JOINT RANGE OF MOTION

The amount of motion available at a synovial joint is called the **range of motion** (ROM). Normal ROM varies among individuals and is influenced by age, gender, body habitus, and whether motion is performed actively or passively.[1] The type and amount of movement that occurs throughout the ROM is unique to each joint of the body and is dependent primarily upon the shape of the articular surfaces. Other factors include the integrity and flexibility of the periarticular soft tissues.

Joint motion involves rotation or translation of one articular surface relative to the other about an axis known as the instantaneous, helical, or screw axis. Both rotation around the joint axis and translation along the instantaneous axis must occur to provide normal joint kinematics.[2] Joint movements that are produced actively through voluntary activation of muscles or passively through an external moment applied by a clinician to simulate active motions are known as **physiological movements**. Physiological movements occur around and along the instantaneous axis. For example, reaching overhead primarily involves the physiological movement of flexion at the shoulder. The majority of actual movement that occurs with shoulder flexion is rotation, which is a turning movement, about the instantaneous axis of the glenohumeral joint. However, some translation or a straight glide occurs to a lesser degree along the axis for most synovial joints. (i.e., rotation is the primary movement, while translation to a lesser degree along the axis). ROM activities are prescribed to prevent loss of physiological movements.

Isolated translational movements are known as **accessory movements** and they cannot normally be produced through volitional effort. Just as some translation occurs with physiological movements, some degree of rotation is produced when performing accessory movements. These translational movements are used in manual therapy procedures known as joint mobilizations and are used in combination with ROM exercises to restore joint kinematics.

BOX 1 STOP *and* THINK

Why are both physiological and accessory movements important to restore following joint injury?

In addition to joint integrity, adequate muscle length and other soft tissue extensibility must be maintained to optimize joint function.[3] Most muscles cross more than one joint to allow for shortening over one joint and lengthening over the other during movement. However, there is potential for a muscle or muscle group to become excessively lengthened or shortened when it crosses more than one joint. If a muscle is rendered weak because it is shortened as much as it can be as it crosses each joint, it is said to be actively insufficient. An example of this occurs when an individual lies prone with the hip extended or in neutral, and the individual is asked to perform a "leg curl" to flex the knee as much as possible. **Active insufficiency** occurs in the hamstrings in this example because they are shortened over the knee and hip at the same time, and full flexion of the knee may be difficult. The hamstring muscles may even spasm when such a maneuver is attempted without allowing hip flexion to occur to lengthen the hamstrings as they cross the hip joint. Figure 1 illustrates this principle. **Passive insufficiency** occurs when a muscle limits joint motion by being stretched or lengthened as an antagonistic muscle is contracting. If we use the example of the individual

Figure 1 Therapist must be aware of patient positioning to prevent muscle insufficiency when addressing strengthening or stretching of two joint muscles. In this example, the patient is flexing his knee while his hip is maintained in extension, the hamstring muscle group will become progressively less effective as it reaches maximum shortness with contraction (active insufficiency).

lying prone again, as the hamstring muscles act to flex the knee joint, the rectus femoris muscle of the quadriceps femoris group can limit knee flexion because of passive insufficiency. Both active and passive insufficiencies occur in healthy, nonpathological muscles, but ROM loss because of these is temporary. That is, as soon as the joint positions change to prevent active or passive insufficiency of the muscles, full joint ROM can be achieved.

BOX 2 STOP *and* THINK

Why should passive insufficiency be promoted at the hand and wrist (extrinsic flexors) in a patient with quadriparesis?

Conditions that may cause adaptive shortening of muscle, periarticular connective tissue, and skin will result in a more permanent loss of ROM. These conditions include prolonged immobilization, restricted mobility, connective tissue or neuromuscular diseases, tissue pathology because of trauma, and congenital or acquired bony deformities.[4] Limitation of joint ROM because of soft tissue adaptive shortening is known as **contracture** (Table 1). Contractures involving musculotendinous units are known as myostatic contractures. Pseudomyostatic contractures occur when there is hypertonicity from a lesion involving the central nervous system. The muscles in this situation are resistant to passive stretch and thus give the appearance of a myostatic contracture. Periarticular contractures result from intraarticular pathology, such as adhesions, chronic joint effusions, arthritis, or arthrosis.

While ROM activities are prescribed to maintain existing joint and soft tissue mobility, stretching activities are used to treat joint contractures because of muscle (myostatic contractures) or other soft tissue shortening. Fibrous changes in periarticular tissue respond to stretching as well. However, the longer the periarticular contracture exists, the more difficult it is to reestablish ROM and tissue length. Once the tissue is replaced with scar tissue that does not respond, the contracture becomes irreversible.

This lesson will focus on the ROM and stretching techniques used to maintain and restore physiological joint movements. The reader should keep in mind that these should be used in combination with manual therapy techniques (joint mobilizations) when necessary to restore accessory movements.

Impairments in ROM

Disease, injury, immobilization, overuse, and aging may result in pathological joint ROM. Excess motion beyond the physiological motion expected is known as **hypermobility** and may be caused by an inability of bone or soft tissue to limit motion. Hypermobile joints are not necessarily pathological, but are more susceptible to injury and pain because there are increased tensile and shear forces. Increased rotation and translation contribute to the tension and shear forces generated. Management of hyper-mobility is therefore aimed at joint protection. For example, a person with a total knee arthroplasty who complains of a feeling of instability while walking and whose knee demonstrates hypermobility in an anterioposterior direction when examined, may benefit from the use of exercises and functional activities that encourage co-contractions of the knee extensors and flexors. The co-contractions may help stabilize the knee in the anterior-posterior direction. Since the focus of this lesson is to apply the concepts of ROM and stretching, treatment of hypermobility will not be discussed further.

TABLE 1 ■ Common Conditions That Affect Joint Range of Motion

ADAPTIVE MUSCLE SHORTENING	PERIARTICULAR DISEASE	SKIN
Tight hamstrings associated with posterior pelvic tilt	Adhesive capsulitis	Burns
Tight trunk extensors associated with increased lordosis and anterior pelvic tilt	Systemic lupus erythematosus	Scleroderma
Hip and knee flexion contractures with tight hip adductors, flexors, and hamstrings	Rheumatoid arthritis	

BOX 3 STOP *and* THINK

What are the clinical concerns of a hypermobile joint?

Limited motion or **hypomobility** may be caused by bony or cartilaginous blocks or by the inability of the soft tissues (joint capsule, ligaments, tendon, muscle) to elongate sufficiently to allow physiological ROM (Figure 2). Motion may be limited either when the patient or individual performs the movement independently **(active motion)** or when the examiner performs the movement **(passive motion).** Muscle contraction or spasm, muscle weakness, neurological deficit or pain may cause differences in joint range of motion between active and passive motions.

Joint End-Feel

Passive joint motion is normally halted by tissue tension at end range or by tissue approximation, such as contact of one bone to another or by one body segment touching another body segment. When performing an examination of passive motion, an examiner should pay close attention to the sensation or "feel" at the end of the available passive movement. This sensation is known as an **end-feel**. Healthy joints have end-feels that are defined as "normal" for a given joint. A joint with an "abnormal" end-feel means that the "normal" end-feel that should be expected for that particular joint is not present. The use of the end-feel assessment can help classify the stage or severity of pathology for a particular joint and also provide prognostic information (Table 2).

BOX 4 STOP *and* THINK

Why are end-feels important?

Capsular Pattern

A specific pattern of limitation or restriction may be found when the clinician examines passive range of motion of the joint. This pattern is unique to a given joint (e.g., shoulder vs. hip) and usually represents capsular contraction, but may also represent muscle spasm, generalized osteophyte formation, or chondrocalcinosis. The pattern is described with the most limited movement listed first, followed by the next limited movement or movements. For example, at the glenohumeral joint, the capsular pattern is external rotation - abduction - internal rotation. This means that external rotation is the most restricted of all passive movements of the glenohumeral joint, followed by abduction and then internal rotation (Table 3). The reader is referred to the work of

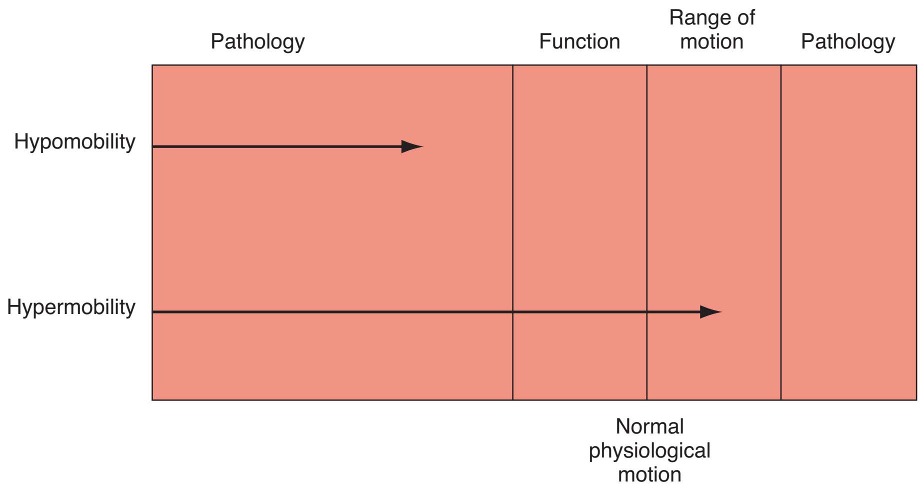


Figure 2 Hypermobility and hypomobility represent the extremes of available joint range of motion.

