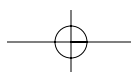


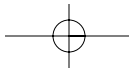
Replacement Chapter 22

Functional Soft-Tissue Examination *and* Treatment *by* Manual Methods

Third Edition

Warren I. Hammer





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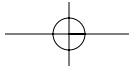
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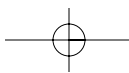
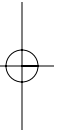
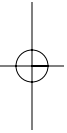
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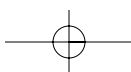
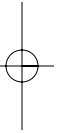
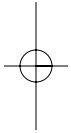
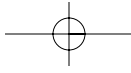
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This Chapter 22 replacement includes additional material from the Second Edition of *Functional Soft-Tissue Examination and Treatment by Manual Methods* that was omitted from the Third Edition.





CHAPTER 22

Active Release Techniques®: Logical Soft Tissue Treatment

P. Michael Leahy

Active Release Techniques® treatment is a collection of soft tissue techniques for examination, diagnosis, and treatment of soft tissue disorders.¹⁻³ These disorders fall under the category of cumulative injury disorder (CID), which is defined later in this chapter. Much like any manipulation, it must be learned in a practical setting with hands-on instruction. Although it would be impossible to include enough information in this chapter to allow the reader to place these techniques into practice, it is possible to present enough information to allow the discerning reader to make an informed decision on whether to pursue hands-on training. A small percentage of the information will be offered here in hopes of stimulating critical thought. It is also hoped that the availability of greatly improved outcomes will become evident.

Approximately 2 years are required to attain real proficiency, although most will agree that on the surface it appears that only minimal time would be necessary. This is where the major challenge to the professions exists. Those that go halfway will not achieve results that justify the use of the protected names. For the benefit of our patients, you are encouraged to use this chapter as a first step in the learning process.

ACCURATE DIAGNOSIS

The diagnosis of soft tissue disorders has traditionally been inadequate. A diagnosis of bursitis of the shoulder is inadequate for several reasons: It is not complete enough to indicate the exact tissue involved. Which bursa is it? More important, it does not convey the mechanism of injury. Bursitis of the shoulder is almost never the beginning or the end of the problem. Usually there is a biomechanical imbalance

of the shoulder that leads to altered mechanics, resulting in friction on a bursa such as the subacromial bursa. Another question therefore arises: What is the original injury and to which tissue may it be attributed? A three-part diagnosis is therefore necessary.

1. Nature of lesion: tear, adhesion, hypertension, etc.
2. Exact tissue involved: subscapularis, median nerve at the pronator teres, ligament of Struthers, arcade of Frohse, etc.
3. Result of lesion: peripheral nerve entrapment, altered mechanics, facet syndrome, lymphatic edema, etc.

In addition to normal methods of examination the practitioner adds a much more detailed examination by palpation. The examination may be made much more efficiently with a thorough understanding of soft tissue biomechanical principles. An example is the swimmer with anterior shoulder pain at the biceps tendon or intertubercular sulcus. With knowledge of shoulder and swimming mechanics the practitioner would immediately test and palpate the subscapularis and almost always find the underlying cause of the symptoms there.

The history of injury, sites of pain, strength of individual muscles, range of motion, soft tissue motion and relative motion, tissue texture, and tension will quickly lead to the cause of the problem. Learning soft tissue mechanics is a lengthy process but yields great dividends.

Much referred pain may be traced to anatomic origins or biomechanical relationships. Pain at the anterior shoulder, for example, is often caused by an infraspinatus injury on the posterior shoulder that causes hypertension along the muscle to its insertion. Constant hypertension leads to cellular hypoxia at the insertion, where circulation is poor

even under normal circumstances and pain/hypersensitivity is the result. There are many examples of how “referred” pain can be caused in this manner.

SOFT TISSUE CHANGES WITH INJURY

The soft tissues undergo a series of events after injury. The inflammatory phase may last 24 to 72 hours and can be recognized by examination and recent history of injury.⁴ Muscle tension may exist as a result of pain and tissue irritation. The swelling associated with inflammation is very fluidlike. The texture is more like a contained fluid and less like a firm mass with a palpable texture. The fluid is usually much more movable than that of a lymphatic edema and is not confined to distinct borders like those of a cyst.

After inflammation, the muscles become altered in tension and texture. Hypertense fibers are palpable and the overall texture seems to be “stringy” because of nonhomogeneous hypertension among the fascicles. This might be described as a “guitar string texture.”

The actual tear or defect in a muscle or fascia is evident from the beginning but after about 3 days the edges are more defined and palpable. After this period the edges become enlarged as the injury begins to heal with a fibrous process. This fibrous process leaves an adhesion that changes texture over time. From guitar string fibers in a muscle it changes to lumpy then leathery. The defect in the tissue gets very firm then spreads out into the surrounding tissue, leaving the ill-defined leathery area. All these changes occur in order. These changes are most easily identified within muscle tissue. The changes for other soft tissues are similar.

Soft Tissue Changes After Injury Over Time⁵

Inflamed	24–72 hours
“Stringy” muscles, lesion defined	2 days–2 weeks
Lumpy tissue	2 weeks–3 months
Leathery tissue, changes slowly	3 months and beyond

At this point it is necessary to define CID, as most of what is treated in terms of soft tissue injury is actually CID.

CUMULATIVE INJURY DISORDER DEFINED

Cumulative injury is a group of injuries to the muscles, tendons, bones, blood vessels, fascia, and/or nerves. It is helpful to understand that CID results from acute injury, repetitive injury, or a constant pressure/tension injury, all of which lead to what we term the cumulative injury cycle. It is the existence of the cumulative injury cycle that separates

this disorder from other injuries of the neuromusculoskeletal system.

Injury Types

Acute Injury

The muscle and fascial tearing from an acute injury immediately results in inflammation. The white blood cell—fibrinogen—adhesion process easily sets in.⁴ If this is not treated correctly, the cumulative injury cycle may be initiated.

Repetitive Motion Injury

Repetitive motion appears to be related to specific physical factors.⁶ In order to understand how repetitive motion injuries happen it is helpful to review the model of repetitive motion.⁵ This is a proportional model or guide and not an exact or linear equation.

$$\text{Model of repetitive motion: } I \approx \frac{NF}{AR}$$

In this model the factors are

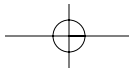
- I** = insult to the tissues
- N** = number of repetitions
- F** = force or tension of each repetition as a percentage of maximum muscle strength
- A** = amplitude of each repetition
- R** = relaxation time between repetition (lack of pressure or tension on the tissue involved)

Damage to tissues results from (High repetitions) (high and is proportional to force) (Small motions) (short relaxation time)

Vibration, for example, results in **N** that is very high, **A** that is very low, and **R** that is very low. The result is a total insult to the tissues that is very high. Posture that is poor and unchanging results in constant high forces in the musculature. **F** is high, **A** is near zero, and **R** is essentially zero. The total tissue insult is therefore high. A person who is very weak will use a higher percentage of maximum strength to accomplish a given task. **F** is high and total tissue insult is high.

