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An Overview of Multidirectional Instability of the Shoulder

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AN OVERVIEW OF MULTIDIRECTIONAL INSTABILITY

OF THE SHOULDER

By

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This Independent Study, submitted by Troy Ivesdal in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

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ABSTRACT

Multidirectional instability (MDI) of the shoulder is an increasingly recognized clinical entity to physical therapists, yet it remains poorly defined and not fully understood. The clinical importance of correct diagnosis is necessary for rehabilitation and surgical procedures. The purpose of this paper is to address the issue of MDI and its importance in the field of physical therapy.

General anatomy will be presented with attention given to biomechanics which may lead to this pathology. Etiological factors will be discussed including symptoms and proper diagnostic procedures for instability. Finally, treatment of multidirectional instability will be reviewed with a focus on proprioceptive exercises for the shoulder complex.

This literature review will give physical therapists an in-depth look at MDI of the shoulder. This review may also promote further research to determine the most beneficial physical therapy rehabilitation program for multidirectional instability.

CHAPTER I

INTRODUCTION

The shoulder is the most mobile joint in the body and is very complex in its function, which can make diagnosis very difficult. Diagnosis of instability of the shoulder is particularly challenging. Not only is there anterior and posterior instability to consider, the concept of multidirectional instability has recently been introduced.

Classification of instability is based on an algorithmic approach compiled of many factors including: direction, degree, chronology, cause, frequency, and volition. Thomas and Matsen[1] use the acronyms TUBS and AMBRI for the majority of instability classifications. The TUBS acronym represents: Traumatic instability, Unidirectional in nature, Bankart lesion, and the condition usually responds to Surgery. The AMBRI acronym represents patients with: Atraumatic causes, Multidirectional in nature, usually present Bilaterally, and which respond to Rehabilitation. These descriptions of conditions are by no means something on which we should base our diagnosis and treatment, but something that classifies types of instability.

As stated before, instability can be unidirectional or multidirectional. Multidirectional instability is instability in multiple planes and predominantly in two planes--either inferior and anterior or inferior and posterior. It is uncommon for patients to have instability in all planes--anterior, posterior, and inferior.

It is very important to correctly diagnose instability of the shoulder. Correct diagnosis is necessary for rehabilitation purposes so all forms of instability are treated appropriately. An accurate diagnosis is also needed for surgical approaches. Research has shown unrecognized instability to be one of the larger causes of failure of surgical repairs of anterior glenohumeral dislocation.[2]

The management of multidirectional shoulder instability can be either conservative or operative. The conservative treatment should be tried before an operative approach. Conservative treatment consists of strengthening the shoulder complex to stabilize the joint. A surgical procedure, inferior capsular shift, is done if conservative measures fail. Conservative and surgical intervention will be discussed more intensively later.

The purpose of this paper is to address the issue of multidirectional instability and its importance to physical therapists. Anatomy and biomechanics will be addressed and attention will be given to the biomechanics that may lead to this pathology. Etiological factors will be discussed, along with symptoms and proper diagnostic procedures for instability. Treatment of multidirectional instability will also be reviewed.

CHAPTER II

ANATOMY AND BIOMECHANICS

There are numerous components that contribute to making the shoulder joint a more stable joint. As stated before, the shoulder exhibits the greatest amount of mobility of any joint and this inherently makes the shoulder unstable. The anatomic factors that help stabilize the osseous structures are the joint geometry, the ligamentous restraints, and the dynamic stabilizers of the shoulder complex. These three components work in unison to successfully provide stability, but if one of these lines of defense falters, instability may arise.

Joint Geometry of the Humeral Head and Glenoid

The shoulder's articular geometry has always been perceived to be less important as a stabilizing factor compared to other joints. This is due to the small area and relative shallowness of the glenoid compared to the humeral head.[3-6] The joint geometry of the glenohumeral joint allows for maximum mobility. The convex head of the humerus fits into the concave glenoid fossa representing a ball and socket joint. The humeral head is much larger than the small glenoid fossa. The surface of the glenoid fossa is only one-third to one-fourth that of the humeral head.[3-6] This relationship translated to only 25-35%. The humeral head is, therefore, only in contact with the glenoid fossa at any given time during motion.[2] The surface area mismatch may also be expressed by the glenohumeral index (maximum diameter of the glenoid/maximum diameter of the humeral head).[6] This ratio is about 0.75 in the sagittal plane and is about 0.6 in the more critical transverse plane. It has been suggested that lower values of the index would indicate glenoid dysplasia and are associated with anterior instability.[4] This theory has not been supported by further research.

The humeral head faces medially, posteriorly, and superiorly in regard to the shaft of the humerus, and is normally retroverted with respect to the shaft at an angle of 25-35 degrees.[4] A high retroversion angle has been implicated as a causative factor in recurrent anterior dislocations.[4] Radiographically, however, there has been no difference found in the retroversion angle between normal shoulders and shoulders with anterior instability. In theory, a humeral head with a low retroversion angle would probably present itself with posterior instability, although this has not been proven.

The glenoid fossa faces slightly superior, anterior, and lateral. The glenoid articulation demonstrates a retroversion angle averaging seven degrees with respect to the plane of the scapula in most normal shoulders.[3] Saha[3] has shown that there are significant variations in the shape and the contour of the fossa. He has emphasized the importance of the retroverted orientation of the fossa for stabilization of the glenohumeral joint.

The relative position of the glenoid fossa suggests different instabilities. Saha[3] suggested that an anteversion of the glenoid was associated with anterior instability of the joint. Randell and Gambrioli[4] used computed tomography (CT) to perform glenohumeral osteometry. Using 50 normal subjects and 40 patients with recurrent anterior dislocations, they found no significant differences in the glenohumeral index, glenoid anteroposterior orientations, and humeral retrotorsion. No cases of anteversion of the glenoid fossa in either stable or unstable shoulders were noted.

Brewer et al[4] measured the retroversion of the glenoid in ten adolescents with posteriorly unstable shoulders. They concluded that excessive retroversion is a developmental deformity and is considered the primary etiology of posterior instability of the shoulder.[3] Basmajian[6] felt the position of the articular surface also could contribute to inferior stability for the glenohumeral joint during a resting position. Basmajian felt the superior tilt of the articular surface, along with the effect of the superior capsule and anterior superior glenohumeral ligament, contributed to this inferior stability. Therefore, if any of these components are altered, inferior instability and multidirectional instability could result.

Glenoid Labrum

The glenoid labrum is a rim of fibrocartilage attached around the glenoid fossa. The labrum is lined by a synovial membrane internally and is attached to the capsule externally.[4] The labrum is continuous with the periosteum of the scapular neck. It is a fibrous structure that forms a ring around the periphery of the glenoid and also acts as an anchor point on the glenoid for the capsuloligamentous structures.[6]

It has been a widely held belief that the labrum adds stability by increasing the depth of the glenoid.[4-6] Soslowsky and associates[6] and Bowen and associates[6] feel that the labrum may contribute with stability by increasing surface area and acting as a load bearing structure for the humeral head.

