# Chapter 8

# REHABILITATION OF THE COMBATANT WITH MUSCULOSKELETAL DISORDERS

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## INTRODUCTION

Musculoskeletal injuries are commonly encountered in the physiatrist's office. Most of these injuries occur as a result of occupational or sports related endeavors. Many of the rehabilitation principles and techniques for these disorders, as well as the overall approach, are also applicable to the injured soldier. In times of war, musculoskeletal injuries similar to those seen in civilian populations can and do occur, often at extremely high rates. General Pershing stated in 1918 during World War I that he wanted no more men with "flat feet, weak backs, and lack of muscular development" sent overseas until they had received special training to restore them to normal.[1] An aggressive, nonsurgical rehabilitative approach to these musculoskeletal ailments is essential in order to decrease injury time, to prevent long term disability, and to reconstitute our forces. The focus of this chapter will be to outline and discuss the pathophysiology of musculoskeletal injuries; the acute, subacute, and chronic management of musculoskeletal disorders; and the specifics of rehabilitation of these disorders as they particularly relate to wartime management.

## PATHOPHYSIOLOGY OF MUSCULOSKELETAL INJURIES

In order to guide specific rehabilitation of a given disorder, some basic knowledge about injury and repair of damaged tissue is essential. In the military setting, musculoskeletal injuries may occur due to one of three mechanisms: (1) soft tissue failure, (2) overload, and (3) direct trauma. Musculoskeletal soft tissues include tendon, ligament, muscle, bursae, bone, and nerve. Each of these tissues has a specific function, mechanism of injury, and healing response to a given injury. We will first describe the response of specific tissues to injury and then detail in depth overload injuries. Direct trauma will be discussed as indicated under specific injuries and their rehabilitation. Bone and nerve injuries will be discussed in other chapters and, therefore, not described in this chapter.

### Tendon Injuries

The primary function of tendons is to transmit muscle force to the skeletal system with limited elongation. Tendon tissue is composed of dense fibers of connective tissue with very high tensile strengths in which the fibers are arranged parallel to each other in the direction of the tensile force of the muscle.[2] A tendon is most likely to be injured when (*a*) tension is applied quickly, (*b*) tension is applied obliquely, (*c*) the tendon is under tension before loading, (*d*) the attached muscle is maximally innervated, (*e*) the muscle group is stretched by exterior stimuli, or (*f*) the tendon is weak in comparison with the muscle.[3,4] These are conditions that surface often during wartime, and in particular, during vigorous training or combat situations. Clinically, tendon injuries, especially tendinitis, are quite common, particularly those of the supraspinatus and bicep tendons used in overhead activities, patellar, iliotibial band (ITB), and Achilles tendinitises used in lower extremity activities.

Tendon healing after injury occurs in three phases (Table 8-1). The first phase is inflammatory, which occurs in the first 48 to 72 hours and is highlighted by influx of vasoactive substances, chemotactic factors, and degradative enzymes.[5] Details of the inflammatory reaction can be found elsewhere.[5–9] This phase is important in the healing process of injured tissue. Nonsteroidal antiinflammatory drugs (NSAIDs) are often used acutely in the treatment of soft tissue injuries. Carlstedt and associates[10,11] have found that in animal models, treatment with indomethacin increased the tensile strength in healing tendons possibly by increasing the cross linkage of collagen molecules.

The second phase is a reparative, or collagen production, stage. This phase starts within the first week and is characterized by collagen proliferation produced by newly infiltrated fibroblasts and cel-

**TABLE 8-1**

**PHASES OF TENDON HEALING**

| Phase | Time | Predominant feature |
|---|---|---|
| Inflammatory | First 48–72 h | Acute inflammatory reaction |
| Reparative | 72 h–3 wk | Collagen proliferation phase |
| Maturation | 3+ wk | Maturation and remodeling |

lular fibrin matrix. During this phase, collagen fibrils are laid down in a random pattern, and thus possess little strength.[2]

During the final phase of healing—maturation or remodeling—the mechanical strength of the healing tendon continues to increase because of remodeling and organization of the fiber architecture along the direction of muscle force. Unless specific stresses are placed upon the healing tissue, newly produced collagen will become useless scar tissue.[12–14] Such stresses can be accomplished even in the acute setting with continuous passive motion machines, or bracing, which allows some motion in one plane, that is, knee orthosis with free, or even limited, range of flexion and extension available.

Chronic repetitive microtrauma can advance beyond the state of inflammation and tendinitis to a condition of degenerative change and cell damage; this is the major component of the pathologic picture termed *tendinosis*.[15] Some common examples of clinical musculoskeletal injuries, where cell degeneration is more prevalent than inflammation, include elbow *epicondylitis* [16] plantar fasciitis,[17] patellar *tendinitis,* [18] and Achilles *tendinitis*.[16] The clinical manifestations of tendinosis are the result of a degenerative process, rather than an acute event.[19] The adaptive changes of the musculoskeletal system that occur can be clinically detected both locally and at the site of symptoms or injury, and distally in other links in the kinetic chain.[19]

The distinction between tendinitis and tendinosis has ramifications in the rehabilitation process. Antiinflammatory medications (to be discussed later) will have a greater role in tendinitis than they will in tendinosis. Local changes due to chronic tendon injury, that is, tendinosis, are usually clinically manifested as any or all of the following: (*a*) inflexibility in the involved muscle-tendon group, (*b*) weakness in the involved muscle or surrounding muscle, or (*c*) muscle strength imbalance between force generator and force regulator in the force couple.[19] All of these implications focus the goal of rehabilitation away from relief of the symptoms of the "itis," and toward restoration of function that is lost with the "osis."[1]

**Ligament Injuries**

Skeletal ligaments are highly specialized, dynamic, dense connective tissues that connect bones.[2] Ligaments function both as passive mechanical structures in stabilizing joints, and as neurosensory structures for providing proprioception to muscles and joints. Ligament injuries can occur by contact or noncontact mechanisms.[20] In either case the injury is often the result of a large force, often suddenly and rapidly applied, placed on a given ligament. These injuries result from acute overload at the insertion interface. Details on healing injured ligaments is reviewed elsewhere.[21] In general, however, the same phases of healing as described for tendon repair occur. Elastin fibrils, which are the most prominent component of ligaments and give ligaments their tensile strength, are stimulated to proliferate with stretching. Ligamentous tissue that is immobilized has poor tensile strength.[22,23] Emphasis on early motion and prevention of long term immobilization in the rehabilitation process will allow for greater ultimate strength of the healed ligament. Nevertheless, although the quantity of ligament may remain quite good after injury and healing, the quality of that tissue is never as good as it was preinjury.

Ligament injuries are the most common injuries to joints, most particularly to the knee; in most studies, they account for 25% to 40% of all knee injuries.[24,25] These injuries will be discussed in more detail in the section describing specific knee injuries. With the rehabilitation of ligament injuries, a point to keep in mind is that some studies have shown that increased activity level has a beneficial effect on the strength properties of bone–ligament complexes.[26,27] It stands to reason that the same effect of exercise may also result with bone-tendon complexes.[2]

**Muscle Injuries**

Muscle injuries can occur from a variety of mechanisms. Frequently muscles are injured during eccentric contraction, or activation of the muscle while it is being lengthened by an opposing force greater than the force in the muscle.[2] Therefore, strengthening muscles in both a concentric and eccentric mode is initially essential both in the prevention of muscle injuries, and postinjury during rehabilitation to prevent recurrent injury. Failure often occurs at the myotendinous junction.[28] Muscle tissue damage triggers an initial inflammatory phase followed by subsequent phases of tissue healing, repair, and remodeling, similar to that stated above for tendon injuries. If enough tissue damage occurs, clinical symptoms and signs (pain, swelling, and discoloration) develop.[29] In the overt or subclinical type of muscle injury, the tissue may repair and remodel, but concomitant changes in muscle function, that is, strength, strength balance (agonist vs antagonist), flexibility, and proprioception occur.[30] The signs and symptoms of injury

abate, but these functional deficits persist. The same functional changes may occur if there is a subclinical muscle injury. Other injuries to muscle can occur from lacerations and direct trauma or contusion. Such injuries can diminish muscle strength, limit joint motion, and lead to myositis ossificans. These types of injuries are quite common in the athletic and working populations and are often a cause of lost time from activity. The mechanism, pathophysiology, and location of muscle injury are well-described in other sources.[28,31–33]

Two important areas to consider in the rehabilitation of muscle injuries are (1) the effects of immobilization and (2) the effect of stretching and warm up. Muscle strength and loss of strength secondary to immobility is well recognized and described.[34,35] Loss of strength can delay the rehabilitation process and the injured soldier's return to active duty. In a study[36] using rabbits, it has been shown that muscle immobilized in a shortened position developed less force and stretched to a shorter length before tearing than did the nonimmobilized contralateral control muscle. Muscle immobilized in a lengthened position exhibited more force and needed more change in length to tear than in nonimmobilized controls. Therefore, when immobilization is necessary for any period of time, it should be done with the muscle in a lengthened, or at least a neutral, position. Secondly, a warm up or conditioning period has been shown to be effective in altering the biomechanical properties of muscle in a way that may be effective in avoiding injury.[37] A flexibility training program may have a beneficial effect on reducing the severity and cost of treating joint injuries.[38]

### Bursae Injuries

Bursae are sacs formed by two layers of synovial tissue that are located at sites of friction between tendon and bone (pes anserinus bursa) or skin and bone (prepatellar, olecranon bursae). In their normal state, they contain a thin layer of synovial-like fluid and may actually communicate with an adjacent synovial sac (suprapatellar bursa). Bursae are typically injured with overuse, and repetitive trauma types of activities that cause either friction of the overlying tendon or external pressure. They may also become inflamed from degeneration and calcification of an overlying tendon, which leads to a chemical bursitis, as in subacromial bursitis secondary to calcific supraspinatus tendinitis.[3] When injured, the bursa will become inflamed, with resultant effusion and thickening of the bursal wall.

## OVERVIEW OF REHABILITATION PRINCIPLES

The background for the rehabilitation of musculoskeletal disorders has been described in terms of the pathophysiology of musculoskeletal injuries. There are certain general principles that apply to the rehabilitation of musculoskeletal disorders. The rehabilitation plans for the injured soldier with a musculoskeletal disorder must be oriented toward restoration to function, not just relief of symptoms. The five goals of such a rehabilitation plan can be stated as (1) establishment of an accurate diagnosis, (2) minimization of deleterious local effects of the acute injury, (3) allowance for proper healing, (4) maintenance of other components of general fitness, and (5) return to normal combat function.

### Establishment of Accurate Diagnosis

Without a precise diagnosis, thorough rehabilitation is not possible. Because of the varied presentation of symptoms, recognition is sometimes delayed. Even if recognition of the injury is made, the need for a complete and accurate diagnosis is still present. Symptoms of musculoskeletal injuries are usually pain, swelling, anatomical deformity, or decreased military performance. The symptoms or dysfunction that the soldier is experiencing may be directly related to the musculoskeletal injury, or may be a distant manifestation of a musculoskeletal injury in another part of the kinetic chain that accomplishes the activity.

Complete and accurate diagnosis of the injury can be established by identifying which of the five components of the musculoskeletal injury complex are present in each injury. In each injury, there are five separate areas that may be identified as contributing to the production or continuation of symptoms.[39] These components have an effect on either musculoskeletal anatomy or musculoskeletal functions; they are (1) *tissue complex injury* (the area of actual anatomical disruption); (2) *clinical symptom complex* (that group of symptoms that are causing acute pain, swelling, and dysfunction); (3) *functional biomechanical deficit* (the combination of muscle inflexibilities, weakness, and imbalance that causes inefficient mechanics); (4) *functional adaptation complex* (the functional substitutions that the soldier employs as a result of the injury in order to maintain performance); and (5) *tissue overload complex*

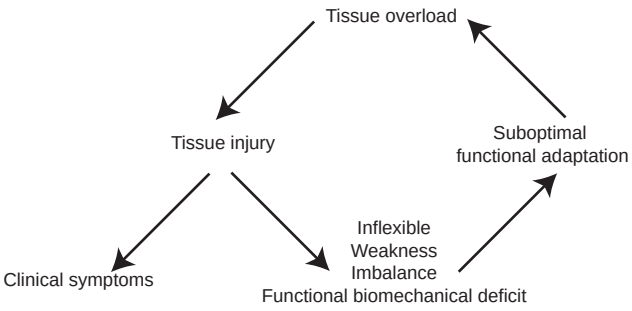



**Fig. 8-1.** Vicious overload cycle. Source: Kibler WB, Chandler TJ, Pace BK. Principles for rehabilitation after chronic tendon injuries. In: Renström PAFH, Leadbetter WB, eds. Tendinitis I: Basic concepts. *Clin Sports Med.* 1992; 11(3):663.

(that group of tissues that may be subject to tensile or eccentric overloads, which may cause or continue symptoms or disability) Figure 8-1.

These components are actually parts of a negative feedback loop, or vicious cycle, that is operative in muscle and musculoskeletal injuries (see Figure 8-1).[19] Depending on the soldier's intensity or duration of continued use, cycling within the loop may continue for varied periods of time before actual clinical symptoms are manifest. During this time, the soldier's function may be fairly normal, but his efficiency may not be optimal. A thorough evaluation of each soldier with respect to inflexibilities, weaknesses, or imbalances will demonstrate the deficits and allow the beginning of diagnostic and therapeutic processes. Specific diagnostic evaluation will guide specific rehabilitation, that is, anatomical diagnosis and diagnosis of functional deficits must be made. This will allow a holistic approach to the total effect an overload injury has on the entire kinetic chain and the total function of the soldier, and will guide rehabilitation back to normal function.

### Minimization of Deleterious Local Effects of the Acute Injury

The initial steps in minimizing deleterious local effects of the acute injury are the control of inflammation and pain. Cryotherapy (the use of ice or other methods of cold application) decreases arteriolar and capillary blood flow and muscle spasm, and is applied to control edema and reduce pain.[40–49] Ice can be applied in the form of crushed ice in a plastic bag, an iced immersion tub, or an ice massage.[43,46,50–52] Limiting the initial development of joint effusion will speed the recovery process.[53] The length of time the cryotherapy must be administered to an injured muscle is directly dependent on the depth of overlying fat; it may vary from 10 to 30 minutes.[54,55] Ice and frozen gel may provide more consistent and longer duration cooling.[56] As with any modality, care must be employed to avoid complications such as burns to anesthetic areas or injury to superficial nerves.[57]

Compression must be concomitant with ice and elevation of the injured area. Ace bandages or other forms of local compression dressings will decrease the degree of the acute inflammatory response and the overdistension of soft tissues due to hemorrhage and exudate. Similarly, no weight bearing, or decreased weight bearing, may initially be important to decrease the degree of inflammatory response in lower extremity injuries. Crutches, canes, and walkers may be helpful in this manner. Likewise, the use of a sling or splint may be necessary for upper extremity injuries.

Early judicious use of antiinflammatory medications and pain medications may greatly speed recovery. The antiprostaglandin effect of NSAIDs has some potential benefit during the acute phase of musculoskeletal injuries. The drugs may minimize the local side effects of the injury by limiting the extent of the inflammatory response, as well as providing pain relief. The duration of the analgesic effect of the NSAID may be different from that of the antiinflammatory effect.[58] Acetylsalicylate may need to be avoided during the early phases of injury because its antiplatelet effects persist for the life of the platelets and may increase hemorrhage. NSAIDs also have antiplatelet effects; however, those are dose related.[14,59] Early use of NSAIDs in acute ankle inversion injuries has been shown to have no deleterious effect on the mechanical integrity of the healing tissue, and in fact, was shown to speed rehabilitation.[60] During the early inflammatory phase, potent glucocorticoids must be used cautiously, because their powerful antiinflammatory effect may inhibit the normal healing process and thereby prolong rehabilitation.[14,61–63] After 1 to 2 weeks, during the proliferative phases of healing, glucocorticoids may be helpful to reduce ongoing inflammation and edema. Glucocorticoids can be safely given orally in commercially available, tapering dose packets, or in a daily dose of prednisone, starting with 70 mg and decreasing by 10 mg per day for 7 days. When using prepackaged dose packets, the physician should evaluate for an adequate amount of corticosteroid in the preparations in order to get a good antiinflammatory response. Glucocorticoids should be used in situations where immediate an-

tiinflammatory response is critical, such as an acute combat situation or when standard antiinflammatory medications are not working. Ten to 20 mg of a corticosteroid (triamcinolone or equivalent) mixed with a short acting anesthetic can be injected into areas of inflammation, yielding a strong antiinflammatory response. Injection of corticosteroids into tendons should be avoided because of the risk of tendon rupture.[61,62] However, injection into tendon sheaths, bursae, or inflamed joints can rapidly decrease inflammation and give substantial pain relief. Some commonly injected areas are:

- Subacromial bursae.
- Lateral or medial epidondyle.
- Tendon sheath of abductor pollicis longus (APL), extensor pollicis brevis (EPB) (de Quervains tenosynovitis).
- Carpal tunnel.
- Greater trochanter.
- Knee joint (intrarticular).
- Plantar fascia.

Opiate and nonopiate analgesic medication can be very helpful in the acute phases of musculoskeletal injuries. To produce an analgesic response, it is important to properly administer the dose of medication, to give it at scheduled times, and to administer it for a predetermined length of time, such as 3 days or 1 week. Too often, inadequate doses of medication are given for fear of making the patient drug dependent.

The use of other therapeutic modalities besides ice can play a large role in the acute management of musculoskeletal disorders. Electrical galvanic stimulation can be very useful for reduction of edema.[57] Higher pulse rates of galvanic stimulation that will produce a tetanic muscle contraction can also be used for pain control. Transcutaneous electrical nerve stimulation (TENS) is helpful for acute pain problems.[57] In fact, TENS units have found the greatest utility in the treatment of acute, painful conditions. Therapeutic modalities, although of significant usefulness in the treatment of acute musculoskeletal disorders, probably have limited usefulness and availability in a combat situation. Occasionally, portable TENS units may be easily transported to the combat zone.

**Allowance for Proper Healing**

For proper healing to take place, immobilization for specific periods of time may occasionally be necessary. Immobilization may be accomplished by taping, bracing, padding, or casting. Prefabricated joint immobilizers and splints are quite useful (Figure 8-2). Slings, cervical collars, and back braces may also be helpful. The period of immobilization or protection of the injured structure will be individualized to fit the nature and extent of the injury. In general, however, gentle mobilization should be initiated even within the first 24 hours after an acute injury and increased as pain and swelling diminish.

Early motion and exercise are essential to proper rehabilitation of musculoskeletal disorders and to speed the healing process.[64–67] Collagen fiber growth and realignment can be stimulated by early tensile loading of muscle, tendon, and ligament.[68] The formation of adhesions between repairing tissue and adjacent structures can be limited by early motion.[69] Proprioception is better maintained and recovers faster with early motion.[68] Optimal conditions for healing depend on a very fine balance between protection from stress and return toward normal function at the earliest possible time.[64,70]

Regaining flexibility allows for proper healing. Decreased joint flexibility can result from muscle spasm, pain and resulting neural inhibition, connective tissue adhesions and contracture, or intraarticular blockade. Treatment of the decreased flexibility is predicated on the specific cause. Muscle spasm is often alleviated with cryotherapy and elec-

**Fig. 8-2.** Prefabricated joint immobilizers and splints for the knee and thumb or wrist.

trical stimulation. Pain and its consequent neural inhibition can be relieved with electrical stimulation modalities, soft tissue mobilization, and massage. Soft tissue and connective tissue changes can be alleviated with static stretch and proprioceptive neuromuscular facilitation (PNF) techniques.[71–73] Intraarticular blockade may be secondary to intraarticular fibrosis or mechanical blocking from such entities as a torn meniscus. Occasionally, increased flexibility can be obtained with intraarticular injection of a local anesthetic. Other times, surgery may be necessary.

Range-of-motion programs may be classified as passive, active, and active assistive. During the first 3 to 5 days after an injury, passive techniques are used while inflammation and edema are quite significant. The early controlled motion helps to decrease soft tissue edema and neural inhibition of tissues. Static stretch and PNF techniques (such as contract relax, the contraction of contralateral extremity, and contraction of antagonistic muscle) can all be effective in improving flexibility.[73–75]

Active assistive programs allow active motion to begin with the assistance of either another (ie, a therapist) or with devices (such as pulleys). The purpose of these activities is to enhance further mobilization of the injured tissues as active muscle firing starts to occur. Gradually these techniques are upgraded to include more active participation on the part of the injured soldier until he is completely active throughout the entire range-of-motion. Once full joint range-of-motion is attained, the injured soldier can proceed to a full flexibility program. Often the subjective complaints of the patient will determine the degree to which he progresses through the phases of mobilization.

The important points in restoring joint and soft tissue extensibility are to avoid overstressing healing tissue, and to recognize the effect of joint instability. It is essential to avoid excessive biomechanically induced stress on the healing structures while mobilizing and strengthening tissues appropriately. For example, an acutely injured and surgically repaired anterior cruciate ligament (ACL) should be mobilized as soon as possible to avoid articular degeneration. However, too rapid mobilization and early stressing of the extensor mechanism may hamper rehabilitation and proper tissue healing. Vigorous mobilization also needs to be avoided in situations where joint instability exists. For example, with acute anterior glenohumeral dislocations, it is often advantageous to avoid abduction and external rotation for the first 4 to 6 weeks after injury to allow some soft tissue healing to occur in the anterior glenohumeral joint. Later, flexibility techniques may be beneficial to avoid excess loss of external rotation of the shoulder.

The effects of immobilization on muscle is well documented.[76–78] There may be up to a 20% decrease of muscle strength after 1 week of muscle immobilization and another 20% decline in residual strength every subsequent week of immobilization.[79] Atrophy may occur even faster if the muscle is immobilized in a shortened position.[80] Type I fibers are particularly affected by immobilization, with up to a 47% decrease by the fifth week.[81–83]

The side effects from immobilization extend beyond the muscle. If an associated joint is excessively immobilized, significant joint capsule, cartilage, subchondral bone, and bone-ligament changes occur.[22] Protracted rehabilitation (greater than 1 year) may be necessary to regain function of the structures.[84] After 8 weeks of immobilization, some tissues, such as articular cartilage, may never completely return to normal.[80,85]

Once the joint and soft tissue extensibility has been attained, the injured soldier is ready to start strength training. Strength training can be started even when full range-of-motion has not been attained. However, any strength gains will be specific only for that range-of-motion. Strengthening programs are divided into isomet rics, manual resistance, isotonics, and isokinetics. These programs can be divided into concentric and eccentric contractions. Details of strength training can be found elsewhere.[86,87] The goal of all strengthening exercises is hypertrophy of muscle and the enhancement of recruitment, and firing of the motor units.[86]

The most important aspects of any strength program are specificity and overload. Muscles are activated for activities in a dynamic way with alteration of concentric and eccentric contractions. Training muscles with static contractions (isometrics) or against a set amount of resistance (isotonics) may improve the overall strength of the muscle group, but may not be transferable to specific activities in the combat field.[88–90] If hypertrophy is to occur, muscles must be subject to loads greater than the usual stresses of daily activity. The goal of a resistance program for increased strength is to overload, not overwhelm, the muscles.[87] Overloading the muscle too rapidly will result in reactive inflammation changes and associated synovitis. The four basic factors in overload are (1) intensity, (2) volume, (3) duration, and

(4) rest. Details of strength training are discussed elsewhere.[86,91–93]

Isometric exercises are used early in the acute injury phase. In isometric exercises, no joint motion occurs, therefore, strengthening can occur concomitantly with joint protection. This type of contraction helps maintain muscle tone and a pattern of contraction. Isometric contractions should be held for at least 6 seconds, with rest periods between 10 and 20 seconds to ensure proper muscle blood flow and to remove substrate of muscular contraction.[73] The isometric contractions should be carried out frequently during the day, utilizing sets of 10 to 20 repetitions.[88] Except in acute musculoskeletal injury rehabilitation, isometric exercises are not routinely used for strength training because of their ability to strengthen muscles only at one point of the range-of-motion.

Because of cross education of the neuromuscular system, exercise of the contralateral side is also important early after an injury. After exercising the contralateral side of the body, the immobilized (nonexercised) side has demonstrated strength increases of up to 30%.[94] Manual resistance exercises are begun as soon as the joint can be safely moved without threat of further injury. The therapist manually resists whatever effort the patient is able to exert. Contractions should be carried out in a pain-free range-of-motion.[95]

Since most strength training is done in a dynamic manner, isotonic and isokinetic exercises are integral in a proper rehabilitation program for musculoskeletal disorders. Sophisticated isokinetic equipment, which allows the control of speed while maintaining a constant force, will probably not be available in the combat zone.[96,97] However, isotonic exercises, where a constant resistance is applied, can be accomplished with free weights, sand bags, cans of food, water bottles, or whatever else is available in the field.

