The acute first-time anterior shoulder dislocation (AFASD)
te Slaa, R.L.

Citation for published version (APA):

General rights
It is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), other than for strictly personal, individual use, unless the work is under an open content license (like Creative Commons).

Disclaimer/Complaints regulations
If you believe that digital publication of certain material infringes any of your rights or (privacy) interests, please let the Library know, stating your reasons. In case of a legitimate complaint, the Library will make the material inaccessible and/or remove it from the website. Please Ask the Library: http://uba.uva.nl/en/contact, or a letter to: Library of the University of Amsterdam, Secretariat, Singel 425, 1012 WP Amsterdam, The Netherlands. You will be contacted as soon as possible.
Chapter 1

General introduction and aim of this thesis
1.1 Introduction on acute first-time anterior shoulder dislocation

"The motions of the shoulder joint are more extensive than those of any other joint in the body, it being capable of moving in every direction; and its dislocations are, consequently, more frequent than those of all other joints in the body collectively...."

These words were written by Sir Astley Cooper in the introduction to his chapter on dislocations of the shoulder (1832). The anterior shoulder dislocation is still the most common dislocation in human joints with a reported incidence of 1 to 2 percent in the general population. The prognosis of recurrence after an acute first-time anterior shoulder dislocation (AFASD) in young patients is reported to be between 17% and 96%, with a mean of 67%. Age and athletic participation are prognostic factors in recurrence. An unstable shoulder with a limited range of motion is very disabling for young athletes engaging in overhead activities. The best treatment of acute first-time anterior shoulder dislocation (AFASD) in young patients is still a matter of debate. Some studies describe the arthroscopic evaluation or eventual treatment of AFASD. However, most of these studies have a short follow-up to be of value. A Swedish study conducted by Hovelius (1996) is the only published long-term (10-years) follow-up study of patients with AFASD. We may regard this study as the “natural history” of patients with AFASD.

The most common mechanism of a traumatic initial dislocation is an indirect anteriorly directed force applied to the posterior aspect of the externally rotated abducted and extended arm, which levers the humeral head anterior in relation to the glenoid, occuring when the patient’s arm is in the overhead position. Occasionally, the initiating event may be a direct blow to the posterior aspect of the proximal humerus. The patient’s age is the most important factor in determining the prognosis. Patients who are younger than 25 years of age at the time of initial dislocation are more likely to suffer recurrent dislocation, whereas patients older than 50 are more likely to suffer rotator cuff tears following the initial dislocation.

Anterior shoulder dislocation is diagnosed on the basis of history, physical and radiographic examinations. The patient may present with a slight abducted arm and may be unable to rotate or fully abduct the arm. Patients often support their forearm of the affected shoulder with the unaffected arm. The humeral head may be palpated on the anterior aspect of the shoulder; whereas the posterior aspect may appear hollow.

There is extensive literature available on the pathology of shoulder dislocation, which dates back many years. There are a number of Dutch thesis in the medical literature that were written during the twentieth century. Leguit wrote his thesis on “Habitude Schouderluxaties” in 1942. He advocated the extra-articular bone graft procedure for treatment, as described by Noordenbos in 1938.
In 1966 Bijlsma wrote his thesis proposing a modification of the Noordenbos procedure.\textsuperscript{16} Sanders (1978) wrote about recurrent shoulder dislocations.\textsuperscript{91} In his thesis he reviews the results of the operative treatment of the recurrent anterior dislocation of the shoulder in 105 patients. Sijbrandy reported in 1984, on dislocations of the shoulder in a Dutch medical journal.\textsuperscript{107} Recently, Visser (1998) wrote a thesis on nerve injury following shoulder trauma (fractures of the proximal humerus and anterior shoulder dislocation).\textsuperscript{111}

1.2 Types of glenohumeral dislocation

The glenohumeral dislocation can be subdivided into anterior, posterior, inferior and superior, according to the position of the humeral head in relation to the glenoid.\textsuperscript{23}

**Anterior shoulder dislocations**

The anterior shoulder dislocations are divided into 4 subgroups, the subcoracoid, subglenoid, subclavicular and intrathoracic.\textsuperscript{23,67} The most common type of anterior shoulder dislocation is the subcoracoid type. This type accounts for up to 90 percent of anterior shoulder dislocations. The second most common type is the subglenoid. It is relatively uncommon by comparison to the subcoracoid type. Both types account for almost 97\% of the anterior shoulder dislocations. The remaining two types are the subclavicular and intrathoracic types, both of which are extremely rare. The usual mechanism of injury which causes subcoracoid dislocations is a combination of shoulder abduction, extension, and external rotation. The head of the humerus is displaced anteriorly in relation to the glenoid and inferior to the coracoid process.

In subcoracoid dislocations, the head of the humerus lies anterior to and below the glenoid fossa, are often associated with fractures of the greater tuberosity and damage to the rotator cuff.

In subclavicular dislocations, the head of the humerus lies medial to the coracoid process, just inferior to the lower border of the clavicle. The latter dislocations are rare, but when they do occur the rotator cuff is usually severely disrupted.

**Figure 1: Subcoracoid shoulder dislocation**

In intrathoracic dislocations, the head of the humerus lies between the ribs and the thoracic cavity and are equally rare and are usually associated with an avulsion of the rotator cuff or a fracture of the greater tuberosity.\textsuperscript{72,93}

These rarer types of dislocation are usually associated with severe trauma. Besides a high incidence of fracture of the greater tuberosity and rotator cuff tears, neurological, pulmonary and vascular complications can occur.
General introduction and aim of this thesis

Posterior shoulder dislocation

Clinical assessment of posterior shoulder dislocation appears to be difficult, which is obvious by the fact that it is a frequently missed diagnoses on initial presentation. The literature refers to misdiagnosed instances of posterior shoulder dislocations as high as 79%. Improved knowledge of the clinical symptoms and available diagnostic techniques will result in more frequent and earlier diagnoses. It is for this reason that this subject will be extensively discussed in this introduction.

Posterior dislocation of the humeral head is uncommon, as opposed to anterior shoulder dislocation. The incidence ranges from 1.5 to 4% of all shoulder dislocations. According to Rockwood and Rowe posterior shoulder dislocation is easily misdiagnosed on the initial presentation in 50-79% of all cases. A retrospective study showed that the average time between the sustained injury and the diagnosis is one year (ranging from 1 week to 10 years).

The physician who assesses the patient initially might easily miss this particular dislocation due to the fact that incidence rates are low and due to unfamiliarity with the characteristic deviations that are associated with this pathology. This type of dislocation is usually caused by violent muscle contraction such as that caused by a seizure or an electric shock. Also a fall on an outstretched arm in flexion and adduction or an acute injury to the anterior shoulder may cause a posterior shoulder dislocation.

Examination shows that the patient presents with the arm held with the shoulder internally rotated or in adduction. Further inspection reveals a flattening of the anterior shoulder contour and a prominent coracoid process. Abduction is limited and external rotation is impossible. This occurs when the humeral head is locked in internal rotation behind the glenoid, which then causes the external rotation to be obstructed. Consequently, limited external rotation is the single most important symptom in these patients.

In other words, each patient presenting with limited external rotation of the shoulder following trauma is suspect of a posterior dislocation until counter-evidence is obtained through adequate X-ray imaging. The diagnosis can be missed if X-ray imaging involves only AP projection. One should realise that with an accurate AP projection, the head of the humerus should never show a false projection with the rim of the glenoid.

Figure 2: Patient with posterior shoulder dislocation and limited external rotation.
AP view of a posterior dislocation will reveal a relatively smaller contour of the humeral head. (Figure 3) Furthermore, the internally rotated position causes to obscure the sharp contour of the greater tuberosity.

