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Closed Kinetic Chain Exercises of the Shoulder Joint: Biomechanics and Clinical Implications

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CLOSED KINETIC CHAIN EXERCISES OF THE SHOULDER JOINT;
BIOMECHANICS AND CLINICAL IMPLICATIONS

by

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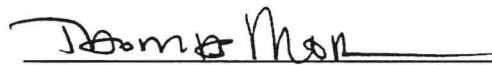University of North Dakota, 1993

An Independent Study

Submitted to the Graduate Faculty of the

Department of Physical Therapy

School of Medicine

University of North Dakota

in partial fulfillment of the requirements

for the degree of

Master of Physical Therapy

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May
1994

This Independent Study, submitted by Todd L. Gentzler in partial fulfillment of the requriements 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.

_____
(Faculty Preceptor)

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(Graduate School Advisor)

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(Chairperson, Physical Therapy)

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PERMISSION

Title   **Closed Kinetic Chain Exercises of the Shoulder Joint; Biomechanics and Clinical Implications**

Department   Physical Therapy

Degree   Master of Physical Therapy

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Signature _Todd J Gentzler_

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ABSTRACT

The shoulder is a complex joint with many structures located in a small area. The purpose of this paper is to first analyze the shoulder joint mechanics and allow for a more complete understanding of this structure. The anatomical features are defined, including muscles, tendons and ligaments, their attachments and innervations. These structures are often predisposed to injury or pathology.

The second part of this paper introduced a new treatment concept: Closed Kinetic Chain Exercises of the shoulder joint. Closed kinetic chain exercises are defined, including the purpose and rationale behind this new treatment. Closed kinetic chain exercises add variability and versatility to shoulder rehabilitation. It is important to understand that not every patient will be able to perform these exercises, as they are relatively aggressive. Using closed kinetic chain exercises in conjunction with established protocols related to shoulder pathology, however, appear to be ideal in permitting the patient or athlete to return to full function.

CHAPTER 1

INTRODUCTION

The shoulder is a complex joint with many structures located in a small area. With its many movements, there are several lesions that can occur either inside or outside the joint capsule. The shoulder region is composed of three synovial joints, the glenohumeral, acromioclavicular and sternoclavicular, and two physiologic joints, the scapulothoracic joint and coracoacromial arch. These joints, along with the ligaments, musculotendinous cuff, and major muscle movers in the area, must work as one entity to produce the various ranges of motion possible in the shoulder joint. Dysfunction in any one of these structures can result in injury.[1,2]

A complete understanding of the anatomy and various pathologies, along with versatile exercises for each patient is essential to establish full function of the shoulder joint. Physical Therapists must be able to implement a wide array of modalities and exercises to complement the normal healing process of the shoulder joint. The purpose of this review is to introduce a new treatment concept termed Closed Kinetic Chain exercises for the shoulder. This form of

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rehabilitation has traditionally been utilized for the lower extremity, but is now incorporated into rehabilitation of the upper extremity as well.

The system is considered to be closed if both ends are connected to an immobile framework, preventing translation in the proximal or distal joint center. Closed kinetic chain exercises are ideal because they cause axial loading and compression in the joint, increasing non-contractile stability. This helps improve joint stability through greater joint congruency and active contraction of the muscles. [9,15] To effectively balance the compression and shear forces, shoulder elevation to a 90 degree (standard push-up) position is optimal.

It is important to understand that closed kinetic chain exercises are not substitutes for standard shoulder protocols. They should be used in conjunction with standard treatments. Closed kinetic chain exercises are considered to be relatively aggressive and should not be used in the extreme early phase of rehabilitation.

With a greater appreciation of closed kinetic chain exercises and its implications in physical therapy, clinicians will be able to utilize this new treatment concept to its fullest, establishing complete function and preventing setbacks in the rehabilitation program.

CHAPTER 2

ANATOMY

Glenohumeral Joint

The glenohumeral joint is a synovial ball-and-socket joint between the humeral head and the glenoid fossa of the scapula. The glenohumeral joint capsule has a volume twice as great as that of the humeral head, allowing for slightly more than one inch of humeral head distraction from the glenoid fossa.[2,18,19] The shallowness of the fossa and the disproportionate size and lack of congruence of the articular surfaces make the joint inherently unstable.[2,18] Stability is primarily dependent on the capsule, ligamentous structures, and the musculotendinous cuff. The ligament restraints can be considered no more than a mere thickening of the joint capsule. With the intrinsic weakness in the joint, it is susceptible to both degenerative changes and derangement.[2,18]

Stability

Some joint stability is provided by a fibrocartilaginous rim, called the glenoid labrum, which surrounds the glenoid fossa. The labrum is lined by synovial membrane internally, is attached to the capsule

externally, and is continuous with the periosteum of the scapular neck.[2,18,49] The labrum helps deepen the glenoid cavity, thus contributing to the stability of the joint.[2,18] In a study on 25 cadavers the average depth of the socket, including the labrum, was 9 mm in a superior-inferior direction and 5 mm in an anterior-posterior direction.[18] The labrum contributes to approximately 50% of the total depth of the socket.[2,10,12,13,18] Boundaries of the labrum including a blending posterior with the tendon of the long head of the biceps brachii and anteriorly with the inferior glenohumeral ligament. This also contributes to the fossa's total articular surface.

The capsule is attached medially to the margin of th eglenoid fossa and laterally to the circumference of the anatomical neck of the humerus. It is think and large which allows for the humeral head to distract from the glenoid. The capsule contributes little to the stability of the joint. The capsule is strengthened by ligaments and the insertion of the rotator cuff tendons.

The coracohumeral ligament originates from the base and lateral border of the coracoid process and passes laterally to insert on the humeral tuberosities.[2,34,35,49] This ligament becomes taut during external rotation of the humerous between 0 and 60 degrees of arm elevation. This ligament provides support for the dependent arm by resisting the downward pull of gravity on the humeral head.[18]

The three glenohumeral ligaments--superior, middle and inferior--are considered to be thickened areas of the anterior, inferior and posterior joint capsule.[2,14,49] The inferior glenohumeral ligament (IGHL) is the most prominent structure of the anterior capsule.[49] It attaches to the anterior, inferior and posterior margins of the labrum medially and to the anatomical surgical neck of the humerous laterally. It also makes up an axillary pouch and anterior-superior band. A posterior-superior band has also been noted. The axillary pouch runds from the inferior one-third of the humeral head to the inferior two-thirds of the anterior glenoid.[49] The posterior-superior band and axillary pouch support the joint in the upper ranges of elevation and prevent anterior subluxation and dislocation in this part of the range. The posterior band also acts as a stabilizer against posterior subluxation of the head udring movements of abduction and internal rotation. both bands limit inferior translation of the humeral head during arm elevation. The anterior band becomes taut with internal rotation.[2,14,18,49] The inferior glenohumeral ligament attachment to the anterior glenoid and labrum is the easiest place to identify this ligament. It is located in a plane between the glenoid and subscapularis tendon.[49]

The middle glenohumeral ligament (MGHL) runs from the anterior humeral neck, medial to the lesser tuberosity, to the upper half of the glenoid and scapular neck.[49] It lies

inferior to and is intimately attached to the subscapularis tendon. This tendon and the MGHL are important anterior stabilizers of the glenohumeral joint and function to limit lateral rotation of the humerus between 0 and 90 degrees of elevation.[8] The MGHL is most easily identified as it passes the superior border of the intraarticular portion of the subscapularis tendon. Its attachment to the glenoid is actually medial to the insertion to the IGHL. The MGHL will vary considerably in width and thickness.

The superior glenohumeral ligament (SGHL) runs from the humeral neck on the medial ridkge of the intertubercular groove to the anterosuperior glenoid labrum.[8,49] It also inserts partially onto the base of the coracoid process. The SGHL is normally much smaller than the other two ligaments, but a wide variation in size is also noted.[49] The primary function of the SGHL is prevention of inferior displacement of the humeral head in the dependent arm while in the adducted position.[8]

Mobility

Stability of the glenohumeral joint is also dependent on muscle control provided by the musculotendinous cuff.[6-8,10] The rotator cuff tendons include the subscapularis tendon anteriorly, the supraspinatus tendon superiorly, and the infraspinatus and teres minor tendons posteriorly. The four tendons blend with each other and with the capsule to attach to the lesser tuberosity, greater tuberosity and the

transverse humeral ligament.[10] The origin of the suderaspinatus is the medial two-thirds of the supraspinous fossa, it inserts on the sueprior facet of the greater tubercle of the humerus. The infraspinatus originates on the medial two-thirds ofthe infraspinuous fossa, and has an insertion on the middle facet of the greater tubercle of the humerus. The teres minor originates on the proximal two-thirds of the lateral border of the scapula. It inserts in the inferior facet of the greater tubercle of the humerus. The subscapularis originates on the medial two-thirds of the subscapular fossa and inserts on the lesser tubercle of the humerus.[26,34,36,48]

The rotator cuff muscles are considered to be the fine tuners of the glenohumeral joint and shoulder girdle, whereas the latissimus dorsi, deltoid, trapezius, serratus anterior, levator scapulae, rhomboids and pectoral muscles are the powerful rotators and abductors. All of the rotator cuff muscles contribute to some degree of glenohumeral abduction; but the supraspinatus and deltoid are the primary abductors.[6,8,10,19]

The supraspinatus also functions to compress the glenohumeral joint and acts to glide the huemral head vertically.[10] The infraspinatus, teres minor and subscapularis muscles also aid in abduction. The rotator cuff as a whole acts as a force couple to depress the head of the humerus and downwardly move the humerus along the

fossa, while externally rotating the humerus and abducting the shoulder.[10,34,35,37] The deltoid will be the primary abductor but without full use of the rotator cuff, the head of the humerus will be "jammed up" into the suprahumeral arch. The infraspinatus, considered to be the next most active rotator cuff muscle behind the supraspinatus, acts as a primary external rotator. The teres minor also contributes to external rotation of the glenohumeral joint. The subscapularis is named as the primary internal rotator of the cuff. The supraspinatus and infraspinatus muscles are innervated by the suprascapular nerve (C5, 6), the teres minor in innervated by the axillary nerve (C5, 6), and the subscapularis is innervated by the upper and lower subscapular nerves (C5, 6).[48]

Acromioclavicular Joint

The acromioclavicular joint consists of two major ligaments and a joint meniscus, which is sometimes absent. It is a plane synovial joint between a small convex fact on the lateral end of the clavicle and a small concave facet on the acromion of the scapula. Both surfaces are covered with fibrocartilage.[2] The medial end of the clavicle rises just above the acromion, and joint surfaces are angled in an inferior, medial direction, resulting in a tendency for the acromion to be driven under the clavicle during times of excessive force.[18]

Joint integrity is maintained by surrounding ligaments rather than a bony configuration.[2,18] The primary functions of the acromioclavicular joint are maintaining the relationship between the clavicle and the scapula in the early stages of upper extremity elevation and allowing the scapula additional range of rotation on the thorax in the later stages of limb elevation.[2]

The superior and inferior acromioclavicular ligaments maintain integrity of the acromioclavicular joint, while the coracoclavicular ligament is the primary joint stabilizer. The coraclavicular ligament is divided into two sections, the lateral portion, or the trapezoid and a medial portion, or the conoid.[2,18] A fall on an outstretched hand tends to translate the scapula medially, and the small acromioclavicular joint alone cannot prevent scapular motion without resulting in joint dislocation. As the scapula and coracoid process move medially, the trapezoid ligament tightens, transferring the force of impact to the clavicle and, ultimately, to the strong sternoclavicular joint. The trapezoid ligament runs from the anterior part of the coracoid process and passes upward and laterally to attach to the inferior surface of the clavicle. The conoid portion lies posterior and medial to the trapezoid ligament and runs upward and slightly backward from the upper surface of the coracoid process to attach to the undersurface of the clavicle.[2] The coracoclavicular ligament is responsible fo

producing longitudinal rotation of the clavicle, which is necessary for full range of motion in elevation of the upper extremity.