TABLE 2 ■ Normal and Abnormal End-Feels Assessed With Passive Range of Motion

NORMAL END-FEEL	DEFINITION	EXAMPLE	ABNORMAL END-FEEL	DEFINITION	EXAMPLE
Hard (bone to bone)	Hard bony sensation at the end of expected range	Elbow extension	Hard (bone to bone)	Hard bony sensation before the end of expected range	Restriction because of osteophyte formation
Soft tissue approximation	Slightly yielding compression, which stops further movement at end of expected range	Elbow flexion in well-developed muscular individual	Spasm	Abrupt end to movement either early or late in the expected range	Acute inflammation
Firm, tissue stretch	Springy or elastic resistance felt at the end of expected range	Ankle dorsiflexion	Springy block	Springy or elastic resistance felt before the end of expected range in joints with fibrocartilage	Meniscal tear
			Soft capsular or boggy	Springy or elastic resistance felt before the end of expected range and increasing with increasing movement	Synovitis
			Hard Capsular	Abrupt, springy or elastic resistance felt before the end of expected range	Chronic conditions, adhesive capsulitis
			Empty	No sensation of end feel; motion may be stopped because of pain	Tumor, acute bursitis

TABLE 3 ■ Normal Range of Motion and Capsular Patterns

JOINT/MOTION	AAOS* ROM VALUES	CAPSULAR PATTERN OF LIMITATION
Shoulder		ER>Abd/Flex>IR
Flexion	180	
Extension	60	
Abduction	180	
Adduction	0	
Internal Rotation	70	
External Rotation	90	
Elbow		Flexion > Extension
Flexion	150	
Extension	0	
Proximal Radioulnar		Pronation = Supination
Pronation	80	
Supination	80	
Distal Radioulnar		
Pronation	80	
Supination	80	
Wrist		Equal limitation in all directions
Flexion	80	
Extension	70	
Radial Deviation	20	
Ulnar Deviation	30	
Carpometacarpal		Abduction > Extension
Flexion	15	
Radial Abduction (Extension)	20	
Palmar Adduction	–	
Palmar Abduction	70	
MCP		Flexion > Extension
Flexion	90	
Extension	45 hyperextension	
PIP	80	Flexion > Extension
Flexion	100	
Extension	0	
DIP		Flexion > Extension
Flexion	90	
Extension	10 hyperextension	
Hip		IR/ER > Abd > Flexion > Extension > Adduction
Flexion	120	
Extension	30	
Abduction	45	
Adduction	30	
Internal Rotation	45	
External Rotation	45	
Knee		Flexion > Extension
Flexion	135	
Extension	0	

TABLE 3 ■ Normal Range of Motion and Capsular Patterns—cont'd

JOINT/MOTION	AAOS* ROM VALUES	CAPSULAR PATTERN OF LIMITATION
Talocrural		Plantarflexion > Dorsiflexion
Plantarflexion	50	
Dorsiflexion	20	
Subtalar		Supination > Pronation
Inversion	5	(Inversion and eversion are frontal plane
Eversion	5	components of pronation and supination)
MTP		Extension > Flexion 1st MTP
Flexion	45	Flexion > Extension 2nd – 5th MTP
Extension	70	

From Van Ost L: Goniometry: An interactive tutorial, Slack Inc, Thorofare, NJ, 2000; and Cyriax JH, Cyriax PJ: *Cyriax's illustrated manual of orthopedic medicine,* ed 2, Butterworth/Heinemann, 1993.
*American Academy of Orthopedic Surgeons: Joint motion: Method of measuring and recording, AAOS, Chicago, 1965.

Magee for a more complete listing of joint capsular patterns.[5]

BOX 5 STOP *and* THINK

What structures can limit joint ROM besides the capsule?

Role of Loss of ROM and Flexibility in Function

Loss of ROM may or may not be associated with loss of function. Several researchers have studied the amount of joint motion necessary for activities of daily living.[6-8] Therefore it is possible to glean a general idea of loss of function from specific loss of motion. However, it is difficult to predict the exact limitation in function caused by loss of joint motion because compensatory motions at the joints above and below the limited joint can allow the person to accomplish a desired activity in spite of the loss of joint motion. For example, excessive shoulder external rotation can be used to substitute for limited radioulnar supination.

When limitation of joint motion is because of soft tissue shortening, stretching exercises have been found to be effective.[9-13] The underlying premise behind the use of stretching is that longer muscles will have the ability to undergo a greater excursion and therefore provide for increased joint function. The muscle tissue includes both contractile muscle fibers and the noncontractile muscle components, such as the connective tissue surrounding muscle fibers.[14] Both these components provide resistance to muscle stretch.

A muscle that crosses only one joint is arranged so that it cannot be excessively lengthened or shortened relative to its optimal length for generating tension. Excursion of a single joint muscle is limited by the joint motion that is available. However, a muscle that crosses two or more joints may undergo a greater change in length during movement over the full ROM at all joints crossed by the muscle. Most multijoint muscles normally function in their midrange of available excursion where ideal length-tension relationships exist. However, the excursion range for multijoint muscles is greater than the limits of any single joint that they cross.[15] Stretching exercises are most often prescribed for multijoint muscles, which is the muscle group most vulnerable to the effects of adaptive shortening.[4]

PATIENT VIGNETTE 1

Scleroderma

Vignette provided by Chris L Wells, PhD, PT, CCS, ATC

Vicky Alexia is a 42-year-old woman who was brought to the emergency department by ambulance. Her neighbor found her unresponsive in her apartment. She was diagnosed with sepsis from a severe urinary tract infection and admitted to the intensive care unit.

PMH: Vicky was diagnosed with scleroderma 5 years ago and currently suffers from several of the associated complications of scleroderma, including multiple ulcers on her fingers and toes from Raynaud's syndrome, severe gastric esophageal reflux, and pulmonary fibrosis.

SOCIAL: She lives alone in a one-bedroom apartment. She is employed as a medical transcriptionist within her home and self-employed as a painter. She is fully independent at this time, but has a neighbor who will intermittently help with shopping and cleaning.

A therapy consult was written for ROM and positioning. Upon initial evaluation, the patient was sedated. A mechanical ventilator supported her breathing and she was receiving medications to support her heart function, maintain her level of sedation, and antibiotics. She was being fed through a nasogastric feeding tube.

Scleroderma or systemic sclerosis is a rare disease that affects multisystems and is characterized by intense fibrosis.[16,17] Scleroderma is characterized by tightness and thickening of the skin, digital pitting, pulp loss, sclerodactyly, and pulmonary fibrosis. The cause of this disease is unknown, but there are several theories. It has been suggested that there is a hormonal influence to this disease because there is an increased onset during pregnancy. It is possible there is a genetic link but this theory has little evidence. It has been documented that there is an increased incidence of scleroderma in males who have been exposed to silica, coal dust toxic oil, and organic solvents.[17,18]

Scleroderma can affect multiple organs. Interstitial pulmonary fibrosis occurs in 74% of the cases of scleroderma. The inflammatory process activates alveolar macrophages that stimulate fibroblastic activity and collagen formation.[16,19] With the presence of capillary destruction, there is a risk for the development of pulmonary hypertension. Up to 65% of the diagnosed cases of scleroderma will have pulmonary hypertension. The skin becomes thick and tight, which typically leads to joint contractures, and Raynaud's syndrome may lead to digital amputation caused by vascular insufficiency. Patients can also have difficulty with secretory glands that reduce saliva and tears, and sexual dysfunction. Anorexia, malnutrition, diarrhea, reflux, and osteoporosis are associated with the involvement of the gastrointestinal system. Renal dysfunction and failure is associated with vascular insufficiency because of fibrosis. Finally a small percentage of patients may present with cardiac involvement: pericarditis, pericardial effusion, arrhythmias, and on rare occasions left ventricular heart failure.[18]

The prognosis of patients with pulmonary involvement secondary to scleroderma is very dismal. If the diffusion capacity is less than 40%, there is a 95% mortality rate at 5 years opposed to

75% if diffusion capacity is greater than 40%. With the progressive pulmonary hypertension and right heart failure, survival rates are even smaller.[16,20]

PT Evaluation: Passive range of motion assessment revealed a restriction in bilateral shoulder flexion (up to 100°) and abduction (up to 95°). The therapist was unable to completely achieve full finger extension with a 20° degree loss of extension at the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints. The therapist also reported a 10° hip flexion contracture and a 5° plantarflexion contracture. Lower trunk rotation was met with soft tissue resistance after 40° of rotation of the lower extremities and pelvis. The end-feel of the restricted joints was firm, except for PIP and DIP joints, which were firm to hard end-feel. During the assessment, the patient's vitals were stable with heart rate averaging 125 beats/min, blood pressure averaging 100/50s, and saturation greater than 93%.

Therapy Orders: Therapy to be scheduled three to five times a week for ROM, stretching, and family training. Therapy will progress functional training once medically appropriate.

Treatment included the following:

PROM was conducted in multiplane directions for all extremities and lower trunk rotation for a set of 20 repetitions. Vicky's family was instructed in a general PROM program and was told to ask the bedside nurse for clearance to begin the exercises for safety reasons.

The therapist performed grade II and III joint mobilization for bilateral ankles, shoulders, and hands to increase dorsiflexion, shoulder elevation, and finger extension, respectively.

Vicky was positioned in supine and a slow, prolonged stretch was applied for shoulder flexion and abduction, knee extension, dorsiflexion, and lower trunk rotation. From a sidelying position, prolonged stretch was applied to stretch hip flexors and shoulder horizontal abduction, and upper and lower trunk rotation. Each stretch was held for at least 1 minute and was repeated for five repetitions.

As Vicky's medical condition improved, her sedation medications were discontinued and she began weaning from the mechanical ventilator. Range of motion was progressed to AAROM and AROM based upon her heart rate, blood pressure, oxygen saturation, and subjective reports. Stretching and joint mobilization were continued to improve available ROM of shoulders, hips, and ankles. Mobility of the trunk was progressed in prone and sidelying positions over pillows to assist in decreasing the work of breathing. Finally, proprioceptive neuromuscular facilitation techniques were introduced to enhance joint and soft tissue mobility, and active ROM.

