In This Chapter

About The Authors

John G. Aronen, M.D., FACSM, is an orthopedic sports medicine specialist. Retired from the Navy in 1996, Dr. Aronen is a consultant for the Center for Sports Medicine, Saint Francis Memorial Hospital in San Francisco. Dr. Aronen is a selected member of the American Orthopedic Society for Sports Medicine and the American Medical Society for Sports Medicine, and a fellow of the American College of Sports Medicine. Following two years of specialty training in sports medicine, Dr. Aronen founded and served as the head of the Sports Medicine Division of the Department of Orthopedic Surgery at the United States Naval Academy and head team physician from 1979 to 1987. Dr. Aronen then founded and directed a four-week CME/GME course for primary care providers and second-year physician assistant students in the "Evaluation and Management of Musculoskeletal Injuries Commonly Seen in Military Personnel."

Kent A. Lorenz, M.S., CSCS, NSCA-CPT, is an exercise physiologist and program coordinator with the Center for Optimal Health and Performance at San Diego State University. He is also a lecturer and researcher within the School of Exercise and Nutritional Sciences, and uses his strength and conditioning and personal training background to develop exercise programs and classes for adults to help them maintain functional capacity and independence.

CHAPTER 15

Arthritis

John G. Aronen

Kent A. Lorenz

Arthritis is a general term that refers to joint inflammation. The two primary forms are **osteoarthritis (OA)** and **rheumatoid arthritis (RA).** OA results from a degeneration of synovial fluid and generally progresses into a loss of **articular cartilage**, which typically presents itself as localized joint pain and a reduction of **range of motion (ROM)** (Buckwalter & Martin, 2006). Buckwalter and Martin (2006) report that approximately 20 million Americans have OA, and the World Health Organization (WHO) estimates that about 10% of the world's population over the age of 60 has the disease. Of those who have OA, 80% will have limitations of movement and 25% cannot perform major **activities of daily living (ADL),** with the most common sites affected being the knees, hips, spine, hands, and feet (Buckwalter & Martin, 2006).

While OA is the most common affliction of the joints, RA is another condition the ACE-certified Advanced Health & Fitness Specialist (ACE-AHFS) may see among his or her clientele. RA is a chronic autoimmune disease that results in inflammation of the **synovium,** leading to long-term joint damage, chronic pain, and loss of function or disability (Arthritis Foundation, 2007). RA progresses in three stages, with the first being a swelling of the synovial lining, resulting in pain, warmth, stiffness, redness, and swelling of the joint. The second phase is a rapid division and growth of cells, which causes the synovium to thicken. In the third and final stage, the inflamed cells release **enzymes** that break down bone and cartilage, causing the affected joint to lose structure and alignment, leading to more pain and a further decrease in function. RA is a chronic disease that typically worsens with time, resulting in further physical limitations of the involved joints. In comparison to osteoarthritis, RA, being an autoimmune disease and having a more systemic effect, may manifest itself in the development of heart and lung disease and **diabetes.** As with OA, the quality of life for individuals with RA can be improved with exercises that are designed to maintain muscle strength, joint range of motion, and cardiovascular function. The exercise recommendations that are presented in this chapter are specific to OA, but people with RA are encouraged to perform low levels of activity that does not increase inflammation. Certain modifications, such as the use of wrist straps or ankle or wrist weights and the performance of lower-intensity and higher-duration activities, may be needed to accommodate certain clients. Helmick et al. (2008) estimate that 1.3 million Americans suffer from RA, down from 2.1 million in 1995, suggesting a reduction in the prevalence of the disease.

While this chapter introduces both forms of arthritis, the focus from this point on will be on the identification and treatment of OA. As with all chronic conditions, the ACE-AHFS should communicate with his or her clients to discuss what types of activities they are able to do—and how much. If someone presents with some of the signs and symptoms of OA or RA, and he or she has not sought medical advice, the ACE-AHFS should refer the individual to a medical professional before working with him or her. In more severe cases, where the majority of weightbearing activity is painful or limited, the ACE-AHFS may want to refer to a physical therapist or occupational therapist.

Medical conditions (e.g., illnesses, injuries) are typically placed into one of two categories based on their suspected cause, or **etiology**—primary or secondary. The determination of a condition's category is based on whether the underlying cause for the problem can be identified. If the underlying cause for a problem cannot be identified, the problem falls into the primary category (i.e., the individual has the problem, but physicians cannot determine why or what is causing or contributing to the problem). For primary problems, treatment and management must be directed at the symptoms associated with the condition. Unfortunately, while this approach may provide resolution of the symptoms, the underlying cause will continue to contribute to the natural progression of the problem.

If the underlying cause that contributed to the onset of the condition and/or continues to contribute to the condition can be identified, the condition is categorized as secondary. With a problem that is secondary, the emphasis of the treatment and management program must be directed at eliminating or minimizing the underlying cause. Failure to recognize that proper management of a secondary condition includes management of both the symptoms and, more importantly, the underlying cause(s) of the symptoms, will result in only short-term relief from the presenting symptoms, as the underlying cause is allowed to continue to contribute to the natural progression of the problem.

Injuries to a joint occur either acutely or insidiously (i.e., over a prolonged period of time). Any injury to a joint that causes detrimental changes to its structural integrity becomes the starting point for the onset and progression of osteoarthritic changes. Because there are typically detrimental changes to the structural integrity of the knee with an acute injury, such as a sprain of the anterior cruciate ligament, a tear of a meniscus, or patellar dislocation, the starting point for the onset of osteoarthritic changes is the time of the injury. In many acute injuries, due to the severity of the initial changes to the structural integrity of the joint, the starting point for osteoarthritic changes is also the starting point for the discomfort and/or swelling associated with the osteoarthritic changes, constant reminders to the individual that he or she no longer has an entirely healthy or normal joint. For others, the initial changes to the structural integrity of the joint are not severe enough to result in the onset of discomfort and/or swelling from the time of the injury. The starting point for changes to the structural integrity with an injury of insidious onset, as may be seen in the knees and hips with a steady regimen of distance running, is ill defined, as it does not come on acutely, but rather over time. Unfortunately, with the normal progression of degenerative changes that occurs in osteoarthritic joints, discomfort and/or swelling will become evident sooner or later in the majority of cases.

Structural integrity of a joint refers primarily to the following:

- Articular cartilage, which consists of hyaline cartilage, is free of pain fibers and covers the portions of bone that articulate with each other within a joint. Articular cartilage is also referred to as chondral cartilage.
- Subchondral bone, which underlies the articular cartilage, must be healthy to provide appropriate structural support to the articular cartilage overlying it.
- Discomfort and/or swelling are the earliest symptoms or physical findings that indicate that changes have occurred to the structural integrity and that the joint is no longer an entirely healthy or normal joint. Typically, the amount of discomfort and/or swelling is an indicator of the severity of changes to the structural integrity of the joint.

Epidemiology

Prior to the 1980s, OA, often referred to as **degenerative joint disease (DJD),** was believed to occur only in men who suffered an injury to a joint, most commonly the knee, while involved in a contact sport. Because the onset of OA was thought to be caused by the initial injury to the knee, the OA that occurred following a documented knee injury was classified as a secondary problem. Additionally, little concern was given to the initial treatment and long-term management of the etiology, which would have slowed down the progression of the undesirable changes to the structural integrity. Typically, the athlete would remain active in sports, only expediting the "unavoidable" progression of the acute changes, resulting in a chronically painful and functionally deficient knee. Unfortunately, due to an avid desire to return an athlete to all activities rather than be realistic and make modifications in lifestyle that may prolong the life of the injured joint, too little progress has been made in the appropriate initial treatment and long-term management.

Two other factors came into play regarding OA in the late 1970s and early 1980s, the first being the sudden surge of adolescent and teenage girls into injury-producing sports. As the number of participants in sport dramatically increased, the number of significant acute injuries, not only to the knee but to other joints as well, resulted in a large increase of symptoms and findings compatible with osteoarthritis in younger and middle-aged athletes. Additionally, it was noted that not only could the onset of OA [through findings noted on **arthroscopy, magnetic resonance imaging (MRI),** or symptoms compatible with OA] occur following an acute injury to a joint, but it could also occur following persistent microtrauma to the structural integrity of a joint, as seen in distance runners. In the early 2000s (approximately 20 to 25 years following the surge in female participation in sports), a dramatic increase in individuals with physical and radiographic findings compatible with significant osteoarthritic changes in the knees and hips began to appear. The following characteristics were common in this group:

- A female participating in sports that were only sparsely available to the female community prior to 1980, but sprang into popularity in the 1980s (e.g., basketball, volleyball, softball, soccer, distance running)
- An individual incurring a knee/hip injury or simply having a history of following a compulsive daily running regimen
- An individual treated with one of the increasing number of surgical procedures designed to address these injuries, followed by aggressive rehabilitation programs

With emphasis placed on early surgical intervention and aggressive rehabilitation, the high rate of attrition from sports entirely due to injuries at an early age suddenly came more into focus. Experts had assumed that injured joints would naturally degenerate with time and that lifestyle modifications of young athletes would have little or no effect on the outcome of the process of degeneration.

It is slowly becoming understood that the progression of the initial changes to the structural integrity of a joint can be "slowed down" through alterations in the individual's daily lifestyle and through rehabilitation programs designed to regain and maintain normal strength and flexibility of the muscles surrounding the joint. These programs are designed to slow the progression of the degenerative changes already existing in the joint. Each exercise incorporated into a rehabilitation program for an individual with OA must be evaluated for the amount of force it places on the vulnerable joint, because, although the individual may be able to perform the exercise without any pain, this is no guarantee that the exercise is not doing more harm than good over an extended period of time.

Another factor in the increase in prevalence of OA was the changing of dietary and exercise habits. Since the 1980s, the United States went from being one of the leanest and fittest countries in the world to the other end of the spectrum. It soon became apparent that people were living longer and presenting with significant arthritic changes in hips and/or knees that had never experienced acute trauma. Although from this group it appeared that a primary form of OA was associated with normal aging, 52% of patients who required total knee **arthroplasty** and 36% of those who required total hip arthroplasty were **overweight** or obese (Namba et al., 2005).

Although there are some individuals who will develop OA with no identifiable underlying causes, the vast majority of OA is secondary in nature—secondary to trauma and/or **obesity.** Therefore, exercise programs for individuals with OA must keep forces on the osteoarthritic joint to a minimum, as clients strive to retain the strength and flexibility necessary for a joint to function normally.

Physical Symptoms and Findings Associated With Osteoarthritis

To understand the symptoms experienced with OA, the ACE-AHFS must have knowledge of the anatomical structures of a joint. Furthermore, the ACE-AHFS must understand the role each structure plays in normal joint functioning and the contributions each makes to the physical symptoms experienced and the physical findings noted on examination).

The role of a joint, or **articulation,** is to allow motion between bones at a specific site. Because of its high frequency of injury and because it is a common site of OA, the knee will be used in this discussion.

A **capsule** fully encloses each joint, so that fluid produced in the joint is retained in the joint. Lining the capsule is a **synovial membrane** that consists of **synovial cells.** There are two types of synovial cells, type A and type B. The lubrication system, which sounds very simplistic, is actually very sophisticated. The type A cells are secretory in that they produce the **synovial fluid** that acts as a lubricant for the joint. The natural viscosity of the synovial fluid minimizes the degenerative process normally seen between two healthy structures that repetitively articulate with each other. The type B cells are phagocytic, in that they are responsible for the debridement (removal) of the "worn out" synovial fluid and any excess fluid (synovial fluid and/or blood) that may have accumulated in the joint. The articular cartilage of the knee is entirely separate from the two **menisci,** which are made up of fibrocartilage and function to provide shock absorption and stability to the knee.

The articular cartilage is unquestionably a key anatomical structure. Osteoarthritis begins and ends with changes to the structural integrity of the articular cartilage. There are a few properties unique to articular cartilage:

- It has no blood supply and thus cannot heal if injured.
- Because it lacks a blood supply, the role of providing nourishment to the articular cartilage is carried out by the synovial fluid, which is able to enter and exit the articular cartilage at will through microscopic pores in the surface.
- The articular cartilage is void of pain fibers.

The contributions of the articular cartilage to a normal, healthy joint include the following:

- When the surface of the articular cartilage is pristine and covered with synovial fluid, the **coefficient of friction** between the two articulating surfaces is almost zero.
- Because it lacks pain fibers, the articular cartilage prevents the subchondral bone, which has an abundance of pain fibers, from experiencing pain related to the normal transmission of force across joints on a daily basis. Without articular cartilage, the joint would be basically bone on bone—which would be very painful.
- It has been determined clinically that healthy articular cartilage can tolerate approximately seven times the person's body weight before undesirable and often silent detrimental changes begin to compromise the structural integrity of the articular cartilage, which is why it is so important to avoid activities that place unnecessarily high forces on the joints (Repo & Finlay, 1977).

Initial changes to the articular cartilage involve the changing of the once pristine surface into an uneven, incongruous surface. These changes can occur quickly from acute trauma, such as a torn anterior cruciate ligament, meniscal tear, or dislocated patella. Each of these injuries produce **shear forces** in the joint that damage the articular cartilage. The rating of the severity of damage is based on the amount of articular cartilage involved and the depth of the disruption, which ranges from grade 1 to grade 4. Grade 1 implies only superficial changes to the articular cartilage, while grade 4 implies damage to the point where subchondral bone is exposed. The loss of the pristine surfaces leads to an increase in the coefficient of friction, which hastens damage due to wear and tear on the remaining articular cartilage.

Along with the loss of the pristine surface, the once microscopic pores that allowed the synovial fluid to flow freely into the articular cartilage become enlarged, allowing the escape of chemicals from inside the articular cartilage into the joint. These chemicals are direct irritants to the synovial cells and cause them to become inflamed (chemical synovitis). Once inflamed, the cells produce soreness throughout the knee as well as an excessive amount of synovial fluid, which is experienced as tightness in the knee. The inflammation from the chemicals, with the resultant discomfort and excessive synovial fluid production, typically takes 10 to 14 hours. Thus, the individual can be physically active on the knee in the evening, but will note the diffuse discomfort and tightness the next day.

With the continuous wear-and-tear changes due to the increased coefficient of friction, the articular cartilage becomes thinner, allowing the subchondral bone to experience more of the forces transmitted across the joint. Forces experienced by the subchondral bone result in pain, the amount and frequency of which is dependent on many factors:

- The location of the site of exposed subchondral bone (weightbearing vs. nonweightbearing areas)
- The amount of force placed on the site with physical activity (e.g., minimal with swimming, highest with weightbearing activities)
- The weight of the individual, as higher body mass can increase joint **compressive forces** that are excacerbated by misalignment of the femoral-tibial joint (Felson et al., 2004)

The pain can start out as minimal following activity, but progresses in accordance with the amount and frequency of undesired forces placed on the site, typically to the point where the individual's lifestyle is greatly altered by the pain. Unfortunately, most individuals will not consider making changes in their level of physical activity until they are experiencing constant bone pain.

Normal Course of Symptoms in an Osteoarthritic Joint

In the earliest stages, there may be no symptoms or findings until continued forces are placed on the joint and the degenerative changes subsequently progress. Initial symptoms are next-day discomfort and/or stiffness of the joint from chemical synovitis.

As the changes to the structural integrity increase, the next-day discomfort and/or stiffness will increase in intensity and frequency. As the articular cartilage becomes thinner, forces are transmitted and experienced by the subchondral bone, resulting in bone pain during and after activity. Further progression leads to bone-on-bone contact and constant pain.

Because it is relatively silent in nature in its early stages, OA is comparable to **hypertension** in that they are both silent diseases that continue to worsen without telltale signs. These silent changes frequently have dramatic effects on an individual's health and lifestyle activities if he or she fails to recognize them in the early stages. The diagnostic criteria that are used to identify individuals with OA of the knee as outlined by the Agency for Healthcare Research and Quality are joint pain plus five of the following criteria (Samson et al., 2007):

- Client over 50 years of age
- Less than 30 minutes of morning joint stiffness
- **Crepitus**
- Bony tenderness
- Bony enlargement
- No palpable warmth of synovium
- Erythrocyte sedimentation rate (ESR) <40 mm/hr
- Rheumatoid factor <1:40
- Non-inflammatory synovial fluid

It is highly recommended that the ACE-AHFS speak in detail with his or her client to get a full history before developing an exercise program. If the ACE-AHFS or the client is unsure of the status or progression of OA, the client should be referred to a medical professional.

The task for an ACE-AHFS is to recommend specific exercises for clients with OA that will allow them to remain physically active without doing harm to their existing problem. The ACE-AHFS must understand which individuals are at risk for osteoarthritis and who will therefore need exercise programs designed to protect the joints.

Any individual who has had surgery on a joint involved in the exercise program: The initial injury requiring surgical intervention in the vast majority of cases disrupts the structural integrity of the joint (i.e., the articular cartilage and subchondral bone). This situation must be recognized in the development of an exercise program for these individuals.

Individuals who state that they experience discomfort and/or tightness the day following physical activity: Chemical synovitis is the number-one reason for these symptoms to occur and persist. Temporary relief can be achieved with over-the-counter anti-inflammatory medications, but the symptom and not the etiology itself is being treated. Also, the concern over the possible significant side effects of all anti-inflammatory medications often outweighs the palliative benefits. A safer route to control the discomfort may be the use of a glucosamine sulfate with a low-molecular chondroitin (only found in CosaminDS®). A study by the National Institutes of Health (Clegg et al., 2006) reported better relief of pain and no known side effects with the use of the ingredients found only in CosaminDS when compared to Celebrex® 200 mg a day or a placebo.

Overweight individuals: The excessive forces associated with overweight and obesity result in undesirable changes to the articular cartilage. The ACE-AHFS must not do anything to hasten these changes.

Individuals who walk with an altered gait, especially following participation in a weightbearing activity: This can result in an asymmetrical loading of the joint, which increases the risk of joint degeneration (Buckwalter & Martin, 2006).

Individuals who feel the need to wear a brace with activity: Bracing is often a result of a previous injury or current joint pain, which may have resulted in alterations of the articular structures and can increase the risk of the development of OA (Buckwalter & Martin, 2006).

Osteoarthritis and Exercise

By increasing muscular strength and endurance, enhancing the stability of the joints, improving range of motion, and reducing passive tension of the soft tissue surrounding joints, an ACE-AHFS can help his or her clients improve their quality of life, maintain normal function, and prevent deconditioning. One of the secondary outcomes of OA is a development of other diseases, such as **coronary artery disease,** diabetes, and hypertension, as physical activity becomes too painful to attempt, cardiovascular function declines, and the client becomes **sedentary.** For the high number of overweight or obese clients with OA, a further reduction in physical activity can increase the risk for the development of comorbidities. By encouraging clients to maintain cardiovascular fitness by doing exercise that does not increase joint pain, combined with exercises and treatments to help reduce joint pain, an ACE-AHFS can reduce the impact of OA on pain, day-to-day function, cardiovascular health, and quality of life.

Of the randomized, controlled trials exploring the effects of exercise on OA symptoms, some have used hydrotherapy, tai chi, or other low-impact exercises to reduce the stress on the joints (Ettinger et al., 1997; Fransen et al., 2007; Hinman, Haywood, & Day, 2007; Lund et al., 2008; Wang et al., 2007). This outcome is certainly recommended for individuals who experience pain throughout the day, but for those who are relatively pain free, weightbearing exercise and resistance training can be beneficial in not only reducing pain and disability, but also in maintaining normal everyday function. A study examining the effects of different exercise modes on pain, disability, and performance measures found that 60 minutes of light- to moderate-intensity walking three days per week [50 to 70% **heart-rate reserve (HRR)**], or light- to moderate-intensity resistance training three days per week (two sets of 12 repetitions of nine exercises) had significantly better results than the control group over an 18-month intervention (Ettinger et al., 1997). Similar results were obtained in a study that showed that individuals performing three days per week of light- to moderate-intensity resistance training (three sets of eight to 10 repetitions) had lower pain scores and higher functional abilities compared to those who performed only passive range-of-motion exercises (Mikesky et al., 2006). There was also a dose-response relationship between those who did more exercise (75 to 100% of programmed sessions) and those who did less activity (<40% of programmed sessions), with those in the former group experiencing lower pain and disability scores and having greater performance and fitness scores (Ettinger et al., 1997). However, as with all exercise programs for individuals with limitations, the ACE-AHFS must always be cognizant of the client. If the client is hurting, he or she will not do the exercise, so the ACE-AHFS must base decisions on individual feedback, not general guidelines.

Unfortunately, exercise is not a cure for OA, but maintaining a regular exercise program of resistance and aerobic training can reduce the pain and rate of decline in functional capacity (Ettinger et al., 1997; Mikesky et al., 2006). No evidence exists that properly programmed and managed exercise will increase the rate of joint degeneration, as measured by joint-space narrowing (Mikesky et al., 2006) or pain scores (Ettinger et al., 1997; van Baar et al., 1999). Exercise can help reduce some of the risk factors associated with the progression of OA, including weak quadriceps (Bennell & Hinman, 2005; Mikesky et al., 2006), **valgus** or **varus** knee alignment (knock-kneed or bow-legged), weak hip abductors, and obesity (Issa & Sharma, 2006). By selecting exercises or developing programs that address these conditions, an ACE-AHFS can help reduce pain and functional limitations, as well as slow the progression of OA to keep clients active. Further reductions in quadriceps strength, as well as in the hip abductors and extensors, will accelerate the deterioration of the joint by reducing the ability of the individual to control anterior-posterior motion of the knee, as well as exacerbating structural alignment problems that may lead to asymmetrical wear on the articular cartilage.