Sternoclavicular Joint

The sternoclavicular joint consists of a joint capsule, three major ligaments, and a disc. It is a plane synovial joint in which the bulbous medial end of the clavicle articulates with the cartilage of the first rib and sternum. Fibrocartilage covers both the artiuclar surfaces.[18] The medial end of the clavicle is concave anteroposteriorly and convex cepthalocaudally. An articular disc attaches to a nonarticular portion of th emedial clavicle superiorly, and to the sternum and first rib below, dividing the joint cavity into two compartments. The joint is stabilized anteriorly and posteriorly by the sternoclavicular ligament, which checks anterior-posterior (A-P) movements of the clavicular head.[18] The costoclavicular ligament functions as the principal stabilizer of this joint, acting as an axis for shoulder elevation/depression and protraction/ retraction, serving to stabilize the clavicle against the pull of the sternocleidomastoid muscle and checking proximal elevation of the clavicle.[2] Althought the articular surfaces of the joint are saddle-shaped, it still functions as a ball-and-socket joint with three degrees of freedom. Elevation and depression of the clavicle occur between the medial end of the clavicle and the disc. Protraction and

retraction occur between the disc and the sternum. In addition, the clavicle can rotate aobut its longitudinal axis.[18]

Scapulothoracic Joint

The scapulothoracic muscles work together as synergists acting as a force couple.[2,18,50] A force couple has been described as divergent pulls of force creating pure rotation. In addition, a force couple has been defined as the effect of equal, parallel forces acting in opposite directions that leads to rotatory movement.[10] For normal upper extremity movement, the scapula provides a stable base from which glenohumeral mobility occurs. Stability at the scapulothoracic joint is dependent upon the surrounding musculature.[22]

Serratus Anterior

The serratus is an important scapular stabilizer muscle, taking an origin from the first eight ribs and coursing along the rib cage, attaching to the vertebral border of the scapula. The upper portion attaches to the medial border of the scapula while the lower portion finds an insertion into the inferior angle of the scapula. Because of its multiple attachment sites, the primary role of the serratus is to stabilize the scapula during elevation and to pull the scapula forward around the rib cage. Scapular abduction or protraction is the term used when the scapula moves in an anterior direction on the thoracic cage.

Protraction is involved with activities like pushing or punching. Innervation of the serratus anterior is provided by the long thoracic nerve (C5, 6, 7).[10,22,48]

Rhomboids

The function of the rhomboid major and minor is to stabilize the medial border of the scapula. The rhomboid minor originates on the spinous process of the C7 and T1 vertebrae and inserts on the medial border of the scapula, near the spine of the scapula. The rhomboid major originates on the spinous process of T2 through T5 and inserts just below the rhomboid minor on the medial border of the scapula.

The rhomboids are most active during scapular adduction, or retraction, which is defined as backward rotation of the scapula towards the vertebral column. If rhomboid weakness is present, the scapula isn't able to fully retract the scapula, which is essential for throwing motions as well as swimming strokes. Failure of the scapula to fully retract leads to increased stress on the anterior structures of the shoulder.[22] Strengthening of this muscle is important when rehabilitating patients diagnosed with anterior instability. The innervation of the rhomboids is by the dorsal scapular nerve (C5 and 6).

Trapezius

The trapezius muscle is divided into the upper, middle and lower portions. The origins of these fibers are the

superior nuchal line, external occipital protruberance, ligamentum nuchae and T1-T12 spinous processes. Insertions extend into the distal third of the scapula, acromion process and spine of the scapula. The upper trapezius elevates and upwardly rotates the scapula, the middle trapezius retracts the scapula, and the lower fibers upwardly rotates and depresses the scapula. Innervation of the trapezius is solely from the spinal accessory nerve, XI.

Levator Scapula

The levator scapula originates on the transverse process of C1 through C4. It inserts along the medial border, superior angle near the spine of the scapula. The levaotr scapula elevates and downwardly rotates the scapula. Innervation is provided by the cervical plexus with frequent contribution by the dorsal scapular nerve, C5 and C6.[22]

Pectoralis Minor and Major

The pectoralis minor is an anterior muscle that originates from the second through sixth ribs and inserts into the medial aspect of the coracoid process on the scapula. Innervation of the pectoralis minor is th emedial pectoral nerve, C8-T1. The pectoralis major has two heads, the clavicular head and sternocostal head. The clavicular head originates on the medial third of the clavicle, while the sternocostal head takes an origin on the sternum and upper six costal cartilages. Innervation for the clavicular head is the lateral pectoral nerve, C5, 6, 7. For the

sternocostal head it is the medial pectoral nerve, C8-T1.
The pectoral muscles act together to perform abduction,
depression, downward rotation and upward tilt of the
scapula. They also play a role in huemral flexion,
extension, adduction and internal rotation of the humerus.[48]

Latissmus Dorsi

The fibers of the latissmus dorsi originate on the
spinous process of T6-T12, posterior layer of the
thoracolumbar fascia, inferior angle of the scapula, and
posterior crest of the ilium. It inserts on teh floor of
the bicipital groove. Innervation of the latissmus dorsi is
the thoracodorsal nerve (C6, 7, 8). It primarily functions
to extend, adduct and medially rotate the humerus; it also
serves as a downward rotator of the scapula.[48]

To summarize the duties of the scapulothoracic joint,
the upper and lower fibers of the serratus anterior and the
trapezius form on eof the force couples acting on this
joint, causing upward rotation during elevation of the
humerus. This force couple also serves as a stabilizing
synergist for the deltoid muscle, which acts on the
glenohumeral joint. Upward rotation of the scapula with the
force couple maintains the proper length-tension ratio for
the deltoid.[7,10,11,18,22]

Lower segments of the pectoralis major, pectoralis
minor, levator scapula, rhomboideus major and minor and
latissmus dorsi form a synergistic muscle group that

downwardly rotates the scapula. Fibers of the pectoralis major and minor, latissmus dorsi and lower trapezius also combine to depress the scapula.[48]

Coracoacromial Arch

The final feature of the shoulder is the coracoacromial arch, or subarcomial space. It is also considered a physiologic joint. It provides the protection against direct trauma to the subacromial structures and prevents dislocation of the humeral head superiorly. Its boundaries include the acromium process superiorly, coracoid process anteromedially, acromioclavicular joint superiorly and the rotator cuff and greater tuberosity of the humeral head inferiorly. The arch itself is formed by the coracoid and acromion process of the scapula and coracoacromial ligament that unites them. The base of this triangular ligament attaches to the lateral aspect of the coracoid process and its apex attaches to the superior aspect of the acromion.[18] The subacromial space between the coracoacromial arch and the huemral head contains the rotator cuff tendons and subacromial bursa.

A bursa is defined as a sac or saclike cavity filled with a viscid fluid and situated in places in the tissues where friction would otherwise develop.[51] The bursa separates the acromial arch and the deltoid muscle superiorly from the rotator cuff and biceps tendon below. This allows for smooth gliding between these structures

decreasing the amount of friction as they pass under the arch.

The distance between the inferior surface of the acromion and the huemral head, measure radiographically, averages 9-10 mm in normal shoulders.[18] When there is a decrease in this apce, there is an association of rotator cuff tendon and long head of the biceps tendon tears.[18,37] Compression will occur most often under the anterior one-third of the acromion process, the coracoclavicular ligament, or the acromio-clavicular (AC) joint. Any abnormality decreasing the volume of the subacromial space can lead to impingement of its contents.[16,32] Reduction of this space can be caused by osteophyte formation on the medial surface of the acromion or lateral clavical, or inflammation of the rotator cuff tendons.[19,41,47] A high incidence of rotator cuff tears secondary to impingement can slo be related to the shape and slope of the acromion process. In a cadaver study, Bigliani[19] grouped the acromion by the shape of the inferior surface as: Type I (19%)-flat, Type II (42%)-curved/ concave, and Type III (39%)-hooked anteroinferiorly.[19] Tests showed that a majority of the tears in rotator cuffs are caused by Type III acromion.[1,19,42] A decreased blood supply of the supraspinatus tendon near its insertion may predispose this region to degenerative changes as well.[14,50]

CHAPTER 3

SHOULDER PATHOLOGY

IMPINGEMENT

The term "impingement syndrome" was first described by Neer in 1972.[2] Commonly seen in repetitive overhead athletes (swimmers, baseball pitchers, quarterbacks, etc.), he emphasized that the supraspinatus insertion into the greater tubercle and the bicipital groove lies anterior to the coracoacromial arch with the shoulder in neutral position. When the shoulder is forwardly flexed, these structures must pass beneath the coracoacromial arch, causing greater opportunity for impingement.[2,27,32] The rotator cuff in relation to the coracoacromial arch is only separated by the subdeltoid and subacromial bursa.

The abnormal processes contributing to increased friction, impingement and increased wear of the cuff tendons are factors such as inadequate shape of the coracoacromial arch, allowing unsafe passage of the cuff tendons, abnormal inferior surface of the acromioclavicular joint, abnormal subacromial bursa and improper function of the humeral head depressor mechanisms including the rotator cuff and biceps tendon. Other things such as decreased capsular laxity,

improper function of the scapulothoracic joint and rough
gliding of the cuff tendons add to impingement.[10,41-43,46]

Two types of impingement have been identified, primary
and secondary. Primary impingement may result from
subacromial crowding, posterior capsular tightness, or
excessive superior migration of the humeral head due to
weakness of the humeral head depressors.[10] Neer describes
primary impingement as impingement of the rotator cuff
beneath the arch.[10] He observed that shoulder flexion with
internal rotation contributes to the critical avascular zone
of the rotator cuff under the coracoacromial arch. The test
for impingement is to flex the shoulder to 90 degrees and
forcibly internally rotate. A positive test produces pain.[1]
Bony spur formation was noted on the undersurface of the
acromion, was secondary to repeated impingement between the
coracoacromial ligament and humeral head.