Constant Pressure/Tension Injury

Constant pressure/tension injury factors will decrease circulation and compromise cell recovery to many factors. Cellular retention of calcium, poor repair, and altered function are but a few of the major results.^{7,8} This factor by definition



does not require a repetitive motion or any motion at all. An isometric contraction of a muscle and the muscle tension of poor posture are good examples of this potential injury.

The Cumulative Injury Cycle

The cyclic nature of injury has been described for many years.⁹ The cycle itself is defined by the following factors and their relationships. Each factor causes the next one in the cycle.

Weak and Tight Tissues

Repetitive effort, for example, tends to make the muscles tighten.¹⁰ When a muscle is tight it tends to weaken and when a muscle is weak it tends to be tight. The other soft tissues may also be drawn tightly.

Friction-Pressure-Tension

As a result of weak and tight tissues, the internal forces acting on the tissues rise. Friction, pressure, or tension can be present, or all at the same time. If one or more of these factors is high enough, an acute injury and inflammation can result even without external forces being applied.

Decreased Circulation—Edema

The effect of increased forces on the tissues is to decrease circulation. If pressure is applied over one of the vulnerable low-pressure lymphatic channels the result is edema. External forces in the form of constant pressure or tension injury may also have the effect of decreasing circulation or causing edema.

Adhesion-Fibrosis

Cellular hypoxia from restricted circulation causes fibrosis and adhesions to occur in and between tissues.⁸ When the friction-pressure-tension factor is severe enough or when an acute injury occurs, two additional factors come into play.

Tear or Crush

The physical disruption of the tissues (microscopic or macroscopic) can occur with sufficient forces. This can be an external force acute injury or it can result from severely increased internal forces.

Inflammation

Inflammation results from the tissue injury and begins the adhesion process, at which point the cycle continues to “weak and tight tissues.”⁴

There are many extrinsic factors that affect the cycle but they do so in predictable ways. Smoking, for example, tends to make circulation less efficient and therefore helps perpetuate the cycle. Diabetes has a similar effect. Thyroid deficits tend to increase tension in the musculature and therefore influence the total insult to tissues as well as the weak and tight factor of the cumulative injury cycle. Hormonal changes with hysterectomy, excessive body weight, and pregnancy all lead to predictable changes in the factors involved.⁶

A diagram of the cumulative injury cycle is helpful (Fig. 22–1). The cycle is defined with two possible routes, the inflammatory cycle and the chronic cycle. These may occur simultaneously.

Conditions

The cumulative injury cycle is self-perpetuating and as this downward spiral continues, the symptoms and syndromes of cumulative injury disorder are produced. These may include carpal tunnel syndrome, cubital tunnel syndrome, epicondylitis, tenosynovitis, myofascitis, bursitis, peripheral nerve entrapment, thoracic outlet syndrome, De Quervain’s disease, and others. When the cumulative injury cycle is not present, these syndromes are not a CID.

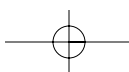
EXAMINATION

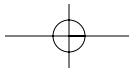
In order to arrive at a proper diagnosis, the soft tissues must be examined for problems in four major categories. One must develop the experience to distinguish normal from altered in each of these areas.

1. Tissue texture
2. Tissue tension
3. Tissue movement
 - joint motion and position
 - soft tissue motion and position
 - gliding of soft tissues over adjacent tissues
 - elasticity of muscle, fascia, ligament, and tendon
 - range of motion for all tissue
4. Tissue function
 - strength and speed of muscle contraction
 - nerve conduction
 - circulation of blood and lymphatics

Tissue Texture

Normal tissue texture requires all adhesions to be released so that the resulting tissue is homogeneous with the surrounding tissue of the same type. All tissues have a texture that can be recognized as normal by palpation. Of





range of motion with appropriate loading performed at or near the maximum elongation of the tissue. Elasticity is important for function. If one looks at the equations of motion that apply to the musculoskeletal system, it is easy to see the extent of this influence.

Joint and soft tissue ranges of motion must be assessed together. For soft tissue motion, the examiner must be able to palpate the gliding motion of tissues over one another. This can be difficult to learn and frequently requires 2 years or more of practice. A good test of this skill is to find the median nerve as it courses posteriorly to or through the two bundles of the pronator teres. The nerve must slide by the muscle and not be significantly pulled by this structure. There are many other sites where a soft tissue must slide over another. It is the rule rather than the exception.

When joint range of motion is limited, the examiner must also establish whether this is due to joint limitation or restriction caused by the soft tissues. This can only be accomplished by palpating the soft tissues while moving the joint to its limits. If the soft tissue is the limiting factor, then the tension that develops in the soft tissue will be excessive, early, and palpable.

Tissue Function

This brings us to the final factor, function. This includes proper strength, speed of contraction, and tonus of muscle as well as proper nerve conduction and vascular function. Venous and lymphatic drainages are the most susceptible because they are low-pressure systems. It takes less pressure over them to cause a blockage. It has been the experience of this author to see lymphatic edema of the upper and lower extremity recede over as short a time as 10 minutes after local blockage is treated. Improvements in other soft tissue functions such as strength, speed, and nerve function are rapid as well.

ALTERED BIOMECHANICS: JOINT AND SOFT TISSUE

When any of the factors are altered, the resulting function of the system is altered. The single-loop negative feedback control system (Fig. 22-2) is the model of how the musculoskeletal system is controlled. Changes in the texture of tissues most often affect the damping coefficient of the system.

The factors affecting the system are as follows:

Damping coefficient ⇒ Adhesion, friction, moments of inertia
Forcing function ⇒ Voluntary contraction

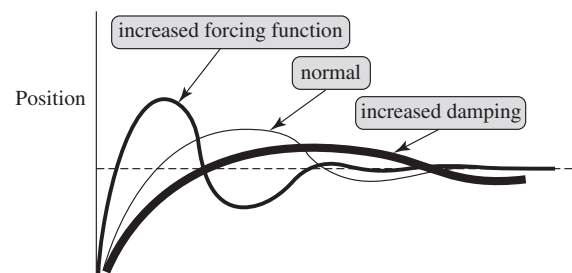


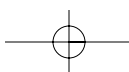
Figure 22-2 The single-loop feedback control mechanism.¹
 Source: Reprinted with permission from P.M. Leahy and L.E. Mock, *Myofascial Release Technique and Mechanical Compromise of Peripheral Nerves of the Upper Extremity, Chiropractic Sports Medicine*, Vol. 6, pp. 139-150, © 1992, Williams & Wilkins.