Neverthless, it is paramount to obtain an axial shoulder X-ray with every “trauma-series” of the shoulder. An axial view shows the position of the humeral head in relation to the glenoid. (Figure 4)

A scapular-Y view is also valuable for this purpose. If additional X-rays show the humeral head in a central position to the glenoid there can be no question of a posterior dislocation. A detailed picture of injuries to the humeral head (especially concerning the extent of the damage) may be obtained through a CT-scan.
When an acute posterior dislocation is diagnosed the shoulder can be reduced without too much difficulty. Posterior dislocations without impression fractures of the humeral head may reduce spontaneously. Fixed posterior shoulder dislocations need to be reduced. Reduction is carried out by applying traction to the adducted arm, internal rotation and 90° elevation while maintaining dorsal pressure on the humeral head. One should never use forced external rotation. After reduction, the arm should be immobilised temporarily with an arm sling or elevation sling. Furthermore, forcefull internal rotation movements of the injured arm should be avoided.

The prognosis regarding an immediately reduced posterior dislocation is, in most cases, good. A recurrence of instability is regarded as a complication of acute posterior shoulder dislocation. This instability may present as a recurring (sub) dislocation in the same direction as the initial dislocation.

Operative reduction is required in the case of posterior dislocations that were sustained longer than 2 to 6 weeks ago, as closed reduction is often no longer a feasible option. Operative treatment of chronic posterior shoulder dislocations should not be considered in patients without pain and patients who are still able to function despite a limited shoulder function. Operative intervention involves an open reduction, the humeral head is placed in normal position in relation to the glenoid and usually the “shortened” m.subscapularis is advanced from the surrounding tissue.

If the humeral head involvement approximately is 20-40%, the subscapular tendon can be placed in this impression defect together with the minor tuberosity. Humeral head defects exceeding 40% are indicative for placement of prosthesis or arthrodesis. Prognosis following an operatively treated chronic posterior dislocation is in most cases significantly worse than immediately reduced shoulders. Most often shoulder function will remain limited following an operation.
Therefore; if an acute posterior shoulder dislocation is recognised and treated early, the prognosis is satisfactory. One should be mindful of an acute posterior shoulder dislocation in each patient that presents with limited external rotation following trauma until counter-evidence is obtained. Additional X-rays, such as axial views are indicated. It is of great importance that this type of injury is diagnosed in an early stage. In order to achieve this goal everyone should be familiar with the clinical picture of this particular injury.

**Inferior shoulder dislocation (luxatio erecta)**

Inferior dislocation of the glenohumeral joint (or luxatio erecta) was first described by Middeldorpff and Scharm in 1859. Inferior dislocations may be produced by a hyper-abduction force that causes abutment of the neck of the humerus against the acromion process, which levers the head out inferiorly. The humerus is then locked with the head below the glenoid fossa and the humeral shaft pointing overhead, resulting in the so-called luxatio erecta. The clinical picture of a patient with luxatio erecta is so clear that it can hardly be mistaken for any other condition. Severe soft tissue injury or fractures of the proximal humerus occur with this dislocation. At the time of surgery or autopsy, various authors have found avulsion of the supraspinatus, pectoralis major, or teres minor muscles together with fractures of the greater tuberosity. Neurovascular involvement is also common.

Lev-El et Rubenstein. (1981) reported on a patient with an injury to the axillary artery and subsequently developed a thrombus that required resection and a vein graft. Rockwood and Wirth found that in 19 patients with this condition, all had a brachial plexus injury and some vascular compromise prior to reduction. The force may be so great as to force the head out through the soft tissues and the skin. Reduction of an inferior dislocation can often be accomplished by traction and counter traction manoeuvres. When closed reduction can not be accomplished, the buttonhole tear in the inferior capsule must be surgically enlarged before reduction can occur.

**Superior shoulder dislocation**

Speed reported that Langier, in 1834, was the first to record a case of superior dislocation of the glenohumeral joint. There is little mention of this type of dislocation in current literature, but occasional cases do undoubtedly occur. The usual cause is an extreme forward and upward force on the adducted arm. With displacement of the humerus upward, fractures may occur in the acromion, acromioclavicular joint, clavicle, coracoid process, or humeral tuberosities. There is an extreme soft-tissue damage of the capsule, rotator cuff, biceps tendon, and surrounding muscles. Clinically, the head rises above the level of the acromion. The arm is short and adducted to the side. Neurovascular complications are usually present.
1.3 Pathology of AFASD

Essential lesion

Broca and Hartmann in 1890 reported their findings of detachment of the anterior labrum and capsule from the glenoid rim (le décollement capsule-periosté-preglenoidien) and described the bony defect in the humeral head in an autopsy specimen.

![Figure 1: Detachment of the anterior labrum and capsule from the glenoid rim.](image)

Although we give credit to Bankart for describing the "essential Bankart lesion". It was Perthes, in 1906 who felt that separation of the capsulolabral avulsion from the glenoid was one of two mechanisms of anterior shoulder dislocation. However, Bankart’s paper in 1938 stressed that the capsulolabral detachment and not capsular rupture would occur at the time of dislocation. He advised to reattach the capsulolabral avulsion back to the glenoid rim.

For many years this Bankart lesion was regarded as the "essential lesion" in anterior shoulder instability. It has been reported at the time of surgery in more than 85% of these cases. Some arthroscopic studies implicate the Bankart lesion as responsible.

There still exists some controversy about what the "essential lesion" is that causes anterior dislocation of the humeral head.

![Figure 2: Blundell Bankart (1879-1951)](image)

Baker et al. established a classification system based on arthroscopic findings of labrum pathology in patients with AFASD. They found in 62% of the cases a Baker III type of lesion. Another 13% had capsular injuries without labral involvement. Recently, Taylor et al. studied patients with AFASD and determined arthroscopically that 97% had evidence of isolated detachment of the capsuloligamentous complex from the glenoid rim and neck, without evidence of intracapsular injury.
In contrast to these arthroscopic studies, two recent cadaveric studies lead to different conclusions regarding the "essential lesion".

Bigliani and Pollock (1992) concluded from their cadaver study that capsular injury ("capsular stretching") is also a significant factor. They reported that an injury to the anterior capsule and ligaments caused stretching and failure of the inferior glenohumeral ligament (IGHL) capsule complex before labral avulsion. In this study the insertion of the IGHL on the glenoid failed most commonly (40%), the humeral insertion in 25% and the ligament midsubstance in 35% of cases.

Speer and Deng (1994) created Bankart lesions in their cadaver model. These lesions contributed minimally to anterior translation of the humeral head. Based on their study, they concluded that a Bankart lesion alone could not be responsible for recurrent anterior instability and that stretching or elongation of the IGHL-capsular structure must also occur.

Besides the Bankart lesion and capsular injury, the acute first-time anterior shoulder dislocation often cause an impression fracture of the posterolateral humeral head (Hill-Sachs lesion) and in some cases an avulsion of the glenoid rim. (Figure 4)

The pathology in older patients appears to be different from that in younger patients. The reported arthroscopic studies have been performed predominantly in younger patients. In contrast, most cadaveric studies have used an older population, allowing comparison difficult.

Figure 4: Anatomical lesions following an anterior shoulder dislocation.
Avulsion of the capsulolabral complex from the glenoid rim and scapular neck (Bankart lesion), attenuation of the capsule and ligaments, impression fracture of the posterolateral humeral head (Hill-Sachs lesion), attenuation of the subscapularis tendon, and humeral avulsion of the IGHL complex (HAGL lesion) have all been suggested as causes of recurrent dislocations.