### Maintenance of Other Components of General Fitness

Once an accurate diagnosis has been made, the deleterious local effects of tissue injury have been minimized, and allowances have been made for proper healing to occur, then the maintenance of other aspects of fitness need to be addressed. For complete rehabilitation to occur, changes in different parts of the kinetic chain after a musculoskeletal injury need to be dealt with. In the rehabilitation of wrist or elbow injuries, shoulders must be strengthened, because the shoulder is the primary stabilizer of the upper limb for distal joint functioning. Hip strength and flexibility may be altered because an ankle injury has caused modification in the gait cycle and resultant proximal limb substitution patterns. A prescription for substitute exercise to maintain general cardiovascular fitness, as well as general strength, will help decrease total rehabilitation time. Along with absolute strength gains, improvement of muscular endurance needs to be addressed. Gaining muscle endurance entails stressing the aerobic pathways to improve the oxidative enzyme capacity of slow-twitch muscle fibers.[98] High repetition, low-resistance exercises, which require greater degrees of muscle endurance, should be integrated into the rehabilitation program. The use of a stationary bicycle, cross-country ski machine, or rowing machine can all increase muscle endurance and are portable enough to be available in a combat hospital. In the field, aerobic conditioning may be accomplished by jumping rope, wind sprints, running hills, step climbing, and swimming, when water is available and safe.

### Return to Normal Military Duty

After adequate flexibility, strength, endurance, and cardiovascular fitness are attained, the injured soldier is then ready to begin specific training or retraining in the development of biomechanical and neurophysiologic skill patterns for the specific activities he will need to perform. The neurophysiologic learning process for developing coordinated skill patterns is based on constant repetition, with focus on perfecting the movement.[73,99,100]

Criteria for return to active duty should include resolution of the tissue injury and clinical symptom complex, functional range-of-motion and adequate muscle strength, and ability to perform specific military duty activities. This usually occurs within 10 to 14 days for simple injuries, but can take up to 6 weeks for more severe injuries or for injuries that develop complications, such as myositis ossificans. Long-standing musculoskeletal problems may result in functional biomechanical deficits and concomitant substitution activity patterns. In these patients, the major focus of rehabilitation must extend well beyond symptom relief. If the functional biomechanical deficits and resultant activity patterns are not addressed, performance drop-off, recurrent injury, or both will occur.[29]

## PRINCIPLES OF REHABILITATION FOR SPECIFIC DISORDERS

### Cervicothoracic Disorders

#### *Cervical Soft Tissue Injuries*

Soft tissue injuries in the cervical region are usually classified with minimal precision. A sprain is an injury to a joint, with possible rupture of some of the ligaments or tendons, but without dislocation or fracture, and occurs from stretching of the supportive soft tissue. A cervical strain is an overload injury to the muscle-tendon unit in the cervical spine caused by excessive force, rotation, or eccentric loading. Most neck muscles do not terminate in tendons, but attach to bone by myofascial tissue that blends into the periosteum. Other cervical soft tissues include the sympathetic nervous system chain, the greater occipital nerve, the vertebral artery, and the interspinous and supraspinous ligaments. In the evaluation of cervical injuries, the most important conditions to rule out are bony lesions, that is, fractures or ligamentous instabilities. In acute severe trauma, plain radiographs of the cervical spine, including anteroposterior, lateral, obliques, and open mouth views, are essential when evaluating acute severe trauma cases for fractures or instability. Often flexion-extension views will also be necessary. On the battlefield, any soldier with severe neck pain or any neurological signs after an acute cervical injury must be assumed to have a fracture or instability until proven otherwise. This situation may require evacuating the soldier to a local hospital for radiographs under spinal precautions, that is, on a spine board with head and neck immobilized.

***Method of presentation.*** A soft tissue injury usually presents as acute traumatic injury secondary to sudden jerking motion of the head and neck, or chronic overload from maintenance of one position of the cervical spine for prolonged periods of time, most often with poor posture.

***Tissue injury complex.*** The tissue injury complex will include supraspinal and interspinal ligaments, cervical paraspinal or anterior cervical musculature (scalenes, sternocleidomastoid, trapezius, erector spinae, levator scapulae).

***Clinical symptom complex.*** This complex presents as neck pain without radiation, exacerbated with movement and resistive motion testing, and relieved by rest and immobility.

***Functional biomechanical deficit.*** With this, the manifestations are loss in the cervical spine of full flexion and rotation or lateral rotation, or both. There is loss of the normal coupled motions in the cervical spine with substitution patterns of movement.

***Functional adaptation complex.*** Here, a clinician will observe marked restriction in cervical range-of-motion with most significant changes at specific segmental levels.

***Tissue overload complex.*** This refers to the specific ligaments or muscles injured.

***Rehabilitation*** (Table 8-2)**.** Early treatment involves control of the inflammatory process as discussed previously. Details of the specific parameters for the use of therapeutic modalities is beyond the scope of this chapter, but can be found in the referenced texts.[52,57] Judicious use of oral corticosteroids may be very helpful. Rarely, an injection of corticosteroid may be indicated in an involved and specifically identified cervical spine or ligamentous structure.[101] A soft cervical collar can improve comfort, assuming no cervical spine instability exists. The wearing of cervical collars should be sharply tapered to avoid dependence on them and prevent atrophy from prolonged use. Total wear of the collar probably should not exceed 10 to 14 days, and weaning should allow increasing daytime removal, with continued use at night to prevent injury during sleep.[102] Occasionally, when only one or two specific myofascial trigger points are present, injection with lidocaine or dry needling, or spray and stretch techniques, may be of benefit for reduction of local pain symptoms.[103,104] When multiple points are

**TABLE 8-2**

**TREATMENT FOR CERVICAL SOFT TISSUE INJURIES**

| Time (d) | Treatment |
|---|---|
| 0–3 | NSAIDs, pain medication, oral corticosteroid |
| 0–10 | Soft cervical collar |
| 3–7 | ROM exercises initiated, isometric strengthening |
| 10–21 | Advanced strengthening and flexibility program |

NSAID: nonsteroidal antiinflammatory drug
ROM: range-of-motion

**Fig. 8-3.** Passive neck stretches for cervical range of motion and flexibility.

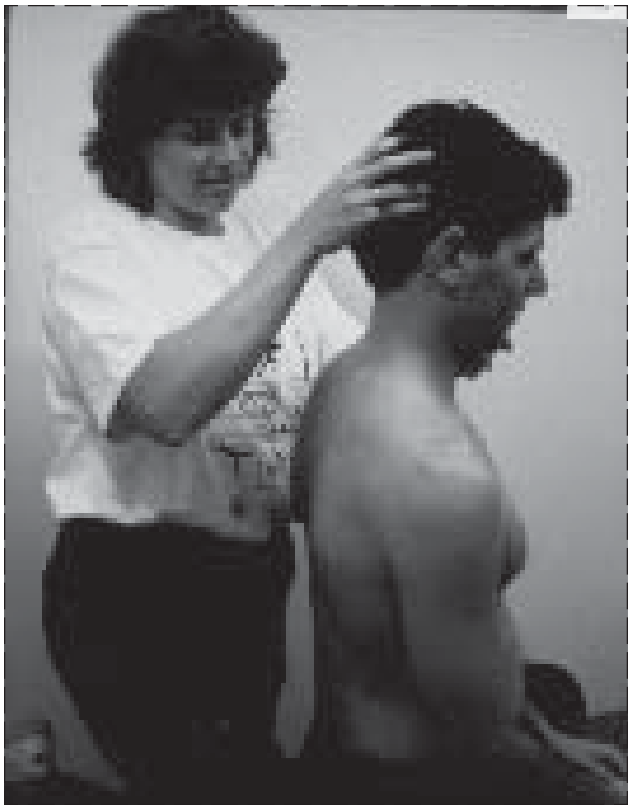


**Fig. 8-4.** Dorsal cervical glides to improve posture. The patient is instructed to tuck the chin to the back of the throat, not to the chest.

present, or symptoms are not well localized to specific, classical trigger points, success is quite variable with these techniques.

Once the acute phase passes (which should be within 3 to 7 days), gentle range-of-motion can begin. Dorsal glides and passive neck stretches are helpful (Figures 8-3, 8-4). Spinal manipulative therapy may be useful during the pain control phase and useful to improve segmental motion.[105,106] Manipulative therapy must be done with some caution, because of potential complications.[107] With sprain injuries, where there is a disruption or stretching of ligaments, traction should be avoided, because it can further stretch damaged ligaments and exacerbate symptoms.

Neck strengthening is an integral part of any cervical spine rehabilitation program. Initially, isometrics should be done with the head in midline only (the neutral position), and resisting forces should be applied perpendicularly to the head from every position. Very slowly, with strengthening in the midline, the head can be taken out of midline after there is no pain. Extremes of head flexion, either anteriorly, posteriorly, or laterally against resistance, are seldom indicated.[108] Of great importance in the overall cervical spine rehabilitation program is shoulder girdle stabilizer strength.[101] This includes the scapular protractors and retractors, as well as truncal/torso stabilizers. These are important to be able to maintain appropriate postural ergonomics by eliminating the head-forward posture associated with a stooped shoulder alignment (Figures 8-5, 8-6).[101]

Following initial cervical isometric exercises, progression to total upper-body isotonic exercises will improve strength and stability of the entire upper torso. Reinforcement of proper posture ergonomics, specifically including cervicothoracic and pectoral girdle posture mechanics, is imperative.[101]

Minor cervical soft tissue injuries should be resolved within 7 to 10 days. More severe injuries may take up to 3 to 4 weeks to resolve. When severe ligamentous injuries cause spinal instability, prolonged treatment with immobilization (up to 6 months), and possibly surgery, may be necessary.

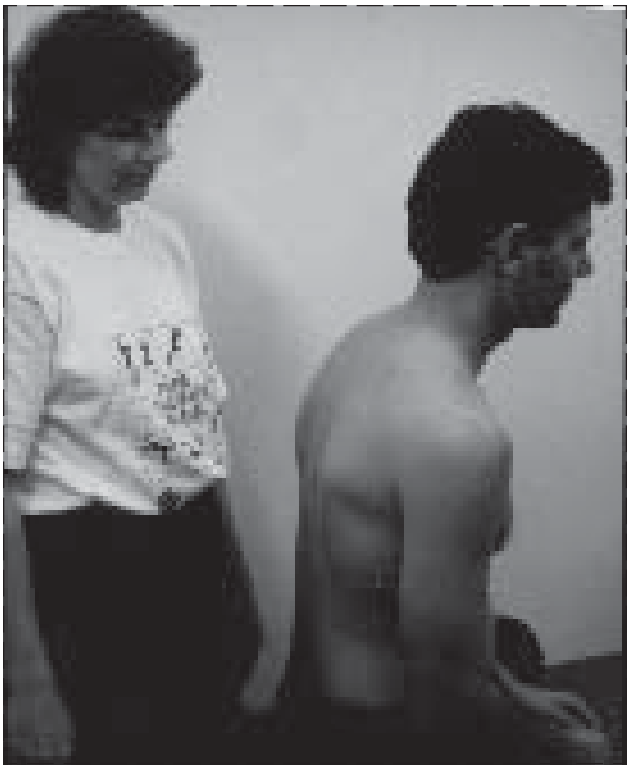


**Fig. 8-5.** Stooped posture with the head forward.

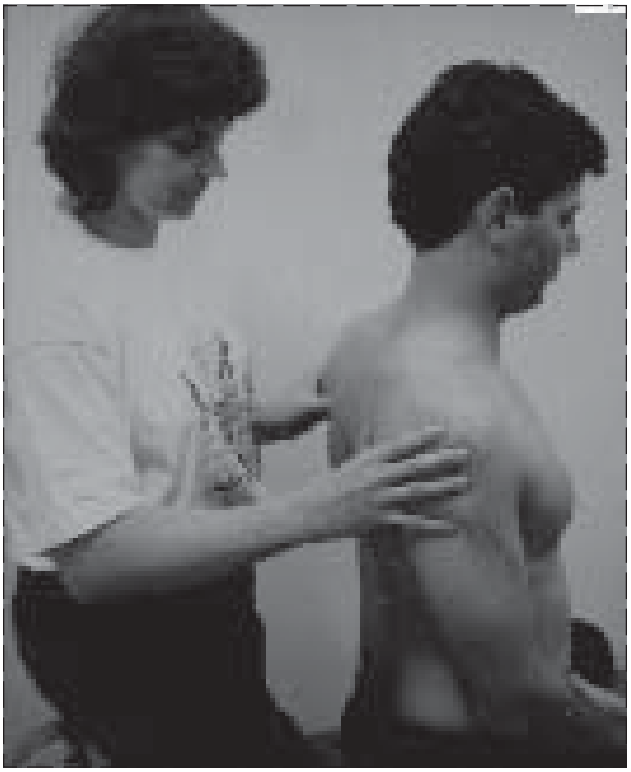


**Fig. 8-6.** Strengthening exercises for scapular retractors/stabilizers. The patient is instructed to squeeze the shoulder blades together with the chin tucked.

### *Cervical Radiculopathy*

Radiculopathy in the cervical area is often the result of chronic stress to the bony, ligamentous, and muscular elements of the cervical spine. Symptoms may be quite subtle and are described as aching, dull, or diffuse in nature. More commonly, symptoms are sharp, piercing, and electric-shock–like with radiation into a specific dermatome of the upper extremity. The most important similar diagnoses to distinguish from cervical radiculopathy are (*a*) peripheral nerve entrapments, such as carpal tunnel syndrome (or high median neuropathy) vs C-6 radiculopathy; (*b*) ulnar neuropathy vs C-8 radiculopathy; (*c*) shoulder and hand tendinitises (ie, rotator cuff pathology) vs C-5 radiculopathy; or (*d*) de Quervain's and extensor tendinitis of the wrist vs C-6 or C-7 radiculopathy. Whenever symptoms of cervical radiculopathy (especially weakness) are progressive, or bowel or bladder dysfunction occur, urgent referral to either a neurosurgeon or orthopedic surgeon is mandatory.

***Method of presentation.*** Radiculopathy may present as acute traumatic injury, or more commonly, chronic overload injury often related to repetitive activities stressing cervical spine musculature.

***Tissue injury complex.*** Disruption of the annulus fibrosis with herniation of nucleus pulposus will cause a mechanical or chemical radiculitis, usually at the level of the foraminal canal or foraminal disease, or both caused by bony degenerative changes or other sources.

***Clinical symptom complex.*** Arm pain or numbness and tingling will usually present in a dermatomal distribution, and be worse with lateral flexion and extension to the ipsilateral side (Spurling's maneuver, Figure 8-7). Also evident may be coughing and sneezing, focal weakness, sensory loss, and diminution of muscle stretch reflexes in a dermatomal distribution.

***Functional biomechanical deficit.*** There will be altered weight distribution across the intervertebral disk. If this occurs gradually, loss of flexion, exten-

**Fig. 8-7.** Spurling's maneuver-axial compression applied to the cervical spine in a side bent and rotated position to close the neuroforamina and reproduce symptoms in a dermatomal distribution.

sion, and lateral rotation and bending motions are obvious, with segmental motion dysfunction.

***Functional adaptation complex.*** Here will be observed the loss of normal coupled motion with lateral flexion and rotation of the cervical spine, abnormal segmental motion patterns (hyper- or hypomobility), and hunched forward posture.

***Tissue overload complex.*** An indicator is fibrosis of annulus fibrosus.

***Rehabilitation*** (Table 8-3). Initially, immobilization (and thus enforced relative rest) of the cervical spine structures should be initiated. If a cervical collar is used, the higher part of the collar should

**TABLE 8-3**

**TREATMENT FOR CERVICAL RADICULOPATHY**

| Time (d) | Treatment |
|---|---|
| 0-14 | Relative rest with a cervical collar |
| 0-7 | Oral corticosteroids, NSAIDs, modalities |
| 0-7+ | Cervical traction |
| 7-10 | Cervical isometrics |
| 10-14+ | Advanced strengthening, cervicothoracic stabilization program |

NSAID: nonsteroidal antiinflammatory drug

**Fig. 8-8.** Proper placement of cervical traction in 25°–30° of flexion.

be worn posteriorly, to maximally open the intervertebral foramen. As stated previously, early tapering should be initiated to prevent disuse atrophy. The collar should initially be worn at all times, except during baths and isometric strengthening exercises. Antiinflammatory medications, particularly oral glucocorticoids, may be used judiciously for an acute condition. Short courses of time-dependent doses of narcotic medications may be helpful, but prolonged use causes side effects. With cervical disk symptoms, a short course of cervical traction is also warranted. A clinical trial of manual cervical distraction is a useful diagnostic maneuver that can predict a successful response to mechanical cervical traction[109] and may be better tolerated than mechanical traction, making it therapeutically useful. Traction in the cervical spine may decrease lordosis, decrease muscle spasm, enlarge the foramina, and distract the vertebral bodies enough to give some pain relief. Proper placement of cervical traction is essential (Figure 8-8).[107] Traction should be initiated in 30° of flexion, starting with 10 to 15 lb, and then increased to 30 lb over time.[110] The reclining and sitting positions are equally therapeutic. However, continuous in-bed, low weight (5 lb) cervical traction, aside from the enforced bedrest, is relatively ineffective.[109]

A short course of passive modalities may help alleviate some pain and allow more aggressive active rehabilitation. These include electrical stimulation, heat packs, and massage. Active exercises are then begun, starting with cervical isometrics and continuing throughout pain free ranges-of-motions. Upgrading posture, that is, chest out-head back, is also an important consideration. Advancement to cervicothoracic stabilization exercises is then begun (Figure 8-9).[111]


**Fig. 8-9.** (**a**) Cervical stabilization exercises—poor positioning with the head and neck hunched forward. (**b**) Proper alignment for cervical posture. The chin is tucked in a gravity resisted position. This exercise is a progression from (**a**). The patient is to hold an isometric contraction in this position for 10 seconds. (**c**) Advancement to single extremity weight bearing while maintaining proper cervical and thoracic positioning. Hold for 10 seconds.

Cervical epidural corticosteriod injections may also be beneficial when radicular symptoms are prominent and oral antiinflammatory agents have not been successful.[112] Epidural corticosteroid injections can be given in the field, if necessary, under sterile conditions, by a physician trained in the appropriate techniques. Ideally, these injections should be done in a hospital situation with close monitoring.

### *Brachial Plexus Injuries*

The major types of brachial plexus injuries that may be seen in the military population include penetrating trauma, traction injuries, and compression injuries.[101,113–115] "Stingers" or "burners" are probably not true anatomically defined plexus injuries, but rather root level injuries, from either traction or foraminal compression.[116] Details of these mechanisms are thoroughly reviewed elsewhere.[113,116]

***Method of presentation.*** Acute traumatic injury will follow a forceful trauma to the head and neck region.

***Tissue injury complex.*** Damage will occur at the cervical anterior and posterior horn nerve root fibers.

***Clinical symptom complex***. Severe neck and shoulder pain will be concomitant with radiation of burning and paresthesia into one of the upper extremities toward the hand. Weakness is occasionally associated, often in the shoulder musculature. The duration of symptoms is variable and frequently lasts less than 1 minute.[116]

***Functional biomechanical deficit.*** There are none (these are acute injuries).

***Functional adaptation complex***. There are none (these are acute injuries).

***Tissue overload complex.*** This will occur at the cervical nerve roots.

***Rehabilitation*** (Table 8-4). Often symptoms of acute brachial plexus injuries (or stingers) resolve rapidly. When sensory symptoms persist, pain control with narcotic and nonnarcotic medications may be necessary to allow the soldier to function with the pain. However, motor findings tend to be the more persistent neurologic abnormalities following a stinger.[116] In any type of brachial plexus injury in a military situation, early rehabilitation focuses on prevention of secondary complications from prolonged immobilization of an injured and significantly weak upper limb. Some splinting or relative immobilization may be necessary when profound weakness is present. Range-of-motion exercises are necessary to prevent adhesive capsulitis. Distal strengthening, in the case of proximal injuries, will be helpful to prevent hand swelling and reflex sympathetic dystrophy. Specific strengthening of involved muscles, initially with isometric and then isotonic exercises, is important. This therapy may be necessary for many months as residual weakness can be prolonged.

**TABLE 8-4**

**TREATMENT FOR BRACHIAL PLEXUS INJURIES**

| Time (d) | Treatment |
|---|---|
| 0–1 | Initiate splinting (if necessary) |
| 0–3 | Early ROM, Codman, and wand exercises |
| 0–7 | Pain medications |
| 3–10+ | Strengthening shoulder girdle, cervical and thoracic spine |

ROM: range-of-motion

### *Thoracic Outlet Syndrome*

Thoracic outlet syndrome (TOS) is a group of disorders attributed to the compression of the neurovascular bundle in the region of the cervical-thoracic dorsal outlet. The neurovascular bundle is a grouping of the brachial plexus nerve fibers and the subclavian vein and artery. Most symptoms of TOS affect the C-8 and T-1 nerve roots, as opposed to cervical nerve root syndromes, which most commonly involve the C-5, C-6, and C-7 levels. Details of the specific disorders that fall under the category of TOS are referenced.[117–121] Contributing factors to TOS include poor posture; muscle strength imbalances, such as weak scapular stabilizers and spinal extensors; tight pectoralis muscles; flexion extension injuries with associated muscle spasm (especially in anterior cervical and shoulder musculature); and emotional stress. The differential diagnosis of TOS includes cervical spondylosis and radiculopathy, shoulder disorders, entrapment mononeuropathies (carpal tunnel syndrome), ulnar neuropathy at the elbow, and myofascial syndromes.

***Method of presentation.*** TOS is usually manifested after chronic overuse in soldiers who use their upper extremities most of the day for activities.

***Tissue injury complex.*** Affected areas are the nerve root fibers of the brachial plexus, usually the lower trunk or medial cord, or less commonly, the subclavian artery and vein, or all three.

***Clinical symptom complex.*** There will be numbness, tingling, and weakness in the affected upper

extremity. Symptoms can be very specific, from only the medial aspect of forearm to affecting the entire upper limb.

***Functional biomechanical deficit.*** The patient will present with inflexibility of scalenes, pectoralis major and minor, and hypomobility of the first rib.

***Functional adaptation complex.*** A hunched forward posture, increased thoracic kyphosis, and increased cervical lordosis may all be evident.

***Tissue overload complex.*** Lumbar spine extensors, scapular stabilizers, and thoracic and cervical spinal extensor muscles are subject to increased stress to accommodate the functional adaptation complex.

***Rehabilitation.*** Thoracic outlet problems are most often related to chronic overuse of the upper extremity, cervical and thoracic spine, and chest musculature with resultant soft tissue inflexibility, muscle imbalances, and altered postures. Treatment starts by correcting drooping shoulders, altering and improving sitting posture, and enhancing body mechanics to attain a "chest out, head back" position.[122,123] Stretching of the pectoralis and scalene muscles is essential.[124] Often soft tissue mobilization of these structures by a qualified therapist is necessary.[123] Increased joint and soft tissue mobilization of the scapula, and scapulothoracic motion is necessary. Joint mobilization of the first rib and clavicle to restore accessory motion of the sternoclavicular and acromioclavicular (AC) joints is necessary to obtain proper upper extremity motion.[125] Strengthening exercises focus on the scapular stabilizers, that is, the serratus anterior, mid trapezius fibers, rhomboids, and erector spinae muscles (Figure 8-10). Often weight reduction and stress reduction will also decrease symptoms. Surgical intervention of either first rib resection or scalenotomy, is necessary on rare occasions.[120] With proper treatment, most cases will start to show improvement within 3 to 6 weeks.

## Shoulder Disorders

### *Rotator Cuff Injuries, Overload, and Tears*

Rotator cuff pathology and associated lesions (such as labrum tears, bicipital tendinitis, and subacromial bursitis) are some of the most common upper extremity musculoskeletal problems seen in the military population. The pathomechanics of this syndrome implicate activities that repeatedly place the arm in overhead positions.[126] The diagnosis of rotator cuff pathology is often straightforward. However, other causes of shoulder pain that can be mistaken for rotator cuff disease include proximal limb nerve entrapments (ie, axillary nerve in the quadrilateral space, musculocutaneous nerve in the biceps muscle, and suprascapular nerve at the supraglenoid fossa), brachial neuritis, AC disease, referred pain from cardiac or gastrointestinal disorders, or cervical radiculopathy.