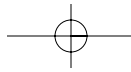
When the effort or muscle contraction is greater, the rate at which position approaches the desired result is faster, but more overshoots are a by-product. When an injury occurs and adhesion is the result, the damping coefficient is increased and the time necessary to achieve the result is longer. The subject will usually increase effort to compensate for this. Peripheral nerve entrapment will alter the forcing function. By this method of analysis, the effects of soft tissue lesions are very predictable.

When the soft tissue structures are injured the sequel to the injury is one of altered biomechanics. It is impossible for a muscle, for example, to undergo a significant tear, develop an adhesion, and then perform normally. Both function and movement on a macroscopic scale are altered. Shoulder biomechanics is a good example.

For the swimmer, weight lifter, or throwing athlete, subscapularis injury is common. This can be sudden or the result of repetitive motion or overuse. As a lesion develops in the subscapularis its function is negatively affected and the force couple mechanism is altered. Because the subscapularis is essential in preventing translation of the humerus anteriorly and superiorly, the result of this deficit is to allow translation of the humeral head superiorly and anteriorly. The humerus does not stay centered in the glenoid fossa. This can cause impingement of the supraspinatus, increased load on the glenoid labrum, and a decrease in performance. Joint mechanics has been directly altered by the soft tissue lesion.

After serious shoulder injury the inflammation process can affect most of the soft tissue in the area. A crepitus under the scapula is a good example of the possible results. An adhesion develops between layers of muscle that must slide over one another. As the fascia develops adhesions, this sliding is restricted and the crepitus develops. A common site is between the serratus anterior and the thoracic cage. If the serratus anterior is treated from an axillary approach and again from a posterior approach, the friction can often be relieved.





There are many sites where proper soft tissue mechanics is imperative. The force couple of the glenohumeral joint is just one. Others may not be quite so obvious. These include the following:

- malposition of carpals due to wrist extensor lesions
- scapulothoracic crepitus
- headaches caused by tissue hypertension at attachments of the cranial aponeurosis
- clicking or locking temporomandibular joint
- lumbar facet syndrome caused by psoas lesion
- recurrent pelvis/sacroiliac subluxation due to psoas/adductor lesions
- patellar-femoral arthralgia
- clicking meniscus of the knee
- clicking hip
- iliotibial band syndrome
- femoral translation on tibia leading to capsular attachment pain
- tennis/golfer's elbow
- "shin splints"
- compartment syndrome

Although most practitioners resist the idea until they have seen it for themselves, all of these conditions are usually resolved with just a few treatments, even if they have been chronic for years.

PERIPHERAL NERVE ENTRAPMENT

Although tissue changes that follow injury apply equally to all the soft tissues, a discussion of peripheral nerve entrapment and CID illustrates these principles very well. This is the area where the best documentation of the efficacy of Active Release Techniques exists. This area also best shows how the system of Active Release Techniques involves step-by-step analysis of mechanisms leading to precise treatment.

Many authors cite various locations of possible nerve entrapment. There are more sites of peripheral nerve entrapment by soft tissue than there are sites of entrapment at nerve roots exiting the spine. Consequently, if we are to successfully address proper nerve function we must address peripheral nerve entrapment sites with at least the same effort as we give to the spine and nerve roots. The following is a list of structures that entrap peripheral nerves that have been shown to respond very quickly to active release techniques. The following are only the most common.

- tunnel of Guyon
- thenar muscles
- carpal tunnel
- wrist flexors
- subscapularis
- serratus anterior
- rhomboids
- shoulder girdle

- pronator teres
- brachioradialis
- wrist extensors
- arcade of Frohse
- supinator
- brachialis
- triceps
- lateral intermuscular septum
- ligament of Struthers
- quadrangular space
- scalenes
- suboccipital muscles
- psoas
- gluteus medius/minimus/piriformis
- adductors
- hamstrings
- peronei
- leg compartments
- ankle retinaculum

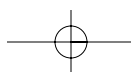
VASCULAR AND LYMPHATIC ENTRAPMENT

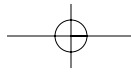
There are numerous sites of entrapment or blockage of circulation. In the extremities it is common for blood circulation to be limited by pressure of a muscle over a vein. It is even more common for a lymphatic vessel to be closed by pressure from an overlying structure. This is because the lymphatic system is one of very low pressure. Fluid is moved along by muscle contraction and movement. If a muscle is injured and is allowed to heal with excessive tension due to scarring, this can easily cause a constant pressure to be applied to the underlying lymphatic vessel. An example of such an event is an adductor strain blocking the hiatus of the adductors. The knee in this case is often susceptible to a lymphatic edema until the pressure is relieved. Other sites include the subscapularis, pronator teres, psoas, popliteus, and leg compartments. The edema is usually relieved with one to three treatments. When resolution is not obtained this quickly, other reasons for lymphatic blockage such as neoplasm and systemic circulatory problems must be considered.

METHODS OF TREATMENT

Much confusion exists with the term *myofascial release*. This term can mean anything from massage to aggressive soft tissue work aimed at "releasing" certain tissues. Unfortunately, much misinformation is still common, so in order to make the issue clear, the term Active Release Techniques treatment was coined to describe the work herein. Although many distinctions apply, the simplest to observe is that specific patient and provider movements are unique to Active Release Techniques treatment. This plus the unique process of following several specific symptom patterns to treatment sites, evaluation, diagnosis, and treatment define the techniques.

In Active Release Techniques the tissue is placed in a shortened position. The lesion is trapped with 1 of 13 contacts, then the tissue is drawn under the contact while the lesion is manipulated. The manipulation, tissue motion,





and contact vary with the requirements of each tissue, each lesion, and the reactions of the tissue during the treatment. There are two methods of utilizing patient motion during examination and treatment, active and passive.

Passive Active Release Techniques Treatment

In some cases it is advantageous to move the structures for the patient. In cases where there is extreme sensitivity, one often finds that when the patient moves the body part a response of muscle tightening in the entire area can occur. Adjacent areas are sometimes affected as well, leading to complication of pain patterns and hypertonicity. In some cases it is also difficult for the patient to understand or duplicate the compound motions required. When either of these is the case, the practitioner should move the body part for the patient.

To accomplish this the practitioner must support the body part in a manner that instills confidence and allows the patient to relax. If the patient begins to resist, the practitioner must reduce contact tension, speed, or another factor to accommodate. Care must be taken to avoid forcing the motion against a muscle contraction, as the muscle that contracts to oppose the motion is most likely the same one that is being treated.

