Biomechanics of Unilateral Overhand Throwing Motion: An Overview

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Abstract: ***Shoulder injury is a common occurrence for the throwing athlete. These injuries are often attributed to faulty throwing technique and/or various congenital/developmental malformations. Understanding the structural interactions associated with the unilateral throw will enable the practitioner to better isolate mechanical flaws predisposing to injury and, in turn, initiate the appropriate treatment program. The vast majority of shoulder injuries respond well to a program of manipulation, stretches, and various exercises applied during specific stages of rehabilitation.***

Key Words: biomechanics, shoulder injury, rehabilitation.

INTRODUCTION

The ability to launch an object along a specific flight pattern is a necessary prerequisite for success in a wide variety of sports. In fact, Bunn (1) mentions the skill of throwing is second only to running as a common element in sport. Because the throwing athlete must often project objects with pinpoint accuracy of speeds exceeding 90 mph, the various articulations of the torso and upper extremity are subjected to tremendous strains. For example, Perry (2) noted that during the early acceleration stage of the overarm pitching motion, torsional strains placed on the anterior glenohumeral joint capsule may exceed 17,000 kg/cm^2. Furthermore, Pappas et al. (3), demonstrated that during the late acceleration phase of overarm throwing, the elbow is extending thru an arc at 500,000°/second/second. Obviously, failure to effectively generate and absorb such extreme forces in a smooth, coordinated fashion may result in chronic tissue damage. The purpose of this article is to review the biomechanics of the unilateral throwing motion and then relate this information to the more commonly seen injuries, their prevention, and treatment.

DISCUSSION

Despite what appear to be dissimilar movement patterns, Atwater (4), using high speed cinematography, demonstrated remarkable similarities between throwing sports (Fig. 1). In all of these sports, the degree of glenohumeral abduction remains somewhat constant while variation in overarm angulation is accomplished primarily by varying degrees of spinal lateral flexion. These kinematic studies demonstrate that whether the athlete is projecting a football, javelin, tennis racket, or baseball, the majority of movement patterns are the same. In order to better describe these movement patterns, various authors have listed between three and six different stages to the unilateral throw (5–9). This paper will subdivide the unilateral throwing

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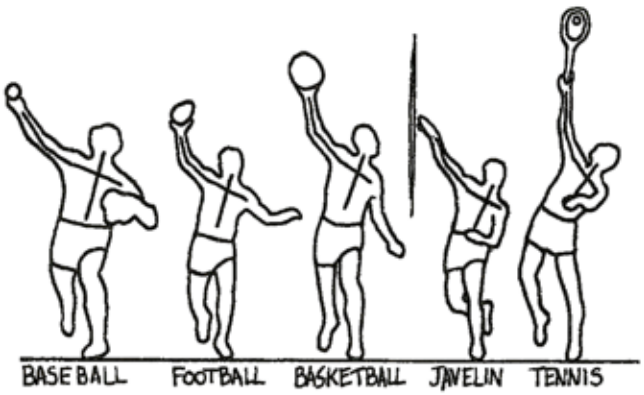

Figure 1. Tracings of photographs taken during release/impact in a variety of sports. Adapted from Atwater (4).

motion into four stages: wind-up, acceleration I, acceleration II, and follow-through. Following is a discussion of the biomechanical events associated with each stage.

WIND-UP

The wind-up stage begins when the athlete first shifts his/her center of mass toward the ipsilateral or pivot foot (i.e., in right handed throwers, this is the right foot) and ends, in baseball pitching, when the ball is removed from the glove (Fig. 2). This stage typically lasts between 0.5 and 1 second depending on the individuals throwing style (3).

The purpose of wind-up is two-fold: it allows the athlete to establish a rhythm and, more importantly, it places all segments of the body into a position where they can contribute to propulsion of the ball. Gowitzke et al. (5) claim that activation of the shoulder external rotators during wind-up stimulates the G.T.O. of these muscles which, in turn, facilitates a stronger contraction of the antagonistic accelerators during later stages. However, the contribution afforded by G.T.O. facilitation must be considered negligible as it has been demonstrated that pitching from a stretch position (a position in which wind-up motions are markedly minimized in order to allow runners less time to steal a base) does not detrimentally affect velocity of the throw (4).

As the wind-up stage ends, body weight is centered over the pivot foot and the contralateral stride leg begins to move forward and upward as the lumbar spine shifts from a slightly extended to a slightly flexed position. As you can see from the chart on the muscle function (Fig. 3), there is relatively little muscular activity during wind-up. Also, the absence of significant torsional strains during this stage allow the shoulder and torso to remain relatively unstressed.

Figure 2. Wind-up.

ACCELERATION I

As with wind-up, acceleration I typically lasts between 0.5 and 1 second (3). It is during this stage that kinetic energy is transferred thru the lower extremity and trunk into the arm and hand (6). This is accomplished as the forward motion of the stride leg begins to rotate the pelvis

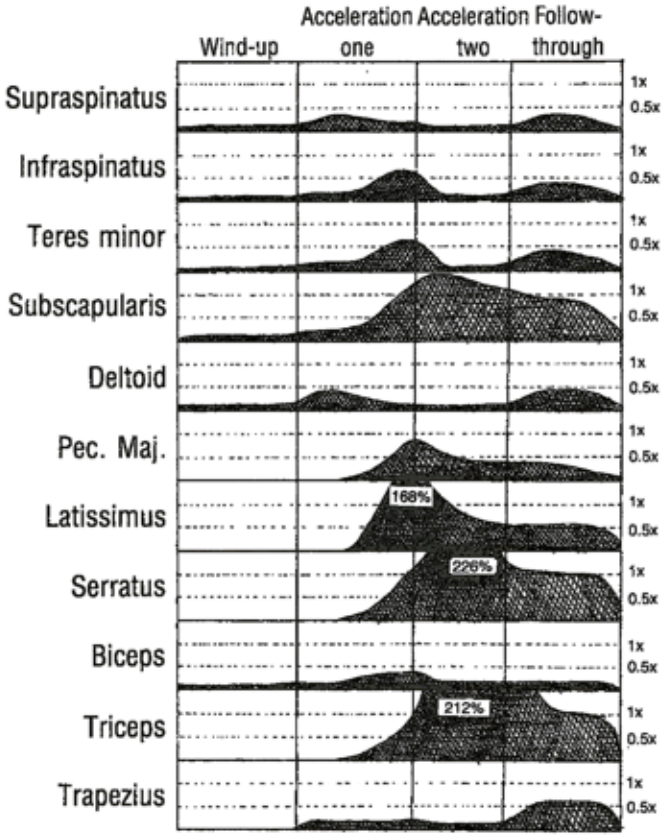

Figure 3. Muscular activity during the unilateral throw. (Based on information from references 10, 17, and 71). X is used to quantify intensity of muscular effort as it represents E.M.G. activity recorded with a single full effort isometric contraction.

toward the left which is immediately followed by a segmental rotation of the trunk, progressing from the pelvis to the shoulders. Braatz and Gogia (6) state, "the pelvis, which is leading the shoulders in opening, exerts a torque effect on the vertebral column, thereby transferring energy thru taut soft tissue structures from the lower extremity to the shoulder and throwing arm." The importance of those lower extremity and trunk motions should not be underestimated as Toyoshima et al. (7) demonstrate 46.9% of the ball's final velocity can be attributed to step and body rotation while 53.1% results from arm action.

Toward the end of acceleration I, the upper trunk reaches its fastest angular velocity of forward rotation, thereby completing the sequential trunk rotation (3). As this occurs, the entire arm lags behind the shoulder line and is forced by inertial forces and the pull of the posterior glenohumeral musculature into a position of extreme external rotation (Fig. 4). It is at this time that impingement of the supraspinatus tendon, the long head of the biceps brachii and/or the subdeltoid bursae may occur as these tissues may become sheared under the coracoacromial arch (Fig. 4A).