***Method of presentation.*** Rotator cuff injuries will show subclinical functional alterations.

***Tissue injury complex.*** The rotator cuff will exhibit tendinitis, a tear, or both.

***Clinical symptom complex.*** There will be impingement with abduction and rotation, and pain with isolated resistance of the supraspinatus, both of which will cause a painful arc from 60° to 120° of abduction.

***Functional biomechanical deficit.*** Deficits will present as internal rotation inflexibility, external rotator muscle weakness, and "lateral scapular slide."[39]

***Functional adaptation complex.*** To compensate, there will be alteration of arm position for overhead activities, such as throwing and lifting; "short arming" of throw; and muscle recruitment from anterior shoulder, forearm, or trunk.

***Tissue overload complex.*** Eccentric overload will occur in posterior shoulder capsule, posterior shoulder muscles, and scapular stabilizer muscles.

***Rehabilitation*** (Table 8-5). The focus of a rotator cuff rehabilitation program is to decrease inflammation, to restore normal shoulder biomechanics, and to achieve adequate strength balance of shoulder girdle musculature. Reduction of inflammation through NSAIDs was discussed earlier in the section on tendon injuries. In a battlefield situation, subacromial bursa injection of corticosteroid can give excellent, rapid reduction of inflammation. However, corticosteroid injections can weaken musculotendinous structures and increase the risk of making a partial tear a complete one.[62] Although these risks are real, on the battlefield, the benefit of significant relief will often outweigh the risk of further damage to the tendon. Cryotherapy, TENS, and ultrasound have all been used successfully to expedite inflammation reduction of the bursa and rotator cuff tendons.[127] To discourage reinjury, the soldier can be advised to use the involved upper extremity only in positions under 90° of abduction and for light activities.

In subacute and chronic rotator cuff injuries, improvement of flexibility should be initiated as soon as any acute inflammation subsides. In particular, tightness of the external rotators of the shoulder, with resultant internal rotation deficits, needs to be

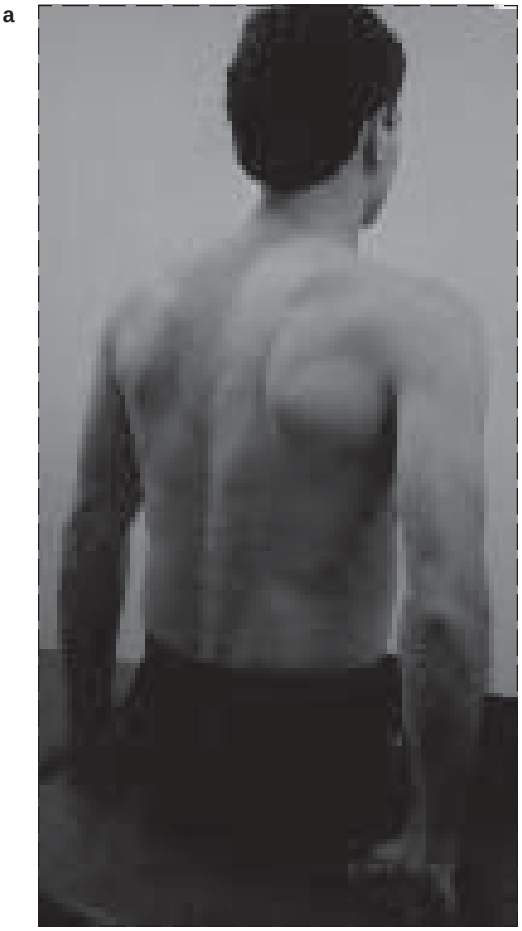
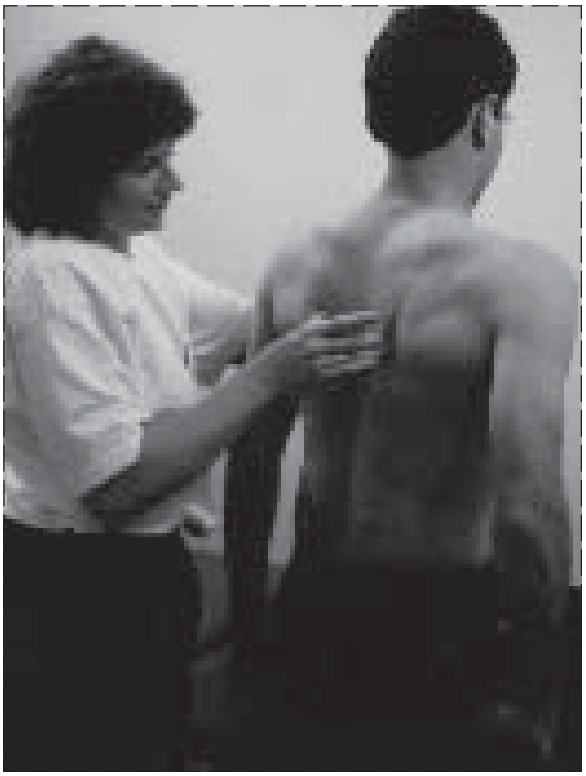



**Fig. 8-10.** (**a**) Poor scapular stabilizer strength with mild scapular winging/protraction. (**b**) Cueing the patient to activate scapular stabilizer muscles—midtrapezius and rhomboids. (**c**) Cueing the patient to activate scapular stabilizer muscles—serratus anterior.



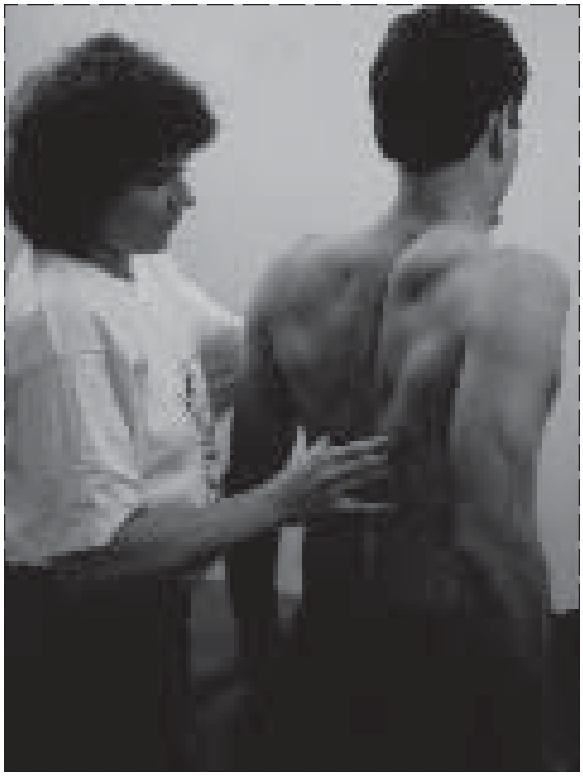



**TABLE 8-5**

**TREATMENT FOR ROTATOR CUFF TENDINITIS**

| Time (d) | Treatment |
|---|---|
| 0–7 | NSAIDs, modalities, injection |
| 3–7 | Flexibility for internal rotation |
| 3–7 | Isometric strengthening of cuff and scapular stabilizers in positions under 90° abduction |
| 7–14 | Isotonic strengthening |
| 21–28 | Overhorizontal strengthening, PNF patterns |

NSAID: nonsteroidal antiinflammatory drug
PNF: proprioceptive neuromuscular facilitation

addressed. Assessment should also be made of the motion of the sternoclavicular, AC, and scapulothoracic articulations, because movements at these joints greatly affect proper rotator cuff biomechanics and function. Deep friction massage may be of some benefit in improving range-of-motion when significant scarring has occurred and neither active nor passive stretching accomplishes the desired results.[128]

Strengthening exercises should start with shoulder isometrics in all planes. Progression is then to isotonic strengthening in the under-horizontal plane for internal and external rotation, scapular stabilizers (midtrapezius fibers, serratus anterior, rhomboids), and the biceps brachii (a humeral head depressor) (Figures 8-11 and 8-12). Emphasis on the scapular stabilizers must be appreciated because in rotator cuff injuries the clinical symptoms do not always correlate with function, and the point of clinical pathology is not always the site of muscle pathology. As described above, the tissue overload complex is the posterior shoulder muscles and the scapular stabilizer muscles. Over-horizontal exercises in nonpainful planes are begun when stretching exercises are pain free. Rehabilitation is then focused on activity specific training. Symptoms should show some improvement with acute measures in 5 to 7 days. Commonly, symptoms will take

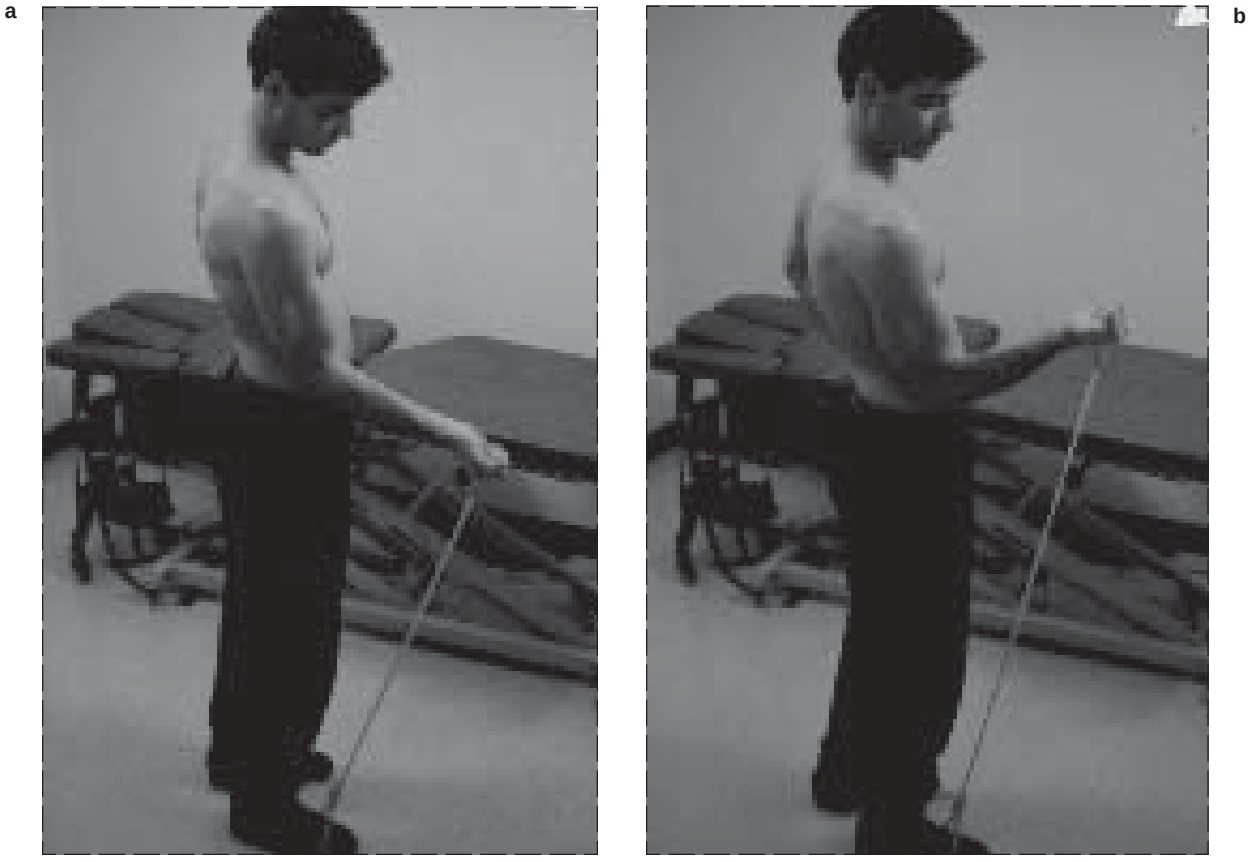



**Fig. 8-11.** Strengthening the biceps brachii muscle with a Theraband.

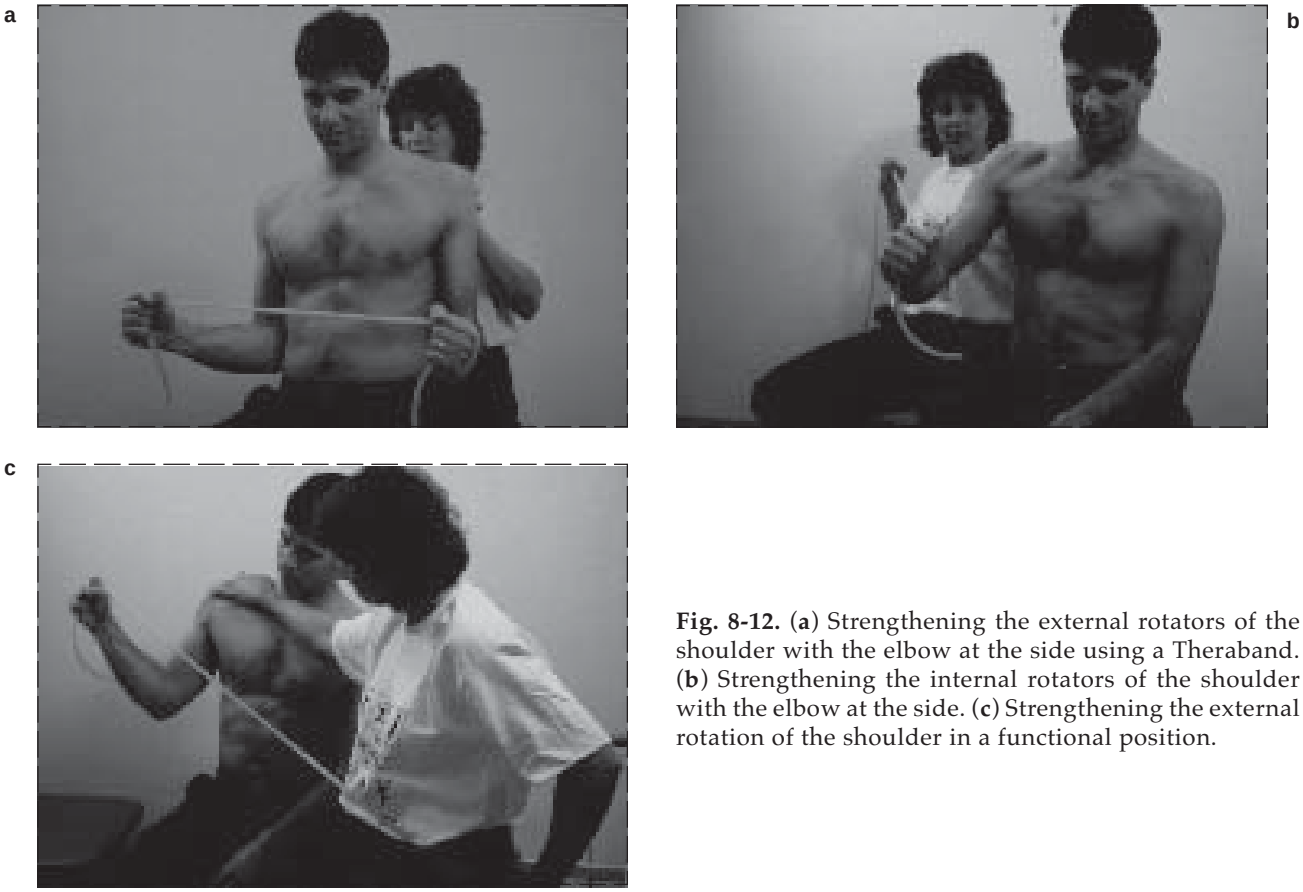



**Fig. 8-12.** (**a**) Strengthening the external rotators of the shoulder with the elbow at the side using a Theraband. (**b**) Strengthening the internal rotators of the shoulder with the elbow at the side. (**c**) Strengthening the external rotation of the shoulder in a functional position.

2 to 4 weeks of rehabilitation before significant changes in flexibility and strength are appreciated.

### *Shoulder Instability: Glenohumeral Joint*

The three articulations of the shoulder girdle (the glenohumeral joint, the AC joint, and the sternoclavicular joint) are all relatively unstable because they allow a wide range of movement in the girdle as a whole. The supporting ligaments, therefore, are relatively lax and are easily stretched.[129]

Subluxation and dislocation of the glenohumeral joint can occur in a military setting when sufficient force is applied to overwhelm the muscular and capsuloligamentous apparatus. The inferior glenohumeral ligament complex is the prime static stabilizer for anterior, posterior, and inferior stability.[129] Often, with chronic subclinical instability, subtle changes occur in the subacromial bursa area and rotator cuff tendon, causing impingement symptoms and producing attritional tears of the glenoid labrum.

***Method of presentation.*** Glenohumeral joint injuries present as an acute traumatic injury, or recurring injury to predisposed tissue, usually caused by a fall with the arm raised and outstretched.

***Tissue injury complex.*** The anterior capsule will be affected, including the anterior glenoid labrum, and superior, middle, and inferior glenohumeral ligaments.

***Clinical symptom complex.*** Pain and gross deformity of the shoulder profile with loss of normal fullness will be evident, along with the loss of mobility and the arm held away from the trunk.

***Functional biomechanical deficit.*** Weakness of anterior shoulder stabilizers (subscapularis and pectoralis major muscles) will occur.

***Functional adaptation complex.*** There is no functional adaptation complex.

***Tissue overload complex.*** The anterior labrum and glenohumeral ligaments will be stressed, especially the inferior glenohumeral ligament.

***Rehabilitation*** (Table 8-6). Restoration of normal structural integrity is the goal of rehabilitation. Descriptions for techniques of acute relocation of a dislocated shoulder are cited elsewhere.[130] Initial treatment for a dislocation will require a period of

**TABLE 8-6**

**TREATMENT FOR SHOULDER DISLOCATION**

| Time | Treatment |
|---|---|
| 0–6 wk | Immobilization in sling |
| 0–3 d | Cryotherapy, modalities, pain medication |
| 0–14 d | Isometrics for elbow, wrist, and hand |
| 7–14 d | Isometrics for shoulder girdle muscles |
| 4–6 wk | Wand, Codman exercises once splint removed |
| 4–6 wk | Passive stretching |
| 6+ wk | Strengthen isometrically and isotonically |

immobilization to allow scarring to occur over the capsuloligamentous structures that were stretched. The optimal time for postreduction immobilization for the first dislocation is 3 to 6 weeks in a sling and swathe.[131,132] Application of ice decreases tissue edema and hemorrhage, and gentle isometrics for the elbow, wrist, and hand are initiated while the shoulder is immobilized. During the period of immobilization, isometric contractions of the shoulder musculature are initiated within the patient's tolerance. Throughout this period the soldier is instructed to maintain the axis of his arm anterior to the midcoronal plane of the body, so as not to encourage anterior instability.[133] Once the sling is removed, exercises to prevent extensive adhesive capsulitis should be started; they should concentrate on gentle, passive, assisted stretching exercises to regain range-of-motion. Strengthening exercises should begin from the under-horizontal (less than 90° abduction) position to encourage stability and diminish mechanical irritation from the injured capsule and ligaments.[134] In any strengthening program, emphasis is placed on the internal rotators of the shoulder because these are the most effective dynamic restraints against anterior instability in the middle to low ranges of abduction.[135,136] Later, strengthening of the external rotators and the remaining shoulder muscles is initiated.

The total period of rehabilitation may vary from 6 weeks to 4 to 5 months. Sufficient external rotation at 90° of abduction (the position at which the shoulder is generally most vulnerable to anterior instability) should be obtained without apprehension to allow comfortable participation in full military activities. Painless range-of-motion, strength, and endurance parity are also necessary for resumption of full military duty.

### *Shoulder Instability: Acromioclavicular Joint*

The AC joint, because of its superficial position, is subject to frequent trauma. Dislocation of the AC joint follows damage to the AC and coracoclavicular ligaments. The cause is usually a fall onto the outstretched arm, elbow, or point of the shoulder so that the joint is forced inward and upward and the scapula is forced caudally.[137] Grade I injuries represent a mild sprain of the AC and coracoclavicular ligaments with no anatomic disruption of either ligament. Grade II injuries represent a partial displacement of the AC joint, less than the width of the clavicle. Grade III injuries represent complete loss of the integrity of the AC ligaments and coracoclavicular ligaments.[138]

***Method of presentation.*** All grades present as acute traumatic injuries.

***Tissue injury complex.*** The AC joint and supporting ligaments will be damaged.

***Clinical symptom complex.*** Pain over the AC joint or proximal shoulder will occur with crossed adduction of the arm at 90° of abduction across the body (Figure 8-13).

***Functional biomechanical deficit.*** There are none.

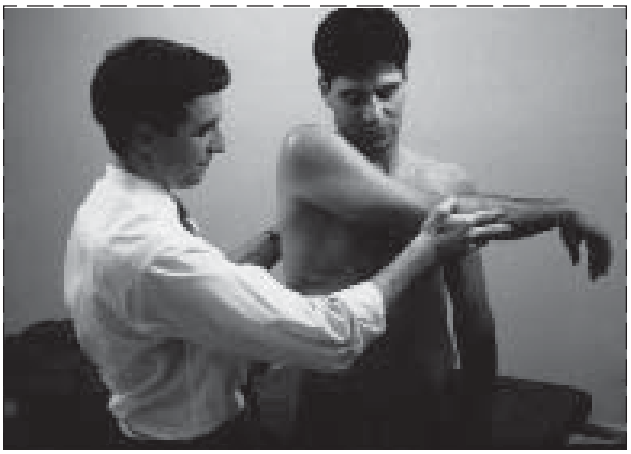


**Fig. 8-13.** Crossed adduction maneuver to check for acromioclavicular joint pathology. The arm is adducted horizontally and then some end range overpressure is exerted. Reproduction of pain over the acromioclavicular joint with this maneuver is a sign of acromioclavicular joint pathology.

***Functional adaptation complex.*** Alteration in scapulothoracic and glenohumeral motion will be present.

***Tissue overload complex.*** Stress will occur to the AC and coracoclavicular ligaments.

***Rehabilitation*** (Table 8-7). To a large extent, rehabilitation will depend on the degree of injury to the AC joint. In any injury, the initial use of ice and antiinflammatory agents may be supplemented with a local injection of anesthetic into the injured joint. This injection may serve both a diagnostic and therapeutic effect. Usually 3 to 5 $cm^3$ of 1% lidocaine will suffice to give good relief of local pain from an AC joint injury. Local padding to prevent direct pressure over the joint can be helpful.

Grade I injuries usually respond to cryotherapy and antiinflammatory medications and padding. Return to full duty should be accomplished in 2 days to 2 weeks. Grade II injuries will require a sling to support the joint until it is asymptomatic, which may range from 1 to 4 weeks. The deformity that is initially present will remain a permanent deformity.[138] Once symptoms begin to abate, a muscle strengthening program, involving the trapezius and the remainder of the shoulder girdle muscles, is initiated; but repetitive activity with the arm above the shoulder should be kept to a minimum for the first 4 weeks of treatment.[139]

Grade III injuries have more limitations of motion and a higher potential for disability. In most cases, nonsurgical treatment is still the preferred course.[138,140] Overall, a 5% to 10% incidence of significant problems can be anticipated with a Grade III AC injury, whether it is treated by closed or open means.[138] Closed symptomatic treatment requires immobilization of the AC joint with a sling for comfort. Once the AC joint is reduced, the soldier is given pain medication and instructions to keep the shoulder quiet, not to remove the sling unless there is severe pain, and report periodically for adjustments of the sling. The sling is discontinued when the symptoms allow, usually in 7 to 10 days.[138] While the soldier is still in the sling, isometric exercises are begun; the sling is loosened for range-of-motion exercises, which are performed over the AC joint with support from the therapist. Progressive resistance exercises are done as tolerated. With closed aggressive treatment, an attempt is made to keep the joint reduced for 6 full weeks, during which time isometric exercises are instituted and periodic checks of sling position are made.[138] If closed techniques do not work, or the results are unacceptable to the patient, referral for surgery is indicated. If dislocation causes a major shift in the scapular position due to the loss of the support of the clavicle, more pain symptoms may be expected. In this case, early surgery may be indicated.