Full range must be employed as much as possible. The practitioner cannot determine the nature and extent of the lesion or improvement unless the full range is explored and treated.

Active Active Release Techniques Treatment

Whenever possible the patient motion should be active. This is important for several reasons. First, the patient will feel more in control and can modify the pace of the treatment as to his or her tolerance. Second, the act of voluntary motion will neurologically reduce the sensation of pain and allow more comfortable treatment. Third, the motions available will more closely follow the ones that are required for the patient to perform a task. A side benefit is that it allows the practitioner to have two free hands for treatment administration such as with the contacts that require a backup.

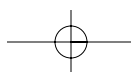
The study of Active Release Techniques involves a learning curve. After only 12 to 24 hours of hands-on training with an experienced practitioner, greatly improved results can be experienced. After 30 days of diligent practice with proper direction the practitioner may be capable of achieving 50% of the desired effects from treatments rendered. This is important because all too often the practitioner will read an article or obtain second-hand information, then assume that mastery of the techniques is obtained. This is falsely confirmed by early successes when compared with

some of the other treatment methods for certain injuries. If the learning process stops at this point, results will never amount to more than a fraction of those cited here. We must all accept that great technique and great results require great effort.

Treatment Rules of Application

Certain guidelines must be followed in order to ensure maximum benefit with minimum complication. If these rules are not understood and followed, patient welfare is compromised.

- **Soft contact:** Use the specific contact taught hands-on. It is impossible to learn the varied methods of contact without hands-on instruction.
- **Work longitudinally:** For many reasons, this method breaks the adhesions in and between tissues effectively. It is most important to establish longitudinal freedom of motion within a muscle, for example. If passes are made across muscle fibers the tissue will slide or roll under the contact before enough tension can be exerted on the fibrous tissue to break it.
- **Active motion whenever possible:** This provides the patient with a sense of control and also blocks pain at the lateral spinal-thalamic tract. More can be accomplished with less discomfort. The methods of active motion are very involved and are designed to maximize relative motion between tissues.
- **Slow motion:** If any motions are fast or quick, the tolerance of the patient is reduced and muscle tension is the reaction. This makes it difficult or impossible to achieve the desired results. The exact motions differ for each tissue and must be taught hands-on.
- **Patient tolerance:** The pressure and number of passes is always limited to patient tolerance. There are several keys or indications of patient tolerance level that must be observed.
- **Tissue tolerance:** Each patient is different, and the tissues react to physical stimulation in a different manner. It is sometimes necessary to delay a treatment because of tissue intolerance. This is indicated by physical as well as nonphysical indicators.
- **Work with lymphatic and venous flow:** Bruising and lymphatic edema are almost completely avoided by using the accurate methods described here.
- **Frequency—alternate days:** Treatment frequency is never more often than alternate days. This leads to tissue intolerance and protracted treatment plans. In some cases an even longer period between treatments is necessary.



Treatments should produce significant results within two or three treatments. Three to five passes are made over any single area of tissue. Sometimes it is necessary to reduce treatment frequency to twice per week or even less when the tissue tolerance is low. If treatment is continued at normal frequency in spite of low tissue tolerance, the number of visits required to resolve the problem goes up and sometimes resolution is impossible. Diminished muscle strength usually changes on the first or second visit. Treatment is not usually initiated during the inflammatory stage. When the injury is new (1 to 3 days), care must be taken to alter the directions of treatment to prevent increasing tension across a defect.

When trying to gauge progress it is important to realize that changes occur very quickly. After studying 223 cases retrospectively it was found that the average number of treatments necessary to resolve carpal tunnel and related syndromes was only six. If the practitioner finds many cases requiring more than 12 visits it is usually improper technique that is to blame.

A very basic explanation of the technique and its application to certain muscles is offered. This explanation does not include many factors that are taught hands-on with video demonstration and are too lengthy for discussion here. Also included is a description of long tract nerve release, a new concept which can be used to increase the relative motion between the nerve and the adjacent tissue (Appendix 22–A).

The Rotator Cuff

Although there are 250 treatment techniques, analysis and treatment of the rotator cuff may illustrate proper use of the techniques. For this purpose a discussion of rotator cuff principles and 4 of the 250 treatments from the forthcoming full text is offered.

The shoulder girdle has very little static or ligamentous stability. Its stability is dynamic and requires almost perfect synergism of all the muscles. Structural defects to any of the bony, cartilaginous, or musculotendinous units involved prevent perfect function. To evaluate and treat shoulder dysfunction adequately, it is essential to understand the difference between the muscles that supply power for shoulder motion (deltoid, latissimus dorsi, and pectoral muscles) and those that supply shoulder stability (the rotator cuff). The rotator cuff (subscapularis, infraspinatus, teres minor, and supraspinatus) acts with the deltoid in force couple mechanism during elevation of the arm to guide the humerus in its movement in the glenoid cavity. At the initiation of abduction, contraction of the rotator cuff acts as a force couple with the deltoid and maintains the depression of the head of the humerus during abduction. If the deltoid were to contract on its own without this depression by the

cuff muscles, the humerus would jam superiorly into the overlying acromial arch.

The glenohumeral joint is an incongruous joint having a shallow concave surface that articulates with a more convex and dissimilar surface. The movement is not around a fixed axis, but rather a gliding motion with a constantly changing axis of rotation. The muscles must not only move the joint, but must also give it stability.

The glenoid labrum is so shallow that the rotator cuff is necessary to hold the humeral head roughly centered in the glenoid labrum. The subscapularis is a powerful anterior stabilizer, making it crucial to that stability.

The Subscapularis

The subscapularis is a large triangular muscle that fills up the subscapular fossa. Its fibers converge and form a tendon that inserts into the tuberosity of the humerus.

When working the subscapularis, place the patient supine with the humerus in the anatomic position (Fig. 22–3A). Flex the elbow and internally rotate the humerus. Using your free hand place the thumb over the anterior side of the latissimus dorsi at the midpoint of the scapula. Palpate the entire subscapularis. At the lateral area you will palpate the latissimus, teres major, and teres minor. Proceeding medially, the entire surface of the scapula is covered with subscapularis muscle. At the medial area, the serratus anterior can be felt as it attaches to the medial border of the scapula. This is often adhered to the subscapularis and can prevent complete palpation of the subscapularis. The superior fibers of the subscapularis are more horizontal and the inferior ones are angled inferiorly and medially. The fiber direction can be felt.

