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Musculoskeletal Injuries in Tennis

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It is estimated that tens of millions of people play tennis in the United States [1,2]. More than half a million are adolescents [3], and growing number of seniors continue to play tennis [4]. The results of epidemiologic studies in tennis players have shown some variability in injury patterns [5–9]. The most common types of injury in tennis players of all ages are muscle and ligament strains or sprains secondary to overuse. These are a particular problem in the adolescent group, because, in general, this group begins playing with a lower level of physical conditioning. Injuries in the younger players are usually not long-standing, and the overuse (chronic) problems seen in the older players, such as patellar tendinosis and tennis elbow, are less common in younger players [10]. Lower limb injuries are twice as common as those of the upper limb or spine in young players, with ankle injuries being the most common [10]. Injuries to the back, neck, and groin occur at a rate roughly equal to that of upper extremity problems [11].

Virtually all the previous studies evaluating patterns of injuries in young tennis players have focused on male players [5,6,8,9,12]. The number of women participating in sports, including tennis, has risen dramatically since the 1970s, however. In general, injury patterns are observed to be more sports specific than gender specific. Nevertheless, female athletes do tend to have more frequent patellofemoral problems, have less upper extremity strength, and begin competition at a lower level of physical conditioning than male athletes [10].

A preliminary study by Kibler and Safran [11] compared injury patterns in elite junior athletes during a 3-year period (1996–1998) at the United States

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Tennis Association (USTA) Tennis Championships. Sixteen- and 18-year-old boys sustained more new injuries during the USTA National Hard Court Championships compared with 16-year-old girls at the USTA Girls' National Championships over the 3 years studied. There was no significant difference in the overall rate of injury (new and recurrent) between boys and girls, however. Boys and girls had similar incidence and prevalence rates of lower extremity injury; however, the number of lower extremity injuries was disproportionately greater in girls compared with boys. Injuries to the abdomen, back, and groin were significantly fewer in female athletes compared with male athletes. Girls had more injuries to the foot, leg, calf, and wrist. Boys sustained more injuries to the ankle, groin, and hand. Boys and girls had a high rate of injury to the back and shoulder. For boys and girls, strains predominated, followed by inflammation and then sprains. Boys had a greater incidence of contusions, abrasions, and lacerations.

Shoulder injuries

Tennis players are susceptible to developing disorders of the shoulder because of the repetitive nature of the game. The most common overuse injury in the upper extremity in young tennis players involves the shoulder, with rotator cuff inflammation being one of the most common injuries in tennis players of all levels [10]. In one study by Lehman [13], shoulder pain was present in 24% of 270 tennis players between the ages of 12 and 19 years. The incidence of shoulder pain increased to 50% in middle-aged tennis players. Overhand throwing and racquet sports place repetitive high-velocity stress on the shoulder joint, gradually resulting in subluxation or movement of the humeral head anteriorly. This subtle instability may lead to recurrent subluxation and impingement of the rotator cuff [14].

Athletes may present with pure and isolated impingement without instability. This is usually seen in older athletes and is considered secondary to degenerative changes within the joint itself. Impingement may also be associated with instability caused by repetitive microtrauma on the labrum and the capsular ligaments. Inherent hyperelasticity can lead to instability and impingement as well. Ultimately, impingement can progress to rotator cuff tears [14].

In contrast to the older player with rotator cuff impingement and degenerative changes, the young tennis player's symptoms are more commonly secondary to instability in the glenohumeral joint [15]. A loss of strength in the external rotators and scapular stabilizers accompanied by a loss of flexibility in the internal rotators has been associated with instability [16–18].

Neer [19] has classified the impingement syndrome into three stages of increasing severity. Stage 1 is generally seen in athletes less than 25 years of age and is characterized by edema and hemorrhage of the subacromial bursa. Stage 2 is seen in athletes older than the age of 25 years, with fibrosis

and scarring of the subacromial bursa. Stage 3 impingement is seen in athletes older than the age of 40 years and involves the rotator cuff, which may be torn [19–21]. Generally, most patients with stage 1 and 2 impingement respond well to conservative treatment. Patients who have failed conservative treatment or have advanced stage 2 or stage 3 impingement may benefit from subacromial decompression [22].

Initial evaluation for tennis players begins with a complete history. It is important to understand the chronology of the symptoms, previous treatment, and training schedule of the player. The amount of time practicing each stroke should be documented, with special focus on the time spent practicing serving, overhead smashes, and volleying [13]. The service and the overhead smash are the strokes that tennis players most commonly cite as being responsible for the onset of shoulder pain [23]. It is also important to ask about neurologic symptoms and mechanical complaints, such as catching and clicking. The sensation of a ''dead arm'' may be related to instability [24]. Shoulder pain may also be referred pain from cervical spinal disorders, such as a cervical herniated disc and cervical radiculopathy.

Physical examination begins with inspection of the shoulder with a comparison with the asymptomatic side to evaluate for atrophy and asymmetry. Tennis shoulder may be observed, which is a drooped and internally rotated shoulder caused by stretching of the large shoulder elevators: rhomboids, levator scapula, and trapezius [25]. Inspection of the posterior aspect of the shoulder should also be performed to observe the scapula stabilizers and to assess scapular protraction [26]. Palpation of the shoulder should be complete, including the sternoclavicular and acromioclavicular joints, clavicle, long head of the biceps tendon, deltoid insertion, greater tuberosity, scapula, and acromion [26].

Range of motion of both shoulders should be evaluated. The dominant shoulder in tennis players usually has decreased internal rotation and increased external rotation, which is related to posterior capsular tightness. One study in cadavers showed increased anterior translation with posterior capsule tightness [27]. Strength testing of the shoulder in abduction, flexion, extension, and internal and external rotation should be performed. Isolation of the supraspinatus can be performed with the arm elevated to 90% in the scapular plane with internal rotation of the humerus (Fig. 1) [26].

Instability of the shoulder should be evaluated. One should inspect for a sulcus sign, which determines the degree of inferior translation. An anterior and posterior apprehension test as well as a load-and-shift test should be performed with the patient in a supine position to evaluate for the degree of instability [26]. Impingement should be checked with the Neer's impingement sign (Fig. 2) and the Hawkins-Kennedy impingement test (Fig. 3). To evaluate for biceps tendon pathologic changes, the Speed's test (Fig. 4) and Yergason's test are helpful.

If a rotator cuff tear is suspected, there are several physical examination findings that could be present. There may be supraspinatus wasting.