Arthroscopic studies and non-invasive studies have provided us with better information about the patho-anatomy of the AFASD. Despite these reports, the nature of the one “essential lesion” following an AFASD remains unclear.

1.4 Arthroscopy and AFASD

Labrum

In 1990 Baker and Uribe reported on a classification system based on arthroscopic findings in a series of 45 patients with AFASD. All patients were younger than 30 years of age. Sixty-two percent had a complete capsule-labral detachment (type III), 25% had a partial labral detachment (type II) and only 6 shoulders (13%) had capsular injuries without labral detachment. Others found a Bankart lesion in almost 100% of cases in studies of arthroscopic evaluation of AFASD. However, Molé (1996) could not find a uniform pathology in his prospective multicentre study of patients with AFASD. He concludes that a large variety of lesions associated with AFASD are visualised during arthroscopy. These findings were confirmed in studies by Baker (1990) and te Slaa (2002). In our arthroscopic study we found a spectrum of labral pathology with a full rupture of the labrum and capsule (Baker III) in 61% of the cases. Furthermore, we could not find a statistically significant correlation between the Baker lesion and shoulder instability at 5-year follow-up.

Hintermann and Gächter (1995), in their prospective arthroscopic study of the unstable shoulder (n=212), found multiple morphologic changes associated with instability of the gleno-humeral joint. They found a normal labrum in 10% of the AFASD. They stated that: “there is no single cause for an unstable shoulder.”
Taylor and Arciero (1997) found type III lesions as described by Baker in 61 of 63 (97%) patients.\textsuperscript{108} (Figure 3) There was no evidence of intracapsular injury in 62 of 63 patients. One patient had an avulsion of the inferior glenohumeral ligament (IGHL) from the neck of the humerus. Only one patient had a type I lesion. Twenty-two percent of the patients had, on the West-Point view as well with arthroscopy, a glenoid rim fracture. Fifty-seven percent of the patients had a Hill-Sachs lesion of which 23 were chondral and 34 were osteo-chondral. There were no rotator cuff tears reported in this study. Noteworthy is that these numbers or percentages of arthroscopic pathology are based on findings in young patients.

Hill-Sachs lesion

The Hill-Sachs lesion is considered to be a pathognomonic sign of anterior glenohumeral instability. Calandra and Baker (1989) published the first report of the arthroscopic evaluation in AFASD (n=32).\textsuperscript{17} They graded the posterolateral compression fractures of the humeral head (=Hill-Sachs lesion) in III grades.

Grade I lesion: a defect in the articular surface (chondral) not including the subchondral bone.

Grade II lesion: a defect in the articular surface including the subchondral bone.

Grade III lesion: a large defect in the subchondral bone. (Figure 4)

Fourty-seven percent of patients with an AFASD were found to have a Hill-Sachs lesion. During arthroscopic examination of the shoulder, the Hill-Sachs lesion should not be confused with the "bare spot", which is a normal finding. Among his arthroscopically treated patients with AFASD, Wheeler (1989) found a Hill-Sachs lesion in all 9 patients: 5 chondral and 4 osteo-chondral.\textsuperscript{116} These high percentages are confirmed consistently in later reports.\textsuperscript{1,2,11,21,71} In our own arthroscopic study, we found a Hill-Sachs lesion in 29 out of 31 patients with an AFASD (94%), comparing well to the given results.\textsuperscript{101}

Table 1 shows the incidence of the Hill-Sachs lesion found by arthroscopy. This varies from 40-100%. Obviously, the incidence of the Hill-Sachs lesion diagnosed at arthroscopy is higher than the incidence based on radiographs reported in past literature.
S.L.A.P. lesion

The S.L.A.P. lesion described by Stephan Snyder et al. is an injury to the superior labrum, which begins posterior and ends anterior. Snyder originally divided this pathology into four distinct types. The most common cause is a compression force to the shoulder due to a fall onto an outstretched arm, with the shoulder in abduction and slight forward flexion. The incidence of S.L.A.P. lesions associated with AFASD varies from 3 - 30% of the cases. (Table 1)

![S.L.A.P. lesion type IV](image1.png)

**Figure 5a: S.L.A.P. lesion type IV**

HAGL lesion

The HAGL lesion (humeral avulsion glenohumeral ligaments) was first described by Wolf et al. (1995) and is an obvious avulsion of the inferior glenohumeral ligament (IGHL) complex from its humeral attachment. This lesion is readily recognised arthroscopically.

Bokor et al. found in their retrospective study of 547 shoulders (operated for instability) 41 cases (7.5%) with a HAGL lesion. In patients with AFASD without a Bankart lesion, a HAGL lesion should always be excluded. Warner (1997) described also the possibility of a HAGL lesion in combination with a Bankart lesion following shoulder dislocation.

![HAGL lesion left shoulder](image2.png)

**Figure 5: HAGL lesion left shoulder.**

23
Table 1 Arthroscopic findings in patients with AFASD

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>N</th>
<th>Age</th>
<th>Hill-Sachs</th>
<th>Glenoid rim(#)</th>
<th>Bankart lesion</th>
<th>SLAP lesion</th>
<th>Rotator Cuff</th>
<th>HAGL lesion</th>
<th>Rec</th>
<th>FU month</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calandra</td>
<td>1989</td>
<td>32</td>
<td>av 22</td>
<td>47%</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Wheeler@</td>
<td>1989</td>
<td>9</td>
<td>men</td>
<td>av 19</td>
<td>100%</td>
<td>55%</td>
<td>ND</td>
<td>ND</td>
<td>22%*</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Baker</td>
<td>1990</td>
<td>45</td>
<td>av 21.2</td>
<td>40%</td>
<td>4%</td>
<td>87%</td>
<td>ND</td>
<td>12%</td>
<td>ND</td>
<td>ND</td>
<td></td>
</tr>
<tr>
<td>Norlin</td>
<td>1993</td>
<td>24</td>
<td>av 22</td>
<td>100%</td>
<td>8%</td>
<td>100%</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td></td>
</tr>
<tr>
<td>Uribe</td>
<td>1993</td>
<td>11</td>
<td>av 20</td>
<td>100%</td>
<td>ND</td>
<td>100%</td>
<td>ND</td>
<td>ND</td>
<td>9%*</td>
<td>19-32</td>
<td></td>
</tr>
<tr>
<td>Arciero@</td>
<td>1994</td>
<td>21</td>
<td>av 20.5</td>
<td>95%</td>
<td>24%</td>
<td>100%</td>
<td>8%</td>
<td>0%</td>
<td>ND</td>
<td>14%*</td>
<td>av 52</td>
</tr>
<tr>
<td>Arciero@</td>
<td>1995</td>
<td>26</td>
<td>av 19.5</td>
<td>88.5%</td>
<td>ND</td>
<td>96%</td>
<td>11.5%</td>
<td>NA</td>
<td>3.8%</td>
<td>0%*</td>
<td>av 19</td>
</tr>
<tr>
<td>Molié</td>
<td>1995</td>
<td>34</td>
<td>av 24.2</td>
<td>100%</td>
<td>3%</td>
<td>91%</td>
<td>30%</td>
<td>23%</td>
<td>ND</td>
<td>20%</td>
<td>24</td>
</tr>
<tr>
<td>Taylor-Arciero@</td>
<td>1997</td>
<td>63</td>
<td>av 19.6</td>
<td>89%</td>
<td>22%</td>
<td>97%</td>
<td>9.5%</td>
<td>0%</td>
<td>1.6%*</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Salmon</td>
<td>1998</td>
<td>17</td>
<td>av 21.6</td>
<td>67%</td>
<td>6%</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>6%*</td>
<td>24-48</td>
</tr>
<tr>
<td>Wintzell</td>
<td>2002</td>
<td>30</td>
<td>15 arth</td>
<td>av 24</td>
<td>100%</td>
<td>20%</td>
<td>100%</td>
<td>13%</td>
<td>ND</td>
<td>20%*</td>
<td>3 of 15</td>
</tr>
<tr>
<td>Kirkley</td>
<td>1999</td>
<td>40</td>
<td>19 arth</td>
<td>&lt; 30</td>
<td>94.7%</td>
<td>ND</td>
<td>100%</td>
<td>11%</td>
<td>0%</td>
<td>ND</td>
<td>16%*</td>
</tr>
<tr>
<td>DeBerardino-Arciero@</td>
<td>2001</td>
<td>49</td>
<td>45men</td>
<td>av 20</td>
<td>100%</td>
<td>ND</td>
<td>98%</td>
<td>ND</td>
<td>4%*</td>
<td>12%*</td>
<td>av 37</td>
</tr>
<tr>
<td>Bottomi-Arciero@</td>
<td>2002</td>
<td>24</td>
<td>24 men</td>
<td>av 21.6</td>
<td>100%</td>
<td>ND</td>
<td>100%</td>
<td>ND</td>
<td>23%</td>
<td>ND</td>
<td></td>
</tr>
<tr>
<td>Te Slaa</td>
<td>2002</td>
<td>31</td>
<td>&lt; 40</td>
<td>94%</td>
<td>excluded</td>
<td>87%</td>
<td>3%</td>
<td>23%</td>
<td>ND</td>
<td>39%</td>
<td>12 of 31</td>
</tr>
</tbody>
</table>