The repetitive strains associated with such an extreme in glenohumeral external rotation frequently lead to developmental changes in shoulder strength and range of motion. Cook et al. (8) evaluated dominant and nondominant shoulder ranges of motion in fifteen college level baseball pitchers and found the throwing shoulder possessed increased ranges of external rotation with concomitantly decreased ranges of internal rotation. While the overall range of motion remained equal to the nondominant side, a greater percentage of the total range of motion in the throwing arm occurred with the humerus in an externally rotated position. Interestingly, Sandstead (9) found that throwing velocity was significantly related to the range of external shoulder rotation. It is theorized that the increased range of external glenohumeral rotation allows the accelerational muscles to internally rotate the humerus thru a larger range for a longer period of time, thereby allowing these muscles to impart added momentum to the ball during the acceleration II stage. Also, the same study by Cook et al. (8) demonstrated the potential for muscular imbalance between shoulder internal rotators and external rotators as the forces of acceleration I and II frequently produce a developmental hypertrophy of the internal rotators.

Jobe et al. (10) note that at the end of acceleration I the glenohumeral capsule has been wrapped tightly in external rotation and is ready to be released during acceleration II like a coiled spring. The soft tissues along the anterior aspect of the glenohumeral capsule are wound-up to an elastic force and the accelerational muscles have been stretched to their maximums (2). Prestretching these muscles at this time allows for the storage of elastic energy which can be returned during acceleration II as the muscles reverse direction of contraction to accelerate the humerus in internal rotation and horizontal flexion. Ciullo and Zarins (11) compare the prestretching of a muscle in order to store and return energy to the cocking of an air rifle. However, it should be emphasized that the stretching of the muscle must be followed by immediate muscular contraction, otherwise the stored energy will be dissipated as heat (12).

At the end of acceleration I, the lumbar spine is in an extended position, and the elbow is flexed 90° with the forearm supinated and the wrist in neutral or slightly extended (3,4). The throwing shoulder is abducted 90°,

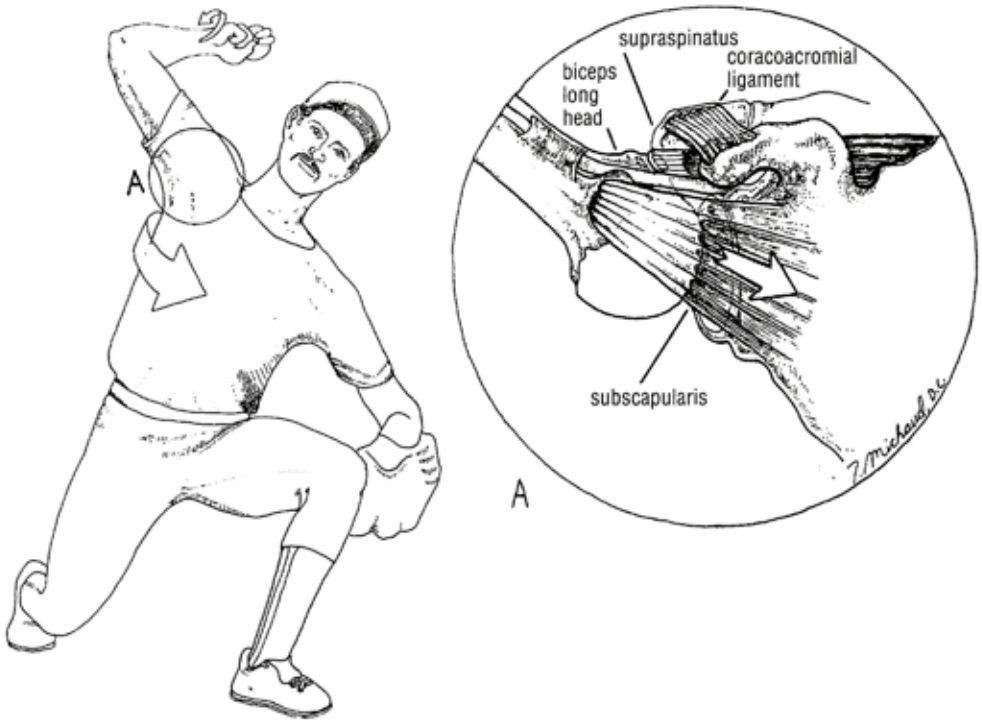

Figure 4. Acceleration I.

externally rotated 90° (or more) and horizontally extended 30°. Shoulder positioning is accomplished primarily by deltoid activity with some "fine tuning" by the rotator cuff musculature (10). Notice in Figure 3 that the subscapularis musculature demonstrates greatest activity during late acceleration I and early acceleration II; this is the last muscle to contract to terminate the exaggerated range of glenohumeral external rotation.

ACCELERATION II

Even though this phase lasts less than one tenth of a second (3), it is a period of time in which injury frequently occurs as the ball is being accelerated from its relatively immobile position present during acceleration I to speeds in excess of 90 mph. Seavers (13) refers to this period as "show time" as it is where virtually all accelerational forces are applied to the ball.

At the beginning of acceleration II the shoulder is externally rotated and horizontally extended and the humeral head is positioned approximately 4 mm posteriorly in the glenoid fossa (14). As acceleration II proceeds the shoulder and elbow move forward as a unit (horizontally flex) as the shoulder almost simultaneously begins to internally rotate (15) (Fig. 5). During this time the humeral head is gliding anteriorly creating a shearing stress on the articular surface and glenoid labrum (14).

Braatz and Gogia (6) note that acceleration II begins as the force of inertia and the pull of the posterior glenohumeral musculature is reversed by the explosive contraction of the accelerational muscles. The authors state this force reversal loads all joints from the vertebral column to the forearm. Perry (2) claims the joint capsule uncoils like a loaded spring with torsional strains exceeding 17,000 kg/cm. Pappas et al. (3) have measured angular velocity of the humeral head during acceleration II at 9,190°/second. These extreme torsional strains have been implicated as causal in the development of spiral fractures of the humerus (16).

Figure 5. Acceleration II.

In skilled throwers the larger accelerational muscles such as subscapularis, latissimus dorsi, and serratus anterior demonstrate peak activity during acceleration II, while the deltoid and the remaining rotator cuff remain surprisingly quiet (17). Gowitzke et al. (5) emphasize the role of serratus anterior during acceleration II claiming scapular protraction is largely overlooked regarding its contribution to forward acceleration of the ball [the scapula protracts approximately 2 inches during acceleration II (5)]. This is consistent with recent E.M.G. findings in which serratus anterior was found to be an extremely important contributor to racket velocity in both tennis serves and forehands (18). Interestingly, Gowen et al. (17) demonstrated that less successful throwers generate much greater activity during acceleration II in the smaller supporting muscles, such as the infraspinatus, teres minor, and supraspinatus.

By the time the ball release has occurred to end acceleration II, the shoulder has internally rotated to a point between 40 and 60° of external rotation and the wrist has flexed to a neutral position. The elbow, which begins acceleration II at 90°, initially flexes to 120° and then finally extends to 25°. Remarkably, during the final 10 to 20 milliseconds prior to ball release, the elbow reaches a peak acceleration of 500,000° second/second (3). Atwater (4) states "the forearm in normal throwing is swung like a whip by the rotary actions of the hip and pelvis and by the forceful contraction of the triceps." The radioulnar joint, depending on the type of pitch thrown, typically begins to pronate approximate 10 milliseconds prior to ball release and continues this motion throughout follow-thru. Although the role of radioulnar pronation remains somewhat controversial, it appears the wrist is pronated slightly before ball release with a screwball and slightly after release with a curveball (4). However, it should be emphasized that regardless of the type of pitch thrown, the radioulnar joint eventually pronates as the pronator teres muscle continues to actively pronate the wrist throughout follow-through (4).