In conjunction with the glenohumeral joint geometry and the glenoid labrum is the concept of concavity compression. The idea refers to the stability afforded a convex object that is pressed into a concave surface. Lippit and Matsen[7] investigated the concavity factor by observing 10 frozen glenohumeral joints in which the muscles and tendons of the deltoid and rotator cuff were resected. Resection of the labrum significantly decreased the compression stability, averaging approximately 20% less resistance to translating forces for each direction. The cadaver study revealed the deeper the glenoid concavity, the greater the translational force required before instability occurred.[7]

Lippit and Matsen[7] proposed, based on these experiments, that stability would be compromised if the glenoid is smaller or flat, if the labrum has become weakened, or when the concavity has been lessened by injury or wear. Lippit and Matsen theorize that atraumatic MDI may be caused by relative flatness of the glenoid articular surface which would cause these patients to have abnormal subluxation in multiple directions.

Ligamentous Restraints

Another component that adds to glenohumeral stability is the ligamentous structures of the shoulder complex. The three glenohumeral ligaments, consisting of the superior, middle, and inferior ligaments, are thickened areas of the anterior, posterior, and inferior joint capsules.[3-5]

The superior glenohumeral ligament arises from the superior glenoid tubercle, the upper part of the glenoid labrum, and the base of coracoid process.[5] The ligament runs inferior and lateral to the humerus between the upper part of the lesser tuberosity and the anatomical neck.[5] The primary function of this ligament is prevention of inferior displacement of the humeral head in the adducted, dependent positioned arm.[4] The ligament also restricts anterior and inferior translation of the humeral head. When this structure is sectioned, the head of humerus will sublux inferiorly.[3]

The middle glenohumeral ligament passes from the anterior margin of the glenoid fossa to the anterior aspect of the anatomical neck and lesser tuberosity of the humerus.[4] The ligament lies under and blends into the subscapularis tendon and becomes tight in external rotation and prevents anterior translation of the humeral head in this position.[5] This structure shows the greatest variation in size and is absent or poorly defined in 30% of shoulders.[2] The middle glenohumeral ligament and the subscapularis tendon function together to limit lateral rotation between 0 and 90 degrees of elevation.[4]

The inferior glenohumeral ligament extends from the anteroinferior labrum and glenoid lip to the lesser tuberosity of the humerus just inferior to the middle glenohumeral ligament.[4] Turkel et al[5] pointed out three parts to the ligament: the superior band, the anterior axillary pouch, and the posterior axillary pouch. Turkel[5] proposed that the superior band was a major stabilizer of the joint. The ligament becomes taut in abduction, extension, and external rotation and limits anterior-inferior translation in this position. O'Brien et al.[6] have redefined this structure as an inferior glenohumeral ligament complex that functions like a hammock supporting the humeral head in the glenoid during abduction and rotation of the shoulder joint.

Another ligamentous structure which contributes stability to the glenohumeral joint is the coracohumeral ligament. It originates from the anterolateral base of the coracoid process and extends two bands over the top of the shoulder, blending with the capsule at the greater and lesser tuberosities. It appears to resist inferior subluxation of the humeral head, but its function is not fully understood.[3]

Musculotendinous Cuff

Stability of the glenohumeral joint is also provided by the rotator cuff muscles. First, the muscles provide a passive role in joint stability. An increased passive arc of motion was demonstrated by several investigators when the muscles were removed.[3] Howell and Galinat[3] have demonstrated that, when the soft tissues and muscles are removed, up to 10 mm of additional superior and inferior translation may occur. Oveson and Nielsen[3] have also shown increased translation, both anterior and posterior, with shoulder muscle release in the cadaver specimen.

The second method of muscular stabilization is compression of the articular surfaces through muscular contraction. Muscular control is primarily by the musculotendinous cuff. The tendons of the cuff muscles (supraspinatus, infraspinatus, teres minor, and subscapularis) blend with and reinforce the joint capsule.[4] These muscles provide active support and can be considered dynamic ligaments.[4] Contractions of these muscles provide a centering of the humeral head in the glenoid fossa.[3] This is independent of balanced muscle activity because the centering phenomenon will still take place even if the anterior muscles contract while the posterior muscles remain relaxed. It would appear that the contraction of the shoulder muscles would produce a tightening of the ligamentous structures. The rotator cuff musculature rotates the shoulder to a stable configuration and tightens the capsular ligaments in the direction opposite the rotation.[3] The rotator cuff tendons blend into the shoulder capsule and promote stability by contracting to produce tension within the capsular ligaments and tighten the capsule. This concept is referred to as dynamic ligament tension.[3]

The last element of dynamic stability is accomplished via neuromuscular control. Individuals can use proprioception to produce muscular contraction and prevent the humeral head from subluxation. The muscles of the rotator cuff and the deltoid work in force couples to stabilize the joint by maintaining the humeral head contact with the glenoid. The rotator cuff muscles need to work in synchrony to maintain these force couple relationships.

Two other mechanisms that add to stability of the joint are limited joint volume and adhesion/cohesion of joint surfaces.[3] Inside a normal joint capsule there is a small amount of fluid (less than 1 cc).[3] This joint normally displays a negative intra-articular pressure which adds a small amount of resistance to distraction and displacement of the humeral head.[3] There also exists a cohesive bond between the humerus and glenoid through viscous and intermolecular forces.[3] This seems to be more of a factor when the gap between the articular surfaces decreases.

CHAPTER III

ETIOLOGY

Multidirectional instability has been simplified into the acronym of AMBRI, which was previously discussed. Clinicians should not limit ithemselves to thinking MDI is always atraumatic in nature. Neer[2] believes two or all three etiological factors are seen in varying proportions: 1) one or more episodes of significant trauma as in wrestling or football, 2) repetitive minor injury and stress on the capsule as in gymnastics and overhead manual labor, and 3) varying degrees of inherent ligamentous laxity, which is usually milder than in Ehlers-Danlos syndrome. Neer[2] feels that all three factors are combined.

Some research suggests that certain individuals may be more predisposed to capsular laxity. Uhthoff and Piscop[6] found capsular redundancy in normal embryos, suggesting that the redundancy seen in patients with instability might be the primary cause rather than the secondary problem. It has been suggested that there might be an intrinsic connective tissue disorder in these patients, causing the capsule to be lax. Belle and Hawkins[6] tried to determine a difference in type III collagen in patients who have MDI. No difference was found, but synthesis of type III collagen in vitro was higher than the skin fibroblasts from the MDI patients. This could relate to the tissue collagen turnover or healing response to injury or microtrauma rather than structural differences. There has not been enough information and data to confirm some of these studies. Neer and Foster[7] also proposed that MDI and inferior stability were due to a stretchy and redundant glenohumeral capsule. The redundant capsule allows excessive glenohumeral angles that exceed the scapulohumeral balance mechanism. Instability will then occur before the capsuloligamentous structures are sufficiently tight to provide stability.[7]

Generalized ligamentous laxity is another factor which may cause MDI, including that of the contralateral shoulder.[6] Neer and Foster[7] observed that 50% of patients with MDI had evidence of hyperlaxity. Hawkins[8] observed only an 8% incidence of hyperlaxity in a multidirectional group. Dubs and Gschwend[9] have suggested that anterior dislocation is more common in lax-jointed individuals.

An article by Emery and Mullaji[9] discusses the relationship between general joint laxity and glenohumeral joint instability. Although they found that the majority of 18 shoulders could be classified as displaying MDI and were above the 50th percentile for general laxity, only three of these shoulders were rated as marked laxity (above the 90th percentile). Warner et al[9] observed 25% of "normal" subjects with no prior history of shoulder pain or dysfunction had hyperlaxity and 22% of the instability group had hyperlaxity.