The superior area must be palpated judiciously, as the chords of the brachial plexus cross here with the blood and lymphatic vessels. Care should be taken to ascertain the condition of the interface between the muscle's fascia and the structures that cross the muscle. The medial chord of the brachial plexus should slide approximately 1.5 cm during abduction and external rotation of the humerus. Elbow flexion and wrist extension further accentuate this gliding. If an adhesion exists between the medial chord and the muscle, the lack of gliding can be felt and there is often a reproduction of paresthesias, which usually ceases within 1 minute after palpation.

The area of injury will have one of the four altered textures. Once the area of injury is determined, place the contact on the skin inferior to the lesion. Move the skin to a point above the lesion (superiorly and laterally) and begin to take up the slack in the muscle by stretching it inferiorly and medially. The contact should arrive at the superolateral border of the lesion when maximum tension is obtained

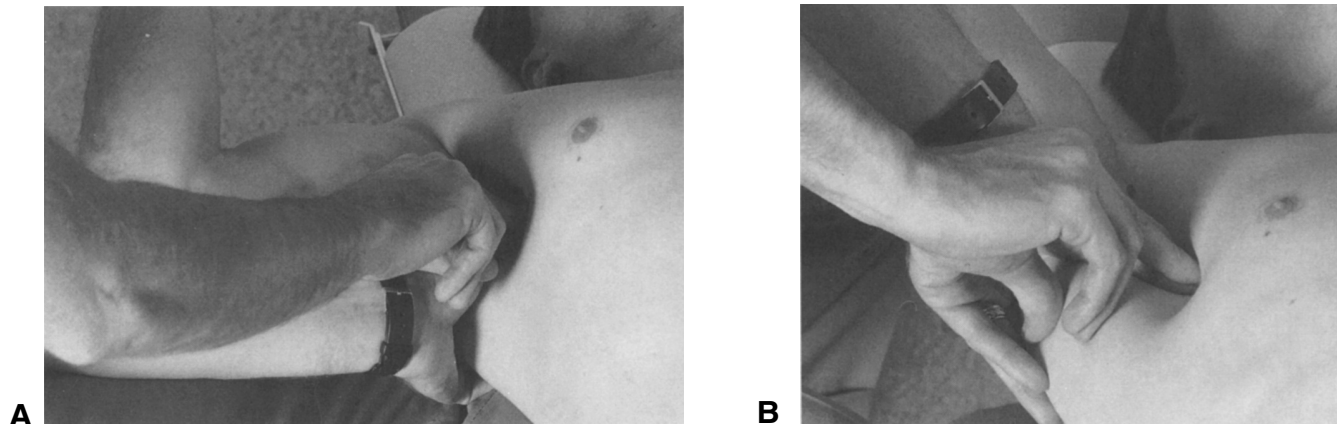
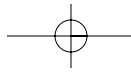


Figure 22-3 Subscapularis treatment. **A**, Beginning position; **B**, ending position.

within the muscle (pull the skin up, then pull the muscle down). Tension within the altered tissue is the objective here, and not compression. Excessive compression will cause excessive discomfort and limit or even preclude effectiveness of treatment. Excessive tension on tissues other than those being treated will have the same effect.

Once the initial contact is established, have the patient begin to abduct the humerus to a point where the arm is draped over the top of the head (Fig. 22-3B). During the motion, the contact is moved a varying amount inferiorly and medially as is warranted by tissue tension and other factors. During this motion the texture of the lesion is more obvious, and other features may become evident. The practitioner must be able to recognize a vein or tear in the fascial covering over the muscle. Failure to recognize this may lead the practitioner to injure the patient. Three to five passes are made over any single tissue area. As the lesion or “adhesion” is broken up, the release is felt by the practitioner but very seldom by the patient. Extremely painful treatment is usually the result of poor technique and should be discontinued as improvement of the condition is unlikely.

The Infraspinatus

The infraspinatus is usually involved to some degree with most rotator cuff problems. Because it is very active in external rotation of the humerus, it is usually found to be a key factor in sports involving this motion. Racquetball and tennis are examples. Because it stabilizes the shoulder and must act to decelerate the humerus in throwing and similar motions, it is also involved in most of the other activities of the shoulder.

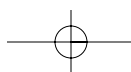
Lesions may be found anywhere along the course of the muscle but are most common on the posterior surface of the scapula. Contact is made with tissue drawn tight on the side of the lesion closer to the humerus. The patient is prone and the arm is taken from the adducted (elbow next to the side) position (Fig. 22-4A) to the fully abducted (full reach above the head) (Fig. 22-4B) and internally rotated position. The muscle will change shape and tension during this motion, so it is necessary to change the contact during the motion to trap the muscle and avoid letting it roll out from under the contact. Pain may be referred to the anterior shoulder during the treatment. Fiber orientation must be taken into consideration, as the superior portions are much more horizontal than the inferior ones. Also be aware that pressure and direction of the stripping motion over the lesion must change as the scapula rotates.

The Teres Minor

The teres minor involves the same humeral motion as the infraspinatus. The contact, however, is more lateral and is more difficult to maintain during the motion. The thumb is usually slightly flexed in order to maintain a more comfortable contact over the tissue and to prevent it from “rolling” out from under the contact (Fig. 22-5).

The Supraspinatus

The supraspinatus is often subjected to much friction under the acromion and the coracoacromial ligament. As a result, the texture of this structure will differ from the most common muscular adhesions. It will feel much tougher and



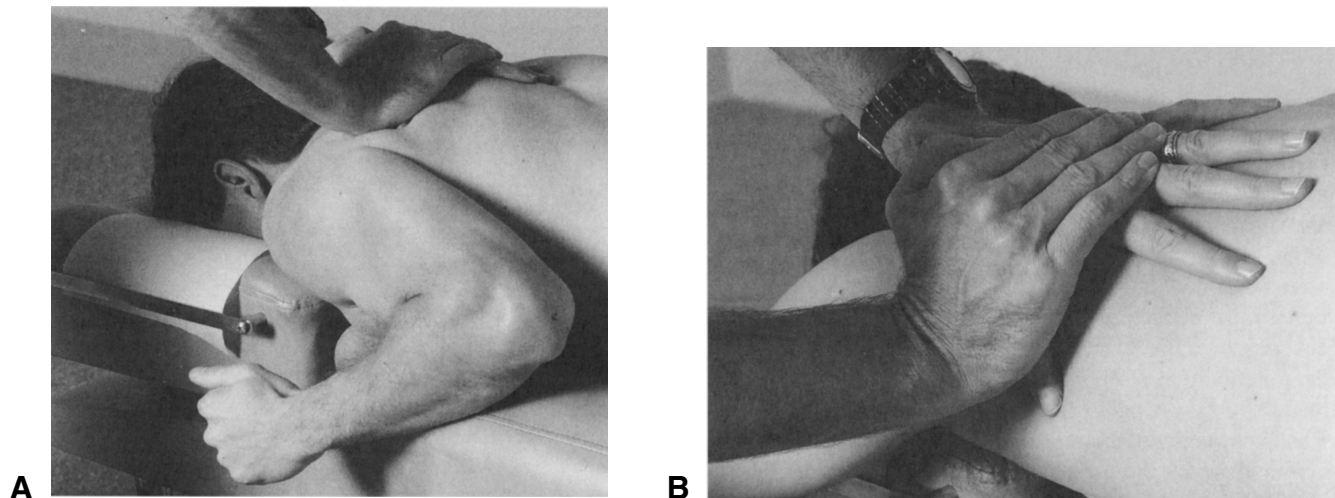


Figure 22-4 Infraspinatus treatment. A, beginning position; B, ending position.