Finally, functional mobility and cardiovascular and muscular endurance training began to incorporate the use of increased ROM and to begin the preparation for discharge. At the time of discharge from the acute care center, active ROM of shoulder flexion was up to 150° and abduction was up to 130°. Vicky was able to extend her metacarpophalangeal and PIP joints to a neutral position, but the DIP joints remained at 10° flexor contractures. Active hip extension was up to 10° and dorsiflexion was in a neutral position. Vicky was able to lie prone on her elbows with her pelvis in contact with the floor and to laterally flex 4½ inches bilaterally. Rotation was restored within normal limits (WNL). Vicky was discharged to an acute rehabilitation facility to complete her rehabilitation with a goal of restoring independence. She returned home after 12 days. Her friend and primary caregiver was instructed in a ROM program, with the goal to maintain the ROM that Vicky had achieved in therapy. She returned to work and painting at 6 weeks after admission and began a cycling exercise program 3 days a week.

BOX 6 STOP *and* THINK

Why should the rectus femoris muscle be stretched in prone instead of standing? Hint: Which position is best for stabilizing the pelvis?

SCIENTIFIC BASIS OF STRETCH

Mechanical Response

The mechanical response of musculotendinous tissue, ligaments, and capsule under load is well known. That is, the deformation of the tissue behaves in a time-dependent manner until reaching a steady state. This is known as "**creep**" and can be explained by the viscoelastic properties of the tissue. Viscoelastic tissues behave both like a fluid and a solid. That means that its behavior with loading is time and rate dependent [21] (Figures 3 and 4). Connective tissue elongation is dependent upon the amount of interweaving of the meshwork of collagen fibers. The greater the amount of interweaving, the less the connective tissue can elongate. Slow, low-intensity stretches can separate adjacent collagen fiber attachments within the meshwork and hypothetically allow for long-lasting or plastic elongation.[9,13,22] Once the load is removed, the lasting effects of stretching are not fully elucidated and may be explained by neurophysiological mechanisms and perhaps molecular mechanisms within the musculotendinous units.[14]

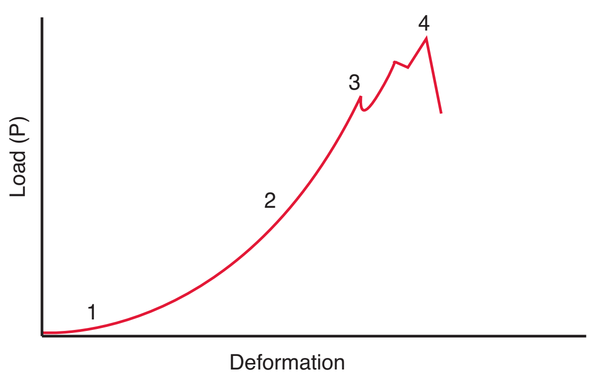


Figure 3 Load-deformation curve for tendon. *1,* "toe region" in which tissue elongation occurs with a small increase in load as wavy collagen fibers straighten; *2,* "linear region," collagen fibers are straightened and stiffness increases rapidly; *3,* end of the linear region in which progressive failure of collagen fibers begins; *4,* ultimate yield point, representing the ultimate tensile strength of the tissue. (Modified from Nordin M, Frankel V, editors: Basic biomechanics of the musculoskeletal system, ed 2, Philadelphia, Lea & Febiger, 1989.)

Joint Receptors

Sensory receptors found in the joint capsule, muscle, ligaments and skin provide input to the central nervous system regarding tissue deformation. Passive stretch maneuvers affect afferent output to the central nervous system via stimulation of the Ruffini's endings of the joint capsule and, to a lesser extent, Ruffini's endings of ligaments and Golgi's endings also found in ligaments surrounding the joint. Ruffini afferents have low mechanical thresholds and are slowly adapting. They respond to tension and provide a signal that the joint is near the limit of motion. They are also present in the skin. Golgi's endings have a high threshold and monitor tension. **Pacinian corpuscles** are another type of joint receptor with low thresholds, and they function to detect acceleration. They also exist in the skin. Finally, free

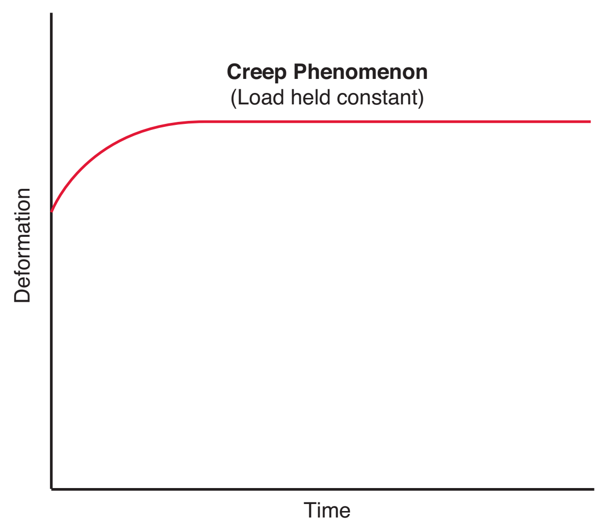


Figure 4 The creep response occurs when loading of ligaments or tendons is halted below the "linear region" of the load. Deformation curve and the load is held constant for a period of time. The deformation of the tissue increases rapidly initially, then slows to a steady state. (Modified from Nordin M, Frankel V, editors: Basic biomechanics of the musculoskeletal system, ed 2, Philadelphia, Lea & Febiger, 1989.)

nerve endings are present in the tissue of the joint. These receptors respond to noxious stimuli, either from abnormal mechanical stress or chemical stress.[23,24] It is important to understand the location of these receptors, their function, and thresholds because they may help or hinder various ROM or stretching techniques.

Muscle Spindle

Passive stretch can also affect afferent output of the muscle spindle and Golgi tendon organ (GTO) to influence alpha motor neurons. The **muscle spindle** is a special receptor consisting of a group of small, unique muscle fibers with Type Ia and II afferent, and gamma and beta efferent neurons. The small muscle fibers within the spindle are known as **intrafusal fibers** and are given this name to distinguish them from the rest of the fibers making up the muscle, otherwise known as **extrafusal fibers**. The type Ia and II afferent endings respond to change in muscle length that occurs when the extrafusal fibers are stretched. Type Ia afferents are responsive to quick and tonic stretch, whereas Type II afferents monitor tonic stretch. Nerve impulses from the intrafusal afferents travel to the spinal cord to inform the central nervous system that stretch has occurred in the muscle. Nerve impulses then return to the extrafusal fibers through the (efferent) alpha motor neuron to initiate muscle contraction to resist the stretch. The other way the spindle provides information about changes in muscle length is through the intrafusal gamma or beta efferents. An action potential of the gamma or beta efferent neurons may be transmitted to the muscle spindle and cause the intrafusal fibers to contract, stretching the area where the Group Ia and II afferents are located. The sensitivity of the spindle can be changed by gamma or beta efferents to increase the response to changes in muscle length or to the rate of change in muscle length.[23-25]

Golgi Tendon Organ

Golgi tendon organs (GTO) are specialized receptors located in a series at the junction between the extrafusal muscle fibers and the tendon proper. The GTO responds to tendon tension either through prolonged muscle stretch or muscle contraction. The sensory afferents of the GTO are known as Type Ib afferent nerve endings. These Ib fibers are attached to the tendon and can override the input of the muscle and therefore inhibit active tension in the muscle (inhibit the alpha motor neuron) and permit muscle elongation[25] (Figure 3-5). Afferent fibers from the GTO have spinal cord connections that play a role in the coordination of spinal reflex actions. Ib afferent output can have an excitatory effect on muscle depending on the sum of all other neural inputs (Figure 6).

It was believed previously that the GTO functioned as a muscle protector by firing only with high tension. For example, a prolonged static stretch would theoretically stimulate the GTO, resulting in

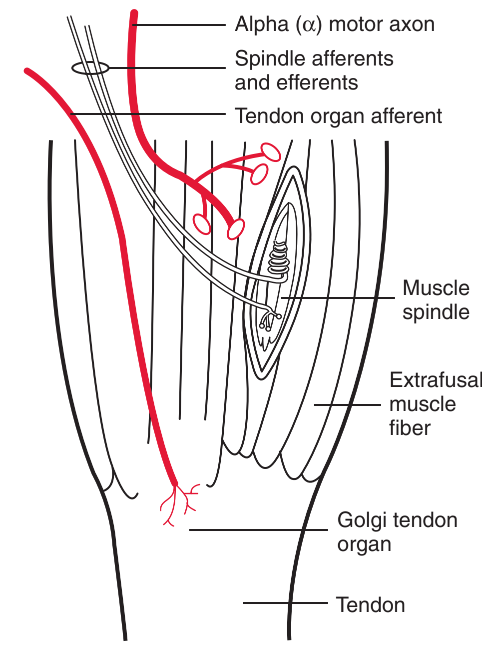


Figure 5 Muscle spindle and Golgi tendon organ. Fibers of the muscle spindle (intrafusal fibers) are activated by stretch. Type Ia and II sensory afferents of the spindle send information about stretch and the stretch velocity to the spinal cord. Nerve impulses then return to the muscle (extrafusal fibers) through the efferent alpha motor neurons that cause the muscle to contract and resist the stretch. The Golgi tendon organs exist in series as the junction between extrafusal muscle fibers and tendons. The sensory afferents of the GTO are known as Type Ib and are sensitive to changes in tendon tension that occur with either muscle stretch or contraction. (Redrawn from Kandel E, Schwartz J, and Jessell T, editors: Principles of neural sciences, ed 3, East Norwalk, Conn, McGraw-Hill, 1991, p. 565.)