FOLLOW-THROUGH

This period begins with ball release and continues until all motion has stopped. This typically takes 350 milliseconds (3). Throughout the follow-through, the shoulder continues to internally rotate and horizontally flex: Gainor et al. (16) note an average of 40° horizontal flexion. As with early acceleration II, the forces acting on the shoulder during follow-through are extreme. For example, when serving a tennis ball, the racket head must be decelerated during follow-through from its peak

speed of 300 mph (19). McCleod (20) estimates the decelerational forces to be twice the accelerational forces but claims they are no more likely to produce injury as they are applied over a longer time period. As is to be expected with such large forces, the follow-through phase is associated with much muscular activity as all of the rotator cuff muscles fire vigorously. The posterior deltoid, trapezius, latissimus dorsi, and triceps are also extremely active as they attempt to decelerate the throwing arm. Injury may result as inertial forces traction the humerus in long axis extension allowing for as much as a 1 inch separation between the humeral head and glenoid fossa (Fig. 6). The repeated tensile strains associated with this motion may eventually lead to traction spurs along the posterior joint as the humerus pulls on the capsule, triceps long head, and teres minor insertion. This may eventually lead to a bony outgrowth at these points (21).

During the latter portions of follow-through, the trunk continues to flex and the scapula continues to protract. Scapular protraction is critical in preventing rotator cuff irritation as it prevents the greater tuberosity from colliding with the coracoacromial arch (22). At the end of follow-through, the pitcher must be in position to field a returned ball.

To summarize, success during the throwing motion is dependent on the athletes' ability to sequentially accelerate and decelerate all aspects of the kinetic chain. Upon studying numerous high speed serial photographs, Atwater (4) makes the observation that as each body segment is being accelerated, its more distal segment first lagged behind (which allows for a prestretching of its supporting musculature), then acquired the speed of the segment moving it and finally accelerated to reach an even greater angular velocity as the proximal segment decelerated. Moorehouse and Cooper (23) developed a schematic diagram to summarize these segmental accelerations (Fig. 7A). These authors emphasize that failure to accelerate each successive segment during peak acceleration of its proximal segment would result in a lessened outcome (Fig. 7, B and C). This is consistent with Atwater's (4) observation that unskilled female throwers tended to move the entire trunk and arm forward as a unit rather than accelerating each segment individually. This movement pattern resulted in a throw that more closely resembled a "fast-push" as these throwers would move the various body segments through much smaller linear and angular ranges.

MECHANISMS OF INJURY

As mentioned earlier, in order to remain injury free, the athlete must be able to generate and absorb large amounts of force in very short periods of time. While acute injury may result from simple overuse with an inability of the involved tissues to adapt to the imposed demands, many chronic injuries result from various congenital and/or developmental disorders. These disorders are discussed in the following paragraphs.

Congenital

While there are numerous congenital malformations capable of producing chronic shoulder injury (such as defects in the shape of the humeral head and/or acromion) possibly the most common malformation responsible for shoulder pain and dysfunction is congenital variation in the shape of the intertubercular groove. In fact, tenosynovitis of the long head of the biceps in the intertubercular groove is considered to be the most common injury to the anterior shoulder (4) and because of its tendency for chronicity has been referred to by baseball pitchers as a "glass-arm" (24).

In a study of anatomical variations in 100 humeri, Hitchcock and Bechtol (25) noted the presence of a supratubercular ridge (the ridge of Meyer) in 67% of the humeri evaluated. This ridge was markedly developed in 8% of the humeri and moderately developed in 59% of the humeri (Fig. 8).

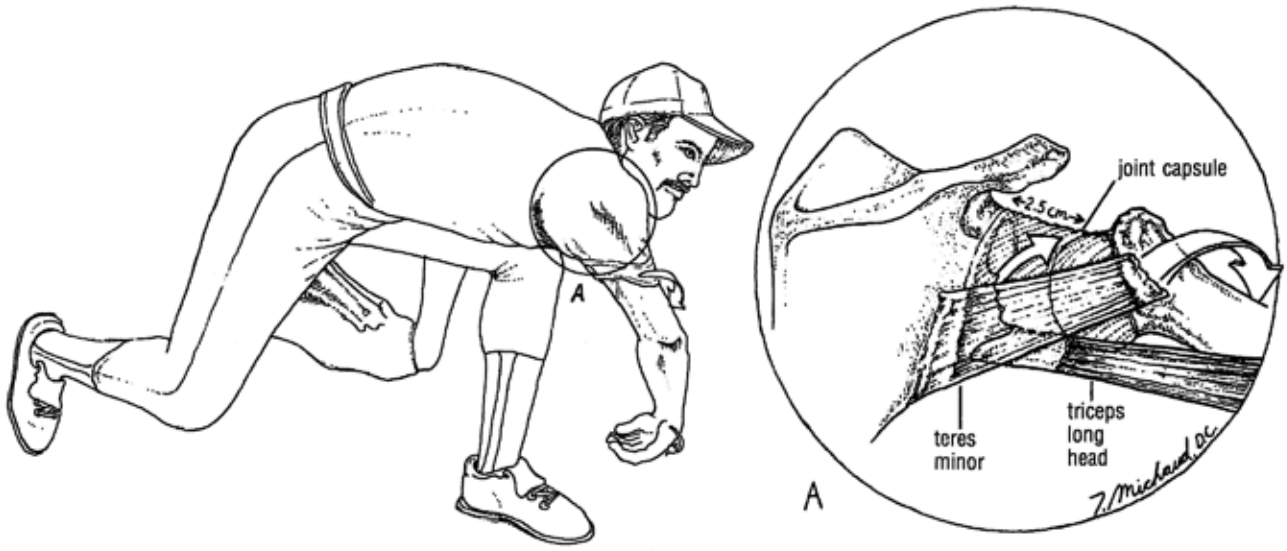

Figure 6. Follow-through [insert A has been adapted from McCleod (20)].

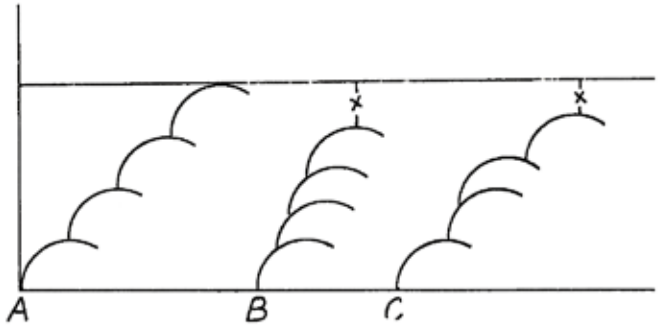

Figure 7. Schematic diagram to demonstrate how the greatest final velocity is achieved when each body segment is accelerated as its more proximal segment reaches peak acceleration (A). In (B) and (C), body parts are accelerated out of synchrony thereby resulting in a lessened outcome (X).

The authors state that the presence of a large supratubercular ridge forces the biceps long head tendon upward, where it pushes against the traverse humeral ligament. The increased pressure of the tendon against its fibrous roof predisposes to an inflammatory reaction as is evidenced by the presence of a spur on the lesser tuberosity of 45% of the individuals possessing a supratubercular ridge (Fig. 8). Note that only 3% of the humeri lacking the ridge showed evidence of a spur.

In addition to the presence of the ridge, the depth of the intertubercular groove (which is expressed as the angle of the medial wall of the groove) also plays an important role in the etiology of shoulder dysfunction. In the same study by Hitchcock and Bechtol (25), 8% of the humeri studied had a medial wall angled at less than 45° (Fig. 9). This formation resulted in insufficient medial stabilization of the biceps long head tendon which predisposed to chronic dislocation of the tendon from its fascial sling. It should be emphasized that neither the supratubercular ridge or shallow bicipital groove will traumatize the tendon unless the tendon is maintained against the lesser tuberosity and medial wall of the groove (25). Unfortunately, the sport-induced overdevelopment of the humeral internal rotator musculature that is frequently seen in throwing athletes may act to maintain the humerus in an internally rotated position during most activities. This internally rotated position of function forces the biceps tendon against the medial wall of the groove where it may be traumatized.