We must realize that glenohumeral instability and glenohumeral laxity are not the same thing. It is useful to define instability as a clinical condition in which unwanted translation of the head of the humerus on the glenoid compromises the comfort and function of the shoulder.[5] By contrast, laxity refers only to the ability of the humeral head to be passively translated on the glenoid fossa.[5] True congenital hyperlaxity causing instability probably is uncommon, as evidenced by the fact that instability is uncommon in children. The presence of MDI confined to one shoulder supports the concept that general joint laxity cannot be the sole factor responsible for such signs. There does not seem to be a relationship between general joint laxity and instability, but there are various opinions.

Another cause of MDI may be the stretching of the capsule ligamentous tissue due to repetitive microtrauma, such as that caused by overhead activities (throwing and swimming).[6] It is possible that after recurrent subluxations the shoulder instability may gradually increase until it presents itself as MDI. Large amounts of translations in any direction may not be symptomatic in the more sedentary individual, but when and if the individual becomes active and is involved in repetitive forces, the translations may start to present as MDI.

Another cause of MDI could be due to abnormal joint anatomy. The concavity factor would be compromised if the glenoid is small or flat, if the labrum is torn or avulsed, or when the concavity has been lessened by injury or wear.[7] Recurrent instability episodes would tend to erode the articular cartilage and further lessen the concavity.

The glenoid center line may not be correctly aligned with the scapula body.[3] Thus, the periscapular muscles may be balanced but would not be able to successfully keep the joint reaction force balanced within the stable arc. As stated before, a ventral tilt of the glenoid is associated with anterior instability and a posterior tilt has been associated with posterior instability. These anatomic deviations would also set an individual up for scapulohumeral imbalance.

Muscle imbalances around the shoulder girdle also may contribute to MDI. Patients with MDI may have external rotator muscle weakness that alters the compressive force of the humeral head into the glenoid concavity. The specialized anatomy of rotator cuff muscles, consisting of the subscapularis, supraspinatus, infraspinatus, and teres minor, as well as the intraarticular long head of the biceps, are situated ideally to actively compress the humeral head into the glenoid cavity. This theory seems to be the one that gives physical therapists the most hope for successful rehabilitation.

Another theory that is related to muscle imbalance as the cause of MDI is an interrupted scapulohumeral balance. Scapulohumeral balance is a theory that proposes that the humeral head is balanced in the glenoid if the net joint reaction force passes through the fossa.[7] As long as the scapula is positioned in such a way that the glenoid fossa encloses the net forces acting on the humeral head, the glenohumeral joint should remain stable. Thus, the periscapular muscles also contribute to stability by aligning the glenoid to the joint reaction force and, if there is a muscle imbalance, there may be a deviation from the glenoid center line.

In a study by Warner et al,[10] Moire topography was used to evaluate scapulothoracic dysfunction. Moire topography is a form of biosterometry and is very useful in depicting the three-dimensional shape of the human body.[10] Static and dynamic Moire evaluations were done on normal and instability subjects. Static Moire evaluation demonstrated an abnormal pattern in 14% of asymptomatic subjects compared with 32% in the instability subjects. The dynamic Moire test demonstrated an abnormal Moire pattern in 18% of asymptomatic individuals and 64% of the instability group. There seems to be a significant association between abnormal scapulothoracic motion and glenohumeral instability; however, whether this represents a primary or secondary phenomenon has yet to be determined.

In Neer and Foster's[11] classic article on MDI, they described three groups of patients who had this diagnosis. The first group had anterior and inferior dislocation with posterior subluxation; the second group had posterior and inferior dislocation with anterior subluxation; and the third group had recurrent dislocation in all three directions. All three groups had laxity of the inferior portion of the capsule. Theoretically, MDI could be instability in just the anterior and posterior directions but the component that almost always seems to be present is the inferior instability. Redundancy of the structures of the inferior portion of the capsule seems to be the major cause of MDI.[11]

Altchek et al[12] observed patients similar to those in Neer and Foster's first group that had anterior and inferior dislocation with posterior subluxation. Unlike Neer and Foster, most of Altchek's patients had a clearly defined anterior Bankart lesion. Neer and Foster stated they had seen other patients who had laxity of the inferior portion of the capsule and a Bankart lesion, but had excluded them from their study. Altcheck et al is not the first to report the coexistence of labral detachment and MDI of the shoulder. This relationship has not been investigated further by others.

After reviewing the literature, there does not seem to be one cause of MDI. Many of the researchers seem to agree that there are multiple factors that may lead to this condition. As stated before, the theories on etiology include traumatic episodes, inherent ligamentous laxity, redundant capsule, muscle imbalance of the rotator cuff and/or the periscapular muscles, repetitive stress on the capsule due to overuse, and abnormal joint anatomy.

CHAPTER IV

DIAGNOSIS AND SYMPTOMS

The predominant symptoms of MDI are pain and weakness.[13] The patient will usually complain of chronic pain about the shoulder and often of pain radiating to the deltoid insertion. If one can elicit a history of discomfort in the shoulder with the arm in several different positions, one must suspect MDI of the shoulder.[14] The pain may not necessarily be in the area of the greatest instability. Initially the pain will be present after activity but as the condition worsens, pain becomes more constant.[13] Localization of the pain to the front or back of the shoulder is less reliable because single plane instability can produce discomfort on the opposite side as well, secondary to the traction placed on the restraining structures.[14]

A complaint of fatigue ache is commonly seen in patients with inferior instability of the shoulder.[14] This complaint may arise when carrying objects, such as books or a brief case, or when working with the arms overhead. This is usually a trademark sign since inferior instability is considered a hallmark of MDI.[11,14] In the late stages of symptomatic involuntary MDI, the patient develops a severe ache of the shoulder during athletics as well as at rest. Night pain is also often present in late stages of MDI. The onset of symptoms associated with MDI may be insidious. Foster[10] separates the MDI patients and their recognition of symptoms into three groups. In approximately one-third of the cases, the athlete may not recall the initial trauma. In another one-third, there will be an episode of mild trauma with chronic aching and weakness from the time of that mild injury. In the remaining third of the athletes, there will be a significant injury causing the onset of symptoms, such as hyperextending and abducting the arm in a football tackle.

Neer[7] believes that MDI may be present in many types of patients, including athletic patients and those who are sedentary and have no history of injury, males as frequently as females, and those in a wide age range. In Neer's series, the average age of patients who had surgery for this condition is 24 years, with ages ranging from 15 to 54 years old. Neer[7] reported that he only operated on one patient who was under 17 years of age and advises against surgery before this age.

There is also the possibility of patients with psychiatric problems. The association of emotional or psychiatric problems with voluntary shoulder subluxation has been clinically described by Rowe.[15] There has been confusion in regard to the terms voluntary and involuntary. When a patient is able to dislocate the shoulder on a voluntary basis with muscular contraction, one must consider associated personality disorders.[15] A patient who can dislocate the shoulder by elevating the arm probably represents an involuntary dislocation. Usually these are painful positions that the patient tries to avoid in functional activities. These position dislocations are usually involuntary, and should not be confused with those patients who intentionally dislocate their shoulders with muscular contraction. This can be a challenging group of patients to assess. The patient will often have minimal to no pain after these shoulder subluxations since they may occur so often. If the patient can continue his/her routine of activities after the instability episode, then one should search carefully for more global problems.[16] Multiple visits to the clinic after enrolling the patient in a conservative rehabilitation program can help in the search for a motivational or emotional base for the patient's shoulder symptoms.