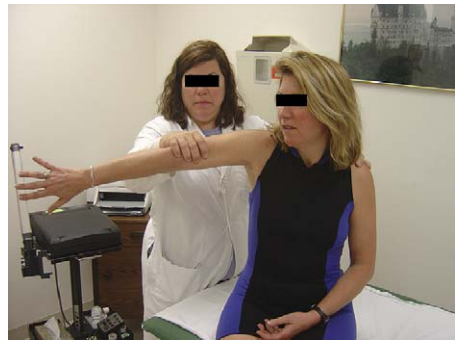

Fig. 1. Supraspinatus or scaption test. The examiner is isolating the supraspinatus by having the patient abduct her shoulder in the scapular plane with internal rotation of the humerus. The patient is abducting her shoulder against resistance.

A painful arc of motion at 90° of abduction and flexion may be seen. Patients typically have a greater passive than active range of motion. Patients may have a positive ''drop arm'' test (Fig. 5). Weakness in abduction and external rotation is also commonly seen [28].

Radiograph testing should include anteroposterior (AP), lateral, and axillary views of the scapula. If the distance between the undersurface of the acromion and the superior surface of the humeral head is less than 6 mm, a rotator cuff tear should be suspected [29]. MRI is the now the ''gold standard'' test in diagnosing shoulder disorders in athletes because it allows visualization of labral, chondral, and rotator cuff pathologic findings [26].

The differential diagnosis of shoulder pain in tennis players most commonly includes subacromial impingement, glenohumeral instability,

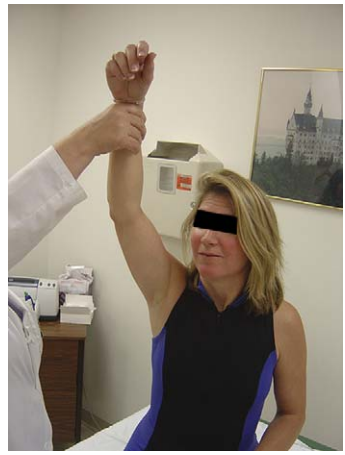

Fig. 2. Neer's impingement sign. With the patient seated, the shoulder is moved passively through forward flexion. Shoulder pain indicates a positive sign.

Fig. 3. Hawkins-Kennedy impingement test. The patient's shoulder is flexed to 90°, and the examiner is forcing the shoulder in an internal rotation without resistance by the patient. Local pain suggests supraspinatus tendinitis.

internal impingement, and rotator cuff tendinosis and tear. Other causes of shoulder pain that should be considered include biceps tendinitis, acromioclavicular (AC) joint arthritis, labral pathologic change, and chondral injury. Cervical disc disease should also be considered, because it can refer pain to the shoulder [26].

Treatment of most overuse shoulder injuries is usually conservative. It includes avoidance of the inciting incident, nonsteroidal anti-inflammatory

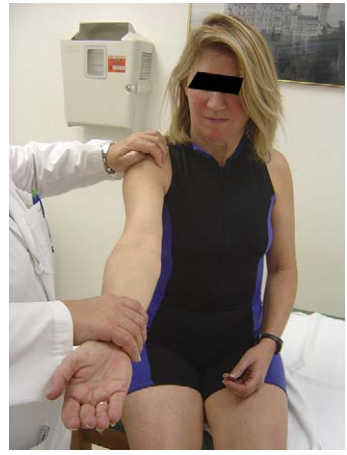

Fig. 4. Speed's test. The patient's elbow is fully extended and supinated, and the shoulder is flexed forward to 45°. The examiner has her left hand on the bicipital groove and her right hand on patient's wrist. The patient is instructed to elevate her arm forward against the examiner's resistance.

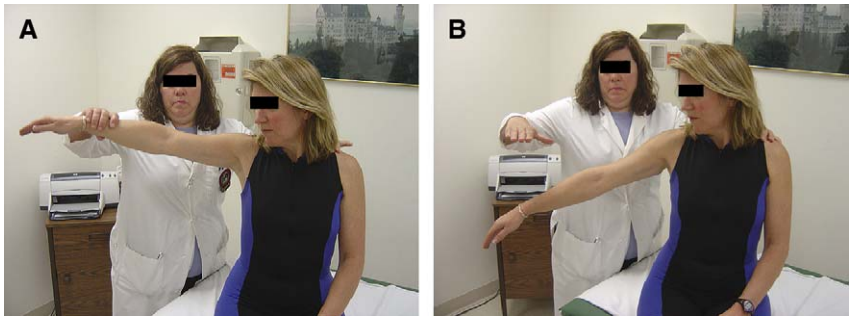

Fig. 5. (*A*) Drop arm test. The patient is seated, and the examiner is abducting patient's arm past 90°. (*B*) Examiner then asks the patient to lower the arm slowly. If there is a tear in the rotator cuff, the arm should fall suddenly to patient's side.

drugs (NSAIDs), and intensive physical therapy. The therapy focuses on strengthening the internal and external rotators of the shoulder using isometric, isokinetic, and isotonic exercises [14]. Proper emphasis must be placed on educating the patient about altered shoulder mechanics and the importance of the rehabilitation. The goal of treatment is to restore the normal mechanics of the shoulder and for the player to be pain-free [30]. Patients who do not respond to therapy may benefit from a local steroid injection into the shoulder. It is recommended that the number of injections be limited to no more than three and that there be a minimum of 3 months between injections [29].

Surgery may be indicated in patients with stage 2 impingement who have not responded to conservative treatment within 3 to 6 months [28]. Early surgical intervention in these patients has been shown to improve function, decrease pain, and prevent progression to complete tears. Even rotator cuff tears in tennis players have been shown to have good clinical outcomes with surgery. In one study, 83% of patients achieved a good or excellent result and were able to return to their presymptomatic level of play without pain [31].

Elbow injuries

Lateral epicondylitis

Lateral epicondylitis (tennis elbow) is extremely common among tennis players. It is estimated to occur with a frequency of up to 50% [32]. Aggravating factors include leading with the elbow during the backhand tennis stroke (Fig. 6) and premature trunk rotation [26,29,32,33]. Novice tennis players with poor technique repetitively extend their wrist on the backhand stroke, leading to lateral epicondylitis. Racquet weight and size also contribute to the development of lateral epicondylitis [26]. Etiologic factors include age older than 35 years, high activity level (three

Fig. 6. Poor technique on the backhand stroke, with the elbow leading ahead of the stroke. This is likely to lead to lateral epicondylitis as more force is placed on the elbow when the racquet strikes the ball.

times or more per week or 30 minutes or longer per session), and demanding activity technique [34].