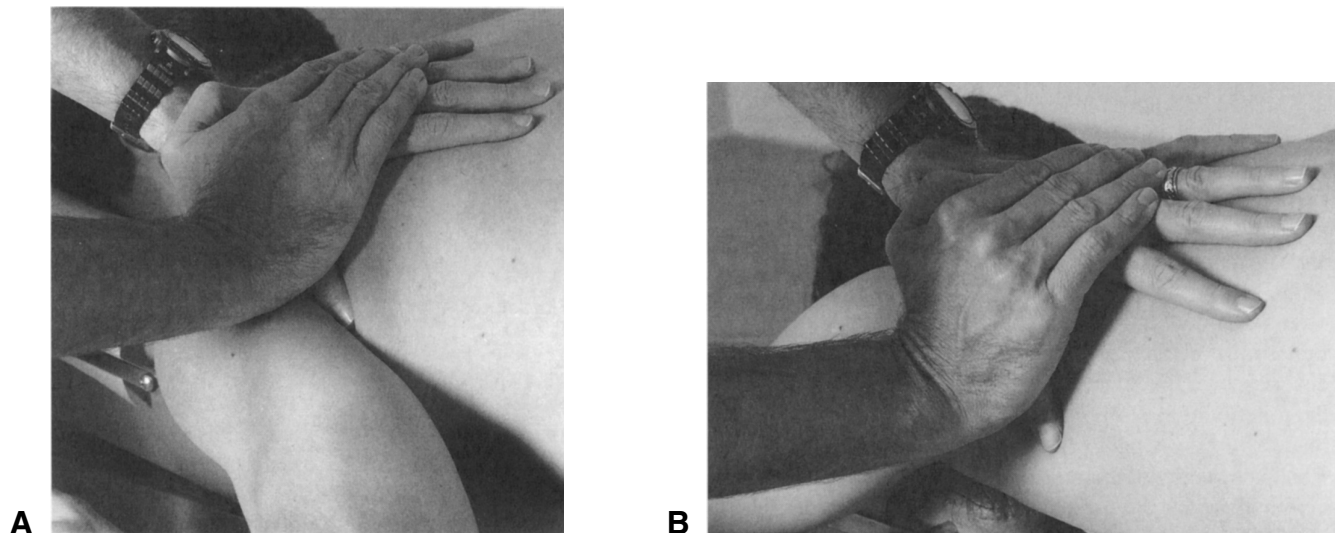


Figure 22-5 Teres minor treatment. A, Beginning position; B, ending position.

“leathery.” The supraspinatus is superficial in most of its course. Almost all areas of this muscle may be treated by taking several passes in different positions, exposing the medial and lateral parts from under the acromion process. Work the majority of the muscle by beginning with the patient seated, arm fully abducted. Use a double-thumb con-

tact just medial to the acromion process and superior to the spine of the scapula, and have the patient slowly lower the arm to the side, then bring the elbow across the abdomen with slight external rotation of the humerus (Fig. 22-6). Hammer suggests moving the arm behind the torso. Work the tissue laterally to the acromion with additional passes.

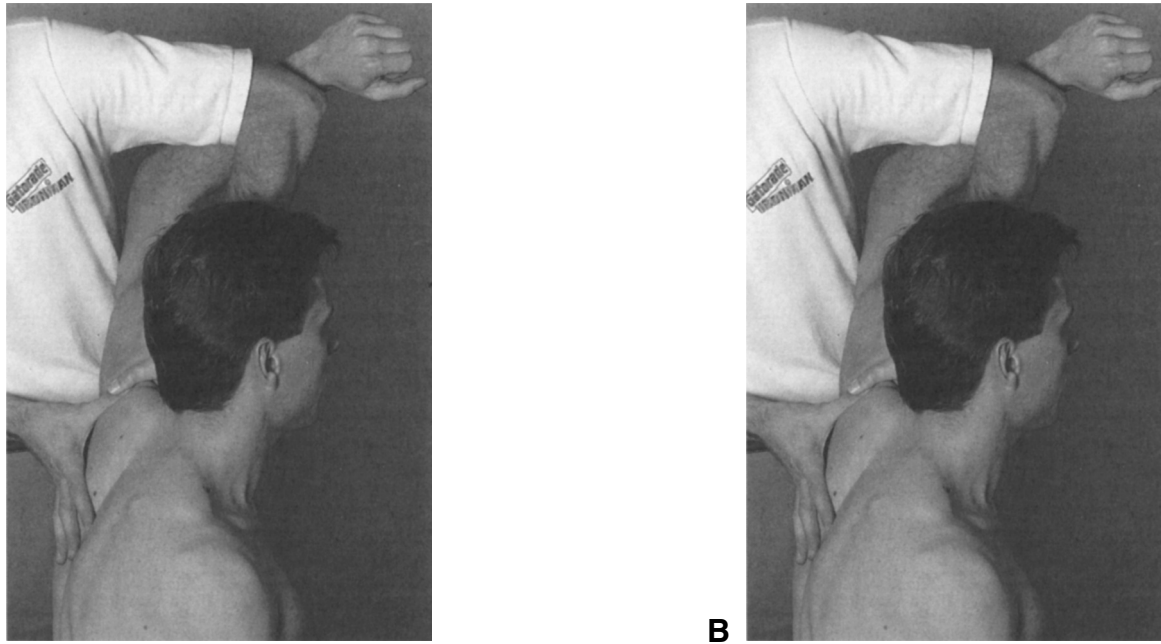
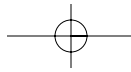


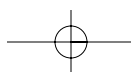
Figure 22-6 Supraspinatus treatment. **A**, Beginning position; **B**, ending position.