@ = authors from “Arciero Group” - West-Point military academy.
* = controlled study non-operative versus operative
ND = not documented
Arth = arthroscopic treatment
Rec = Recurrence
1.5 Recurrence of AFASD

In reviewing the available literature on all natural history studies, Kirkley (1999) found an overall recurrence rate ranging between 17%-96% with a mean of 67%.* Several factors have been suggested regarding the recurrence rate following an AFASD. Factors that might affect the risk of recurrence are age, type of athletic participation, length of immobilisation, rehabilitation, and length of time spent before returning to sports. However, it has been shown clearly that age at the time of the initial shoulder dislocation is the most important prognostic factor for recurrent instability.*

Previous studies have shown high recurrence rates in young patients (< 20 years) varying from 46% to 95%.* A crucial study by Lennart Hovelius has provided us with the only “natural history” study of AFASD with 2, 5 and 10 years of follow-up.*

He also, found a high recurrence rate (62-70%) in the group of patients younger than 22 years of age; but only one-third of these recurrent dislocations needed a stabilisation operation. After a 10-year follow-up, 52% had no additional episodes of dislocation. Fifty-eight (23%) had had a stabilisation operation because of recurrent instability.

*Figure 1: Lennart Hovelius (Gavle, Sweden).

*Figure 2: 10-year prognosis of patients with AFASD: age versus recurrence.
Table 2 shows the recurrence rates in “conservative” or non-operative treated patients. The recurrence rate varies from 0%-95% depending on age.

**Table 2: Recurrence rate in patients with AFASD treated by “conservative” or “non-operative” treatment.**

<table>
<thead>
<tr>
<th>Author</th>
<th>year</th>
<th>number patients</th>
<th>recurrence rate</th>
<th>age</th>
<th>follow-up years</th>
</tr>
</thead>
<tbody>
<tr>
<td>McLaughlin HL</td>
<td>1950</td>
<td>573</td>
<td>90%</td>
<td>&lt;20</td>
<td>0.5-5</td>
</tr>
<tr>
<td>McLaughlin HL</td>
<td>1950</td>
<td>573</td>
<td>60%</td>
<td>20-40</td>
<td>0.5-5</td>
</tr>
<tr>
<td>McLaughlin HL</td>
<td>1950</td>
<td>573</td>
<td>10%</td>
<td>&gt;40</td>
<td>0.5-5</td>
</tr>
<tr>
<td>Rowe CR</td>
<td>1961</td>
<td>324</td>
<td>42%</td>
<td>all</td>
<td>av 4.8</td>
</tr>
<tr>
<td>Rowe CR</td>
<td>1961</td>
<td>324</td>
<td>94%</td>
<td>&lt;20</td>
<td>av 4.8</td>
</tr>
<tr>
<td>Rowe CR</td>
<td>1961</td>
<td>324</td>
<td>74%</td>
<td>20-40</td>
<td>av 4.8</td>
</tr>
<tr>
<td>Rowe CR</td>
<td>1961</td>
<td>324</td>
<td>14%</td>
<td>&gt;40</td>
<td>av 4.8</td>
</tr>
<tr>
<td>McLaughlin HL</td>
<td>1967</td>
<td>181</td>
<td>95%</td>
<td>teenaged</td>
<td>ND</td>
</tr>
<tr>
<td>Henry JH</td>
<td>1982</td>
<td>121</td>
<td>88%</td>
<td>av 19</td>
<td>ND</td>
</tr>
<tr>
<td>Yoneda B</td>
<td>1982</td>
<td>124</td>
<td>17%</td>
<td>21</td>
<td>13</td>
</tr>
<tr>
<td>Aronen JG</td>
<td>1984</td>
<td>20</td>
<td>25%</td>
<td>av 19.2</td>
<td>3</td>
</tr>
<tr>
<td>Simonet WT</td>
<td>1984</td>
<td>116</td>
<td>33%</td>
<td>12-96</td>
<td>av 5</td>
</tr>
<tr>
<td>Simonet WT</td>
<td>1984</td>
<td>116</td>
<td>66%</td>
<td>&lt;20</td>
<td>av 5</td>
</tr>
<tr>
<td>Simonet WT</td>
<td>1984</td>
<td>116</td>
<td>0%</td>
<td>&gt;40</td>
<td>av 5</td>
</tr>
<tr>
<td>Wheeler JH</td>
<td>1989</td>
<td>38</td>
<td>92%</td>
<td>av 18.6</td>
<td>av</td>
</tr>
<tr>
<td>Hoelen MA</td>
<td>1990</td>
<td>54</td>
<td>64%</td>
<td>&lt;30</td>
<td>4</td>
</tr>
<tr>
<td>Vermeiren J</td>
<td>1993</td>
<td>-</td>
<td>68%</td>
<td>&lt;20</td>
<td>4</td>
</tr>
<tr>
<td>Alciero RA</td>
<td>1994</td>
<td>15</td>
<td>80%</td>
<td>av 19.5</td>
<td>2</td>
</tr>
<tr>
<td>Hovelius L.</td>
<td>1996</td>
<td>257</td>
<td>66%</td>
<td>&lt;20</td>
<td>10</td>
</tr>
<tr>
<td>Hovelius L.</td>
<td>1996</td>
<td>257</td>
<td>56%</td>
<td>20-30</td>
<td>10</td>
</tr>
<tr>
<td>Taylor DC</td>
<td>1997</td>
<td>53*</td>
<td>90%</td>
<td>av 19.1</td>
<td>ND</td>
</tr>
<tr>
<td>Arciero RA</td>
<td>1997</td>
<td>17-23</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kirkley A</td>
<td>1999</td>
<td>19*</td>
<td>47%</td>
<td>&lt;30</td>
<td>2</td>
</tr>
<tr>
<td>Wintzell G</td>
<td>1999</td>
<td>15*</td>
<td>60%</td>
<td>av 24</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kraalinger FS</td>
<td>2002</td>
<td>241</td>
<td>16.7%</td>
<td>&lt;20</td>
<td>13-76 months</td>
</tr>
<tr>
<td>Kraalinger FS</td>
<td>2002</td>
<td>241</td>
<td>61.3%</td>
<td>21-30</td>
<td>13-76 months</td>
</tr>
<tr>
<td>Bottoni CR</td>
<td>2002</td>
<td>14*</td>
<td>75%</td>
<td>av 23</td>
<td>3</td>
</tr>
<tr>
<td>men</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>men</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>te Slaa RL</td>
<td>2003</td>
<td>107</td>
<td>64%</td>
<td>&lt;20</td>
<td>46-91 months</td>
</tr>
<tr>
<td>te Slaa RL</td>
<td>2003</td>
<td>107</td>
<td>37%</td>
<td>20-40</td>
<td>46-91 months</td>
</tr>
<tr>
<td>te Slaa RL</td>
<td>2003</td>
<td>107</td>
<td>6%</td>
<td>&gt;40</td>
<td>46-91 months</td>
</tr>
</tbody>
</table>

* = "conservative" part of the controlled study.
ND = not documented
av = average
1.6 Immobilisation and rehabilitation after AFASD.