The differential diagnosis of lateral epicondylitis includes radial tunnel syndrome and posterior interosseous nerve entrapment. These entities can be ruled out with a thorough physical examination and electromyographic testing [34,35]. Other diagnoses that should be considered in the differential are C6 to C7 nerve root compression, posterior impingement with olecranon osteophytes, and radiocapitellar osteoarthritis [32,33].

The pathophysiology of lateral epicondylitis is related to an overuse injury that results in microtearing of the extensor carpi radialis brevis and sometimes the extensor digitorum communis [32]. This begins the inflammatory stage, which leads to the formation of granulation tissue and adhesions at the site of the tear, resulting in pain when tension is put on the region [32,36].

Typical physical examination findings include tenderness and pain to palpation over the lateral epicondyle and extensor tendon origin, pain with resisted wrist extension, and pain with gripping motions [29,32,33,36]. Plain radiographic studies may demonstrate osteophyte formation at the epicondyles, degenerative joint disease, loose bodies, or fractures. Ultrasonographic studies may demonstrate thickening and heterogeneous echogenicity. MRI is useful for demonstrating degenerative thickening of tendons, fibrovascular proliferation, and mucoid degeneration [37].

Treatment consists of nonoperative and operative techniques, of which nonoperative treatment has been found to be successful in relieving symptoms in up to 90% of patients [33,34]. Nonoperative treatment includes rest from aggravating activities, followed by improvement of performance techniques and passive modalities, such as ice, deep friction massage,

bracing, stretching, and sometimes corticosteroid injections. Continued rehabilitation includes a progressive stretching, flexibility, and strengthening program with the goal of a gradual return to the sport with pain-free activity [32,34–36].

Operative treatment is recommended only if symptoms have not resolved with proper nonoperative treatment after 6 months [33]. Several surgical techniques are described, including arthroscopic techniques, all of which involve release of the extensor carpi radialis brevis origin from the lateral epicondyle with or without excision of the pathologic tissue [26,33,34]. After surgery, the same rehabilitation principles apply, with return to competitive sports in 4 to 6 months [33].

Medial epicondylitis

Medial epicondylitis occurs much less frequently than lateral epicondylitis in tennis players [33,36,38]. It is caused by repetitive stress and subsequent muscular overload of the flexor-pronator musculature resulting from overhead tennis serves [29,36,38]. Degenerative changes are usually seen in the pronator teres and flexor carpi radialis muscles, although changes have also been seen in the palmaris longus, flexor digitorum superficialis, and flexor carpi ulnaris [38].

The differential diagnosis of medial epicondylitis includes medial collateral ligament tear, medial elbow instability, and ulnar nerve pathologic change [26,29,33,38]. Again, as in lateral epicondylitis, a careful physical examination and electromyographic studies can aid in a definitive diagnosis. Radiographic studies demonstrate findings similar to those seen in lateral epicondylitis [37]. Physical examination findings typically include insidious onset of pain at the medial epicondyle and tenderness with resisted wrist flexion and forearm pronation [26,29,33,38].

Treatment for medial epicondylitis is similar to that used for lateral epicondylitis. Operative treatment is only considered if there is failure of nonoperative treatment after 3 to 6 months. Unlike the surgical techniques for lateral epicondylitis, those for medial epicondylitis do not include arthroscopic techniques because of the close proximity and potential for damage to the ulnar nerve and ulnar collateral ligament [38].

Wrist injuries

Overuse injuries of the wrist are common in tennis players because of the amount of motion that involves this joint during the tennis stroke [39]. Most wrist injuries in tennis players are attributable to overuse, but direct trauma or an acute rotation episode can lead to wrist pain. Thus, it is important to know the mechanism of injury as well as how long the symptoms have been present and whether they have progressed [26].

De Quervain's tenosynovitis

De Quervain's tenosynovitis is one of the most common tendon problems seen in a tennis player [40,41]. This involves inflammation of the tendons of the abductor pollicis longus and extensor pollicis brevis as they pass through the fibro-osseous tunnel at the level of the radial styloid. Ulnar deviation, particularly with grasp, can lead to inflammation and pain in this area [26]. Physical examination reveals point tenderness over the radial styloid and the first dorsal compartment. A positive Finkelstein's test is pathognomonic for the condition (Fig. 7) [41].

Treatment of De Quervain's syndrome involves splinting and NSAIDs, which generally result in an 80% reduction of symptoms [6]. Should these fail, an intracompartment injection of corticosteroid is usually curative; in rare cases, surgical release is required [42]. Anticipated return to play may be 8 weeks after decompression.

Extensor carpi ulnaris tendinitis

The most common wrist complaint in tennis players is ulnar wrist pain, and this is frequently attributable to extensor carpi ulnar tendinitis. Treatment involves splinting, rest, NSAIDs, occasional corticosteroid injections into the sheath, and attention to technique modification [41]. There are also reports of recurrent dislocation of this tendon. Surgical reconstruction of the sixth extensor compartment fibro-osseous tunnel is recommended for this condition [42,43].

Triangular fibrocartilage injury

The combination of impaction and rotation that occurs with the tennis stroke can lead to triangular fibrocartilage complex injury. Clinically, the patient has pain in the area of the ulnar styloid that worsens with loading

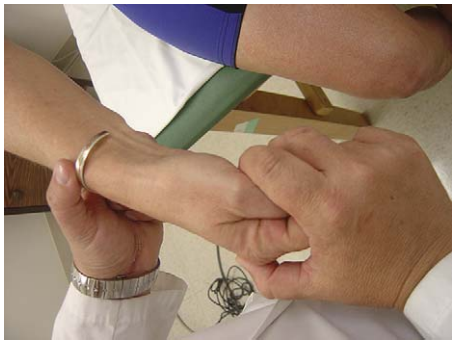

Fig. 7. Finkelstein's test. The thumb is adducted into the palm, and the wrist is passively ulnarly deviated. Pain over the first dorsal compartment is pathognomonic for De Quervain's tenosynovitis.

of the ulnocarpal joint. Plain radiographs are negative. MRI helps to confirm the diagnosis [26]. If nonoperative management with splinting, rest, and NSAIDs is not successful, arthroscopic debridement or repair is indicated [44,45].

Hamate fracture

Fracture of the hook of the hamate is an injury seen less commonly in tennis players [46]. When the grasp is relaxed or the centrifugal force of swinging the racquet overcomes the grasping power, the butt end of the handle can strike and fracture the hook [26]. The patient's history is useful to determine whether the pain was first experienced while swinging the racket. The patient may be able to grip the racket but has difficulty in swinging the racket. Plain radiographs are usually not helpful in diagnosing this entity, although a carpal tunnel view may show the lesion. CT is used to confirm the fracture [41].