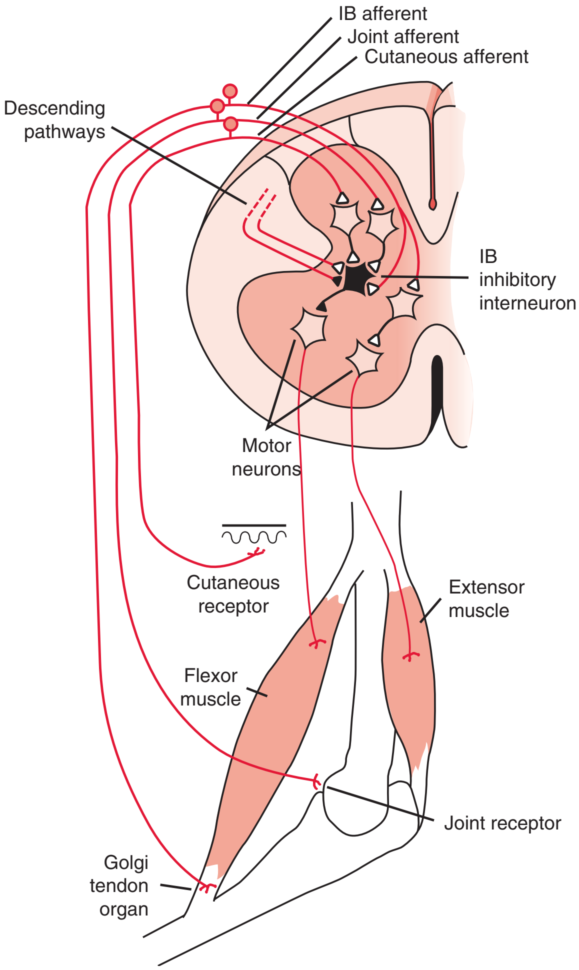


Figure 6 Reflex action of Ib afferent fibers from GTO. Ib inhibitory neurons receive input from GTO, spindles, joint cutaneous receptors, and descending pathways. (Modified from Kandel E, Schwartz J, and Jessell T, editors: Principles of neural sciences, ed 4, New York, McGraw-Hill, 2000.)

inhibition of the muscle being stretched. However, over the last decade or so, the GTO has been recognized as being able to detect very small changes in muscle tension, suggesting that these specialized receptors give the nervous system information about the state of the contraction of the muscle.[26] Afferent inputs from the Golgi tendon organs, cutaneous receptors, and joint receptors converge onto spinal cord interneurons that inhibit motor neurons. This inhibition of the motor neurons allows for control of muscle tension. It is easy to see how this precise control is important for activities, such as lifting and holding a hot cup of coffee. Their role in stretching to increase muscle length is not as clear.[27] Historical knowledge of the GTO may have oversimplified its role in muscle stretching. Further research is necessary to determine how the spinal reflexes respond to prolonged passive stretching.

BOX 7 STOP *and* THINK

Does the GTO function as a protective measure from excessive stretching at end range only?

PRINCIPLES OF ROM EXERCISES

Range of motion activities are used to maintain existing joint and soft tissue mobility, whereas stretching is used to treat limitations in range of motion that have occurred because of adaptive shortening of soft tissue. Of course, neither of these interventions should stand alone but rather in combination with other techniques, such as thermal modalities, relaxation or inhibition techniques, or joint mobilizations.[4,13] When a joint is completely immobilized following trauma, ROM should be maintained at the joints above and below the immobilized joint. ROM exercises should also be used during bedrest to prevent complications of deconditioning (cardiac and respiratory), bone demineralization, decreased circulation, and loss of joint motion. ROM exercises can be performed completely by an external force **(passive),** with the external force and the patient simultaneously **(active assistive),** or completely by the patient **(active)** (Table 4).

Passive ROM

Passive ROM (PROM) is used as a test and measure of the limitations of joint motion to assess tissue irritability, to determine joint stability, and to determine muscle and other soft tissue extensibility. Passive ROM exercises are indicated when the patient is not able to actively move the joint throughout its unrestricted or available range. Examples include weakness or paralysis limiting the ability of the person to voluntarily move through a full joint range or inability to follow instructions to do so. The external force may be provided manually through another individual or gravity, or mechanically through a machine. The clinician needs to consider other factors, such as the patient's position, that may influence the forces placed upon the joint or surrounding muscles. Surgical procedures, such as tendon repairs, often require PROM exercises to prevent excess tensile loads on the area of repair. Research has shown that PROM performed in the supine position results in the least amount of electromyographic activity by the muscles of the shoulder compared with AROM and resistive exercises.[28]

PROM exercises may also be used when active ROM is painful and therefore limited. The external force can be applied in a soothing, controlled fashion to prevent loss of joint motion and soft tissue shortening. However, it is important to note that PROM and passive stretching are not the same. PROM is used to maintain joint integrity and prevent contracture whereas passive stretching is used to treat an already present contracture. PROM exercises may precede passive stretching techniques as a means of relaxing the patient before passive stretching. PROM exercises may also be used to demonstrate movement to the patient before the progression to active ROM. Figure 7 shows an example of PROM using proprioceptive neuromuscular facilitation technique. Remember, the therapist should expose the body part the first time the patient does the exercise to ensure proper performance.

Controlled PROM through the use of a machine, known as **continuous passive motion** or CPM, has been shown to promote healing of cartilage, ligament, and tendon and to prevent joint stiffness following trauma or injury[29] (Figure 8). The use of controlled passive movement through CPM has grown to include treatment following total knee arthroplasty. However, its usefulness following arthroplasty is controversial.[29-34] Several investigators have reported delayed wound healing, a propensity for the development of a flexion contracture, and an extensor lag (less active extension compared with passive extension) associated with the use of CPM following knee arthroplasty.[35,36] Although early postoperative ROM may be improved with CPM, long-term ROM is not superior in patients who have used the CPM.[32-34]

TABLE 4 ■ Types of Range of Motion

TYPE OF RANGE OF MOTION EXERCISE	VOLITIONAL EFFORT OF PATIENT OR INDIVIDUAL	USE OF EXTERNAL FORCE BY CLINICIAN OR DEVICE
Passive	No	Yes
Active Assistive	Yes	Yes
Active	Yes	No

Figure 7 PROM can be complete in one plane or a combination of planes of motion.

Figure 8 PROM exercises applied through an external device known as continuous passive motion (CPM). The external force can be applied in a soothing, controlled fashion to prevent loss of joint motion and soft tissue shortening.

PATIENT VIGNETTE 2

Total Knee Replacement

Vignette provided by Chris L Wells, PhD, PT, CCS, ATC and Fran E. Huber, EdD, PT, OCS

HPI: Mrs. Goodwin is a 55-year-old woman who was involved in a motor vehicle accident 30 years ago. As she was driving, she was broadsided on her side of the vehicle, which caused her sport utility auto to roll several times before coming to rest upside down. Mrs. Goodwin was diagnosed with a right sacral fracture and right tibial plateau fracture. She was in acute rehabilitation for 3 weeks. She eventually regained independence for basic ADLs and returned to her job as a financial adviser 4 months after the injury. The baseline AROM for the right knee flexion was 10° to 100°. Mrs. Goodwin demonstrated a loss of heel strike and continued knee flexion throughout the stance phase and was unable to ascend or descend stairs in a reciprocal fashion. There was 15° genu varus in standing posture.

Over the years Mrs. Goodwin reported a progressive loss of function and ROM, and increasing pain. Consequently she chose to see an orthopedic surgeon for possible surgical intervention, at which time it was decided that she was a candidate for a total joint replacement. She elected to undergo surgery and was referred to physical therapy on post operative day one for gait training with PWB and ROM.

Total knee arthroplasties (TKAs) are surgical approaches used primarily to alleviate pain, correct deformity, and improve function in patients with knee arthritis or arthrosis that has not responded to conservative treatment.[37,38] TKAs are not the only option for these patients; other surgical and conservative options include: activity modification, antiinflammatory medications, intraarticular corticosteroid injections, modifications to footwear, arthroscopic knee debridements, osteochondral allografts, and osteotomy.[37] In the middle-aged patient, as in this scenario, the main concern is to extend the lifetime of the natural knee joint as long as possible. However, procedures with limited chance for success or that may negatively affect the outcome of a future TKA are not recommended.[38] Middle-aged patients were not previously considered candidates for TKA, but as surgical outcomes improved, the indications were expanded.[38] Minimally invasive techniques have gained favor in TKA, including unicompartmental knee replacement, with varying reports of clinical outcome.[37] Over the years, long-term outcomes for middle-aged patients post-TKA have improved, especially with cemented posterior stabilized prostheses.[38] Complications that follow total knee arthroplasties and that are of particular concern to the therapist include extensor mechanism problems (e.g., patellar tendon ruptures and extensor lag), periprosthetic femoral fracture, infection, and stiffness.[37]

PMH: Mrs. Goodwin is a 55-year-old woman who is also being treated for hypertension and hyperlipidemia, BMI 41, and smokes 1 pack per day.

SOCIAL: The patient lives with her husband and youngest son in a multilevel home with the bedroom and bath on second floor and powder room on first.

PT Evaluation: Observation: right knee is ace wrapped and there is a hemovac drain. Her resting vitals were heart rate 103 beats/min and blood pressure was 156/80. Neurological examination is grossly intact. ROM and strength of uninvolved extremities is WNL; Right knee AAROM (Ext - flex) 18° to 47° and reports 6/10 pain with movement. Right ankle dorsiflexed to neutral position. She required moderate assistance for bed mobility; minimal assistance for transfers and gait with a standard walker for 20 feet.

Problem List

1. Hypomobility of the knee joint
2. Disuse atrophy of the thigh and calf muscles
3. Altered ADL
4. Dependent with basic functional mobility
5. Pain with movement

Plan: Patient will be scheduled for therapy twice a day to improve general mobility, ROM, and strength of her right lower extremity.

Specific treatment techniques that were performed to improve mobility and function of right knee included starting on an AAROM program for knee flexion and extension within her pain tolerance. The therapist also began teaching the patient how to perform these AAROM exercises.