The most useful tool for diagnosis of MDI is a physical examination. It is important for the patient to be comfortable and have confidence in the clinician. It may take several visits before an adequate examination is possible with a painful and unstable shoulder. Patients who are compensating for MDI often are tender to palpation along the medial angle of the scapula.[15] Also, patients with MDI or posterior instability may have trigger areas over the levator scapulae muscle, along the rhomboids, or along the trapezius muscle.[15] Patients may also often have diffuse tenderness along the anterior cuff structures and occasionally over the posterior cuff structures. A neurologic evaluation of the patient should be as thorough as possible. The examiner should consider motor, sensory, and reflex changes. Sillman and Hawkins[15] believe that it is uncommon for patients with MDI to have radiating paresthesias often with no organic physical examination signs.

Usually with single plane instability, decreased ROM in the shoulder is observed.[14] The MDI shoulder, in contrast, is quite flexible. Flexibility of the shoulder with flexion greater than 180 degrees, external rotation to near 90 degrees, and internal rotation to the upper thoracic spine levels is common.[14] Flexibility should be compared to the contralateral shoulder as well as other joints, including the distal upper extremity and the patellofemoral joint.

Every examination for shoulder instability should include an examination of the opposite shoulder, along with the fingers, elbows, knees, as an index of generalized joint laxity. As stated before, generalized joint laxity is not the only cause of MDI, but it may be an etiologic factor. The multidirectional shoulder may be the only unstable joint.

It is also important to emphasize that multiple shoulder problems may co-exist in the same shoulder; ie, impingement syndrome, acromio-clavicular arthritis, and shoulder laxity. An element of impingement, particularly anterior impingement, can be the presenting symptoms in MDI.[4] This is followed by increased excursion of the humeral head, causing impingement of the anterior rotator cuff tendons.

Sillman and Hawkins[15] believe there are two major components of the assesssment of stability of the glenohumeral joint. They believe the first component documents the amount of passive translation of the humeral head in the glenoid fossa when stressed by the examiner. It is important to look for the reproduction of the symptom complex. The second component attempts to reproduce the symptoms of subluxation and apprehension by placing and stressing the shoulder in positions of compromise. As mentioned previously, it is sometimes difficult for the patient to relax for the examiner to perform a simple manipulation in the direction of instability to further document the pathology.[13]

Static testing of shoulder excursion should be a routine part of the shoulder examination.[14] The "load and shift test" is used to assess glenohumeral translation.[15] It is important to ensure that the humeral head is initally reduced concentrically when you are assessing the amount of translation.[15] In patients with MDI laxity, the humeral head may have a resting postion that is nonconcentric. The head may be sitting anteriorly, posteriorly, or inferiorly. At the beginning of any stress testing, the humeral head should be pushed into the glenoid fossa to ensure its reduction in neutral position. The stresses should be done in all directions with special attention to the inferior stress and the probable "sulcus sign" with MDI.

Examination under anesthesia has gained popularity within the last decade. This testing is now thought to be the most definite, accurate, noninvasive test of shoulder instability.[16] Arendt[14] states that evaluation under anesthesia is the most accurate assessor of shoulder instability. It is also important to evaluate under anesthesia since muscle guarding, particularly in a heavily muscled patient, can conceal MDI. Passive translocation of the humeral head on the glenoid defines the limits of humeral excursion. Patients may have more than just unidirectional instability present in a shoulder. In a study done by Cofield and Irving,[16] 66 patients who had surgical repair for anterior shoulder instability were examined under anesthesia and revealed the presence of multidirectional instability. All 66 patients had typical anteroinferior translocatability with the arm in the apprehension position. In addition, 48 had straight anterior translocation, 44 had inferior translation, and 27 had posterior translocation, all to a greater degree than in the opposite asymptomatic side.

We as clinicians must always suspect that MDI may be present in many types of patients, such as the young athletic patient and those with obvious generalized ligamentous laxity who have always been inactive. An improper diagnosis of the instability can be very costly. If a diagnosis of recurrent anterior dislocation is made on a patient with MDI and a standard repair for anterior dislocation is performed, the procedure is very likely to fail. Neer[2] states that the procedure will fail in one of two ways: 1) They do not correct inferior instability, leaving the shoulder unstable downward and in the opposite direction, and 2) by tightening the capsule on one side, the humeral head may become fixed in a subluxation in the opposite direction so that it leads to severe arthritic changes. Another reason for failure is a more serious problem because the fixed subluxation may eventually require total shoulder replacement.

The next phase of assessment is to attempt reproduction of the symptom complex with translation or to elicit apprehension with certain provocative positions of impending subluxation or dislocation. Assessing anterior instability is done by positioning the arm in abduction and external rotation.[15] With increased external rotation and controlled general forward pressure exerted against the humeral head, an impending feeling of anterior instability may be produced (apprehension sign). With the arm in this position, a posterior stress may be exerted on the proximal humerus and the apprehension may disappear. This is called the "Fowler sign" or "relocation test".[15] Posterior instability is actually a subluxation rather that a dislocation.[15] If this is recurrent, this usually can be demonstrated by the patient, either by arm position and forward elevation or by selective muscular control in various postions of elevation with applied internal rotation. The examiner may attempt to reproduce the instability by manually duplicating the stressses. Patients with inferior instability may say that the distal traction on the arm reproduces their symptom complex. This may include pain, parathesia, and anxiety and suggest underlying MDI.

Standard radiographic examination is another diagnostic tool used by physicians to determine shoulder instability. Plain films are a necessity and should include true anteroposterior, tangential scapula, and axillary views of the shoulder.[13] A West Point axillary view may also be useful.[13] The classic radiologic findings for unidirectional instability about the shoulder also apply to multidirectional situations. These situations include the Hill-Sachs lesion[13] and bony fragments off the anterior or posterior glenoid rim.[13] In patients with suspected MDI, awake stress views can be used to look for inferior instability.

This is best documented with 15 to 20 pounds of traction in each hand, with both shoulders compared on a single AP view.[14]

Arthrography is also used for evaluations. The technique is improved if used with tomography when analyzing labral tears and capsular pathology.[14] Studies have reported a high correlation between arthrotomographic findings and surgical pathology.[14] Findings on arthrotomography includes abnormalities of the glenoid labrum, glenoid rim changes, and impression fractures.

Conventional computerized tomography (CT) can identify impression fractures, bony changes at the glenoid rim, and loose bodies.[14] It also has been used to study glenohumeral size, shape, and orientation. Arthrography combined with conventional CT is very beneficial when defining specific labral, capsular, and cartilaginous pathology. Labral pathology including intra-substance tearing, detachment from glenoid margin, and attenuation are found with CT arthrography. Capsular pathology includes distortion of capsular reflections, especially at the site of scapular insertion, capsular irregularity and thickening, and occasionally a capsular tear. If both capsular and labral pathology are found, a diagnosis of instability is made radiographically. This must also be correlated with clinical findings. These diagnostic procedures are not only helpful in making a correct diagnosis of instability, but they also help to determine the appropriate surgical technique to correct the pathology. Both CT arthrograms and plain arthrograms have been used to assess capsular volume.

