on a prospective randomised study of patients with stage II impingement syndrome (Brox et al. 1993). It is possible that recovery may be improved and facilitated by effective controlled physiotherapy. The slow recovery of shoulder muscle strengths should also be considered when prescribing a sick leave for a heavy manual worker.

### 6.5 Acromial morphology evaluated by supraspinatus outlet view

Despite the potential value of correlating specific acromial forms with lesions of the rotator cuff described by Bigliani and Morrison (Bigliani et al. 1986), some investigators
have been unable to reproduce these findings. They have questioned the reliability of radiographic acromial morphology assessment in the sagittal plane (Jacobson et al. 1995, Zuckerman et al. 1997, Bright et al. 1997, Liotard et al. 1998). Here, we found no significant differences in the distribution of the different types of acromions between the patients and controls. However, there was a tendency for type III acromions to appear more frequently in patients than in controls. This tendency turned out significant when an objective method of measurement was applied.

In an effort to objectively quantitate and standardise the classification of the curvature of the anterior acromion, Toivonen et al. (Toivonen et al. 1995) devised the measurement of an acromial angle. They found it to range from 5° to 42°, which is comparable to the values observed in our study, 3° to 67°. We also found an increased acromial angle to associate with rotator cuff tears, which is in agreement with the findings of earlier studies (Toivonen et al. 1995, Tuite et al. 1995, Banas et al. 1995).

There is some terminological confusion concerning the slant of the acromion in relation to the scapula and the reference points used to determine it (Chambler & Emery 1997). This inclination has been called acromial tilt (Zuckerman et al. 1992, Kitay et al. 1995, Prato et al. 1998) and acromial slope (Aoki et al. 1986, Edelson & Taitz 1992, Gohlke et al. 1993), and it has been evaluated by different methods. Kitay et al. (Kitay et al. 1995) and Aoki et al. (Aoki et al. 1986) determined it as the angle formed by connecting two lines from the posteroinferior undersurface (angulus acromialis) of the acromion to the tip of the coracoid process and to the anteroinferior acromion. Some other investigators (Edelson & Taitz 1992, Zuckerman et al. 1992, Mallon et al. 1992, Gohlke et al. 1993, Prato et al. 1998) have evaluated the corresponding inclination in relation to the scapular axis. Here, we used the method described by Kitay et al. and Aoki et al using the tip of the coracoid process as a reference point and called this inclination acromial tilt. It was, however, emphasised that the tilt may vary because the length of the tip of the coracoid is affected by the osteophytes pointing the conjoined tendon. Therefore, we also measured the tilt by using the coracoid undersurface curve as a reference point, but this did not alter the results. (Fig. 6.)

The dimensions of the acromion have been found to be different between sexes, but not age-dependent (Nicholson et al. 1996). Here, we found no significant differences in the acromial measures between the controls and the patients, and the measurement of acromial dimensions is therefore a waste of time in view of the diagnosis.

6.6 Reactions of subacromial bursa in different stages of impingement syndrome

6.6.1 Tenascin-C expression

The expression of tenascins in different tissues varies, depending on the developmental stage of the organism analysed, and it changes dramatically under various pathological
conditions (Erickson & Bourdon 1989, McCachren & Lightner 1992, Riedl et al. 1992, Soini et al. 1993, Riley et al. 1996, Kaarteenaho-Wiik et al. 1996). Elevated expression of tenascin is seen in active reparative processes as wound healing, inflammatory lung diseases and keloids (Mackie et al. 1988, Dalkowski et al. 1999, Kaarteenaho-Wiik et al. 2000). In bursal tissue, it also very probably characterises regeneration and, thus, a different phase of reaction compared to fibrosis, being a late reaction. This is in good accordance with our results showing a lack of intercorrelation between tenascin-C expression and fibrosis.

Our study was the first to use the expression of tenascin-C as a marker of tissue lesions in the subacromial bursa. Tenascin-C expression in the subacromial bursa was significantly more pronounced in all of the involved groups than in the control group. It was expressed weakly in the extracellular matrix in the controls, but was found in larger quantities in the stroma of involved tissue. Riley et al. (Riley et al. 1996) studied tenascin-C expression in supraspinatus tendon affected by the impingement syndrome and found it to be increased, especially in degenerative tendons, but not in healthy cadaveric tendons. On the other hand, increased tenascin-C expression might be caused by a mechanical load due to impingement, as it has been shown that the expression of tenascin-C responds to a mechanical load in bone (Webb et al. 1997) and in the osteotendinous junction (Jarvinen et al. 2000). Further support of the mechanical cause might be a lack of correlation between tenascin-C expression and an inflammatory reaction or fibrosis in the bursa. It thus seems that the reaction in the subacromial bursa is associated with the tendon lesion regardless of whether it is degenerative or caused by a mechanical load or compression.