Secondary impingement is described as a decrease in the
subacromial space due to instability of the glenohumeral
joint (weakness of the rotator cuff mechanism) or scapular
instability (weakness of the scapulothoracic muscles
resulting in improper scapulohumeral rhythm). A positive
apprehension and relocation sign indicates anterior
glenohumeral instability, as increased lateral scapular
slide gliding indicates scapulothoracic instability.[10] The
end result of both syndromes are rotator cuff tendon
inflammation and potential rupture. Similar signs and

symptoms may be present in both cases but the mechanism of injury is different. Neer classified the three progressive stages of impingement (Table 1).[52]

Table 1. Progressive Stages of Impingement

Stage I: Edema and Hemmorrhage-
* AA-Generally under 25 years of age
* DD-Subluxation, acromioclavicular arthritis
* SX-Dull ache following activity, night pain
* PP-Reversible
* TX-Conservative

Stage II: Fibrosis and Tendonitis-
* AA-Generally 25-40
* DD-Frozen shoulder, calcium deposits
* SX-Pain with overhead activity, Stage I symptoms increase
* PP-Recurrent pain with activity, not reversible
* TX-Bursectomy, coracoacromial ligament division

Stage III: Bone Spurs and Tendon Rupture-
* AA-Generally over 40
* DD-Cervical radiculitis, neoplasm
* SX-Increased sharp pain, weakness with activity, loss of
    active/passive motion
* PP-Progressive disability, partial/complete rotator cuff
    tear
* TX-Anterior acromioplasty, rotator cuff repair

AA = Age
DD = Differential Diagnosis
SX = Symptom
PP = Prognosis
TX = Treatment

INSTABILITY

The shoulder is one of th emost flexible and immobile joints in the body.[30] The arthrokinematics of the glenohumeral joint predisposes it to instability. The glenoid cavity is small and shallow, the fossa faces forward and laterally, and the external rotators have an advantage over the internal rotators. All of these combine to make the shoulder more susceptible to dislocations in the forward, medial and inferior directions.

The shoulder is the most frequently dislocated joint in the body.[30] Studies show that dislocations caused by twisting or forceful abduction have a higher recurrence rate than those caused by just a direct blow to the shoulder, anterior or posterior.[27,30-32,34] This is primarily caused by detachment of the labrum, known as a Bankart Lesion, which is also associated with recurrent dislocations.

Anterior Instability

Most often, anterior dislocations occur with the arm abducted and externally rotated to 90 degrees. This can occur while attempting to tackle in football, or any abnormal forces to the shoulder while in that unstable position. An example would be a quarterback getting hit while attempting to pass the football. The dislocated shoulder is characterized by a flattened deltoid contour, inability to move the arm, and severe pain. With this

injury, the head of the humerus is forced out past the labrum and upward to rest under the coracoacromial process.

Insidious onset of anterior instability may be repeated stress in the anterior capsule during activities like throwing--resulting in capsule attenuation and ultimately anterior instability, causing asynchronous firing of the scapular rotators and cuff muscles.[2,36,39] It has also been postulated that weakness of the posterior cuff muscles and/or hypertrophy of the itnernal rotators may result in excessive anterior displacement of the humeral head in the early acceleration phase of throwing.[24,25] Stresses placed on the posterior cuff and minor trauma causes what is called subluxation, or partial dislocation that "goes back in by itself." Subluxation in shoulders is often seen in conjunction with dislocations. Anterior subluxation of the glenohumeral joint may develop without a history of trauma and is most commonly seen in throwers.

Posterior Instability

Posterior dislocation or subluxation is not as common as anterior subluxation, but it does occur. Posterior subluxation often results from overuse, such as a traumatic episode occurring while pitching a baseball. This stretches out the posterior capsule. Another example would be a posterior directed force with the arm adducted and internally rotated.[2,23,24] These findings are frequently missed because they aren't as dramatic as with an anterior

dislocation. Indications of a posterior dislocation are characterized by the inability to rotate the arm externally, inability to fully supinate the forearm while it is flexed, and, possibly, glenohumeral subluxation with horizontal adduction.[23]

Multidirectional Instability

Individuals who are multidirectionally unstable pose a difficult problem, not only to the clinician, but also to the surgeon. "These individuals usually have a lax capsule in all directions and typically have generalized ligament laxity."[2,21,31] Some surgeons elect to repair the most prominent instability under anesthesia and carry out the other procedures at a later date.[2]

For treatment with patients with instability, conservative care should be implemented first, concentrating on the affect unstable region. Treatment should focus on the avoidance of the subluxed position. Aggressive flexibility to the shoulder is usually contraindicated because of the attenuated tissues.[2,38] Successful rehabilitation protocols emphasize wand exercises, pulleys, PNF diagonals, plyometrics and Closed Kinetic Chain Exercises. (See Table 2 for shoulder stabilizing structures.)[18]

Table 2. Shoulder Stabilizers

Factors Limiting Anterior Translation

STRUCTURE	FUNCTION
-Coracohumeral ligament and SGHL	-Limits ER between 0 and 60 degrees
-Subscapularis and MGHL	-Stabilizes between 0 and 90 degrees elevation
-IGHL (anterior band)	-Stabilizes above 90 degrees of elevation
-Infraspinatus and teres minor	-Prevents anterior translation of humeral head in abducted and IR position

Factors Limiting Posterior Translation

STRUCTURE	FUNCTION
-Infraspinatus and teres minor	-Static stabilizers in all abduction positions
-Subscapularis	-Prevents posterior translation of humeral head
-Anterior superior capsule	-If disrupted, posterior dislocation occurs
-Retrotilt of glenoid fossa	-If excessive, posterior subluxation occurs

Factors Limited Inferior Translation

STRUCTURE	FUNCTION
-Superior joint capsule and SGHL	-Limit inferior subluxation in dependent position
-Negative intra-articular pressure	-Limits inferior displacement of adducted humerus
-IGHL	-Stabilizes above 45 degrees of abduction

Surgical management of those people who do not respond to conservative care is controversial, and includes techniques such as the Bankart, Bristow and Hill-Sachs repairs.

ACROMIOCLAVICULAR SEPARATION

Injuries involving the acromioclavicular joint (A-C Joint) can be complicated because of activities requiring repetitive overhand use. They can be from a direct trauma, in which the athlete falls on the tip of the shoulder and depresses the acromion process inferiorly, or from a fall on an outstretched arm, in which forces are transmitted superiorly through the acromion process.

The level or extent of the A-C separation is dependent upon whether the acromioclavicular ligaments are traumatized or the main stabilizing coracoacromial ligaments are damaged.[2] A-C injuries have been classified by Nevaiser[2] according to the amount of displacement:[2,47]

Grade I Sprains-incomplete injury to supporting ligaments without any degree of displacement.

Grade II Injury-rupture of the acromioclavicular ligament with sprain of the coracoclavicular ligaments.

Grade III-no contact between the articular surfaces of the clavicle and acromion because of rupture of both acromioclavicular and coracoclavicular ligaments.

Treatment of these injuries is most commonly based on a conservative approach, and often treated nonoperatively.

While there are a number of surgical procedures available, it is dependent upon the extent of damage.

OVERUSE INJURIES

Overuse injuries are usually defined as accumulative microtrauma secondary to repetitive submaximal stress that eventually overloads the tissues and results in an inflammatory process.[46] Degenerative changes and macrotrauma are soon to follow. Overuse injuries are related to overhead movements like throwing, swimming, and lifting. Heavy load movements like lifting and throwing errors are almost always associated with too high of an intensity, too long of a durection and the frequency being too often.

Overuse is related to compression forces causing irritation, inflammation, and degeneration. The impingement syndrome previously mentioned is the most common abnormal compression at the shoulder. Traction forces have also been suggested as a cause as well as a solution to overuse problems. Structures aggravated by overuse are the subacromial bursa, superior glenohumeral capsule, subdeltoid bursa, connective tissue, supraspinatus tendon and the biceps tendon. Treatment of choice for overuse injuries is the conservative choice, with emphasis on PNF diagonals, plyometrics and Closed Kinetic Chain exercises as tolerated by the patient.

ADHESIVE CAPSULITIS

Often called frozen shoulder, this pathology is usually insidious with a gradual increase in pain and loss of range of motion (especially ER, IR, and ABD) and function. The 40-60 age group is the most common patient population affected.[41] The underlying cause is often overuse (rotator cuff, tendonities, bursitis, impingement). This creates a painful, inflamed shoulder leading to fibrosis/scarring and thickening with contracture development of the glenohumerual capsule.[41]

"True" frozen shoulder presents with a capsular pattern which is external rotation least limited, followed by abduction and internal rotation most limited.[41] Capsular end feels are also noted. "Neurological" frozen shoulders are limitations in range of motion with noncapsular pattern seen.[41] There is guarding and splinting and an empty end feel.

Conservative treatment consists of anti-inflammatory and pain medications and modalities to control inflammation and pain. Aggressive independent range of motion home exercise program, mobilization and passive range of motion by a physical therapist and strengthening of muscles includes in a standard rotator cuff protocol and closed kinetic chain exercises are then used. Surgical manipulation is the last resort.

ROTATOR CUFF DISEASE

Much of the initial recognition of rotator cuff disease began in 1934 with Codman's treatise.[14] He indentified a critical zone near the insertion of the supraspinatus tendon where most tears seemed to occur. Several researchers have attempted to explain the initiation of tendon disease in this zone.[14,50]

It was noted that tendinous tissue was considerably less vascularized in adults than in adolescents. Codman noted that the region one-half proximal to the supraspinatus insertion in the greater tuberosity tended to be anemic with a gross appearance of infarction.[14] There is noted development of an area of avascularity in the tendon with the arm in the dependent adducted position near th einsertion of the supraspinatus. As the arm is abducted, this area of avascularity disappears. Because the supraspinatus helps suspend the humerus while the arm is adducted, the phenomenon of "wringout out" of the blood vessels in the tendon further diminishes the blood supply to the tendon. With the arm in the abducted position, the tendon appears to be revascularized.[14]

Repetitive damage to the rotator cuff occurs by compression between the acromion and humeral head. This was identified long after the critical zone was defined.[14] The space is a rigidly defined compartment containing segments of the rotator cuff that is overlaid by the subacromial

bursa and biceps tendon. An outlet is formed at the upper border of the coracoacromial arch. It consists of the coracoid, coracoacromial ligament and acromion. The humeral head lies below it. Attempts to relieve the compression consisted of complete and lateral acromionectomy initially. This resulted in significant weakening of the deltoid and sinus and scar formation. Neer[14] had noted progression of tendon disease, beginning with inflammation and ending with rotator cuff tears. This is the same classification of progression used with his stages of impingement.

These factors all contribute to rotator cuff disease and eventual tears. Conservative approaches are optimal if tears are partial; otherwise, a surgical approach is used.