Programming Guidelines and Considerations

Contraindications and precautions

- Stop immediately if any feelings of joint pain occur during exercise.
- Reduce volume and intensity if next-day tightness or pain is present.
- More frequent, lower-intensity exercise is preferred to a single longer or higher-intensity session.

Guidelines

- Clients should always perform an adequate warm-up (10 minutes) to ensure joint lubrication and increased elasticity of tissues (Hedrick, 1992).
  - ✔They should start with light aerobic exercise to increase systemic blood flow and body temperature.
  - ✔They should perform activation exercises to target specific areas (knees, hips), such as unloaded knee flexion and extension focusing on full ROM.
  - ✔Dynamic flexibility exercises should be performed to maintain elasticity and further increase lubrication (static stretching will cool the body down; the goal is to keep it warm and moving).

Progressions and recommendations

- *Aerobic exercise:* Three to five days per week of light- to moderate-intensity training (50–70% HRR) of lower-impact exercises (e.g., walking, swimming, cycling, inline skating, rowing) for 30 to 60 continuous minutes
  - ✔Multiple, shorter sessions per day may help reduce joint pain.
  - ✔Aqua-therapy or swimming can reduce joint stress while maintaining cardiovascular function and muscular endurance.
  - ✔Clients should gradually progress to longer sessions (increases of 5–10% in duration) when able to comfortably exercise without any fatigue or increasing joint pain, keeping intensity low to avoid higher joint forces.
  - ✔If exercising on consecutive days, clients should switch modes to avoid overuse injuries.
  - ✔The ACE-AHFS should remind clients that proper footwear and softer terrain are important to reduce joint forces during weightbearing exercise.
- *Resistance training:* Two to three days per week of light- to moderate-intensity training

- ✔Clients should perform one to three sets of eight to 12 repetitions.
- ✔They should follow a full-body program using machines or free weights.
- ✔The program should include functional exercises to develop **synergists** as well as overall coordination of the musculature to control and stabilize the joint.
- ✔Clients should begin with isometrics and unloaded movements to increase ROM and develop proper movement patterns.
- ✔Exercise in the water allows for light resistances to help condition muscles through a full, pain-free ROM, while also reducing joint stresses.
- ✔Clients can progress to light resistance (cuff weights, tubing/bands, dumbbells) or bilateral exercises (bodyweight squats).
- ✔Clients should work toward moderate-resistance exercises with as much ROM as can be tolerated, or to unilateral exercises (lunges, step-ups).

- *Flexibility and stress reduction:* ROM exercises daily to keep joints mobile and compliant
  - ✔Clients should perform dynamic flexibility exercises to increase ROM and keep joints lubricated, and static stretching to decrease passive tension (emphasis on the hamstrings, quadriceps, gluteals, gastrocnemius, soleus, and adductors for the lower extremity, and the pectorals, trapezius, latissimus dorsi, deltoids, rhomboids, rotator cuff muscles, biceps, and triceps for the upper extremity).
  - ✔Pilates, yoga, tai chi, and meditation can improve overall flexibility, reduce stress, improve mood or psychological outlook, and reduce pain.
  - ✔**Myofascial release** (foam rollers, massage) can decrease passive tension and break down soft-tissue adhesions that impair normal muscular function.

Osteoarthritis of the knee

- Clients can begin with isometric exercises or light resistance (ankle weights) to strengthen the quadriceps, hamstrings, and gluteals without putting undue pressure on the joint. These exercises can be performed in a pool to further reduce joint pressures.
- Clients can move to bodyweight bilateral exercises (e.g., squats) to develop overall muscular and joint control while encouraging full ROM.
  - ✔Clients can add external resistance to increase muscular strength and endurance if they are pain free.
- Clients can progress to unilateral exercises (lunges, step-ups) to develop muscular control of the joint complex.
  - ✔They should focus on proper control and technique to make sure the patella and femur track correctly.

Osteoarthritis of the hip

- Clients can begin with passive ROM exercises to increase circulation and synovial lubrication, which helps reduce joint compression. Also, aqua-therapy can be used to reduce pressure on the joint.
- Clients can perform exercises lying on the ground to avoid putting too much load on the hips. They should begin with limited-ROM lying hip abduction and extension exercises, plus ROM exercises to strengthen the hip and increase flexibility.
- Clients can progress to bilateral weightbearing exercises with limited ROM (e.g., wall slides, bodyweight squats) to develop the hip complex. They can perform ROM exercises at the end of the session to keep the joint flexible and reduce joint compression and passive tension.
- When the client is able to perform bilateral exercises with limited pain through a larger ROM, he or she can progress to unilateral exercises (e.g., single-leg squat) to further develop the gluteals and hamstrings.

Sample Exercises

All programs should be tailored to meet the individual needs and experiences of each client, but free-weight or body-weight exercises are generally preferred for clients with OA, as they allow for the development of neuromuscular control and conditioning of **antagonists** and synergists to help control and stabilize the joint.

Note that this list of exercises is not meant to be a complete exercise program, but is instead intended to provide sample exercises that can be beneficial in reducing symptoms of OA and ameliorating the progression of the disease.

At the beginning stages of exercise, individuals need to increase quadriceps strength without increasing the risk of joint degeneration. The open-chain terminal 30 degrees knee extension exercise (Figure 15-1) is an effective means of doing just that. This exercise allows for the strengthening of the knee extensors, but by performing only the final 30 degrees the client avoids the high compression forces of the full-ROM knee extension. *Note:* One exercise that should be removed from all exercise programs of those with OA (and for those who want to avoid developing OA) is the full-ROM knee extension. This exercise places tremendous compression forces on the underside of the patella, accelerating the degeneration of the joint.

Once the client has progressed to where he or she can create adequate force and endurance to sustain the contraction shown in Figure 15-1 for 15 to 30 seconds, the client can progress to the closed-chain terminal knee extension (Figure 15-2). This exercise allows for the development of the quadriceps, but also develops the hip extensors and abductors in a stabilizing capacity.

As the client progresses, the addition of bodyweight exercises to condition the lower body is recommended. The isometric or small-ROM wall slide or wall squat (Figure 15-3) is the first exercise that should be added to the program, as it develops the strength of the knee extensors, hip extensors, and abductors, and also helps the client develop the neuromuscular control to help move on to dynamic exercises.

After the client is able to perform the wall slide comfortably, he or she can progress to the bodyweight squat to develop the musculature surrounding the hips and knees (Figure 15-4). The

Figure 15-1
Terminal 30-degrees knee extension (open chain). Starting the knee at 30 degrees of flexion and moving to full extension reduces the compression forces on the underside of the patella, minimizing wear and preventing further degeneration of the articular cartilage. This exercise can be performed with ankle weights or as an isometric hold at the terminal range of motion.

Figure 15-2
Terminal knee extensions (closed chain). The client starts by standing on one leg with the non-supporting leg resting toes-down for support. The client then moves the support leg into 30 degrees of flexion, keeping the shoulders and hips over the heel, and then presses the knee of the supporting leg backward, actively contracting the quadriceps to move into full extension. Resistance bands can be added to increase difficulty.

Figure 15-3
Wall slide/squat. The client begins with the feet comfortably under the hips and the back flat against the wall and slides down the wall until the knees are flexed (staying above 90 degrees or to tolerance) and holds for 2–30 seconds. The client presses through the heels and returns to the starting position.

ACE-AHFS must pay particular attention to the client's knees as he or she performs the exercise. Many clients will allow the knees to move medially (inward), which places strain on the joint and leads to uneven wear of the articular cartilage.

The next progression is the single-leg squat, which places greater demand on the client, especially on the quadriceps, to control the leg and the hip muscles to control and stabilize the pelvis and femur (Figure 15-5). Many people will have difficulty with this exercise, as they do not have the hip strength to maintain proper pelvic alignment or the strength and balance of the legs to maintain correct form. Two common errors are to allow the knees to move laterally (outward) during the eccentric phase of the exercise (descent) and medially (inward) during the concentric phase (ascent). *Note:* Individuals displaying medial motion of the knee often have poorly functioning external hip rotators and extensors (gluteal group) or tight adductors.

Figure 15-4
Bodyweight squat. The client starts with the feet under the hips and the arms out for balance. The client begins the movement by contracting the glutes and hamstrings to pull the hips backward. The ACE-AHFS should remind the client to allow the body to bend naturally at the hips and allow the knees to flex until comfortable. The client must keep the knees in line with the hips and ankles.

Figure 15-5
Single-leg squat. The client begins by balancing on one foot with the other leg flexed behind for counterbalance. The client initiates the movement by pulling the hips back, and then sinks the weight downward over the support-leg heel. It is essential that the patella tracks straight and the knee does not move medially.

One factor that may contribute to both knee and hip pain is the inability to control the hips and pelvis, potentially leading to poor femur-tibia alignment, altered gait patterns, and weakness of the hip abductors. Therefore, development of the spinal, pelvic, and hip stabilizers is important. A simple exercise to accomplish this objective is the hip bridge (Figure 15-6). Particular emphasis should be placed on training the deep spine stabilizers to provide adequate support of the spine and pelvis when engaging in single-leg balance or locomotion activities. Dysfunction in this region often prevents the appropriate recruitment of the prime movers, as the pelvis is unstable and does not provide a good foundation for movements.

Exercises such as the side plank (Figure 15-7), prone plank (Figure 15-8), and prone hip

Figure 15-6
Hip bridge. The client lies supine with the feet about hip-width apart and the shoulders down with the arms out for support. The client presses through the heel and elevates the hips so that they are in a straight line with the knees and shoulders. The client engages the lower back, glutes, and hamstrings to help develop strength and endurance of the hip extensors.

Figure 15-7
Side plank. The client positions the elbow under the shoulder and the knees in line with the hips so that the body is straight, and then lifts the hips off the ground by engaging the deep muscles of the spine (quadratus lumborum). It is important the hips are straight and not "sagging" below the level of the shoulders.

An advanced single-leg version of the hip bridge

For a more advanced and challenging exercise, the client can support him- or herself on the elbow and toes, again with the hips raised so that they are level with the line of the shoulders.

extension (Figure 15-9) develop strength and endurance of these muscles. Not only is stability important, flexibility and adequate ROM are as well, as reducing passive tension along the kinetic chain can reduce tissue stress surrounding the joint, which may be contributing to uneven muscle-recruitment patterns. Using a foam roller or massage is effective, but doing simple hip ROM exercises (Figure 15-10) can be beneficial as well.

It is important to identify any potential muscular dysfunctions that can contribute to the development of OA. By having the client perform calf raises to full plantarflexion (Figure 15-11), perform a standing single-leg balance (Figure 15-12), and walk on the heels and toes (Figure 15-13), an ACE-AHFS can get a basic idea of any muscle-recruitment difficulties. If the client is unable to perform a full-ROM calf raise, he or she may have an underdeveloped gastrocnemius, which may limit the ability to control the knee joint during locomotion. Similarly, the performance of a

Figure 15-8
Prone plank. The client lies prone with the elbows under the shoulders and the feet flexed for support. He or she then lifts the hips off the ground by engaging the gluteals, hamstrings, and spinal extensor muscles so that the hips are level with the shoulders and knees.

The ACE-AHFS can modify this exercise by having the client put the knees down while keeping the hips level with the shoulders.

Figure 15-9
Prone hip extension. The client lies in a prone position with the hands under the chin for support and then lifts the leg with the knee straight by engaging the gluteals and hamstrings. The ACE-AHFS should watch for a "roll" of the torso away from the leg being lifted, as this is a sign of dysfunction of the hip extensors and of the pelvic/spine stabilizers.

standing single-leg balance exercise can identify any hip or pelvic stability problems, as there is increased demand on these muscles during unilateral support tasks. Often, weakness of the spine stabilizers will present itself as a leaning of the hips away from the stance leg, which results

Figure 15-10
Hip ROM complex

The client lies on his or her back and pulls the knee to the chest to feel the stretch in the gluteals and hamstrings.

The client pulls the knee to the same-side shoulder, stretching the hips and hamstrings while also targeting the adductors.

The foot is returned to the center and the knee is dropped to the outside to stretch the adductors. The ACE-AHFS should ensure that the opposite hip is kept stable and does not roll toward the side being stretched.

The client then pulls the knee to the opposite-side shoulder, focusing on the hip extensors and abductors.

The client pulls the leg across the body, stretching the lower back and hip abductors and extensors. Note that the shoulders are kept down and the arm is extended to the side for support.

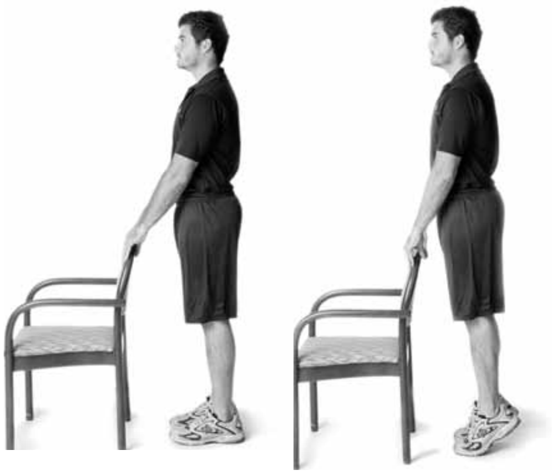


Figure 15-11
Calf raises. Using a chair or other stable object for support, the client rises onto the balls of the feet, coming to full plantarflexion and pausing to fully engage the gastrocnemius. Clients with stronger calves can perform this exercise on one leg at a time.

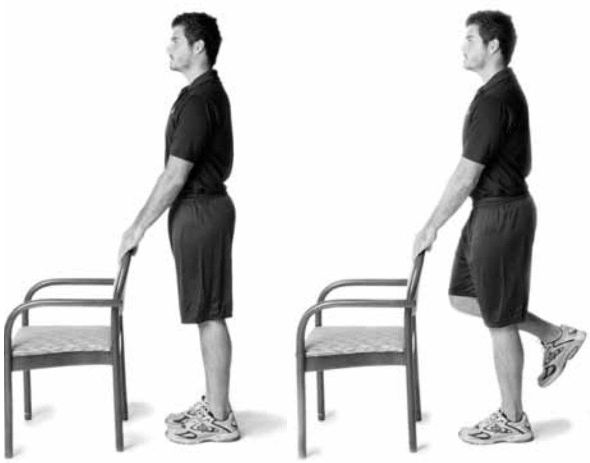


Figure 15-12
Standing single-leg balance. To test the ability of the client to engage the deep spinal stabilizers, obliques, and hip extensors and abductors, the ACE-AHFS can have the client stand facing a table or other stable surface with the feet comfortably under the hips. The client lifts one leg at a time while trying to keep the pelvis level and then holds that position for as long as possible.

Figure 15-13
Heel and toe walking. To assess any potential neurological difficulties created by osteoarthritis of the spine or hip, the ACE-AHFS can have the client walk on the heels and then on the toes. If the client is unable to walk in these positions, this may be evidence of muscle weakness that is caused by joint pain resulting in dysfunction. It may also be evidence of neurological disorders caused by degeneration of the intervertebral discs or the hip joint that leads to pressure on the nerve, resulting in muscular atrophy. These types of diagnoses should be made formally by a physician, but they can give an ACE-AHFS an indication of whether there is some dysfunction.

Figure 15-14
Diagonal arm raise. The client holds a dumbbell in each hand slightly below waist level. He or she then abducts the arm from the shoulder, with the arms at approximately 45 degrees in relationship to the torso until the arm reaches shoulder height. The client can then slowly return to the starting position.

in the loss of balance. Also, weakness of the hip abductors will often result in a rotation of the hips. Finally, any neurological troubles stemming from degeneration of the lumbar discs or nerve compression along the sciatic nerve can be identified if the client has difficulty walking on the heels and toes. Also, conditioning the rotator cuff muscles using the diagonal arm raise exercise (Figure 15-14), along with direct internal and external rotation of the humerus using exercise bands, can reduce the loss of shoulder function that may occur with later development of OA in the cervical spine.

Case Study

A 46-year-old male Navy Seal presents with a history of bilateral knee pain that has been increasing in intensity and frequency over the past four years. There is no history of a significant injury or surgery to either knee. At the time of initial evaluation, his daily activities of lifting weights and running in hard-soled boots on asphalt for 2 miles (3.2 km) [was running 6 miles (9.7 km) a day but steadily decreased his distance over time due to increasing knee pain], were losing appeal to him due to the bilateral knee pain, with swimming being his only well-tolerated activity.

The client was well known for more than 20 years for putting 130 pounds in a rucksack and running in the mountains until bilateral knee pain forced him to stop. Although he attempted to keep running the mountains without the weighted rucksack, the pain was a persistent problem. The client had noted **atrophy** of his quadriceps for years and attempted to build them up with knee extension exercises, squats, and lunges, only to experience more knee pain and visible atrophy of his quadriceps. Finally, he committed himself to seek medical attention. Examination revealed a disproportionate 6'3" 230-pound male (1.9 m; 103.5 kg) with an extremely well-developed upper body, while his lower extremities, most notably his quadriceps, showed significant atrophy. He has very little flexibility of the quadriceps, iliotibial band, adductors, hamstrings, and low-back muscles. The initial interview revealed no history of injuries to his knees. The client could walk a quarter-mile

(0.4 km) if necessary, but running is entirely out of the question. He admittedly avoids stairs and anything that requires him to squat.

He is on a permanent limited duty status until his scheduled retirement date from the Navy due to chronic bilateral knee pain. On examination, his vastus medialis obliques (VMO) are found to be flaccid bilaterally. Other than the gross quadriceps atrophy, the remainder of the examination is non-contributory. X-rays reveal multiple **exostosis** of both the tibiofemoral and patellofemoral joints bilaterally compatible with severe osteoarthritis.

In discussion with the client, he has no desire for total knee replacements except as a "last ditch measure." He has been on a plethora of anti-inflammatories over the years with minimal relief from pain. The treatment regimen is based on many changes in his daily lifestyle, with the end goals of decreasing the pain he experienced and regaining the size and the tone of his quadriceps via low-force, pain-free exercises, and enhancing the flexibility of the muscles of the lower extremities and lower back.

- In accordance with recommendations from his attending physician, he can manage the pain initially with Celebrex, 200 mg a day, along with CosaminDS, three tablets three times a day for the first two months. He and his physician have decided to discontinue the use of Celebrex after the initial two months over concerns of potential significant side effects, while continuing the CosaminDS at two tablets three times a day.
- The client should eliminate boots from his daily life and replace them with soft-soled shoes.
- The client should discontinue all running until he is 100% pain free, and then consider resuming a low-intensity, short-duration running program with input from his physician.
- He can use a bicycle with the seat in a high position for transportation around the base and for exercise.
- The client should avoid stairs and any activity that causes knee pain (such as full squats and lunges) due to the increased forces these movements place on the knee, especially during the descent phase.
- The client should perform exercises with an emphasis on developing strength in the quadriceps and external hip rotators. Terminal knee extensions are appropriate as long as the knee is kept within the final 30 degrees of extension. In this exercise, the client should hold the knee in full extension to see and feel the quadriceps and external hip rotators contract for 10 seconds (see Figure 15-2). Squats performed within the 30-degree range of motion with the back against the wall are encouraged. The squats should be held for 10 seconds with an emphasis on an isometric quadriceps contraction. Aquatic exercise, such as floating on the back and kicking in a pool with fins while not allowing the knees to break the surface of the water, is also indicated. Exercising through a pain-free ROM on elliptical and stair climbing machines is acceptable, since both feet are on a surface (i.e., weightbearing) at all times. He should not use a treadmill, as only one foot is on a surface at all times, thereby forcing one leg to accept the entire body weight with each step. The client should be encouraged to participate in yoga classes as frequently as possible.
- The client should undergo a trial of bilateral Bauerfeind Genutrain knee braces to be worn during waking and non-swimming hours.
- He should double the time spent in the pool, with a portion devoted to kicking on his back with fins.
- The client should have a goal of weight reduction via a modification in dietary habits so that the weight will not simply be regained once his goal of 200 pounds (90 kg) is reached.
- The ACE-AHFS should follow up in two weeks for evaluation specifically of the quadriceps and external hip rotators.

The client was very cooperative due to frustration with prolonged and increasing pain. At the two-week follow up, although his pain was significantly reduced, little gain had been made in the size and strength of his quadriceps and external hip rotators, and thus the client was scheduled for physical therapy daily for 30 sessions of quadriceps electrical stimulation for 20 minutes.

Follow-up at four weeks showed improvement in the size and tone of both quadriceps, along with a decrease in pain. Follow up at six weeks showed much improvement in the size and development of the quadriceps, along with decreased discomfort.

Summary

Osteoarthritis is the most common degenerative joint disease and, if left untreated, can reduce the quality of life for an individual. When used in combination with medical interventions, exercise can help maintain function and reduce pain in an affected joint. Exercises that focus on developing the strength and endurance of muscles that surround the joint can reduce mechanical loading and lessen symptoms. Individual selection of progressive exercises that begin with unloaded isometrics and end with full weightbearing exercises can be an effective method for maintaining joint range of motion, strength, and endurance, and reducing joint pain. The ACE-AHFS must take care to not introduce exercises or activities that have high loads or high strain rates, especially if the client is overweight.