The options for treatment are cast immobilization versus excision of the fragment. Many authors recommend early excision of this fragment because it often leads to nonunion despite proper immobilization [46]. Displaced fractures are best treated with excision to avoid this complication [47]. If unrecognized, this injury can progress to painful nonunion, which can also be treated successfully with excision [48]. Avascular necrosis has been reported after this injury [49].

Back injuries

Low back pain is common in athletes and nonathletes alike. Eighty percent of persons are estimated to be affected by low back pain during their lifetime. Most recover within 1 month of symptoms, and less than 4% have chronic pain for more than 6 months [50]. Among active athletes, back pain has been reported at an incidence of up to 85%, suggesting a relation to sports activity [51–57].

In one report, 38% of 148 professional tennis players missed a tennis tournament because of low back pain. Forty-three players reported chronic low back pain, and 11 of 38 players with acute injuries had injuries to the lumbosacral spine [58]. In a retrospective study of 631 injured athletes who presented to a sports medicine clinic over 8 years, 21% were tennis players. Forty-three percent of the tennis players were diagnosed with lumbar disc prolapse; however, no supportive radiographic or clinical data are presented [59].

Another study by Sward et al [60] involved 30 elite tennis players between the ages of 17 and 25 years who were selected at random without knowledge of previous or present back injuries or symptoms. Fifteen patients reported a history of thoracolumbar back pain of at least 1 week's duration; 30% had moderate pain, and 20% had severe pain. Fourteen players had abnormal

plain film radiographic changes, but these changes did not correlate with a history of low back pain [60,61].

The research available to date suggests that back pain is common in tennis players of all ages. Whether tennis players are at a greater risk of low back injury than the general population or other athletes (other than football players, wrestlers, and gymnasts) is unknown [62].

Lumbar strain

Acute lumbar strain is the most common back injury in tennis players. The muscles at risk in lumbar strain are the erector spinae and multifidus muscles because of repetitive trunk extension and rotation as well as the abdominal muscles from repetitive trunk flexion and rotation. These posterior and anterior muscle groups work synergistically in an alternating concentric-eccentric fashion, allowing smooth trunk movement for the extremes of extension-rotation to flexion-rotation [62].

The typical presentation for lumbar strain is unilateral or bilateral low back pain, often with painful paraspinal muscle spasm. Frequently, the player gives a history of change of intensity or duration of play or a recent change in technique. Pain develops suddenly or progressively over several hours. Pain is usually localized to the back and does not refer. Walking and sitting are uncomfortable but not unbearable [62]. On physical examination, there is typically tenderness over the paraspinal muscles. There may be pain with lumbar range of motion. Lumbar flexion may be painful and limited, from paraspinal and hamstring muscle spasm or from baseline inflexibility. Lumbar extension is not usually pain limited; if pain is severe with extension, other diagnoses should be considered, including facet disease, spondylolysis, and lumbar disc disease [62]. Straight leg raising often reveals taut hamstrings but is negative for radicular symptoms. The results of neurologic examination are normal.

For straightforward presentations of lumbar strain, diagnostic imaging studies are not indicated. For patients who present with subacute or chronic back pain, recurrent pain, or clinical symptoms and signs that are atypical for acute lumbar strain, plain films of the lumbosacral spine should be obtained. MRI may also be warranted. A bone scan should be obtained in young patients with normal plain films who have increased back pain with extension so as to rule out spondylolysis [62].

In acute uncomplicated lumbar strain, initial treatment includes relative rest and pain relief. Local ice application may send impulses that compete with slower pain-producing impulses; muscle spasms may also be temporarily relieved, which allows stretch of the muscle to reduce spasm [50]. Other modalities, such as ultrasound and massage, may complement this treatment approach. NSAIDs provide further pain relief and are generally only needed short term. Once pain is relieved, gradual flexibility, followed by a strengthening protocol, should occur. There should be emphasis on strengthening the

shoulder and lower limbs as well so that the kinetic chain does not have a weak link that places further strain on the lower back muscles. The athlete could return to play within several days of an acute lumbar strain [62].

Lumbar disc degeneration and herniation

Acute lumbar disc herniation may result from a sudden overload of the intervertebral disc with a subsequent acute annular tear. The more common disk pathologic findings seen in tennis players result from repetitive microtraumatic loading of the lumbar disc, especially with repetitive hyperextension and rotation. In this model, lumbar disc degeneration and herniation can be viewed as a continuum of the same process [62].

The stroke placing the greatest stress on the low back is the serve [58]. The lumbar spine hyperextends and rotates with the hitting arm away from the net during the toss (Fig. 8). The trunk then powerfully laterally flexes, and the shoulders and trunk rotate toward the net as forward flexion occurs. It is thought that the serve may place the lumbar disc at increased risk of annular tears because of repetitive rotational forces applied to the discs, especially when coupled with hyperextension. Hypertension increases the shearing effect of the caudal lumbar discs [50]. Repetitive trunk rotation and hyperextension shear the annulus, which can result in an annular tear [62].

Tennis players with an acute lumbar herniated disc or annular tear may present with back pain only, leg pain only, or a combination of the two. The player usually recalls the sudden onset of a distinctly uncomfortable feeling in the low back. There may be a prior history of episodic sudden low back pain lasting days, with asymptomatic periods between such attacks. The

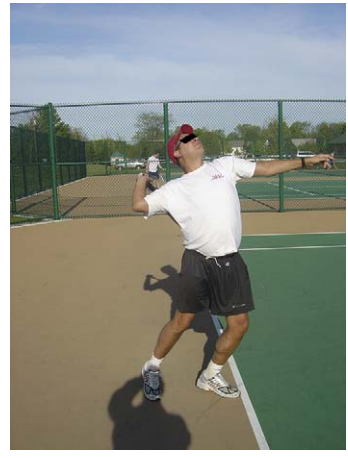

Fig. 8. Of all the tennis strokes, the serve causes the most stress to the back. Repetitive hyperextension occurring during the toss phase of the serve may ultimately lead to annular tears of the disc as well as to facet impingement.

onset of pain may not always follow vigorous activity and may follow a change in normal routine by hours or days. This feeling may persist for hours or days and then may suddenly become severe, with or without referred leg pain [62]. Patients are generally uncomfortable sitting and walking. Patients may reduce their pain by lying with the knees flexed or standing with one leg elevated. Pain may increase with position changes or the Valsalva maneuver [62].

On physical examination, the patient may exhibit a lumbar list, with spinal curvature concave to the side of pain. Lumbar flexion is usually pain limited. Lumbar extension is usually pain-free if the facets are not compromised. Straight leg raising is usually positive ipsilateral to the side of the pain. The results of neurologic examination are often normal unless there is significant nerve root compromise [62].