CHAPTER 4

SHOULDER REHABILITATION

The rehabilitation approach with shoulder pathology deals with different approaches, protocols and theories. I will focus on a basic protocol to be used as a guideline when treating shoulder pathology. This will give an idea of exercises used, and the progressive stages. It is important not to use a "cook-book" approach and to individualize treatment, adding variability with each patient. Functional activities are also a very important addition to treatment.

Conservative Rotator Cuff Protocol

Indications for this protocol are pathologies such as rotator cuff tendonitis, biceps tendonitis, bursitis, impingement, small rotator cuff tears, partial thickness tears and adhesive capsulitis.

Program Guidelines[3]

A. Phase I (0-6 weeks)-preserve healing/repair

B. Phase II (6-12 weeks)-restore ROM/strength

C. Phase III (3-6 months)-restore function, return to full activity

Phase I (0-6 weeks)[4,5]

-Sling (6-8 weeks)-Use of ABD pillow more common now

30

-Codman's exercises

-Gentle pain free isometrics, avoiding stressed
 abduction at first, and external rotation

-Passive ROM, mobilization, and supine wand
 exercises

-Elbow ROM, bicep/tricep co-contractions

-Grip strengthening exercises

Phase II (6-12 weeks)[4,5]

The goal is to start increasing strength and
quality of motion. The sequence may be
overlapping but it is important to remember that
no two people will progress at the same time.

-ROM within functional limits by 8-10 weeks

-Multiple-assistive ROM - Wand, pulley, upper body
 ergometer (UBE)

-Theraband exercises (ER, IR, ABD, flex, extend,
 add; all without pain)

-Begin isotonic exercises without weight and
 progress to light weight or theraband as
 tolerated.

    1)   flexion
    2)   abduction
    3)   extension
    4)   empty can
    5)   horizontal abduction
    6)   horizontal adduction
    7)   internal rotation
    8)   external rotation with towel roll
    9)   shoulder shrugs
   10)   serratus push
   11)   bicep curl

Phase III (3-6 months)[4,5]

This stage begins with more aggressive

strengthening exercises and the implementation of

closed kinetic chain exercises. Figures 1-5

demonstrate examples of these exercises.

The following aggressive shoulder protocol using the cable column and free weights complements the use of closed kinetic chain exercises. They are as follows:

Days 1 and 3

-Pull Downs, reverse grip on "pull down" machine

-Pull Downs, front grip on "pull down" machine

-Bent over rowing with free weights

-Shoulder shrugs with free weights

-Empty can with free weights

-Forward flexion with free weights

-PNF diagonals on the cable column

-Internal/external rotation with theraband

-Abduction in short range with theraband

Days 2 and 4

-Triceps with "pull down" machine, front and
 reverse grip

-Biceps with "pull down" machine, front grip and
 also with free weights

-Full extension on "pull down" machine for
 latissmus dorsi

-Upright rows on cable column or free weights

-BTE or Cybex internal/external rotation

These exercises are to be used in conjunction with the closed kinetic chain exercises.

Figure 1

    <u>The push up with a plus</u> - (A,B) The plus phase of this exercise (B) helps to facilitate scapular protraction and enhances serratus anterior strengthening while keeping the shoulder joint in a 90 degree elevated position.
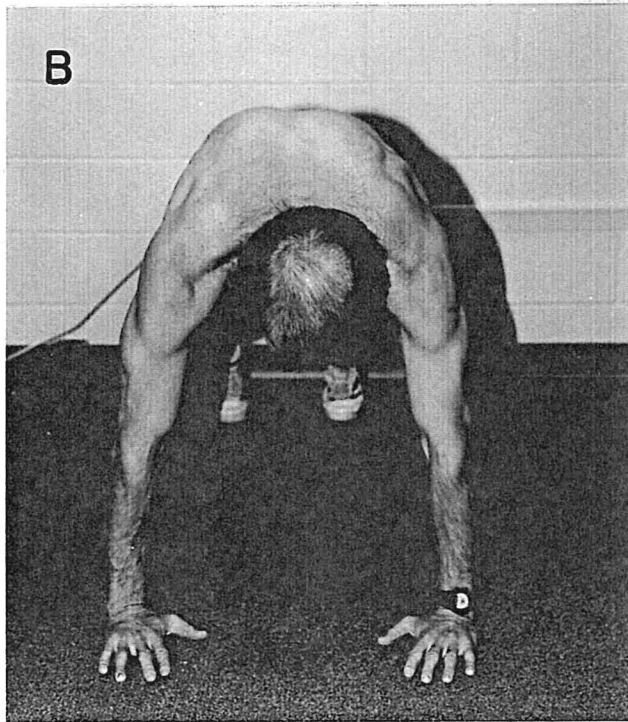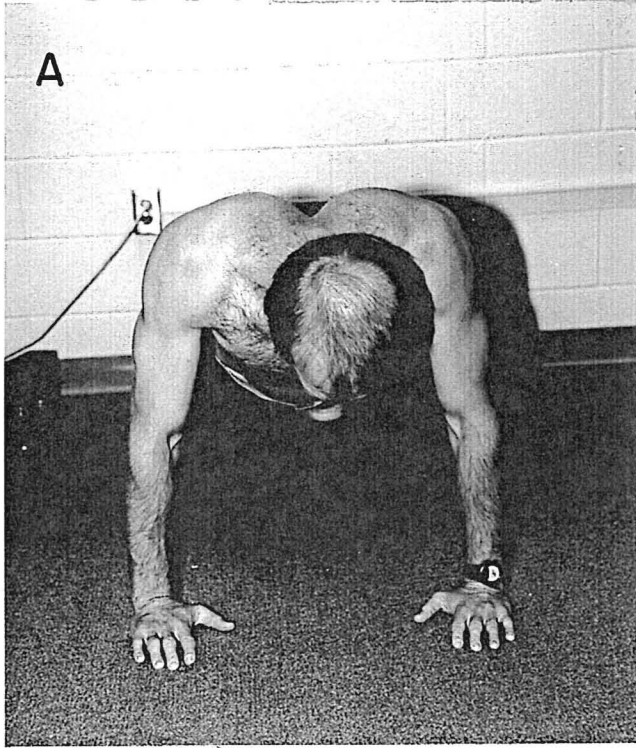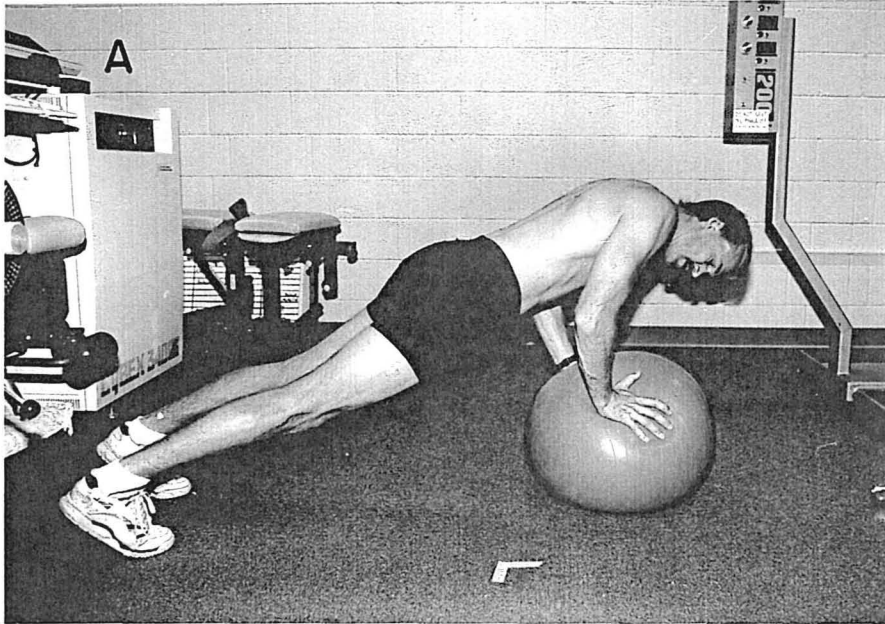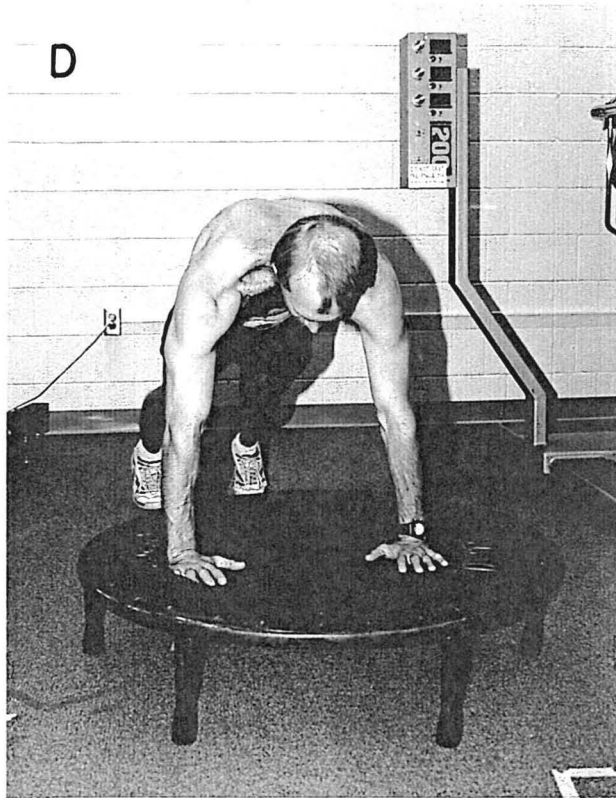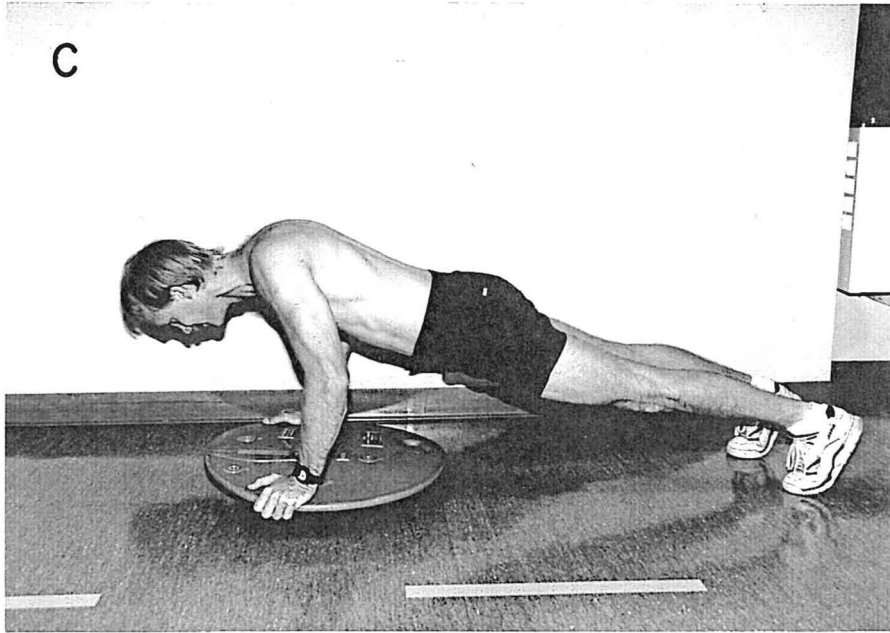
Figure 2
The push up with a plus can be performed on the floor (A,B), wall (C), or any other immobile surface, while a sitting push up (D) facilitates strengthening of the scapular depressors.
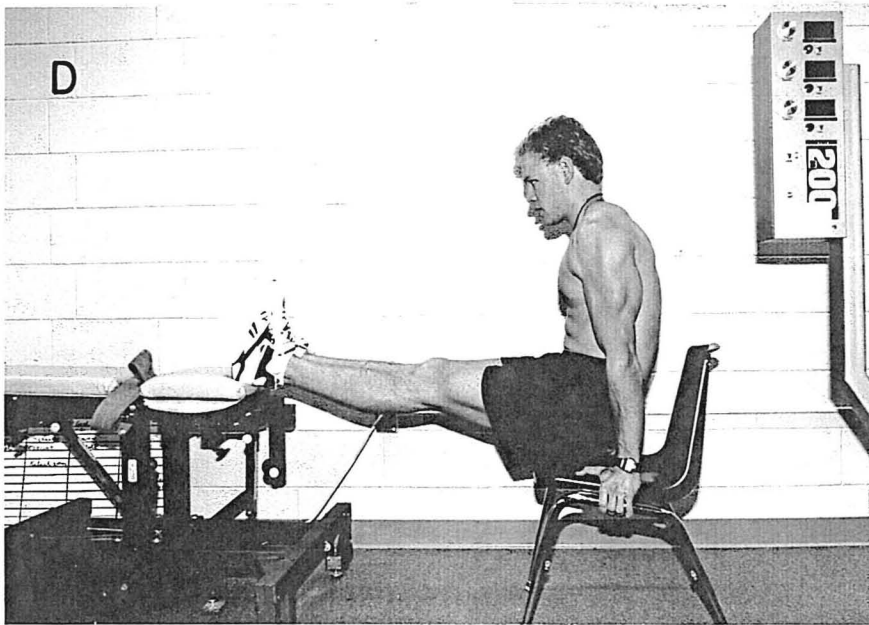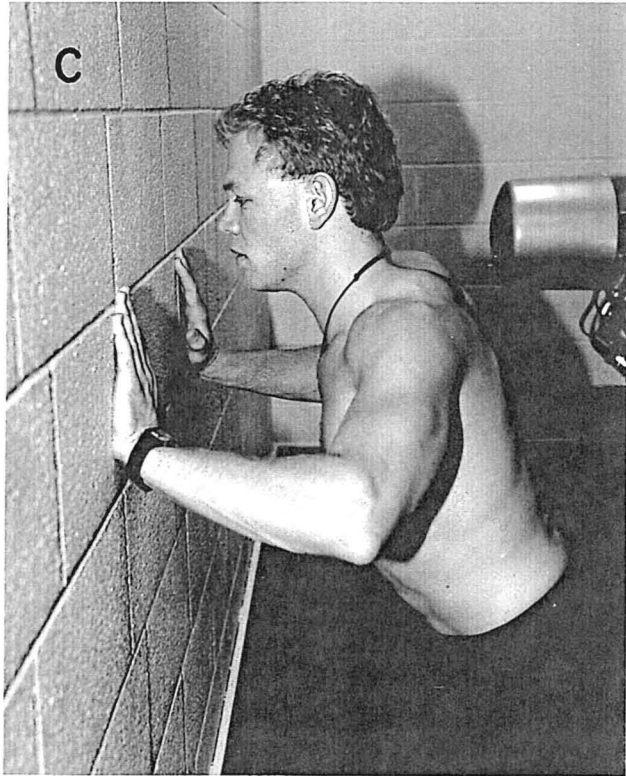
Figure 3