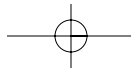
OUTCOMES EXPECTED

In general, changes in tissue texture are evident during treatment. The provider should feel adhesions separate or “break loose” during the passes over the tissue. Circulation and function of the tissues should improve immediately. The four categories of objectives should be attained at least in part on each visit. It is unacceptable to continue treatment without measurable gains on every visit unless exceptional and identifiable factors delay the onset of improvement. Severity or chronicity of the condition is usually not such a factor. Changes in the treatment program are often required to accommodate for a plateau in progress.

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APPENDIX 22–A

Active Release Techniques®: Long Tract Nerve Release

Many manual methods have been devised for the treatment of muscle, and a few have been developed for the treatment of peripheral nerves. Some are designed to “stretch” the nerve, and some are meant to make the nerve “glide.” These methods have proved to be valuable, although much more should be done to find a way to separate a peripheral nerve from an adjacent tissue more effectively. When an adhesion develops between a nerve and a muscle, for example, normal motion of the body can cause a traction neurodesis. This process causes neurologic symptoms distal and sometimes proximal to any of the “tunnels” through which the nerves pass. The adhesion and traction neurodesis process also causes neurologic symptoms at sites independent of the traditional tunnel sites.

For many years, active release technique (ART) treatment protocols were designed to release the nerve from its adjacent muscle, fascia, tendon, or bone. This was achieved by treating the adjacent tissue while concentrating the applied tension to the spot where the nerve passed through or under the tissue. In most cases, this would cause a relative motion between the nerve and the tissue.

In order to increase the relative motion between the nerve and the adjacent tissue, an entirely new concept must be employed. It is not enough to simply stretch or glide the nerve. We must concentrate on moving the nerve as far as possible while holding a tension on the adjacent tissue in the opposite direction. We must cause as much relative motion as possible. In general, this means shortening or relaxing the nerve on one side of the contact site and stretching the nerve on the other side of the contact site.

The concept is simple, but the application can be difficult because the positions and motions can be confusing.

Only a thorough understanding of the principle and dedicated practice of each treatment protocol can make these procedures useful. Active Release Techniques LLC is responsible for instruction in patented protocols.

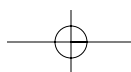
Treatment protocols have been designed for all the long-tract nerves, including the radial, median, ulnar, sciatic, posterior tibial, peroneal, femoral, and saphenous nerves. The nerve is moved in both directions through the adjacent tissue, so a total of 56 protocols are employed. The original ART protocols are performed first, so that the pressures on the nerve are minimized before the advanced protocols are used.

The treatment protocols are diagnostic. The provider must be able to assess the movement of the nerve past the adjacent tissue. It is common but not necessary to duplicate the patient’s symptoms during treatment. The provider should be judicious in the amount of tension applied to the nerve in such cases. It is also worthy to note that the modulus of elasticity of a nerve is such that the tension increases abruptly near the end of its motion, so anticipation and slow motions are necessary.

Although a complete description is not possible here, the basic procedure for the median nerve at the pronator teres and the saphenous nerve at the sartorius/gracilis are presented.

MEDIAN NERVE AT THE PRONATOR TERES/FLEXOR DIGITORUM SUPERFICIALIS

Step 1: Place the body in position so that the nerve is moved proximally. In this position, on the distal side of the entrapment site, the nerve is relaxed, and on the proximal side, the nerve is tractioned as far as possible (Fig. 22–7A).



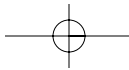


Figure 22-7 A and B A) Position the body so that the median nerve is moved proximally. B) Place the contact on the distal edge of the pronator teres next to the median nerve and push the muscle proximally, adjacent to the nerve. The contact should not compress the nerve.

- Flex the thumb and the next two fingers.
- Flex and pronate the wrist.
- Extend the elbow.
- Abduct and extend the shoulder.
- Laterally flex the neck away.

Step 2: Place the contact on the distal edge of the pronator teres next to the median nerve and push the mus-

cle proximally, adjacent to the nerve. The contact should not compress the nerve (Fig. 22-7B).

Step 3: Move each joint so that the nerve moves distally past the muscle (Fig. 22-8A).

- Laterally flex the neck to this side.
- Adduct the shoulder and forward flex slightly.
- Flex the elbow.
- Extend and supinate the wrist.
- Extend the thumb and the next two fingers.

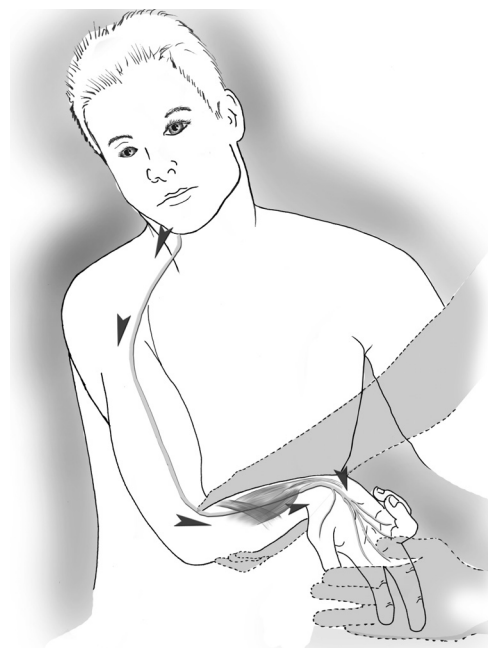
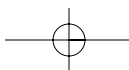


Figure 22-8 A and B A) Move each joint so that the nerve moves distally past the muscle. B) Move the contact to the proximal side of the muscles and push the muscle distally.



Step 4: Move the contact to the proximal side of the muscles and push the muscle distally (Fig. 22–8B).

Step 5: Move the nerve proximally again by pushing the muscle distally. This brings the patient back to the original position (see Figs. 22–7A and B).

- Flex the thumb and the next two fingers.
- Flex and pronate the wrist.
- Extend the elbow.
- Abduct and extend the shoulder.
- Laterally flex the neck away.

SAPHENOUS NERVE AT THE SARTORIUS/GRACILIS

Step 1: Place the body in position so that the nerve is moved proximally. In this position, on the distal side of the entrapment site, the nerve is relaxed, and on the proximal side, the nerve is tractioned as far as possible (Fig. 22–9A).

- Dorsiflex the ankle.
- Flex the knee.
- Extend the hip.
- Flexed the spine.

Step 2: Place the contact between the distal gracilis and the sartorius and push the muscle proximally. The contact should not compress the nerve (Fig. 22–9B).