If disc pathologic change is strongly suspected, plain radiographs and MRI should be obtained. Plain films may reveal spondylolisthesis or subtle instability with flexion and/or extension views [62]. Plain films are usually normal in acute lumbar disc herniation. MRI can demonstrate acute, subacute, and chronic disc changes and reveals the relation of any disc pathologic change to the spinal canal, neural foramen, and nerve roots [62]. If the tennis player has sensory or motor deficits on physical examination, electrodiagnostic studies should be performed.

Initial treatment in players with lumbar disc disease includes rest and pain control with NSAIDs. In patients with severe pain from acute lumbar disc herniation, oral or epidural corticosteroids may provide further pain relief [63,64]. Opioid medications may also be given short term in players with severe pain. Physical therapy should include proper trunk and abdominal flexibility and strengthening exercises as well as ways to unload the lumbar disc. Lumbar stabilization techniques in which the athlete is trained to become aware of the lumbar spine position during all body positions and exercise routines are extremely important. If the athlete does not correct faulty body mechanics or stroke mechanics, he or she is likely have a recurrence of the injury [62].

Facet impingement and arthropathy

In a normally functioning lumbar spine, the facets should not approximate in a weight-bearing manner. Although acute hyperextension may cause facet impingement, a healthy intervertebral disc should prevent this from occurring [65]. Facet impingement and facet arthropathy are usually a continuum of lumbar degenerative disc changes, thereby allowing approximation of these posterior elements. When the disc becomes narrowed, up to 70% of the compressive force usually applied to the disc is transferred to the facet joints. Players with lumbar degenerative disc changes who hyperextend their lumbar spine while serving are at an increased risk of acute facet impingement and chronic facet arthropathy [62].

Players with facet impingement usually present with low back pain that may refer to the buttocks region. Referral of pain into the legs does not usually occur unless there is significant disc pathologic change or foraminal narrowing as well. Acute hyperextension may precipitate facet impingement; however, the player usually gives a history of previous back pain, and he or she may have had several bouts of back pain with asymptomatic periods. Physical examination may reveal spinal curvature convex on the side of the pain. Lumbar extension may exacerbate the pain. A straight leg raise test is negative, and the results of the neurologic examination are normal [62].

The diagnostic workup should begin with plain films of the lumbar spine, which should include oblique views for best visualization of the facet joints. MRI is also valuable because it can demonstrate the extent of facet disease and also evaluate discogenic pathologic change. Treatment for facet disease, which is usually a continuum of lumbar disc pathologic change, should be treated similar to lumbar disc disease. Strict attention should be directed to avoid hyperextension and rotation so that the facets do not assume a weight-bearing role [62].

Knee injuries

Knee injuries are also common in tennis players because of the sharp side-to-side movements required. The most common injuries seen include patellofemoral dysfunction, jumper's knee, meniscal injuries, and bursitis [66].

Patellofemoral dysfunction

Patellofemoral dysfunction is a common cause of anterior knee pain that is produced by mechanical overload and abnormalities in the knee extensor mechanism. Other factors involved are general ligamentous laxity and an abnormal limb alignment or Q angle [29,66]. The Q angle, which is the difference between the resultant line of pull of the quadriceps muscle and the anatomic position of the ligamentum patellae, is normally 13° to 18°. When this angle is increased, it contributes to the development of patellofemoral dysfunction.

Pain often presents insidiously and is described as dull, aching, and difficult to localize. It is usually located anteriorly around the peripatellar region, although it sometimes radiates into the retropatellar region. Initially, the pain resolves after the activity is stopped; however, with increasing severity, the pain remains after the activity and is eventually present continually. There is sometimes a sensation of weakness in the quadriceps reported by the patient [29].

On physical examination, there may be atrophy of the vastus medialis and tight hamstring muscles. A positive patellar inhibition test is pathognomonic of patellofemoral dysfunction. The test is performed with the patient supine and the leg extended. The examiner pushes the patella distally in the

trochlear groove. The patient is instructed to contract the quadriceps while the examiner continues to apply resistance against patellar excursion. In cases of patellofemoral dysfunction, there is pain with movement and the patient relaxes the leg immediately. The medial and lateral retinaculum should be examined for tenderness. Hypermobility of the patella should be checked. The apprehension test is useful in diagnosing acute patellar subluxation or dislocation. In an acute patellar dislocation, there is usually a hemarthrosis present [29,66].

Radiographic examination is useful in ruling out other bony pathologic findings and should include standing AP, lateral, tunnel, and skyline views [66]. Rehabilitation should include a stretching program that targets the iliotibial band and hamstrings. Strengthening should focus on the quadriceps. Patellar taping may also be done. Surgery should only be considered after 8 to 9 months of conservative care, because realignment of the patellofemoral joint is considered a major procedure [29,66].

Jumper's knee

Jumper's knee is another term for patellar and quadriceps tendinitis. It is an overload injury that occurs in sports that require repetitive jumping or bending. The repetitious activity results in fatigue type tissue microtearing [66]. The presentation and progression of pain follow the same pattern as that of patellofemoral dysfunction, except that tendon rupture may occur in the final stage [66].

Physical examination reveals point tenderness and occasional swelling over the involved area. There are no palpable defects found on physical examination. There may be quadriceps atrophy and patellar hypermobility present on examination. Plain radiographs may demonstrate a number of changes, including cystic radiolucency or irregularity at the inferior pole of the patella, calcification at the tibial tubercle, or a bony avulsion at the superior pole of the patella, among other findings. Ultrasonography is useful for showing tendon thickening, decreased echogenicity, and calcification of the tendon. MRI is useful for demonstrating chronic degenerative changes of the tendon [37,66]. Treatment is typically conservative and includes activity modification, passive modalities for pain, and quadriceps strengthening exercises. Surgery is only rarely indicated and usually reserved for cases of tendon rupture [66].

Meniscal injury

Meniscal injuries usually occur when the athlete is standing on the leg in a semiflexed position and a torsional stress is applied to the knee, as happens during pivoting. The medial and lateral meniscus may be injured, although involvement is usually limited to the posterior horn. Initial presentation includes pain over the affected side of the knee and swelling that is usually

serous in nature. The patient may complain of buckling of the knee and joint locking [66].

Physical examination may reveal joint line tenderness, quadriceps muscle atrophy, and positive McMurray (Fig. 9) and Apley (Fig. 10) tests. Plain film radiographic evaluation should include standing AP, lateral, tunnel, and skyline views. MRI is useful for demonstrating soft tissue details [37,66].