    <u>Push ups on uneven objects</u> - This stresses the scapular
stabilizers and enhances joint loading, proprioception,
muscle and ligament balance. This can be done on objects
such as a physioball (A), balance board (B), BAPS board (C),
and trampoline (D).

C

D
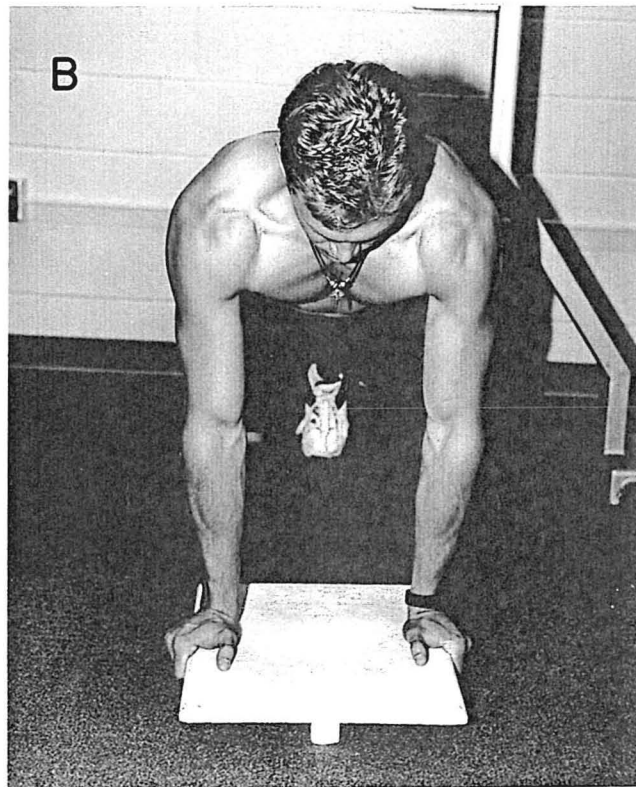
Figure 4

   <u>Tripod balance and wall push ups</u> - (A,B) This helps to
facilitate co-contraction of the antagonists and anterior
strengthening. Weight shifts can be employed early in the
rehabilitation program to enhance dynamic stability of the
shoulder and may be enhanced with manual resistance given to
the patient by the therapist (B).
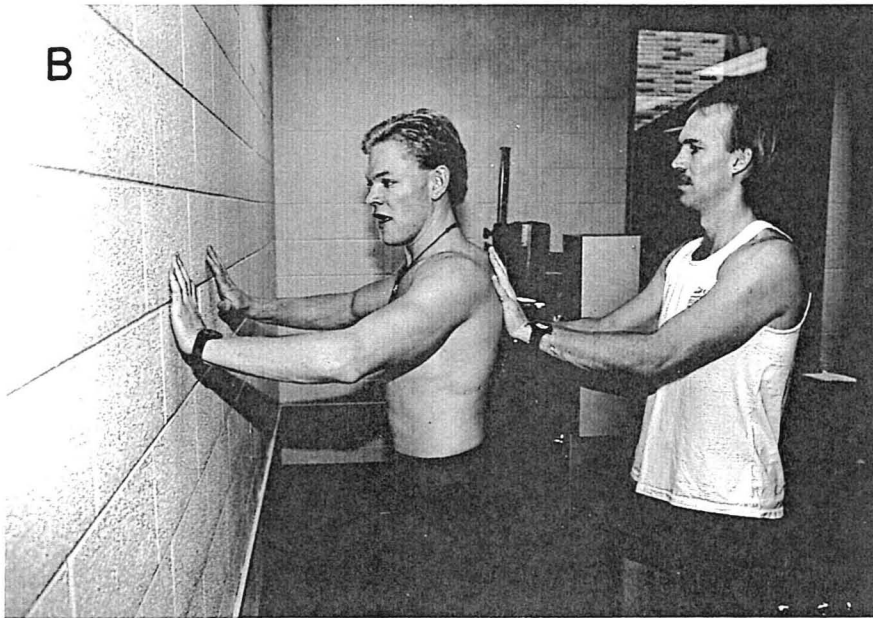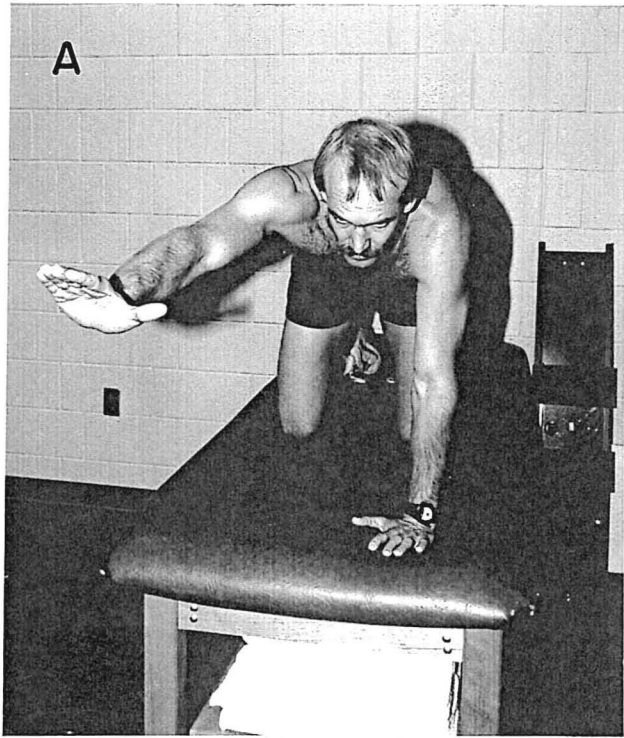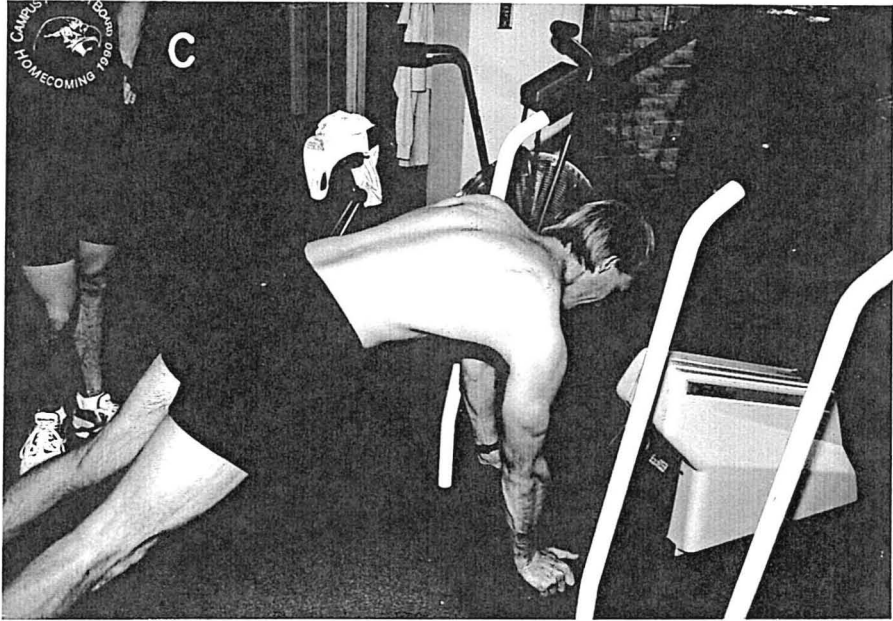
Figure 5
    Manual resistance given to patient by the therapist in tripod (C) and quadruped (D) balancing.
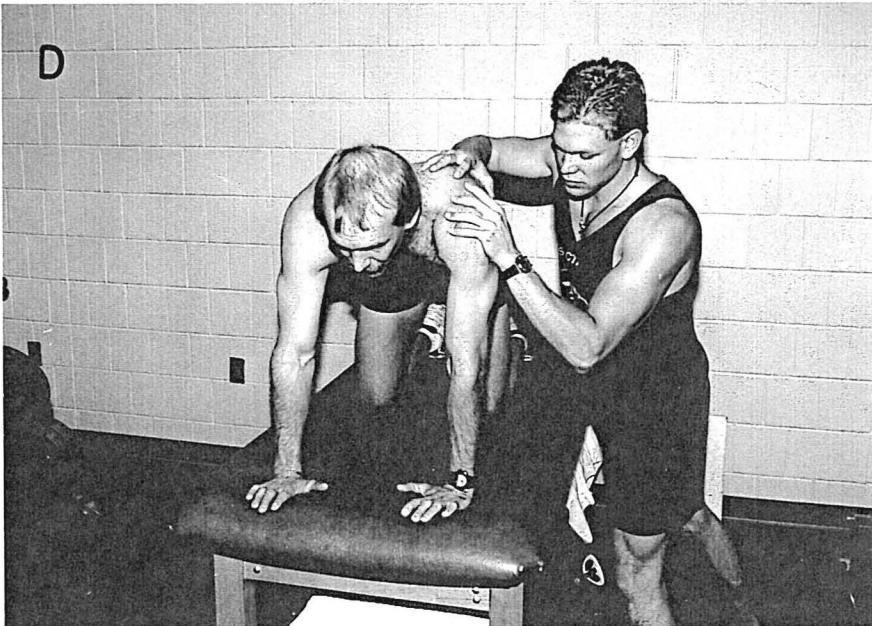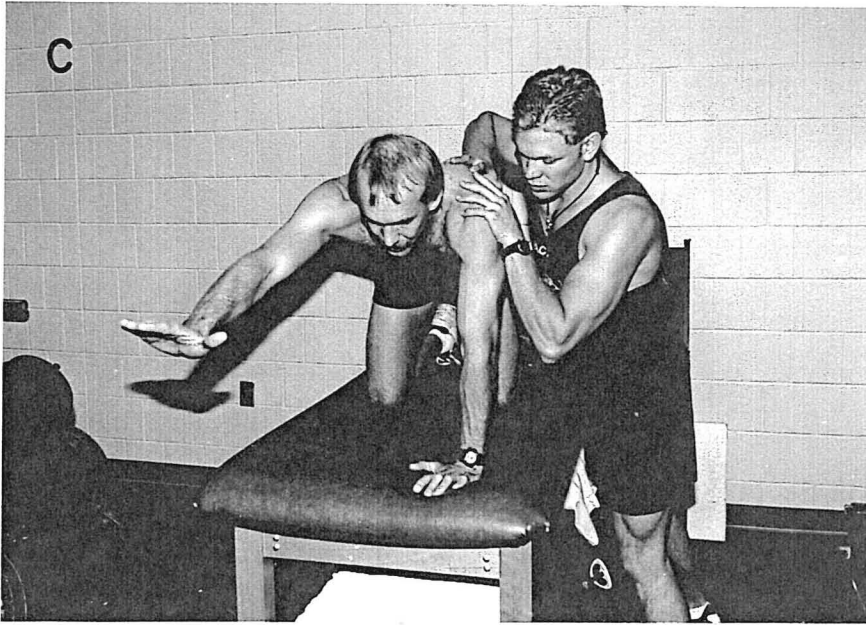
Figure 6