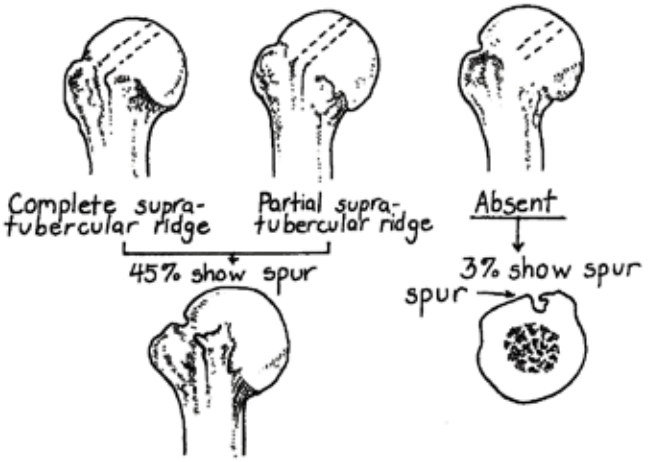

Figure 8. The Supratubercular ridge [adapted from Hitchcock and Bechtol (25)].

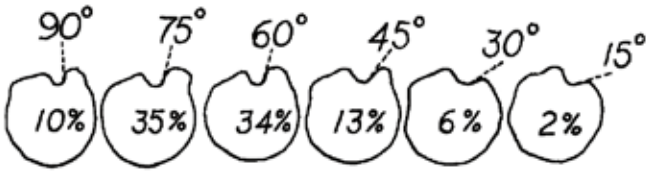

Figure 9. Variation in angle of the medial wall of the biceps groove [adapted from Hitchcock and Bechtol (25)].

Developmental

Shoulder injuries resulting from developmental changes in the supporting tissue are more likely to produce problems in the experienced athlete as the majority of individuals with congenital malformation do not progress beyond the early stages of their athletic careers. As mentioned, a common developmental imbalance seen in the throwing athlete is overdevelopment of the glenohumeral internal rotators with an increased range of external rotation and a decreased range of internal rotation (8). In addition, a recent study of trunk strength in various elite athletes demonstrated a sport-induced overdevelopment of the trunk flexors in tennis players (theoretically resulting from stresses associated with the accelerational phases of the service motion) with normal or even less than normal amounts of trunk extensor strength (26). This information is clinically significant as this particular imbalance is frequently seen in populations with chronic low back pain (27,28) and could help to explain the prevalence of low back pain among professional tennis players. Care must be taken when designing a rehabilitation program for these individuals as overemphasis of abdominal exercises to prevent hyperextension injury of the lumbar spine would only amplify the imbalance.

Possibly the most common mechanism for developmental shoulder injury is the repetitive overload of the rotator cuff musculature during the acceleration II phase. This is particularly true if the athlete begins training with an inadequate warm-up: Safran et al. (29) demonstrate that warming a muscle with isometric contractions may reduce the risk of injury as it increases the elasticity of the muscle-tendon junction (which would allow for improved dampening of the tensile forces associated with acceleration II). However, if the stresses associated with the various stages of the throwing motion exceed the muscles ability to adapt to these forces, breakdown will occur. The muscle-tendon junction is most frequently injured as it is structurally the weakest point of the muscle/tendon/bone complex (29). Microtears at the myotendinous junction may result in chronic injury as the inflammatory response frequently produces a neurological inhibition of the involved muscle which often leads to disuse and atrophy. This is particularly troublesome

when the rotator cuff musculature is involved as weakness of these muscles allows the powerful deltoid to function unopposed in creating a superior shear of the humeral head (Fig. 10). The resultant upward excursion of the head in the glenoid fossa may result in impingement of the supraspinatus, biceps long head and/or subacromial bursae between the humeral head and the coracoacromial arch. Because the humerus must drop inferiorly (30) and externally rotate (31) in order for the greater tuberosity to clear the acromion during glenohumeral abduction, physical activity with the humerus maintained in an elevated position will not only limit the range of abduction possible, but will also produce a chronic shearing of the various tissues trapped between the humeral head and the coracoacromial arch. Prolonged activity with the humeral head in this position will lead to degenerative changes with inflammation, pain, and eventual loss of function (32). As cited by Simon and Hill (33), Howard (34), initially presented the idea that trauma can cause a vascular granular tissue reaction which frays the involved tendon causing it to eventually rupture under stress. The avascular sections of the supraspinatus and biceps long head tendons (which are referred to as the critical zones of these tendons) are particularly prone to injury as a cell-mediated inflammatory response at these critical zones results in lysozomal breakdown of connective tissue (33). (It should be emphasized, however, that Neer (35) feels that 95% of all rotator cuff tears are initiated by impingement wear rather than circulatory impairment.)

In an overview of the healing process, Kellet (36) states that the remodeling phase of tissue repair involves the incorporation of immature type three collagen fibers. Because these fibers possess fewer cross links between their tropocollagen units, they are less able to resist tensile strains and are therefore more likely to rupture with the application of forces during the throwing motion. In addition to mechanically weakening the tendon, the process of repair frequently results in a granular reaction capable of increasing the diameter of the tendon. Unfortunately, the enlarged tendon is even more prone to impingement shear (with continued fibrosis) as it takes up more space between the humeral head and the coracoacromial arch. Atwater (4) makes an important observation regarding impingement wear in throwing athletes as she states a sport-induced hypertrophy of the muscles located underneath the coracoacromial arch predisposes these tissues to entrapment during the various stages of the throwing motion. Other mechanisms for developmental impingement include spur formation at the anterior/inferior acromioclavicular joint, hypertrophy of the coracoacromial ligament and/or enlargement of the anterior/inferior acromion (33).

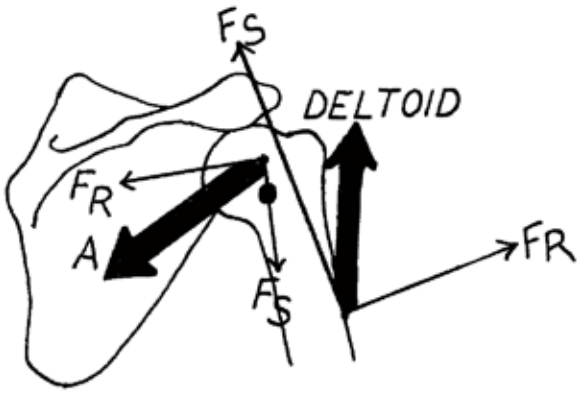

Figure 10. Force couple of rotator cuff and deltoid muscles: the combined pull of the rotator cuff muscles (arrow A) is applied above the glenohumeral axis for abduction (black dot) and can be resolved into forces that abduct the humerus (Fr) and forces that shear the humerus inferiorly (Fs). The combined pull of the deltoid, which is applied below the axis for abduction, can be resolved into forces that abduct the humerus (Fr) ad forces that shear the humerus superiorly (Fs). Normally, the inferior shear of the rotator cuff musculature will negate the superior shear of the deltoid [adapted from Shenkman and Rugo De Cartaya (31)].

Besides producing obvious damage to the tissues located directly below the coracoacromial arch, impingement wear may also result in an adhesive capsulitis of the glenohumeral joint secondary to disuse. Even when subtle, a limited range of glenohumeral motion may produce injury to the neighboring structures because of the faulty compensatory movement patterns. For example, a decreased range of glenohumeral horizontal flexion will result in an increased range of compensatory scapular protraction during the follow-through phase. The increased lateral excursion of the scapula necessary to allow the upper extremity to cross in front of the torso will strain the rhomboids and trapezius musculature (which are eccentrically contracting to decelerate the exaggerated scapular motions) and, if sufficient, will lead to a tractioning of the suprascapular nerve capable of producing a neuropraxis. Because the suprascapular nerve innervates infraspinatus and supraspinatus, prolonged tractioning of this nerve will eventually lead to weakness and atrophy of these muscles. This only adds to the shoulder pain and dysfunction as it decreases rotator cuff stabilization of the glenohumeral joint. In a well-written article on rehabilitation of the pitching shoulder, Pappas et al. (37) emphasize that failure to correct biomechanical problems elsewhere in the body may result in secondary injuries such as rotator cuff and labral tears. Possibly the best example of this relates to an injury suffered by a pitcher named Dizzy Dean. During the 1937 all-star game, Dizzy Dean, a successful pitcher for the Cardinals, suffered a broken big toe when he was struck by a line drive. Unfortunately, Mr. Dean did not allow this injury to heal completely and he returned to competitive play 2 weeks later wearing a splint and an oversized shoe. Apparently, in an attempt to accommodate the injured toe, Dizzy Dean made subtle alterations in the mechanics of his throwing motion. Even though these were seemingly insignificant changes, the altered style resulted in a shoulder injury that led to the end of a successful career. For this reason, injuries resulting from faulty compensatory movement patterns have become known as the "Dizzy Dean Syndrome" (38).