**TABLE 8-7**

**TREATMENT FOR ACROMIOCLAVICULAR DISLOCATION**

| Time | Treatment |
|---|---|
| 0–3 d | Cryotherapy, NSAIDs, pain medication |
| 0–7 d–6 wk | Local padding, sling use |
| 5–10 d | Strengthen trapezius, shoulder girdle |

NSAID: nonsteroidal antiinflammatory drug

## Elbow Disorders

### *Epicondylitis: Medial and Lateral*

Medial and lateral epicondylitis are common chronic, repetitive overuse disorders seen in the military population. It is generally accepted that the primary pathology on the lateral aspect involves a microtear at the origin of the extensor carpi radialis brevis; and less commonly, the extensor carpi radialis longus and the anterior portion of the extensor communis tendon. With both, there is a formation of subsequent fibrosis and granulation tissue as a consequence of repeated trauma.[141–143] Repetitive concentric contractions of these muscles, shortening as they maintain tension to stabilize the wrist, produce chronic overload, which results in the symptoms of lateral epicondylitis.[141] Medial epicondylitis involves the pronator teres, flexor carpi radialis, and occasionally the flexor carpi ulnaris, all of which arise from the medial epicondyle of the humerus and from the fascia over it.[141] Differential diagnosis of medial and lateral epicondylitis includes cervical radiculopathy, nerve entrapment syndromes (particularly the radial nerve at the lateral elbow), or proximal radioulnar joint injuries.[141–143]

***Method of presentation.*** The insidious onset will have gradually increasing symptoms, or acute exacerbation of chronic injury, or both.

***Tissue injury complex.*** Microtears and tears will be evident in the extensor carpi radialis brevis and longus tendons, or in the flexor carpi radialis and pronator teres tendons, with angiofibromatous hyperplasia.

**TABLE 8-8**

**TREATMENT FOR EPICONDYLITIS-MEDIAL AND LATERAL**

| Time (d) | Treatment |
|---|---|
| 0–7 | Cryotherapy, antiinflammatory modalities |
| 3–5+ | Counterforce bracing |
| 3–14 | Flexibility program for involved muscle |
| 7–14 | Eccentric and concentric strengthening |
| 21–28 | Corticosteroid injection (if necessary) |

***Clinical symptom complex.*** Pain at the epicondyle can radiate distally into the forearm. Occasionally there will be weakness of grip strength and tenderness to palpation over insertion points of involved muscles. Pain will occur with resisted motion of the involved muscle.

***Functional biomechanical deficit.*** With lateral epicondylitis, there will be extensor muscle inflexibility, extensor weakness, pronation contracture, and decreased shoulder external rotation strength. The medial epicondylitis will show flexor-pronator inflexibility and weakness.

***Functional adaptation complex.*** Alteration in grip positions on military equipment will be evident, as will more use of the shoulder in throwing motion.

***Tissue overload complex.*** Lateral epicondylitis will have stress at wrist extensor (especially extensor carpi radialis brevis) and shoulder external rotators; with the medial epicondylitis, wrist flexor-pronator tendons will be affected from eccentric overload.

***Rehabilitation*** (Table 8-8). Initially, copious use of cryotherapy and judicious use of antiinflammatory medication are indicated, usually for the first 2 to 3 weeks. With epicondylar pain, the flexibility of the involved tight muscles can be improved by fully extending the elbow and either palmar flexing or extending the wrist with increasing pressure against a table (Figure 8-14). Stretching should be done several times a day. Initial strengthening can be done isometrically, with resistance from the other hand at multiple angles of wrist flexion and exten-

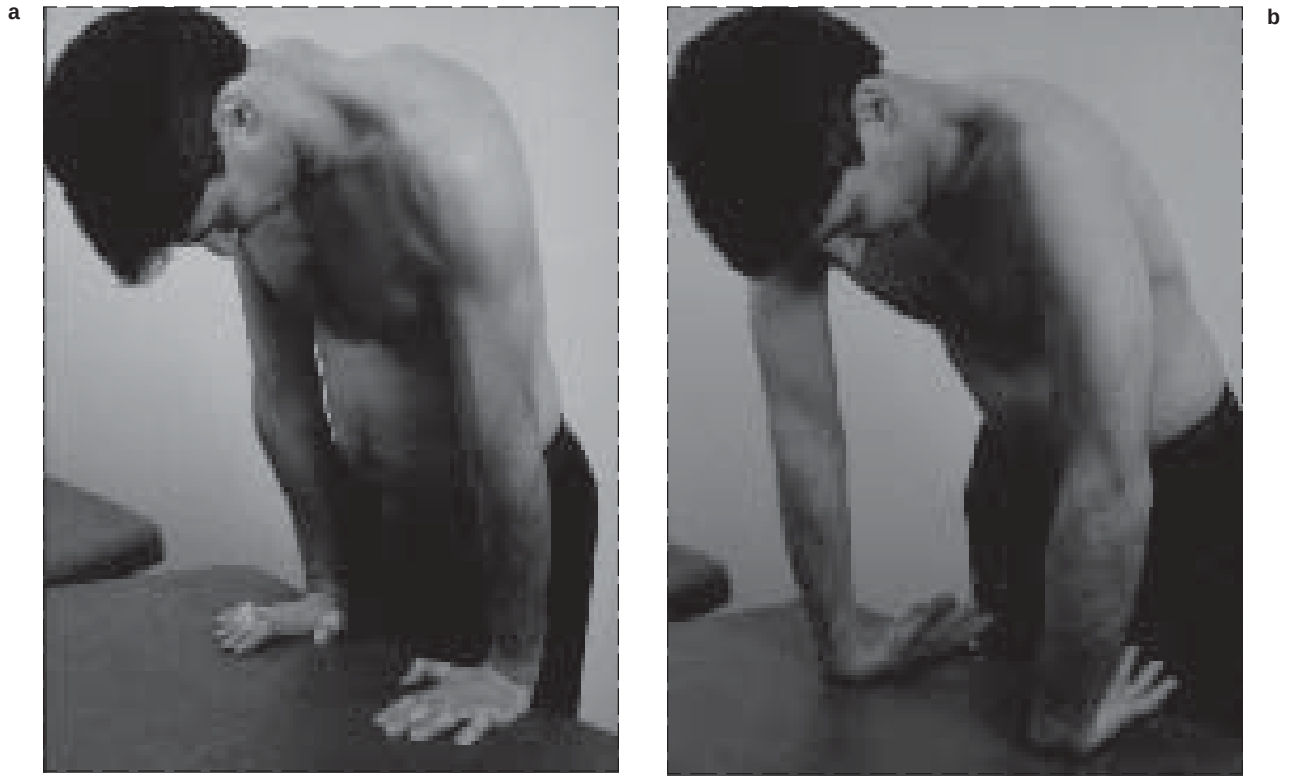



**Fig. 8-14.** (**a**) Stretching of the medial epicondylar muscles (wrist flexors and pronators). (**b**) Stretching of the lateral epicondylar muscles (wrist extensors and supinators).

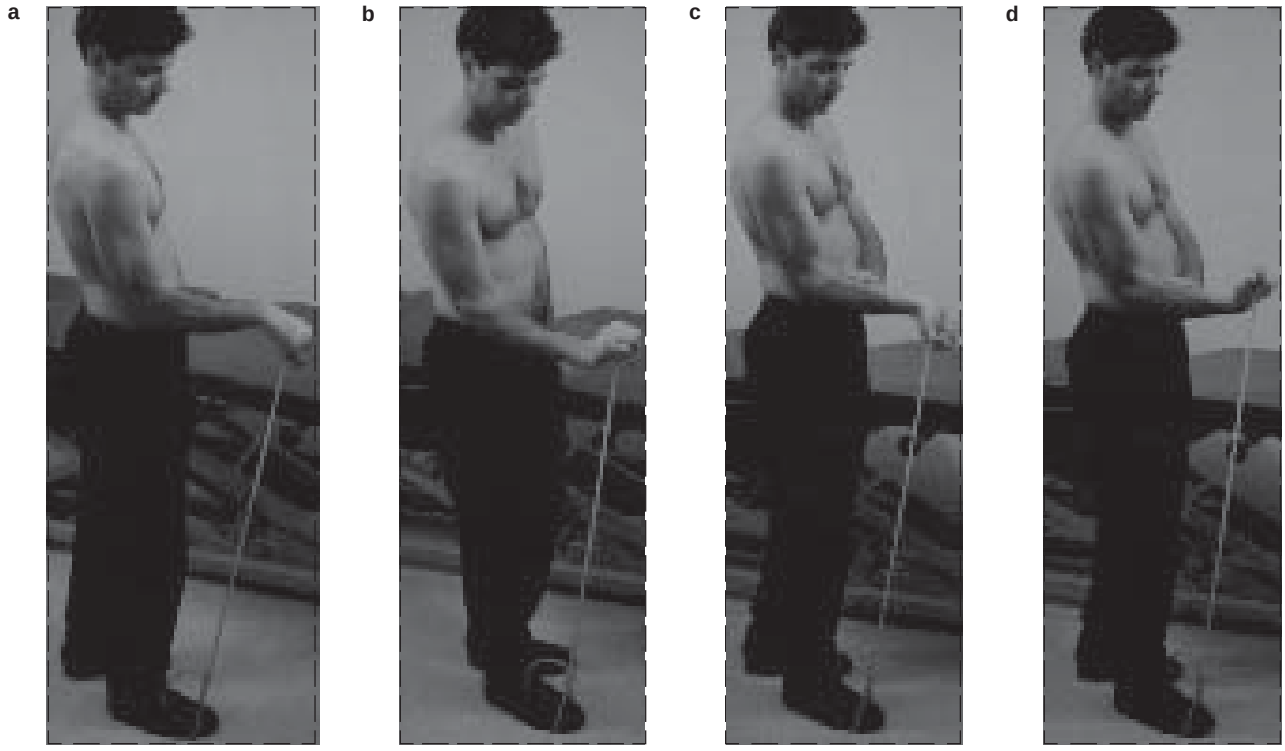



**Fig. 8-15.** (**a, b**) Strengthening wrist extensors using a Theraband. (**c**, **d**) Strengthening wrist flexors using a Theraband.

sion. Progression can then be made to wrist strengthening with the use of elastic bands or free weights (Figure 8-15). Ultimately, strengthening should be done both eccentrically and concentrically, such as with a weight tied to a piece of wood, which is slowly raised and lowered only with wrist motion (Figure 8-16).

Use of ultrasound or other heating modalities may give some pain relief, as well as loosen any scar tissue to allow better flexibility. Electric stimulation modalities may also help with pain reduction and prevention of muscle atrophy. Counterforce braces may also be helpful by distributing the forces around the elbow over a greater surface area.[144–146] For lateral epicondylitis, the brace is applied firmly around the forearm over the wrist extensor muscle mass at the elbow. It is tightened enough so that when the patient contracts the wrist extensors, they do not obtain a full contraction of the muscle; that may relieve tension on the attachment of the extensor tendon.[141]

If symptoms are not significantly reduced over a period of 3 to 4 weeks, a corticosteroid injection in the painful area may be helpful. The steroid is mixed with a local anesthetic and injected into the subaponeurotic space at the point of maximal tenderness. Vigorous activity of the involved forearm should be avoided for 2 weeks after an injection. If the patient receives some relief but still has pain that limits function, injections may be given once again. When conservative measures fail and the soldier is significantly disabled by epicondylitis, surgical release of the involved fibers should be considered.

### *Medial Capsuloligamentous Injuries*

The medial aspect of the elbow is supported by the medial collateral ligament, the medial joint capsule, and the muscle mass. In overhead activities, such as throwing, the elbow may be subject to intense valgus-tension stress. Any of the structures on the medial aspect of the elbow may become injured. Tension on the medial aspect of the elbow is first resisted by the overlying flexor-pronator muscles. These muscles may tear, or a partial avulsion of one of the tendons or muscle insertions may occur, causing valgus overload injuries. Repetitive, violent stresses will involve the deeper capsule and ligament. Tension stress that the capsule and ligament put on the ulna and humerus can lead to spur formation and, ultimately, compression of the ulnar nerve. The differential diagnosis of medial capsuloligamentous injuries include ulnar neuropathies,

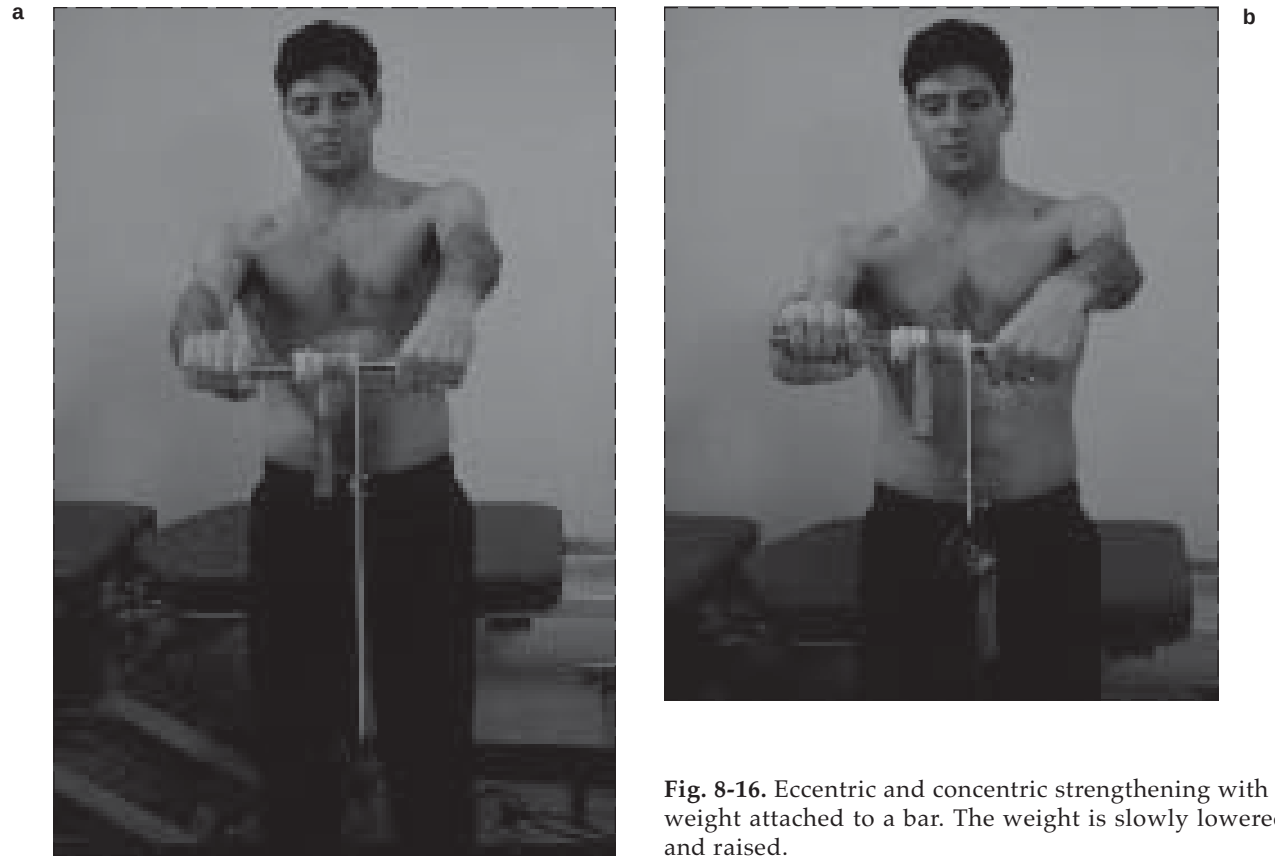



**Fig. 8-16.** Eccentric and concentric strengthening with a weight attached to a bar. The weight is slowly lowered and raised.

radioulnar joint lesions, degenerative joint disorders of the elbow, cervical radiculopathy (especially C-8 lesions), lower trunk brachial plexus lesions, and TOS.

***Method of presentation.*** The method of presentation will be acute exacerbation of a chronic injury.

***Tissue injury complex.*** Injury will occur in the medial collateral ligament, and the flexor-pronator muscle mass.

***Clinical symptom complex.*** During overhead activities, such as throwing, pain will be present along the inner aspect of the elbow. Tenderness over the medial aspect of the elbow can be intensified by applying valgus stress to the elbow at 20° to 30° of flexion. Valgus laxity may exist.

***Functional biomechanical deficit.*** The flexor-pronator will be inflexible and weak.

***Functional adaptation complex.*** There will be a loss of terminal extension of elbow (inability to do a full push-up).

***Tissue overload complex.*** Medial capsuloligamentous structures will be stressed, especially the anterior oblique band of the medial collateral ligament.

***Rehabilitation*** (Table 8-9). Treatment will begin with relative rest and the judicious use of antiinflammatory medications. In most cases, symptoms should resolve in 7 to 14 days. Antiinflammatory modalities, such as ultrasound and electrical stimulation, are useful adjuncts to early treatment. Occasionally, a local injection of 3 to 5 $cm^3$ of anesthetic over the tender area may give temporary relief if immediate relief is necessary, such as in a battlefield situation. Therapy should be directed to stretching the flexor and pronator muscles of the forearm to improve range-of-motion. Regaining and maintaining a nor-

**TABLE 8-9**

**TREATMENT FOR MEDIAL CAPSULOLIGAMENTOUS INJURIES**

| Time (d) | Treatment |
|---|---|
| 0–3 | Antiinflammatory medications and modalities, taping |
| 3–7 | Stretching flexor, pronator muscles |
| 7–10 | Strengthening eccentrically, concentrically elbow flexors |

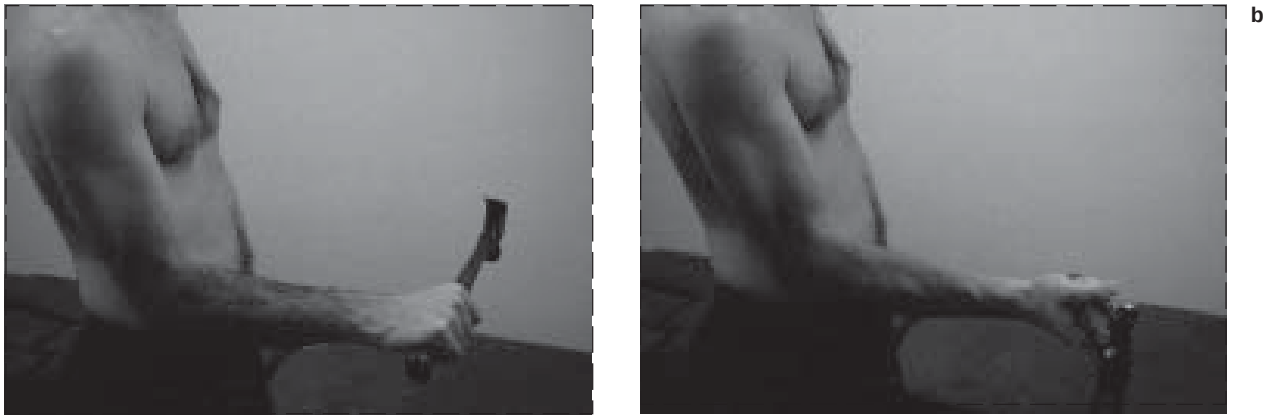



**Fig. 8-17.** Strengthening supination and pronation of the forearm by alternating forearm positions with a weighted object.

mal range-of-motion requires stretching in flexion, extension, pronation, and supination. Strengthening is also initiated, both concentrically and eccentrically. These type of strengthening exercises can be done as outlined before, with epicondylitis. Pronation and supination can be strengthened using a hammer or similar tool to produce torque throughout the full range-of-motion (Figure 8-17). Grip and shoulder exercises are also initiated.

Rehabilitation programs for elbow problems should always address proximal stability at the shoulder to allow the elbow to be properly placed in space to function appropriately. Ice may be applied immediately after exercises. If major soft tissue injuries have occurred, care must be taken to avoid early aggressive stretching because of the risk of traumatic myositis ossificans.[147] If a soldier's return to active duty is required before rehabilitation is completed, taping and bracing of the medial aspect of the elbow may be necessary for additional support. In the case of an acute rupture of the medial collateral ligament, with instability, surgical repair is indicated.

## Hand and Wrist Disorders

### *De Quervain's Tenosynovitis*

De Quervain's disease is a tenosynovitis of the first dorsal compartment. The first dorsal compartment contains the tendons of the abductor pollicus longus and the extensor pollicus brevis. These tendons are prone to inflammation from repetitive hand and wrist motions. Any of the tendons and muscles of the hand and wrist can become inflamed, especially with activities that require a forceful grasp coupled with ulnar deviation or repetitive use of the thumb.[148] Accurate and early diagnosis of tenosynovitis of the digital extensors is important, especially in the case of the extensor pollicus longus because this tendon tends to rupture.[149] The differential diagnosis of de Quervain's tenosynovitis includes distal radius stress fracture, radial neuropathy, cervical radiculopathy (especially C-6 lesions), and degenerative joint disorders of the wrist.

***Method of presentation.*** This tenosynovitis shows the acute exacerbation of a chronic injury.

***Tissue injury complex.*** Damage will occur to the synovial sheath of the abductor pollicus longus and to the extensor pollicus brevis.

***Clinical symptom complex.*** Pain and swelling will be one-half inch proximal to the radial styloid, and over the radial aspect of the wrist with ulnar deviation. There will be a positive Finkelstein's test (Figure 8-18), and an occasional palpable nodule.

**Fig. 8-18.** Finkelstein's test. Ulnar deviation of the closed fist reproduces radial sided pain over the extensor pollicus brevis and the abductor pollicus longus.

**TABLE 8-10**

**TREATMENT FOR DE QUERVAINS TENOSYNOVITIS**

| Time (d) | Treatment |
|---|---|
| 0–3 | Rest, splinting in slight extension, cryotherapy, NSAIDs |
| 3–7 | Corticosteroid injection |
| 3+ | ROM exercises within pain free range |
| 4–7 | Isometric strengthening around wrist |
| 7–14 | Isotonic strengthening |

NSAID: nonsteroidal antiinflammatory drug
ROM: range-of-motion

***Functional biomechanical deficit.*** Along with adduction, the thumb will be inflexible.

***Functional adaptation complex.*** There are none.

***Tissue overload complex.*** The abductor pollicus longus and extensor pollicus brevis tendons will have eccentric loading.

***Rehabilitation*** (Table 8-10). Initial treatment for de Quervain's tenosynovitis (the same for any of the tendinitises of the wrist and hand) consists of rest, splinting (in mild extension), use of cryotherapy, and oral antiinflammatory agents. Corticosteroid injections into the tendon sheath, if coupled with rest, can also be effective. Soluble steroids such as dexamethasone are preferable to insoluble steroids, which tend to leave a deposit. Use of 0.5 mL of dexamethasone (40 or 80 mg/mL) and 0.5 mL of 2% lidocaine is suggested.[148] Repeated injections, that is, more than three, should be avoided.[150] Early, pain-free range-of-motion of the wrist is important to avoid soft tissue contracture and scarring. Strengthening should be started isometrically and progressed throughout the entire range-of-motion, concentrically and eccentrically, as tolerated. Ultrasound treatment, followed by stretching, may be particularly helpful in chronic cases where extensive soft tissue shortening has occurred. Most cases of de Quervain's tenosynovitis should respond to appropriate treatment within 14 to 21 days. If conservative treatment fails, or if the condition becomes chronic, surgical decompression may be necessary.[151]

### *Ulnar Collateral Ligament Injury*

Injury to the ulnar collateral ligament of the thumb can be quite common. Abduction stress to the thumb while the metacarpophalangeal joint is near full extension can tear the ulnar collateral ligament. This type of injury is most often described in skiing accidents.[152] Early recognition and proper treatment is necessary to prevent instability and decreased functional use of the hand. Classifications of injury are as follows: grade I and grade II lesions are degrees of partial disruptions of the ligament; grade III lesions represent complete ligamentous disruptions. Joint stability is best evaluated by stress testing, but should always be preceded by conventional roentgenograms, when available, to determine if a large, undisplaced intraarticular fracture is present. Injuries are classified by stress testing the metacarpophalangeal joint in slight flexion and in full extension to see if any opening of the joint occurs. Comparison to the uninjured thumb is essential since there is a great variation in metacarpophalangeal range-of-motion from person to person. Differential diagnosis, in chronic cases where no history of acute injury is present, include carpometacarpal arthritis of the first digit, carpal tunnel syndrome, adductor pollicus brevis or flexor pollicus brevis strain, and C-6 radiculopathy.

***Method of presentation.*** This is an acute injury.

***Tissue injury complex.*** The tissue involved is the ulnar collateral ligament of the thumb, usually at the insertion point to the proximal interphalangeal joint.