    <u>Using a fitter or Stairmaster</u> - This can be used to
facilitate both dynamic and static control of the
glenohumeral joint and scapular depressors. Starting on
knees (A), and progressing to more strenuous activity on the
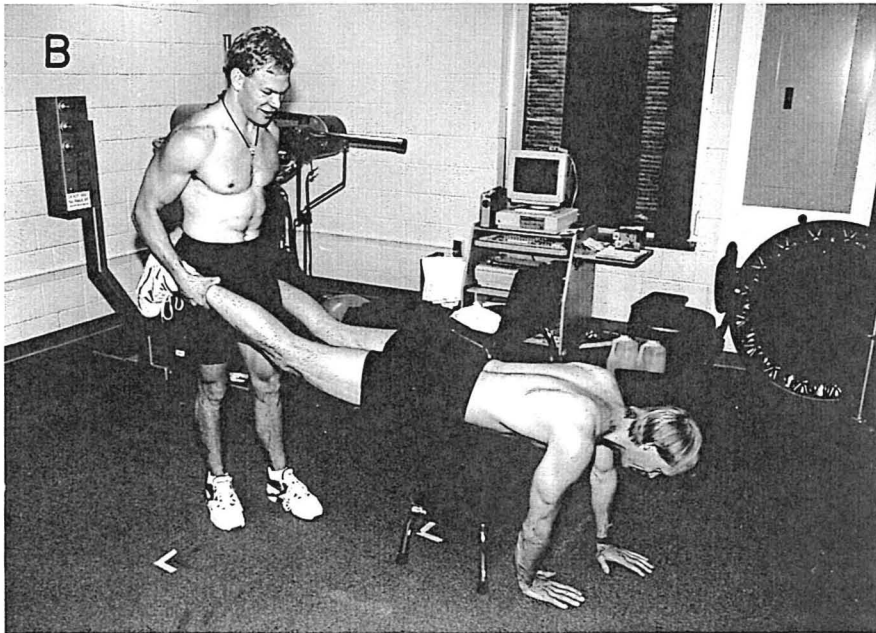toes with both machines (B,C).
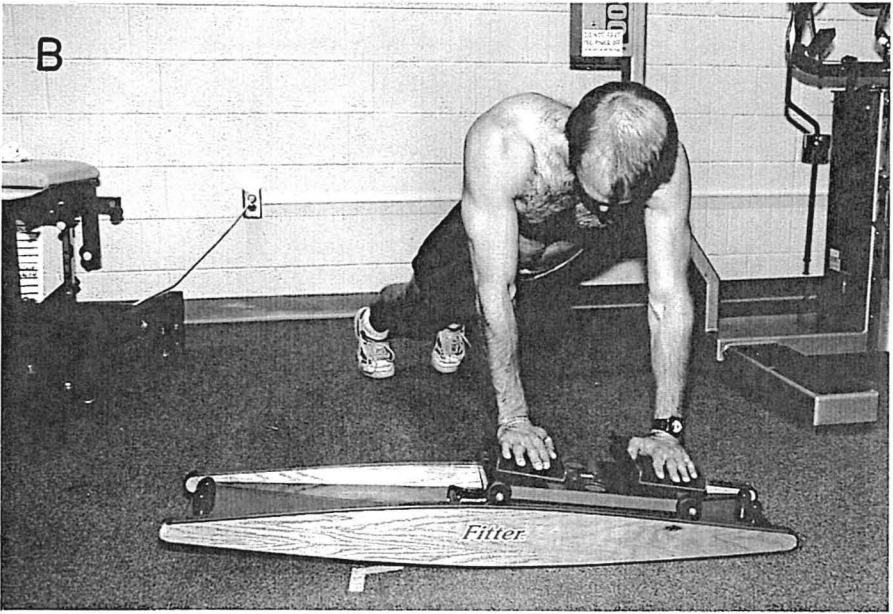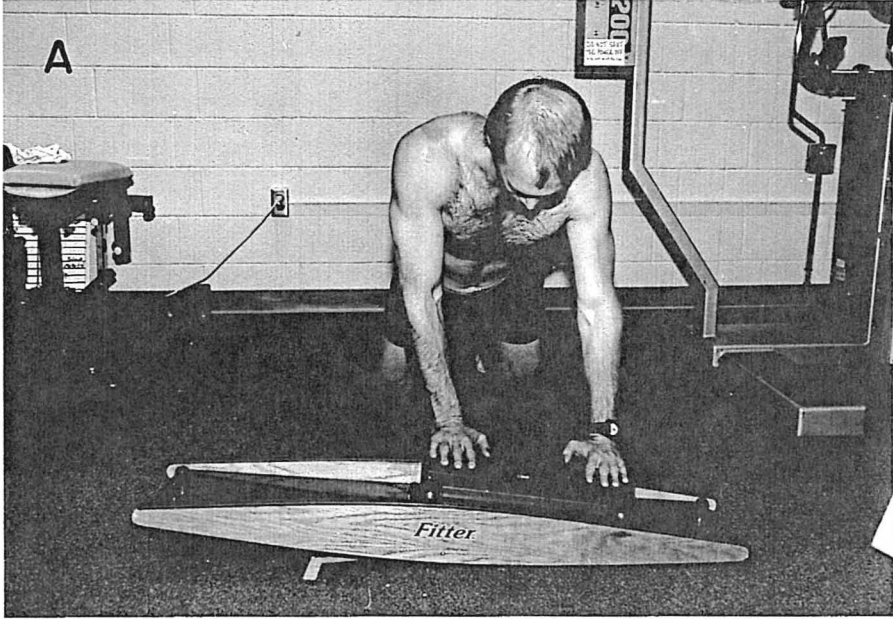
Figure 7