A final consideration in this discussion of developmental injuries relates to abnormal mechanics of the spine. As mentioned earlier, variation in overarm angulation during the unilateral throw is accomplished pri-

marily by laterally flexing the trunk away from the throwing arm (Fig. 1). In order for the throwing athlete to maintain his or her head in the sagittal plane (which allows the athlete to better focus on the target) the cervical spine will "typically" laterally flex in the opposite direction; i.e., towards the throwing arm. (Note that "typically" is emphasized as there is much individual variation in head positioning during the unilateral throw). Because of the coupled movement patterns present in the various portions of the spine, right lateral flexion of the head and neck produce ipsilateral rotation (right rotation) of the second cervical through the third thoracic vertebrae, while left lateral flexion of the dorsal spine (T3 and below) produce contralateral body rotation (which also happens to be right rotation) of the thoracic vertebrae (39).

These ideal coupled movement patterns act to minimize intervertebral torsional strains as the mid and lower cervical vertebrae and the dorsal vertebrae are all rotating in the same direction. An important consideration is that the upper cervical vertebrae (specifically C1 and C2) counteract the cumulative right rotation of the mid and lower cervical spine by rotating to the left (40). This again allows the throwing athlete to maintain a face forward position although it gives the illusion that only pure lateral flexion is taking place in the cervical spine.

The described movement patterns were referred to as ideal because in order for them to occur, the axes of motion for the individual vertebrae must maintain the proper spatial relationships throughout their ranges of motion (joint dysfunction cannot be present) and the dorsal spine must be maintained in a neutral or extended position: As Grieves (39) points out, when the dorsal spine is maintained in a flexed position (as in the kyphotic athlete) the thoracic vertebral bodies will rotate ipsilaterally, not contralaterally with lateral flexion. This being the case, or if joint dysfunction is present in the upper cervical or dorsal spine, excessive torsional strain will be placed on the lower cervical articulations as the left rotating dorsal spine (or nonrotating dorsal spine) meets the right rotating lower cervical spine. (Of course, all of these motions would be reversed in the left-handed athlete). If these faulty movement patterns are allowed to continue over a period of time, degenerative changes to the involved discs and neural arches may follow (41). If degenerative changes were already present (as in many older weekend athletes seen in practice) or if the faulty movement patterns were extreme, the excessive anteroposterior segmental translation associated with extension and rotation could mechanically irritate the nerve and if sufficient, traction the involved nerve root (which is fixed by ligamentous attachment to the transverse process gutter). This can initiate a three stage process of neural damage as described by Lee (41).

First, the excessive tractioning of the nerve root allows the dura to create a tension level capable of compromising the venous drainage in the vasa nervorum (39,42). The dura, unlike the nerve root, has no elongation potential and therefore creates a compressive tensile force when tractioned (43). According to Sunderland (42), the early signs of nerve root ischemia include hypersensitivity of the related dermatomes with spontaneous firing of the large myelinated fibers (particularly the gamma motoneurons). Because the gamma motor neurons regulate sensitivity of the muscle spindle complex, the first stage of nerve root tractioning is frequently associated with dense palpable chords of muscle (i.e., trigger points) which are preceived by the patient as focal points of muscle tension. As described by Travell and Simons (44), the increased muscle tension will impair blood flow around the contracted muscle fibers, producing a favorable environment for the accumulation of waste products, specifically histamine and serotonin (which are released by degranulated mast cells and platelet cells, respectively). Because histamine and serotonin are neuroirritants, a pain-spasm-pain cycle would begin which theoretically inhibits the return of ATP to the contracted sarcomere. This produces a state of functional rigor mortis in the involved fibrils which perpetuates the cycle. Lee (41) claims these neurologically induced trigger points can be palpated in the segmental spinal musculature as well as the peripherally-related musculature. The author emphasized that because of the neurological origin of the contracted muscle tissue (i.e., not all trigger points result from direct mechanical damage to the sarcoplasmic reticulum), treatment must include restoration of normal cervical biomechanics.

In the second stage of this process, the resultant nerve root hypoxia produced by dural compression damages the capillary endothelium which allows proteins to leak into the endoneural spaces. This may lead to the formation of intraneural scar tissue (via increased fibroblastic activity) and demyelination of specific nerve fibers (39,42). Cannon and Rosenbleuth (45) claim that such demyelination, even when minor leads to an increase in the surface area of portions of the muscle fiber that are sensitive to acetylcholine. The increased surface area leads to a supersensitivity because the muscle may respond with contraction as neighboring motor and even sensory nerves discharge. The involved muscle is clinically detectable by its state of chronic tension and by its accelerated rate of fatigue. In the case of C6 nerve root irritation resulting from compensatory hypermobility of the C5-C6 vertebral segment, the resultant intraneural scar tissue formation will damage the suprascapular nerve (spinal segments C5-C6) which in turn results in a chronic state of tension and increased fatigability in muscles innervated by that nerve; e.g., infraspinatus and supraspinatus. This, of course, can lead to shoulder injury because of decreased muscular stabilization. Also, even slight mechanical irritation of the nerve root will make the entire peripheral nerve more susceptible to injury because of the double crush hypothesis. As stated by Upton and McComas (46), compression at one point along a nerve will make the distal aspects of that nerve more prone to neuropathy because the compressed section allows for a lessened axoplasmic flow to the remaining nerve which results in a decrease in nutritional support and an associated hypersensitivity. The effect of multiple compression points is cumulative.

The third stage of neural damage is rarely seen in competitive athletes as it involves the more advanced stages of denervation in which the normal type I and II

collagen fibers in skeletal and soft tissue are replaced with the weaker, immature collagen fibers. This predisposes those tissues to breakdown with even minor amounts of stress. In his outline of the three stages of nerve damage (which was used to describe the correlation between cervical dysfunction and tennis elbow), Lee (41) emphasized that in order to be effective the treatment program must be geared toward restoring both the peripheral tissue lesion and the primary spinal lesion. The author states that any treatment program aimed solely at restoring function to the contracted muscle filaments (i.e., cross-frictional massage, ischemic compression, spray and stretch, etc.) will be destined to fail unless optimal mechanics and posture are restored to the cervical spine. As demonstrated by Carrick (47), aberrancy of the normal coupled movement patterns present in the cervical spine can be associated with neurological deficits, and chiropractic manipulation of the dyskinetic vertebrae will restore proper biomechanics and allow for both subjective and objective improvement in the clinical picture of the cervical radiculopathy; i.e., improvement in sensation, motor power, and deep tendon reflex. In an earlier study relating peripheral soft tissue lesion to dysfunction in the cervical spine, Gunn and Milbrandt (48) demonstrated that tennis elbow often represented a reflex localization of pain from radiculopathy in the cervical spine and that treatment of the cervical spine appeared to give relief in the majority of patients. It should be emphasized that although initiated by neural irritation, peripheral soft tissue lesions eventually develop a "life of their own" as they are capable of eliciting an exaggerated autonomic response that acts to perpetuate the lesion (42).

EVALUATION AND TREATMENT

Although a few of the more severe types of congenital malformations will require surgical reconstruction if athletes are to continue with their career, the vast majority of these deformities will respond favorably to a well-designed program of conservative care. Keep in mind that a shallow intertubercular groove and/or supratubercular ridge will only produce symptoms if the humerus is maintained in an internally rotated position of function. Nirschl (49) even downplays the significance of developmental deformities such as enlargement of the anterior inferior acromion, hypertrophy of the coracoacromial ligament and/or spur formation at the anterior inferior acromioclavicular joint. The author feels that even in these situations, the primary cause for dysfunction is intrinsic musculotendinous overload and that proper function in the majority of situations can be restored without surgical intervention. Because this is most often the case, such developmental deformities should initially be considered coincidental, not causal.