***Clinical symptom complex.*** Pain and swelling will occur in the medial aspect of the thumb.

***Functional biomechanical deficit.*** Stability of the thumb will be lost.

***Functional adaptation complex.*** There will be a decreased grip strength.

***Tissue overload complex.*** The ulnar collateral ligament will be overloaded.

***Rehabilitation*** (Table 8-11). For a partial grade I or grade II ligament injury in which there is no in-

**TABLE 8-11**

**TREATMENT FOR HAND-ULNAR COLLATERAL LIGAMENT INJURIES**

| Time | Treatment |
|---|---|
| 0–21 d | Immobilize in spica cast |
| 21–28 d | Active ROM exercises outside of splint |
| 6–12 wk | Protective taping or silicone cast for duty |

ROM: range-of-motion

stability, treatment is immobilization in 20° of flexion in a spica cast for 3 to 4 weeks.[149,153] The interphalangeal joint is left free to allow for active motion to prevent scarring of the extensor mechanism. A removable splint is fabricated after 3 to 4 weeks, and active exercises are allowed several times a day.[153] The splint may be removed at 5 to 6 weeks for normal activity. If there is severe swelling after the initial injury, a molded volar gutter splint may be used for the first weeks until the swelling subsides (Figure 8-19). This may be followed by the application of a thumb spica cast. For participation in military duty, the thumb is protected for a total of 3 months by either taping it to the index finger in adduction or by fabricating a silicone cast.[153,154] Surgery can be reserved for cases in which there is later disability or in which the diagnosis of instability is delayed for weeks after injury[155]; or it can be performed on any unstable joint.[146,156] For many weeks after immobilization, range-of-motion exercises will be important because of soft tissue contracture.

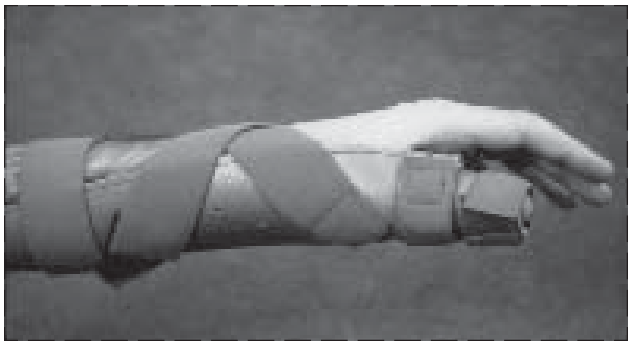


**Fig. 8-19.** Prefabricated volar gutter splint for ulnar collateral ligament injuries.

### Lumbar Spine Disorders

Lumbar spine problems in the military population are quite common, and may parallel prevalence and incidence figures in the general population. Occupational and workplace factors clearly contribute to the development of low back pain, and in some cases, low back disability. Intrinsic to most occupations with a high risk of low back pain is a composite of repetition and force, and workers in occupations requiring high repetition/high force activities are more likely to sustain injuries. These types of activities are quite common in the theater of combat. The physical demands and potential injury mechanism of any particular job can be segregated into chronic repetitive overload such as bending, twisting, and vibration; or acute dynamic overload such as heavy lifting, slips, and falls. Details of these mechanisms of injury can be found in numerous publications, which are cited in the references section of this chapter.[157–166]

#### *Diagnosis*

Establishing a specific diagnosis in the acutely or subacutely injured soldier can lead to directed treatment that may allow quick, nonsurgical resolution of symptoms and early return to duty. In general, an adequate assessment of back pain requires (*a*) an intrinsic knowledge of spinal biomechanics and the degenerative spinal cascade, (*b*) integration and proper interpretation of imaging and electrophysiologic studies with clinical decision making, and (*c*) a healthy index of suspicion for both missed diagnoses, and signs and symptoms of disability and nonorganicity. An excellent review of these issues can be found in an article by Weinstein and Herring.[167] Identification of the tissue injury complex and the method of presentation of the injury are some initial steps. A complete history and physical examination should be performed for all soldiers with back pain to ensure that referred intraabdominal or intrathoracic pain is not the cause of the symptoms. Likewise, a history of fevers, chills, or constitutional symptoms should alert the clinician to the possibility of a tumor or infection of the spine. Iritis, uveitis, urethritis, a history of recent sexually transmitted disease (chlamydia) or a history of psoriasis raises the possibility of a spondyloarthropathy (Reiter's syndrome, ankylosing spondylitis). However, the vast majority of spine problems result from biomechanical and anatomic factors as well as repetitive overload, and are described below.

To categorize lumbar spine dysfunctions in a clinically useful way, the classification system of Kirkaldy-Willis and associates[168] may be helpful. Separating the three-joint complex into its component parts, namely discogenic and posterior element (joint), allows appreciation of different clinical presentations in the various phases of degeneration (dysfunction, instability, stability), and an understanding of the interaction of the individual components that lead to various types of spinal stenosis (Figure 8-20). Each phase will be discussed in regards to pathomechanics of the disk and joint, presenting symptoms and signs, and pertinent clinical correlates. It should be noted that the initial presentation of symptoms can occur anywhere along this continuum, implying that subpain threshold

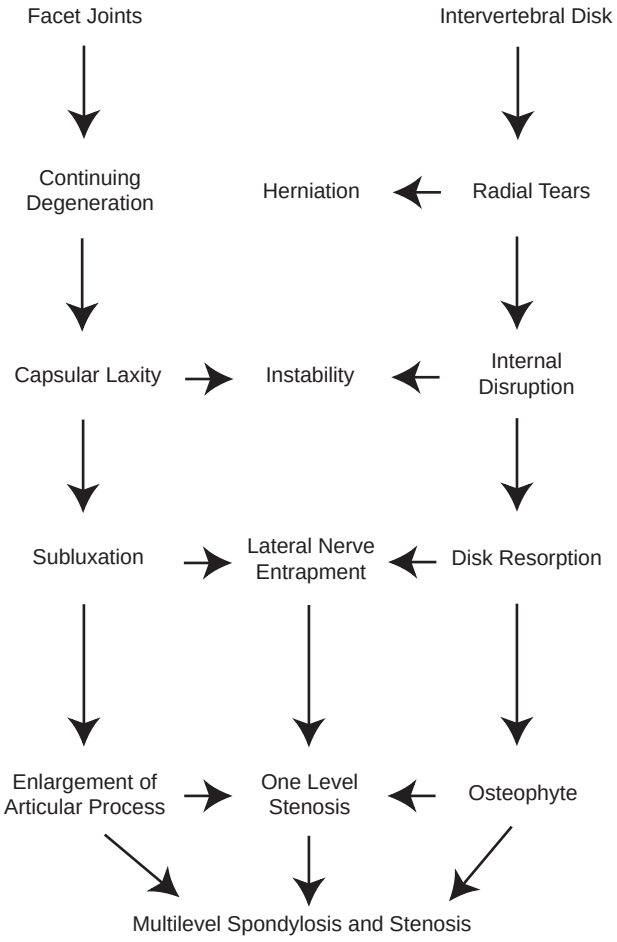



**Fig. 8-20.** Kirkaldy-Willis' degenerative cascade of the lumbar spine. Adapted with permission from Kirkaldy-Willis WH, Wedge JH, Yong-Hing K, Reily J. Pathology and pathogenesis of lumbar spondylosis and stenosis. *Spine*. 1978;3(4):320.

degenerative changes occur throughout life.[169–171] Also, degenerative changes in one motion segment may predispose to a similar process in adjacent segments. In one individual, different phases of the degenerative cascade can be found in different motion segments of the lumbar spine. Not uncommonly, dysfunctional (or hypomobile) segments might occur adjacent to a hypermobile segment. Lastly, the aging process does not always correlate with the clinical phase of degeneration.

***Phase I***. Phase I of the degenerative cascade is described as segmental dysfunction: a state of abnormally reduced, albeit subtle, movement of the motion segment. The initial clinical presentation in this phase usually reflects joint dysfunction as opposed to disk herniation. Facet joint dysfunction can include reactive synovitis and articular cartilage degeneration, resulting in joint pain, inflammation, and hypomobility. Abnormally sustained muscle contraction, especially of the short segmental extensors and rotators, can contribute to limited joint motion. The clinical presentation may be one of the classic sprain-strain syndrome. Low back pain may be worse with static standing, walking, extension, or extension combined with rotation; it may be somewhat relieved with flexion. However, even flexion may irritate a restricted joint capsule or inflexible musculotendinous unit in spasm. Local tenderness and muscle spasm, limited range-of-motion, and normal neurological examination are usual findings. Typically, posterior element dysfunction results in either nonradiating low back pain or referred pain to the buttock or proximal thigh.[172–174]

In this first phase, disk degeneration, including annular fiber tears, does occur. Joint dysfunction may not allow adequate load bearing and thus, transfers increased stress across the intervertebral disk. Radial tears are more likely to result in disk protrusion or herniation in the latter stages of dysfunction, but circumferential tears may also be painful because the outer annulus is supplied with nociceptive fibers. Classic discogenic radiculopathy can occur acutely, however, especially with a sudden, dynamic overload.

The typical presentation of discogenic low back pain is pain exacerbated by flexion oriented activities (sitting and bending,) activities that increase shear stress across the annulus (twisting), and activities that cause a valsalva-type maneuver (coughing or sneezing). The disk protrusion or herniation is usually in the posterolateral direction where the annular fibers are not well protected by the posterior longitudinal ligament, and where shear forces are greatest with forward and lateral bending. This clinical picture is not always present, however, and experience has shown that atypical discogenic pain can be seen, especially in young recruits and people with central disk protrusions or herniations. The usual clinical features of lumbar radiculopathy include leg pain greater than back pain, and paresthesias or weakness or both in a given dermatomal distribution; these are exacerbated by flexion activities, and relieved with extension activities. The degree of low back pain associated with disk protrusion or central herniations may be substantial, possibly due to the large number of free nerve endings in the posterior longitudinal ligament. The ratio of back pain to lower extremity pain varies, but with an extruded or free fragment resulting in radic-ulopathy, unless it is massive, low back pain may be minimal or absent. Lower extremity pain

may also diminish, and a neurologic deficit, that is, motor, reflex or sensory (or both) will become the predominant clinical feature.

***Phase II.*** Phase II refers to a condition of excessive segmental motion, segmental hypermobility and, occasionally, frank segmental instability. This is a phase that is clinically more difficult to conceptualize. The objective findings of a "degenerative lumbar disk" by imaging studies presents a challenge to the practitioner in defining the source of pain, especially lumbar radicular pain or radiculopathy. As the border between these phases is somewhat arbitrary, a patient may who has never had discogenic low back pain (or for that matter, has never before been symptomatic) may enter Phase II.

Abnormalities of the facet joints include capsular laxity and joint subluxation as defined by Kirkaldy-Willis and associates.[168] Such movement may not be detectable by standard radiography, including lateral flexion and extension roentgenograms as no translation may occur, but the instantaneous center of rotation may move abnormally.[175] This also relates to our clinical impression that the quality of motion is more representative of pathology than is the quantity. Measuring gross range-of-motion allows evaluation of soft tissue extensibility and an ability to monitor the effects of treatment but does not provide information about segmental motion that could be obtained by a skilled manual examination. Observing dysrhythmia, a catching sensation or painful arc with recovery phase from forward bending, can be indicative of hypermobility. Another sign of a hypermobile segment is tenderness or spasm, or both, that is elicited by applying a torsional stress across a specific segment, such as, lateral springing on the spinous process (Figure 8-21).

**Fig. 8-21.** When lateral springing of a spinous process reproduces the patient's low back pain, it is suggestive of a hypermobile segment.

Disk related abnormalities in Phase II can include internal disk disruption and narrowing of the intervertebral disk space. An internally disrupted disk evolves as the process of degeneration progresses. Multiple annular tears develop that allow random distribution of nuclear material throughout the disk. A disk that is internally disrupted may be a pain generator. The source of the pain may result from hyperexcitability of the annular nociceptive fibers, or from neurobiomechanical factors. In this phase the disk is much less tolerant of torsional stress. Typically, torsional and compressive loads occur simultaneously and the compressive force assists with stabilizing the three-joint complex by loading the annular fibers.[176] As the annular fibers become less competent, torsional load results in a greater degree of "free play," and potentially a higher degree of symptoms. The reduction in disk space height promotes facet joint laxity, reduction in the size of the intervertebral foramen, and lateral recess narrowing. Radiculopathy can occur secondary to a variety of causes, including direct impingement on the lumbar or sacral nerve roots from a herniated disk, dynamic lateral entrapment due to narrowing of the lateral recess, and primary radiculitis due to neurobiochemical factors associated with an internally disrupted disk.

Radicular pain from any of these causes can occur in the absence of back pain. Hip girdle pain (buttock or trochanteric area, or both), thigh, or leg pain all may be precipitated through a number of dynamic clinical tests that cause forward displacement of the superior articular facet toward the vertebral body, thus narrowing the lateral recess medial to the intervertebral foramen.

***Phase III.*** The process of segmental stabilization occurs over time. Pathomechanically, the facet joints become fibrosed, enlarged, and arthrosed. The intervertebral disk becomes increasingly degenerated and desiccated, allowing approximation of the ver-

tebral end plates and osteophyte formation. This combination of anterior and posterior changes can manifest in ankylosis of the motion segment, although lesser degrees of spondylosis are common. Limited range-of-motion and stiffness may become the predominant features, not unlike those observed in the dysfunctional stage. Low back pain may no longer be prominent, although severe degenerative changes may not permit this process of autofusion and, therefore, continuation of symptoms can occur. Symptomatic degenerative facet joints usually cause a low degree of aching low back pain, possibly bilaterally and sometimes at multiple levels.

In this phase, spinal nerve root entrapment is relatively common. Fixed lateral stenosis, central stenosis, and degenerative spondylolisthesis can all result in radicular signs and symptoms. Bilateral and multilevel radiculopathy may be seen. Central and lateral stenoses can coexist at the same or different levels. Neurogenic or pseudoclaudication is the typical presentation of lumbar radiculopathy in this phase.[177] The dermatomal symptoms of paresthesias, dysesthesias and myotomal distribution of muscle cramping and weakness, consistently occur with erect posture or exercise, including standing and walking. Typically these symptoms resolve with flexion maneuvers, such as forward bending or sitting. The mechanism of symptoms may represent venous engorgement of the cauda equina. Differentiation from true vascular claudication of the lower extremities is possible by the lack of (or the delayed onset of) symptoms when the lower extremities are exercised in a flexion posture, such as stationary bicycle riding. Unlike radiculopathy associated with disk herniation, straight leg raising is usually unremarkable and may be a clinical sign to differentiate spinal stenosis from the more unusual presentation of a disk herniation in this phase. The prevalence of disk herniations diminishes in people over the age 55[178] and these are typically far lateral herniations reflecting biochemical alterations of the disk, reduction in elasticity, and degenerative bony constraints. Far lateral herniations may have clinical features not unlike spinal stenosis.

On the battlefield, imaging techniques are not available for assisting in diagnosis of lumbar pain. However, in combat hospitals some imaging is available, and its greatest usefulness will be to rule out associated fractures as a cause of or contributing factor to low back pain. As with any diagnostic tool, imaging techniques of the lumbar spine must be utilized and interpreted in the context of clinical relevance.[179] More advanced imaging techniques may be available at corps level hospitals. In evaluating and treating low back disorders, it is very useful to have knowledge of the relative specificity and sensitivity of various techniques, including plain radiographs, computed tomography (CT) scans, multiplanar reformatted CT scan, myelograms, myelograms combined with CT scans, magnetic resonance imaging (MRI), discography with and without CT scans, and bone scans and single photon emission computed tomography scans (SPECT).[169,171,180–194] Although no radiographic equipment is available in the field of combat, knowledge of the radiographic tests that are useful for specific disorders is helpful in treating low back disorders, including military personnel, as well as injured soldiers in triage hospitals.

In general, plain radiographs of the lumbar spine have low specificity and sensitivity in regards to intrinsic lumbar spine pathology.[195] As previously reviewed, lateral flexion-extension roentgenograms may not provide additional information regarding lumbar hypermobility[183,187] or instability, except in cases of spondylolysis. Facet fracture or spondylolysis may not be appreciated without oblique radiographs, and although plain radiographs may not determine the age of such bony abnormalities, further specific imaging (ie, a SPECT bone scan)[182] can be directed.

The role of noncontrast enhanced CT scans is probably limited in the evaluation of clinically significant abnormalities, unless fracture or other primary bone pathology is being considered. CT scans have low specificity, as shown by the fact that 35% of asymptomatic individuals had radiologist-determined abnormalities on CT scans.[171] Although CT scans may reveal disk herniations, occasionally the distinction between disk material and thecal sac is difficult. Further, extruded disk fragments can be missed and the source of a migrated fragment may be difficult to determine. Typical lumbar CT scans are limited to axial images through the lowest three disk spaces, often skipping the lateral recesses and rarely evaluating the higher lumbar disks or conus medullaris. Myelography, combined with postmyelogram CT imaging, is an effective means of evaluating lateral stenosis, assuming the images are obtained in an overlapping manner, and can also evaluate for lateral disk herniations.[196] Myelography alone will typically miss any lesion that is lateral to the pedicle, such as a far lateral herniation or impinging lateral osteophyte.

With estimates of up to 99% sensitivity, high resolution MRI is extremely reliable in identifying lumbar disk herniations.[184,192] The sensitivity may diminish when evaluating for spinal stenosis, annular fi-

ber tears and even degenerative disks. MRI scans may have limited specificity, providing too many false positives, as demonstrated by Boden and colleagues[169] in a study of abnormal MRI findings in asymptomatic people. Nevertheless, MRI is an excellent tool for evaluating disk herniations at multiple levels, and it frequently supports the possibility of internal disk disruption as an etiology to chronic pain.

Selective injections, including epidural injection, facet joint and selective nerve blocks, are an extremely useful adjunct to other clinical diagnostic evaluation tools. These injections can also potentially provide therapeutic benefit if administered with a corticosteroid. From a clearly diagnostic viewpoint, selective injections can aid in identifying clinically significant lumbar spine pain generators. Differentiating the qualitative and quantitative contribution of the posterior element, discogenic, or radicular pain is the goal of diagnostic selective blocks. Determining a specific pain generator requires precise needle localization, by use of fluoroscopy and contrast dye.[197–202] However, in a combat area, using proper technique and a skilled clinician, these injections can be done with fairly accurate localization to allow some useful information as to the relative proportion of pain generated by a given structure. If necessary, a soldier who requires further evaluation can have these studies done with fluoroscopy and contrast agents in a major medical center.

Another type of injection that can be helpful, particularly in a combat situation, is a trigger point injection. When specific, reproducible myofascial tender (trigger) points are prominent and appear to be causing significant pain and disability, the pain-spasm cycle can be broken to provide temporary relief by either dry needling or a local injection of 1 to 3 $cm^3$ of lidocaine. These injections work best when the number of tender or trigger points are limited (fewer than three), are well localized, and easily reproducible. Trigger point injections are generally very safe and easy to perform and can be done in a combat field situation.

### *Rehabilitation*

Rehabilitation principles can be applied to all spinal disorders whether these occur in acutely injured or chronic pain patients, injured soldiers in the field of combat, nonsurgical, or postsurgical patients. The benefits of appropriate rehabilitation techniques become more apparent with evidence that even large, extruded disk herniations have, in some circumstances, spontaneously resolved over 1 to 2 years without surgery.[203,204] In field hospitals in the communication zone, aggressive nonsurgical rehabilitation of spine disorders takes on even greater importance in reconstituting our forces in a timely manner.

Any successful spine rehabilitation program relies on adequate diagnoses, which address (*a*) pathoanatomy, (*b*) stage of healing, (*c*) functional phase of spinal degeneration, and (*d*) identification of secondary soft tissue changes. Selected spinal disorders will be discussed in terms of their presentation; tissue injury; and clinical symptom, functional biomechanical, adaptation, and tissue overload complexes. In this way appropriate specific rehabilitation issues will be discussed as predicated on specific anatomic, biomechanical, and functional diagnoses. The section on rehabilitation of acute muscle strain outlines the principles of spinal rehabilitation that are applicable to all disorders of the lumbar spine and should be included in the specific rehabilitation programs for the specific disorders listed below.

***Acute discogenic pain.*** The method of presentation is as an acute injury, occurring with flexion and rotation of the lumbar spine with a patient history of previous episodes of low back pain that have usually resolved unremarkably within 3 to 5 days.

***Tissue injury complex*** includes annular fibers of the disk and chemical or mechanical (or both) irritation of the anterior and dorsal spinal roots, or posterior longitudinal ligament and other pain-sensitive intercanal and foraminal structures, or all of these.

The ***clinical symptom complex*** will be low back and leg pain. Leg pain is usually much more pronounced than back pain when herniation of the disk occurs, whereas more back symptoms occur when there is an annular tear without protrusion of the disk. Pain can occur at any level of the lumbar spine, most commonly at L-4–L-5 and L-5–S-1 areas. Pain is usually worse with flexion and flexion and rotation, and is often relieved with extension.

***Functional biomechanical deficit*** will show soft tissue inflexibilities (muscle, fascia, ligament) due to spasm or tightness, and segmental hypomobility.

***Functional adaptation complex*** will include the loss of normal lumbopelvic rhythm, increased lumbar lordosis, increased loading of posterior elements, and lateral pelvic shift. This will result in ***tissue overload complex,*** including annulus fibrosis, nucleus pulposus, and to the supporting paraspinal ligaments and musculature.

***Rehabilitation*** procedures are shown in Table 8-12, but education is probably the most important com-

**TABLE 8-12**

**TREATMENT FOR LUMBAR SPINE-ACUTE DISCOGENIC PAIN**

| Time (d) | Treatment |
|---|---|
| 0–1 | Proper positioning, 90°/90° traction, education |
| 0–3 | Bedrest, antiinflammatory modalities |
| 0–7 | NSAIDs, pain medications |
| 3–10 | Begin extension exercises |
| 3–10 | Traction (often in extension) |
| 10+ | Flexibility and strength training progressed |
| 7–14 | Walking, cross-country ski machine |

NSAID: nonsteroidal antiinflammatory drug

ponent in any back care program, including this acute injury.[205] Instruction in sitting and standing (maintenance of lordosis) and body mechanics is aimed at protecting injured structures and preventing further injury. Review of activities of daily living and nightly living allow practical application of these principles. A short course of bedrest may be appropriate. Prolonged absolute bedrest greater than 3 days has not been shown to reduce disability or dysfunction.[206] In the early stages, modalities may be helpful. Cryotherapy and electrical stimulation are effective for pain control and antiinflammatory effects. The use of NSAIDs and other analgesic medications is advocated early in the treatment of acute discogenic injuries, especially within the first 1 to 2 weeks, as has been previously discussed.

Initial exercises, movement into flexion or extension, depend upon which activity centralizes low back pain (ie, less radicular pain), or does not exacerbate low back pain. Usually, extension exercises are begun by lying prone, with support under the stomach to maintain a neutral position; and progress as tolerated to lying prone, unsupported, with support under the chest; to lying prone on elbows; and then to press-ups (Figure 8-22).[207–210] Lateral trunk shifts must be corrected before initiating extension exercises, or symptoms are likely to increase. Patients can be instructed in self-correction techniques. Theoretically, extension exercises may be effective in reducing pain by decreasing tension in the posterior annular fibers; increasing mechanoreceptor input, which activates the gate mechanism[211]; decreasing tension on the nerve root[212]; changing intradiscal pressure[213]; and allowing anterior migration of the nucleus pulposus.[208] Repeated extension posturing in standing, for use after sitting and forward bending activities should be taught. Contraindications to extension exercises include segmental hypermobility or instability; large or uncontained herniation; bilateral sensory or motor signs, or both; significant increase in low back pain unless associated with concomitant reduction in radicular pain; and increase in radicular sensory disturbance. If hypermobility exists at a segment adjacent to a disk herniation, manual blocking of extension at that level can be applied by the therapist, and patients can be taught to generally reduce motion at the lower lumbar segments. Care must be taken to prevent secondary hypermobility at the thoracolumbar segment, however, which can also occur if extension exercises are emphasized in a patient with lumbar segmental hypomobility. Hyper- and hypomobility are often clinically difficult to assess unless the examining physician has good manual and palpatory skills. Occasionally, hypermobility may be seen on flexion/extension radiographs of the spine where increased motion is occurring at specific segments with varying positions. However, in general, if the specific type of exercise program is increasing the severity and duration of symptoms, it should be avoided and other types of exercises should be contemplated.