The traditional form of treatment following a successful reduction of the shoulder generally consists of some form of immobilisation, such as a sling or collar and cuff with the injured arm in internal rotation. There remains some controversy on the role and duration of immobilisation following first-time anterior shoulder dislocations and the ability to affect the rate of recurrence. In older literature based on the healing of the labrum pathology, the recommended duration of immobilisation varies from 3-6 weeks, depending on age, first-time versus recurrence and associated injuries.\textsuperscript{86,114}

Watson-Jones (1948) reported that he found no case of a recurrent dislocation in several hundred cases of AFASD treated by complete immobilisation for more than 4 weeks.\textsuperscript{114} Kiviluoto et al. (1980) showed a lower recurrence rate of 22\% in 27 patients younger than 30 years of age with a 3 week immobilisation of the shoulder compared with a recurrence rate of 50\% in 26 patients of the same age and with an immobilisation of only 1 week.\textsuperscript{57} They recommended longer immobilisation (3 weeks) for patients younger than 30 years of age and shorter immobilisation (1 week) for patients older than 30 years.

Yoneda et al. (1982) reported a recurrence rate of only 17\% in 124 young athletes treated with a more prolonged period of immobilisation (5 weeks) and limited range of motion for an additional 6 weeks.\textsuperscript{121}

Aaronen and Regan (1984) found a recurrence rate of 20\% in United States Navy men who had also a specific aggressive rehabilitation program after the initial dislocation.\textsuperscript{4}

In contrast to these, in many reports, the incidence of recurrence appears to be insensitive to the type and duration of the immobilisation of the shoulder after an AFASD.\textsuperscript{40,43-45}

Simonet and Cofield (1984) concluded that immobilisation had no effect on the dislocation recurrence rate.\textsuperscript{97} The only significant factor in treatment which influenced the final result was a restriction on returning to sports or full activity for more than 6 weeks.

In his prospective study (10-years) Hovelius et al. (1996) followed 257 patients (all less than 40 years of age) following a first-time dislocation.\textsuperscript{45} After reduction approximately half were treated with immobilisation for 3 to 4 weeks; the others were treated with early range of motion. They concluded that immobilisation did not influence the rate of recurrence. This has been emphasised by others, who could not establish a positive relationship between immobilisation and the chance of recurrence following an AFASD.\textsuperscript{36,55,57,68,86}

The question of whether the position of immobilisation after AFASD could influence the recurrence rate is still under investigation. Itoi et al. (1999), based on their cadaver - and MRI study, concluded that immobilisation of the arm in external rotation allows a better approximation of the Bankart lesion to the glenoid than does in internal rotation.\textsuperscript{48,49} They have already proposed a prospective study to verify the clinical benefit of their observations.
We could not find any scientific nor any biomechanical data about the usefulness of brace treatment to prevent recurrence following AFASD, despite claims of success by many of the manufacturers. The purpose of bracing is to prevent abduction and external rotation, thereby avoiding the position that places the patient at risk for dislocation. This is almost impossible in sports, such as basketball, baseball and volleyball, where a high level of shoulder mobility is required.

Rehabilitation
Following the initial immobilisation, a shoulder rehabilitation program is implemented. We could not find any randomised-controlled trials (RCTs) nor reviews on this subject. Only a few retrospective studies were found in PubMed (Medline 1966-2002). When considering the role of physical therapy, it has produced varied results among investigators. In the series of Yoneda (1982) and Aaronen (1984) 75-83% of the patients were treated successfully with immobilisation and physical therapy. Simone and Cofield (1984) recommend an intensive rehabilitation program before returning to athletic activity. Others were only able to show a 17% success rate in traumatic shoulder dislocations. The group of Arciero and Taylor (1998) also showed poor results after rehabilitation alone. Hovelius reported that age at time of initial dislocation was more important than rehabilitation or length of immobilisation. To restore the range of motion is the initial goal in any shoulder rehabilitation program. Following an acute injury and appropriate initial immobilisation, one should attempt to decrease the amount of pain. Pendulum exercises are begun as soon as the immobilisation device is removed.

1.7 Associated lesions of AFASD

1.7.1 Fractures

Greater tuberosity fractures (GT)
Greater tuberosity fractures in association with anterior shoulder dislocations are not unusual. About 15-35% of all anterior shoulder dislocations have a GT fracture. Johnson and Bayley (1982) found a 24% incidence of these fractures following anterior shoulder dislocation in their series of the Royal National Orthopaedic Hospital in London. In his thesis, Visser (1998) reported that out of 77 patients with an AFASD, 14 patients had a fracture (18%). Twelve of these 14 patients had a GT fracture. Two had a glenoid rim fracture. According to Hovelius (1983) these GT fractures are 4 times more common in patients older than 30 years.
In recent studies involving a large number of patients with acute first-time anterior shoulder dislocations, Saragaglia (2001) and Robinson et al. (2002) found a GT fracture in 22% and 17%, respectively. According to Robinson, none of the patients with an isolated GT fracture had a redislocation within six weeks. However, those with a fracture of both the glenoid rim and the GT (2%), had an increased risk of early redislocation (relative risk = 35.5). The risk on recurrence is very low following AFASD with an isolated GT fracture.

**Glenoid rim fractures**

Anterior glenoid rim fractures following anterior shoulder dislocations are well known. The incidence ranged from 3% to 32%.

Avulsions of the glenoid labrum and glenoid rim fractures are more commonly seen in young individuals following a major injury.

In patients over the age of 35 years, traumatic instability tends to be associated with fractures of the greater tuberosity and rotator cuff tears. The indication for conservative or surgical treatment is controversial. A limited number of reports on this subject are available in the literature. Because large fragments (more than 25-33%) of the glenoid can lead to recurrent instability, operative reposition is advised. Rowe (1956), found in his series of 500 shoulder dislocations fracturing of the anterior glenoid rim in 5.4% of the cases. The recurrence rate in this group was 62%.

The aforementioned studies of Saragaglia (2001) and Robinson et al. (2002) found a glenoid fracture in three- and five percent, respectively. According to Robinson, the patients with an isolated glenoid rim fracture have higher relative risk of a redislocation within six weeks. Seven out of eleven patients (64%) with a fracture of both the glenoid rim and the GT had a redislocation within six weeks. This tendency increases with age at the time of the initial traumatic dislocation.
Hill-Sachs Lesion, (posterolateral humeral head impression fractures)

Hermodsson (1934) described the first well-documented case of this “typical defect” that occurred following an AFASD.\textsuperscript{37} He suggested that the defect was a frequent occurrence. Hill and Sachs (1940) published an article in which they stated that this “typical defect” was actually a compression fracture caused by the cortical bone of the anterior glenoid on the posterolateral side of the humeral head.\textsuperscript{38} They also described the so-called “line of condensation”, which is a sharp line detectable on radiographs of the shoulder with the arm in internal rotation.