Although an impression of increased volume can be made, variation in capsular volume and what constitutes an abnormality has not been defined.

Isokinetics can also be used as a diagnostic tool. Foster[13] reports the use of the Cybex exercise machine as a method of quantitating the direction of the greatest instability. Particular attention is paid to the shape of the curve, the range of motion as documented by graph output, the rate of rotation, and comparison of the curve with the other arm.[13]

Ozaki[17] studied the glenohumeral movements of the involuntary inferior and multidirectional instabilities by means of cineradiography. Ozaki[17] used the devised parameters of shoulder center edge and glenoid angles to compare normal shoulders to shoulders with inferior and multidirectional instability. The shoulder center edge (SCE) angle indicates the centralization of the humeral head toward the glenoid cavity. As the SCE value decreases, the lateral deviation of the humeral head is recognized and the glenohumeral joint is predisposing. The glenoid angle indicates the anatomical superior inclination of the glenoid cavity, scapular abduction, and shoulder external rotation. As the glenoid angle value becomes smaller, the glenohumeral stability is enhanced. In the shoulders with the involuntary inferior and multidirectional instabilities, the SCE angle did not increase with abduction, compared to the normal shoulders. At 180 degrees of abduction, there was a significant difference between the normal shoulder (17.56+ or - 5.2) and the involuntary inferior and MDI instabilities (11.5 ± 20.2). The value of the glenoid angle decreases slowly with abduction and reached minimum value at 180 degrees of abduction (57.2 ± 13.2).

The two parameters used in this study are worthwhile indicators of the involuntary inferior and MDI of the shoulder.[17] The movements of involuntary inferior MDI showed not only an excessive excursion and sliding motion at the glenohumeral joint but also a deterioration of scapular abduction and external rotation with the arm progressively abducted. The roentgenogram of the shoulder joint with the arm maximally elevated indicates whether or not there is an involuntary inferior and MDI, and at this postion, these devised parameters can be diagnostically useful for this lesion.

Jalovaara et al[18] investigated the use of autotraction stress roentgenography. The authors felt that roentgenography may be useful for diagnosing recurrent anterior subluxations and anteroinferior multidirectional instabilities. The anterior and inferior shifts found multidirectional instabilities, averaging 27 mm and 26 mm, respectively, were significantly greater than the recurrent subluxations, but the difference was not great enough for accurate differential diagnosis in individual cases. Stress is induced by anteroinferior traction concentrated on the shoulder. The patients grasped their knees with both hands and stretched their shoulders by extending their flexed hip.

There are a number of problems that challenge the understanding of this clinical entity. These problems include: 1) There is no uniform classification for shoulder instability. 2) There is no agreeable grading system. 3) There are numerous new tools to evaluate the shoulder diagnostically and therapeutically.

4) Multiple problems may co-exist in the same patient; i.e., acromioclavicular arthritis, tendinitis, shoulder instability, and cervical spine disease.

CHAPTER V

CONSERVATIVE TREATMENT

Conservative treatment for MDI is the initial treatment of choice. The conservative treatment for MDI consists of shoulder rotation strengthening exercises.[13,14] Foster[13] states that the emphasis should be placed on strengthening the muscles on the side of the joint of greatest instability. Foster reports that internal rotators should be strengthened when the greatest instability seems to be anterior and inferior. The external rotators should be strengthened when the greatest instability is posterior and inferior. Both internal and external rotators should be strengthened if there are true multidirectional dislocations with anterior, posterior, and inferior instability. Arendt[14] states strengthening the rotator cuff muscles below the horizontal plane is the cornerstone of general strengthening program of the shoulder and upper torso.

The strengthening program should be preceded by an attempt to quiet any inflammation when present with relative rest and anti-inflammatory medication.[14] Any deficiencies in range of motion or muscle weakness should be detected and corrected as part of the conservative approach.

Another part of the conservative treatment may require a change of activity. For athletes involved in repetitive overhead activities, evaluation must include analysis of upper extremity mechanics, review of training methods, and correcting the technique of patient's individual stroke/throwing style when appropriate.[14,15] For instance, a butterfly swimmer should change to another stroke. A swimmer's stroke should be analyzed with respect to body roll, position of the shoulder at water entry phase, degree of internal rotation at pull-through phase, and degree of external rotation during the out-of-water phase.[14] Arendt[14] states that often times a swimmer's shoulder symptoms will decrease by increasing body roll, limiting the extremes of shoulder rotation, and/or alternating the side to which one breathes. The motion of the baseball pitcher's delivery should also be changed if there are increased symptoms.

The patient should be monitored clinically with repeated physical examinations during the period of conservative treatment. Foster[13] states that the patient may maintain a clinical sensation of instability, but the pain and discomfort will gradually resolve. At the six-month mark, if the patient is pain free, he may resume prior activities, but this may need to be permanently changed if the symptoms start to recur.

Wilk and Andrews[3] report aggressive rehabilitation as the first line of treatment for shoulder instability. The type and length of the rehabilitation program is dependent on several factors: 1) severity of injury, 2) stage of condition (acute or chronic), 3) age of patient, 4) type of instability (traumatic or atraumatic), 5) range of motion and strength status, and 6) level of activity to which the patient plans to return. Wilk and Andrews'[3] protocol for non-operative treatment is for general shoulder instability, not necessarily MDI. A general instability program should be more than adequate for MDI.

Wilk and Andrews[3] employ a four-phase approach to their treatment program. The program begins with the Acute Motion Phase. In this phase, the goals are to reestablish pain-free ROM, retard muscular atrophy, and diminish the patient's pain and inflammation. Immobilization, with the use of a sling, may be used if there has been a traumatic injury. Employment of immediate motion and strengthening exercises which are pain-free is important to stimulate collagen synthesis and organization of collagen fibers. Initiation of isometrics early in the rehabilitation program is critical in establishing humeral head control and preventing rotator cuff muscular atrophy and cuff shutdown. Modalities may be employed to calm the inflamed shoulder.

Phase II, the intermediate phase, is initiated by Wilk and Andrews[3] when the patient has minimal pain, full ROM, and has a manual muscle test which demonstrates a "good" status of the shoulder musculature. The key goals of Phase II are to reestablish normal arthrokinematics and improve the neuromuscular control of the shoulder complex.

Wilk and Andrews[3] take a more global approach to strengthening the shoulder complex in contrast to Foster[13] who took a more detailed approach to strengthening each side of the shoulder for a certain type of instability. The patient is placed on a program that exercises all the muscles about the shoulder in an attempt to establish global stability via the dynamic stabilizers.

In addition, with any instability, the patient is placed on a vigorous strengthening program for the biceps brachii (long head portion), supraspinatus, and deltoid. These muscles are important humeral head stabilizers and prevent inferior subluxation and excessive superior humeral head migration.

Also in this phase, scapular strengthening exercises are emphasized to provide a stable base of motion. Often the patients with MDI exhibit significant scapular weakness and excessive scapular motion due to collagen deficiencies.