A continuous passive motion machine was set up in the patient's hospital room. Mrs. Goodwin was instructed in how to use the machine. She was advised to use the machine at 2 hours in the morning and before going to bed and 1 hour periods throughout the day to manage pain and stiffness. The machine was initially set at 15° to 50° of motion and she was instructed in increasing the motion by 2° to 5° degrees as tolerated. The CPM was initially started at 15° flexion to address the long term loss of extension preoperatively. The ROM was progressed aggressively to avoid the persistence of a flexion contracture. At the time of discharge from the hospital, Mrs. Goodwin's ROM for her right knee was (ext - flex) 13° to 67° degrees and dorsiflexion was up to 7°. She was independent with basic functional mobility and able to walk 75 feet with axillary crutches. She was able to ascend and descend stairs with close supervision.

Physical therapy was continued at an outpatient clinic 3 weeks after the operation to improve ROM and strength of the right lower extremity. Mrs. Goodwin's AROM at the time of the initial evaluation was (ext - flex) 10° to 75° and dorsiflexion of the ankle was actively to a neutral position. She was instructed in stretching of the Achilles tendon complex, with a strap in both a knee flexed and knee extended position. She was also instructed in patellofemoral joint mobilization in an inferior and superior direction. Once the surgical incision was completely healed, friction massage was taught to the patient.

Basic ROM exercises were progressed to include active motion with her sitting and standing. At the end of outpatient therapy, postoperative week 8, mini-squats were introduced. The therapist instructed her to begin these standing (closed kinetic) exercises by placing 10 to 20 lbs. onto her right lower extremity. The weight was increased as she displayed tolerance to the motion and there were no signs of exacerbation of her edema.

The therapist began proprioceptive neuromuscular facilitation exercises to further facilitate end range of motion. Contract-relax was used to facilitate knee extension and hold-relax was used to improve flexion. Finally, slow reversal facilitation exercises were completed at end of motion followed by full active ROM to promote full ROM.

Mrs. Goodwin was discharged from outpatient physical therapy after 6 sessions completed over 8 weeks. She was independent in a home exercise program, which included stretching of her quadriceps, hamstrings, and Achilles' tendon complex. She was advised to increase her walking until she could walk 30 minutes a day for general exercise. She was fully independent with ADLs and functional mobility, including transfers to and from the floor and reciprocal motion ascending and descending stairs. She used a straight cane when she went for extended walks, and walking on very uneven surfaces. Her AROM of the right knee is (ext - flex) 0° to 105°.

The patient case presented above provides the reader with several important educational points:

1. The importance of maintaining ROM of associated joints after a surgical procedure to prevent avoidable contractures from occurring.
2. Appropriate therapeutic exercise is critical in maintaining and improving ROM.
3. Appropriate management of a hypomobile joint can result in dramatic improvement in ROM.
4. Regaining end-range ROM is much more difficult than obtaining motion early within the range.
5. Successful treatment incorporates both self-exercise and manual therapy performed by a trained therapist.

Active Assistive and Active ROM

Active assistive ROM (AAROM) is used when a person can voluntarily contract the muscles to move a joint but needs the assistance of an external force to complete the motion. AAROM provides assistance to weak muscles for progression of strengthening and to ensure full joint motion. AAROM exercises can eventually be progressed to **Active ROM** (AROM) exercises, whereby the patient is completely responsible for the movement that occurs. Active ROM is also useful before the progression of an aerobic conditioning program to improve cardiovascular and respiratory responses in a slow and controlled fashion. This is particularly useful for the patient with a deconditioned state that results from prolonged bed rest. AAROM and AROM exercises accomplish the same goals of passive ROM with the addition of the therapeutic value of muscle contraction to aid in the prevention of muscle atrophy, increase circulation, prevent thrombus formation, and to initiate the coordination and skills necessary for functional activities. Increasing repetitions and sets of AAROM and AROM can be used to progress strength and endurance of weakened muscles. However, AAROM and AROM cannot maintain or increase strength in muscles that grade fair or better. It is important to note that coordination or motor skills will not be developed except in the movement patterns in use[4] (Figure 9).

Figure 9 A patient can use a straight cane or uninvolved extremity to assist the ROM of the involved extremity.

Each type of ROM (passive, active assistive, and active) can be performed in straight cardinal planes of flexion, extension; abduction, adduction; internal rotation, external rotation; or with combinations of movement planes. The combinations of movement typically employ **proprioceptive neuromuscular facilitation** (PNF) diagonal patterns. These are used to attempt to simulate "normal" movement and to increase efficiency of the treatment.[39] In other words, two or more planes of movement are combined in using a PNF diagonal pattern and this approach may shorten treatment duration.

BOX 8 STOP *and* THINK

Which motion is harder to gain, early motion within the range or end-range motion? Why?

Precautions and Contraindications to ROM Exercises

Symptoms and signs from too much motion or force used during ROM exercises may include increased pain, increased warmth, increased swelling (effusion), and redness. Pain and swelling are perhaps the best indicators of how hard to push ROM. The healing state of tissue is paramount in making a clinical judgment about whether or not to use passive or active ROM. Tissues violated during surgery need to be protected. Active or passive tension on repaired tissue should be minimized during early to mid recovery. For example, when an open rotator cuff repair is performed and the subscapularis is incised and repaired, no passive external rotation or active internal rotation of the glenohumeral joint should be used immediately following surgery. The mechanism of injury during trauma also dictates what ROM activities are contraindicated immediately following the incident. For example, passive or active ROM in the directions of joint dislocation should be avoided until the soft tissues have had adequate time to begin the process of repair and therefore promote the return of joint stability. Proper progression of ROM activities according to phases of tissue healing is discussed in the next section.

Precautions need to be taken when completing AAROM and AROM exercises for patients who are critically ill. If the patient is suffering from uncontrolled heart failure or angina at rest, then even AAROM may be contraindicated. This is also true for the patient whose body is producing excessive amounts of lactic acid, either because of severe respiratory failure or a systemic infection. Vitals should be monitored closely when treating these patients and it is advised to begin with AAROM and progress intervention as the medical condition improves (Table 5).

PRINCIPLES OF STRETCHING

Loss of joint ROM because of adaptive shortening of soft tissue (contracture) may be treated with stretching. Prolonged immobilization, connective tissue diseases, arthritides, trauma, surgery, inflammation, hemorrhage, and burns can lead to the production of dense fibrous tissue. This dense fibrous tissue replaces healthy soft tissues, including muscle, tendon, joint capsule, and skin and therefore causes loss of normal joint ROM. Muscle strength can also be affected by the subsequent loss of joint motion because it disrupts the normal muscle length tension relationships. However, it is important to note that muscle tightness can be present without a joint contracture. Multijoint muscles are particularly vulnerable to developing tightness, especially in those individuals who do not regularly perform stretching exercises.

BOX 9 STOP *and* THINK

Why are multijoint muscles more vulnerable to developing tightness?

Static Stretching

Stretching may be applied manually to a segment or joint as a passive maneuver in which an external force is applied to lengthen shortened tissue while the patient is relaxed. This technique is often referred to as "**static**" stretching and is usually applied as a low-intensity stretch to allow for gentle elongation. Theoretically, there is less opportunity to cause injury with this approach because it allows the stretch response of the muscle spindle to subside, uses less force compared with other techniques, and allows for subject relaxation[40] (Figure 10). However, studies have shown a low level of muscle activity to exist by EMG monitoring during static stretch.[41,42]
Stretching may also be applied mechanically through an external force, such as traction and pulley systems, dynamic braces, or serial casting. These are often recommended when joint contractures exist.

An individual can perform passive stretching exercises without the assistance of another person or device. Although this seems like a contradiction in terms (i.e., passive vs. active), it really isn't. In other words, the external force attempting to lengthen shortened muscle or other soft tissue is still provided by the patient, but it is passive in the sense that the affected muscle is to remain in a relaxed state. Passive stretching exercises conducted independently by the patient are often referred to as "flexibility exercises."

How long to hold a static stretch to achieve maximal benefit and carryover has not been fully investigated. Most clinicians prescribe a short duration stretch to be held for at least 15 to 30 seconds, but it is not uncommon to manually hold a stretch position up to 2 minutes. The effectiveness of the stretch

TABLE 5 ■ Precautions and Contraindications for ROM Exercises

Pain
Acute local infection
Acute surgical procedure
Effusion
Joint instability
Medical instability

Figure 10 A slow, low intense static stretch will allow the patient to relax and result in elongation of the trunk musculature.

may be influenced by factors other than duration and frequency, with muscle type and the involvement of other soft tissue also needing to be considered when deciding how long to hold a stretch.[9,24]

Proprioceptive Neuromuscular Facilitation

Stretching through the use of **proprioceptive neuromuscular facilitation** (PNF) is theoretically based on the principle of stretch activation of the muscle spindle and GTO. PNF activities involve the activation of either agonist or antagonist muscles immediately preceding a stretch. These PNF techniques theoretically inhibit the muscle, either through activation of the GTO, known as **autogenic inhibition,** or by the Sherrington principle of **reciprocal inhibition** in which maximal activation of one muscle inhibits activation of another muscle–its antagonist.[43,44] Several studies have shown PNF techniques to be superior to either static or ballistic stretching, but it is not clear which PNF techniques are the most effective.[39,42,45] This is partly true because the literature is confusing regarding the terminology of PNF techniques and partly true because there is a paucity of randomized controlled studies. There is, however, EMG evidence to suggest that PNF stretching techniques cannot work solely on the principles of autogenic and reciprocal inhibition.[46]

Studies comparing the effectiveness of PNF stretching to static stretching are inconclusive at this time. Both stretching techniques have been shown to increase hamstring length compared with a control. However, a comprehensive and well-controlled investigation comparing the two techniques using the same stretching parameters is necessary. Sady and colleagues[42] compared ballistic, static, and PNF autogenic inhibition stretching techniques over a 6-week stretching program. They reported an increase in hamstring length with only the autogenic PNF stretching group. The static stretching group, however, only performed 6-second stretches. Based upon Moore's and Hutton's[47] investigation, 6-second stretching may not be adequate time to see a change in hamstring length using static stretching.