Rehabilitation is initially conservative (eg, rest, passive modalities for pain, crutches, bracing), followed by progressive stretching and strengthening of the quadriceps. After the patient has full pain-free range of motion, no evidence of mechanical blockage, is nontender, and has full strength, he or she can begin jogging in a straight line and progress to running in a straight line, followed by cutting as tolerated. Conservative management is not appropriate in cases of frank locking, which is usually indicative of a ''bucket-handle'' tear. Postoperative rehabilitation follows the same principles as in conservative rehabilitation, with the exception that isometric exercises for the quadriceps and hip abductors and adductors are instituted immediately after meniscectomy [66].

Bursitis

Bursae are membranous sacs that serve a protective function in the knee joint, diminishing the friction between two adjacent structures as they slide over each other. Bursae may become irritated and inflamed by constant friction during overuse or by acute trauma [66].

Bursitis usually presents at the anteromedial proximal tibial metaphysis and the posteromedial corner of the knee. The differential diagnosis includes meniscal injury and medial collateral injury. Physical examination reveals

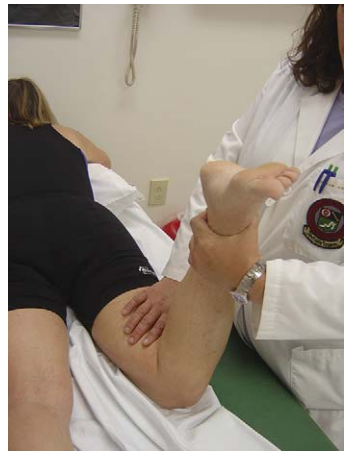

Fig. 9. Apley's test. The patient is prone, and the knee is flexed to 90°. The examiner is grasping the ankle and placing downward pressure while internally and externally rotating the leg. Pain or crepitus on either side of the knee indicates a meniscus injury on that side.

Fig. 10. McMurray's test. The patient is supine. The examiner then flexes the knee. The knee is extended with external rotation of the tibia on the femur and then extended with internal rotation. A palpable or audible click indicates an injury of the meniscus.

tenderness at various locations, depending on which bursa is inflamed. Treatment is conservative, consisting of relative rest, passive modalities for pain, bracing with a Neoprene sleeve, and progressive stretching and strengthening exercises [66].

Leg injuries

Leg injuries common in tennis include tennis leg, Achilles tendonitis, shin splints, and cramps. These injuries are common in sports that require constant running and repetitive jumping. Achilles tendonitis and rupture usually occur when a foot that has been in plantarflexion is forced into dorsiflexion with the knee extended [29,67,68].

Tennis leg

Tennis leg is a partial tear at the musculotendinous junction of the medial belly of the gastrocnemius muscle [69–71]. The mechanism of injury involves sudden ankle dorsiflexion in the involved leg, where the knee was previously extended and the ankle was plantarflexed. In tennis, this position is usually encountered in the back leg of the athlete during the follow-through of a tennis serve. Predisposing factors are increasing age, inadequate stretching, fatigue, and prior muscle injury. The differential diagnosis includes deep vein thrombosis, popliteal cyst, and tumor [66,71].

Sudden intense calf pain is the typical presentation. Physical examination findings initially include a palpable gap in the muscle, with calf tenderness and painful restricted ankle dorsiflexion. As time passes, the palpable gap

disappears secondary to increased calf swelling [66,67,72]. Treatment is conservative, with immediate attention to diminishing the amount of swelling. Patients often use crutches or heel lifts for a short time, followed by initiation of stretching and strengthening of the posterior calf muscles. Previous activity levels may not be resumed until the athlete can ambulate without pain, usually 2 to 12 weeks after the injury [67,68,71].

Achilles tendonitis

Achilles tendonitis progresses through different stages depending on the severity of the injury. Initially, the paratendon becomes inflamed, thickened, and edematous. With continuation of the aggravating activity, the paratendinitis spreads to the tendon. This leads to weakened tensile strength of the tendon and, ultimately, tendon rupture, which can be partial or complete [29,68].

Patients with Achilles tendonitis initially complain of pain only after strenuous activity. With continued exercise, pain is present during the activity. The pain is localized to the inferior aspect of the posterior calf. The patient may complain of weakness during activity and morning stiffness. Physical examination may demonstrate decreased ankle dorsiflexion and tight hamstring muscles, tenderness to palpation 2 to 6 cm above the insertion of the tendon on the calcaneus, and a thickened nodular area of tenderness on the tendon that moves when the ankle is flexed. Treatment is conservative, with rest, physical therapy that emphasizes stretching, and progressive resistance strengthening of the calf muscles [29].

On physical examination, the patient with a rupture can usually walk flat-footed but without push-off. Acutely, a palpable gap in the tendon may be felt, but this soon becomes masked because of bleeding into the pseudosheath. A positive Thompson test is pathognomonic of a complete rupture [67,68]. MRI is most helpful in detailing the extent of a tear [37].

Treatment of a rupture is nonsurgical or surgical. Factors to be considered in deciding whether to operate include the length of time to diagnosis, age, athletic level of the patient, and quality of the tendon. Goals of the rehabilitation program in both cases are to restore the normal length and strength of the muscle tendon complex. It is important to note that the risk of rerupture is higher with nonoperative treatment [68].

Medial tibial stress syndrome

Medial tibial stress syndrome (shin splints) refers to pain that is located at the posteromedial border of the lower one third of the tibia. It is thought to arise from muscles that normally absorb shock; however, because of fatigue, the shock is transferred to the bone, resulting in periostitis and eventually shin splints. The location of the pain is commonly found along the posteromedial border of the tibia. The pain is exacerbated by active resisted plantarflexion. Radionuclide imaging is helpful in diagnosing shin splints. MRI

has also been helpful in defining the extent and degree of bone involvement in shin splints. Treatment is primarily rest, although taping or use of a pneumatic splint may also be done [68,71].

Foot and ankle injuries

Common ankle and foot injuries in tennis include ankle sprains, plantar fasciitis, "tennis toe," and degenerative joint disease of the first metatarsophalangeal joint [67,68].

Ankle sprain

The most common ankle sprain is an inversion injury causing damage to the lateral ligaments (anterior talofibular, calcaneofibular, and posterior talofibular). First-degree sprains involve only the anterior talofibular ligament, whereas second-degree sprains also have involvement of the calcaneofibular ligament. A third-degree sprain involves all three ankle ligaments and requires operative intervention [67,68].

Diagnosis is made from the patient's history and physical examination. The injury occurs when there is excessive supination and inversion of the plantarflexed foot while the tibia is externally rotated. Tenderness, swelling, and ecchymosis are present in variable amounts, depending on the severity of the sprain. Anterior drawer and talar tilt tests should be part of the physical examination [68].