As described by Pappas et al. (37), comprehensive conservative treatment should incorporate a sequential four stage rehabilitation program designed to 1) restore the active and passive ranges of motion to all joints in the kinetic chain; 2) reestablish synchrony of motion; 3) increase strength and endurance, and; 4) ensure progressive return to the throwing motion. These authors repeatedly emphasize that restoring flexibility is an essential prerequisite to a strengthening program as premature initiation of resistance exercises will only reinforce the existing muscle imbalances and faulty biomechanics. The examination techniques suggested by Pappas et al. (37) include a sitting evaluation to observe shoulder elevation (the injured shoulder is usually lower) and attention to symmetry of scapulohumeral rhythm while the patient is abducting the arms. The authors recommend supine positioning of the patient to evaluate shoulder motions in combined abduction and horizontal flexion. These motions are extremely important to the throwing athlete as they are essential components of the wind-up and follow-through phases, respectively. To test horizontal flexion, the practitioner stabilizes the scapula and moves the humerus medially, maintaining the arm in the transverse plane (Fig. 11). A range of less than 45° is considered significant as it may produce compensatory scapula protraction during follow-through. Combined abduction is tested by stabilizing the scapula after it has shifted to the edge of the thorax (which allows for the normal 60° of scapulo-thoracic motion) and then abducting the shoulder to 150°. If adequate glenohumeral motion is present, the humerus can then be brought

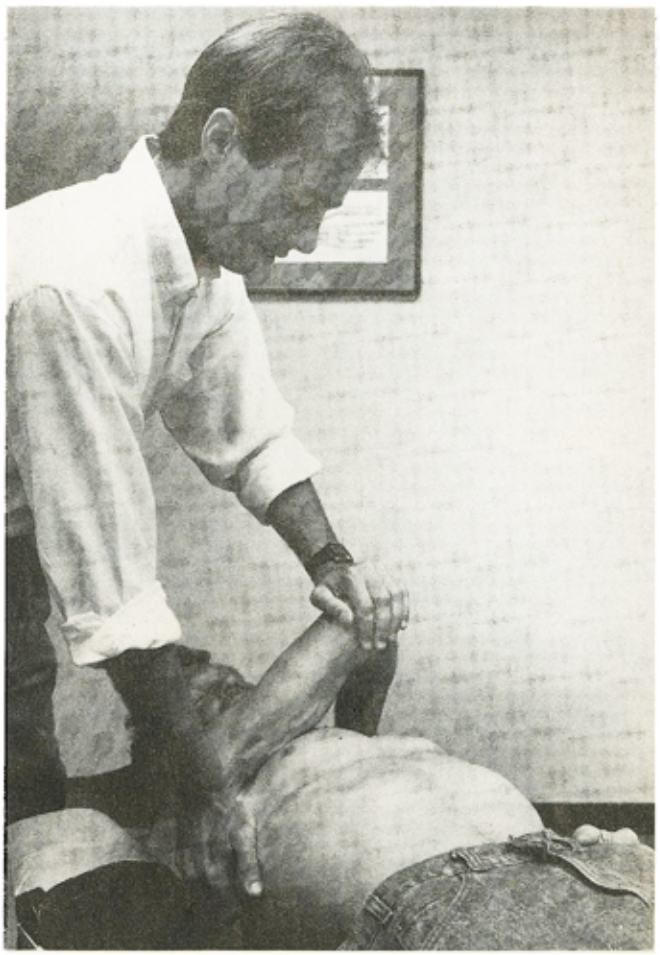

Figure 11. Test for horizontal flexion. Note the examiner stabilizes the scapula to ensure pure glenohumeral motion.

down to the table (Fig. 12). A decrease in this range will disallow the normal acceleration one position which, in turn, results in a lessened velocity of throw with the potential for injury as compensatory motions develop.

If found deficient, these and other ranges can usually be restored thru a combination of static and dynamic stretches. The use of hold-relax stretches and other proprioceptive neuromuscular facilitation (P.N.F.) techniques have been widely advocated because they more quickly increase range of motion (50–53) and improve the ability of the myotendinous junction to resist tensile strains (29). However, some recent studies have demonstrated that maximal resistance hold-relax stretches produce a lingering after-discharge that could detrimentally effect muscle tension (54,55). From personal experience, this after discharge can be minimized by using less than maximal resistance while performing these stretching techniques. While many authors feel that maximum contraction is necessary to stimulate the golgi tendon organ's reflexive relaxation of the agonist (56,57), this has never been conclusively demonstrated. Holt (58) feels that the agonists relaxation following isometric contraction stems not from an increase in information from the golgi tendon organ but rather from a decrease in information from the muscle spindle. Apparently, the isometric contraction somehow lessens the afferent flow of impulses from the spindle complex. The exact mechanism for the lessened discharge remains to be proven.

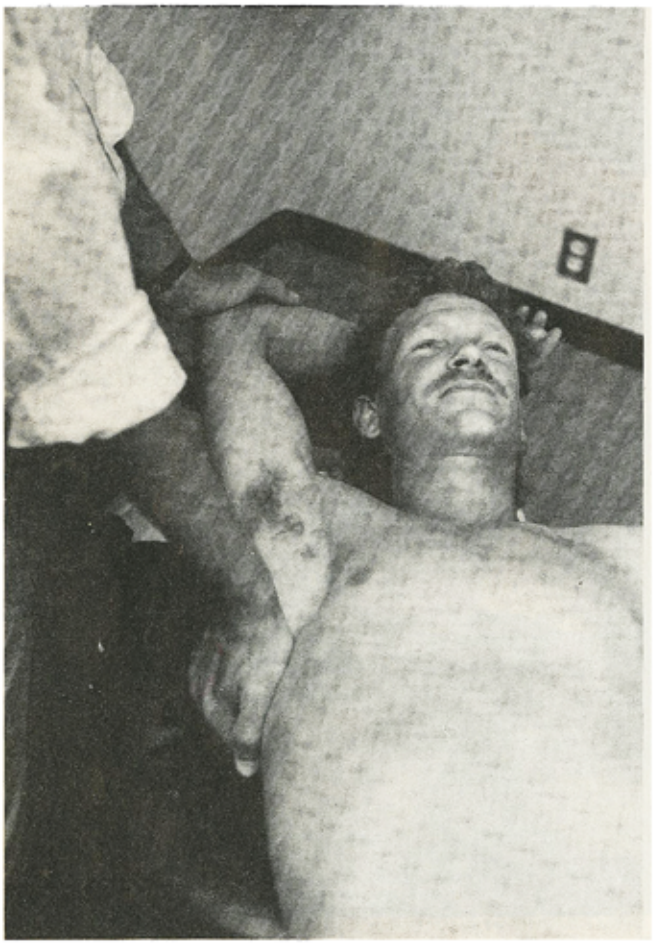

Figure 12. Combined abduction test.

In addition to the use of P.N.F. stretches, the use of conventional static stretches should always be a consideration as a recent comparison of P.N.F. versus static stretches on hamstring flexibility (59) demonstrated that static stretches produced significant reductions in oxygen consumption with corresponding improvements in gait economy. The decreased oxygen consumption during gait was related to an improved antagonist response. The authors state that the "static stretching procedure prepared the subject for more economical gait by applying the end-range stretch in the same plane that the muscles are going to be used." The results of this study suggest that static stretching of the upper extremity musculature may be an effective way to improve endurance in the throwing arm and shoulder.

Regardless of which stretching techniques the practitioner chooses to restore the lost ranges of motion, care must be taken not to overstretch the glenohumeral joint capsule. While horizontal flexion is limited by the chest wall, overzealous stretching of the humerous beyond the horizontal level of the table during the combined abduction stretch can produce a laxity of the glenohumeral joint capsule that may eventually lead to anterior labral tears and/or chronic bicipital groove tendinitis (37).