References

Arthritis Foundation (2007). *Rheumatoid Arthritis.* http://www.arthritis.org/disease-center.php?disease_id=31.

Bennell, K. & Hinman, R. (2005). Exercise as a treatment for osteoarthritis. *Current Opinion in Rheumatology,* 17, 5, 634–640.

Buckwalter, J.A. & Martin, J.A. (2006). Osteoarthritis. *Advanced Drug Delivery Reviews*, 58, 150–167.

Clegg, D.O. et al. (2006). Glucosamine, chondrointin sulfate, and the two in combination for painful knee osteoarthritis. *New England Journal of Medicine,* 35, 8, 795–808.

Ettinger, W.H. et al. (1997). A randomized trial comparing aerobic exercise and resistance exercise with a health education program in older adults with knee osteoarthritis: The Fitness Arthritis and Seniors Trial (FAST). *Journal of the American Medical Association,* 277, 1, 25–31.

Felson, D.T. et al. (2004). The effect of body weight on progression of knee osteoarthritis is dependent on alignment. *Arthritis and Rheumatism*, 50, 12, 3904–3909.

Fransen, M. et al. (2007). Physical activity for osteoarthritis management: A randomized controlled clinical trial evaluating hydrotherapy or Tai Chi classes. *Arthritis and Rheumatism*. 57, 3, 407–414.

Hedrick, A. (1992). Exercise physiology: Physiological responses to warm-up. *National Strength & Conditioning Association Journal,* 14, 5, 25–27.

Helmick, C.G. et al. (2008). Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. *Arthritis and Rheumatism.* 58, 1, 15–25.

Hinman, R.S., Haywood, S.E., & Day, A.R. (2007). Aquatic physical therapy for hip and knee osteoarthritis: Results of a single-blind randomized controlled trial. *Physical Therapy*, 87, 1, 32–43.

Issa, S.N. & Sharma, L. (2006). Epidemiology of osteoarthritis: An update. *Current Rheumatology Reports,* 8, 1, 7–15.

Lund, H. et al. (2008). A randomized controlled trial of aquatic and land-based exercise in patients with knee osteoarthritis. *Journal of Rehabilitation Medicine*, 40, 2, 137–144.

Mikesky, A.E. et al. (2006). Effects of strength training on the incidence and progression of knee osteoarthritis. *Arthritis and Rheumatism,* 55, 5, 690–699.

Namba, R.S. et al. (2005). Obesity and perioperative morbidity in total hip and total knee arthroplasty patients. *Journal of Arthroplasty,* 20, 7, Suppl. 3, 46–50.

Repo, R.U. & Finlay, J.B. (1977). Survival of articular cartilage after controlled impact. *Journal of Bone & Joint Surgery,* 59, 3, 1068–1076.

Samson, D.J. et al. (2007). *Treatment of Primary and Secondary Osteoarthritis of the Knee.* Rockville, Md.: Agency for Healthcare Research and Quality, U.S. Department of Health and Human Services. Publication No. 07-E012.

van Baar, M.E. et al. (1999). Effectiveness of exercise therapy in patients with osteoarthritis of the hip or knee: A systematic review of randomized clinical trials. *Arthritis and Rheumatism,* 42, 7, 1361–1369.

Verity, L. (2010). Exercise prescription in patients with diabetes. In: *ACSM's Resource Manual for Guidelines for Exercise Testing and Prescription* (6th ed.). Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.

Wang, T.J. et al. (2007). Effects of aquatic exercise on flexibility, strength and aerobic fitness in adults with osteoarthritis of the hip or knee. *Journal of Advanced Nursing*, 57, 2, 141–152.

Suggested Reading

American College of Sports Medicine (2009). *ACSM's Exercise Management for Persons With Chronic Diseases and Disabilities* (3rd ed.). Champaign, Ill.: Human Kinetics.

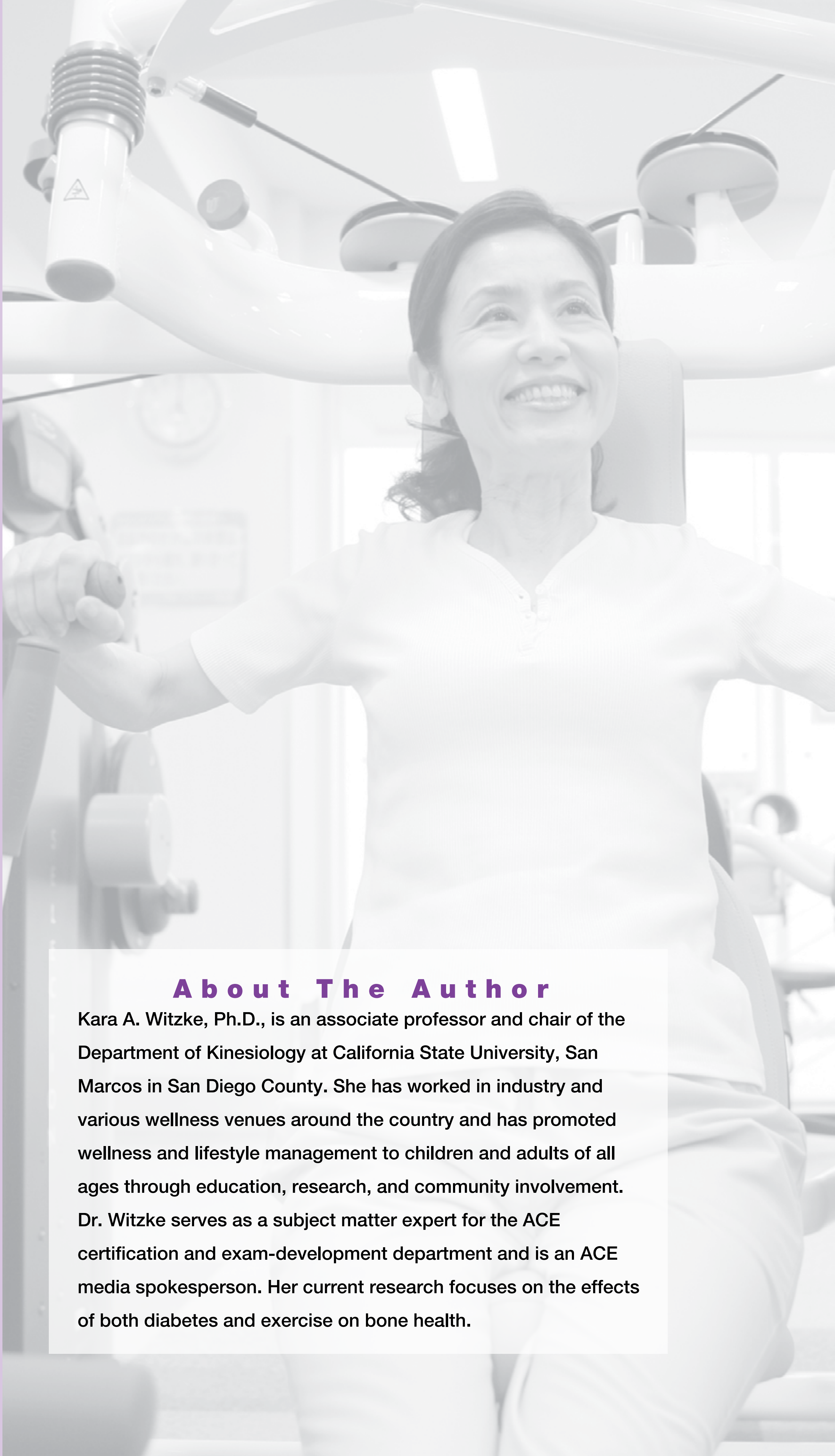
Arthritis Foundation (1999). *PACE: People with Arthritis Can Exercise: Instructor's Manual.* Atlanta: Arthritis Foundation.

Ettinger, W.H. et al. (1997). A randomized trial comparing aerobic exercise and resistance exercise with a health education program in older adults with knee osteoarthritis: The Fitness Arthritis and Seniors Trial (FAST). *Journal of the American Medical Association,* 277, 1, 25–31.

Helmick, C.G. et al. (2008). Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. *Arthritis & Rheumatism,* 58, 1, 15–25.

van Baar, M.E. et al. (1999). Effectiveness of exercise therapy in patients with osteoarthritis of the hip or knee: A systematic review of randomized clinical trials. *Arthritis and Rheumatism,* 42, 7, 1361–1369.

In This Chapter



About The Author

Kara A. Witzke, Ph.D., is an associate professor and chair of the Department of Kinesiology at California State University, San Marcos in San Diego County. She has worked in industry and various wellness venues around the country and has promoted wellness and lifestyle management to children and adults of all ages through education, research, and community involvement. Dr. Witzke serves as a subject matter expert for the ACE certification and exam-development department and is an ACE media spokesperson. Her current research focuses on the effects of both diabetes and exercise on bone health.

CHAPTER 16

Osteoporosis and Osteopenia

Kara A. Witzke

O**steoporosis** is defined conceptually as a condition of generalized skeletal fragility (very low bone mass) that increases the risk of fracture with minimal trauma. **Osteopenia** refers to reduced skeletal mass (low bone mass) that, while not as severe as osteoporosis, may still warrant close monitoring to ensure that the condition does not worsen.

Epidemiology of Osteoporosis and Fractures

Osteoporosis is the most prevalent disease affecting the skeleton. It is characterized by low bone mass and deterioration of the microarchitecture of the bone, resulting in structural weakness and an increased risk for fracture. It is one of the most important public health issues in America because of its prevalence. It is estimated that more than 50% of all women and 20% of all men over the age of 50 will suffer an osteoporotic fracture at some time in their lives (U.S. Department of Health and Human Services, 2004). One in six women will experience a hip fracture, the most devastating type of osteoporotic fracture, compared with only a one-in-nine risk of developing breast cancer (van Staa et al., 2001).

Osteoporotic fractures are most common in the proximal femur (hip), vertebrae (spine), and distal forearm (wrist). However, osteoporosis is really a systemic disease, and individuals with low bone mass are at an increased risk of all types of fractures. Adults who fracture are 50 to 100% more likely to fracture again in a different location (Wu et al., 2002). Hip fractures are by far the most devastating type of fracture due to their strong association with low bone mass, the cost to repair, and the level of disability that often accompanies them even post-surgery. Hip fracture incidence rates increase exponentially with age in both men and women, due to both age-related declines in bone density (a surrogate measure for bone fragility) and an increase in falls, which are responsible for more than 90% of all hip fractures.

In 1990, it was estimated that 1.7 million people worldwide suffered a hip fracture, with annual direct and indirect costs associated with these fractures exceeding $131.5 billion. In the United States alone, these hip fractures imposed costs of more than $20 billion, and these numbers are expected to rise. It is estimated that the number of worldwide hip fractures alone may exceed 6.3 million by the year 2050 (Cummings & Melton, 2002). In addition, an estimated 33.6 million Americans (80% of them women), have osteopenia, a condition of low, but not very low, bone mass. The usefulness of labeling such individuals, whose **bone mineral density** (**BMD**) may actually be within a “normal” range, has been questioned, but osteopenia can be viewed not unlike **prehypertension,** impaired fasting glucose, and borderline high cholesterol in defining an intermediate-risk group with somewhat uncertain boundaries. Although fracture risk is still greater in individuals with osteoporosis rather than osteopenia, the much larger number of persons with osteopenia means that this group represents a substantial portion of the population at risk for fracture (Khosla & Melton, 2007).

Because women live longer and experience more age-related bone loss and falls than men, their incidence of hip fracture is also about two to three times that seen in men (Cummings & Melton, 2002). Age-adjusted rates of hip fracture are highest in Scandinavian and North American populations, and seven times lower in southern European countries and in Asian or African populations (Melton & Cooper, 2001). It should be noted that although Asian populations are at an increased risk for osteoporosis, they suffer relatively few fractures, probably due to decreased rates of falling.

The epidemiology of vertebral fractures is different than that of hip fractures. While only about one-third of all vertebral fractures identified by x-ray come to a specialist's attention, less than 10% result in hospital admissions. These fractures do cause pain, disability, and increased **kyphosis** of the upper spine. Most vertebral fractures do not result from falls, but rather from routine activities such as bending or lifting light objects (Cooper et al., 1992).

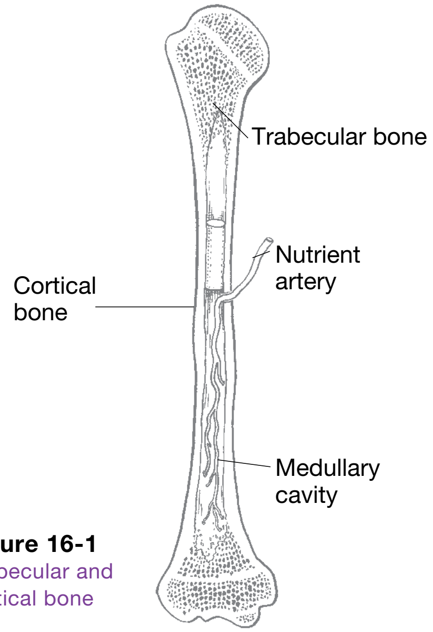


Figure 16-1
Trabecular and cortical bone

Organization of Bone

Bone is a unique tissue with enormous responsibilities. The simple task of supporting loads imposed on it requires that bone have incredible strength and resilience, while also being lightweight and adaptable so that locomotion is not a metabolic burden. Bone consists of an organic component (20 to 25% by weight), an inorganic component (70 to 75% by weight), and a water component (5% by weight). The organic component is primarily composed of type I collagen and some bone cells, while the inorganic component is almost all mineral (crystalline calcium hydroxyapatite).

The human skeleton is composed of two types of bone: cortical (compact) and trabecular (spongy). An important difference between **cortical** and **trabecular bone** is in the way the bone matrix and cellular components are arranged. Calcium comprises 80 to 90% of cortical bone volume, but only 15 to 25% of trabecular bone volume. Cortical bone forms a dense shell around all bones and constitutes the thick shafts of long bones, while trabecular bone is found in the vertebrae and in the ends of long bones (Figure 16-1). Trabecular bone forms a lattice-like network, which greatly increases its surface area for metabolic activity. As a result, trabecular bone undergoes far more remodeling cycles during an individual's lifetime than does cortical bone (Marcus, 1987). A remodeling cycle consists of a bone **resorption** (removal) stage that is followed by a period of new bone formation. Through this coupled process, bone is constantly renewed.

During growth and young adulthood, the rate of bone formation is faster than the rate of bone resorption, leading to an overall gain in bone mineral. This is called **bone modeling.** Modeling improves bone strength not only by adding mass, but also by expanding the inner and outer diameters of bone. This allows bone to adapt its shape according to the loads imposed, which maximizes its strength and resistance to fracture. **Bone remodeling,** on the other hand, is a locally coordinated activity of **osteoclasts** (bone resorption cells) and **osteoblasts** (bone formation cells), whereby bone can both prevent and repair damage caused by everyday loading. A key feature of remodeling is that it replaces damaged tissue with an equal amount of new bone tissue.

In the aging and osteoporotic skeleton, however, the balance between the amount of bone being resorbed and the amount begin formed is unequal, favoring bone resorption. This causes a net loss of bone that eventually causes bone strength and integrity to be compromised (Figure 16-2).

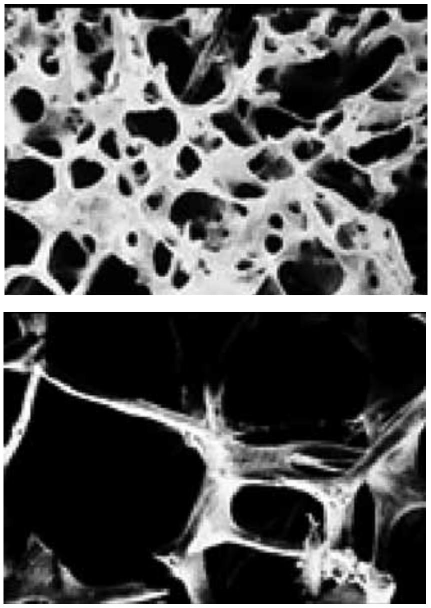


Figure 16-2
Normal versus osteoporotic trabecular bone

Source: Images courtesy of GE Healthcare.

Questions	Yes	No
Do you have a small, thin frame and/or are you Caucasian or Asian?		
Have you or a member of your immediate family broken a bone as an adult?		
Are you a postmenopausal woman?		
Have you had an early or surgically induced menopause?		
Have you taken high doses of thyroid medication or used glucocorticoids $\geq$5 mg a day (e.g., prednisone) for three or more months?		
Have you taken, or are you taking, immunosuppressive medications or chemotherapy to treat cancer?		
Is your diet low in dairy products and other sources of calcium?		
Are you physically inactive?		
Do you smoke cigarettes or drink alcohol in excess?		

Note: The more times an individual answers "yes," the greater the risk for developing osteoporosis.

Figure 16-3
Osteoporosis risk assessment questionnaire

Source: Reprinted with permission from *Osteoporosis: Can It Happen to You?* (2008). National Osteoporosis Foundation, Washington, DC, 20037. All rights reserved.

Factors That Affect Bone

While the effects of physical activity on bone are undeniable, these positive effects only account for approximately 10% of bone mineral in the population as a whole. The remaining 90% of bone mineral is accounted for by a combination of other factors such as genetics, gender, age, race, hormones, lifestyle factors (e.g., smoking, alcohol, caffeine), nutrition, medication, and soft-tissue composition (i.e., lean and fat mass). These factors interact with each other and their degree of influence varies based on the stage of life and the skeletal site. Figure 16-3 provides a osteoporosis risk assessment questionnaire.

Age, Sex, Genetics, and Race

Bone mineral is accrued at various rates throughout early childhood and adolescence, and then diminishes with aging. This normal, age-related process causes a net loss of approximately 5 to 10% of bone mineral per decade and begins some time after the cessation of longitudinal growth and the achievement of peak bone mass (in the third decade) (Snow-Harter & Marcus, 1991). Bone resorption in women is especially rapid during the first five years following menopause (if pharmacotherapy is not implemented), and may cause a loss of 3 to 5% of overall bone mass per year. These age-related bone mineral decrements are not as pronounced in men, primarily because men reach a higher peak BMD, have larger bones that afford a biomechanical resistance to fracture, and do not experience the same rapid postmenopausal bone loss as women. For individuals diagnosed with osteoporosis, it is often impossible to expect to build back significant amounts of bone mineral.

BMD appears to be controlled by a combination of several genes, which may also display important interactive effects with environmental factors (such as physical activity and calcium intake). Nevertheless, familial studies have shown that BMD is strongly influenced by parental bone mass (McKay et al., 1994; Tylavsky et al., 1989), and these assumptions are supported by research that shows very little variation between identical twins (Krall & Dawson-Hughes, 1993). Although various genes [such as those encoding the vitamin-D receptor, collagen Ia1, LDL receptor-related protein 5 (LRP5), and estrogen receptor] have some relation to BMD, attempts to relate them to fracture risk have generally been unsuccessful (Sambrook & Cooper, 2006).

Race implies similar genetic characteristics in a population, perhaps even among individuals in a similar environment, that interact with genes to cause the expression of a particular trait. Studies have confirmed that compared with whites, black adults have higher BMD at both the hip and spine and an increased cortical thickness. Lower fracture rates at both the hip and spine have also been reported in many studies (Aloia et al., 1996; Griffin et al., 1992). Although rates of bone loss appear similar for black and white individuals, it is estimated that African-American women have a 50% lower risk of fracture than Caucasian women (Bohannon, 1999). Similar lower fracture rates have also been shown for Hispanic and Asian populations (Maggi et al., 1991).

Hormones

The endocrine system is highly involved in the regulation of the biologic processes that control bone. The hormones involved in these processes belong to one of two classes, either "controlling" or "influencing" serum calcium levels and the levels of other agents related to bone. Controlling hormones include **parathyroid hormone (PTH),** vitamin D, and **calcitonin.** These hormones induce responses based on plasma concentrations of calcium. Influencing hormones, such as **estrogen, progesterone, testosterone, growth hormone, insulin-like growth factor I (IGF-I), corticosteroids,** and **thyroid hormone,** also modify calcium metabolism, but in response to other factors besides plasma calcium concentrations.

Estrogen has both direct and indirect effects on bone. Directly, estrogen decreases bone remodeling (turnover) through a complex interaction with the estrogen receptor on osteoblasts and by inhibiting other hormones that would normally stimulate osteoclast production. In this way, estrogen maintains bone mass by limiting resorption. Estrogen may also exhibit indirect effects on bone through the parathyroid gland, gut, and kidneys. Specifically, estrogen may lower the sensitivity of PTH to serum calcium levels that would promote mineralization by reducing bone turnover. It may also increase reabsorption of calcium via the kidneys by stimulating vitamin D and calcitonin production, which would also limit bone turnover.