The classic Williams flexion exercises[214] may decrease the compressive load to the posterior disk and open the intervertebral foramen, thus, its long history of use in acute disk presentations. Indeed, flexion exercises may be better tolerated in central

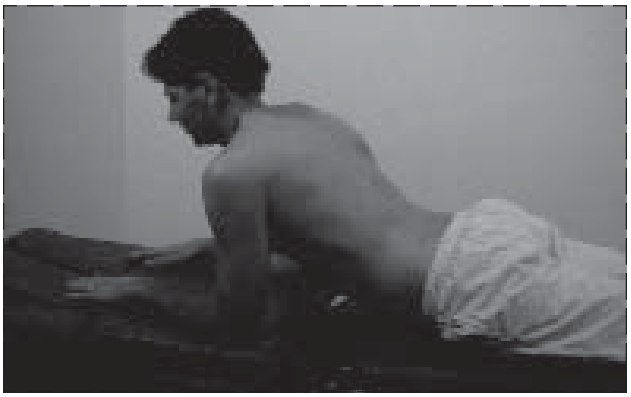


**Fig. 8-22.** Extension positioning of the lumbar spine with the patient prone and on the elbows.

disk herniations, but acute dural tension is most likely aggravated by flexion postures. Flexion does increase intradiscal pressure and may not be the best position for soldiers with acute disk symptoms. Therefore, in patients with acute discogenic pain, extension exercises, rather than Williams flexion exercises, would probably be the preferable regimen, unless a central disk protrusion is present.

Traction may be an effective treatment for acute discogenic low back pain.[215] Traction can be applied manually, mechanically (typically on an intermittent basis), through the use of gravity (either in an upright or inverted position), by means of autotraction (in which the patient controls the force), and in specific positions of side-lying, usually for far lateral disk herniations. Traction applied in a prone lying position, and neutral to extended lumbar postures can be maintained. Inversion traction has the added advantage of combining extension exercises with the benefits of traction.[215] Although originally felt to decrease intradiscal pressure, actual reduction in pressure is only to 20% to 30%.[213] Traction may be more effective in allowing vertebral body separation, decreasing compressive forces on the lumbar nerve roots by widening the foramen, improving blood flow to the nerve roots, and stretching spinal musculature. The applied force needed to adequately distract the vertebral bodies in a horizontal plane is 25% to 50% of body weight.[215] Less force does not overcome the friction component, and more force will pull the entire body. Inversion gravity traction can distract the lumbar vertebral bodies by 0.3 to 4.0 mm, which can reduce symptoms. Side effects of inversion therapy, however, are not benign and include hypertension, headaches, gastrointestinal reflux, and ruptured berry aneurysms.[216]

Early activity is encouraged. Walking, swimming, or using a cross-country ski machine helps to maintain muscle tone and cardiovascular endurance, provides extension for the lumbar spine, maintains joint mobility, and increases blood flow to the injured segment. Specifics of flexibility and strength training will be discussed later in this chapter.

***Acute Posterior Element Pain.*** Acute posterior element pain is often an acute injury that occurs with the extension and rotation of the lumbar spine, and is usually related to a torsional load on the spine.

***Tissue injury complex*** includes the zygapophyseal joints and surrounding synovium and joint capsule. Also, the posterior longitudinal ligament or intraspinous and supraspinous ligaments may be involved.

**TABLE 8-13**

**TREATMENT FOR LUMBAR SPINE-POSTERIOR ELEMENT PAIN**

| Time (d) | Treatment |
|---|---|
| 0–3 | Cryotherapy, flexion postures, NSAIDs, pain medication |
| 0–3 | Relative rest |
| 3–10 | Progressive flexion exercises |
| 3–10 | Traction (in flexion postures) |
| 7–14 | Walking, exercise bike, treadmill |

NSAID: nonsteroidal antiinflammatory drug

***Clinical symptom complex*** includes nonradiating low back pain, or referred pain to the buttock or proximal thigh will be present, but rarely below the knee.

***Functional biomechanical deficit*** will present as abnormal pelvic tilt and hip rotation secondary to tight hamstrings, hip rotators and quadratus, and weak erector spinae and hamstrings.

If the condition is chronic, the ***functional adaptation complex*** will appear as flattening of lumbar lordosis, rotation or side bending at the sacroiliac or thoracolumbar area.

Initial ***tissue overload complex*** will be at the synovium and capsule of zygapophyseal joints; when the injury is more chronic, the articular cartilage of zygapophyseal joints will be involved.

***Rehabilitation*** for acute facet joint pain is outlined in Table 8-13, and the initial treatment stages are similar to acute disk pain, that is, education, ice, relative rest, and maintaining positions of comfort. These positions are typically accomplished through neutral to flexed postures. Greater relief may be obtained by hook lying, or 90°/90° positions, that is, hips and knees both flexed at 90° (Figure 8-23). Flexion exercises are theoretically effective by decreasing facet joint compressive forces, stretching hip flexors and lumbar extensors, and strengthening abdominal and gluteal muscles.

Pelvic tilts, or flattening the low back and decreasing the degree of lumbar lordosis, should be performed in multiple positions, including bent knees, straight legs, and standing. Pelvic tilts unload the facet joints and aid in pelvic awareness. Single knee-to-chest maneuvers help stretch the contralateral hip flexors and ipsilateral extensors,

**Fig. 8-23.** The 90°/90° position to decrease the load on the intervertebral disc and lumbar spine with the hips and knees flexed at 90°.

while double knee-to-chest positions promote stretching of the lumbar and hip extensors. Stretching the hamstring and internal and external rotator hip is also essential. Partial sit-ups strengthen the abdominal muscles. Contraindication to flexion exercises include segmental hypermobility or instability, increase in low back or peripherilization of pain into the lower extremity, or both.

Intermittent mechanical traction in a 90°/90° lying position or inversion gravity traction may be helpful in unloading the posterior elements, lubricating the joints, improving joint nutrition, and reducing pain through mechanoreceptor input. When traction equipment is not available, such as in a combat situation, simply positioning the patient in the 90°/90° position can be helpful. Sustained or static traction will often exacerbate symptoms due to stretch of the facet joint capsule. Prone lying positions should also be avoided.

Maintenance of aerobic activity is essential. It can be accomplished by a stair climbing machine, a treadmill at a slight incline, or a stationary bicycle, if available. These also help to maintain a neutral-to-flexed posture.

### *Pars Interarticularis Injury*

Assessment of the soldier, particularly the young recruit, with localized lumbosacral discomfort should alert the physician to the possibility of spondylolysis or spondylolisthesis. Injury of the pars interarticularis is particularly common in active younger individuals who perform repetitive activities in lumbar extension, flexion, and rotation. These lesions include pars stress reaction, spondylolysis (isthmic) and spondylolisthesis. A pars stress reaction is a bony irritation to the pars interarticularis without the presence of a lytic lesion.[217] This prespondylolytic state may progress to a true spondylolysis. Spondylolisthesis occurs if this defect is bilateral, and forward slippage of the superior vertebral body on the one below occurs, particularly L-5 on S-1.

If a pars injury is clinically suspected (see *clinical symptom complex*, below), a radiographic assessment is indicated. Plain radiographs with an oblique view may show the spondylolytic defect with or without slippage. Not all pars interarticularis injuries will be apparent on radiograph.[218,219] A planar bone scan may demonstrate increased uptake in the pars region when plain radiography is normal. Even when the plain radiograph demonstrates a defect in the pars interarticularis, the planar bone scan is helpful to determine if the fracture is acute or chronic.

A pars interarticularis injury will present as a chronic overload injury, with the *tissue injury complex*, including pars interarticularis, either unilateral or bilateral, usually at L-4 and L-5, where maximum loading and shear occur.[220–222]

The ***clinical symptom complex*** will cause localized lumbosacral discomfort, which will be worse with extension and partially relieved with flexion. This is more common in soldiers whose activities require repetitive flexion, extension, and rotation. A normal neurologic examination is usually indicated.

***Functional biomechanical deficit*** will be hamstring tightness, and the ***functional adaptation complex*** will be loss of lumbar lordosis. The ***tissue overload complex*** will be pars interarticularis.

***Rehabilitation*** is outlined in Table 8-14. Management is often controversial and ranges from rest and restriction of activity to rigid immobilization. It is

**TABLE 8-14**

**TREATMENT FOR LUMBAR SPINE-PARS INTERARTICULARIS INJURY**

| Time | Treatment |
|---|---|
| 0–3 mo | Restrict repetitive extension exercises |
| 0–10 d | Pain medication, NSAIDs |
| 0–6 wk–6 mo | Antilordotic bracing |
| 10+ d | Flexion exercises, stretching hamstrings |

NSAID: nonsteroidal antiinflammatory drug

determined by the severity of symptoms. In cases of symptomatic spondylolysis, Micheli[223,224] favors a rigid polypropylene brace constructed with zero degrees of lumbar flexion (a modified Boston overlap brace) to be worn 23 hours a day for 6 months. Bracing from 6 weeks up to 3 months has also been suggested.[224] Unilateral defects may have a greater chance of healing than do bilateral defects. The presence of a bilateral pars defect on plain radiographs may indicate a significant decrease in the chance for bony healing, even with immobilization.[225] According to Wiltse (personal communication 1991), if the bone scan or SPECT scan demonstrates increased uptake in the *absence* of plain radiographic evidence of a pars defect, longer immobilization may be warranted due to a greater chance of bony healing. Whether relative rest alone or bracing, or both, are utilized, a thorough program of spinal rehabilitation is essential before the soldier's return to military activities.[226] Such programs emphasize hamstring stretching and antilordotic positioning and strengthening. If back pain persists despite bracing and rehabilitation, or if neurologic symptoms develop (or both), surgical intervention is most likely necessary.

**TABLE 8-15**

**TREATMENT FOR LUMBAR SPINE-STRAIN/ CONTUSION**

| Time (d) | Treatment |
|---|---|
| 0–2 | Relative rest, back education |
| 0–3 | Antiinflammatory modalities, pain medications |
| 3–10 | Initiate activity, regain flexibility |
| 10–21 | Improve joint mobility |
| 14–28 | Therapeutic exercises |
| 28+ | Cardiovascular conditioning, maintenance program |

### *Acute Muscles Strain and/or Contusion*

The method of presentation will be acute traumatic injury, with the *tissue injury complex* including the muscle tissue or thoracolumbar fascia.

***Clinical symptom complex*** will include localized lumbosacral discomfort, usually from a blow to the back; a tearing sensation while lifting or other traumatic event; or there will be a more subtle history of aggravation, such as a constant repetition of a new activity.

***Functional biomechanical deficit*** will include segmental hypomobility of the three-joint complex secondary to muscle spasm and guarding.

***Functional adaptation complex*** will show loss of active and passive segmental and combined motions; the ***tissue overload complex*** will be muscle myofilament.

***Rehabilitation*** is outlined in Table 8-15. Initial treatment should focus on limited periods of rest (less than 3 days) with early gentle activity within the pain free range. Progression is then made to mild activities and mobilization in the subacute phase. Ice massage, cold packs, and electrical stimulation are often helpful. Restoration of full function (strength and mobility) and normal posture should be the most important aspects of treatment. Regaining soft tissue flexibility and segmental motion is the first goal in the subacute phase. This can be accomplished through a variety of manual therapy techniques, including myofascial release,[227] joint mobilization or manipulation,[228,229] muscle energy techniques,[227] and stretching.

In all lumbar problems, maximization of the lower extremity muscular flexibility is especially important to allow normal lumbar motion. Due to their attachments to the pelvis, the hip flexors, extensors, and rotators have a great influence on positioning the lumbar spine. Adequate hip muscle flexibility allows for hip joint motion independent of lumbar segmental motion and is essential for the use of proper body mechanics and posture. Poor flexibility will cause excessive stress to be transmitted to the lumbar motion segments and sacroiliac joints.[230] Typical patterns of lower extremity inflexibility include hamstring, gluteus maximus and gastrocnemius-soleus, hip flexor, tensor fascia, and quadriceps groups. Tight hip flexors (iliopsoas) and quadriceps (rectus femoris) often cause extension and rotation hypermobilities in the lumbar spine. If the iliopsoas is contracted in a shortened position, the pelvis is maintained in excessive anterior tilt, placing the hip extensors (gluteus maximus and hamstrings) at a mechanical disadvantage. Therefore, early recruitment of lumbar extensor muscles (erector spinae) occurs and will result in increased shear or torsion stress to the intervertebral disk. If hip rotators are tight, abnormal kinetic chain motions in rotation occur, causing more stress at the joints and sacroiliac area and over the extensor muscle attachments. Self-stretching techniques should be taught as early as possible to allow ac-

tive involvement in the rehabilitation program. It is essential to stretch with as neutral a pelvic position as possible, since excessive anterior and posterior pelvic tilt will diminish the benefits of these flexibility exercises.

After muscle flexibility is achieved, optimal joint mobility must be obtained. Assessing spinal segmental motion and restoring motion to restricted or hypomobile segments again demands precisely applied manual techniques.[228,229,231] Hypomobility can result from impairment of the soft tissue supporting structures (ie, muscle hypertonus or fascial restriction) or from the intrinsic components of the three-joint complex (ie, lumbar disk degeneration or facet joint dysfunction). Adjacent hypomobile segments can cause increased stress on injured segments, and injured segments may cause hypermobilities at adjacent noninjured joints. Mobilization techniques[105,227] do not provide long-term relief to painful spinal segments primarily caused by discogenic abnormalities, and do not reduce disk herniations. However, temporary relief may occur from mechanoreceptor stimulation, stretch of adhesions, or restoration of shortened muscle length. Muscle energy techniques can be utilized with mobilization to improve segmental mobility.[227] These techniques use the patient's own isometric muscle contraction to relax hypertonic muscles by resetting the gamma gain in the muscle spindles. A series of isometric muscle contractions is elicited at varying degrees of joint positioning. The series of isometric contractions can also be utilized to mobilize a joint and may be less painful than passive mobilizations.

Once appropriate muscle flexibility and joint mobilization is accomplished, appropriate exercise is prescribed. This can often be determined by comparing flexibility side to side with the asymptomatic extremity, or to what the initial degree of flexibility was in the spine when the soldier presented the complaints. Exercises can be used to control pain (possibly through endorphin release), optimize tissue repair and regeneration, and improve muscle performance. Exercise to improve the function of the spinal muscles is generally known as spinal stabilization.[232,233] Stability of the lumbar spine is provided by bony architecture, disk mechanics, ligamentous support, muscular strength, endurance, and coordination. Optimal muscle strength can protect the spinal motion segment from chronic repetitive shear stress or acute dynamic overload. The concept of spine stabilization implies a muscle fusion. Spine stabilization exercises use force couple muscles, acting in concert around a mobile area, to provide a stable base, smooth motion, and efficient force modulation and transfer in the lumbar spine. By flexing, extending, and rotating the spine, the various abdominal, pelvic, and trunk muscles that attach to the thoracolumbar fascia act as an abdominal corset. Increased intraabdominal pressure does not appear to be the mechanism of stabilizing the disk and joint, but rather the reduction of shear forces across the three-joint complex (the posterior elements and the intervertebral disk) through abdominal cocontraction and tension generation in the thoracolumbar fascia and midline ligaments.

The muscular stabilizers of the spine include the intersegmental muscles (ie, multifidi, rotatores, interspinalis) abdominal muscles (particularly the transversus abdominus and internal obliques), latissimus dorsi, erector spinae, iliopsoas, and quadratus lumborum. The role of the smaller intersegmental muscles in stabilizing the lumbar spine is controversial; however, it is felt that they are effective as stabilizers, can balance shear forces, and apparently produce rotation, although not as primary rotators.[116,234–236] The multifidi also secondarily maintain lumbar lordosis by the nature of the force vector posterior to the vertebral bodies. The multisegmental muscles of the spine have been shown to be more efficient prime movers.[235]

Any spinal exercise must address stability before movement, endurance, and strength. Following spinal injury, the intersegmental muscles act as postural stabilizers (or tonic) of the spine, administering to fatigue first and then atrophy. Therefore, the initial stabilization exercises are directed toward those muscles that can control individual segmental mobility. Typically, these are manually resisted exercises of the trunk and are limited to short arcs performed in rotation, flexion, extension, and sidebending. The next phase of stability training involves direct and indirect strengthening of muscle groups through a variety of exercises performed in a neutral spine posture. Neutral spine is defined as the midpoint of available range between anterior and posterior pelvic tilt, not the absence of lordosis. The advantages of neutral positioning are (*a*) its loose-packed position, which decreases tension on ligaments and joints; (*b*) its allowance of more balanced segmental force distribution between the disk and facet joints; (*c*) its closeness to the center of reaction, allowing movement into flexion or extension quickly; (*d*) it provides the greatest functional stability with axial loading; and (*e*) it is usually the position of greatest comfort.[116] Training begins with exercises designed to help locate neutral spine in a variety of body positions, which increases awareness of lumbar and pelvic motion. These are

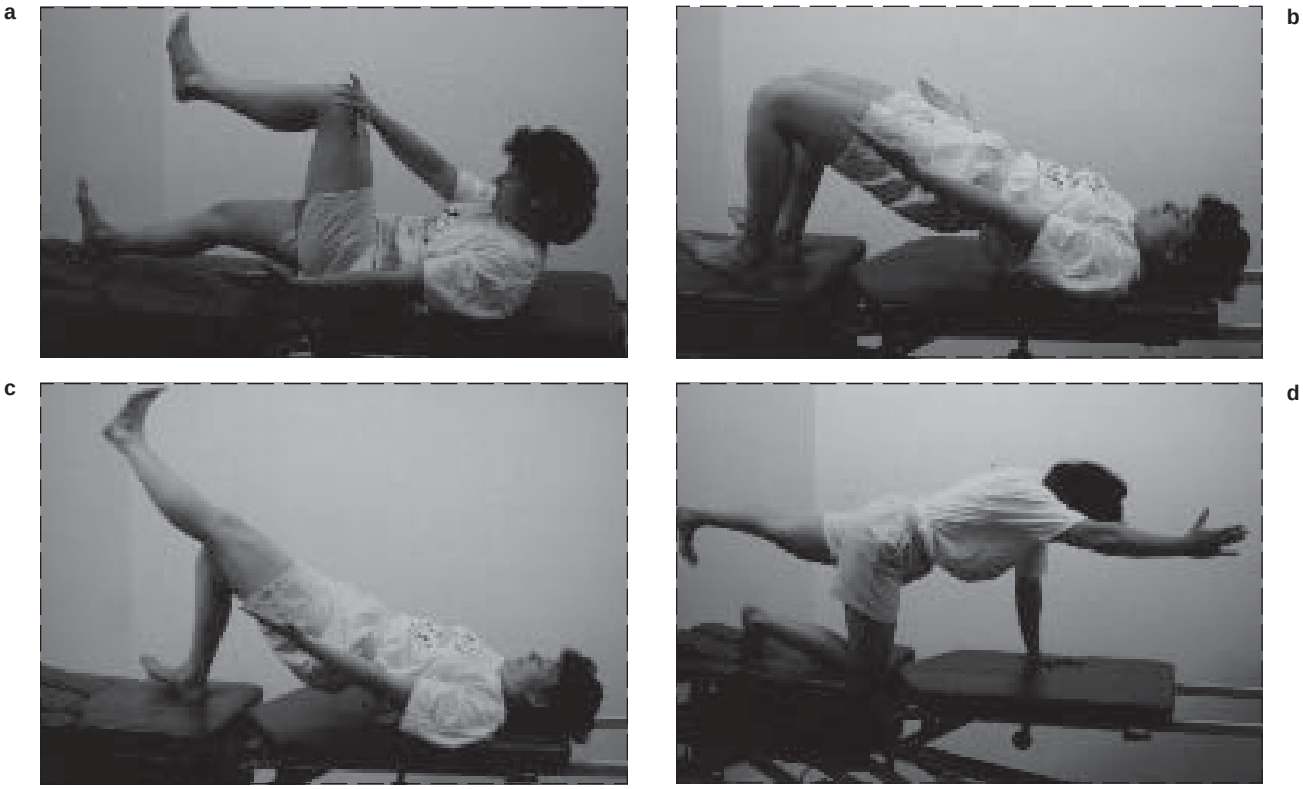



**Fig. 8-24.** Spine stabilization exercises for the low back. (**b**) **S**traight bridging of the back. The patient is instructed to keep the back straight to strengthen the gluteals and spine extensors, and using the abdominal muscles to maintain a neutral spine position. (**c**) Advanced bridging, progression to single leg stance for proximal stability. (**d**) Proximal stabilization of the "down" arm and leg with dynamic extensor strengthening of the "up" arm and leg.

followed by exercises of the extremities while maintaining a neutral spine, and later with the addition of resistance to the extremities, either manually or through use of weights. These exercises are performed slowly with the emphasis on precise pelvic control, which will facilitate neuromuscular coordination and enhance endurance and strength gains (Figure 8-24). These neutral spine stabilizing exercises will also emphasize the smaller postural stabilizers.

Strengthening prime movers, including abdominals and erector spinae, ultimately is required. Historically, abdominal exercises, especially sit-ups, have been emphasized as part of a low back exercise program. Sagittal plane sit-ups are utilized in the stabilization routine, but are limited to partial curl-ups lifting the head and upper body only. During this initial phase, the obliques and rectus abdominus are activated, whereas in the second half of a full sit-up, the iliacus and rectus femoris provide the main muscle force.[233] Lower extremity strength is necessary, as well as these muscles working in a coordinated manner with the trunk muscles for most functional activities. This is especially true during lifting, when the gluteal and hamstring muscles are the prime posterior rotators of the pelvis and trunk. Quadriceps strengthening is also important; it allows adequate support of body weight during use of proper body mechanics such as squatting. Torque to the lumbar spine is intrinsic to most activities, especially in work, athletic competition, and military duty.

After proper strength training and stabilization activities have been initiated, cardiovascular conditioning becomes essential. Improved cardiac fitness has been recognized to protect certain workers with high physical demand from back injury.[237] At hospitals in the Communication Zone (COMMZ), cardiovascular conditioning can be attained with aerobic exercises, such as running. Although not now available in the COMMZ, light portable cross-country ski machines could be used for this purpose.

The lumbar spine problems discussed in this section are in no way a comprehensive presentation of the myriad disorders seen in patients with lumbar spine complaints. Congenital disorders, metabolic

and neoplastic entities, as well as degenerative spinal conditions, such as spinal stenosis and spondylosis, have not been discussed. It is crucial to remember that if symptoms persist in a soldier who is believed to have a contusion or strain, the physician must reassess the diagnosis and consider other etiologies for the back symptoms (disk spondylolysis, posterior element dysfunction, and so forth). Most causes of low back pain resolve within 4 to 6 weeks. The aim of this section has been to focus on acute injuries that would be most commonly encountered in the field during war, and would need rapid evaluation and aggressive nonsurgical rehabilitation using limited resources. As stated previously, the discussion on specifics of strength, flexibility, and endurance pertains, in many respects, to all lumbar spine disorders and should be instituted in any spinal rehabilitation program.

**TABLE 8-16**

**TREATMENT FOR HIP-TROCHANTERIC BURSITIS**

| Time (d) | Treatment |
|---|---|
| 0–4 | NSAIDs, modalities, ice, friction massage |
| 2–14 | Stretch ITB, hip external rotators, flexors, abductors, extensors |
| 7–14 | Strengthen hip adductors |

ITB: iliotibial band
NSAID: nonsteroidal antiinflammatory drug

## Hip Disorders

### *Trochanteric Bursitis*

Bursitis over the greater trochanter is caused by friction where the ITB passes over the bony prominence of the greater trochanter. In response to friction, the potential space of the bursa becomes inflamed and fills with fluid. It is usually caused by excessive activity and insufficient stretching of the hip musculature. Femoral neck and ischial stress fractures, strain of the hip abductors, and referred back pain can mimic trochanteric bursitis.

***Method of presentation.*** Bursitis is an acute injury or a chronic exacerbation of a preexisting condition.

***Tissue injury complex.*** This will occur to the greater trochanteric bursa.

***Clinical symptom complex.*** There will be tenderness just posterior to the greater trochanter, aggravated by contraction of the tensor fascia lata with the hip abducted against resistance.

***Functional biomechanical deficit.*** There will be an inflexible ITB and hip external rotators, and weak adductors.

***Functional adaptation complex.*** Increased external rotation at the hip will result in altered gait and running patterns.