Although rarely described in mainstream medicine's rehabilitation programs, chiropractic adjustment to the spine and extremities should always be considered if treatment is to be comprehensive. By restoring proper coupled movement patterns to the spine and accessory joint motions to the extremities the adjustments can improve the quantity and quality of afferent information [which may negate an aberrant autonomic response (42)] as well as decrease the need for mechanical compensation elsewhere in the kinetic chain. The impressive work by Carrick (47) and Gunn and Milbrandt (48), clearly demonstrates the importance of a thorough spinal exam when treating any peripheral lesion. The efficacy of treating spinal hypomobile segments in order to improve extremity function has been demonstrated in other studies (60,61) in which mobilization of fixed lumbosacral and sacroiliac articulations produced immediate increases in hamstring power as tested with isokinetic dynamometry. Although not tested, it seems likely that for the same neurological mechanisms, manipulation of a dyskinetic cervical spine could result in immediate improvements in muscular function of the upper extremity.

If shoulder motion is limited by the presence of adhesions at the myotendinous junctions, many practitioners advocate the use of cross-frictional massage. According to Cyriax (62), this technique, which involves vigorous massage applied perpendicular to the direction of the muscle fiber, results in a break-down of the weaker type three collagen fibers thereby allowing for improved flexibility. The author recommends isolating the involved tendon with specific manual muscle tests (the fibrotic myotendinous junction typically produces pain when isometrically tensed). Unfortunately, this method can result in frequent false positive results as many of the test positions create a shearing force which may

produce pain from impingement, not from stressing the fibrotic myotendinous junction. This is particularly true when muscle testing supraspinatus and infraspinatus. Bowling et al. (63) claim that the false positives associated with manual muscle testing can be minimized by "careful performance of procedures and by correlating test results with other portions of the examination." It should be stated that not all practitioners feel cross-frictional massage is a valid technique for restoring function. Carrick (64) claims that such techniques destroy golgi tendon organs and can be responsible for chronic injury secondary to decreased mechanoreceptor function. Hammer (65) disagrees and claims that favorable long-term followup on thousands of cases has failed to demonstrate such damage. On the contrary, he feels that cross-frictional techniques favorably stimulate mechanoreceptors and that the temporary anesthesia associated with such stimulation serves as an excellent tool for differentially diagnosing the involved tissue.

Once the appropriate manual therapies have restored adequate ranges of motion to the involved tissues, the second stage of rehabilitation regarding synchrony of motion can be evaluated. One method of evaluation requires the patient be positioned prone with both arms comfortably hanging off the sides of the table. The patient is then asked to externally rotate and horizontally extend the shoulders while they are positioned at 90° abduction (Fig. 13). This action requires the coordinated contraction of posterior deltoid, infraspinatus, teres minor, the rhomboids, and middle trapezius, and is an important part of rehabilitation as it duplicates muscular and joint interactions during wind-up. A standing version of this motion can be used as a starting exercise as the athlete moves through this pattern at least twice daily with 25 repetitions each time (Fig. 14). If the scapulohumeral rhythm is adequate and movements are symmetrical, the athlete can begin isometric exercises where he moves through all phases of the throwing motion isometrically tensing the torso and upper extremity musculature every few inches for 10 seconds throughout the range. This initiates the physiological response in conditioning that will be important during the later stages of rehabilitation.

These isometric exercises can be initiated simultaneously with supraspinatus setting exercises in which the athlete sits upright in a chair and abducts the shoulder against slight resistance from a partner (Fig. 15). It is imperative that forceful supraspinatus contraction occur in the 30 to 45° range so as to properly depress and stabilize the humeral head within the glenoid cavity. When proper supraspinatus activity is accomplished in the 30 to 45° range, the athlete may continue through a 90° arc of abduction. If pain is felt at anytime during the 90° range of abduction, the athlete should return to the setting exercises in the 30 to 45° range. When the supraspinatus setting exercises can be properly performed throughout a 90° range of abduction, and when the exercise in Figure 14 can be accomplished with distinct contractions noted in all of the shoulder muscles, then and only then can progressive resistance exercises begin. Supraspinatus should be exercised in three different positions: 1) shoulder abduction to 90° with arms straight out to the side and shoulders externally rotated; 2) abduction to 90° with arms in slight horizontal flexion in a position of neutral rotation, and; 3) abduction to 90° with arms at 30° of horizontal flexion and shoulders internally rotated. Infraspinatus and teres minor can be exercised by using the position demonstrated in Figure 13. The patient is instructed to maintain his head and chest on the table while sequentially externally rotating then horizontally extending the shoulders. Pappas et al. (37) suggest that all exercises initially begin with very low weights and high repetitions; i.e., 1 pound weights with 25 repetitions. When a specific exercise can be performed without muscle substitution, the weight can be increased in 1 or 2 pound increments up to a maximum of 5 pounds.

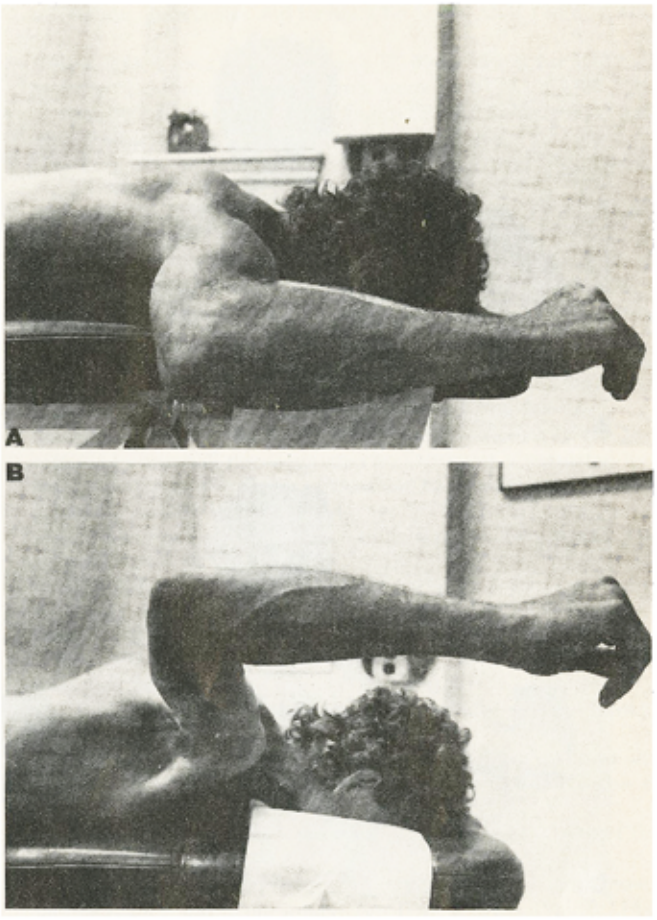

Figure 13. The patient is asked to (A) externally rotate then (B) horizontally extend the shoulder.

Exercises can also be designed to duplicate the stresses of acceleration one and two by having the patient move thru these phases with resistance offered by surgical tubing or a pulley/weight system. This brings up a very important concept regarding rehabilitation. In all situations, the designed exercises should attempt to reproduce in every respect the movements associated with that individuals ideal throwing form. Numerous studies (66–68) have demonstrated that the efficacy of strength training is specific to the angle in which the limb is exercised, the velocity of the limb and the type of contraction used during the exercise (i.e., concentric vs.