Evidence has been presented about the influence of oral contraceptives on bone mass. In a systematic review of 86 studies published between 1966 and August 2005 that reported on fracture or BMD outcomes by use of combined hormonal contraceptives, researchers report that studies of adolescent and young adult women generally found lower BMD among users of combined oral contraceptives than among non-users. Evidence for premenopausal adult women suggested no differences in BMD between oral contraceptive users and non-users, and use in perimenopausal and postmenopausal women generally preserved bone mass, while non-users lost BMD (Martins, Curtis, & Glasier, 2006). Oral contraceptive pills are often prescribed to female athletes for treatment of menstrual irregularities, but it is still fairly unclear whether BMD improves in these women. It seems as though estrogen-containing pills are more beneficial (or less harmful) to bone in premenopausal women than progesterone-only derivations such as depot medroxyprogesterone acetate (Depo Provera®) (Curtis & Martins, 2006).

During the third trimester of pregnancy, estrogen levels are high, but during lactation, mothers become hypoestrogenic as prolactin becomes a dominant hormone. Maternal bone lost as a result of breastfeeding seems to be recoverable upon weaning (Eisman, 1998). However, in women with low bone mass prior to pregnancy, additional calcium supplementation during pregnancy and lactation is warranted (Funk, Shoback, & Genant, 1995).

Lifestyle Factors

Lifestyle factors such as smoking and alcohol consumption may adversely affect bone. Smoking seems to have a detrimental effect on both pre- and postmenopausal bone density via an increase bone resorption and decreased calcium absorption (Tudor-Locke & McColl, 2000).

Excessive alcohol consumption also appears to exert a direct toxic effect on bone. While studies consistently show bone irregularities in alcoholics, moderate alcohol consumption may be associated with a slight increase in bone via increases in **estradiol** concentrations. It does not appear that moderate alcohol consumption is deleterious to bone density, but women should always be cautioned against the potential effects of an alcoholic lifestyle on bone (Tudor-Locke & McColl, 2000).

Caffeine increases urinary excretion of calcium for at least three hours after ingestion and has been associated with changes in bone remodeling (Massey & Whiting, 1993). It does appear that calcium balance decreases with every cup of coffee ingested, but that these effects are offset by calcium intake. Carbonated cola beverages, on the other hand, do not appear to adversely affect short-term calcium balance, although more studies are needed in this area, since it is possible that the phosphorus contained in these beverages may adversely affect bone (Calvo & Park, 1996; Smith et al., 1989).

The effects on bone of positive lifestyle factors such as exercise are discussed later in the chapter.

Nutrition

Dietary calcium provides the essential building blocks for bone formation. Calcium also plays an important role in muscle contraction and intra- and extracellular ion **homeostasis.** Without an adequate daily intake, calcium is withdrawn from the bones to maintain normal blood levels (9 to 11 mg/dL). Adequate calcium intake is especially important during growth, when the skeleton is still forming and prior to the fourth decade of life when peak bone mass may still be influenced (Recker et al., 1992).

Availability of calcium for absorption is a result of many factors. Calcium from dairy products is highly absorbable, whereas some plant forms are less so. Large intakes of dietary **fiber** can interfere with calcium absorption and dietary protein and sodium intake can increase excretion of calcium. If vitamin D levels are insufficient, parathyroid hormone secretion is increased, which increases activity of the bone resorbing osteoclasts. The **Dietary Reference Intake (DRI)** for both calcium and vitamin D are reported in Table 16-1. Dietitians recommend food as the primary source of calcium and vitamin D. Dairy products and fortified foods such as bread, cereals, and orange juice are good sources of both nutrients. Table 16-2 lists some of the most common dietary sources of calcium.

Table 16-1
Dietary Reference Intakes (DRI) for Calcium and Vitamin D Across the Lifespan

Age (males and females)	Calcium (mg/day)	Vitamin D (IU/day)
1–3	500	200
4–8	800	200
9–18	1300	200
19–50	1000	200
51–70	1200	400
71 and over	1200	600

Source: American Dietetic Association (2006). *Nutrition Fact Sheet: Calcium and Vitamin D: Essential Nutrients for Bone Health.* Chicago: American Dietetic Association.

Table 16-2
Common Sources of Dietary Calcium and Vitamin D

Foods and Beverages	Calcium (mg)	Vitamin D (IU)
Milk, low-fat or non-fat, 1 cup	301	98
Calcium- and vitamin D–fortified orange juice, 1 cup	350	100
Fruit yogurt, low-fat, 1 cup	372	100
Cheddar cheese, low-fat, 2 oz.	236	*
Salmon, pink, canned with soft bones, 3 oz.	208	530

*Not a significant source of the nutrient indicated.

Note: Sunlight causes the skin to make Vitamin D and, for most people, 15 minutes of sunlight several days a week (with the hands and face exposed) is enough.

Source: American Dietetic Association (2006). *Nutrition Fact Sheet: Calcium and Vitamin D: Essential Nutrients for Bone Health.* Chicago: American Dietetic Association.

While dietary sources of calcium are the most bioavailable forms in the body, supplements may provide a primary source of these nutrients in individuals who are unable to achieve adequate dietary intakes. Supplements should be evaluated on the basis of their elemental calcium content (usually between 200 and 600 mg per tablet or chew), and not on the overall milligrams of calcium compounds, such as calcium carbonate or calcium citrate. Calcium carbonate supplements (e.g., Tums®, Viactiv®) may be less expensive and are best taken with food, which promotes an acidic environment in the gastrointestinal tract that aids calcium absorption. Calcium citrate or calcium citrate malate supplements (e.g., Citracal®), while sometimes more expensive, do not cause constipation, a side effect experienced by some individuals using calcium carbonate supplements. Many of these forms also contain vitamin D, which aids in the intestinal absorption of calcium.

Although it is widely accepted that dietary intake of calcium is related, at least in part, to optimal bone health, an association between calcium intake and BMD and fracture is not necessarily so straightforward. Part of the complexity lies in the fact that not all calcium ingested passes directly into the bone reservoir. Calcium retention efficiency in children peaks at about 30% for girls and 36% for boys. Retention rates are inversely related to intake, however, so that when intakes are low, retention rates are relatively higher to compensate for the reduced amount of mineral coming into the body. In randomized controlled trials that show increased bone mineral accrual in children supplementing with calcium, benefits achieved are soon lost when supplementation is withdrawn (Lee et al., 1996). Thus, bone changes following supplementation in children may be transient.

During the pre- and postmenopausal years in women, and throughout men's lives, dietary calcium may have a smaller influence on BMD than genetic and other environmental factors. Part of the problem in interpreting the research in this area is in the difficulty of measurement of lifetime dietary intake and the confounding effects of other nutrients. It is reasonably clear, however, that dietary calcium supplementation has a greater positive effect on BMD than no treatment (postmenopausally), but it probably has less of an effect on bone than either hormone replacement therapy or pharmacological interventions (Khan et al., 2001).

Special Considerations

The Female Athlete Triad

The term "female athlete triad" describes a condition consisting of a combination of disordered eating, menstrual irregularities, and decreased bone mass in athletic women. This combination of factors may increase a woman's risk of osteoporosis and premature fracture. The pattern of the triad is more typical in athletes who believe that they will receive a performance advantage by having lower body weight (e.g., gymnasts, dancers). Dieting behavior usually becomes very restrictive and the pathogenic weight-control behaviors predispose a woman to menstrual dysfunction and eventually compromised bone mass. In this way, the triad disorders are interrelated and the existence of one component is linked, directly or indirectly, to the others (Beals & Meyer, 2007).

The mechanism of bone loss in athletic women with menstrual disturbances has been debated and the original position stand published in 1997 by the American College of Sports Medicine (ACSM) has been revised. ACSM's October 2007 position stand reflects newer evidence that estrogen deficiency is probably not the primary mechanism of bone loss in these athletes, but rather a combination of low estrogen and, more importantly, chronic undernutrition that reduces the rate of bone formation (Nattiv et al., 2007). A well-designed study by Zanker and Swaine (1998) evaluated biochemical markers of bone turnover in active women. Their work showed that women distance runners with long-term amenorrhea have reduced bone formation, compared to eumenorrheic runners and sedentary age-matched eumenorrheic women. Low BMD appears to be much less responsive to estrogen therapy in premenopausal amenorrheic women. This work helps explain why treatment with estrogen in amenorrheic women has not led to the same gains in bone mass as it has with postmenopausal women.

Screening for the triad requires an understanding of the relationships among the three components. Athletes who present with one of the components of the triad should always be evaluated for the others. If an ACE-certified Advanced Health & Fitness Specialist (ACE-AHFS) suspects that a client may have an eating disorder (or restrictive/purging behaviors) and/or menstrual dysfunction, the client should be referred to the appropriate healthcare provider for follow-up.

Menopause

Menopause marks a time of dramatic change in reproductive hormone secretion in women, characterized by estrogen and progesterone deficiency that causes menstrual cycles to cease. The average onset of natural menopause is about 45 to 50 years of age. Clinically, menopause is retrospectively diagnosed when a woman has not had a menstrual period for one year. Changes and symptoms, which usually start several years earlier, as marked by the "perimenopausal" period, include:

- A change in periods—shorter or longer, lighter or heavier, with more or less time in between
- Hot flashes and/or night sweats
- Trouble sleeping
- Vaginal dryness
- Mood swings
- Trouble focusing
- Less hair on the head and more on the face

The early phase of postmenopausal estrogen and progesterone withdrawal that occurs during the first three to five years after menopause is characterized by rapid bone loss (up to 3 to 5% of total bone mass per year), increased circulating plasma levels of calcium, and increased renal calcium excretion (Borer, 2005). Estrogen replacement therapy is most effective at reducing bone loss during this early menopausal period, while calcium supplementation is less effective. After the first five years following menopause, the rate of bone loss decreases back to premenopausal rates of loss (about 1% of total bone mass per year).

Clinical Criteria and Diagnostic Testing

The operational definitions of osteoporosis and osteopenia relate to BMD scores, which are usually measured by **dual-energy x-ray absorptiometry (DEXA)** (Table 16-3).

The National Osteoporosis Foundation guidelines indicate that BMD testing should be performed on:

- All women aged 65 and older regardless of risk factors
- Younger postmenopausal women with one or more risk factors (other than being white, postmenopausal, and female)
- Postmenopausal women who present with fractures (to confirm the diagnosis and determine disease severity).

Note: Medicare covers BMD testing for the following individuals aged 65 and older:

- Estrogen-deficient women at clinical risk for osteoporosis
- Individuals with vertebral abnormalities
- Individuals receiving, or planning to receive, long-term **glucocorticoid** (steroid) therapy
- Individuals with primary **hyperparathyroidism**
- Individuals being monitored to assess the response or efficacy of an approved osteoporosis drug therapy

Medicare permits individuals to repeat BMD testing every two years.

Table 16-3
Diagnostic Categories of Bone Mineral Density

Diagnostic Category	Criterion
Normal	A value for BMD or BMC that is within 1.0 SD of the reference mean for young adults
Low bone mass (osteopenia)	A value for BMD or BMC that is more than 1.0 but less than 2.5 SD below the mean for young adults
Osteoporosis	A value for BMD or BMC that is 2.5 SD or more below the mean for young adults.
Severe osteoporosis (established osteoporosis)	A value for BMD or BMC that is 2.5 SD or more below the mean for young adults in combination with one or more fragility (low-trauma) fractures.

Note: BMD = Bone mineral density; BMC = Bone mineral content; SD = Standard deviation

Source: World Health Organization

Scientists and researchers are interested in the measurement of bone's shape and size (anatomy), strength (biomechanics), and metabolic activity (biochemistry), and the development and adaptation capabilities of bone. Clinicians see bone a little differently, because they view bone as tissue that provides structure and permits the body to move. They generally are interested in preserving or reestablishing those functions and in predicting fracture risk. In this case, bone imaging *in vivo* provides the most important information about bone. The most commonly used methods for imaging bone for clinical purposes include DEXA, **quantitative ultrasound (QUS),** and **quantitative computed tomography (QCT).**

DEXA is probably the most widely used method of clinical evaluation of BMD and risk for fracture. DEXA uses a low-dose x-ray that emits photons at two different energy levels. BMD is calculated based on the amount of photon energy attenuated (absorbed) by the different body tissues (bone, muscle, and fat). Bone attenuates the most energy, followed by muscle and then fat, based on their relative tissue densities. In this way, DEXA can not only measure bone density, but also **body composition,** and is arguably the new "gold standard" against which other techniques are compared. Regional sites such as the lumbar spine, proximal femur (hip), and distal wrist are the most commonly measured sites for assessment of fracture risk. BMD is reported in units equal to bone mineral content divided by the area of the region of interest (g/cm^2). In this way, BMD is not a true "volumetric" density, but rather an "areal" density. The advantages of DEXA, when compared to other methods, include its ease of measurement (five to 10 minutes), low radiation exposure (about equal to the amount received flying across the country), high accuracy and precision, and the ability to measure small changes in BMD over time. Disadvantages of DEXA include the fact that it provides no measure of bone architecture, because it does not distinguish between trabecular and cortical bone. It is therefore difficult to determine material properties of bone, including bone strength, using DEXA.

QUS is another popular method of determining bone status, especially as a "field test" due to its portability and the fact that no ionizing radiation is used. Two ultrasound transducers are positioned on each side of the tissue to be measured (commonly the heel). QUS does not measure bone density, but rather speed of sound (SOS) and broadband ultrasound attenuation (BUA). SOS is expressed as the quotient of the time taken to pass through the bone and the dimension that it passed through (expressed in m/s). Exactly what QUS measures is still a bit of a puzzle, but it is thought that the parameters measured relate to bone density and bone microarchitecture, including trabecular number, connectivity, and orientation. Prospective studies have shown that calcaneal QUS measurement can predict fracture risk in postmenopausal women (Huang et al., 1998; Thompson et al., 1998), and it could be argued that QUS measures characteristics of bone strength that are potentially independent of bone density.

QCT provides two advantages over DEXA in that it can provide a three-dimensional measure (true volumetric density; mg/cm^3) of trabecular and cortical bone. For this reason, it can also be used to study the anatomical structure of trabeculae, which makes it quite attractive for researchers in particular, though it is also useful for clinicians to monitor age-related bone loss and to follow patients with osteoporosis or other metabolic bone diseases. However, this technology has several disadvantages, including poor precision and accuracy and radiation doses 125 times higher than a standard regional DEXA scan. It is also limited to peripheral regions and the lumbar spine, so it not useful for determining qualities of bone at the hip. Newer peripheral quantitative computed tomography (pQCT) units that are typically used in research to measure bone in the tibia/fibula region have improved precision and accuracy, but still expose the patient to higher radiation doses than DEXA and cannot be used at clinically relevant fracture sites such as the hip and spine.

Treatment of Osteoporosis and Osteopenia

Since most fractures occur as a result of falls, reduction in the risk for falls is an important goal of clinicians, as well as the ACE-AHFS. Strategies to reduce fracture risk should emphasize lifestyle modifications such as optimal nutrition, smoking cessation, moderate alcohol and caffeine intake, and exercise that provides adequate bone loading. Emphasis should also be placed on reducing the risk of falls, including improving home safety by reducing tripping hazards; maintaining eyesight through regular vision check-ups and updates for prescription lenses; and improving muscle strength, power, and balance. Hip protector pads are sometimes used to help dissipate the forces sustained in a fall, although compliance in wearing the hip pads is an issue. On a mechanistic level, drugs can be used to help reduce bone resorption or increase bone formation, and, although they do nothing to mediate fall risk, they could help prevent a fracture in the case of a fall.

Non-pharmacological Treatments

An optimal diet to improve the prevention and management of osteoporosis includes adequate caloric intake (to avoid malnutrition), and adequate calcium and vitamin D intake. Adults should follow the American Dietetic Association's (ADA) DRI guidelines for dietary calcium that generally recommend calcium intakes between 1000 and 1200 mg/day and vitamin D intakes of 200 to 600 IU (see Table 16-1). Many women diagnosed with osteoporosis take some form of calcium and vitamin D supplement. A meta-analysis of studies using vitamin D to reduce fracture risk concluded that vitamin D reduced the risk of hip fracture by 26% and non-vertebral fracture by 23% in those individuals with vitamin D deficiency (Bischo-Ferrari et al., 2005). There is some controversy regarding whether the appropriate level of serum vitamin D needed for bone health is 50 or 80 nmol/L, but in those with serum levels lower than 50 nmol/L, doses higher than 1000 IU vitamin D per day could be needed to reach serum levels of 80 nmol/L (Sambrook & Cooper, 2006). Regardless, the ACE-AHFS should always refer clients to a registered dietitian or physician for recommendations regarding vitamin supplementation.

Pharmacological Treatments

Most of the drugs with current Federal Drug Administration (FDA) approval for the management of postmenopausal osteoporosis are called "antiresorptives." These include estrogens, calcitonin, bisphosphonates, selective estrogen receptor modulators (SERMs), and others, including isoflavones and parathyroid hormone (Table 16-4).

After the publication of the Women's Health Initiative study in 2002 (Rossouw et al., 2002), the role of long-term postmenopausal hormone therapy (both estrogen alone and estrogen and progesterone in combination) for the prevention and management of osteoporosis became controversial. The study population consisted of women 50 to 79 years old, many of whom had cardiovascular risk factors. Women were not specifically selected for low bone mass, as is the case in most osteoporosis trials. While both trials did find substantial reductions in subsequent osteoporotic fractures, controversy arose when the results showed an elevated risk for stroke and cardiovascular events with combined hormone therapy, especially if women over the age of 70 began treatment. Although the data suggest a different risk profile for combination therapy versus estrogen alone, the results support the recommendation that hormone therapy should be avoided in favor of alternative antiresorptive agents, and that hormone therapy should remain an option only for short-term early use around menopause in symptomatic women at risk for fracture (Sambrook & Cooper, 2006).

It is worth noting that isoflavones, plant-derived compounds with a chemical structure similar to estrogen (e.g., soy), also have physiological effects similar to estrogen. Women interested in using isoflavones in lieu of estrogen for hormone replacement therapy should be referred to their healthcare providers. Like estrogen, isoflavones exert an acute vasodilatory action and may alter physiological responses to exercise, although studies confirming this effect used larger doses of isoflavones than is commonly used in estrogen replacement therapy (Rosano et al., 1993).

Table 16-4
Antiresorptive Medications Used to Prevent Bone Loss

Drug class	Name of Drug	Brand Name
Estrogens	Estrone sulfate	Ogen®
	Conjugated estrogen	Premarin
	Transdermal estrogen	Estraderm®
	Estropipate	Ortho-Est®
	Esterified estrogen	Estratab®
	Conjugated estrogen + medroxyprogertone acetate	Premphase® Prempro®
Calcitonin	Synthetic salmon calcitonin	MiaCalcin® Calcimar®
Bisphosphonates	Alendronate	Fosamax®
	Risedronate	Actonel®
	Etidronate	Didronel®
	Ibandronate	Boniva®
	Zolendronate	Zometa® Reclast®
	Tiludronate	Skelid®
	Pamidronate	Aredia®
SERMs	Raloxifene	Evista®
	Tamoxifene	Nolvadex®
	Droloxifene (in phase III trial)	
	Levormeloxifene (in phase III trial)	
	Arzoxifene (in phase III trial)	
	Lasofoxifene (in phase III trial)	
Others	Stronitium ranelate (in phase III trial)	Protelos® (in Europe)
	Isoflavones (plant-derived)	
	Tibolone or Ipriflavone (synthetic)	Livial®
	Calcitriol	Rocaltrol®
	Sodium fluoride	
	Parathyroid hormone	Forteo®

Calcitonin is a hormone that inhibits osteoclastic activity, thus reducing bone resorption. Calcitonin derived from human, pig, salmon, and eel have all been used in studies of osteoporosis and have shown effectiveness in both increasing low bone mass and decreasing fracture risk in postmenopausal women (Iwamoto et al., 2002). Side effects may include headaches and flushing. Calcitonin is commonly administered via nasal spray in 200 IU dosages.

Bisphosphonates, another class of antiresorptives, arguably represent one of the most significant advances in the treatment of osteoporosis since the mid-1990s. Oral bisphosphonates are generally well tolerated, although they may cause gastrointestinal intolerance in some individuals. It is very important that clients remain upright for at least 30 minutes following oral dosing to avoid esophageal discomfort. This class of drugs has been shown to reduce the risk of vertebral fractures by 40 to 50% and non-vertebral fractures (including hip fractures) by 20 to 40% (Guyatt et al., 2002). They function by inhibiting the action of osteoclasts (formation remains the same), thereby slowing bone resorption. Despite their impressive potential to reduce fractures, new studies are questioning their safety. These drugs remain in the skeleton for decades, and bone turnover can be affected for up to five years after the drugs are discontinued. Since the natural purpose of bone remodeling is to repair microdamage sustained as a result of everyday wear and tear, it is suspected that bone not permitted to resorb and renew may become brittle. While the fracture data is positive, these studies have only followed patients three to five years into treatment and the optimum duration of therapy remains unclear (Keen, 2007). Furthermore, there have been reports of the rare but serious disorder of **osteonecrosis** of the jaw associated with bisphosphonate use, mainly in patients receiving high doses in combination with cancer treatment. This area needs more investigation to understand the mechanism of this disease.