***Tissue overload complex.*** This will occur at the ITB proximally at the hip.

***Rehabilitation*** (Table 8-16). Initial treatment consists of NSAID medication and modalities and stretching of the ITB, external rotators of the hip, quadriceps, and, often, the hip flexors. Stretching of the gluteus maximus, which inserts into the ITB, is also important. Strengthening the hip adductors and correcting an imbalance between abductors and adductors can decrease trochanteric bursitis symptoms. In severe cases, a few days of cane-assisted ambulation may be helpful. Ice and deep friction massage, as well as contrast baths, may be of some benefit.[238] Corticosteroid injections can be used in recalcitrant cases. Occasionally, a leg length discrepancy, causing abnormal pelvic tilt, can be an aggravating factor in trochanteric bursitis and may benefit from a shoe lift or orthotics. Also, correction of excessive posterolateral heel wear can decrease symptoms. In most cases, symptoms will usually respond to treatment in 7 to 10 days. Chronic cases may require extended treatment.

### *Iliotibial Band Syndrome*

The iliotibial band or tract is a thickened portion of fascia lata that passes down the lateral aspect of the thigh and inserts into Gerdy's tubercle on the lateral tibial condyle.[23] Inflammation and symptoms can occur in any of three locations: (1) over the bony prominence of the greater trochanter, as discussed above; (2) most commonly, over the lateral femoral condyle; and (3) at the insertion at Gerdy's tubercle into the tibia. Factors associated with ITB syndrome are tibia vara, hyperpronation, cavus foot, worn outer soles of shoes, and ITB contracture.[239] The differential diagnosis includes hip pathology; meralgia paresthetica; upper lumbar radiculopthy; knee disorders, especially lateral collateral ligament injuries and lateral meniscal tears; and popliteus tenosynovitis.

ITB syndrome presents as an acute exacerbation of chronic overuse injury, and the ***tissue injury complex*** will include the proximal portion of the ITB

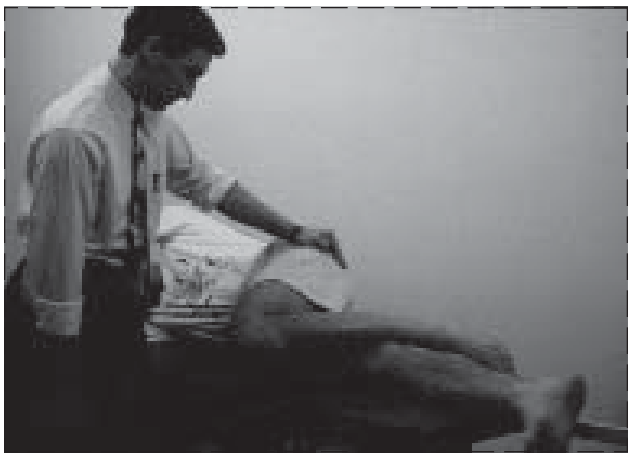


**Fig. 8-25.** Modified Ober test to evaluate for inflexibility of the iliotibial band (ITB). The bottom leg is extended. The top hip is flexed. The greater trochanter is perpendicular to the table surface. The degree of ITB tightness is measured as the number of fingerbreadths between the medial part of the knee and the table surface in this position.

over the greater tuberosity, the distal portion over the lateral femoral condyle, or insertion to the tibia.

***Clinical symptom complex*** includes (*a*) localized pain, usually over the lateral femoral condyle, and worse with running activities, especially on banked surfaces or hills; (*b*) abduction against resistance; and (*c*) a positive Ober's test (Figure 8-25).

**The *functional biomechanical deficit*** will be at the inflexible ITB, with the ***functional adaptation complex*** presenting as increased lateral patellar tracking, external rotation at hip, internal rotation of the leg, and functional pronation of the foot.

***Tissue overload complex*** will be varus loading on the lateral aspect of the knee.

***Rehabilitation*** is outlined in Table 8-17. Therapy for ITB syndrome includes application of ice with ice massage for up to 20 minutes at a time, followed by active stretching of the ITB, the tensor fascia lata, the gluteus maximus, and often the hip flexor muscles (Figure 8-26). Antiinflammatory medications and modalities are used as available. Local injection of anesthetic and corticosteroid can be helpful, especially at the lateral femoral condyle, where the injection is made both anterior, posterior, and deep to the ITB.[239] Correction and support of the functionally pronated foot must be addressed. Strengthening exercises for the adductors of the hip are important to counteract the abduction moment caused by a tight ITB. Strengthening of the gluteus maximus and tensor fascia lata are also important to avoid overuse of these muscles that form the ITB.

**TABLE 8-17**

**TREATMENT FOR ILIOTIBIAL BAND SYNDROME**

| Time (d) | Treatment |
|---|---|
| 0–2 | Ice massage, NSAIDs |
| 2–14 | Stretch ITB, TFL, gluteus maximus |
| 7–10 | Strengthen adductors, gluteus maximus, TFL |
| 14–21 | Local injection (if necessary) |

ITB: iliotibial band
NSAID: nonsteroidal antiinflammatory drug
TFL: tensor fascia lata

Reduction in mileage running, along with a course of antiinflammatory medication, will frequently cause rapid reduction of pain within less than 1 week.[240] However, more difficult cases may take as long as 6 weeks to resolve.[241] Rarely, surgery may be necessary for recalcitrant cases.

### *Hamstring Strain*

Hamstring strains are a frequent cause of disability in the military population. The injury may occur anywhere along the muscles, but is more common at the junction of the proximal muscles and tendons. The mechanism involves forced flexion of the hip with the knee extended.[242] Sudden, forced change in the musculotendinous length may result in strain or rupture at the junction of the muscle and tendon. Hamstring strains are associated with inadequate hamstring flexibility, inadequate warm-up, exercise fatigue, poor muscular coordination, abnormal or excessive pelvic tilt, previous injury, and imbalance between hamstring and quadriceps strength.[243–245]

Hamstring strain generally presents as an acute traumatic injury with a ***tissue injury complex*** involving the myotendinous junction disruption in the upper third of muscle secondary to eccentric overload.[28]

**The *clinical symptom complex*** includes pain in the upper third of thigh, swelling, bruising, mass in the thigh, muscle weakness, and tenderness on bending or prolonged sitting.

**The *functional biomechanical deficit*** will be decreased knee extension, increased hip flexion, and worsened hamstring, that is, quadriceps strength ratios.



**Fig. 8-26.** (**a**) Stretching exercise for the internal rotators of the hip and the hip extensors by placing the foot (ankle) on the knee and pulling it toward the chest. (**b**) Stretching exercises for the hip flexors. The patient is instructed to maintain a posterior pelvic tilt while actively extending the hip.

**The *functional adaptation complex*** will be decreased stride length with running, and the ***tissue overload complex*** will be at the myotendinous junction.

***Rehabilitation*** is outlined in Table 8-18. Initial treatment for acute hamstring injury in the proximal midthigh or distal thigh should be rest and application of ice directly to the injured area, compression wraps with the knee in flexion to decrease edema and hemorrhage, medication, and non–weight bearing.[239] During the initial 24 to 48 hours, icing, with the knee in extension, should be done for 20 minutes at a time 3 to 4 times a day to put early, gentle stretch on the hamstrings. Isometric strengthening should also be instituted early, and advanced to progressive resistance exercises as tolerated. Within 3 to 5 days, electric stimulation and gentle passive massage and stretching should be initiated. Modified flexion exercises (Figure 8-27) are started as early as pain permits to progressively stretch the healing hamstring muscle. Activity can begin when the soldier can perform the modified Williams flexion exercise of the bent knee to straight leg raise and have the leg raised beyond a 90° angle from the perpendicular while keeping the knee perfectly extended and the toes dorsiflexed.[246] Generally, for first degree strains, where the injury is mild and there is only slight loss of extension, this point will be reached in 4 or 5 days. For a second degree strain, which has more significant swelling, pain, and loss of flexion, this point is usually reached in 7 to 10 days. Third degree injuries, where the loss of extension is greater than 45° and a more severe injury has occurred, may take 3 to 4 weeks to reach the point where activity can begin. To avoid recurrent injury, the musculotendi-

**TABLE 8-18**

**TREATMENT FOR HAMSTRING STRAIN**

| Time (d) | Treatment |
|---|---|
| 0–3 | Ice, compression, decrease weight bearing |
| 3–7 | Electric stimulation, passive ROM exercises, modified Williams stretching |
| 7–21 | Progressive stretching of hamstrings |
| 14–21 | Begin isometrics of hamstrings, quadriceps |
| 14–28 | Stationary bicycling |
| 21+ | Eccentric and concentric strengthening of hamstrings |

ROM: range-of-motion

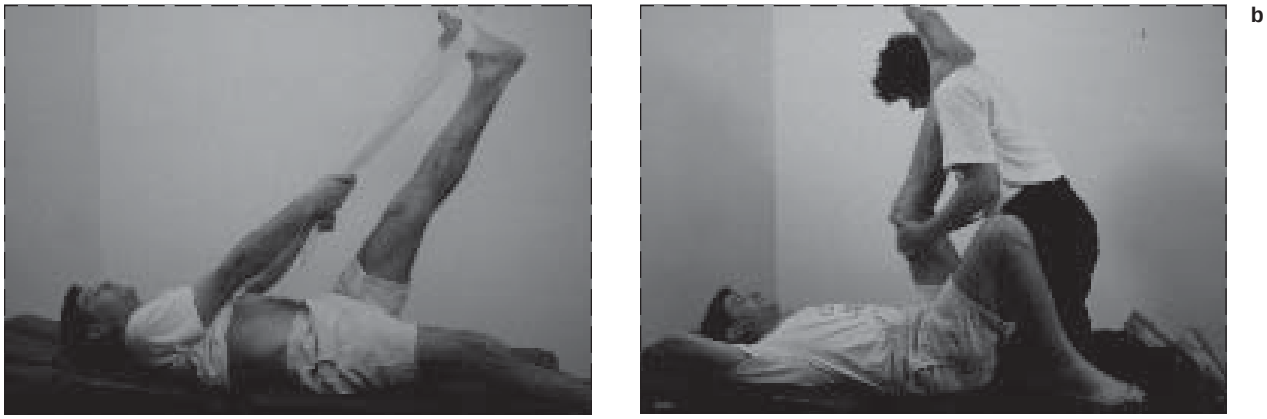



**Fig. 8-27.** Flexion exercises to stretch the hamstrings. (**a**) The straight leg raise as shown is difficult to hold for the 30 seconds required to have a beneficial effect. Alternatively, a useful exercise consists of lying in the doorway with the heel on the frame. (**b**) Therapist-assisted hamstring stretches. Ideally, the knee is not allowed to bend at all during this exercise.

nous unit must be slowly stretched to reestablish its full length before significant strengthening occurs. Stationary bicycle riding and more aggressive eccentric and concentric strengthening can proceed. Return to full activity requires restoration of muscle length and strength, with muscle strength to be at least 90% of the strength of the uninvolved side, and no pain experienced with isometric contraction.[247]

## Knee Disorders

### *Ligament Injuries: Anterior Cruciate Ligament*

The ACL functions to prevent both excessive anterior movement of the tibia on the femur, and abnormal tibial internal and external rotation. It is needed to maintain the normal biomechanical function of the knee; its loss not only produces abnormal kinematics, but frequently results in major degenerative changes.[248–250] The mechanism of injury can be by hyperextension of the knee, varus stress with internal rotation, and extremes of valgus and external rotation at the knee. Injuries are graded, from 1 to 4, by the degree of anterior displacement of the tibia with respect to the femur on the Lachman or Anterior Drawer tests. Grade 1 injuries have 0 to 5 mm of displacement; grade 2, 5 to 10 mm; grade 3, 10 to 15 mm; and grade 4, greater than 15 mm of displacement. Surgical vs nonsurgical treatment is controversial.[249–253] However, postoperative complications are more likely if surgery is done within the first 2 weeks after injury, when the knee is acutely inflamed and range-of-motion is restricted.[254] In a war situation, the initial important considerations are whether (*a*) the knee is stable enough to proceed with conservative, nonsurgical rehabilitation and (*b*) there are associated injuries, such as meniscus tear or tibial plateau fracture. The focus here will be on the nonsurgical and postsurgical issues that need to be addressed to adequately rehabilitate an anterior cruciate deficient (or reconstructed) knee.

***The method of presentation*** is as an acute injury, with the ***tissue injury complex*** including the ACL part, or all, of the two major bundles (posterolateral, anteromedial).

***The clinical symptom complex*** will be a sudden pop on the knee after rotational injury, occurring with either applied external force or simple axial loading. This will result in the acute onset of knee effusion, and a giving-way sensation.

***The functional biomechanical deficit*** will be the loss of rotational stability of the knee, especially anterolaterally.

***Functional adaptation complex*** will be the loss of ability to rotate on the foot in the midstance position; the ***tissue overload complex*** is the ACL.

***Rehabilitation*** is outlined in Table 8-19. The anterior cruciate deficient knee has abnormal motion under stress or when involved in aggressive physical activities. This abnormal motion can be in a rotatory direction or in the anteroposterior direction, and in most cases, results in synovitis, which inhibits the rehabilitation program.[255] In any ACL rehabilitation program, the important factors to consider are the specificity of the treatment exercises, the positioning of the knee, the role of antagonist and agonist muscles, proprioceptive retraining, and the use of closed chain vs open chain exercises.[256,257]

**TABLE 8-19**

**TREATMENT FOR ANTERIOR CRUCIATE LIGAMENT**

| Time | Treatment |
|---|---|
| 0–7 d | Early ROM exercises, weight bearing as tolerated with immobilizer, isometric quadriceps and hamstring cocontraction |
| 7–14 d | Full extension, hamstring curls, one-legged quarter squats, step-ups, calf raises |
| 3–6 wk | Discontinue immobilizer, leg presses, stationary biking |
| 6–8 wk | Jump rope, figure-8 drills, bracing for cutting or pivoting activity |

ROM: range-of-motion

As soon as practical, acute injury of the ACL, without other major ligamentous or cartilage damage, can be treated with NSAIDs and physical therapy. This will reduce inflammation and restore full range-of-motion.[248] To control pain, crutches and immobilizing splints may be helpful, but are rarely necessary for more than a few days. When available, it may be helpful to use therapeutic modalities, such as interferential current or a TENS unit to decrease pain, cryotherapy to decrease edema and pain, and electric stimulation to retard atrophy and neuromuscular reeducation. Either postoperatively or acutely after injury, it is important to begin early passive full extension of the knee and weight bearing as tolerated.[254] Avoidance of hyperextension of the knee is sought to decrease the stress on the injured or repaired ligament. Postoperatively, or within the first week after injury, abduction and adduction straight leg raises, extension straight leg raises, and hamstring curls are initiated. Quadriceps exercises can be delayed for 2 to 3 weeks, and initial use should only be isometrically to avoid an increase in anterior translational forces on the torn ACL or the postoperative healing graft. Active extension exercises can be initiated at about 6 weeks after acute injury or postsurgery. Once swelling and pain have subsided, isometric hamstring and quadriceps strengthening are initiated with the knee resting on a rolled towel. Cocontraction of the quadriceps and hamstring muscles is needed to dampen the anterior shear force from the quadriceps contraction on the torn or healing ACL. Initially, straight leg raises are avoided because they may increase the anterior subluxation of the tibia on the femur.[258] Isometric internal and external tibial rotation exercises, which are added once the patient has 90° of knee flexion, have been shown to decrease abnormal tibial rotation.[259]

Isotonic exercises for the upper body, the contralateral leg, the hips, and the ankles, can be started early in the rehabilitation process. Also, cardiovascular endurance training with one-legged cycling and rowing, or upper extremity ergometry can be used. Neuromuscular and proprioceptive retraining become more important as good, closed chain kinetic strength is developed. Closed chain kinetic strengthening involves activities such as a partial squat, where the foot is fixed while leg muscles are being exercised. Active hamstring control, which reduces the pivot shift, was present in 95% of patients with ACL injuries who successfully avoided surgery.[251] Balance board activities are an excellent source of proprioceptive training. Later in the rehabilitation program, activity specific drills need to be incorporated to prepare the soldier for returning to full, unrestricted duty. Often a derotation type of brace may give the soldier more proprioceptive feedback when returning to active duty. Total rehabilitation time is variable, ranging from a few weeks for a mildly strained ACL to 6 to 8 months for a reconstructed ACL with concomitant injuries to the menisci, collateral ligaments, or other structures that complicate the rehabilitation process.

### *Ligament Injuries: Medial Collateral Ligament*

The medial collateral (MCL) or tibial collateral ligament (TCL) is the primary restraint to medial joint opening.[260,261] The mid-third medial capsule is a secondary restraint to valgus stress and is usually torn with more force than necessary to tear the tibial collateral ligament alone.[262] The mechanism of injury to the MCL is most commonly a contact force to the lateral or posterolateral aspect of the knee. The collateral ligament complexes, due to their extracapsular environment with extensive blood supply, have a high potential for spontaneous healing. Lesions of the MCL are graded from 1 to 3, with grade 1 injuries being a stretch to the MCL fibers; grade 2, an incomplete tear of the MCL; and grade 3, a complete ligamentous disruption.

***The method of presentation*** is as an acute, usually traumatic, injury, and the tissue injury (***tissue injury complex***) will be to the medial (tibial) collateral ligament.

***The clinical symptom complex*** may include acute medial knee pain, swelling and tenderness over the site of injury, usually within 24 to 36 hours follow-

ing the injury. Occasionally, there will be complaints of the knee giving way into valgus, usually from a valgus force applied to the knee with external tibial rotation.

***The functional biomechanical deficit*** will be a decrease or loss of medial stability of the knee when valgus force is applied.

***Functional adaptation complex*** will present as ambulation with a flexed knee (avoidance of a full knee extension that stresses MCL fibers), and with the ***tissue overload complex;*** stress will occur on the MCL, and in the quadriceps and hamstring muscles.

***Rehabilitation*** is outlined in Table 8-20. Following collateral ligament injury, there are time constraints for ligament healing. Appropriate restrained joint motion will allow physiologically tolerable stresses, improved cartilage nutrition, diminished muscular atrophy, and enhanced stimulation of collagen healing. All grades of medial collateral ligamentous injuries can be treated nonoperatively.[257, 263–270] Initially, ice is used for pain and edema reduction. For incomplete tears, early motion is advocated, usually in a hinged cast brace, which is initially set between 30˚ and 90˚ of flexion. Full extension is avoided during early treatment because this position puts increased tension on the healing ligament. Full weight bearing with the use of crutches can be employed for several days until the acute symptoms subside. Progression to full weight bearing and free range-of-motion is advocated as the swelling and pain decrease.[262] This may be within 3 to 5 days in a grade 1 injury, or 2 to 6 weeks with a grade 2 injury.[271,272] Grade 3 injuries can be immobilized in a cast for 2 weeks, followed by a cast brace treatment for 4 additional weeks.[271] Isometric quadriceps strengthening, straight leg raises and strengthening of the hip flexors, adductors, and abductors can be initiated early. Progressive resistive exercises are begun when full range-of-motion is obtained, and an isolated grade 3 lesion of the MCL will also respond well to nonoperative treatment and actually regain strength in a significantly shorter period of time than will patients who have had surgery.[272] Care must be taken during hip exercises to avoid valgus stress of the knee. During isotonic training of hip musculature, weights will need to be placed proximal to the knee to avoid such valgus stresses. Grade 3 MCL tears may require a cast brace from 30˚ to 90˚ of flexion for 2 to 6 weeks. Conditioning on a stationary bicycle, when available, is helpful in MCL rehabilitation, because of the varus moment at the knee with cycling and the subsequent reduced stress on the MCL. Active duty is resumed when the knee has full range-of-motion, good muscular control, and no tenderness. Total rehabilitation time can be as short as 2 weeks or as long as 8 to 10 weeks.[258]

**TABLE 8-20**

**TREATMENT FOR KNEE-COLLATERAL LIGAMENT INJURY**

| Time (d) | Treatment |
|---|---|
| 0–14 | Cast brace 10º internal rotation of tibia, 30º–90º flexion (grades 2 and 3) |
| 0–14 | Multiple angle isometric strengthening of quadriceps, hamstrings, adductors, abductors, along with straight leg raises |
| 14–21 | Open hinge brace to 10º–90º (grades 2 and 3) |
| 21–28 | Remove brace, weight bearing, squats (partial), step-ups, toe raises, heel raises |
| 28+ | Light jogging |

### *Patellofemoral Pain Syndrome*

Patellofemoral pain syndrome is one of the most common musculoskeletal injuries seen in the athletic population. Similarly, these problems are seen in the military population. Repeated stresses applied to the patellofemoral joint in deceleration maneuvers are a common cause of injuries to this joint.[272] Many predisposing factors to patello-femoral pain exist, including vastus medialis obliquus dysplasia, vastus lateralis hypertrophy, extensor mechanism malalignment, high and lateral patellar posture, increased Q-angle, and bony deformity.[273–275] The differential diagnosis of patello-femoral pain includes referred pain from the hip and low back, osteochondritis dessicans of the femur or patella, bone tumors (especially in cases of unilateral symptoms), osteoarthritis, inflammatory joint disease, meniscal pathology, or a synovial plica.

The method of presentation is as a chronic overload injury, and ***the tissue injury complex*** will include the patellar cartilage and synovium, and tendon insertion into the patella.

***The clinical symptom complex*** may include peripatellar pain (which will be worse with increased knee flexion), positive "theater" sign (pain upon arising after sitting for a prolonged period of time), crepitus, pain when descending stairs, and patellar compression.

***The functional biomechanical deficit*** will include insufficiency of medial quadriceps musculature; inflexibility of the ITB, lateral retinaculum, hamstrings, and gastrocnemius muscles; alteration in patellofemoral tracking; and hamstring muscle weakness.

***The functional adaptation complex*** may present as a knee flexion contracture, increased pain with running and axial loading of the knee, and jumping from the opposite leg, with the ***tissue overload complex*** being in the lateral retinaculum, and patellar tendon.

***Rehabilitation*** is outlined in Table 8-21. Symptomatic treatment may start with antiinflammatory medications and modalities. Lower extremity malalignment problems, such as genu varum, tibia vara, hindfoot varus, and forefoot pronation, which if present, can cause a compensatory subtalar joint pronation and obligatory internal tibial rotation, must be corrected. Orthotics are often used for this. Flexibility exercises must focus on many lower extremity muscles, in particular the ITB, because of its insertion into the lateral aspect of the patella and laterally deviating forces on the patella. The hamstring and gastrocnemius muscle must also be exercised because of their propensity to shorten and increase the patellofemoral joint reaction forces with increasing flexion of the knee. This compression has been documented to rise sharply after 30° of knee flexion and can reach eight-fold the body weight with a full squat.[276] Manual medial glide and tilt of the patella may be employed specifically to stretch the tight lateral retinaculum.[273] Initial strengthening of the quadriceps and hip flexors is done isometrically and with straight leg raises.[277] Short arc (–30° extension to 0°) quadriceps exercises may be helpful, although the selectivity in strengthening only the vastus medialis obliquus is debatable. Often, multiple angle isometrics of the quadriceps can strengthen the quadriceps without the significant increase in patellofemoral joint reaction forces that occurs with isotonic exercises. Imbalances between the medial and lateral rotators, and adductors and abductors of the hip need to be addressed, because they may lead to excessive medial rotation and adduction of the hip during the stance phase of gait with an associated increased valgus vector at the patellofemoral joint. Closed chain kinetic exercises, with cocontraction of quadriceps, hamstring, and gastrocnemius-soleus muscles are important to reduce excessive forces across the patella. Furthermore, partial squats (one quarter of a full squat) will also eccentrically load the knee, which will be more physiologic in nature. MacConnell[278] has described an approach to neuromuscular reeducation of knee musculature combined with patellar taping that may be very effective for patellofemoral pain problems. The use of a knee sleeve with fenestration for the patella has also been occasionally helpful.[279]

**TABLE 8-21**

**TREATMENT FOR KNEE-PATELLOFEMORAL PAIN**

| Time (d) | Treatment |
|---|---|
| 0–3 | NSAIDs, antiinflammatory modalities, correct alignment |
| 3–14 | Flexibility for ITB, hamstrings, gastrocnemius-soleus |
| 7–14 | Isometric strengthening, ie, SLR, multiple angle isometrics, adductors and abductors strengthening, taping |
| 14–28 | Closed chain kinetic exercises |

ITB: iliotibial band
NSAID: nonsteroidal antiinflammatory drug
SLR: straight leg raise

## Leg, Foot, and Ankle Disorders

### *Medial Tibial Stress Syndrome*

Medial tibial stress syndrome, or shin splints, can result from a number of different factors that cause pain and discomfort in the anteromedial or posteromedial part of the lower leg.[241] This usually occurs after repetitive overuse in walking and running, and these conditions include tibial stress fractures (to be discussed subsequently), posterior tibial tendinitis, and periostitis, stress changes at the attachment of the soleus fascia, and compartment syndromes.[280–283] Factors contributing to shin splints include weak anterior leg muscles, improper footwear (hard heel and minimal cushion), varus foot, forefoot imbalance, weak posterior leg muscles, tight heel cords with equinovarus, hypermobile, pronated feet, increased heel eversion, tibia vara, subtalar varus, and forefoot supination.[284]

***The method of presentation*** will be as a chronic overuse injury, and the ***tissue injury complex*** will include the posterior tibial tendon; attachment of soleus fascia, anterior, anterolateral, or deep posterior compartments of the leg; and the middle third of the tibia.