A Hill-Sachs deformity is an osteochondral defect in the superior aspect of the posterolateral margin of the humeral head that results from impaction of the humeral head on the rim of the glenoid at the time of anterior dislocation of the shoulder.

The Hill-Sachs lesion is considered to be pathognomonic for anterior glenohumeral instability. The Hill-Sachs lesion is more often detected after an acute first-time anterior shoulder dislocation, if the correct radiographic views are taken.

There have been several publications in the available literature describing the best radiographic view to demonstrate the presence of this lesion.\textsuperscript{37,38,78,88} Hill and Sachs (1940) and Hermodsson (1934) emphasised the importance of an AP view with the arm in maximum internal rotation.\textsuperscript{37,38} A careful radiographic evaluation is necessary to demonstrate this defect. This defect is usually seen on the AP view with the arm in 50 to 60 degrees of internal rotation. Clinical studies of Pavlov and Warren et al. (1985) and Rozing and de Bakker et al. (1986) support the usefulness of the Stryker notch and maximal internal rotation views.\textsuperscript{78,88}
However we should realise that these conventional radiographs will give an under estimation when comparing to, for example, arthroscopic evaluation.

Visser reported in his thesis that in 48% of the AFASD, he found a Hill-Sachs lesion on the 45°-cranio-caudal view. He could not find this impression fracture in 42% of the cases. Thirty-one out of 37 Hill-Sachs lesions were "small" (= a small irregularity of the cortex) and in six patients the Hill-Sachs defect was large.

On MRI this deformity is detected as an indentation in the humeral head. If there has been a recent episode of anterior instability, there may be high signal intensity in the cancellous bone surrounding the deformity on a T2-weighted image. A cyst sometimes forms adjacent to a Hill-Sachs deformity in patients who have a history of multiple dislocations.

There is ongoing debate about whether the Hill-Sachs lesion will produce a higher recurrence rate. The matter still generates controversy; however, some authors consider a large Hill-Sachs lesion a "positive" prognostic factor for recurrence.

**Coracoid process fractures**

In contrast to the above-mentioned fractures, the coracoid process fracture, first described by Desault in 1768, is a rare injury. The reported incidence is between three percent and thirteen percent for fractures around the shoulder. Most reports of coracoid fractures are isolated cases. Despite the fact that dislocation of the shoulder is a common disorder, we could only find 10 reported cases in the literature on shoulder dislocations, which had a simultaneous fracture of the coracoid process.

We have published material in the past concerning the rare combination of a fracture of the greater tuberosity and coracoid process following an AFASD with a glenoid rim fracture. Most coracoid fractures are minimally displaced and usually heal well with conservative treatment.

**1.7.2 Vascular injury**

Because of the elasticity of the axillary artery, vascular injury associated with anterior shoulder dislocation is an unusual but potentially serious occurrence. The exact incidence is not known, but more than 200 cases of shoulder dislocation and axillary artery injury have been reported on reviewing the literature to date.

Vascular damage tends to occur more frequently in elderly patients with stiffer and more vulnerable vessels. In the literature, vascular injury occurs most often when chronically dislocated shoulders in elderly patients are reduced by closed methods. In the series of Calvet, this gave a profound mortality of 50%.
Chapter 1

RECURRENT OR HABITUAL DISLOCATION 
OF THE SHOULDER-JOINT.

BY 
A. S. BLUNDELL BANKART, M.C.CANTAB., F.R.C.S., 
ORTHOPAEDIC SURGEON, MIDDLESEX HOSPITAL; SURGEON, ROYAL 
NATIONAL ORTHOPAEDIC HOSPITAL AND HOSPITAL FOR 
EPILEPSY AND PARALYSIS, MAIDA VALE.

Recurrent dislocation of the shoulder-joint is an uncommon condition, the real nature of which appears to be little understood. It may almost be said to be peculiar to athletes and epileptics—a rather curious association, which, as I shall show, is not without etiological significance. Most of the former class are powerful, healthy, athletic young men to whom the frequently recurring dislocation from trivial causes is a great and serious disability. In epileptics the dislocation sometimes recurs with every fit. The dislocation is nearly always anterior. I have only seen one case of posterior recurrent dislocation. That case was in a woman, and she was not operated upon.

Figure 1: Citation of Bankart (1923) from his article “recurrent or habitual dislocation of the shoulder joint.”

Simonet and Cofield (1984) found a correlation between participation in sport and a higher recurrence rate in young patients with AFASD.97 There was an 82% recurrence rate among athletes compared with only 30% among non-athletes.

Wheeler and Arciero (1989) also found high recurrence rates among 85% of non-operatively treated patients in a population of highly trained military personnel.116 Hovelius and others, however, could not support this aforementioned theory.40,45,58 Hovelius (1996) showed that different levels of athletic activity at the time of the first dislocation did not influence the rate of recurrence after ten years.45 Other authors such as, Hoelen et al. and recently Kralinger have also found no positive correlation between sports activity and recurrence rate. 40,58

We should realise that the last three mentioned studies were performed in the general patient population in contrast to the selected patient groups used in that of Wheeler and Arciero.116 High sport activity level and high endurance in military personnel provided higher recurrence rates. When considering return to normal functional activity, some studies have shown that many patients do not appear to achieve the same level of sports activity as they had prior to AFASD.60,82
1.9 Treatment of AFASD

Because of the low recurrence rates in the older patients, a "conservative" approach is still justified here. The choice of treatment of the AFASD in the young patient is still a matter of debate. Few studies, conducted during the past few years, particularly from the group of Wheeler and Arciero, have shown a significantly reduced recurrence rate in patients with AFASD who have undergone arthroscopic stabilisation. They found recurrences in 85% of the non-operatively treated patients versus 13% in the surgically treated group. There are however, three points of weakness of importance in their studies. Firstly, it should be noted that their group consists of a patient population of highly trained military personnel (cadets from West Point academy), which is not representative of a general patient population. Secondly, in their studies the groups were not randomised and finally, the duration of the follow-up studies was limited to 2 years. It was only, until the year 2001 that their 2 to 5-year follow-up results were published. Of importance is the fact that they have changed their stabilisation technique three times in the last 10 years. Unfortunately, to date, there are no long-term follow-up studies about the arthroscopic stabilisation of the AFASD.

The publications of Wintzell et al. also reported on their experience of arthroscopic lavage of the shoulder after an AFASD. In these studies, with a short follow-up (0.5, 1, 2 years), they found a significantly lower recurrence rate after lavage without stabilisation, compared to group treated by traditional methods. We found in our 5-year follow-up study on AFASD an increasing recurrence and instability rate following arthroscopy. This in contrast to Wintzell's theory concerning the lavage method as treatment for the young patient with AFASD.

The literature was searched for evidence supporting an operation directly following an AFASD, as treatment options for the prevention of instability. We found only two prospective randomised clinical trials, comparing arthroscopic stabilisation with a traditional, non-operatively treated group of patients younger than 30 years of age. In the first study by Bottoni et al. (2002), two groups of young athletes with AFASD were compared. Fourteen out of 24 patients were treated conservatively with an instability rate of 75%. Of the nine operatively treated patients, only one (11.1%) developed instability. The follow-up was 36 months. The study involved a small and selected group of patients in a military academy.