SERMs represent a class of agents that, while similar in structure to estrogen, exert their effects only on target tissues. The most studied is raloxifene (Evista®) and its effects on markers of bone turnover have been more modest than with bisphosphonates, and its effect on non-vertebral fractures such as the hip have not been marked. For this reason, it is recommended for use in women with milder osteoporosis or in those with osteoporosis primarily in the spine. Side effects include hot flashes and an increased risk of venous thrombosis similar to that associated with hormone therapy.

Parathyroid hormone is an anabolic hormone that, unlike antiresorptive drugs that reduce bone resorption, acts mainly to stimulate bone formation. Although clinical trials showed a 65% risk reduction for new vertebral fractures and a 53% reduction for non-vertebral fractures, benefits to BMD receded after discontinuation. Therefore, this drug is recommended for short durations of less than two years (Sambrook & Cooper, 2006).

Strontium ranelate is an antiosteoporotic agent that is used in the European Union but is not yet approved for use in the U.S., though it is undergoing **phase III clinical trials**. Although its exact mechanism is unclear, it is the first drug that apparently increases bone formation while also reducing bone resorption. Vertebral fracture rates are reduced by about 50% and non-vertebral rates by about 16%, with even higher rates of fracture reduction seen in individuals with the weakest bones (Reginster et al., 2005).

Surgical Intervention

While surgical procedures for treatment of osteoporosis are not available, once fracture has occurred, surgery is often needed to repair damage to the bones. Despite advances in orthopedic surgery, anesthesia, and perioperative care, hip fracture surgery is still associated with complications in up to one-third of patients. The risk of nonunion (failure of the fracture to heal) and osteonecrosis are of particular concern. Data from well-designed outcome studies indicate that the most predictable, durable, and cost-effective procedure for an active older patient with a femoral neck fracture is total joint **arthroplasty.** However, not all patients are candidates for this procedure. In addition, the potential complications of such an invasive surgery, including mortality, may be more difficult to manage and more severe than those associated with less radical procedures (Schmidt et al., 2005).

A newer treatment option for vertebral fractures involves the injection of a special bone cement into the compressed body of the vertebrae. Studies have shown increased bone strength (Steens et al., 2007) and pain relief (Afzal et al., 2007; Steens et al., 2007) in patients treated with this technique.

Exercise

Weightbearing exercise provides one of the most viable, potent tools for both prevention and management of osteopenia and/or osteoporosis. In addition to the ability to improve indices of falling that may lead to fracture, properly planned weightbearing exercises also provide a direct stimulus to bone that improves its strength and structure. Clearly, a well-planned exercise program should provide the foundation from which the disease can be effectively addressed. This topic is discussed in greater detail in the remaining sections of this chapter.

Physiological/Physical Responses to Exercise Training

In general, cross-sectional research demonstrates that physically active individuals of all ages enjoy better skeletal mass than their inactive peers. The magnitude of this difference depends on the mode and intensity of the activity, when the activity was initiated during the lifetime, and for how many years it was performed. The data clearly show that loading exercises performed prior to puberty have the greatest influence on bone. Likewise, increases in bone mass of pre- and postmenopausal women have usually been modest, and the best adaptations occur in those with the lowest starting bone mass values.

Physical Activity and Bone Response in Children and Adolescents

Childhood (prior to puberty) appears to be the time when the skeleton is most responsive to bone-loading activities. Sports that require participants to begin physical training at an early age provide useful information about the role of physical activity in bone growth and mineral accretion. The volume and intensity of training performed by highly motivated young athletes often exceeds five to 24 hours per week. Studies on young gymnasts, whose bodies regularly experience ground reaction forces of 15 times bodyweight, confirm that these types of forces can induce a change in BMD that is between 30 and 85% higher than controls for the whole body, spine, and legs (Bass et al., 1998). Randomized controlled trials in prepubescent children have similarly shown that jumping exercises performed for as little as seven months can confer large differences in bone mineral content of 3 to 4% at the spine and hip between exercise and control groups (Fuchs, Bauer, & Snow, 2001). Furthermore, there is evidence that impact exercise performed during the years before puberty may produce changes in bone that are sustained into adulthood (Fuchs & Snow, 2002). Even weightlifting activities that produce high skeletal loads via muscular pull in the absence of impact have the potential to positively influence bone. In a cross-sectional study of 15- to 20-year-old Olympic weightlifters, forearm bone mineral content was 40 to 50% higher in these athletes versus controls (Virvidakis et al., 1990).

Longitudinal studies also support the observation that children who are generally more physically active than their sedentary counterparts, even in the absence of targeted bone-loading exercise, display higher BMD. A six-year study following the bone mineral accrual in children passing from childhood into adulthood found a 9% and 17% greater total body bone mineral content for active boys and girls, respectively, over their inactive peers (Bailey et al., 1999).

Physical Activity and Bone Response in Premenopausal Women

Not unlike active children, active adults tend to have higher BMD than sedentary adults. These differences have been observed for all regions of the skeleton, regardless of the measurement device used. Some activities may not incorporate loads that apply a sufficient stimulus to bone to produce an adaptive response. Those who participate in activities with high force and load magnitudes, such as gymnastics, jumping, and power lifting, display higher BMD than those who participate in low-intensity or non-weightbearing activities like swimming and cycling. Even though swimming provides muscular pull on bones, it does not appear that this level of loading is adequate to offset the many hours of skeletal *unloading* individuals experience while buoyant in the water (Bellew & Gehrig, 2006).

The most successful exercise interventions in this age group have incorporated jumping exercises that create ground reaction forces of up to six times body weight. These data also suggest that exercises must be performed for a minimum of six months to elicit a significant bone response, but it may take up to nine months or longer (Khan et al., 2001). Another important feature of bone-loading exercises done during the adult years is that unlike results observed in children, these activities must be continued if the individual is to maintain the increases in bone mass. The principle of **reversibility** definitely applies to adult bone mass. There are no studies in premenopausal women that have demonstrated permanent bone gain as a result of short-term training.

Physical Activity and Bone Response in Postmenopausal Women

Bone mineral density of the hip predicts an individual's risk for fracture, and most intervention studies in postmenopausal women have found small, positive effects on hip BMD. Many of the "benefits" to bone described in these studies are due to an observed maintenance of bone in the exercise group with a concomitant decrease in bone in the control group. In this age group, however, even maintenance of bone is a very positive thing, as it may translate into a reduced fracture risk. Intervention studies using only strength training in postmenopausal women have shown mixed results. Nelson and colleagues (1994) had 50- to 70-year-old, previously untrained, estrogen-depleted women train at 80% of **one-repetition maximum (1 RM)** for one year. They showed a 0.9% and 1% gain in BMD in the strength-training group for the hip and spine, respectively, compared to –2.5% and –1.8% BMD losses in controls. Furthermore, they showed that indices of falling, such as muscle mass, muscle strength, and dynamic balance, also improved, which may have implications for fracture risk as well, especially if the exercises are maintained. These results have since been replicated by others and suggest that exercise intervention in this age group can maintain BMD, but rarely serves to add substantial amounts of bone. Similar results have been found for hip BMD using high-impact exercises. Kohrt, Ehsani, and Birge (1997) conducted a study comparing the effects of strength training versus high-impact training on bone. They found that while both programs improved lumbar spine BMD, only the impact group augmented hip BMD. They concluded that it may be better to recommend exercises that generate impact forces on bone over those that generate muscle forces. However, because strength training reduces risk factors for falling, both types of activities should be considered when designing programs for individuals at risk for fracture.

During the late postmenopausal years, when the rate of bone loss has slowed compared to early postmenopause (if estrogen is not replaced), calcium supplementation becomes more important as a way to compensate for reduced estrogenic actions on intestinal calcium absorption and renal calcium excretion (Borer, 2005). There is also evidence that exercise has a synergistic effect on calcium retention in the skeleton and may help to ameliorate bone loss (Specker, 1996). Clients should be encouraged to obtain at least 1200 mg of calcium per day, through either dietary or supplement sources.

Physical Activity and Bone Response in Men

There is a surprising lack of intervention studies on the effects of exercise on bone in men. This is unfortunate, since the number of men with osteoporosis and related fractures is increasing. The few intervention studies that do exist indicate that the response of the male skeleton to exercise is similar to that of women, but is not complicated by the abrupt withdrawal of reproductive hormones in late adulthood. Similar to the results of studies in women, more rigorous training conveys more benefit to the skeleton, while low-intensity exercises, such as walking and moderate-intensity running, afford little benefit (Beck & Snow, 2003).

Summary of Bone's Response to Loading, Hormonal Intervention, and Dietary Intervention

The literature in this area supports eight basic principles related to how bone responds to exercise loading, hormonal intervention, and dietary intervention, which are summarized as follows (Borer, 2005):

- The best time to load bone is prior to puberty. Improvements in bone mass during this time are more dramatic and evidence suggests that they may cause an increase in peak bone mass that persists into adulthood.
- Bone requires dynamic, rather than static, loads to improve its size, shape, and/or density.
- Bone requires loads over and above normal daily loading to improve its size, shape, and/

or density. Bone must sense an overload stimulus if it is to adapt. Higher stresses produce higher bone strains, and these can be accomplished by higher force magnitudes and/or faster application of force. This may partially explain the ineffectiveness of walking programs and low-to-moderate intensity weight-training programs in producing positive gains in bone mass.

- Bone's response is proportional to strain frequency. Bone is maintained both with less frequent mechanical loads of higher intensity and with higher frequency loads at lower intensity.
- Bone's response is improved with brief, intermittent exercise, and may require six to eight hours of recovery between intense loading sessions. The number of loads need not be high (anywhere from five to 50 impacts can be beneficial) to produce the desired response.
- Bone requires an unusual loading pattern to improve. Exercise that loads the skeleton in unusual, uncustomary ways, produces more dramatic responses than those using normal loading patterns.
- Bone requires abundant available nutrient energy if it is to respond. Caloric restriction negatively impacts bone via suppression of key **anabolic** hormones.
- Bone requires abundant calcium and vitamin D availability. This is more important before puberty and after menopause, when the anti-resorptive effects of estrogen are suppressed and vitamin D intake may be inadequate. Evidence also suggests synergistic effects of exercise and calcium and vitamin D on bone during these times.

Programming and Progression Guidelines

Recommendations for Children and Young Adults

- Choose weightbearing activities such as basketball, soccer, volleyball, and gymnastics over non-weightbearing activities such as swimming and cycling. Although these weightbearing activities specifically target bone, it should be understood that a well-rounded program should also contain activities to promote cardiovascular health.
- Emphasize activities and movements that develop muscular strength and power, such as running, hopping, skipping, and jumping. These activities are easily incorporated into games and regular physical-activity classes and should maximize movement and minimize inactive time.
- Remember the principles of **specificity** and **overload.** The skeletal response to exercise is greatest at the site of maximum stress, and the training load must be greater than that encountered on an everyday basis.
- Adequate energy intake is essential for proper growth and bone development. Girls especially should be educated about the importance of a healthy diet and the dangers of menstrual dysfunction.
- Youth should avoid substituting soft drinks for milk. Calcium intake during growth is essential for healthy bones.

The FITT principle for children and young adults is as follows:

Frequency: Several bouts of bone-loading exercise are more effective than one long bout.

Intensity: High-intensity activities with high strain rates promote stronger bones than endurance-type activities.

Time: The number of strain cycles can be small (e.g., 50 to 100), so the duration can be short (five to 10 minutes depending on the types of activities chosen).

Type: A variety of loading patterns applied in unusual ways is more beneficial than activities that mimic everyday activities. Static loads (e.g., isometrics) do not promote increased bone accrual.

Recommendations for Premenopausal Women and Middle-aged Adult Men

- Choose weightbearing activities such as running, group fitness classes (including aerobic and/or muscle-conditioning classes), basketball, soccer, volleyball, and martial arts over non-weightbearing activities such as swimming, cycling, and rowing. Although these weightbearing activities specifically target bone, it should be understood that a well-rounded program should also include activities to promote cardiovascular health.

- Emphasize activities and movements that develop muscular strength and power, such as running, hopping, skipping, and jumping. These are easily incorporated into games and regular physical-activity classes and should maximize movement and minimize inactive time.
- Remember the principles of specificity and overload. The skeletal response to exercise is greatest at the site of maximum stress, and the training load must be greater than that encountered on an everyday basis.
- Simple jumping seems to provide an adequate stimulus for bone, and is a safe and appropriate loading modality for younger women and older nonosteoporotic women (Bassey & Ramsdale, 1994; Bassey et al., 1998; Heikkinen et al., 2007; Winters & Snow, 2000). Studies in premenopausal women using 50 daily jumps (two-footed, using arms for propulsion, bare/stocking feet, bent-knee landing) have shown positive gains in bone mass at the hip. This is a very simple, yet effective method for incorporating high-impact activity into an existing exercise program (Bassey & Ramsdale, 1994).
- High-intensity strength-training exercises (8 RM) should also be included in a well-rounded exercise program, as they have been shown to benefit bone and multiple indices of falling and fracture risk.
- These clients should perform strength-training exercises in a standing (weightbearing) position, using free weights and/or a weighted vest when possible. Doing so will challenge the **vestibular system,** involve stabilizing muscles, and translate to **activities of daily living (ADL)** much more effectively than seated activities. Weighted vest loads of 7 to 15% of the client's body weight are an effective means of loading the skeleton during exercises performed while standing (e.g., lunges, squats, stair steps, calf raises).

Recommendations for Non-osteoporotic Postmenopausal Women and Older Men

Based on the available research, it is not only advisable to recommend exercises that will directly benefit bone, but also those that will reduce the risk for falling. Because falls cause more than 90% of hip and 50% of spine fractures, fall prevention should be central to an exercise program for older adults in general. Muscle weakness, postural instability, and poor functional mobility are important risk factors for falls.

- Any program should be individualized based on the physician's recommendations and the client's current health/fitness status, joint concerns, medication use, and ability level. Having the results from a bone density test is valuable to help determine risk for osteoporosis and fracture.
- Low-intensity activities, such as walking, impart very low bone loads and are not recommended as an effective strategy for the prevention of osteoporosis in postmenopausal women. If walking is performed as a primary exercise modality, it should definitely be accompanied by high-intensity strength training (using 8 RM as a guide).
- Older adults can perform high-intensity strength-training exercises (8 RM), as they have been shown to benefit bone and multiple indices of fracture risk.
- These clients can perform strength training exercises in a standing (weightbearing) position, using free weights and/or a weighted vest when appropriate and possible. Doing so will challenge the vestibular system, involve stabilizing muscles, and translate to improvements in ADL much better than seated activities. Heavy loads (>10% of body weight) in a weighted vest should be avoided if spine BMD status is unknown.
- Clients should focus on lower-body muscle groups, but must not neglect the upper body, as these muscles are important for daily living and maintaining independence.
- High-impact jumping (>2.5 times body weight) and other plyometric exercises should probably be avoided in these individuals, especially if bone status is unknown.

Plyometrics

Plyometric exercises provide a means for incorporating a variety of different movements, including medial/lateral movements that overload the skeleton. **Plyometrics** are specialized jumping exercises associated with high-impact loads and forceful muscular takeoffs, and include various exercises specifically designed to increase muscular strength and power. They are based on the premise that increasing eccentric preload on a muscle will induce the **myotatic stretch reflex,** thereby causing a more forceful concentric contraction. Plyometrics range in difficulty and intensity level from simple stationary jumping to traveling drills, such as hopping and bounding, to high-intensity box jumps (Chu, 1998; Radcliffe & Farentinos, 1999). An inherent benefit of utilizing these types of activities is that they require little equipment, small blocks of time, and are generally safe for adolescents and healthy adults to perform. Care should be taken to make sure clients have adequate leg strength to land properly before incorporating these types of high-intensity plyometric activities.

A proper progression of plyometric exercises is important to ensure adequate muscular strength to maintain proper body mechanics during execution and landing. A plyometric program should begin with one to two sets of 10 repetitions of five to seven different exercises and progress slowly to the more strenuous activities, adding sets and repetitions as tolerated. All exercises should be performed on a medium-hard surface such as grass (preferred), group fitness room floor, or carpet.

It is important to note that plyometrics should not be performed by osteoporotic individuals or by older adults with joint concerns. Clients can perform the sample exercises presented in Figures 16-4 through 16-10 in succession, minimizing time spent on the floor. This method maximizes muscle preload and ensures optimal gains in power.

There are also several more advanced plyometric exercises that can be used with more fit clientele. When performing alternating leg bounds, the knee on the lead leg drives up and forward, lands, and then the opposite knee drives up and forward. The client should emphasize maximum height and distance, and

Figure 16-4
Squat jump—The client jumps as high as possible, using the arms for propulsion, and then lands in a squat position.

Figure 16-5
Stride jump—Starting from a front lunge position, the client drives the hips upward and lands with the opposite leg forward, again in a lunge position.

Figure 16-6
Split jump—Starting from a standing straddle position, the client jumps up, quickly brings the legs together, and lands in the same standing straddle position.

Figure 16-7
Double-leg butt kicks—From a standing position, the client jumps with both legs, kicks the buttocks, and lands on both feet, emphasizing maximum distance.

Figure 16-8
Ankle hops or hop progressions—The client performs basic, small, double-leg hops as quickly as possible.

Figure 16-9
Cone or hurdle hops—The client performs large, high, two-footed hops over barriers such as cones or low hurdles. The goal is to minimize the time spent on the ground.

Figure 16-10
Box jump progression—Using several 4- to 8-inch steps (group fitness benches work well) spaced two to three feet apart, the client jumps onto and off of the succession of steps, spending as little time on the floor as possible. Pauses, if any, should be made on top of the bench rather than on the floor.

move the bent arms in a backward circular motion for added propulsion. This exercise also can be performed as a same-leg bound, where the lead leg cycles around and is the only leg to touch the ground.

A variety of advanced plyometric exercises can be performed on a flight of stairs, ranging from standard running, "bounding" (skipping several stairs at a time) a sideways approach. To perform a sideways stair exercise, the client stands sideways on two successive steps, with the trailing leg straight, and pushes the lead leg up to the next step. He or she then moves the trailing leg up to the next step, pushes off, and so on. This exercise represents a sort of "seesaw" motion and strengthens the hip abductors and adductors.

Recommendations for the Osteoporotic Client

To date, there have been very few intervention studies in people with osteopenia or osteoporosis, so specific recommendations are difficult for this group. Clients diagnosed with osteoporosis, with or without a history of vertebral fractures, should not engage in jumping activities or deep forward trunk flexion exercises such as rowing, toe touches, and full sit-ups (Beck & Snow, 2003). In this group of individuals, a regular walking program, combined with resistance training that targets balance and upper- and lower-body muscle strength, may help to improve muscle strength and coordination, thereby reducing fall risk.

The body-weight resistance exercises shown in Figures 16-11 through 16-24 may be useful for osteoporotic clients. The exercise session should begin with an eight- to 15-minute warm-up of gentle stretching and range-of-motion exercises, followed by five to 10 minutes of aerobic activity at 60 to 75% of maximum predicted heart rate.

If osteoporotic clients are limited by severe pain, exercise options may be limited. It may be advantageous to begin exercise with a warm pool-based program, which, while non-weight-bearing, can improve flexibility and muscle strength in deconditioned clients. These clients may also want to discuss with their physicians the use of calcitonin (a hormone beneficial to bone, administered via nasal spray that has been shown to help reduce pain).

Figure 16-11
Wall arch

Figure 16-13
Wall slide/wall sit

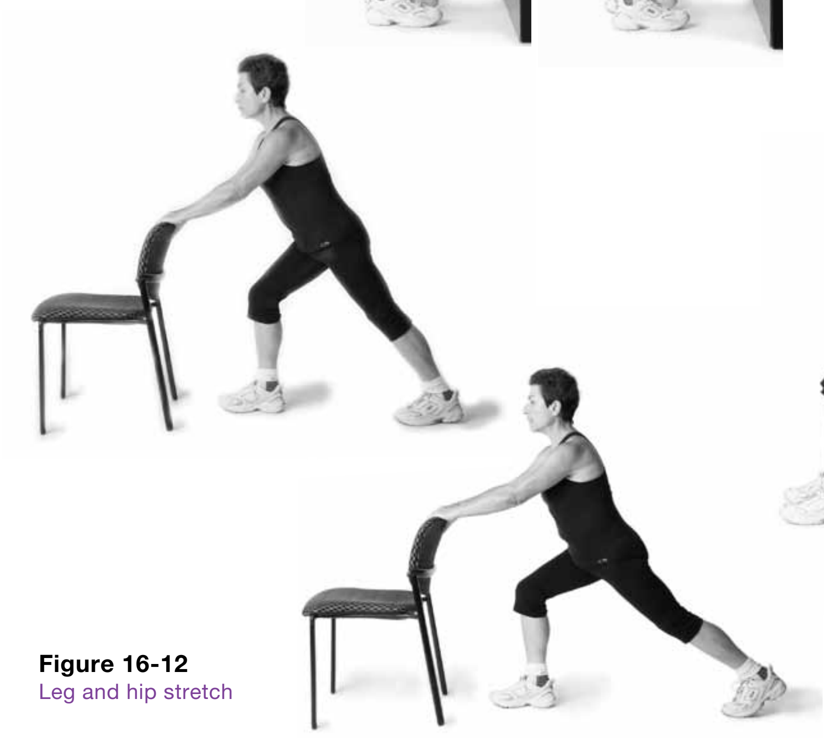


Figure 16-12
Leg and hip stretch

Figure 16-14
Chair rise

Figure 16-15
Standing back bend

Figure 16-16
Seated posture correction

Figure 16-17
Prone trunk lift

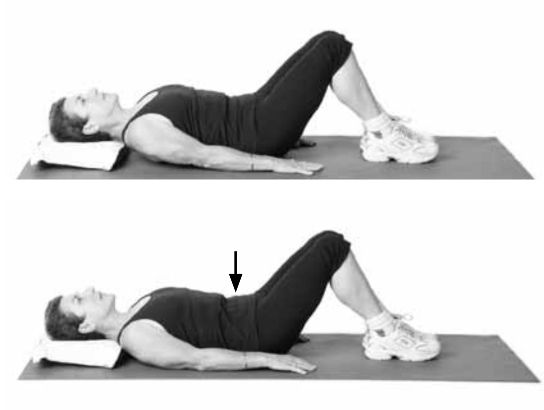


Figure 16-18
Abdominal strengthening

Figure 16-19
The glute bridge

Figure 16-20
Side-lying knee lift

Figure 16-21
Prone leg lift

Figure 16-22
All-fours leg lift

Figure 16-21
Sitting stretch

Figure 16-22
Cat stretch

Case Studies

Case Study 1

Rhonda is a thin, fair-skinned, 35-year-old marathon runner who has come to an ACE-AHFS for a strength-training program after finding out that her 50-year-old mother has osteoporosis. She currently runs about 75 miles per week (121 km/week), but would like to add a weightlifting regimen if it will help reduce her risk for osteoporosis. In the pre-screening assessment, the ACE-AHFS asks Rhonda to complete an osteoporosis risk questionnaire (see Figure 16-3) , which shows that she is at high risk. The ACE-AHFS also discovers that Rhonda has not had a menstrual period for two years (since she increased her training volume to compete in marathons) and only had a period every other month for three years prior to that. She has had two stress fractures in the past 12 months, but managed to continue her physical activity with non-weightbearing exercise while they healed. She is very concerned about her dietary fat intake, and claims that she consumes about 1200 calories per day. She is very eager to begin her strength-training program.