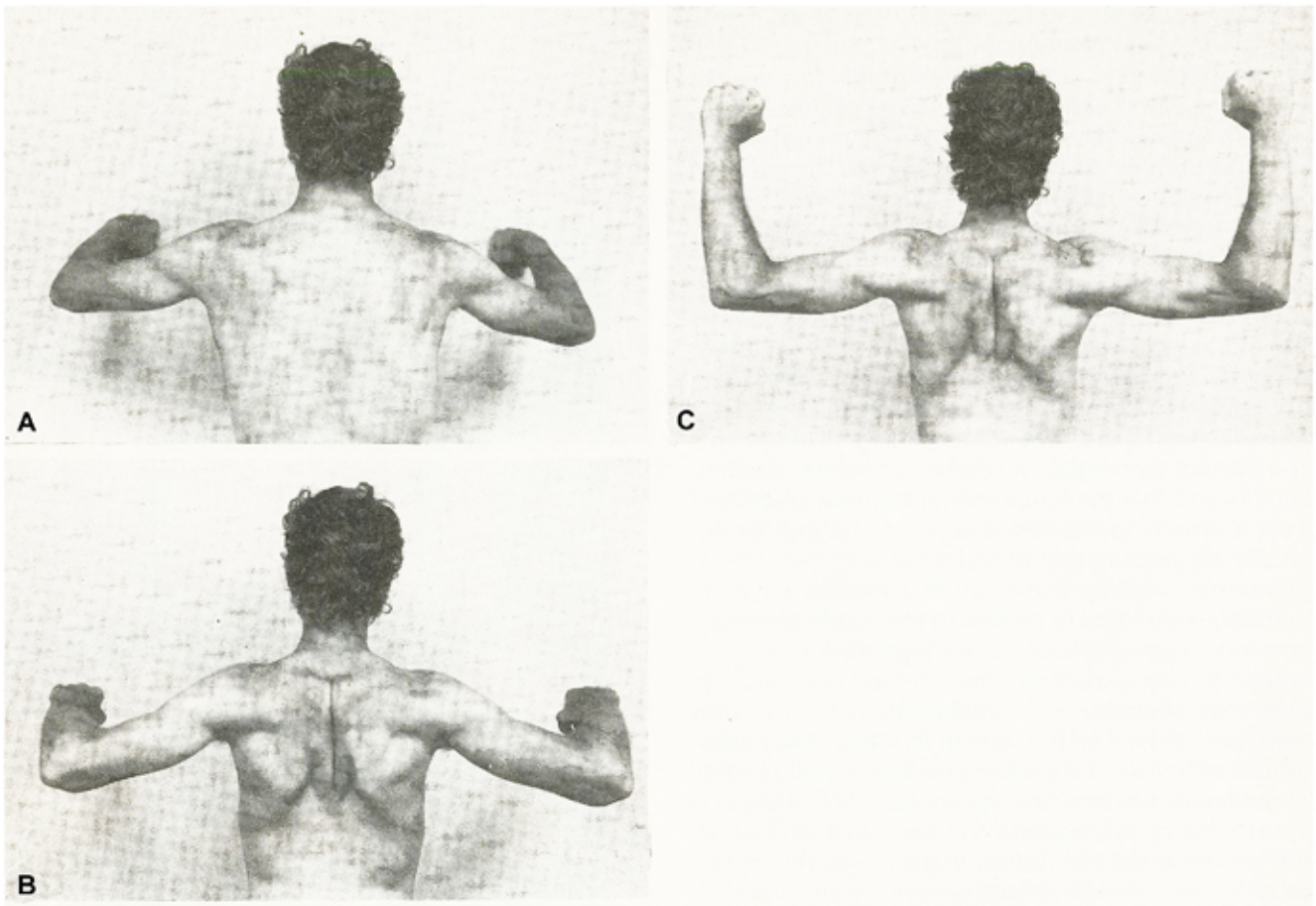

Figure 14. An early exercise requires the patient (A) abduct the shoulders to 90° (B) horizontally extend and (C) externally rotate the shoulders.

eccentric) and that failure to duplicate the patterns used in sport will result in a lessened outcome. This has been demonstrated in throwing athletes as concentric exercises for the shoulder muscles have produced greater ball velocities with tennis serves than eccentric exercises using the same weight and speed (69). Even more impressive is a study by Nelson et al. (70), in which P.N.F. exercises produced a two-fold rate of improvement in the distance of a volleyball throw when compared to a traditional weight training program; i.e., 25% improvement in distance versus a 12.8% improvement after an 8-week training program. Keep in mind that eccentric exercises should always be used to duplicate the motions during follow-through. Because isokinetic dynamometry cannot duplicate the three dimensional movements involved with sporting activities, they are unable to strengthen muscles in their position of function and are therefore less effective (and much more expensive) than a well-designed home exercise program.

The final stage of rehabilitation, progressive return to throwing, should begin with mirror throwing, in which a 1 pound weight is placed in the throwing hand and the athlete is told to move through each phase of the throwing motion in front of a mirror. During this process, the individual is told to concentrate on specific joint interactions during each phase, particularly on the horizontal extension and external rotation occurring during acceleration I. The hand weight used during this exercise provides an inertial force that allows for ideal concentric conditioning of the shoulder internal rotators during acceleration II and eccentric conditioning of the shoulder external rotators during follow-through.

The mirror throwing exercises can be initiated simultaneously with short distance throws in which the athlete can play catch at distances not to exceed 30 feet for 5 to 15 minutes daily. Accuracy, not speed, should be the objective of this exercise. When the athlete is able to throw short distances for 20 minutes without discomfort, long distance arc throwing can begin. This involves slow high arc throws with gradual increases in distance from 30 to 150 feet. Again, accuracy and not speed should be emphasized as the athlete should concentrate on the fluidity of the motion.

The final stage of rehabilitation involves progressive increases in speed which should be monitored on an individual basis. Return to competitive sport should not be considered until the prior level of performance has been achieved without discomfort. Throughout the final phase of rehabilitation, full body exercises should be initiated, which may include any of the popular trunk and extremity strengthening machines, rowing, running, push-ups, and/or swimming. Such exercises should be

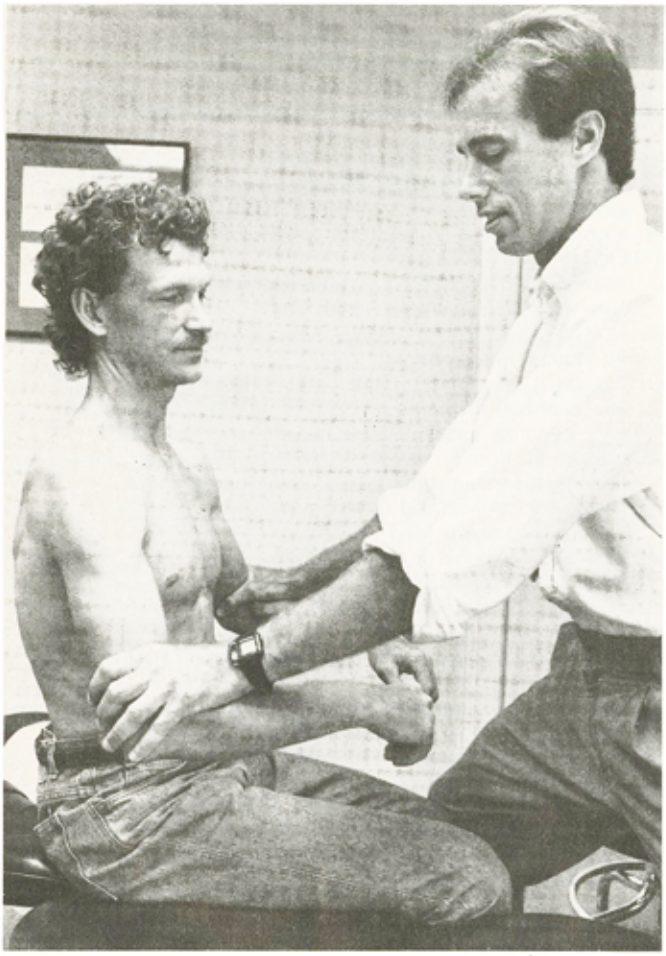

Figure 15. Supraspinatus setting exercise.

continued throughout the season. Lastly, flexibility should be maintained as the athlete continues with a well-designed stretching routine both before and after exercise.

CONCLUSION

It is hoped that this article demonstrates that a thorough evaluation of the entire kinetic chain is necessary if the throwing athlete is to achieve optimal performance. This evaluation should include a chiropractic evaluation of the spine and extremities, along with a full body evaluation of flexibility, strength, and endurance. The importance of a sequential rehabilitation program beginning with the restoration of flexibility and ending with a progressive return to sport is recommended as essential.

Acknowledgements

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