The function of the scapula and surrounding musculature is vital to the overall normal function of the shoulder. Rotator cuff strengthening is usually the rehabilitation choice for many pathologies, but one must remember the rotator cuff muscles arise from the scapula. Weakness of the anchoring muscles that control the position of the scapula may lead to altered biomechanics of the glenohumeral joint. Paine and Voight[19] believe that three basic activities to include when designing a program are scapular pinches, shrugs, and punches. These three basic activities can be complemented by many other scapular strengthening exercises. These exercises address the serratus anterior, middle trapezius, rhomboids, upper trapezius, and levator scapula. Press-ups can also be done to strengthen the lower trapezius and pectoralis minor.

These three exercises can be done with manual resistance, dumbbells, surgical tubing, and/or isokinetic devices. Shoulder shrugs, an upper trapezius/levator scapula strengthening exercise, should probably be performed with surgical tubing in patients with MDI. Long axis distraction, when using heavy dumbbells, may exaggerate the inferior glide of the humeral head and should be avoided. An alternative method of strengthening is the use of manual resistance on the top of the scapula and clavicle. This method removes the stress applied to the inferior capsule of the glenohumeral joint.

Also in Phase II, various manual resistance patterns, such as proprioceptive neuromuscular facilitation (PNF), drills can be performed. The D_2 flexion/extension UE pattern with rhythmic stabilization applied can be very beneficial. This type of drill appears to improve dynamic humeral head control by activating the stabilizing element of the rotator cuff musculature. Later on, the topic of proprioceptive training will be discussed more in depth.

In Phase III, the Advanced Strengthening Phase, all exercises are performed at a slow, controlled rate of contraction followed with high speed contractions. This type of exercise program can be beneficial for the athletic patient. Exercises used include eccentrics, plyometrics, and isokinetics. Wilk and Andrews[3] also emphasized end range strengthening through rhythmic stabilization.

The last phase of the program represents the gradual return to unrestricted functional work and sport activities. It is important for the clinician to continue to follow the patient's progress and to encourage the patient to follow through with dynamic strengthening, neuromuscular control, and proprioceptive awareness drills.

Brostrom et al[20] conducted an experiment studying the effect of shoulder muscle training in patients with recurrent dislocations. Thirty-three shoulders in 29 patients with recurrent shoulder dislocations, of both traumatic and nontraumatic type, were studied. The patients suffered from muscle weakness, and also atrophy of the supraspinatus and infraspinatus muscles. The patients used an isokinetic pulley-weight system to increase strength, coordination, and endurance of the rotator cuff and deltoid. At follow-up one year after completion of the program, all shoulders except five had improved. In the two patients with MDI, improvement of stability after training was slight and of short duration. In conclusion, the authors stated that training of muscle strength, coordination, and endurance should be considered, both in patients with nontraumatic and traumatic type of instability. Factors indicating a less effective result of training were an abnormal skeletal anatomy and/or a multidirectional instability. The stabilizing effect provided by the training, in patients with MDI, was of short duration.

Burkhead and Rockwood[21] reported the effects of a specific rehabilitation program for the shoulder on a group of patients who had traumatic or atraumatic instability and MDI of the shoulder. The rehabilitation program consisted of exercises to strengthen the deltoid, rotator cuff, and scapular stabilizer muscles. The diagnosis and classification of the shoulders into the traumatic and atraumatic groups were based on a carefully taken history, a physical examination, and evaluation of radiographs.

The results of the rehabilitation program showed a substantial difference in the number of satisfactory responses between patients who had traumatic and atraumatic instability. Of the shoulders that had traumatic instability, 15% had a good or excellent result. The shoulders classified as atraumatic subluxation had good or excellent results in 83% of the cases.

The authors reported that in each subgroup, the patients who had posterior instability responded better than those who had anterior instability. The authors felt that because many patients who have posterior instability have a component of hyperlaxity, it appears that muscle strengthening exercises can accommodate for ligamentous and capsular laxity of the shoulder.

The fact that exercises improve the dynamic stability of the shoulder and often can negate the need for operation is not a new concept. Others have found good results with a conservative treatment for dislocation.[21] In the study by Burkhead and Rockwood,[21] more than 80% of the patients with atraumatic subluxations responded to exercises. This is helpful in encouraging patients to continue the exercise program. Neer[2] recommends conservative treatment for one year prior to surgical shoulder reconstruction for MDI.

CHAPTER VI

SURGICAL AND POST SURGICAL TREATMENT

Anatomical pathology should be corrected if possible. Lesions to glenoid rim and labral pathology are often problems.[14] Large labral defects should be repaired if substantial intersubstance fraying and tearing have not occurred. Large bony labral and/or bony defects can be found in one direction with subsequent stretching of the structures in the opposite direction. This can lead to MDI with the most profound instability in one plane. Correction of the labral and bony defect combined with the capsular shift described by Neer and Foster[11] is the most appropriate surgical intervention.

Surgical reconstruction is reserved for those who fail with conservative treatment. Arendt[14] states that before surgery is performed, one must feel confident that: 1) the patient's symptoms are due to his/her shoulder laxity; 2) there are no underlying emotional or motivational factors; and 3) laxity is known.

The principle of the capsular shift is to detach the capsule from the neck of the humerus and shift it to the opposite side of the inferior portion of the neck of humerus. This procedure will not only obliterate the inferior pouch and capsular redundancy on the side of the surgical approach but also reduce laxity on the opposite side.[2,3,11]

Through an anterior approach, the Neer Capsular Shift Procedure requires making a T-shaped incision to the anterior capsule.[2,3,11] The inferior capsular flap is then shifted superiorly and the corresponding superior capsular flap is then reattached inferiorly. This results functionally in double breasting the anterior capsular layer. As a consequence, there is some increased strength to this anterior buttress. In addition, the volume of the glenohumeral capsule is significantly decreased which effectively increases joint stability in multiple directions. The inferior capsular redundancy, a common finding in inferior instability, is particularly reduced with this technique. Patients with MDI with a severe posterior component would be surgically approached from the posterior side of the shoulder. In this way, the capsular redundancy prominent on the posterior side of the shoulder joint would be significantly reduced along with the volume of the entire glenohumeral capsule.[2,3,11]

Lebar and Alexander[22] reported on the results of a capsular shift procedure performed on 10 active-duty patients with an average of 28 months followup. Nine anteroinferior capsular shifts and one posteroinferior capsular shift were performed. Postoperatively, the patients were placed in a sling for six weeks and active-assisted exercises were performed between six to eight weeks. From 8 to 12 weeks, active motion and isometrics were started and PREs were added at 12 weeks postoperatively. Improvements in pain, function, and stability occurred postoperatively in nearly all patients. Loss of total elevation and external rotation were minimal and an average of three vertebral segments of internal rotation were lost. A history of an acute traumatic event was indicative of greater improvement in pain and stability. Only one patient required further surgery for recurrent instability and all but two patients reported overall improvement. Both patients had had a previous instability repair. Lebar and Alexander[22] stated that lifestyle changes that preclude active military service may be necessary even with good surgical outcome.

Neer and Foster[11] had promising results in their preliminary report. The results were graded as satisfactory or unsatisfactory. A satisfactory result meant there was no recurrence of dislocation or subluxation, no significant pain, full activities, normal strength on manual testing compared with contralateral shoulder, and within 10 degrees of full elevation and 40 degrees of rotation compared with the contralateral shoulder. Results are unsatisfactory if criteria are not met. Thirty-two shoulders were followed for more than one year, 17 of them for more than 2 years. Only one of these shoulders received an unsatisfactory result.