Alternative closed kinetic chain exercises with advancement and variability. Progressing patients such as athletes back into their specific sport can be used by the following: wheeled chair crawling (A) and wheelbarrel walking (B).
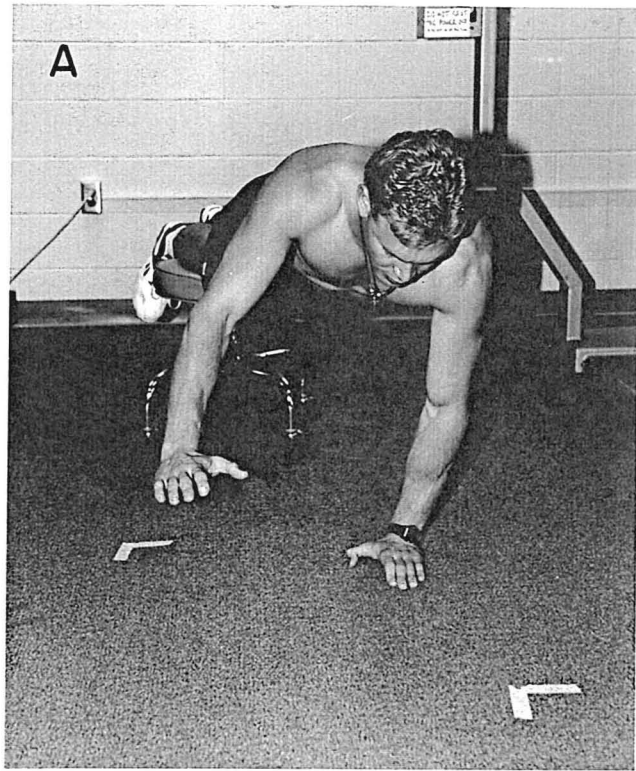
Figure 8
     Wheelbarrel walking through an obstacle course (C), and
standard treadmill activities (D).
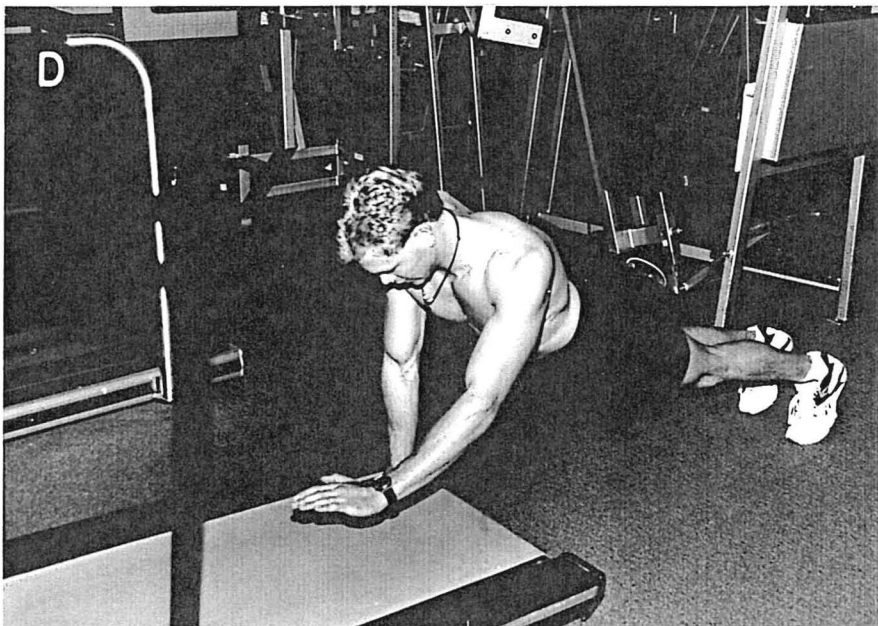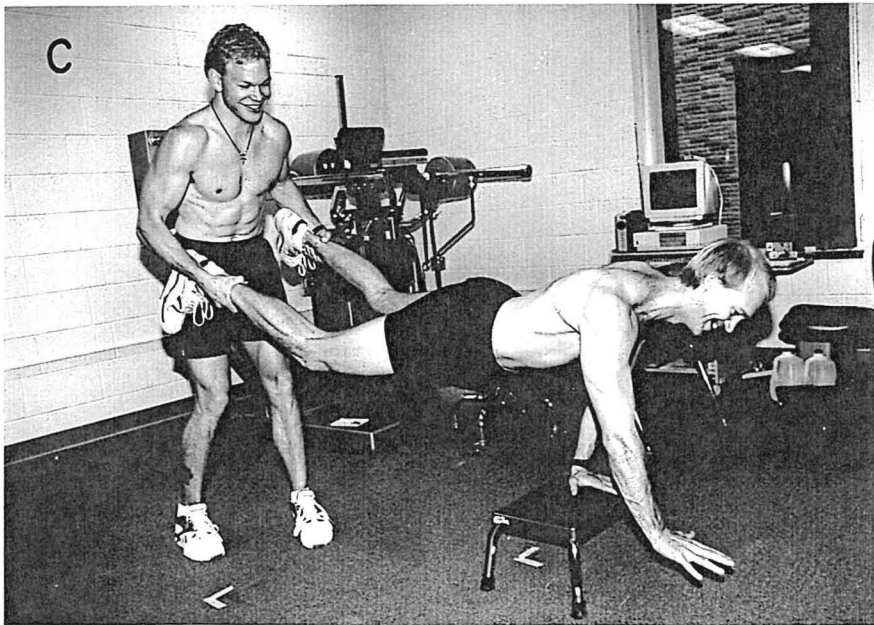
Figure 9
     Adding variable skills to treadmill activities such as
arms side-to-side (E) and carioca step (F).

CHAPTER 5

THE CLOSED KINETIC CHAIN
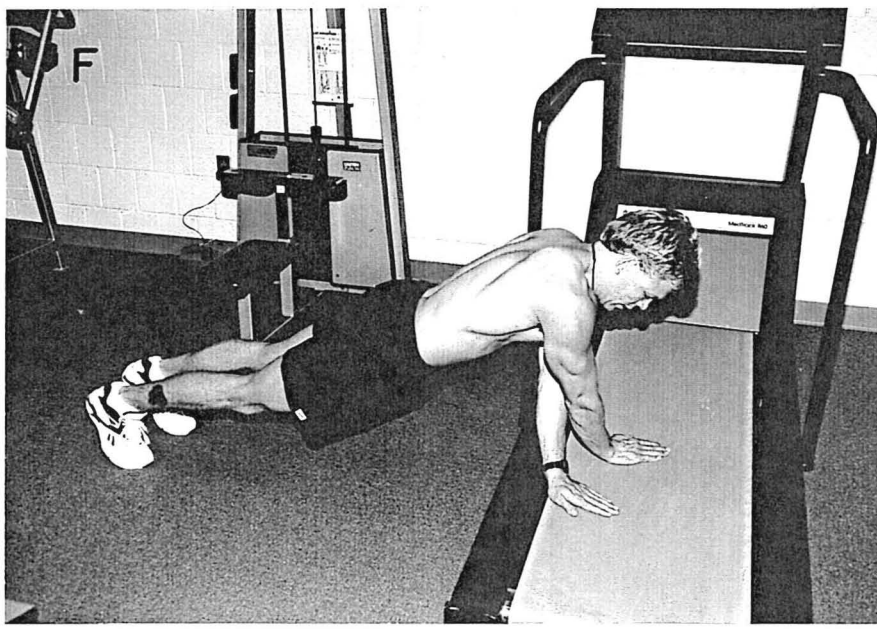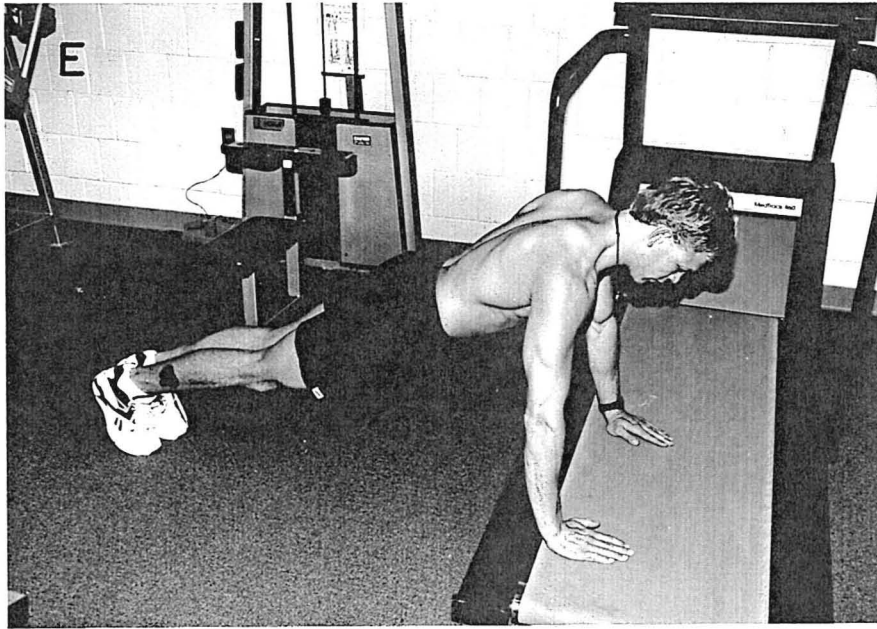
General progression of forces is included in the SAID Principle: "Specific Adaptations to Imposed Demands." This implies that the cell adapts in highly specific ways to the demands of stress imposed upon it.[43,45] Another progression is the overload principle.[43] As adaptation to stress occurs, progressively increased stress must be applied to ensure continued adaptation. This is the point when therapists should utilize the closed kinetic chain exercises.

Kinesthesia, as defined by Newton,[7] is the ability to discriminate joint position, relative weight of body parts, and joint movement including direction, amplitude, and speed. The objective of a kinesthetic rehabilitation program is to facilitate the shoulder's performance of a complicated skill without conscious guidance. The concept of closed kinetic chain exercises has been recently popularized for the lower extremity, but a similar concept can also be employed in the upper extremity.[8] Closed kinetic chain exercise is defined as fixing the distal segment to an immovable object.[2,7-9,40] This prevents

translation of either the proximal or distal joint center.
A system is created when movement at one joint produces
movement at all other joints in a predictable manner,
essentially called the closed kinematic chain.[2,7-9,40] These
exercises provide joint approximation forces, promoting co-
contraction about the joint and also enhancing dynamic joint
stability. The closed kinetic chain exercises cause axial
loading and compression in the joint, therefore increasing
noncontractile stability. The increased joint stability
through greater joint congruency and through active
contraction of the muscles help to control the stability of
the stability of the shoulder.

The extremities can be thought of as rigid, overlapping
segments in a series. Steindler[9] proposed that although a
closed kinematic chain never occurs in the extremities, two
types of kinematic chain exist under different limb loading
conditions.[9] It was observed that when the foot or hand
meets considerable resistance, muscle recruitment and joint
motion differ from that seen when the foot or hand meets
considerable resistance, muscle recruitment and joint motion
differ from that seen when the foot or hand is completely
free to move. Steindler[9] felt the difference was
significant enough to warrant distinguishing the two
conditions with separate terms.[7,9] Specifically, an open
kinetic chain exists when the peripheral joint of the
extremity can move freely, such as when waving the hand or

moving the foot forward in the swing phase of gait.[9] With the existence of a closed kinetic chain, the foot or hand meets resistance such as in the chin-up or coming up from a squat.[7-9] Steindler pointed out that a true closed kinetic chain only existed during an isometric exercise, since, by definition, neither the proximal or distal segment can move in a closed system.