6.6.2 Vascularity

Ishii et al. (Ishii et al. 1997) examined vascularity (angiogenesis) and cellularity (mostly fibroblasts and occasionally lymphoblasts) as a combined measure of the “bursal reaction”, while we evaluated and presented them as separate parameters of bursal involvement. As far as we can see, this distinction allows a more refined analysis of the course of the pathological and healing process at different stages of impingement syndrome.

6.6.3 Fibrosis

The most significant difference in the histological parameters between the controls and patients was that fibrosis was present significantly more frequently in the involved groups. An increased amount of bursal fibrosis in impingement syndrome has also been observed in the previous studies (Rahme et al. 1993, Kronberg & Saric 1997). Rahme et al. (Rahme et al. 1993) found bursal fibrosis in 80% of their patients with supraspinatus tendinitis but in only 15% of cadaveric controls. Kronberg and Saric (Kronberg & Saric 1997) reported severe fibrosis in 10, moderate in 8 and no fibrosis in 1 of 19 patients with
tendinitis stage impingement syndrome and no fibrosis in 5 cadaveric control shoulders. In the study of Ishii et al (Ishii et al. 1997), fibrosis was evaluated as part of the bursal reaction and turned out to be more pronounced next to the rotator cuff tear than in specimens obtained further away from the lesion. They also found the reactions to be significantly slighter in tendinitis than in the presence of a rotator cuff tear. This was also the case in our study, where the specimens were taken from the deltoid side of the bursa, which probably reflects better the general reaction of the bursa than the specimens obtained from lesion sites.

### 6.6.4 Inflammatory cells

Santavirta et al (Santavirta et al. 1992) found in their immunohistochemical study one mild and six moderate inflammatory cell involvements in the 12 subacromial bursas biopsied at acromioplasty for supraspinatus tendinitis or rotator cuff tear. They suggested that inflammation of the subacromial bursa is a potentially important cause of pain in impingement syndrome. In our study, inflammatory cells were seen only rarely. This is in agreement with the findings reported by Ishii et al (Ishii et al. 1997), who observed no plasma cells and only occasional lymphocytes in bursal specimens taken from patients with tendinitis or a tear of the rotator cuff.

### 6.6.5 Thickness

The thickness of the bursa has not been used as a specific parameter of bursal reaction in the earlier studies, probably because it is difficult to measure due to uncertainties in the definition of the cutting plane. However, Rahme et al (Rahme et al. 1993) evaluated it as part of fibrosis, and it proved to be increased. Fibrosis and bursal thickness also tended to be related in the present study.

### 6.6.6 Haemorrhage

Previous reports have not paid much attention to the presence of hemorrhages in the subacromial bursa. Ishii et al (Ishii et al. 1997) noticed hemorrhages in 15% of their cases of tendinitis stage impingement syndrome. In this study, bursal hemorrhages occurred most frequently in association with tendinitis and partial tears, but were uncommon in the controls and in full-thickness tears.
6.6.7  *Subacromial bursa in the pathomechanism of the impingement syndrome*

Our study sheds some light on the role of the subacromial bursa in the pathomechanism of the impingement syndrome. Based on histological studies, it has been suggested that bursal involvement is secondary to the damage of the rotator cuff tendon (Ulhoff & Sarkar 1991, Ishii *et al.* 1997) and the bursa is, similarly to the peritoneum, only secondarily involved, as postulated by Codman (Codman 1990). We did not examine the changes in tendons, but the fact that the increased tenascin-C expression reflecting bursal reactions was more pronounced at the tear stage than at the tendinitis stage of the disease supports the previous assumptions. However, the reaction of the subacromial bursa may be the origin of the symptoms of impingement syndrome, as suggested by some other studies, which focused on the origin and mediators of impingement pain (Santavirta *et al.* 1992, Rahme *et al.* 1993, Gotoh *et al.* 1998).

The attitude to bursal resection during rotator cuff surgery has been under discussion. Ishii *et al.* (Ishii *et al.* 1997) recommended repair of the bursa during RC tear repair. On the other hand, Kronberg and Saric (Kronberg & Saric 1997) and Rahme *et al.* (Rahme *et al.* 1993) reported a better outcome in the patients whose bursa had been removed at the tendinitis stage. In this study, we observed that the fibrosis phase of the bursal reaction is dominant compared to the regeneration phase, as indicated by tenascin-C expression at the tendinitis stage of impingement. Thus, we feel that the resection of bursa is justified, as it has been suggested that bursal fibrosis impairs the outcome (Rahme *et al.* 1993, Kronberg & Saric 1997). At the tear stage, the regenerative process in the bursa, determined by tenascin-C expression, is so notable as to be potentially important for the healing of the tear. Its restoration is therefore recommended.