The rehabilitation program following a capsular shift procedure is based on five key factors: 1) the type of shift procedure preformed, 2) tissue integrity of the patient, 3) type of patient, 4) desired activity level, and 5) the patient's rehabilitation potential.[3] The rate of progression is slower for a posterior capsule shift as compared to the anterior procedure. The program is also slowed if the patient exhibits significant joint laxity and collagen deficiency, such as elbow hyperextension, thumb hyperabduction, and excessive contralateral shoulder laxity. The rehabilitation of an overhead activity athlete is generally much quicker than that of an nonoverhead athlete. During the rehabilitation program, the physical therapist should frequently assess the joint stability dynamic control and accessory motions at the sternoclavicular and the scapulothoracic joints. The ultimate goal is to return the patient to prior activities as quickly as possible (usually about six months) while maintaining a stable shoulder joint.[3]

The following rehabilitation program is utilized by Wilk and Andrews[3] and is accelerated for the overhead athlete and is generally three to four weeks faster than for nonoverhead athletic patients. Phase I is the first four weeks post-op and is considered the Protection Phase.[3] The goals in this phase are: 1) allow healing of the sutured capsule, 2) begin early protected ROM exercises, 3) prevent muscular atrophy, and 4) decrease post-op pain and inflammation. Postoperatively, the patient is placed into a shoulder immobilizer for two to three weeks. The patient performs all ROM exercises to tolerance. Isometric strengthening exercises are performed also.

Phase II, the Intermediate Phase, runs from week five to end of week ten.[3] Accomplishment of full, nonpainful range of motion by the end of week ten is the goal in this phase. Other goals are to improve strength and neuromuscular control and normalize arthrokinematics. Aggressive joint mobilization techniques are employed along with vigorous stretching exercises and self stretches of the capsule.

Week 10 to 16 is considered the Dynamic Strengthening Phase or Phase III.[3] The goals in this phase are to improve the athlete's strength, power, and endurance with the goal of preparing them to return to sport activities. The criteria to enter Phase III are full, nonpainful ROM, no pain or tenderness, and shoulder musculature strength 70% of the contralateral side. In this phase, the emphasis of strength training is on power, high speed, and high energy exercises emphasizing plyometrics and eccentrics.

The last phase that Wilk and Andrews[3] present is the Throwing Phase, which begins at approximately week 20 to 26. An interval throwing or sports program is employed as the athlete continues to improve the strength of the shoulder complex.

The rehabilitation, as stated before, would be modified for different patient types, but this is a very comprehensive protocol. All of the factors must be taken into consideration when initiating rehabilitation.

CHAPTER VII

PROPRIOCEPTIVE EXERCISES

The shoulder is finely controlled by muscular attachments and proprioceptors found within the joint capsule and musculotendinous unit. Coordinated function of the muscles around the shoulder is essential for athletic function. Without appropriate neuromuscular control, the shoulder can become dysfunctional and unstable.[23]

Shutte and Happel[24] have stated that alteration in joint innervation caused by athletic trauma can occur and markedly affect joint function. Shoulder joint kinesthesia can also be adversely affected as a result of athletic trauma. A cause of recurrent instability that has not been investigated in depth is the loss of peripheral sensory perception and neuromuscular control.

Kinesthesia is the perceived sensation of the position and movement of joints and muscles that plays an important role in coordination of muscular control of peripheral joints.[23] Freeman et al[25] suggested that functional instability of the foot/ankle resulted from muscular incoordination consequent to rupture of afferent nerve fibers in damaged ankle joint ligaments and capsules. Increased laxity of joints may be related to delayed protective reflexes. Thus, subtle changes in the sensory system, specifically deficits in shoulder joint and muscle kinesthetic sensibility, may predispose the glenohumeral joint to instability and, therefore, to reinjury.

Smith and Brunolli[25] examined kinesthesia in normal (uninjured) shoulders and in shoulders with a history of glenohumeral joint dislocations. Three tests were used to measure kinesthesia in both shoulders of all subjects during one testing session. The angular reproduction test was used to examine the subject's ability to reproduce an angle when the shoulder was placed in intermediate ranges of lateral rotation. The threshold to sensation of movement test was used to examine the threshold to the sensation of movement. The angular displacement before the subject perceived a change of position was recorded as the threshold to sensation. Finally, the end-range reproduction test was used to examine the subject's ability to reproduce an angle at the end range position of shoulder lateral rotation. The results of the study showed that the involved shoulders demonstrated greater average kinesthetic deficits in all three tests when compared with the uninvolved shoulders.

Smith and Brunolli's[25] findings of kinesthetic deficits after glenohumeral joint dislocation indicate that clinicians should consider shoulder treatment programs that include kinesthetic rehabilitation. Clinicians should also consider a proprioceptive and kinesthetic rehabilitation for patients who have MDI in the conservative approach and also in the post surgical rehabilitation.

Tyler and Hutton[23] have shown that coactivation firing may protect joints from compressive and distractive forces. Hasan and Stuart[23] have stated that centrally mediated stabilization based on the afferent feedback has the advantage that it can be temporarily turned off in the interest of maneuverability. This is important with regard to the shoulder because the shoulder joint requires not only extreme neuromuscular control, but it also must maintain excessive amounts of motion to be dynamically effective. It has been suggested that functional stability of other joints may be enhanced through improved kinesthesia skills and proper muscular coordination. Whether shoulder joint stability can be enhanced through this type of rehabilitative training has not yet been documented.

The objective of a kinesthetic and proprioceptive rehabilitation program is to facilitate the shoulder's performance of a complicated skill without conscious guidance.[26] Wilk and Arrigo[26] utilize a sequence that involves both open and closed kinematic chain conditioning to facilitate proprioceptors to enhance stability and dynamic control. They usually begin with closed kinetic chain exercises because they cause axial loading and compression in the joint, therefore increasing noncontractile stability. This causes contraction of agonist/antagonist muscle groups, thereby creating increased dynamic joint stability.

Closed kinetic chain exercises allow strengthening of the shoulder in a closed pack position which will result in less tensile stress of the capsular ligaments and facilitate cocontraction of dynamic stability structures.[23] Enhancement of static stability in a closed kinetic chain helps to "educate" the proprioceptors to balance the shoulder girdle musculature when functioning dynamically.

The goal of open chain strengthening is to provide proximal control of the scapulothoracic joint and to facilitate a stable base of support for glenohumeral mobility.[23] Proprioceptive neuromuscular facilitation exercises utilize specific skilled sensory input from the clinician to bring about or facilitate a specific activity or movement pattern.[26] A commonly performed pattern is the D2 flexion/extension pattern for the U/E with rhythmic stabilization techniques applied at various points in the ROM. The patient can be challenged at ranges where the shoulder is more unstable in the later stages of rehabilitation. The drills enhance the dynamic stability elements of the glenohumeral joint through isometric control of the humeral head through the glenohumeral musculature.

Various parameters are considered with Wilk and Arrigo's[26] program in terms of progression of the patient; such as, 1) submaximal to maximal effort, 2) slow to faster speeds in execution of the activity patterns, 3) known to unknown patterns, 4) different positions of the shoulder and arm, and 5) stable to increasingly unstable surface areas.