The second RCT study, which had a minimum of 24 months follow-up, Kirkley et al. (1999) found a statistically significant difference in rate of redislocation. Sixteen percent of the surgically treated patients (n=20) had a dislocation whereas 47% of the non-operatively treated patients (n=20) had a redislocation. There was also a statistically significant difference in disease specific quality of life, in favour of the operated group. They found convincing evidence that the overall shoulder function (WOSI scale) was better after two years in the stabilised group than in the non-operatively treated group, in patients younger than 30 years.
of age. These results suggest that the idea of direct stabilisation in young athletic patients with AFASD is appropriate. This was recently challenged by the same author, however, after presenting their long term results at the Canadian Orthopaedic Association meeting in June 2002. They found that 31 of 38 AFASD who were followed for an average of 6.5 years had a redislocation rate of 50% at 24 months for the non-operated group and 15% for the immediate arthroscopically stabilised group. They found no difference between both groups on the WOSI scale after 6.5 years of follow-up. In the 15 patients treated conservatively, 8 patients eventually required surgery (53%). In the 16 patients treated with immediate surgical stabilisation 20 surgeries were required at long term follow-up. All but 1 patient in each group returned to their original sports activity. The authors noted that early surgical stabilisation decreases the redislocation rate, but taking this approach resulted in a higher rate of surgery in the group treated with the arthroscope - approximately 50%. In other words, of the initially operated group 50% more surgery was performed in patients younger than 30 years with an AFASD who were initially treated surgically.

There are studies suggesting early arthroscopic stabilisation in the young competitive athlete, however, the follow-up is often short. Others suggest using open techniques in favour of the arthroscopic techniques in patients with collision sports, because of high recurrence rates. The fact that the group of Wheeler, Arciero, and Taylor have not published their long-term results yet, and that their arthroscopic technique varies each time, should make one cautious.

One should realise, that not all studies in the past have shown an evident Bankart lesion as an essential lesion. Furthermore, the results of arthroscopic stabilisation in general have variable success rates with success rates ranging from 59%-100%.

Another important aspect often used in the debate, is that the high level sportsmen as patient, represents a “very small” sample of the population, presenting themselves to an average orthopaedic surgeon. These high level sportsmen often want to have their operation out of the sporting season. An appropriate approach would be to encourage patients to “live with the injury and see if it becomes a disability”. If not, a predictable (open) stabilisation can be performed offering a low recurrence rate.

In summary, it is the authors personal opinion that one should not routinely recommend direct stabilisation for the young patient with AFASD. One should wait for the results of longer follow-up studies of the primary (arthroscopic) stabilisations. In the specific (small) group of (high level) young sportsmen (< 25 years) one should educate and discuss the benefits of any surgical intervention in the light of an eventual recurrence and hence, re-operation.
Indications for "direct" operations in patients with AFASD are:
1. A not reducable shoulder dislocation.
2. Greater tuberosity fractures with large displacement.
3. Glenoid fractures with a large fragment (>25-33%)
4. Vascular injury
5. Instable shoulder because of massive cuff lesion
6. Young (<25 year) high level sportsmen

Table 3: Results of surgical treatment of patients with AFASD

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Study</th>
<th>Aantal N</th>
<th>Recur rate</th>
<th>Age</th>
<th>Treatment</th>
<th>FU mnth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheeler JH @</td>
<td>1989</td>
<td>Retros</td>
<td>9</td>
<td>22%</td>
<td>&lt;21</td>
<td>Arthrosc. Staple + debrid.</td>
<td>14</td>
</tr>
<tr>
<td>Uribe JW</td>
<td>1993</td>
<td>Prosp</td>
<td>11</td>
<td>11%</td>
<td>av 20</td>
<td>Arthrosc. Suture Caspari</td>
<td>av 24.5</td>
</tr>
<tr>
<td>Arciero R @</td>
<td>1994</td>
<td>Prosp</td>
<td>21</td>
<td>14%</td>
<td>av 20.5</td>
<td>Arthrosc. Suture Morgan</td>
<td>av 32</td>
</tr>
<tr>
<td>Arciero R @</td>
<td>1995</td>
<td>Prosp</td>
<td>26</td>
<td>0%</td>
<td>av 19.5</td>
<td>Arthrosc. Bioabs.Tissue tack</td>
<td>av 19</td>
</tr>
<tr>
<td>Hehl G</td>
<td>1996</td>
<td>Retros</td>
<td>30</td>
<td>7%</td>
<td>av 26.1</td>
<td>Arthrosc. Suture Morgan</td>
<td>av 22</td>
</tr>
<tr>
<td>Wintzell G</td>
<td>1996</td>
<td>Prosp</td>
<td>30</td>
<td>13%</td>
<td>av 24</td>
<td>Arthrosc. Lavage</td>
<td>12</td>
</tr>
<tr>
<td>Taylor-Arciero @</td>
<td>1997</td>
<td>Prosp</td>
<td>63</td>
<td>ND</td>
<td>av 19.6</td>
<td>Arthrosc. ???</td>
<td>ND</td>
</tr>
<tr>
<td>Kirkley A</td>
<td>1999</td>
<td>RCT</td>
<td>40</td>
<td>16%</td>
<td>&lt;30</td>
<td>Arthrosc. Suture Caspari</td>
<td>24</td>
</tr>
<tr>
<td>Wintzell G</td>
<td>1999</td>
<td>Prosp</td>
<td>30</td>
<td>20%</td>
<td>av 24</td>
<td>Arthrosc. Lavage</td>
<td>24</td>
</tr>
<tr>
<td>Larrain MV</td>
<td>2001</td>
<td>Prosp</td>
<td>46</td>
<td>4%</td>
<td>av 21</td>
<td>Arthrosc. Suture 22 // bone anchor suture 6</td>
<td>4-18</td>
</tr>
<tr>
<td>De Berardino TM @</td>
<td>2001</td>
<td>Prosp</td>
<td>49</td>
<td>12%</td>
<td>av 20</td>
<td>Arthrosc. Bioabs.Tissue tack</td>
<td>av 37</td>
</tr>
<tr>
<td>Bottoni CR @</td>
<td>2002</td>
<td>RCT</td>
<td>10</td>
<td>11.1%</td>
<td>av 21.6</td>
<td>Arthrosc. Bioabs.Tissue tack</td>
<td>av 36</td>
</tr>
</tbody>
</table>

@ = authors from "Arciero Group"
RCT = randomised controlled trials
Prosp = prospective study
Random = randomised
Retro = retrospective study
ND = not documented
Arth = arthroscopic treatment
Av = average
1.10 Glenohumeral osteoarthrosis following AFASD.

"There is relatively little information in the orthopaedic literature on the subject of glenohumeral arthrosis following dislocation. The incidence of arthrosis after dislocation is unknown."

This was stated in an article by Samilson and Prieto in 1983. Even though 20 years have passed since this was written, little progress has been made on this subject other than the study of the natural history by Lennart Hovelius (1996). In a general population degenerative changes in the glenohumeral joint are rare.

The development of post-dislocation "arthropathy" was described by Samilson and Prieto (1983). They believed that the prevalence of osteoarthrosis following dislocation of the shoulder was unknown and that neither the number of dislocations nor the defects of the glenoid rim or humeral head was related to the severity of the osteoarthrosis. However, we should realise that the grading of glenohumeral "arthropathy" by this method is unreliable as was shown in the study of Ilg and Emery (2001). In his long-term follow-up study Hovelius (1996) found that 23 of 208 shoulders (11%) had mild arthropathy and 18 of 208 (9%) had moderate or severe arthropathy. A few of the shoulders with arthropathy had no recurrence. Interestingly is the fact that shoulders that had a single recurrence had approximately the same degree of arthropathy as was noted in the shoulders that had multiple recurrences or operatively treated dislocation.