Rhonda is probably displaying characteristics of the female athlete triad. The ACE-AHFS should immediately be concerned that she has not menstruated in two years, and had irregular periods for some time before that. Her high training volume and low caloric intake probably have contributed to her **amenorrhea.** A visit to her physician is definitely in order before the ACE-AHFS can begin working with her. The ACE-AHFS should talk to Rhonda about concern over her training volume and associated stress fractures, and discuss amenorrhea, but must not diagnose the problem. The ACE-AHFS should also educate Rhonda on the usefulness of a bone-density assessment so that she may talk with her physician about getting one. The ACE-AHFS should also ask Rhonda about her dietary intake and recommend that she consult with a qualified nutritionist.

Once Rhonda returns with her physician release and the results from her bone-density assessment, the ACE-AHFS is ready to design a training program for Rhonda. If her bone mass is below normal (osteopenic), the ACE-AHFS should implement a conservative program of strength training and stationary jumping. Additional plyometric exercises should not be added for at least three months to ensure that Rhonda tolerates the jumping without injury. She can begin with upper- and lower-body resistance exercises for the major muscle groups using a weighted vest or dumbbells for resistance. Since she is in good physical condition, she can begin with two sets of 10 repetitions, using 8% of her body weight in the vest, or 75% of her 1 RM. Every two to three weeks, Rhonda can gradually increase her resistance. The ACE-AHFS should recommend that Rhonda weight train two days per week, substituting one of her running days for a weight-training day.

Case Study 2

Fiona is a 70-year-old, postmenopausal woman with a small build who has just been told by her physician that she has osteoporosis. She has lost 1.5 inches (3.8 cm) in height and has upper-back pain, but has been cleared by her physician to begin an exercise program. She performed some upper-body rubber tubing exercises with a physical therapist, but is more concerned about a hip fracture. She is otherwise sedentary and has chosen not to use estrogen replacement therapy, although she has heard positive things about "these new bone drugs" from her friends. She wants the ACE-AHFS to tell her which bone drug she should begin taking, and to start her on a strength-training program to help slow her bone loss. She is on an antidepressant drug that sometimes makes her dizzy, especially when she forgets to wear her glasses. She took a bad fall in her home last month when she tripped over her small dog, and is concerned that she is having trouble climbing the stairs in her home.

Since Fiona has a physician's release to begin working with the ACE-AHFS, she is ready for screening and program development. Although the ACE-AHFS can educate Fiona about the new anti-resorptive drugs, all questions about whether or not they are right for her should be directed to Fiona's physician. She should continue performing the regimen her physical therapist prescribed for her to help maintain upper-back flexibility and strength. Since she has a recent history of falling and experiences episodes of dizziness, the ACE-AHFS should assess her functional mobility using an older adult fitness battery (see Chapters 14 and 21 and Appendix F) (Rikli & Jones, 2001). The ACE-AHFS also should help Fiona assess safety in her home and suggest that she wear stable shoes and her glasses while inside to prevent another tripping incident. A bell on her dog might also be a good idea. She also should secure all area rugs in her house and might consider installing handrails in the bathroom. Fiona definitely needs lower-body strength training, especially if she is to continue to climb the stairs in her home. The ACE-AHFS should implement the exercises presented in this chapter for osteoporotic adults, but should not include any jumping activities, since Fiona's osteoporosis is already established. It is important to make sure that Fiona has her eyeglasses on while training to maximize her safety and minimize her fear and risk of falling.

Summary

Bone is a dynamic, metabolically active tissue that responds to both use and disuse by adapting the amount of mineral to accommodate daily loading patterns. Of the two types of bone in the human body, trabecular bone is more susceptible to the deleterious effects of osteoporosis. Because of its high trabecular content, the neck of the femur is a common osteoporotic fracture site; hip fractures often cause a loss of independence and death in many cases. Osteoporosis prevention and treatment strategies include estrogen replacement therapy or other pharmacological agents, increased calcium intake, and exercise.

Bone is most responsive to mechanical loading during growth, and is progressively less responsive as an individual ages. Bone-loading exercises are beneficial to the skeleton at all stages of life and should be incorporated into a well-rounded program for all clients, especially those at risk for osteoporosis. The types of exercise that are most beneficial to bone are those that sufficiently overload bone using high-force magnitude rather than a high number of low-force repetitions. High-force magnitude can be produced through direct impact loading of the bone, as with jumping, or through strong muscular contractions that bend bone, as with strength training. Nonweightbearing, non-impact exercises such as swimming and rowing do not sufficiently overload bone to increase bone formation or slow bone loss. Similarly, weightbearing exercises that are not significantly different from daily loading patterns (in normally ambulating individuals), such as walking, also do not provide a stimulus for new formation.

The frequency and intensity of the exercises should take into account the client's bone status (preferably from a bone density scan), physical and functional status, medication use, and hormonal status, as well as the overall goals for the program. If the client is at risk for osteopenia or osteoporosis, it is wise for the ACE-AHFS to obtain a physician's clearance for strength-training exercises and/or any type of impact exercise prior to beginning a program.

References

Afzal, S. et al. (2007). Percutaneous vertebroplasty for osteoporotic fractures. *Pain Physician,* 10, 4, 559–563.

Aloia, J.F. et al. (1996). Risk for osteoporosis in black women. *Calcified Tissue International,* 59, 6, 415–423.

Bailey, D.A. et al. (1999). A six-year longitudinal study of the relationship of physical activity to bone mineral accrual in growing children: The University of Saskatchewan bone mineral accrual study. *Journal of Bone Mineral Research,* 14, 10, 1672–1679.

Bass, S. et al. (1998). Exercise before puberty may confer residual benefits in bone density in adulthood: Studies in active prepubertal and retired female gymnasts. *Journal of Bone Mineral Research,* 13, 3, 500–507.

Bassey, E.J. & Ramsdale, S.J. (1994). Increase in femoral bone density in young women following high-impact exercise. *Osteoporos International,* 4, 2, 72–75.

Bassey, E.J. et al. (1998). Pre- and postmenopausal women have different bone mineral density responses to the same high-impact exercise. *Journal of Bone Mineral Research* 13, 12, 1805–1813.

Beals, K.A. & Meyer, N.L. (2007). Female athlete triad update. *Clinical Sports Medicine,* 26, 1, 69–89.

Beck, B.R. & Snow, C.M. (2003). Bone health across the lifespan: Exercising our options. *Exercise and Sport Science Review,* 31, 3, 117–122.

Bellew, J.W. & Gehrig, L. (2006). A comparison of bone mineral density in adolescent female swimmers, soccer players, and weight lifters. *Pediatric Physical Therapy,* 18, 1, 19–22.

Bischo-Ferrari H.A. et al. (2005). Fracture prevention with vitamin D supplementation: A meta-analysis of randomized controlled trials. *Journal of the American Medical Association,* 293, 2257–2264.

Bohannon, A.D. (1999). Osteoporosis and African-American women. *Journal of Women's Health and Gender-based Medicine,* 8, 5, 609–615.

Borer, K.T. (2005). Physical activity in the prevention and amelioration of osteoporosis in women: Interaction of mechanical, hormonal and dietary factors. *Sports Medicine,* 35, 9, 779–830.

Calvo, M.S. & Park, Y.K. (1996). Changing phosphorus content of the U.S. diet: Potential for adverse effects on bone. *Journal of Nutrition,* 126, 4 Suppl., 1168S–1180S.

Chu, D.A. (1998). *Jumping into Plyometrics*. Champaign, Ill.: Human Kinetics.

Cooper, C. et al. (1992). Incidence of clinically diagnosed vertebral fractures: A population-based study in Rochester, Minnesota, 1985–1989. *Journal of Bone Mineral Research,* 7, 2, 221–227.

Cummings, S.R. & Melton, L.J. (2002). Epidemiology and outcomes of osteoporotic fractures. *Lancet,* 359, 9319, 1761–1767.

Curtis, K.M. & Martins, S.L. (2006). Progestogen-only contraception and bone mineral density: A systematic review. *Contraception,* 73, 5, 470–487.

Eisman, J. (1998). Relevance of pregnancy and lactation to osteoporosis? *Lancet,* 352, 9127, 504–505.

Fuchs, R.K., Bauer, J.J., & Snow, C.M. (2001). Jumping improves hip and lumbar spine bone mass in prepubescent children: A randomized controlled trial. *Journal of Bone Mineral Research,* 16, 1, 148–156.

Fuchs, R.K. & Snow, C.M. (2002). Gains in hip bone mass from high-impact training are maintained: A randomized controlled trial in children. *Journal of Pediatrics,* 141, 3, 357–362.

Funk, J.L., Shoback, D.M.. & Genant, H.K. (1995). Transient osteoporosis of the hip in pregnancy: Natural history of changes in bone mineral density. *Clinical Endocrinology (Oxford),* 43, 3, 373–382.

Griffin, M.R. et al. (1992). Black-white differences in fracture rates. *American Journal of Epidemiology,* 136, 11, 1378–1385.

Guyatt, G.H. et al. (2002). Summary of meta-analyses of therapies for postmenopausal osteoporosis and the relationship between bone density and fractures. *Endocrinology Metabolism Clinics of North America,* 31, 3, 659–679, xii.

Heikkinen, R. et al. (2007). Acceleration slope of exercise-induced impacts is a determinant of changes in bone density. *Journal of Biomechics,* 40, 13, 2967–2974.

Huang, C. et al. (1998). Prediction of fracture risk by radiographic absorptiometry and quantitative ultrasound: A prospective study. *Calcified Tissue International,* 53, 5, 380–384.

Iwamoto, J. et al. (2002). Effects of five-year treatment with elcatonin and alfacalcidol on lumbar bone mineral density and the incidence of vertebral fractures in postmenopausal women with osteoporosis: A retrospective study. *Journal of Orthopedic Science,* 7, 6, 637–643.

Keen, R. (2007). Osteoporosis: Strategies for prevention and management. *Best Practice & Research Clinical Rheumatology,* 21, 1, 109–122.

Khan, K. et al. (2001). *Physical Activity and Bone Health*. Champaign, Ill.: Human Kinetics.

Khosla, S. & Melton III, L.J. (2007). Clinical practice: Osteopenia. *New England Journal of Medicine,* 356, 22, 2293–2300.

Kohrt, W.M., Ehsani, A.A., & Birge, Jr., S.J. (1997). Effects of exercise involving predominantly either joint-reaction or ground-reaction forces on bone mineral density in older women. *Journal of Bone Mineral Research,* 12, 8, 1253–1261.

Krall, E.A. & Dawson-Hughes, B. (1993). Heritable and life-style determinants of bone mineral density. *Journal of Bone Mineral Research,* 8, 1, 1–9.

Lee, W.T. et al. (1996). A follow-up study on the effects of calcium-supplement withdrawal and puberty on bone acquisition of children. *American Journal of Clinical Nutrition,* 64, 1, 71–77.

Maggi, S. et al. (1991). Incidence of hip fractures in the elderly: A cross-national analysis. *Osteoporosis International,* 1, 4, 232–241.

Marcus, R. (1987). Normal and abnormal bone remodeling in man. *Annual Review of Medicine,* 38, 129–141.

Martins, S.L., Curtis, K.M., & Glasier, A.F. (2006). Combined hormonal contraception and bone health: A systematic review. *Contraception,* 73, 5, 445–469.

Massey, L.K. & Whiting, S.J. (1993). Caffeine, urinary calcium, calcium metabolism and bone. *Journal of Nutrition,* 123, 9, 1611–1614.

McKay, H.A. et al. (1994). Familial comparison of bone mineral density at the proximal femur and lumbar spine. *Bone and Mineral,* 24, 2, 95–107.

Melton, L.J. & Cooper C. (2001). Magnitude and impact of osteoporosis and fractures. In: Marcus, R., Feldman, D., & Kelsey, J. (Eds.) *Osteoporosis.* San Diego, Calif.: Academic Press.

Nattiv, A. et al. (2007). American College of Sports Medicine position stand: The female athlete triad. *Medicine & Science in Sports & Exercise,* 39, 10, 1867–1882.

Nelson, M.E. et al. (1994). Effects of high-intensity strength training on multiple risk factors for osteoporotic fractures: A randomized controlled trial. *Journal of the American Medical Association,* 272, 24, 1909–1914.

Radcliffe, J.C. & Farentinos, R.C. (1999). *High-powered Plyometrics*. Champaign, Ill.: Human Kinetics.

Recker, R.R. et al. (1992). Bone gain in young adult women. *Journal of the American Medical Association,* 268, 17, 2403–2408.

Reginster, J.Y. et al. (2005). Strontium ranelate reduces the risk of nonvertebral fractures in postmenopausal women with osteoporosis: Treatment of Peripheral Osteoporosis (TROPOS) study. *Journal of Clinical Endocrinology Metabolism,* 90, 5, 2816–2822.

Rikli, R.E. & Jones, C.J. (2001). *Senior Fitness Test Manual*. Champaign, Ill.: Human Kinetics.

Rosano, G.M. et al. (1993). Beneficial effect of oestrogen on exercise-induced myocardial ischaemia in women with coronary artery disease. *Lancet,* 342, 8864, 133–136.

Rossouw, J.E. et al. (2002). Risks and benefits of estrogen plus progestin in healthy postmenopausal women: Principal results From the Women's Health Initiative randomized controlled trial. *Journal of the American Medical Association,* 17, 3, 321–333.

Sambrook, P. & Cooper, C. (2006). Osteoporosis. *Lancet,* 367, 9527, 2010–2018.

Schmidt, A.H. et al. (2005). Femoral neck fractures. *Instructional Course Lectures,* 54, 417-45.

Smith, S. et al. (1989). A preliminary report of the short-term effect of carbonated beverage consumption on calcium metabolism in normal women. *Archives of Internal Medicine,* 149, 11, 2517–2519.

Snow-Harter, C. & Marcus, R. (1991). Exercise, bone mineral density, and osteoporosis. *Exercise and Sport Science Review,* 19, 351–388.

Specker, B.L. (1996). Evidence for an interaction between calcium intake and physical activity on changes in bone mineral density. *Journal of Bone Mineral Research,* 11, 10, 1539–1544.

Steens, J. et al. (2007). The influence of endplate-to-endplate cement augmentation on vertebral strength and stiffness in vertebroplasty. *Spine,* 32, 15, E419–E422.

Thompson, P.W. et al. (1998). Quantitative ultrasound (QUS) of the heel predicts wrist and osteoporosis-related fractures in women age 45–75 years. *Journal of Clinical Densitometry,* 1, 3, 219–225.

Tudor-Locke, C. & McColl, R.S. (2000). Factors related to variation in premenopausal bone mineral status: A health promotion approach. *Osteoporosis International,* 11, 1, 1–24.

Tylavsky, F.A. et al. (1989). Familial resemblance of radial bone mass between premenopausal mothers and their college-age daughters. *Calcified Tissue International,* 45, 5, 265–272.

U.S. Department of Health and Human Services (2004). *Bone Health and Osteoporosis: A Report of the Surgeon General.* Rockville, Md.: U.S. Department of Health and Human Services, Office of the Surgeon General.

van Staa, T.P. et al. (2001). Epidemiology of fractures in England and Wales. *Bone,* 29, 6, 517–522.

Virvidakis, K. et al. (1990). Bone mineral content of junior competitive weightlifters. *International Journal of Sports Medicine,* 11, 3, 244–246.

Winters, K.M. & Snow, C.M. (2000). Body composition predicts bone mineral density and balance in premenopausal women. *Journal of Women's Health and Gender-Based Medicine,* 9, 8, 865–872.

Wu, F. et al. (2002). Fractures between the ages of 20 and 50 years increase women's risk of subsequent fractures. *Archives of Internal Medicine,* 162, 1, 33–36.

Zanker, C.L. & Swaine, I.L. (1998). Relation between bone turnover, oestradiol, and energy balance in women distance runners. *British Journal of Sports Medicine,* 32, 2, 167–171.

Suggested Reading

Bailey, D.A. et al. (1999). A six-year longitudinal study of the relationship of physical activity to bone mineral accrual in growing children: the university of

Saskatchewan bone mineral accrual study. *Journal of Bone Mineral Research,* 14, 10, 1672–1679.

Beals, K.A. & Meyer, N.L. (2007). Female athlete triad update. *Clinical Sports Medicine,* 26, 1, 69–89.

Beck, B.R. & Snow, C.M. (2003). Bone health across the lifespan: Exercising our options. *Exercise Sport Science Reviews,* 31, 3, 117–122.

Chu, D.A. (1998). *Jumping Into Plyometrics*. Champaign, Ill.: Human Kinetics.

Fuchs, R.K., Bauer, J.J., & Snow, C.M. (2001). Jumping improves hip and lumbar spine bone mass in prepubescent children: A randomized controlled trial. *Journal of Bone Mineral Research,* 16, 1, 148–156.

Khan, K. et al. (2001). *Physical Activity and Bone Health*. Champaign, Ill.: Human Kinetics.

Kohrt, W.M., Ehsani, A.A., & Birge, S.J., Jr. (1997). Effects of exercise involving predominantly either joint-reaction or ground-reaction forces on bone mineral density in older women. *Journal of Bone Mineral Research,* 12, 8, 1253–1261.

Marcus, R. (1987). Normal and abnormal bone remodeling in man. *Annual Review of Medicine,* 38, 129–141.

Nattiv, A. et al. (2007). American College of Sports Medicine position stand: The female athlete triad. *Medicine & Science in Sports & Exercise,* 39, 10, 1867–1882.

Nelson, M.E. et al. (1994). Effects of high-intensity strength training on multiple risk factors for osteoporotic fractures: A randomized controlled trial. *Journal of the American Medical Association,* 272, 24, 1909–1914.

United States Public Health Service, Office of the Surgeon General. (2004). *Bone Health and Osteoporosis : A Report of the Surgeon General*. Rockville, Md., U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General.

In This Chapter

ACE would like to acknowledge the contributions to this chapter made by Sabrena Merrill, M.S., fitness-industry consultant, author, and educator.

About The Author

Jennifer Solomon, M.D., is board certified in Physical Medicine and Rehabilitation and fellowship trained in Spine and Sports Medicine. She specializes in non-operative treatments for sports and spine injuries, including electrodiagnostics. Dr. Solomon is assistant attending physiatrist at the Hospital for Special Surgery's Women's Sports Medicine Center and clinical instructor at the Weill Medical College of Cornell University. She serves as team physician for St. Peter's College and has covered several sporting events, including the NYC Marathon, tennis tournaments, and various races. Dr. Solomon is also a team physician for the United States Federation Cup Tennis Team and a medical consultant for La Palestra Center for Preventative Medicine. Dr. Solomon is a member of the American Academy of Physical Medicine and Rehabilitation, the North American Spine Society, and the American Association of Electrodiagnostic Medicine. She has published more than 15 articles and chapters on a variety of spine and sports medicine topics.

CHAPTER 20

Low-back Pain

Jennifer Solomon

Epidemiology

Low-back pain (LBP) plagues modern society and is a significant source of cost and disability. Estimates from the National Health Interview Survey (NHIS), which surveyed more than 31,000 adults, revealed that about 25% experienced LBP in the past three months (Deyo et al., 2006). Studies have shown that more than 80% of Americans suffer from at least one episode of back pain during their lifetime (Anderson, 1997). While these episodes vary in length and intensity, historical records estimate that at any one time there are 1.2 million adults disabled as a result of their LBP (Wong & Transfeltd, 2007). A significant proportion of these individuals seek assistance from a healthcare professional. In fact, back pain is the second most common complaint heard in doctors' offices.