Plyometrics are activities that utilizes the proprioceptors. Plyometrics are characterized by powerful muscle contraction in response to rapid dynamic loading or stretching of the involved muscles.[27] The mechanism by which plyometrics may increase muscular performance centers around neuromuscular coordination. The neurological system may be enhanced to become more automatic. Weighted balls can be used to create a dynamic overpressure into the apprehensive positions. This would require the patient to control the movement in a dynamically challenging exercise. The performance enhancement observed from stretch-shortening exercises appears to occur from neural adaptation, increasing speed of the myotatic reflex, desensitization of the golgi tendon organ, or enhance neuromuscular control and not from morphological changes in the muscle.[27] The benefits seem optimal for a patient with decreased proprioception.

As stated previously, there has been no present data that report increasing shoulder stability after proprioceptive and kinesthetic training. Davies[28] investigated the acute effects of fatigue on shoulder rotator cuff internal and external rotation kinesthesia. Angular reproduction tests were done and then subjects performed isokinetic exercise of shoulder IR/ER until an operational definition of fatigue occurred. Subjects rested for one minute and then the post-tests were performed similar to the pre-test. No significant differences with the acute effects of fatigue on the kinesthesia were noted. Further research in this area would be beneficial to increase the knowledge on the effects of shoulder kinesthesia.

Proprioceptive/kinesthetic rehabilitation seems to have an important place in all types of rehabilitation but may be more important in an unstable joint like the shoulder. Through more research, the effects of these exercises can be better understood and more efficiently used. The use of these exercises gives physical therapists hope for a treatment of multidirectional instability of the shoulder.

CHAPTER VIII

CONCLUSION

Multidirectional instability is a problem that is now recognized by physical therapists. Multidirectional instability is a complex subject, and perhaps this is the reason for the lack of attention to it in the past. This condition remains poorly defined and incompletely understood.

It is important for physical therapists to have a good understanding of anatomy and biomechanics of the glenohumeral joint. Special attention needs to be directed towards the components that contribute to stability of this joint. Proper knowledge of anatomy and biomechanics will also help with the rehabilitation program.

The most important step in treatment of MDI is its initial recognition. Therapists need to be aware of the causes of this problem. As stated before, there are various theories on the cause of MDI, but many believe that there are multiple factors that may lead to this condition. Clinicians must also have an understanding of the symptoms that are involved with this type of instability and not mistake this problem for single plane instability.

The treatment program of multidirectional instability can be either conservative or operative. The inferior capsular shift procedure is performed if the conservative attempt fails. A proprioceptive program may also play an important role in the rehabilitation of a shoulder with multidirectional instability. It is evident that further research is needed in this area.

REFERENCES

1. Thomas SC, Matsen FA. An approach to the repair of glenohumeral ligament avulsion in the management of traumatic anterior glenohumeral instability. J Bone Joint Surg. 1989;71A:506-510.

2. Neer CS. Involuntary Inferior and Multidirectional Instability of the Shoulder: Etiology, Recognition, and Treatment. Instructional Course Lectures. 1985;34:232-238.

3. Wilk KE, Andrews JR. Current concepts in the treatment of shoulder instability. Orthopaedic Physical Therapy Home Study Course, LaCrosse, Wis; July 1993, 1-20.

4. Culham E, Peat M. Functional anatomy of the shoulder complex. J Ortho Sports Phys Ther. 1993;18:342-349.

5. Matsen FA, Harryman DT, Sidles JA. Mechanics of glenohumeral instability. Clinics in Sports Med. 1991;10:783-788.

6. Driscoll SW. Atraumatic instability: pathology and pathogenesis. In: Matsen FA, Fu FH, Hawkins RJ, eds. The Shoulder: A Balance of Mobility and Stability. Vale, Colo: American Academy of Orthopaedic Surgeons Symposium.

7. Lippitt S, Matsen F. Mechanisms of glenohumeral joint stability. Clin Orthop. 1993;293:20-28.

8. Warner JJP, Mecheli LJ, Arslanian LE, Kennedy J, Kennedy R. Patterns of flexibility, laxity, and strength in normal shoulders and shoulders with instability and impingement. Am J Sports med. 1990;18:366-374.

9. Emergy RJH, Mullaji AB. Glenohumeral joint instability in normal adolescents. J Bone Joint Surg. 1991;73-B:406-408.

10. Warner JJP, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Scapulothoracic motion in normal shoulders and shoulders with glenohumeral instability and impingement syndrome. Clin Orthop. 1992;285:191-199.

11. Neer CS, Foster CR. Inferior capsular shift for involuntary inferior and multidirectional instability of the shoulder. J Bone Joint Surg. 1980;62-A:897-902.

12. Altchek DW, Warren RF, Skyhar MJ, Ortiz G. T-plasty modification of the Bankart procedure for multidirectional instability of the anterior and inferior types. J Bone Joint Surg. 1991;73-A:105-112.

13. Foster CR. Multidirectional instability of the shoulder in the athlete. Clinics in Sports Medicine. 1983;2:355-367.

14. Arendt EA. Multidirectional shoulder instability. Orthopedics. 1988;11:113-120.

15. Silliman JF, Hawkins RJ. Classification and physical diagnosis of instability of the shoulder. Clin Orthop. 1993;291:7-19.

16. Cofield RH, Irving JF. Evaluation and classification of shoulder instability. Clin Orthop. 1987;223:32-42.

17. Ozaki J. Glenohumeral movements of the involuntary inferior and multidirectional instability. Clin Orthop. 1989;238:107-111.

18. Jalovaara P, Myllyla V, Paivansalo M. Autotraction stress roentgenography for demonstration anterior and inferior instability of the shoulder joint. Clin Orthop. 1982;284;136-143.

19. Paine RM, Voight M. The role of the scapula. J Orthop Phys Ther. 1993;18:386-391.

20. Brostrom L, Kronberg M, Nemeth G, Oxelback U. The effect of shoulder muscle training in patients with recurrent shoulder dislocations. Scand J Rehab Med. 1992;24:11-15.

21. Burkhead WZ, Rockwood CA. Treatment of instability of the shoulder with an exercise program. J Bone Joint Surg. 1992;74-A:890-896.

22. Lebar RD, Alexander H. Multidirectional shoulder instability clinical results of inferior capsular shift in an active-duty population. Am J Sports Med. 1992;20:193-198.

23. Davies GJ, Dickoff-Hoffman S. Neuromuscular testing and rehabilitation of the shoulder complex. J Orthop Sports Phys Ther. 1993;18(2):449-458.

24. Schutte MJ, Happel LT. Joint innervation in joint injury. Clin Sports Med. 1990;9(2):511-517.

25. Smith R, Brunolli J. Shoulder kinesthesia after glenohumeral dislocation. Phys Ther. 1989;69:106-112.

26. Wilk KE, Arrigo C. Current concepts in the rehabilitation of the athletic shoulder. J Orthop Sports Phys Ther. 1993;18(1):365-37.

27. Wilk KE, Voight ML, Keirns MA, Gambetta V, Andrews JR, Dillman CJ. Stretch-shortening drills for the upper extremities: theory and clinical application. J Orthop Sports Phys Ther. 1993;17(5):225-239.

28. Davies GJ, Lawson K, Jones B. The Acute Effects of Fatigue on Shoulder Rotator Cuff Internal/External Rotation Isokinetic Power and Kinesthesia. Presented at the 1993 American Physical Therapy Association's Annual Conference, Cincinatti, OH, June, 1993 (in press). Phys Ther. 1993.