Treatment for first- and second-degree sprains is identical, although complete recovery is longer for second-degree sprains. Initial rehabilitation consists of rest, ice, compression, and elevation (RICE) as well as protected weight bearing. A normal heel-toe gait is emphasized. Dorsiflexion and plantarflexion are started early to maintain range of motion, and strengthening and proprioceptive exercises are added progressively. Protective splints may be used after full weight bearing is attained to help prevent further injuries, because ankle sprains are often recurrent and lead to permanent instability [68].

Plantar fasciitis

Plantar fasciitis results from an overload of the plantar fascia at its insertion into the medial calcaneal tuberosity. Excessive pronation of the foot and tightness of the gastrocnemius-soleus muscle complex are thought to contribute to plantar fasciitis [67,68].

The diagnosis is made from the patient's history and physical examination. Pain is present at the medial calcaneal insertion of the plantar fascia and sometimes radiates distally toward the ball of the foot. Initially, the pain is worse in the morning with the first few steps and then resolves. With time, the pain occurs throughout activity. Plain radiographic films

may demonstrate a calcaneal spur, which occurs because of chronic inflammation at the insertion of the plantar fascia [67,68].

Treatment is conservative with rest, ice, orthotics to correct biomechanical abnormalities of the foot, heel cups, and corticosteroid injections. Surgery is rarely indicated [67,68].

Tennis toe

Jamming the toes against the toe bumper of the shoe when the athlete abruptly stops causes tennis toe. The great toe is most commonly affected. Pain is caused by pressure attributable to bleeding under the nail and can be relieved by drilling a small hole in the nail. Treatment includes keeping the toenails short and wearing shoes that are properly sized [36,68].

Excessive dorsiflexion performed in a repetitive manner may cause degenerative joint disease of the first metatarsophalangeal (MTP) joint. The athlete may complain of pain during push-off. There is usually localized tenderness, swelling, and decreased dorsiflexion of the involved toe. Plain film radiographs demonstrate narrowing of the MTP joint and subchondral sclerosis. Treatment is geared toward symptomatic management and may include icing and taping the toe. Surgical treatment includes debridement of the MTP joint and sometimes arthrodesis of the MTP joint in 15° to 20° of dorsiflexion [67,68].

Summary

Tennis is popular in the United States, with millions participating in the sport. Unlike many other sports, young, middle-aged, and elderly persons are actively playing tennis. Most injuries that are seen are secondary to overuse. Faulty stroke mechanics are often involved in the development of specific injuries seen in tennis players. Fortunately, most injuries respond to conservative treatment and do not require surgery. Most players are able to return to their previous level of activity after appropriate treatment.

References

[1] Maylack FH. Epidemiology of tennis, squash, and racquetball injuries. Clin Sports Med 1988;7:233–43.
[2] Koplan JP, Siscovick DS, Goldbaum GM. The risks of exercise: a public health view of injuries and hazards. Public Health Rep 1985;100:189–95.
[3] Gregg JR, Torg E. Upper extremity injuries in adolescent tennis players. Clin Sports Med 1988;7:371–85.
[4] Leach RE, Abramowitz A. The senior tennis player. Clin Sports Med 1991;10:283–90.
[5] Hutchinson MR, Laprade RF, Burnett QM. Injury surveillance at the USTA boys' tennis championships: a six year study. Med Sci Sports Exerc 1995;7:826–30.
[6] Kibler WB, McQueen C, Uhl TL. Fitness evaluations and fitness findings in competitive tennis players. Clin Sports Med 1988;7:403–16.

[7] Kibler WB, Chandler TJ. Racquet sports. In: Fu FH, Stone D, editors. Sports injuries: mechanism, prevention, and treatment. Baltimore (MD): Williams & Wilkins; 1994. p. 278–92.
[8] Reese LA, Fricker PA, Maguire LM. Injuries to elite young tennis players at Australian Institute of Sport. Aust J Sci Med Sport 1986;18:11–5.
[9] Winge S, Jorgensun U, Neilson L. Epidemiological studies in Danish championship tennis. Int J Sports Med 1989;10:368–71.
[10] Bylak J, Hutchinson M. Common sports injuries in young tennis players. Sports Med 1998; 26(2):119–32.
[11] Kibler WB, Safran M. Musculoskeletal injuries in the young tennis player. Clin Sports Med 2000;19:781–92.
[12] Kibler WB, Chandler TJ, Uhl TL, et al. A musculoskeletal approach to the preparticipation physical examination: preventing injury and improving performance. Am J Sports Med 1989;17:525–31.
[13] Lehman RC. Shoulder pain in the competitive tennis player. Clin Sports Med 1988;7: 309–27.
[14] Jobe F, Bradley J. The diagnosis of non-operative treatment of shoulder injuries in the athlete. Clin Sports Med 1989;8:419–38.
[15] Nirschl RP. Rotator cuff tendonitis: basic concepts of pathoetiology. In: Barr JS, editor. Instructional course lectures 38. Park Ridge (IL): American Academy of Orthopedic Surgeons; 1989. p. 439–45.
[16] Jobe FW. An EMG analysis of the shoulder in throwing and pitching, a preliminary report. Am J Sports Med 1983;11:3–5.
[17] Jobe FW, Motnes DR, Tibone JE. An EMG analysis of shoulder in pitching, a preliminary report. Am J Sports Med 1984;12:218–20.
[18] Kibler WB. The role of the scapula in throwing motion. Contemp Orthop 1991;22:525–32.
[19] Neer CS II. Impingement lesions. Clin Orthop 1983;173:70–7.
[20] Ciullo JV. Swimmers shoulders. Clin Sports Med 1986;5:115–37.
[21] Jackson DW, Graf BK. Decompression of the coracoacromial arch. In: Jackson DW, editor. Shoulder surgery in the athlete. Baltimore (MD): Aspen Publishing; 1985. p. 51–63.
[22] Kuhn J, Hawkins R. Surgical treatment of shoulder injuries in tennis players. Clin Sports Med 1995;14:139–61.
[23] Priest JD. The shoulder of the tennis player. Clin Sports Med 1988;7:387–402.
[24] Rowe CR, Zarins B. Recurrent transient subluxation of the shoulder. J Bone Joint Surg [Am] 1981;63:863–72.
[25] Priest JD, Nagel DA. Tennis shoulder. Am J Sports Med 1976;4:28–42.
[26] Marx R, Sperling J, Cordasco F. Overuse injuries of the upper extremity in tennis players. Clin Sports Med 2001;20:439–51.
[27] Harryman DT, Sidles JA, Clark JM, et al. Translation of the humeral head on the glenoid with passive glenohumeral motion. J Bone Joint Surg [Am] 1990;72:1334–43.
[28] Beach W, Caspari R. Arthroscopic management of rotator cuff disease. Orthopedics 1993; 16:1007–15.
[29] Barry N, McGuire J. Overuse syndrome in adult athletes. Rheum Dis Clin North Am 1996; 22:515–30.
[30] Ellenbecker TS. Rehabilitation of shoulder and elbow injuries in tennis players. Clin Sports Med 1995;14:87–110.
[31] Bigiliani LU, Kimmel J, McCann PD, et al. Repair of rotator cuff tears in tennis players. Am J Sports Med 1992;20:112–7.
[32] Peters T, Baker C. Lateral epicondylitis. Clin Sports Med 2001;20(3):549–63.
[33] Kandemir U, Fu F, McMahon P. Elbow injuries. Curr Opin Rheumatol 2002;14:160–7.
[34] Nirschl R, Ashman E. Elbow tendinopathy: tennis elbow. Clin Sports Med 2003;22: 813–36.
[35] Whaley A, Baker C. Lateral epicondylitis. Clin Sports Med 2004;23:677–91.