While the majority of acute back pain improves over time, some people develop recurrences, while others experience continuous pain. Acute pain can be thought of as pain resulting from a specific trauma or disease that responds to traditional modes of treatment, such as pain relievers. In contrast, chronic pain represents the lasting effects once an inciting event has faded, and is generally resistant to usual treatments (Wong & Transfeltd, 2007). Acute pain and chronic pain not only have individual physiologic mechanisms, but may also have varying psychological, societal, and economic implications. People with LBP report symptoms of **depression, anxiety,** and insomnia more frequently than those without it (Morris, 2006). LBP is the second leading cause of work absenteeism, following upper respiratory infections (Wong & Transfeltd, 2007). In an analysis of total healthcare expenditures in 1998, $90.7 billion was spent on individuals with back pain, which was 60% more than was spent on their back pain–free counterparts (Luo et al., 2004).

Factors Contributing to LBP

Researchers have attempted to discern specific factors that contribute to the onset and persistence of LBP, and while there is much discrepancy, some consistencies exist. These risk factors can be divided into individual, activity, and psychological categories.

Individual Factors

Age and Sex

LBP occurs most commonly in the 30- to 55-year-old age group (Wong & Transfeltd, 2007). Herniated disks are most common in those between the ages of 30 and 40, a time when the water content of intervertebral disks has decreased. However, many individuals will have had at least one episode of back pain earlier in life (Rubin, 2007). In fact, LBP in adolescence appears to be a risk factor for similar symptoms in adulthood. The prevalence of back pain increases with age until age 60, with older individuals experiencing greater rates of chronic or intermittent pain (Rubin, 2007).

Overall, men and women appear to be equally affected. However, older women have a higher prevalence of back pain than older men, which may be secondary to the greater rates of

osteoporosis of the spine seen in women (Bressler et al., 1999). In contrast, men are more involved in heavy work, and therefore have higher rates of occupational back pain.

Body Type

While body weight may play some role in the development of back pain, the data to prove a strong association is currently lacking (Leboeuf-Yde, 2000). Likewise, height and body build have not been shown to have a strong correlation with LBP.

Conditions affecting posture such as **scoliosis, kyphosis,** and leg-length discrepancies are not predisposing factors for LBP (Wong & Transfeltd, 2007). It is unclear, however, how congenital vertebral abnormalities such as **spina bifida occulta** or **transitional vertebra** contribute to LBP.

Smoking

Smoking is thought to be a significant risk factor, though the exact mechanism remains unclear. It is thought that smoking may decrease blood flow to the intervertebral discs, leading to a deficit of nutrients and/or a lack of sufficient oxygen, which may lead to accelerated cell death. Other effects of smoking may include an increase in the rate of development of osteoporosis, fractures, and degenerative changes in the spine (Manek & MacGregor, 2005). In addition, chronic coughing may be an indirect link between smoking and back pain.

Activity-related Factors

Occupation

Certain types of occupational activities appear to predispose individuals to LBP, including heavy lifting, carrying, pulling, pushing, prolonged walking or standing, driving, and working night shifts (Hurwitz & Morgenstern, 1997). High exposure to whole-body vibration is also a risk factor for back pain. Specific professions that are at higher risk include sales, clerical work, repair service, and transportation (Bahr et al., 2004). Work-related stress and dissatisfaction are also associated with the development of LBP.

Exercise

Studies that have surveyed athletes to determine which sports have higher rates of back pain have yielded conflicting results. However, athletes involved in certain activities, such as cross-country skiing and rowing, may have higher rates of LBP than non-athletes (Borenstein, Wiesel, & Boeden, 2004).

The protective role of specific exercise regimens in preventing LBP is less clear, as are associations with **overweight** and **obesity** (Leboeuf-Yde, 2000). In general, those who engage in regular recreational physical activity appear to be less likely to have back pain at any given time and are less likely to develop future pain (Rubin, 2007). Not surprisingly, some high-intensity or repetitive exercises (i.e., golf and tennis) may, in fact, predispose or worsen an existing condition. While physical fitness does not completely prevent LBP, it may improve functional outcomes by decreasing recovery time. Unfortunately, many people with LBP choose not to exercise and actually believe that doing so would be detrimental to their condition.

Psychological Factors

In general, depression, anxiety, and insomnia are strongly correlated with back pain. They can be both predisposing (e.g., psychogenic muscle tension, work distress) and resulting factors of existing back pain. Chronic back pain sufferers are six times more likely to be depressed than individuals without pain (Currie & Wang, 2004). Depression may affect an individual's ability to cope with pain. Fear-avoidance personality variables (exaggerated pain or fear that activity will cause permanent damage) and passive coping techniques (avoidance, withdrawal, wishful thinking) are positive predictors of chronic back pain symptoms. The inability to determine an exact cause of pain or to effectively relieve symptoms may result in further depression.

Overview of Conditions

The possible etiologies of LBP are numerous. Mechanical causes account for approximately 97% of cases, whereas 2% of cases are "visceral disease," or disease of the internal organs and structures, such as

pancreatitis, prostatitis, and **aortic aneurysm.** The remaining 1% of back pain results from non-mechanical spinal conditions such as tumors, infections, and rheumatologic disorders (Deyo & Weinstein, 2001). This section describes some of the most common mechanical back pain diagnoses, along with their causes and typical symptoms.

Lumbar **strain** or **sprain** is a non-specific term often used to describe mechanical LBP. Typically, a strain refers to a muscle injury in which the fibers are abnormally stretched, while a sprain refers to a torn ligament. A more descriptive and accurate term for most of these cases is **discogenic back pain**, which refers to dysfunction or degeneration of lumbar intervertebral discs. This phenomenon is a universal process of aging that leads to chemical and physical changes in the disc. Discs are comprised of an outer ring, the **annulus fibrosus,** and an inner gel, the **nucleus pulposus.** Over time, discs lose their water content and acquire gradual fibrotic changes that limit mobility.

While these changes occur in everyone, the presence of symptomatic back pain is multifactorial. Microtrauma may lead to small tears or cracks in the annulus and result in inflammation and pain around annular nerves. Also, gradual narrowing of the disc height may cause an unstable surface where the facet joints articulate, leading to an overloading of those structures and, ultimately, pain. Generally, discogenic pain is caused by activities that increase pressure within the disc, such as coughing, sneezing, sitting, and bending forward. Discogenic pain can be acute or chronic.

Herniated discs account for approximately 4% of all mechanical LBP (Deyo & Weinstein, 2001). As previously noted, aging affects discs by decreasing the nearly 80% water content of the nucleus and making the annulus more prone to tears. A herniation occurs when there is a tear in the annulus and a subsequent extrusion of the nucleus through this annular defect. Individuals with herniated discs complain of sharp or throbbing LBP, which is worse with movement but improved with lying down. Herniated material may extend out far enough to compress the nerve roots that exit the spinal cord at that level, causing pain, numbness, tingling, or weakness. These are signs and symptoms of lumbar **radiculopathy,** or nerve root impairment. **Sciatica** refers to radicular symptoms that follow the path of the sciatic nerve, down the posterior aspect of the thigh, lower leg, and foot. Generally, there is no acute event associated with the onset of sciatica, which may be exacerbated by standing, sitting, sneezing, heavy lifting, or having a bowel movement. In some cases, the sciatic symptoms may be equivalent to, or worse than, the LBP itself.

Another 3% of mechanical pain is secondary to **spinal stenosis,** which results from narrowing of the central spinal canal, either by bone or soft tissue (Deyo & Weinstein, 2001). Degenerative changes of the facet joints and intervertebral discs are largely responsible for this process. Thus, spinal stenosis typically arises in persons over age 50. Symptoms arise when the narrowing causes compression of the spinal cord or spinal nerves. Most commonly, spinal stenosis sufferers complain of cramping, pain, numbness, or weakness in their back or legs. However, some will report leg pain only. The typical pattern is termed **pseudoclaudication,** which refers to pain in the buttock, thigh, or leg that occurs with standing or walking and is relieved by rest in a lying or sitting position, or by flexing forward at the waist.

Approximately 4% of mechanical LBP occurs from osteoporotic **compression fractures** (Deyo & Weinstein, 2001). Spinal fractures generally result from major trauma, such as a fall from a great height or a motor vehicle accident. However, individuals with **osteopenia** or osteoporosis, in which bone density is reduced, can acquire compression fractures from less significant trauma, even from something as simple as a sneeze or cough. Up to 20% of the time, compression fractures due to osteopenia or osteoporosis may be asymptomatic and found incidentally on imaging performed for other reasons (Wong & Transfeltd, 2007). The remaining 80% of osteoporotic compression fracture cases experience a sudden onset of pain that is usually diffuse in nature.

Spondylolysis, another type of fracture, is a common asymptomatic finding in 5% of the general population (Yu & Garfin, 1994). Spondylolysis is essentially a stress fracture in the posterior aspect of the spine, the **pars intrarticularis**, where the vertebral body and posterior elements join together. The fifth lumbar vertebra is the most common location for spondylolysis. The condition typically occurs in children or young adults involved in sports and is a result of compressive forces or hyperextension. Symptoms can be unilateral or bilateral, acute or chronic, and range in severity from mild to immobilizing.

Spondylolysis can lead to **spondylolisthesis,** which is an anterior displacement of a vertebra relative to the one below it. The most common symptom is LBP, which may or may not be associated with an acute injury and is worse with flexion but relieved by extension. Leg pain can also occur with spondylolisthesis if spinal nerve roots are irritated as they leave the canal.

Diagnostic Testing

Generally, all clients with back pain should receive a thorough physical exam from a physician. In all of the previously described conditions, there are common warning signs that necessitate immediate attention, including fever, loss of bowel or bladder control, unexplained weight loss, history of cancer or recent infection, and intravenous drug use.

After careful evaluation, the physician may choose to pursue further evaluation with an imaging study. Plain radiographs or x-rays are often used as an initial test, as they are the best option for evaluating bony changes such as fracture, tumor, spondylolisthesis, and disc-space narrowing. **Computed tomography (CT)** and **magnetic resonance imaging (MRI)** are used to visualize spinal stenosis and herniated discs and may be used to rule out conditions such as infection or cancer. However, CT and MRI may also detect asymptomatic abnormalities. Therefore, careful correlation with the individual's subjective history and physical exam is essential when using any imaging technique.

Treatment

Since the natural history of LBP is variable, treatment goals depend on the specific condition. Even though studies have shown that one-third of patients presenting to primary care offices with non-specific back pain significantly improve within one week, and two-thirds improve within seven weeks, up to 40% will experience a recurrence within six months (Carey & Garrett, 1999). Thus, a significant proportion of back-pain patients experience a relapsing and remitting course of pathology. Still others may develop a chronic disabling or persistent condition. Multiple factors may determine the overall prognosis, but each diagnosis also inherently includes general expectations for the extent and timing of recovery. For example, pain from acute herniated discs has a favorable prognosis, with only 10% of patients having significant pain after six weeks. However, painful symptoms in approximately 70% of patients with spinal stenosis remain stable, while only 15% improve and the remaining 15% gradually worsen (Deyo & Weinstein, 2001).

Treatment options for patients with LBP have evolved and, in some cases, become a source of much debate. Because of the ubiquitous nature of LBP in modern society, many patients approach their diagnosis with several preconceived ideas about treatment, formed from a combination of lay press articles and recounted experiences from friends and relatives. However, for almost all conditions, current thinking among health professionals is one of active recovery. Bed rest is contraindicated, as it has been shown to not only delay recovery, but also worsen symptoms. Understanding a client's condition is essential for him or her to actively participate in improving current symptoms and preventing future recurrences.

The following sections present an overview of current treatment options, with a focus on non-surgical choices.

Non-pharmacologic Therapy

In the acute setting, clients often fear that the pain they feel with activity is a sign of further injury, or even permanent disability. However, clients can be reassured that returning to daily activities has actually been shown to be an integral part of treatment and recovery. Basic recommendations include avoiding specific movements or activities that provoke pain and limiting bed rest to times of severe pain only. Because there is little evidence that specific back exercises are useful in the acute setting, clients are generally encouraged to engage in low-stress aerobic activities such as walking. Heavy lifting and prolonged sitting and standing should be avoided. In cases of **subacute** and chronic LBP a physical therapist can design specific exercises that focus on conditioning the core musculature in an attempt to improve current symptoms and avoid future recurrences. Physical therapy serves to reeducate patients on proper posture and alignment as well as correct current muscle imbalances. Using popular exercise modalities, such as yoga and Pilates, as maintenance therapy may be appropriate for certain individuals. Exercise is covered in more depth later in the chapter.

Application of ice in patients with acute back pain is thought to decrease inflammation and swelling, while the rationale for heat application is to reduce muscle spasms. Other modalities such as ultrasound and **transcutaneous electrical nerve stimulation (TENS)** have conflicting evidence for effectiveness (Brosseau et al., 2002).

Many LBP sufferers turn to alternative therapies such as massage and acupuncture. While these therapies may aid in pain control for some individuals, they are not substitutes for exercise for ultimate symptom relief and prevention. However, as they generally have few side effects, those who are interested in these techniques may be encouraged to integrate massage or acupuncture into their treatment plans.

Spinal manipulation refers to adjustment of the spine using twisting, pushing, or pulling movements and is performed predominantly by chiropractors. Individuals interested in chiropractic services should have a thorough evaluation of their symptoms by their primary care physician and have an understanding of their diagnosis before choosing to try this modality.

Pharmacologic Therapy

Pain relievers such as acetaminophen (Tylenol®) and **nonsteroidal anti-inflammatory drugs (NSAIDs)** are often used in the short-term for acute cases of LBP. Narcotics and muscle relaxants are also options for pain, especially night-time symptoms, but their side effects and dependence profiles make them more appropriate for cases of severe pain. Occasionally, chronic pain may also be treated with antidepressants.

Neuropathy or neuropathic pain, which clients may experience as burning, "pins and needles," or "electric shock" sensations, can be treated with two newer drugs, gabapentin (Neurontin®) and pregabalin (Lyrica®). These medications work directly on the **central nervous system** and serve as nerve stabilizers. The mechanisms of action of gabapentin and pregabalin are unknown, but it is believed that they involve binding to voltage-dependent calcium ion channels.

Injections

Spinal injections, another option for chronic pain relief, can also be used as a diagnostic tool to localize symptoms. Typically, local anesthetics and/or **corticosteroids** are used to relieve pain and decrease inflammation. Injections can target several different structures:

- Trigger point injections target areas of muscle that are painful and fail to relax.
- Facet injections target facet joints on the posterior aspect of the spine that form where one vertebra overlaps another. Pain from facet joints can cause localized spinal pain or refer pain to adjacent structures.
- Epidural injections target the epidural space inside the spinal canal that contains, among other structures, spinal nerve roots.

These injections are thought to be particularly helpful for patients with radicular signs and symptoms such as sciatica. Sacroiliac joints are also occasionally injected in patients with accompanying buttock and thigh pain or sacroiliac dysfunction. Both facet and epidural injections can be done under **fluoroscopy,** or x-ray guidance, to ensure that the medications reach the correct location.

Surgery

Rarely, if conservative treatments have failed or patients develop progressive and limiting neurologic symptoms, spinal surgery is considered. The two most common types of lumbar surgery are decompression and spinal fusion. Decompression surgery, either a **laminectomy** or a **microdiscectomy,** aims to relieve impingement of the nerve root by removing a small piece of bone and/or disc material. These procedures, which are most commonly used for spinal stenosis or herniated discs, create a wider spinal canal and, therefore, more space for the spinal nerves. Spinal fusion surgery uses a bone graft to fuse two vertebral segments together. This, in turn, stops abnormal or excessive motion at the joint that is thought to be generating pain. Fusion is most often used for degenerative disc disease, spondylolysis, and spondylolisthesis.

Role of Exercise in Managing LBP

For most individuals with LBP, exercise will be the cornerstone of their treatment program. However, clients with LBP, especially chronic sufferers, may have developed fear-avoidance behaviors and negative beliefs about their abilities to exercise or perform certain activities. Therefore, clear communication between the ACE-certified Advanced Health & Fitness Specialist (ACE-AHFS) and the client is essential for facilitating a positive outcome from the exercise program. The overall goals of symptom relief and return to function must be discussed, and expectations must be set prior to beginning exercise. In some cases, complete resolution of all symptoms is not a realistic goal. In such cases, clients must view exercise as a way of overcoming their LBP rather than eradicating it. Discussing specific physical-activity objectives [e.g., work duties, **activities of daily living (ADL),** sport skills] allows the client and ACE-AHFS to create long-term goals.

Rehabilitation

Specific back exercises in the setting of acute LBP are generally not considered to be effective, and may even be detrimental to the healing process (Malmivaara et al., 1995). Instead, the exercise program for those with acute pain focuses on walking and resuming normal daily activities as soon as possible. While modifications to avoid strenuous activities such as running and heavy-lifting are reasonable, periods of immobility, including bed rest and prolonged sitting or standing, are not recommended.

Once the acute phase is over, exercise becomes a central focus of physical therapy. Chronic back pain sufferers can learn basic, life-long adjustments that both improve current symptoms and decrease the chance of recurrence. Examples include being mindful of maintaining posture, creating ergonomically correct work spaces, and committing to frequent movement and position changes when behind a desk or on a long car ride.

In the rehabilitation setting, the goal of physical therapy is to focus on specific exercises that are designed to increase strength, endurance, flexibility, and **aerobic capacity.** Physical therapy provides an opportunity to not only perform exercises, but also to learn correct technique, as exercise will be a life-long requirement for the back pain sufferer to maintain the benefits of therapy. In a meta-analysis of specific exercise strategies on outcomes in chronic LBP, programs that had the greatest impact on pain and function were individually designed, at least partially supervised, and included greater than 20 total hours (Hayden, van Tulder, & Tomlinson, 2005). The authors also found that stretching had the greatest impact on pain, while strengthening resulted in the greatest functional improvements.

Therapeutic exercises for LBP generally are classified as spinal flexion, extension, or stabilization. The theoretical goals of flexion exercises are to open the intervertebral **foramina** and facet joints, strengthen the abdominal muscles, and stretch the back extensors (Wendell, 2001). Extension exercises aim to improve motor coordination, strengthen the back extensors, improve mobility, and, perhaps, shift disrupted nuclear material to a more normal position (Borenstein, Wiesel, & Boeden, 2004). Isometric stabilization exercises promote abdominal strength and co-contraction of trunk muscles by a series of moves that ultimately result in a posterior pelvic tilt. While individual assessment of each client presenting with LBP is the key to a successful program, some commonalities among LBP sufferers exist. For example, while individuals without LBP generally have 30% greater spinal extensor strength than spinal flexor strength, those with LBP often have extensors that are weaker than flexors (Borenstein, Wiesel, & Boeden, 2004).

Aerobic fitness is thought to impede the onset of LBP, as several studies show a link between low levels of physical activity and symptoms of back pain (Leboeuf-Yde, 2000). The exact mechanism by which aerobic fitness improves overall spine health is not known. One theory is that aerobic exercise leads to increased diffusion of nutrients into the avascular space of the intervertebral disk. In addition, increases in the capillary network of the surrounding tissues as a result of aerobic fitness may increase disc nutrition (Borenstein, Wiesel, & Boeden, 2004). Aerobic exercise may also increase pain tolerance by increasing endorphins, which leads to a decreased incidence of depression. Within the first two weeks of an acute painful attack, the individual should begin a regimen of low-stress aerobic exercise. Activities such as walking, stationary biking, and swimming are good initial options for increasing physical stamina.

Core stability training is emphasized in LBP programs. The trunk, or core, of the body refers to the musculoskeletal structures associated with the cervical, thoracic, and lumbar spine, as well as the shoulder and pelvic girdles. Exercise training aims to develop the strength, coordination, and endurance of the core muscles, which in turn leads to more stability and efficiency during dynamic movements. Individuals without these competencies have excess movements in individual vertebrae and are more likely to generate greater forces, which can lead to low-back injury.

Wendell (2001) describes three stages of exercise programs: centralization, lumbar stabilization, and dynamic stabilization. Centralization is said to occur when active motion of the lumbar spine causes symptoms to either resolve or move from the periphery to the lumbar spine. Symptoms most commonly localize with either flexion, usually in individuals with degenerative or stenotic conditions, or extension, usually in individuals with lumbar disc herniation. For these patients, the rehabilitation program can focus on exercises using the centralizing movement (either flexion or extension) with the goals of decreasing the severity of symptoms and increasing activity level.

Much debate exists regarding the efficacy of choosing these types of patient-specific exercises over using a generalized program for all patients with LBP. A randomized control trial by Long, Donelson, and Fung (2004) placed 230 subjects in three separate groups after they were assessed for centralization to determine their "directional preference." Subjects were taught lumbar exercises that either matched their directional preference or opposed their directional preference, or were given a generalized program of commonly used exercises. The group that performed matched exercises had more symptom improvement and higher activity levels, among other variables. The authors concluded that these patient-specific exercises were most effective in providing pain control, thus giving weight to the popular clinical practice of creating tailored treatment plans, especially in the centralization, or early phase, of rehabilitation (Long, Donelson, & Fung, 2004).