6.7  *Pressures in different locations of the subacromial space*

Our results suggest that subacromial pressure is highest under the anterolateral acromion, increases during abduction in impingement patients and is elevated in shoulders suffering from impingement. These findings further elaborate the earlier knowledge and are, by and large, in agreement with the previous studies. Flatow *et al.* (Flatow *et al.* 1994), using stereo-photogrammetric analysis, demonstrated that the acromial undersurface and the rotator cuff tendons are in closest proximity between 60 degrees and 120 degrees of elevation in cadaveric shoulders and that the main area of increased contact is the anteroinferior part of the acromion. Zuckerman *et al.* (Zuckerman *et al.* 1992) evaluated subacromial pressure indirectly, using three-dimensional computer modelling, which showed that the volume of the subacromial space decreased when the prominence of the anterior aspect of the acromion increased. However, they did not measure the pressure. Wuelker *et al.* (Wuelker *et al.* 1995a, Wuelker *et al.* 1995b), using a dynamic cadaveric model, found that the peak forces under the coracoacromial arch occurred between 51° and 82° of elevation and were most pronounced underneath the acromion. They pointed
out that measurements of anatomic specimens may not be fully applicable to the clinical situation. Jerosch et al (Jerosch et al. 1989) suggested in their cadaveric study, where the measurement was carried out with a compression film, that mechanical subacromial compression only occurs in shoulder joints with a Bigliani acromion type III (Morrison & Bigliani 1987) and/or in joints with imbalance of the deltoid/rotator cuff relation. In our study, acromions were classified as type II and we could not consider the muscular imbalance because of the anesthesia. Sigholm et al (Sigholm et al. 1988) studied subacromial bursal pressures in healthy volunteers using a microcapillary infusion method. The pressure, which averaged 8 mm Hg at rest, increased when the arm was lifted to 39 mm Hg and, with a 1 kg weight, to 56 mm Hg. Their method reflects only the total pressure of the bursal pouch (Nordt, III et al. 1999), not local pressures under the acromion, as does our method. Nordt et al (Nordt, III et al. 1999) posted that fully abducted and cross-reach positions generate the highest impingement pressures, while acromioplasty decreased significantly (60%–100%) the anterior edge subacromial pressures. These studies are very much in accordance with our findings showing that the measured pressures were highest in 90° of abduction in the anterolateral part of the subacromial space. These papers, in line with our findings, lend support to the use of Neer’s acromioplasty in the treatment of impingement syndrome (Neer 1972). Excision of the anterior undersurface of the acromion, under which the subacromial pressure is concentrated, decreases the pressure and explains the good effect of acromioplasty in this disease.

The steady increase in subacromial pressure observed under the anterolateral part of the acromion during abduction at up to 90° in impingement syndrome shoulders fits very well with the classical clinical symptom of this disease, i.e. a painful arc (Calvert 1997). Our findings thus seem to support the "extrinsic" theory (Neer 1983, Morrison & Bigliani 1987, Bigliani & Levine 1997) of the pathogenesis of impingement pathology. It should be considered, however, that increased subacromial pressure might also be of secondary origin, i.e. caused by spur formation secondary to the primary degenerative process in the cuff, as suggested by the supporters of the "intrinsic theory" (Uhthoff et al. 1987, Ozaki et al. 1988).

Generally, the shoulder impingement syndrome can be manifested in many forms. It seems that it has at least two different pathomechanisms, which we should be able to differentiate. The diagnosis of the disease is still mostly clinical, and good criteria for evaluating the precise need for surgery are lacking. As a treatment option, a tailored rehabilitation program can help to avoid unnecessary surgery.
7 Conclusions

1. The good outcome after open acromioplasty is not permanent in all cases, suggesting that a degenerative process is involved in the pathogenesis of impingement syndrome.
2. Shoulder muscle strength is restored to near normal within one year after open acromioplasty. This suggests that mechanical compression plays a role in the pathogenesis of impingement syndrome.
3. Variation in the shape of the acromion, as evaluated by a routine supraspinatus outlet view, is associated with impingement syndrome, but this association is weak and its validity in the diagnosis of impingement syndrome is therefore a minor adjunct to the other diagnostic methods.
4. Tenascin-C is a more general indicator of bursal reaction compared to the conventional histological markers, being especially pronounced at the more advanced stages of impingement. It seems that the bursal reaction is an essential part of the impingement pathology at all stages, and that this reaction shows different phases, as indicated by the different parameters used in this study.
5. Impingement pain may be caused by compression onto the inflamed subacromial bursa or pressure ischaemia in the rotator cuff.
6. The local pressures in the subacromial space were elevated in impingement syndrome, supporting the mechanical theory.
7. Mechanical (extrinsic) and degenerative (intrinsic) factors are involved in the pathogenesis of impingement syndrome.
References


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