Sullivan and colleagues[48] compared static self-stretching with a PNF technique, which used both autogenic and reciprocal inhibition over a 2-week stretching program. They reported no difference in the two stretching groups. Osternig et al[45] compared static, PNF reciprocal inhibition, and PNF autogenic inhibition techniques during one testing session. They reported greater mean increases in acute hamstring length with the PNF techniques compared with the static stretching group; however, they did not perform any statistical analysis to determine if actual differences exist among the treatment groups.[45]

Ballistic Stretching

Ballistic stretching involves quick, repetitive, bouncing movements. For the majority of patients, this type of stretching is not recommended because of potential for soft tissue injury. Bandy notes that ballistic stretching has fallen out of favor among clinicians for this reason and also because of activation of the afferent nerve fibers of the muscle spindle.[49] Afferent activation of the muscle spindle in this case may be counterproductive because it causes contraction of the muscle simultaneously as it is stretched. Malone on the other hand argues that a ballistic stretching program is indicated for the athlete who is accustomed to high-velocity movements.[50]

A good examination with appropriate tests and measures will help the treating clinician discern the nature of the limitation. Both the joint and its soft tissues may need to be addressed. If the problem is loss of joint accessory motions, joint mobilizations are indicated. Commonly, however, both problems concomitantly exist so that ROM, joint mobilizations, and stretching may be indicated.

Precautions and Contraindications for Stretching

There are numerous precautions for stretching. First and foremost, one should remember that range of motion varies among healthy individuals and a joint should never be stretched beyond its "normal" range. This "overstretching" may result in hypermobility.

Since a period of prolonged immobilization will cause connective tissues to lose tensile strength, stretching should proceed with caution.[41,51] If a person experiences joint or muscle soreness or swelling lasting more than 24 hours, then it can be concluded that the exercise was completed with too much force. Similarly, a newly united fracture should be protected.[4] It is important to protect the fracture site by holding the proximal segment, supporting the fracture site, and avoiding rotation during the stretch.

Stretching should not be used immediately following a fracture near a joint. Once the fracture has united, stretching should proceed with caution as described in the previous paragraph. The fracture site should be protected from high-intensity stretching exercises and the area between the fracture site and the joint must be stabilized. If there is a bony block to the joint, no stretching should be performed.

Bone also needs to be protected if osteoporosis is suspected or known. Such is the case with prolonged use of steroids, bed rest, immobilization, and aging.[52] Aggressive stretching should be avoided; long lever arms should not be used to apply the force; and rotation should be avoided (Figure 11).

If acute inflammation exists, no stretching should be performed. Edematous tissue should not be stretched as stretching can be a source of continuous irritation to the tissue and may result in the persistence of edema. Similarly, if an infectious process is suspected, no stretching should be used.

Ballistic stretching should not be used in the nonathlete.[50] However, even in the athlete, ballistic stretching should be reserved for the athlete who is highly conditioned and should not be used during early season training or in the injured athlete because of the potential of reinjury.[50]

Caution should be used in stretching following acute traumatic brain injury and acute blunt trauma that results in a hematoma, especially over long bones, such as the mid humerus or mid femur. These acute traumatic conditions expose the patient to the development of myositis ossificans.[52] Early treatment of myositis ossificans requires rest until the pain and inflammation subside, followed by PROM and then AROM. Activity is advanced when the bone stabilizes in size and appearance as shown by radiography. Several months should pass before progression to aggressive stretching because myositis ossificans can be made worse with passive stretch at high force.[53,54]

Figure 11 Care must be taken to stretch without compressing the anterior vertebral body.

Contractures caused by fibrosis of soft tissue can be refractory to conservative attempts of stretching. High-intensity, vigorous stretching may cause tissue trauma and therefore act to worsen joint contractures. Surgical release may be necessary for periarticular musculotendinous tissue. Manipulation under anesthesia following total knee arthroplasty may be recommended before the formation of excessive scar tissue replaces dead or damaged tissue during the repair phase of healing.[55]

Sometimes contractures aid in functional abilities and should not be stretched. Such is the case in persons with quadriplegia who use tenodesis of the long finger flexors and wrist extensors to grasp objects with their hands (Table 6).

As stated previously, neither of these treatments (ROM, stretching) should be used in isolation. This is particularly true of stretching exercises. Strengthening should be included with a stretching program at some point so that a balance can be achieved between strength and flexibility. The reader should once again keep in mind that it may be necessary to include joint mobilizations for the person with a joint contracture because accessory motions may also need to be restored for return of physiological joint ROM.

TABLE 6 ■ Precautions and Contraindications for Stretching

Age of subject
Fracture and newly healing fracture
Osteoporosis and other bone diseases
Acute local inflammation
Hematoma
Myositis ossificans and hypertrophic ossification
Integrity of joint
Hypermobility
Contractures that promote functional mobility

TECHNIQUES FOR ROM EXERCISES

Preparation

The examination and evaluation of the patient will determine the application of passive, active assistive, or active ROM. Regardless of the type of ROM exercises used, several principles remain unchanged. First, the patient should be positioned as comfortably as possible with the joint and segments treated unclothed and free to move. This may require proper draping to protect patient modesty. Second, the treating clinician should position him or herself using proper body mechanics. Third, the treating clinician's hand placement should be as near the joint as possible to allow for control of the joint and its adjacent body segments. If the joint is painful or there is an unstable segment (e.g., recent fracture), the grip may need to be modified. Additional support is necessary if there is a flail limb because of severe sensorimotor impairment.

Movements Used

Motions can be performed in cardinal planes, a combination of planes using combined patterns of movement following proprioceptive neuromuscular patterns, or within a muscle's range of elongation. The motion should not be forced beyond the available range. All motions should be smooth and rhythmical. There is no hard and fast rule for the number of repetitions. Kisner and Colby recommend 5 to 10 repetitions, depending on the "...objectives of the program, and the patient's condition and response to the treatment."[4]

If AAROM is used, the treating clinician should demonstrate the motion desired using PROM first. If PROM is contraindicated, the clinician can demonstrate the desired motion on the uninvolved extremity (Figure 12, *A-C*). A firm hand placement should be maintained to assist or guide the patient through the movement. Assistance throughout the entire available ROM may not be necessary. That is, weakness may not exist throughout the entire ROM and the patient may only require assistance at the beginning and end ranges of motion. When the healing state of the tissue is advanced enough to allow AROM, exercises should be progressed to prevent further loss of function.

ROM Progression

The patient's response to ROM exercises should be monitored during and after the treatment intervention. Pain, quality of movement, and changes should be noted in ROM. Vital signs should be monitored in persons with known cardiorespiratory disease or with deconditioning because of bed rest. As with any treatment intervention, the treatment should be changed or progressed according to the patient's response to treatment.

BOX 10 STOP *and* THINK

How does a therapist decide when to use straight cardinal planes for ROM versus PNF diagonal patterns?

STRETCHING TECHNIQUES

The type of stretching performed will be determined partly by the examination and evaluation and partly by the preference of the treating clinician. For example, autogenic inhibition (neuromuscular) techniques cannot be applied to muscle that is denervated or severely weakened.

Static Stretching

Passive stretching alone (without the use of volitional contraction by the patient) may be applied manually or mechanically (Figure 13, *A,B*). The technique of manual passive stretching involves a low-load stretch, usually lasting from 15 to 30 seconds, and several repetitions during a treatment session. A slow and steady stretch is imperative to prevent a monosynaptic stretch reflex from occurring that would increase tension in the tight muscle (Figure 14). Both contractile and noncontractile tissues are elongated, attempting to move the joint beyond the available range. Lengthening the duration of the hold does not appear to improve results of low-load passive stretching in young adults.[9] However, Feland and colleagues found that a duration of 60 seconds was more effective at increasing knee ROM (hamstring stretch) in adults 65 years or older.[24] Contractures require passive stretch of a longer duration. This type of passive stretching requires a mechanical device to

Figure 12 **A,** Passive ROM of the shoulder performed by the therapist. The motion should not be forced beyond the available range. **B,** Passive ROM can be advanced to active assistive ROM. Hand placement should still be maintained to assist or guide the patient through the movement. Assistance throughout the entire available ROM may not be necessary. **C,** ROM can be performed in cardinal planes as shown in A and B, or combination of planes using combined patterns of movement following proprioceptive neuromuscular patterns or within a muscle's range of elongation.

apply the external force, such as traction and pulley systems or dynamic braces. If traction is used, the external load is typically between 5 and 15 lb.[4] The duration of the stretch may be 20 to 30 minutes or for several hours.[22,46,56]

Neuromuscular Techniques

The underlying premise of neuromuscular techniques such as Proprioceptive Neuromuscular Facilitation (PNF) is that in order for a muscle to be elongated, it must be inhibited or relaxed. **Hold-relax** (also referred to as "**muscle energy**") requires the patient to perform an isometric contraction of the muscle to be elongated just before it is passively lengthened. It is postulated that the muscle will relax after contraction because of "autogenic inhibition" through firing of the Golgi tendon organ. **Contract-relax** applies the principle of "reciprocal inhibition" in which a concentric contraction of the muscle or muscle group