To balance compression and shear forces of the glenohumeral joint, strengthening of the shoulder in a closed pack position results in less tensile stress of the capsular ligaments and facilitates co-contraction of dynamic stabilizing structures.[7] Studies show that in order to effectively balance compression and shear forces, shoulder elevation at 90 degrees is the optimal position. This is because the head of the humerus is stabilized centrally in the glenoid fossa, causing distraction forces and mechanical deformation to be minimal.[7] The use of static stability in closed kinetic chain exercises helps to "educate and train" the proprioreceptors in the shoulder girdle. This balances the shoulder girdle musculature when functioning dynamically. The involvement of the sternoclavicular and acromioclavicular joints in the scapulohumeral rhythm is an unavoidable consequence of their involvement in a closed kinematic chain with the scapulothoracic articulation. Motion at the scapulothoracic joint inescapably produces motion at the sternoclavicular and acromioclavicular joints.[10]

CONCLUSION

There has been much skepticism over the appropriateness
or function of closed kinetic chain exercises because there
is essentially no reason to do this form of exercise.
Generally, we don't use our upper extremities in a closed
kinetic chain fashion, except with crawling. I feel that
adding this form of treatment regimen in conjunction to the
standard treatment protocols, including open kinetic chain
exercises, is ideal. The different activities of closed
kinetic chain exercises improve proprioreception, joint
approximation forces and enhance dynamic joint stability by
adding a co-contraction of all the structures around the
joint. This essentially is working on increasing the
stability of what is most common, an unstable joint. The
active contraction of all the muscles in the shoulder
complex stabilize and balance shoulder structures,
preventing substitution of a stronger group that overrides
the weaker muscle groups. This form of exercise also
reduces the tensile stress on joint ligaments and tendons,
aiding in the healing process and improving the integrity of
the joint.

Closed kinetic chain exercises would not be used to
replace old treatment protocols, nor would it be one of the

first exercises performed. It is essential that the therapist adhere to proper treatment guidelines for each individual patient, as well as protocols set forth by the orthopedic surgeon.

Closed kinetic chain exercises for the upper extremity are new forms of treatment receiving a lot of early recognition as well as clinical utilization. Upper extremity closed kinetic chain exercises are used for a variety of pathologies ranging from impingement and instability to rotator cuff tears and neuropathies. By progressing each patient with appropriate protocols, gradually adding closed kinetic chain exercises as the patient tolerates aids in achieving full and complete function. Further studies are needed to enhance and elucidate the full potential of the exercises and follow-up treatments must be done before their usefulness can be validated.

Physical Therapists must recognize that every patient is different and unique and that all rehabilitation programs should be individualized. Although some of these exercises may appear to be aggressive, returning the patient back to full function is essential. The patient's age, strength, diagnosis, progression and desired goals should be carefully considered to adapt a proper treatment for each and every patient.

REFERENCES

1. Magee DJ. Orthopedic Physical Assessment. 2nd ed. Philadelphia, PA: WB Saunders Co; 1992:90-142.

2. Andrews J, Harrelson GL. Physical Rehabilitation of the Injured Athlete. Philadelphia, PA: WB Saunders Co; 1991:367-441.

3. Finke CK. Rehabilitation of Traumatic Injuries. Shoulder Seminar Handout, Feb. 1993.

4. Hooke NC. Diagnosis and Treatment of Shoulder Injuries in the Athlete. Nurs Clin North Amer. 1991; 26:199-209.

5. Adapted Protocols from Campbell County Memorial Hospital, Gillette, WY 1993.

6. Pink M, Jobe FW. Shoulder Injuries in Athletes. Clinical Management. 1991; 11:39-47.

7. Davies GJ, Dickoff-Hoffman S. Neuromuscular Testing and Rehabilitation of the Shoulder Complex. JOSPT. 1993; 18-:449-458.

8. Wilk KE, Arrigo C. Current Concepts in Rehabilitation of the Athletic Shoulder. JOSPT. 1993; 18:365-378.

9. Palmitier RA, An Kai-Nan, Scott SG, Chao EYS. Kinetic Chain Exercises in Knee Rehabilitation. Sport Med. 1991; 11:401-412.

10. Kamkar A, Irrgang JJ, Whitney SL. Nonoperative Management of Secondary Shoulder Impingement Syndrome. JOSPT. 1993; 17:212-223.

11. Wilk KE, Voight ML, Keirns MA, Gambetta V, Andrews J, Dillman CJ. Stretch Shortening Drills for the Upper Extremities: Theory and Clinical Application. JOSPT. 1993; 17(5):225-239.

12. Dillman CJ, Fleisig GS, Andrews JR. Biomechanics of Pitching with Emphasis on Shoulder Kinematics. JOSPT. 1993; 18:402-408.

13. Savoie FH. Athroscopic Examination of the Throwing Shoulder. JOSPT. 1993; 18:409-412.

14. Meister K, Andrews JR. Classification and Treatment of Rotator Cuff Injuries in the Overhead Athlete. JOSPT. 1993; 18:413-421.

15. Brewster C, Moynes Schwab D. Rehabilitation of the Shoulder Following Rotator Cuff Injury or Surgery. JOSPT 1993: 18:422-432.

16. Litchfield R, Hawkins R, Dillman, CJ, Atkins J, Hagerman G. Rehabilitation of the Overhead Athlete. JOSPT. 1993; 18:433-441.

17. Sillman JF, Dean MT. Neurovascular Injuries to the Shoulder Complex. JOSPT. 1993; 18:442-448.

18. Culham E, Peat M. Functional Anatomy of the Shoulder Complex. JOSPT. 1993; 18:342-350.

19. Ho CP. Applied MRI Anatomy of the Shoulder. JOSPT. 1993; 18:351-359.

20. Baker CL, Liu SH. Neurovascular Injuries to the Shoulder. JOSPT. 1993; 18:360-364.

21. Boublik M, Hawkins RJ. Clinical Evaluation of the Shoulder Complex. JOSPT. 1993; 18:379-385.

22. Paine RM, Voight M. The Role of the Scapula. JOSPT. 1993; 18:386-391.

23. Hurley JA, Anderson TE, Dear W, Andrish JT, Bergfield JA, Weiker GG. Posterior Shoulder Instability. Surgical Versus Conservative Results with Evaluation of Glenoid Version. Am J Sports Med. 1993; 20:396-400.

24. Glousman RE. Instability Versus Impingement Syndrome in the Throwing Athlete. Orthop Clin North Am. 1993; 24:89-99.

25. Andrews JR, Kupferman SP, Dillman CJ. Labral Tears in Throwing and Racquet Sports. Clin Sports Med. 1991; 10:901-911.

26. Sillman JR, Hawkins RJ. Current Concepts and Recent Advances in the Athlete's Shoulder. Clin Sports Med. 1991; 10:693-705.

27. Warner Jj, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Patterns of Flexibility, Laxity, and Strength in

Normal Shoulders with Instability in Impingement. Am J Sports Med. 1990; 18:366-375.

28. Moseley JB Jr, Jobe FW, Pink M, Perry J, Tibone J. EMG Analysis of the Scapular Muscles During a Shoulder Rehabilitation Program. Am J Sports Med. 1992; 20:128-134.

29. Schultz JS, Leonard JA Jr. Long Thoracic Neuropathy from Athletic Activity. Arch Phys Med Rehabil. 1992. 73:87-90.

30. Yu J. Anterior Shoulder Dislocations. J Fam Pract. 1992; 35:567-571, 575-576.

31. Burkhead WZ Jr, Rockwood CA Jr. Treatment of Instability of the Shoulder with an Exercise Program. J Bone and Joint Surg Amer. 1992; 74:890-896.

32. Warner JJ, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Scapulothoracic Motion in Normal Shoulders and Shoulders with Glenohumeral Instability and Impingement Syndrome. A study using Moire topographic analysis. Clin Orthop. 1992; 285:191-199.

33. Fields KB, Rasco T, Kramer JS, Cates R. Rehabilitation Exercises for Common Sports Injuries. Am Fam Physician. 1992; 45:1233-1243.

34. Harryman DT, Sidles JA, Harris SL, Matsen FA. The Role of the Rotator Interval Capsule in Passive Motion and Stability of the Shoulder. J Bone and Joint Surg Amer. 1992; 74:53-66.

35. Clark J, Sidles JA, Matsen FA. The Relationship of the Glenohumeral Joint Capsule to the Rotator Cuff. Clin Orthop. 1990; 254:29-34.

36. Kursunoglu, Brahme S, Resnick D. Magnetic Resonance Imaging of the Shoulder. Radiol Clin North Am. 1990; 28:941-954.

37. Soslowsky LJ, Flatow EL, Bigliani LU, Mow VC. Articular Geometry of the Glenohumeral Joint. Clin Orthop. 1992; 285:185-190.

38. Terry GC, Hammon D, France P, Norwood LA. The Stabilizing Function of Passive Shoulder Restraints. Am J. Sport Med. 1991; 19:26-34.

39. Howell Sm, Kraft TA. The Role of the Supraspinatus and Infraspinatus Muscles in Glenohumeral Kinematics of

Anterior Shoulder Instability. Clin Orthop. 1991;
263:128-134.

40. Sachs Ra, Danile DM, Stone ML, Garfen RF.
Patellofemoral Problems After Anterior Cruciate
Ligament Reconstruction. Am J Sport Med. 1989; 17:760-
765.

41. Filzen D. Differential Diagnosis of the Complicated
Patient. Outline from Minnesota Sport and Spine Rehab,
Feb. 1993.

42. Askew M. Shoulder Pathologies. Outline from Orthopaedic
Associates of Fargo, ND; Red River Valley Sports
Medicine Institute, Feb. 1993.

43. Filzen D. Rehabilitation of Overuse Injuries. Outline
from Minnesota Sport and Spine Rehab, Feb. 1993.

44. Blair DF, Willis RP. Rapid Rehabilitation Following
Anterior Cruciate Ligament Reconstruction. Athletic
Training. Spring 1991; 26:32-42.

45. Finke CK. Rehabilitation of Traumatic Injuries. Outline
from Shoulder Seminar, Feb. 1993.

46. Finke CK. Overuse Injuries of the Shoulder. Outline
from Shoulder Seminar, Feb. 1993.

47. Finke CK. Traumatic Injuries of the Shoulder. Outline
from Shoulder Seminar, Feb. 1993.

48. Rothstein JM, Roy SH, Wolf SL. The Rehabilitation
Specialist's Handbook. Philadelphia, PA: F.A. Davis
Company; 1991:69-92.

49. O'Connell PW, Gordon NW, Mileski RA, Lautenschlager E.
The Contribution of the Glenohumeral Ligaments to
Anterior Stability of the Shoulder Joint. Am J Sports
Med. 1990; 18:579-585.

50. Mohr TM. The Shoulder Joint. Notes in Muscle Function
in Health and Disease; Spring 1992. Grand Forks, ND.

51. Dorland's Illustrated Medical Dictionary. Philadelphia,
PA: WB Saunders Co; 1988.

52. Neer CS. Impingement Lesions. Clin Orthop. March, 1983;
173.