**TABLE 8-22**

**TREATMENT FOR MEDIAL TIBIAL STRESS SYNDROME**

| Time (d) | Treatment |
|---|---|
| 0–7 | Cryotherapy, NSAIDs, evaluate biomechanics |
| 3–10 | Stretching gastrocnemius-soleus, posterior tibialis |
| 7–14 | Strengthen posterior tibialis, FHL, FDL, gastrocnemius-soleus; proximal limb strengthening |

FDL: flexor digitorum longus
FHL: flexor hallucis longus
NSAID: nonsteroidal antiinflammatory drug

**The *clinical symptom complex*** will be anterior leg pain with activity, often localized over a 3 to 6 cm area of tenderness over the posteromedial edge of the distal third of the tibia, and is generally relieved with rest.

***The functional biomechanical deficit*** may be inflexibility and weakness of ankle plantar flexion, and weakness of ankle dorsiflexors or invertors, or both.

***The functional adaptation complex*** will present as a functional pronation of the foot, and the tissue overload complex will be at the plantar flexors and invertors of the feet, the attachment of soleus muscle to medial tibia, and the bony cortex of the tibia.

***Rehabilitation*** is outlined in Table 8-22.The common denominator in all the potential causes of medial tibial stress syndrome is repetitive overload to leg structures; therefore, the cornerstone of rehabilitation is to modify activity and provide relative rest of the injured area. Relative rest may vary from a few days, in a very mild case, to 4 to 8 weeks in severe cases.[285] Modification of active duties is indicated; however, aerobic capacity and general fitness need to be maintained with other forms of activity, particularly nonweight bearing ones, such as the use of an exercise bicycle or cross-country ski machine. During the acute period, the area of pain and tenderness should receive ice treatments for 10 to 15 minutes, 2 to 3 times a day.[284] Antiinflammatory medications are also useful. Stretching of the gastrocnemius-soleus and tibialis posterior muscles is stressed. When pain and tenderness are diminished, strength needs to be developed, concentrically and eccentrically, in the posterior tibialis, anterior tibialis, flexor hallucis longus, and flexor digitorum longus.[284] Proximal limb strength at the hip and knee also needs to be maintained. When predisposing factors exist, evaluation of foot anatomy and mechanics is essential to prevent recurrence of problems.[286] Orthotics may be beneficial in such cases. An orthosis prevents or reduces compensatory pronation by use of a medial heel and forefoot wedge. With proper treatment, most symptoms will subside in 2 to 4 weeks. If symptoms persist, a further diagnostic work-up at a military hospital may be warranted.

### *Stress Fractures*

Stress fractures are defined as a partial or complete fracture of bone caused by an inability to withstand nonviolent stress that is applied in a rhythmic, repeated, subthreshold manner.[287,288] Stress fractures can occur in any area where bony architecture is overloaded, causing an inability of the bone to remodel in response to the mechanical stress placed on it. The clinical manifestation is often pain. Running activities and sports cause stress fractures in the fibula and tibia; jumping activities affect the pelvis, femur, calcaneus, and patella.[284] In military recruits, the incidence of metatarsal stress fractures during basic training is 40%.[287,289] The fibula is the site of approximately 25% of the stress fractures in civilian athletes, as opposed to only 2% in military recruits. In the military population during basic training, tibial stress fractures are found in 20% of personnel, calcaneus fractures in 30%, femur fractures in 3%, and spine stress fractures in 5%.[287] A 4-year study on stress fractures of the lower extremity in basic training soldiers showed common male stress-fracture sites were the metatarsals (66%), calcaneus (20%), and lower leg (13%). In females, the common sites were the calcaneus (39%), metatarsals (31%), and lower leg (27%).[290] After the onset of stress fractures, roentgenograms taken before 3 to 4 weeks are almost always negative. During the early period of symptoms, a bone scan can be diagnostic. A positive result scan can be seen as early as 2 to 8 days after the onset of symptoms.

***The method of presentation*** is as a chronic overuse injury, and the ***tissue injury complex*** will be the local bone.

***The clinical symptom complex*** will include the gradual onset of local pain in the area of the fracture at the conclusion of activity. This is relieved with rest, but pain with percussion of the affected bone away from the fracture site generally produces pain at the fracture site.

***The functional biomechanical deficit*** and the ***functional adaptation complex*** will vary, depending on the site of fracture.

***The tissue overload complex*** will occur at the bone.

***Rehabilitation*** of stress fractures will also vary, depending on the location of the fracture, but it generally involves decreasing the stress applied to the bone.[284,291] Pester and Smith[290] found that modification of the physical training program to eliminate continuous, high impact activities during high-risk weeks (ie, weeks 2, 4, and 6) resulted in a 12.9% drop in stress-fracture incidence. Other limited weight bearing activities can be substituted to maintain cardiovascular fitness, such as the use of a cross-country ski machine or bicycling, walking, and swimming.

Rehabilitation of fibular stress fractures should include no running for a minimum of 3 weeks, appropriate stretching of the heel cord and ankle, and lower extremity strengthening exercises to avoid ankle stiffness or weakness after the fracture has healed. In rare cases, cast immobilization or partial weight bearing may also be considered for 2 to 3 weeks until inflammation resolves.[284]

Tibial stress fractures usually involve the proximal or medial third of the tibia.[292] Since this bone bears five sixths of the body's weight, these fractures require longer rehabilitation time. Generally, it is necessary to stop running activities for at least 4 to 8 weeks. Flexibility and strength issues are similar to those for fibular stress fractures. Caution should be observed with anterior tibial cortical stress fractures because these frequently have delayed union, nonunion, and fracture completion.

Stress fractures are also common in the foot and ankle regions.[287,293–295] Treatment focuses on relative rest of the affected limb. However, movement should be maintained in surrounding joints, as well as the affected joints, to avoid effects of immobilization. An effective determinant for healing in foot and ankle stress fractures, is when a patient can hop on the affected limb without pain.[296]

Femoral neck and pubis stress fractures are common in females and people doing significant distance running. Femoral neck fractures will require many months of touch-down weight bearing with crutches. Occasionally this type of fracture will require prophylactic pin fixation. Due to persistent physical activity or inadequate treatment, completion of this fracture may lead to avascular necrosis of the femoral head.[238] Pubic rami stress fractures heal within 2 to 5 months.[288]

For most fractures, treatment should be from 6 to 8 weeks. Medication other than analgesics is rarely necessary. Ultrasound treatment is contraindicated because it aggravates the pain in the area of the fracture.[288] Ice and massage may be appropriate for pain reduction, and bracing may occasionally be necessary to limit any motion that would aggravate the injury. Surgery is considered in those bones in which a complete fracture would have serious consequences.[288] These include a displaced fracture at the tarsal navicular, a femoral neck fracture (as discussed above), or possibly a stress fracture of the fifth metatarsal shaft because of its frequent progression to a nonunion in an active soldier.

### *Inversion Sprains*

Injuries to the ankle, particularly inversion sprains, are among the most common musculoskeletal problems seen in the military population. The ankle is much less stable in plantar flexion, owing to the narrowing, wedge-shaped posterosuperior surface of the talus and the distal extension of the lateral malleolus.[297] The most common mechanism of injury, plantar flexion and inversion, may cause injury to the anterior talofibular, calcaneofibular, posterior talofibular (with increasing rotational component), and tibiofibular ligaments (with severe injuries).[298] The differential diagnosis of an acute inversion injury to the ankle include acute rupture of the peroneal retinaculum with subluxation of the peroneal tendons, fracture of the distal fibula, osteochondral fracture of the dome of the talus, or fracture of the anterior process of the os calcis.

***The method of presentation*** is as an acute traumatic injury, and the *tissue injury complex* will involve the anterior talofibular, calcaneofibular, and posterior talofibular and tibiofibular ligaments.

***The clinical symptom complex*** will include pain with active or passive inversion, and localized lateral ankle pain.

***The functional biomechanical deficit*** will present as decreased inversion stability of the ankle, especially in plantar flexion.

***The functional adaptation complex*** will be functional pronation of the forefoot, and the ***tissue overload complex*** will be to the lateral ligamentous complex of the ankle.

***Rehabilitation*** is outlined in Table 8-23. Immediate use of ice and compression will greatly decrease edema and hemorrhage and accelerate the rehabilitation process. Use of cryotherapy for 20 minutes every 2 to 3 hours during the first 48 hours

**TABLE 8-23**

**TREATMENT FOR INVERSION SPRAIN**

| Time (d) | Treatment |
|---|---|
| 0–1 | Early ROM exercises, writing alphabet with foot in the air |
| 0–3 | Ice, compression, bracing |
| 3–7 | Elastic tubing, resistance band exercises |
| 7–14 | Proprioception, closed chain kinetic exercises |

ROM: range-of-motion

after injury is advised. After the acute swelling subsides, electric stimulation, Jobst-type pump, or contrast baths can decrease residual and recalcitrant edema. Early ankle motion can be started on the first day of rehabilitation with active motion (nonweight bearing) in the form of 2 or 3 sets of writing the alphabet with the ankle 3 to 4 times a day. This will decrease edema, provide motion to prevent contracture, and start strengthening all planes of the ankle. Use of elastic ankle supports or inflatable air stirrup splints can give excellent support to an injured ankle during the rehabilitation process. As edema subsides, strengthening with resistance tubing is begun in the directions of inversion, eversion, dorsiflexion, and plantar flexion. Weight bearing should progress as tolerated, and once comfortable, weight bearing closed chain kinetic exercises can begin (partial squats and toe raises), as well as proprioceptive retraining with a balance board (Figure 8-28). Proximal limb strengthening is also important because of the significant degree of weakness in the linkage system noted with distal limb problems. Strengthening needs to be done in both concentric and eccentric modes to more accurately simulate physiological stresses.[299] These include exercises for the hip abductors and adductors, and for the knee flexors and extensors. Stretching exercises, such as with a slant board for

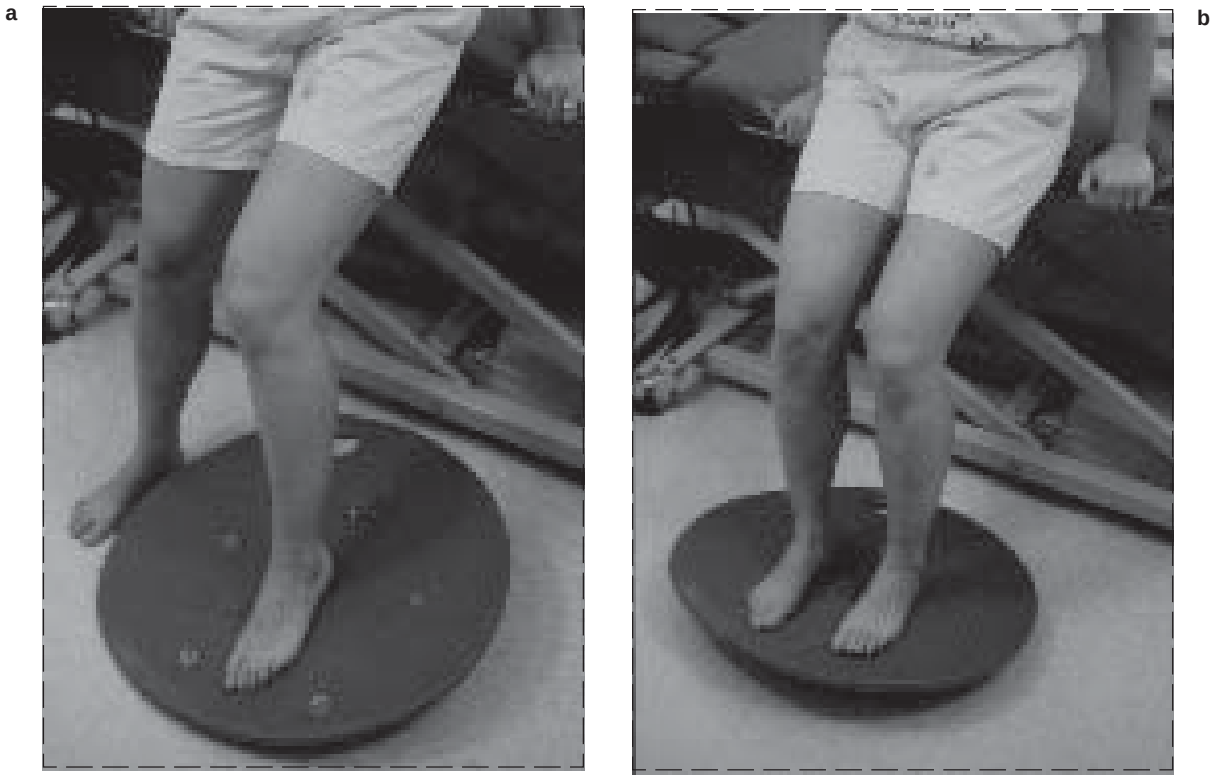



**Fig. 8-28.** (**a**) Balance board training for proprioception and ankle strengthening with one leg. The patient is instructed to slowly press the outer rim of the board against the ground, first in a clockwise rotation and then in a counterclockwise rotation. (**b**) Balance board training for proprioception and ankle strengthening with two legs. The instructions are the same as those for one-legged balance board exercises. These exercises can be done initially while supporting the upper extremities and later advanced to unsupported for more proprioceptive and balance training.

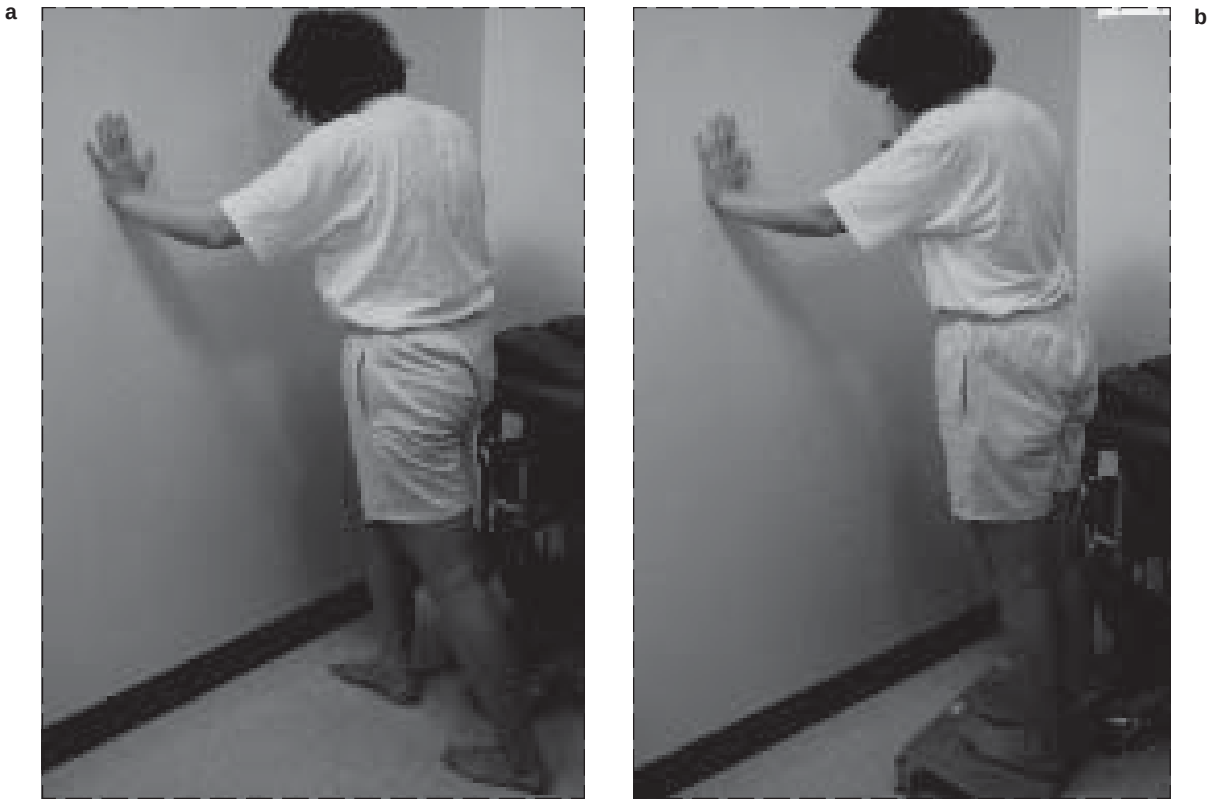



**Fig. 8-29.** Wall stretches for the gastrocnemius-soleus muscles. (**a**) The front leg is maintained in a flexed position to stretch the soleus muscle, the back leg is extended to stretch the gastrocnemius. The heels are to remain in contact with the ground. (**b**) A slant board is used in conjunction with the wall stretches.

the gastrocnemius-soleus group are important (Figures 8-29). Hamstring, quadriceps, ITB, and hip flexor flexibilities are also stressed.

### *Achilles Tendinitis*

Achilles tendinitis results from repeated episodes of microtrauma to the Achilles tendon, resulting from uneven or inconsistent loading of the gastrocnemius-soleus system, which is not able to undergo an efficient eccentric contraction.[300] These episodes can occur during marching or running on uneven terrain. Microtears in the Achilles tendon result. These tears occur in the area of least vascularity, 2 to 6 cm above the insertion of the tendon, where the tendon is susceptible to chronic inflammation and rupture.[301,302] Anatomic factors that can contribute to Achilles tendon problems include excessive pronation, hindfoot varus, forefoot varus, tight heel cords, and tibia vara.[298] Training errors can include increase in training mileage; a single severe running session; increase in training intensity; running on hills, uneven, or slippery terrain; and resumption after an extended period of inactivity.[298] The differential diagnosis of Achilles tendinitis includes retrocalcaneal bursitis, os trigonum fracture, or plantaris or medial gastrocnemius muscle rupture.

***The method of presentation*** is as an acute exacerbation of a chronic problem, and the ***tissue injury complex*** is a myotendinous junction of the Achilles tendon.

***The clinical symptom complex*** will present as pain 6 to 8 cm proximal to the insertion of the Achilles tendon into the calcaneus, and will be worse with resisted plantar flexion.

***The functional biomechanical deficit*** will include weak ankle dorsiflexors and inflexible plantar flexors, with the ***functional adaptation complex*** being the functional pronation of the foot and increased knee flexion.

***The tissue overload complex*** will include the gastrocnemius-soleus muscle groups and common Achilles tendon.

***Rehabilitation*** for Achilles tendinitis is outlined in Table 8-24. Initial treatment consists of antiin-

**TABLE 8-24**

**TREATMENT FOR ACHILLES TENDINITIS**

| Time (d) | Treatment |
|---|---|
| 0–3 | NSAIDs, cryotherapy, heel lift |
| 3–7 | Stretching on incline board |
| 7–14 | Strengthening ankle dorsiflexors, plantarflexors, proximal limb muscles |

NSAID: nonsteroidal antiinflammatory drug

flammatory medications, ice baths, and gentle stretching on an incline board. Often, one-half inch heel lifts placed in the soldier's shoes can give temporary relief of pain. To prevent an abnormal gait, heel lifts need to be placed in both shoes, even if only one ankle is symptomatic. Placing a rubber Neoprene pad over the Achilles tendon can keep the affected area warm and give symptomatic relief. In chronic conditions, ultrasound and other heating modalities are helpful prior to stretching to help loosen up the scarred, chronically inflamed tissue. Soldiers in whom Achilles tendinitis reaches a chronic stage require a long time to heal; generally, a 4 to 6 week reduction in active duty is mandatory.[303] When in a situation that requires maximum functioning, despite significant symptoms from Achilles tendinitis, taping of the tendon can be done. Techniques are described elsewhere[303] and should be reserved for situations where activity is necessary and taping the Achilles is necessary to prevent rupture. Aerobic, noninjured leg, and upper body conditioning is mandatory.

### *Plantar Fasciitis*

Plantar fasciitis results from repeated traction on the plantar fascia at its insertion into the calcaneus.[304] Microtears and inflammation of the plantar fascia at the calcaneus can result from limited ankle dorsiflexion owing to a tight gastrocnemius-soleus complex.[305] The normal biomechanics of running dictate that the foot strike the ground in the supinated position with subsequent forefoot pronation in foot-flat and return to supination at toe-off. In individuals with pronated feet, stress to the medial aspect of the plantar fascia during running is increased, thereby increasing the chance of plantar fasciitis.[298] The differential diagnosis of plantar fasciitis includes entrapment of the medial calcaneal branch of the tibial nerve, plantar arch strain, tarsal tunnel syndrome, and calcaneal stress fracture.

***The method of presentation*** is as a chronic injury, with the ***tissue injury complex*** being the tensile overload of plantar fascial insertion into the calcaneus.

***The clinical symptom complex*** presents as a point tender pain located along the medial plantar fascia just distal to the calcaneus, which is worse on arising in the morning or after sitting for awhile and then standing, running, or walking.

***The functional biomechanical deficit*** will include plantar flexor inflexibility, plantar flexor peak torque weakness, and a flexor endurance weakness, leading to functional pronation.[30]

***The functional adaptation complex*** will manifest as running on metatarsal heads, decreased stride length, decreased mileage, and foot inversion.

***The tissue overload complex*** will be eccentric overload of plantar flexors due to continued running.

***Rehabilitation*** of plantar fasciitis is outlined in Table 8-25. Initial treatment focuses on the use of cryotherapy and early relative rest of the injured foot. An ice massage or slush bath for 20 minutes, several times a day can temporarily alleviate discomfort, although it may not be successful for long periods of time.[306–308] Antiinflammatory medications are also useful. The injection of steroidal medication into the calcaneal attachment can help control inflammation.[307] The injection is given at the site of maximal tenderness, and it is rarely necessary to give more than 3 weekly injections. Heel pads and arch supports control excessive forefoot pronation and decrease symptoms. Biomechanical factors, such as increased pronation of the foot, can be corrected with an in-shoe, shock-absorbing, medially posted orthosis, or by Low Dye taping.[298,309] Stretching the Achilles tendon, hamstrings, and the plan-

**TABLE 8-25**

**TREATMENT FOR PLANTAR FASCIITIS**

| Time (d) | Treatment |
|---|---|
| 0–3 | Cryotherapy, address biomechanics |
| 0–7 | Steroid injection, heel pads |
| 3–7 | Achilles, hamstring, plantar fascia stretching, ankle dorsiflexion strengthening |

tar fascia are essential components of rehabilitation.[304,310] Aerobic, noninjured leg, and upper body conditioning should be maintained throughout the rehabilitation program. Plantar fasciitis may take 6 to 10 weeks to resolve. Running type activities are usually resumed when tenderness over the plantar fascia, morning stiffness, and pain with weight bearing have abated.[298] Patients who remain symptomatic 6 to 9 months after initiation of care are candidates for surgical intervention.

## CONCLUSION

Musculoskeletal injuries are among the most common injuries encountered in soldiers. A rational approach to their rehabilitation is essential. Understanding the pathophysiology of musculoskeletal injuries lays the groundwork for a focused rehabilitation program. Rehabilitation requires (*a*) establishing an accurate diagnosis, (*b*) minimizing the deleterious effects of the acute injury, (*c*) allowing for proper injury tissue healing while maintaining other components of fitness, and ultimately, (*d*) return to normal function while preventing reinjury.

Principles of rehabilitation of specific disorders have been discussed and outlined. Understanding the method of presentation of specific injuries, the tissues injured and overloaded, the clinical symptoms present, and the biomechanical adaptations of an injury are important ingredients in the rehabilitation program. Approximate time frames for treatment options have been addressed. Rehabilitation of musculoskeletal injuries must go beyond the resolution of symptoms to prevent recurrent problems.

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