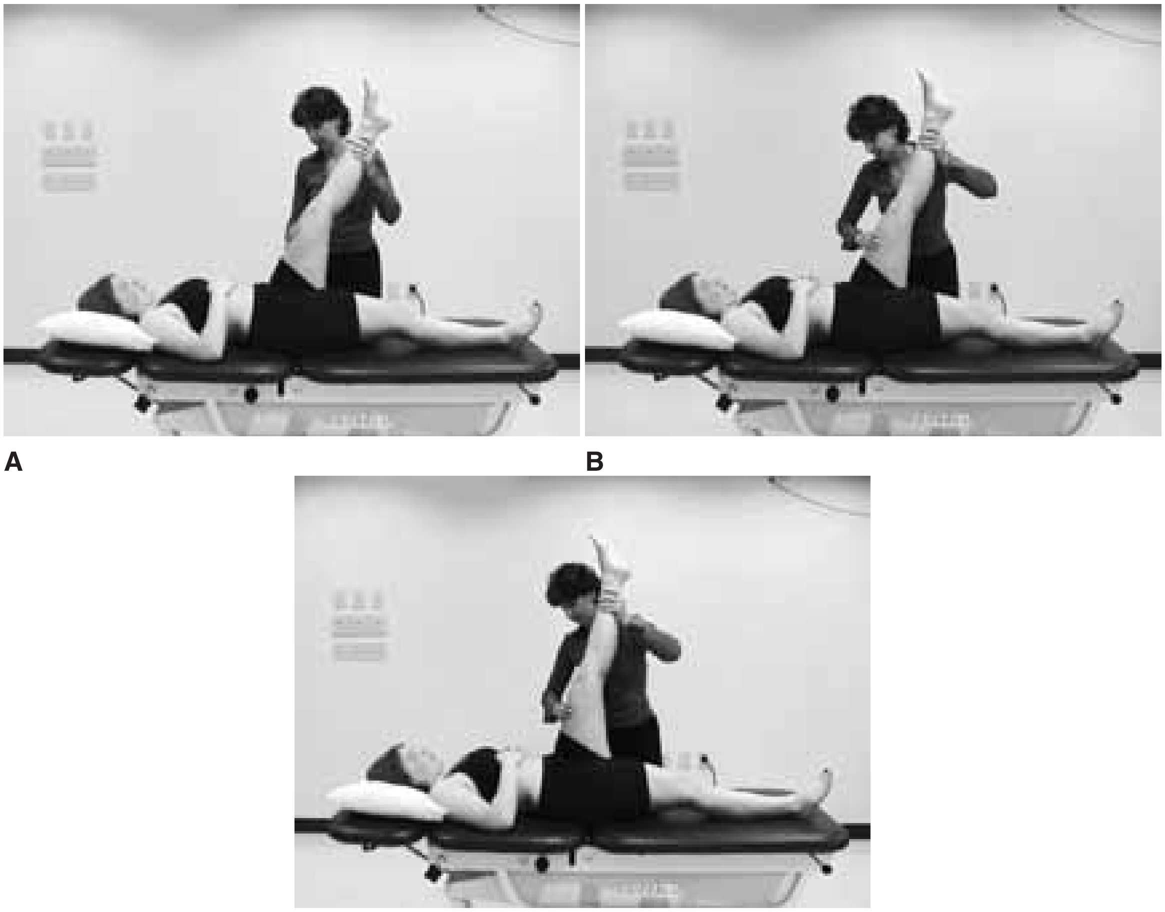
A B

Figure 13 Passive stretch alone (without the use of volitional contraction by the patient) may be applied manually **(A)** or mechanically **(B)**. **A,** Shows the technique of manual passive stretching to the hamstring muscle group. This technique involves a low-load stretch, usually lasting 15 to 30 seconds, and several repetitions during a treatment session. **B,** A low-load, prolonged stretch can be applied continuously through the use of a mechanical device. The device shown is known as an "extension board" and is used to facilitate knee extension.

Figure 14 Slow stretch to the Achilles complex is critical to prevent the recruitment of the muscle in the presence of spasticity.

located on one side of the joint causes inhibition or relaxation of the muscle or muscle group on the opposite side of the joint (Figure 15, *A-C*). Various combinations of the techniques can be applied and the reader is referred to the work of Voss and colleagues for more information about PNF.[39] The literature supports the use of PNF, but whether a specific technique is best for stretching is not known. The underlying mechanisms for their use are not fully clear either.[39,43,44] If stretching includes the use of PNF through hold-relax, then the patient should be asked to perform an isometric contraction of the tight muscle from a comfortably lengthened position. The contraction should be maximal and held for 5 to 10 seconds, or submaximal and held until the muscle starts to fatigue. Submaximal contraction may be preferred by the clinician to maintain better stabilization and control. The patient should then relax so that the segment and/or joint can be moved through the newly achieved range. The process should be repeated several more times after a brief rest period.

If stretching includes PNF techniques of **contract-relax-contract**, then the patient in this case, not the clinician, moves the segment and/or joint through the newly achieved range. The patient again is positioned with the muscle in a comfortably lengthened position and is asked to perform a concentric contraction of the muscle or muscles on the opposite side of the joint from the tight muscle (Figure 16). Resistance applied by the clinician should be minimal and allow motion (hence concentric rather than isometric). Excessive resistance in this application is



A B C

Figure 15 Hold-relax is a neuromuscular stretching technique that applies the principle of "autogenic inhibition," in which a concentric contraction of the muscle or muscle group located on one side of the joint causes inhibition or relaxation of the muscle or muscle group on the opposite side of the joint. The joint is passively moved to the end point in the range and then an isometric hold of the agonist is performed. The patient is then instructed to relax and passive movement into the new range of the agonist pattern follows. **A,** The patient's hip extensors (hamstrings) are stretched passively to end range. **B,** The patient is instructed to perform an isometric contraction of the hip extensors. **C,** The therapist then stretches the hip extensors into the new range.

counterproductive because it may cause contraction of the tight muscle and therefore hinder relaxation. It is important that the clinician remember to allow the segment and/or joint to move as the resistance is applied.

When the patient stretches independently, body weight can be used as the force providing the stretch. The patient should be instructed in body segment positions that also use gravity as a means to assist with the stretch (Figure 17). The patient can also be instructed in techniques using active inhibition where appropriate (Figure 18).

Before any stretching session, a brief warm-up period is recommended to promote tissue extensibility

Figure 16 Contract-relax: This technique allows for the increase in knee extension through the active contraction of quadriceps and the inhibition of the hamstrings.

Figure 17 The use of the patient's body weight can provide the external force to allow for an effective stretch.

and help prevent tissue injury. This warming of the tissues may be accomplished through low-intensity, active exercise (such as 5 minutes riding a stationary bike with low resistance and speed) or through the use of superficial or deep heat modalities.[13] The warmth of superficial modalities may provide the additional benefit of helping the patient relax before initiation of stretching. The patient should be advised that relaxation is paramount and relaxation procedures may need to be used before stretching. Massage may also be of benefit to increase circulation and aid in relaxation.

Figure 18 Self-stretch: The patient is shown stretching the levator scapulae muscle.

During treatment, the patient should always be positioned to promote comfort. Restraints to movement should be minimized. Clothing may need to be removed to uncover the area and appropriate draping applied. The body part should be stabilized and the joint should first be moved through the available range just to the point of the restriction. It may be necessary to then change hand placement to grasp the proximal and distal joint segments when possible. This grasp should be firm but not uncomfortable for the patient. Usually the proximal segment is stabilized and the distal segment moved. Devices such as mobilization straps or another person may be necessary to assist with stabilization while the treating clinician applies the stretch.

Gentle joint distraction (Grade I mobilization) should be used to avoid joint compression during the stretch. The joint should be taken to the point of limitation and then moved slightly beyond that point. The stretch should be gentle, slow, and sustained. No ballistic or bouncing stretches should be used because they are counterproductive. That is, bouncing will facilitate the monosynaptic stretch reflex and may also result in tissue damage. The patient should experience the tension or pulling of the tight structures but should not feel anything greater than mild discomfort. This again is counterproductive because it impedes relaxation and may result in tissue injury.

The stretched position should be held at least 15 to 30 seconds during which time tissue tension should ease and allow the segment to be moved further.

The stretch force should be released as slowly as it is applied. The clinician(s) and the patient should then rest briefly. This process should be repeated several times. It is unrealistic and potentially dangerous to expect to gain full range within one or two treatment sessions. It may take several weeks to obtain the desired results.[22] Bandy has shown that static stretching can produce a change in tissue length in as little as 2 to 4 weeks.[49]

Multijoint muscles should be stretched over one joint at a time to start and then progressed to all joints crossed when appropriate. Sometimes it is inappropriate to stretch over all joints crossed by the muscles. For example, the long finger flexors should never be stretched over the wrist, metacarpophalangeal, and interphalangeal joints simultaneously in someone with quadriplegia. The reason for this is that finger joint flexion should accompany wrist extension to promote tenodesis and provide assistance with grip during functional activities. Again, results of stretching should take several weeks and must be continued for maintenance of flexibility.

BOX 11 STOP *and* THINK

Do stretching exercises need to be continued once the desired goal has been achieved?

Ballistic Stretching

Ballistic stretching consists of repetitive bouncing movements performed by the individual only. The force during a ballistic stretch is not produced by a therapist or outside force. Ballistic stretch should be reserved for the athlete and even then, its use is debatable.[50] The technique of ballistic stretching should be preceded with a brief warm-up as described in the previous section, followed by static stretching. The individual then begins bouncing movements using muscles considered to be antagonistic to the muscle(s) being stretched. One should not get the idea that ballistic stretching involves bouncing that is not controlled. It is very important for the individual using this technique to understand that motion and force, while rapidly occurring, is controlled. Otherwise, this type of stretching is counterproductive because of neurophysiological inhibitory influences and has the potential to be harmful to tissue. Malone proposes a means of ballistic stretching in which the velocity of stretch and range of stretch are progressively increased. The reader is referred to his work for additional information.[50]

SUMMARY

This lesson presented the scientific bases for ROM and stretching exercises used to maintain and restore joint motion. Case examples are consistent with those presented elsewhere in this text but with discussion germane to the topics of ROM and stretching only. The reader is cautioned that ROM and stretching may need to be used in combination with manual therapy techniques (joint mobilizations) to restore accessory movements.