Singer et al. (1995), in a long-term follow-up study of the results of fourteen Bristow procedures, found mild arthropathy in six patients, moderate arthropathy in one, and severe arthropathy in three. They postulated that it was the primary dislocation that had initiated the arthropathy and that later recurrences were of minor importance. This corresponds well with Hovelius’s findings and may suggest that the degenerative process starts when the initial dislocation occurs.

Follow-up studies longer than 25 years following AFASD are needed to clarify the influence of the primary trauma or the operation on the development of any arthropathy. In Sweden, shoulder dislocation is a rather common disorder; the prevalence in the general population is approximately 2 percent (200,000 people). Nevertheless, Hovelius stated that a few Swedish orthopaedic surgeons have performed an arthrodesis or a total shoulder replacement in patients who have post-dislocation "arthropathy."

Neer et al. (1982) reported the results of 273 total shoulder replacements and found that 10 per cent of the shoulders had had operative treatment for osteoarthrosis after recurrent dislocation.
However, several authors have described the occurrence of glenohumeral osteoarthrosis after operative shoulder repairs due to recurrent dislocations. Van der Zwaag and Rozing (1999) found in their retrospective study of 66 Putti-Platt repairs, with a mean follow-up period of 22 years, evidence of a radiographic glenohumeral osteoarthrosis in 61% of the studied shoulders. Thirty-five percent of examined shoulders had mild arthrosis.

Advanced glenohumeral osteoarthrosis is a reported late sequela of anterior instability repairs. It is more common following non-anatomic repairs resulting in limited external rotations such as the Magnuson - Stack and Putti Platt repairs or after complicated instability repairs involving metal fixation materials in the joint. It is obvious that longer follow-up studies are required to determine whether the AFASD initiates the start of the degenerative process. Furthermore, instability repairs should attempt to restore normal anatomy without resulting in limitations to external rotation.

1.11 Costs related to AFASD

The costs of the AFASD are medical costs (diagnosis and treatment) and costs related to the inability to work. Very little information is available in the literature about these costs related to the AFASD.

A search in PubMed (Medline 1966-2002) on shoulder dislocations and the items: costs / economic / finance, produced only one relevant study. In this study by Matthews (RCT) he compared intra-articular lidocaine (IAL) with intravenous sedation before reduction of the shoulder. He found a reduction in effective costs of 62% in the group with IAL, mainly due to a shorter period of time spent in the emergency department.

Despite the fact that it was not mentioned in the PubMed (Medline 1966-2002) search, we found some radiographic studies, which suggest that restricting radiographs in the uncomplicated patient with an AFASD will significantly reduce the waiting time in the emergency- and radiology department and therefore reduce the hospital costs. (chapter 8). We did not find studies evaluating costs and reduction methods in AFASD.

Work participation is rarely mentioned in reports on AFASD. This was evaluated more extensively. In our retrospective study of AFASD performed in Delft 34% of the patients did not return to their former place of work. However, in 5 out of 107 shoulders (5%) the injured shoulder was the reason to cease or change work. (chapter 10) We do not have exact information on precisely how long a patient with an AFASD is unemployed, nor when they return to work, and what the costs are related to unemployment.
The data from the Dutch GAK (Gemeenschappelijk Administratie Kantoor) can offer some indication. Unfortunately the GAK Trauma statistics no longer provide up to date statistical information regarding work and trauma and the last available information dates from the period 1992-1993. The GAK statistics provides information on patients who are receiving sickness benefits for more than 6 weeks. According to the figures available, the stream into social security (WAO) in 1992 was 43 out of 527 (8.1%) and in 1993 57 out of 586 (9.7%) in all cases of shoulder dislocation. (Table 4 and Fig 1) In 1992 these 43 patients with a shoulder dislocation represented 0.1% of all cases, that eventually stream in to the social security (WAO) after one year.

### Table 4: Percentage of patients with diagnosis 831.1 (shoulder dislocation) in 1992 and 1993 related to length of illness.

<table>
<thead>
<tr>
<th>period</th>
<th>total</th>
<th>6-13 wks</th>
<th>13-26 wks</th>
<th>26-52 wks</th>
<th>&gt;52 wks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1992</td>
<td>527</td>
<td>308 58.4%</td>
<td>125 23.7%</td>
<td>51 9.6%</td>
<td>43 8.1%</td>
</tr>
<tr>
<td>1993</td>
<td>586</td>
<td>319 54.4%</td>
<td>138 23.5%</td>
<td>72 12.3%</td>
<td>57 9.7%</td>
</tr>
</tbody>
</table>

### Shoulder dislocation

![Figure 1: Curve of patients entering social security (WAO) with diagnosis 831.1 (shoulder dislocation) in 1993 related to length of illness](image)

The GAK administration registered three million people in a year with an new “illness”. There were a total of seven million new reports of illness in the Netherlands. The GAK number should be multiplied by 7/3 to obtain the gross national number. No exact details concerning the situation at present are available, due to the changes in the health and insurance system. Due to the scarcity of the information available in the Netherlands, an America registration system can be used to provide us with guidelines on this issue. This information can be
obtained from the following website: http://www.disabilitydurations.com. This represents the *Official Disability Guidelines* and is based on all employed people in the USA. Their data represents the most recently published material. In their tables we found the following data and guidelines on return to work and length of disability following a shoulder dislocation (ICD 9 831): Their **Return-to-Work Summary Guideline** is as follows:

On average, a working patient with a shoulder dislocation should be able to return to work after 16 days. After 83 days, however, this patient will be at risk of not returning to work at all.

Furthermore, as can be seen in table 2, 50% of the whole group is back at work after 16 days following a shoulder dislocation. After 87 days 100% of the working patients will have returned to work.

**Table 2**

<table>
<thead>
<tr>
<th>10%</th>
<th>20%</th>
<th>30%</th>
<th>40%</th>
<th>50%</th>
<th>60%</th>
<th>70%</th>
<th>80%</th>
<th>90%</th>
<th>100%</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>13</td>
<td>14</td>
<td>15</td>
<td>16</td>
<td>21</td>
<td>34</td>
<td>41</td>
<td>83</td>
<td>87</td>
<td>27.4</td>
</tr>
</tbody>
</table>

*(Calendar days away from work.)*

Noteworthy is that the data obtained from the GAK, as well as from the official disability guidelines comprise all dislocations together. In other words, the number of AFASD occurrences are combined with the number of recurrent dislocations.
Chapter 1

1.12 Aim of this thesis

The central aim of this thesis is to study some aspects of the acute first-time anterior shoulder dislocation. The following questions were formulated:

1. What is the epidemiology of the acute first-time anterior shoulder dislocation (AFASD)?

2. Which reduction method for AFASD is safe and has the highest success rate?

3. What is the standard treatment of patients with acute first-time shoulder dislocation (AFASD) in the Accident & Emergency Departments (A&E) of Dutch hospitals?

4. What is the incidence and clinical significance of abnormalities seen on pre- and post-reduction radiographs taken in the emergency department in patients with AFASD?

5. What is the natural history (prognosis) of the patients with AFASD?

6. What is the intra-articular pathology of the shoulder in patients with AFASD and can this pathology predict the recurrence - and instability rate.
References


56. Kirkley A. Late results of randomized clinical trial comparing the effectiveness of immediate arthroscopic stabilisation versus immobilisation and rehabilitation in first traumatic anterior dislocations of the shoulder. Presented at the 115th annual meeting of the American Orthopedic Association in conjunction with the Canadian Orthopedic Association. June 2002


General introduction and aim of this thesis


General introduction and aim of this thesis