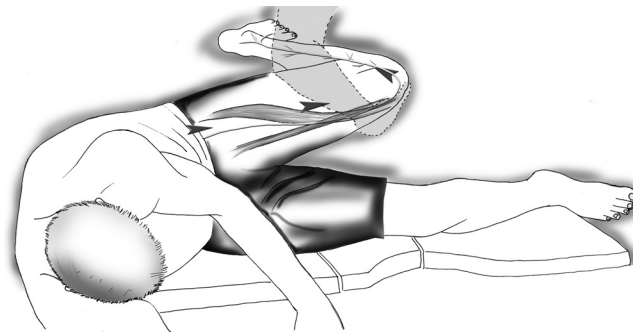
Step 3: Move each joint so that the nerve moves distally past the muscle (Fig. 22–10A).

- Extend the spine. Flex the hip.
- Extend the knee.
- Plantar flex and evert the foot.

Step 4: Move the contact to the proximal side of the muscles and push the muscle distally (Fig. 22–10B).

Step 5: Move the nerve proximally again by pushing the muscle distally. This brings the patient back to the original position (see Figs. 22–9A and B).

- Dorsiflex the ankle.
- Flex the knee.
- Extend the hip.
- Flex the spine.



A



B

Figure 22–9 A and B A) Position the body so that the nerve is moved proximally. B) Place the contact between the distal gracilis and the sartorius and push muscle proximally. The contact should not compress the nerve.

MENISCAL ENTRAPMENT OF THE KNEE

The meniscus of the knee must slide anterior with knee extension and posterior with knee flexion. The medial and lateral meniscus follow exactly the displacements of the femoral condyles (see Menisci in Chapter 7, Fig. 7–12). Lateral tibial rotation causes the lateral meniscus to be pulled anteriorly and the medial meniscus to recede while medial tibial rotation causes the medial meniscus to move forward while the lateral meniscus recedes. If the meniscus is not free to slide then it can be trapped or pinched during these motions. The patient will describe an internal pain or “pinching” during extension or flexion. If severe the patient will describe a “locking” or restricted motion. It is with these cases that there is a danger of tearing the meniscus. When pinched the meniscus will tend to become inflamed.

The medial meniscus is attached to the tibia via the capsule and ligaments of the knee.

Injuries to the medial meniscus occur about 20 times more often than to the lateral due to its more generous attachments to surrounding tissues. Most notably it is fused

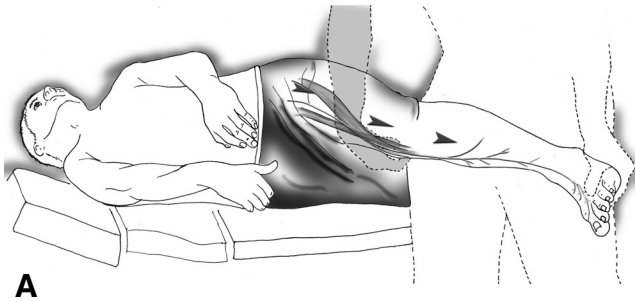
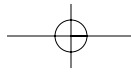
**A****B**

Figure 22-10 A and B A) Move each joint so that the nerve moves distally past the muscle. B) Move the contact to the proximal side of the muscles and push the muscle distally.

with the medial collateral ligament and the capsule. The medial collateral ligament is covered partly by the pes anserinus and tendon of the semimembranosus.

The lateral meniscus is attached to the femur via the two menisofemoral ligaments. It is in contact with the capsule which is in contact with the oblique popliteal and arcuate popliteal ligaments as well as the tendon of the popliteus. The lateral meniscus is much more mobile than the medial.

Because so many tissues are connected to the menisci directly or indirectly it is easy to see why the movement of a meniscus is influenced by virtually every tissue that crosses the knee. In order to evaluate meniscal movement one should consider each muscle, tendon and ligament that crosses the knee.

When a pinching or entrapment of the meniscus is evident by symptoms one must first ascertain the direction of relative immobility and the position of the knee that causes it. With the knee placed in this position the meniscus is then manually pushed in the direction that it is restricted. Simply find the position and direction that it will not move into then put it in that position and make it move with manual pressure.

Loss of Posterior Medial Meniscus Motion

With the patient supine, place the contact hand over the anterior half of the medial menisci. Flex the knee as far as comfortably possible and feel for a lack of posterior movement of the meniscus under the femur at the end of flexion. If the meniscus stops moving before the end of flexion it will feel like it stops and bulges out under the contact. This procedure will detect a lack of posterior movement of the meniscus relative to the femur.

If the medial meniscus does not move posterior enough, use the contact over the meniscus to push it into more posterior glide or stretch as full flexion is reached. Rotate the tibia laterally. Be careful to avoid using the lower leg as too strong a lever for flexion. A mild pressure into flexion is all that is necessary. The pressure on the meniscus must be slow and firm but not compressive (Fig. 22-11). The knee should be flexed while the tibia is laterally rotated.

Loss of Anterior Medial Meniscus Motion

For a medial meniscus that does not move anterior enough with knee extension the meniscus will stop moving before the end of extension and will feel like it stops and bulges out under the contact. Place the contact on the posterior half of the menisci and extend the knee in the same manner and feel for a lack of anterior motion of the meniscus. Be careful to avoid using the lower leg as too strong a lever for extension. Treat by extending the knee with the tibia rotated medially. A mild pressure into extension is all that is necessary. The pressure on the meniscus must be slow and firm but not compressive.

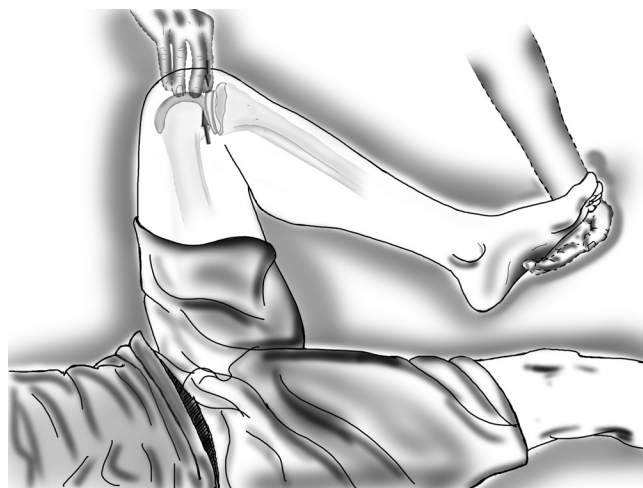
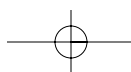
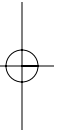
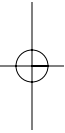
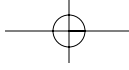


Figure 22-11 Flex the knee comfortably feeling for a lack of posterior meniscal movement before the end of flexion. Contact the anterior half of the meniscus and use a mild pressure during the flexion. The pressure should be slow and firm but not compressive.





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