STUDY QUESTIONS

1. Explain the difference between physiological and accessory joint motions. What are the clinical terms used to describe each of these?
2. Why are multijoint muscles more prone to adaptive shortening?
3. What is the difference between active and passive insufficiency? Give examples of each.
4. What examination findings suggest that loss of ROM is because of a bony block?
5. What are the indications for PROM exercises and AROM exercises?
6. What duration of time should be used to perform a static stretch of the ankle plantarflexors?
7. Explain how ballistic stretching can be performed in a controlled fashion.
8. Explain the technique of stretching near an area of a newly united fracture.
9. Explain why AROM exercises cannot increase strength in muscles with a good or normal grade.
10. Explain why neuromuscular techniques, such as PNF, may not work solely on the principles of autogenic inhibition and reciprocal innervation.

REFERENCES

1. Norkin C, White D: Basic concepts. In Norkin C, White D, editors: Measurement of joint motion: A guide to goniometry, ed 2, Philadelphia, FA Davis, 1995.
2. Lee M, Gal J, Herzog W: Biomechanics of manual therapy. In Dvir Z, editor: Clinical Biomechanics, Philadelphia, Churchill Livingstone, 2000.
3. APTA. Guide to physical therapist practice: What types of test and measures do physical therapists do, Phys Ther 2001; 81(1):S84-S92.
4. Kisner C, Colby L: Stretching. In Kisner C, Colby L, editors. Therapeutic exercise: Foundations and techniques, ed 2, Philadelphia, FA Davis, 1990.
5. Magee D: Orthopedic physical assessment, ed 3, Philadelphia, WB Saunders, 1997.
6. Morrey B, Askew L, An K: A biomechanical study of normal elbow motion, J Bone Joint Surg 1981; 63A:872.
7. Laubenthal K, Smidt G, Kettlekamp D: A quantitative analysis of knee motion during activities of daily living, Phys Ther 1972; 52:34.
8. Johnson R, Smidt G: Hip motion measurements for selected activities of daily living. Clin Orthop 1970; 72:205.
9. Bandy W, Irion J, Briggler M: The effect of time and frequency of static stretching on flexibility of the hamstring muscles, Phys Ther 1997; 77:1090.
10. Bonutti P, Windau J, Ables B: Static progressive stretch to reestablish elbow range of motion, Clin Orthop 1994;6:128.
11. Steffan T, Mollinger L: Low load prolonged stretch in the treatment of knee flexion contractures in nursing home residents: Phys Ther 1995; 75:886.
12. Worrell T, Smith T, Winegardner J: Effect of hamstring stretching on hamstring muscle performance, J Orthop Sport Phys 1994; 20:154.
13. Lentell G, Hetherington T, Eagan J et al: The use of thermal agents to influence the effectiveness of a low load prolonged stretch, J Orthop Sport Phys 1992; 16(5):200.
14. DeDeyne P: Application of passive stretch and its implications for muscle fibers, Phys Ther 2001; 81:819.
15. Chleboun G: Muscle structure and function. In Levangie P, Norkin C, editors: Joint structure and function: A comprehensive analysis, ed 3, Philadelphia, FA Davis, 2001.
16. Minai O, Dweik R, Arroliga A: Manifestations of scleroderma pulmonary disease, Clin Chest Med 1998; 19(4):713-727.
17. Silman A: Epidemiology of scleroderma, Ann Rheum Dis 1991; 50:846-853.
18. Steen V: Clinical manifestations of systemic sclerosis, Semin Cutan Med Surg 1998; 17(1):48-54.
19. Fishman A, editor: Pulmonary disease and disorders: Companion handbook, ed 2, New York, McGraw-Hill, 1994.
20. Bulpitt K, Clements P, Lachenbruch P et al: Early undifferentiated connective tissue disease: III. Outcome and prognostic indicators in early scleroderma (Systemic sclerosis), Ann Inter Med 1993; 118:602-609.

21. Mow V, Proctor C, Kelly M: Biomechanics of articular cartilage. In Nordin M, Frankel V, editors: Basic biomechanics of the musculoskeletal system, ed 2, Philadelphia, Lea & Febiger, 1989.
22. Light K, Nuzik S, Personius W et al: Low load prolonged stretch vs high load brief stretch in treating knee contractures, Phys Ther 1984; 64:330.
23. Enoka R: Single joint system components, In Enoka R, editor: Neuromechanics of human movement, ed 3, Champaigne IL, Human Kinetics; 2002:233-238.
24. Feland J, Myrer J, Schulties S et al: The effect of duration of stretch of the hamstring muscle group for increasing range of motion in people aged 65 years or older, Phys Ther 2001; 81(5):1110.
25. Griggs P: Articular neurophysiology. In Zachazeski J, Magee D, Quillen W, editors: Athletic injuries and rehabilitation, Philadelphia, WB Saunders Co, 1996.
26. Gregory J, Brockett C, Morgan D et al: Effects of eccentric muscle contractions on Golgi tendon organ responses to passive and active tension in the cat, J Physiol 2002; 538(1):209-218.
27. Pearson K, Gordon J: Spinal reflexes. In Kandel E, Schwartz J, Jessell T, editors: Principles of neural science, ed 4, New York, McGraw-Hill, 2000.
28. McCann P, Wooten M, Kadaba M et al: A kinematic and electromyographic study of shoulder rehabilitation exercises, Clin Orthop 1993; 288(3):179-188.
29. Salter R: Research: An overview of continuous passive motion. In Salter R, editor: Textbook of disorders and injuries of the musculoskeletal system, ed 3, Baltimore, Williams & Wilkins, 1999.
30. Mancinelli C, Blaha J: Rehabilitation after revision total knee arthroplasty, Sem Arthroplasty 1996; 7(4):226.
31. Nadler S, Malanga G, Zimmerman J: Continuous passive motion in the rehabilitation setting, Am J Phys Med Rehabil 1993; 72:162.
32. Romness D, Rand J: The role of continuous passive motion following total knee arthroplasty, Clin Orthop 1988; 226:239.
33. Vince K, Kelly M, Beck J: Continuous passive motion after total knee arthroplasty, J Arthroplasty 1987; 2:281.
34. Wasilewski S, Woods L, Torgerson W: Value of continuous passive motion in total knee arthroplasty, Orthopedics 1990; 13:291.
35. Maloney W, Schurman D, Hangen D: The influence of continuous passive motion on outcome in total knee arthroplasty, Clin Orthop 1990; 256:162.
36. Ritter M, Gandolf V, Holston K: Continuous passive motion versus physical therapy in total knee arthoplasty, Clin Orthop 1989; 244:239.
37. Archibeck MJ, White RE. What's new in adult reconstructive knee surgery [electronic version]. Journal of Bone and Joint Surgery. 2003. 85-A(7) 1404-1411.
38. Hansenn A, Stuart MJ, Scott RD, Scuderi GR: Surgical options for the middle aged patient with osteoarthritis of the knee joint [Electronic version] Journal of Bone and Joint Surgery 2000 82A (12) 1768-1781.
39. Voss D, Ionla M, Myers B: Proprioceptive neuromuscular facilitation, ed 3, Philadelphia, Harper & Row, 1985.
40. Kottke F, Pauley D, Ptak R: The rationale for prolonged stretching for correction of shortening of connective tissue, Arch Phys Med Rehabil 1966; 47:345.
41. Noyes F: Functional properties of knee ligaments and alterations induced by immobilization, Clin Orthop 1977; 123:210.
42. Sady S, Wortman M, Blanke D: Flexibility training: Ballistic, static or proprioceptive neuromuscular facilitation? Arch Phys Med Rehabil 1982; 63:261.
43. Grimwood P, Appenteng K: Effects of afferent firing frequency on the amplitude of the monosynaptic EPSP elicited by trigeminal spindle afferents on trigeminal motoneurons, Brain Research 1995; 689:299.
44. Prochazka A, Gillard D, Bennett D: Positive force feedback control of muscles, J Neurophysiol 1997; 77:3226.
45. Osternig L, Robertsion R, Troxel R et al: Differential responses to proprioceptive neuromuscular facilitation (PNF) stretch techniques, Med Sci Sports Exerc 1990; 22(1):106.
46. Bohannon R: Effect of repeated eight minute muscle loading on the angle of leg raising. *Phys Ther* 1984; 64:491.
47. Moore M, Hutton R: Electromyographic investigation of muscle stretching techniques, Med Sci Sports Exerc 1980; 12:322-329.
48. Sullivan M, Dejulia J, Worrell T: Effects of pelvis position and stretching method on hamstring muscle flexibility, Med Sci Sports Exerc 1992; 24:1383-1389.
49. Bandy W. Stretching activities for increasing muscle flexibility. In Bandy W, Sanders B, editors: Therapeutic exercise: Techniques for intervention, Philadelphia, Lippincott Williams & Wilkins, 2001.
50. Malone T, Garrett W, Zachazewski J: Muscle: Deformation, injury and rehabilitation. In Zachazewshi J, Magee D, Quillen W, editors: Athletic injuries and rehabilitation, Philadelphia, WB Saunders, 1996.
51. Carlstedt C, Nordin M: Biomechanics of tendon and ligament. In Nordin M, Frankel V, editors: Basic biomechanics of the musculoskeletal system, Philadelphia, Lea & Febiger, 1989.
52. Salter R: Fracture and joint injuries: General features. In Salter R, editor: Textbook of disorders and injuries of the musculoskeletal system, Baltimore, Williams & Wilkins, 1999.

53. Sanders B, Nemeth W. Hip and thigh injuries. In Zachazewski J, Magee D, Quillen W, editors: Athletic injuries and rehabilitation, Philadelphia, WB Saunders, 1996.
54. Van Susante J, Buma P, Kirn H et al: Traumatic heterotopic bone formation in the quadriceps muscle: No progression by continuous passive motion in rabbits, Acta Orthop Scand Suppl 1996; 67:450-454.
55. Fox J, Poss R: The role of manipulation following total knee replacement, J Bone Joint Surg 1981; 63(A):357.
56. Bohannon R, Larkin P: Passive ankle dorsiflexion increases after a regimen of tilt table wedgeboard standing, Phys Ther 1985; 65:1676.