[36] Kulund D. Tennis injuries: prevention and treatment a review. Am J Sports Med 1979;7(4): 249–53.
[37] Wilson J, Best T. Common overuse tendon problems: a review and recommendations for treatment. Am Fam Physician 2005;72(5):811–8.
[38] Ciccotti MC, Schwartz M, Ciccotti MG. Diagnosis and treatment of medial epicondylitis of the elbow. Clin Sports Med 2004;23:693–705.
[39] Rettig AC. Stress fracture of the ulnar in the ulna in an adolescent tournament tennis player. Am J Sports Med 1983;11:103–6.
[40] Posner MA. Injuries to the hand and wrist in athletes. Orthop Clin North Am 1977;8: 593–618.
[41] Rettig A. Wrist problems in the tennis player. Med Sci Sports Exerc 1994;26:1207–12.
[42] Osterman AL, Moskow L, Low DW. Soft tissue injuries of the hand and wrist in racquet sports. Clin Sports Med 1988;7:329–48.
[43] Rayan GM. Recurrent dislocation of the extensor carpi ulnaris in athletes. Am J Sports Med 1983;11:183–4.
[44] Osterman AL. Arthroscopic debridement of triangular fibrocartilage complex tears. Arthroscopy 1990;6:120–4.
[45] Osterman AL, Terrill RG. Arthroscopic treatment of TFCC lesions. Hand Clin 1991;7: 277–81.
[46] Stark HH, Jobe FW, Boyes JH, et al. Fracture of the hook of the hamate in athletes. J Bone Joint Surg [Am] 1977;59:575–82.
[47] Rettig ME, Dassa GL, Raskin KB, et al. Wrist fractures in the athlete: distal radius and carpal fractures. Clin Sports Med 1998;17:469–89.
[48] Carter PR, Eaton RG, Littler JW. Ununited fracture of the hook of the hamate. J Bone Joint Surg [Am] 1977;59:583–8.
[49] Failla JM. Osteonecrosis associated with nonunion of the hook of the hamate. Orthopedics 1993;16:217–8.
[50] Calliet R. Low back pain syndrome. Philadelphia: FA Davis; 1981.
[51] Ferguson R, McMaster J, Stanitski C. Low back pain in college football linemen. Am J Sports Med 1974;2:63–9.
[52] Jackson D, Wiltse L, Cirincione R. Spondylolysis in the female gymnast. Clin Orthop 1976; 117:68–73.
[53] Semon R, Spengler D. Significance of lumbar spondylolysis in college football players. Spine 1981;2:172–4.
[54] Szot Z, Boron Z, Galaj Z. Overloading changes in the motor system occurring in elite gymnasts. Int J Sports Med 1985;6:36–40.
[55] Sward L, Hellstrom M, Jacobsson B, et al. Back pain and radiographic changes in the thoraco-lumbar spine in athletes. Spine 1990;15:124–9.
[56] Eriksson K, Nemeth G, Eriksson E. Low back pain in elite cross-country skiers. A retrospective epidemiological study. Scand J Med Sci Sports 1996;6:31–5.
[57] Kujala U, Taimela S, Erkintalo M, et al. Low back pain in adolescent athletes. Med Sci Sports Exerc 1996;28:165–70.
[58] Marks MR, Haas SS, Wieses SW. Low back pain in the competitive tennis player. Clin Sports Med 1988;7:277–87.
[59] Chard MD, Lachmann MA. Racquet sports: patterns of injury presenting to a sports injury clinic. British Sports 1987;21:150–3.
[60] Sward L, Hellstrom M, Jacobbson B, et al. Anthropometric characteristics, passive hip flexion and spinal mobility in relation to back pain in athletes. Spine 1990;15:376–82.
[61] Hellstrom M, Jacobsson B, Sward L, et al. Radiologic abnormalities of the thoracolumbar spine in athletes. Acta Radiol 1990;31:127–32.
[62] Hainline B. Low back injury. Clin Sports Med 1995;14:241–65.
[63] Benzon HT. Epidural steroid injections for low back pain and lumbosacral radiculopathy. Pain 1986;24(3):277–95.

[64] Green LN. Dexamethasone in the management of symptoms due to herniated lumbar disc. J Neurol Neurosurg Psychiatry 1975;38:1211–7.
[65] Haher T, Felmy W, Devlin V, et al. The contribution of the three columns of the spine to rotational stability: a biomechanical model. Spine 1989;14(7):663–9.
[66] Gecha S, Torg E. Knee injuries in tennis. Clin Sports Med 1988;7(2):435–52.
[67] Leach R. Leg and foot injuries in racquet sports. Clin Sports Med 1988;7(2):359–70.
[68] Zecher S, Leach R. Lower leg and foot injuries in tennis and other racquet sports. Clin Sports Med 1995;14(1):223–39.
[69] Bianchi S, Martinoli C, Abdelwahab IF, et al. Sonographic evaluation of tears of the gastrocnemius medial head (''tennis leg''). J Ultrasound Med 1998;17:157–62.
[70] Delgado G, Chung C, Lektakul N, et al. Tennis leg: clinical US study of 141 patients and anatomic investigation of four cadavers with MR imaging and US. Radiology 2002;224: 112–9.
[71] Blue J, Matthews L. Leg injuries. Clin Sports Med 1997;16(3):467–78.
[72] Millar A. Strains of the posterior calf musculature (''tennis leg''). Am J Sports Med 1979; 7(3):172–4.