In the second stage, lumbar stabilization, the goals of treatment are to further increase activity level and decrease disability. Exercises focus on the muscles that provide support and stabilization of the lumbar spine: transverse abdominis, erector spinae, multifidus, quadratus lumborum, and oblique abdominals. While each muscle has a specific function, they all work in concert to stabilize the lumbar spine during everyday activities.

This concept of lumbar instability causing LBP is based on the hypothesis that instability is a result of three interdependent components: structural changes, muscular changes, and ineffective neural control (Barr, Griggs, & Cadby, 2007). Bone and ligamentous structures provide passive restraint toward the end of the **range of motion (ROM)**, muscles provide support and stiffness at the intervertebral level to sustain common forces, and the neural control system coordinates muscle activity to respond to expected and unexpected forces. Instability can be a result of damage to tissues, insufficient muscular strength or endurance, or poor muscular control—or a combination of all three. Lumbar-stabilization programs are based on the belief that subjects with LBP have different biomechanics than those without back pain, such as dysfunctional superficial and deep stabilizing muscles. Studies have found that subjects with these biomechanical deficits include decreased proprioception, balance, and reaction times (Barr, Griggs, & Cadby, 2007).

Finally, patients progress to dynamic stabilization after developing core strength and endurance in stage 2. The goals of dynamic stabilization therapy are to return the client to full activity. Exercises in this stage are performed on an unstable surface such as a stability ball. Exercises can progress through balancing on a ball while performing upper- and lower-extremity movements holding weights to standing exercises incorporating a trampoline, wobble board, or foam roller. In this stage, clients are reminded to engage their core muscles as learned in stage 2 to maintain spinal stability while performing all dynamic exercises.

The final stages of rehabilitation are geared toward previously set activity goals. The unique actions used in the client's daily activities or desired sports are incorporated into the therapeutic exercises. The movements may be more complex, encompassing sport-specific actions such as catching, throwing, swinging, and lifting.

Post-rehabilitation

Creating an individualized exercise program for a person with a history of LBP requires consideration of his or her specific clinical diagnosis. Initially, an assessment of the client's overall fitness level is also important, as an athlete will have a different starting point than a **sedentary** person. Additionally, understanding the duration and severity of symptoms, as well as any activity limitations, will allow the ACE-AHFS to establish a baseline from which to observe progress.

Precautions

The following are contraindications and modifications to consider when designing an exercise program for clients with a history of LBP:

- Clients should be encouraged to obtain clearance from their physicians before beginning a program, as exercise may not be appropriate for certain individuals with serious conditions such as tumor, fracture, or progressive neurologic deficits.
- Although walking is generally a good choice for aerobic fitness in clients with LBP because it places low compressive loads on the lumbar structures, it may not be suitable for all clients. Because walking places the lumbar spine in a more extended position, clients with spinal stenosis who have symptoms while walking that are relieved with rest, should avoid prolonged walking.
- As previously described, the progression of exercises can be as important as the exercises themselves. In general, the ACE-AHFS should consider working on the muscles that stabilize the spine prior to the muscles that move the spine, to decrease the likelihood that unsupported exercises will cause damage to ligaments.
- Keep in mind that clients in beginning stages may not need additional weight added to exercises. Initially, the weight of their limbs may provide enough of a challenge.
- Know each client's limitations as set forth by his or her physician or physical therapist prior to designing the exercise program. Avoid extreme postures or actions that take the individual beyond his or her normal range of motion. Although creativity is an important motivational factor when designing a program, performing exercises outside of normal body mechanics is not useful and may be detrimental.
- Respect the client's normal spinal curvature when performing trunk exercises. Avoid hyperextension of the spine, which would cause clients to exceed their normal **lordosis.**
- Many of the exercises used in LBP rehabilitation require only subtle movements, such as **abdominal hollowing.** Using extreme movements or momentum is usually unwarranted for individuals with a history of LBP.
- Although a hands-on approach can be beneficial to provide adjustments and guidance, the ACE-AHFS must never physically force a client into a position. Providing extra force to bring a client "deeper" into a stretch can cause serious injury.

Exercise Program Guidelines and Considerations

The balance of this chapter is based on the work of Stuart M. McGill, Ph.D., a renowned expert in spine biomechanics and kinesiology whose books *Low Back Disorders,* Second Edition, and *Ultimate Back Fitness and Performance,* Third Edition, can be purchased through his website: www.backfitpro.com.

Clients with a history of back troubles may desire pain relief and spinal stability (a health objective), while others may seek a performance objective (which may be counter-productive to optimal back health). Some clients may need more stability, while others may need more mobility. Certain exercises will exacerbate the back troubles of some people but may help others. Because each individual has different needs, proficient exercise professionals will need an understanding of the issues, and of the myths and realities pertaining to each issue, to form a foundation for the decision-making process.

The scientific foundation for many "common sense" recommendations offered for back health yield no, or very thin, evidence. For example, it is widely believed that stretching the back and increasing the ROM is beneficial and reduces back problems—however, the scientific evidence shows that, on average, those who have more range of motion in their backs have a greater risk of future troubles (Biering-Sorensen, 1984). Clearly there is a tradeoff between mobility and stability; the optimal balance is a very personal and individual variable. Indeed, the "stability/mobility balance" may shift during a progressive exercise program as symptoms resolve, with advancing age, or as rehabilitation or training objectives change.

Another generally perceived goal of training the back is to increase strength. Strength has little association with low-back health (Biering-Sorensen, 1984). In fact, many people hurt their backs in an attempt to increase strength. It could be argued that this is an artifact, in that some exercise programs intended to enhance strength contained poorly chosen exercises such as sit-ups. Performing sit-ups both replicates a potent injury mechanism (i.e., posterior disc herniation) and results in high loads on the spine. On the other hand, muscle endurance, as opposed to strength, has been shown to be protective against future back troubles (Luoto et al., 1995). Further, for many people, it is better to train for stability rather than stretching to increase range of motion (Saal & Saal, 1989). Investigations into injury mechanisms have revealed that many back-training practices actually replicate the loads and motions that cause parts of the low back to become injured (Axler & McGill, 1997). For example, disc herniations need not have excessive loading on the back to occur; rather, repeated forward flexion motion of the spine is a more potent mechanism. Thus, if full flexion or deviation is avoided in the spine, the risk of herniation is remote.

Injury is caused by damage to supporting tissues. This damage reduces the normal stiffness in the spine, resulting in unstable joints. Thus, while injury results in joint instability, an event characterized by improper muscle activation can cause the spine to "buckle" or become unstable. There is no question that excessive loading can lead to back injury, but instability at low loads is also possible and problematic. For example, it is possible to damage the passive tissues of the back while bending down and picking up a pencil, if sufficient stability is not maintained. Some people recommend that when training, the client should exhale upon exertion. In terms of grooving stabilizing motor patterns for all tasks, this is a mistake. Breathing in and out should occur continuously, and not be trained to a specific exertion effort. This continuous breathing helps the client maintain constant abdominal muscle activation and ensure spine stability during all possible situations.

Further, specific muscle-activation patterns are essential to avoid injury, but have also been documented to become perturbed following injury (Hodges & Richardson, 1996). Pain is a powerful instigator in the deprogramming of normal/healthy motor patterns and the creating of perturbed patterns. The exercises and programs described in this section are based on the latest scientific knowledge of how the spine works and how it becomes injured. In addition, they have been quantified for spine load, resultant spine stability, and muscle oxygenation. These are only a few examples to begin a program. The goals are to enhance spine stability by grooving motion and muscle-activation patterns to prepare for all types of challenges. Of course, other exercises may be required subsequently to enhance daily functioning, but once again, these will depend upon the characteristics and objectives of the individual.

Two other concepts must be emphasized. First, training approaches intended to enhance athletic performance are often counterproductive to the approaches used when training for health. Too many patients are rehabilitated using athletic philosophies or, worse yet, "bodybuilding" approaches designed primarily to isolate and **hypertrophy** specific muscles, and progress is thwarted. Many bad backs are created due to inappropriate performance philosophies. Identifying the training objectives is paramount.

The emphasis should be on enhancing spine health; training for performance is another topic. Second, many of the training approaches that are used at joints such as the knee, hip, or shoulder are mistakenly applied to the back. The back is a very different and complex structure, involving a flexible column with complex muscle and ligamentous support. The spine contains the spinal cord and lateral nerve roots, and musculature intimately involved in several other functions, including breathing mechanics, to give just one example. Many of the traditional approaches for training other joints in the body are not appropriate for the back—either they do not produce the desired result or they create new LBP patients.

Caveats for Designing Exercise Programs for Back Health

- While there is a common belief that exercise sessions should be performed at least three times per week, it appears low-back exercises have the most beneficial effect when performed daily.
- The "no pain, no gain" axiom does not apply when exercising the low back in pained individuals, particularly when applied to weight training.
- General exercise programs that combine cardiovascular components (like walking) with specific low-back exercises have been shown to be more effective in both rehabilitation and for injury prevention. The exercises shown in this chapter comprise only a component of the total program.
- Diurnal variation in the fluid level of the intervertebral discs (i.e., discs are more hydrated early in the morning after rising from bed) changes the stresses on the discs throughout the day. Specifically, they are highest following bed rest and diminish over the subsequent few hours. It would be very unwise to perform full-range spine motion while under load shortly after rising from bed.
- Low-back exercises performed for maintenance of health need not emphasize strength; rather, more repetitions of less-demanding exercises will assist in the enhancement of endurance and strength. There is no doubt that back injury can occur during seemingly low-level demands (such as picking up a pencil) and that motor control error can increase the risk of injury. While it appears that the chance of motor control errors, which can result in inappropriate muscle forces, increases with fatigue, there is also evidence documenting the changes in passive tissue loading with fatiguing lifting. Given that endurance has more protective value than strength, strength gains should not be overemphasized at the expense of endurance.
- There is no such thing as an ideal set of exercises for all individuals. An individual's training objectives must be identified (be they rehabilitation specifically to reduce the risk of injury, optimize general health and fitness, or maximize athletic performance), and the most appropriate exercises chosen. While science cannot evaluate the optimal exercises for each situation, the combination of science and clinical experiential "wisdom" must be utilized to enhance low-back health.
- Clients should be encouraged to be patient and stick with the program. Increased function and pain reduction may not occur for three or more months.

Exercise Program Progressions

In Dr. McGill's *Ultimate Back Fitness and Performance,* Third Edition, a variety of LBP prevention solutions and training progressions are presented. This evidence-based approach for enhancing back health involves five distinct stages, beginning with corrective exercise and building a foundation and ultimately progressing to high-performance training.

The Five Stages for Building the Ultimate Back

According to McGill, building the ultimate back consists of a five-stage process that ensures a foundation for eventual strength, speed, and power training. The five stages are as follows:

- Groove motion/motor patterns, and corrective exercise
- Build whole-body and joint stability
- Increase muscle endurance
- Build muscle strength
- Develop power and agility

Stage 1: Groove Motion/Motor Patterns and Corrective Exercise

The first stage involves identifying disrupted motion patterns and developing appropriate corrective exercises. Determining where to begin the process of finding the most appropriate and suitable exercises for a client with a history of LBP starts with an assessment of the client's current fitness status and an evaluation of which, if any, movements produce pain.

A natural consideration when selecting appropriate exercises is to determine a course of action if an exercise or movement produces pain. Any exercise that causes pain is inhibiting and detracts from proper exercise technique or form. Attempting to "work through the pain" with the back is almost never beneficial. If an individual has pain, he or she is probably doing the exercise incorrectly, or more likely, doing the wrong exercise. The ACE-AHFS should prompt clients to describe tasks, postures, and movements that exacerbate their LBP. After determining which movements are problematic for a client, the ACE-AHFS should develop an exercise program to minimize the exacerbating movements.

Proper motor patterns of the muscles that support the spine enhance the back's ability to withstand the various loads and directional forces that it encounters during daily activities and physical exertion. A client's awareness of his or her lumbar spine, **abdominal bracing,** and gluteal complex activation are all elements in improving motor patterns for appropriate spine function.

An awareness of proper spine position allows a client to adopt a neutral spine wherein the tissues supporting the vertebrae have minimal elastic stress. Teaching clients how to adopt and maintain a neutral spine is one of the first important lessons for those with LBP. Neutral spine may be modified by clients who find relief with slightly more lumbar flexion or extension. This modified position becomes their neutral spine.

Abdominal bracing, or maintaining a mild contraction of the abdominal wall, can help ensure sufficient spine stability. Many fitness professionals mistakenly believe that activating the transverse abdominis and intentionally sucking in the abdominal wall toward the spine (a technique known as hollowing) increases spinal stability and is therefore helpful for back-pain sufferers. However, training the transverse abdominis in this manner actually compromises stability and creates spine dysfunction.

It is not uncommon for individuals to possess sufficient levels of torso flexion and extension strength, but fail tests indicative of torsional, or rotational, control. A simple, low-level test for torsional control starts in the modified push-up position (Figure 20-1a), with one hand placed directly over the other hand (Figure 20-1b). The client's pelvis and ribcage should remain locked throughout the movement. An elevated pelvis, as illustrated in Figure 20-1c, is indicative of poor lumbar torsional control. It would be contraindicated to recommend a rotational cable-pulling exercise, such as the wood-chop exercise, to a client with poor torsional control because of the high level of twisting torque it produces.

For many individuals, learning to "lock" the ribcage on the pelvis is essential for enhancing torsional control, resulting in a reduced potential for injury and optimal functional performance during rotational or twisting movements. Locking the ribcage on the pelvis involves abdominal bracing—or the co-contraction of the abdominal wall muscles without pulling in the navel.

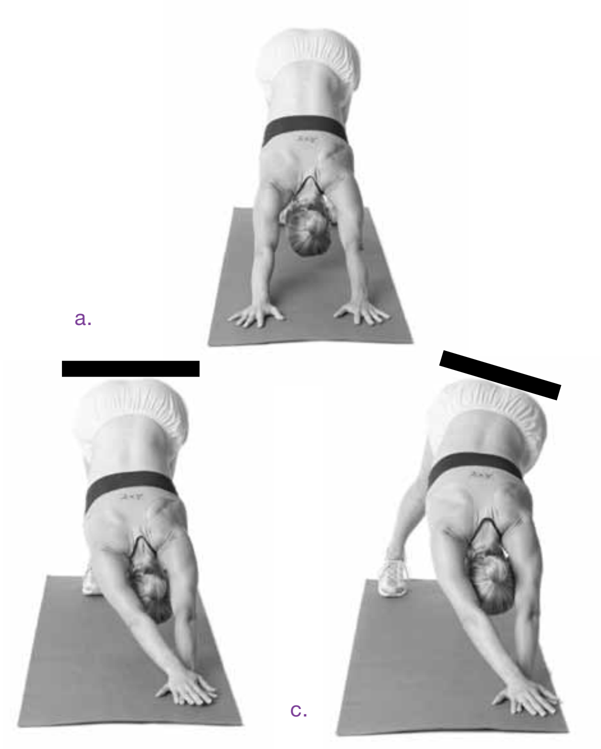

a. b. c.

Figure 20-1
Test for lumbar torsional control

To enhance lumbar torsional control, Dr. McGill developed and used an exercise progression that begins with the wall roll exercise to improve an individual's level of torsional control. The wall roll begins with the client in the plank position with both elbows planted on the wall (Figure 20-2a). The abdominals are braced and the ribcage is locked on the pelvis. The client pivots on the balls of his or her feet while pulling one elbow off the wall (Figure 20-2b). No spine motion should occur throughout the movement.

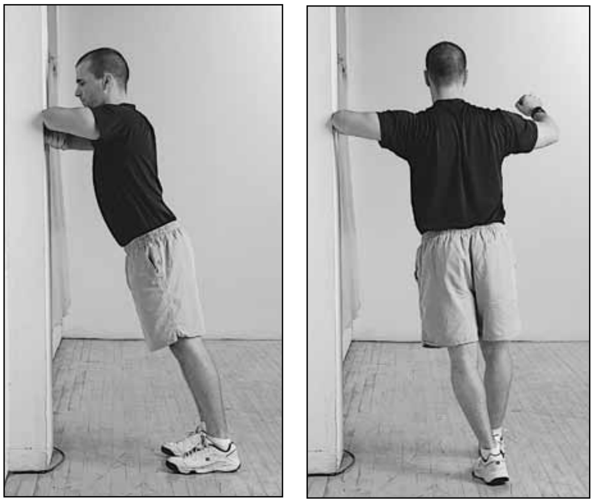

a. Starting position b. Ending position

Figure 20-2
Wall roll

The torsional control exercise progression continues with the floor roll and scramble-up exercise. This exercise begins with the client in contact with the floor with both hands and feet (Figure 20-3a). The ribcage is locked to the pelvis with an abdominal brace. The client pivots, rolls completely (360 degrees), and eventually moves into a sprinting or bounding motion (Figures 20-3b and 20-3c). A neutral spine should be maintained throughout the floor roll and scramble-up movements—no spine motion should occur.

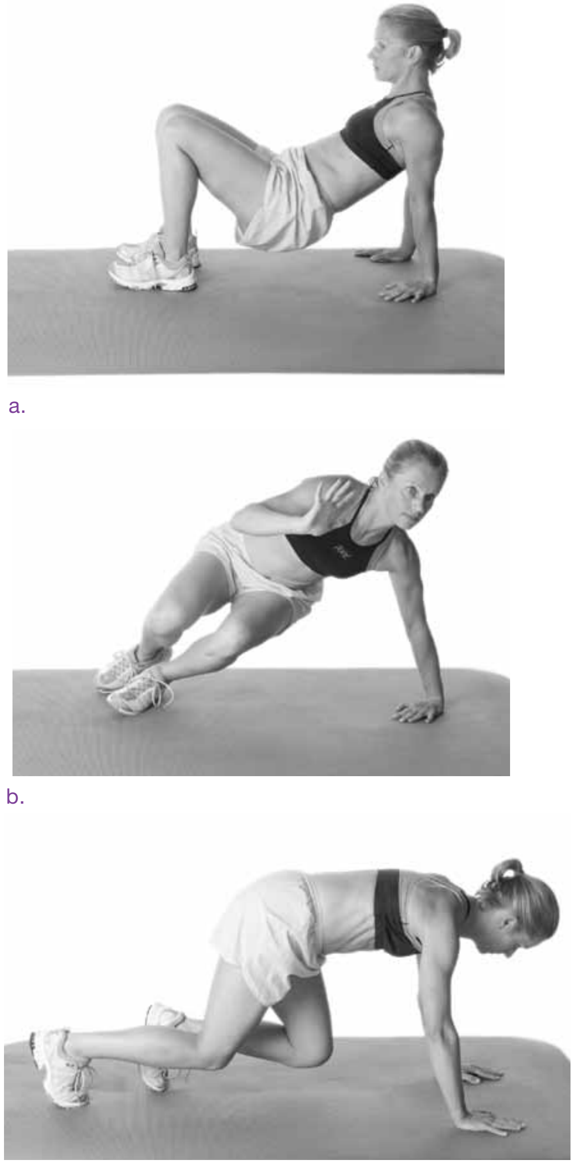

a. b. c.

Figure 20-3
Floor roll and scramble-up exercise

A healthy back also requires healthy gluteal muscle function, since the performance and safety of many movements are dependent on balanced hip power production. The "crossed-pelvis syndrome" is a phenomenon that occurs when the gluteal complex is inhibited during squatting patterns. It is very common in individuals with a history of back problems. Interestingly, it is not known if the crossed-pelvis syndrome exists prior to back problems or is a consequence of having back problems. Regardless of whether the syndrome is a cause or an outcome, individuals with impaired gluteal motor patterns are unable to spare their backs during squatting patterns since they rely on their hamstrings and erector spinae to drive the extension motion. In turn, the erector spinae forces create loads that compress the lumbar spine. It is impossible to achieve optimal squat performance without well-integrated hip-extensor or gluteal patterns. One of the more common reasons individuals fail to properly rehabilitate their backs is due to an emphasis on strength development without first effectively addressing the impaired gluteal patterns.

Retraining of the gluteals cannot be achieved with conventional barbell squat exercises. Performing a conventional squat requires relatively little hip abduction. As a result, gluteus medius activation is minimized and activation of the gluteus maximus is delayed during the traditional barbell squat until lower squat angles are reached. McGill argues, as many other experts do, that the barbell squat is primarily a quadriceps exercise and not a gluteal exercise in the truest sense. Unlike the conventional barbell squat, the single-leg squat elicits almost immediate activation of the gluteus medius and more rapid integration of the gluteus maximus during the squat descent to assist in the **frontal plane** hip drive needed for common activities such as running and jumping.

The ACE-AHFS should begin by instructing unfit clients to learn how to activate the gluteus medius by performing the clamshell exercise. During the clamshell exercise, the client lies on his or her side while anchoring the thumb on the anterior superior iliac spine and reaching around with the fingertips, positioning them to land on the gluteus medius. Opening the knees like a clamshell will allow the individual to feel the gluteus medius activation (Figure 20-4).

The single-leg squat matrix is an example of a more advanced corrective exercise for retraining the gluteals. During the single-leg squat matrix progressions with the leg to the front (Figure 20-5a), side (Figure 20-5b), and rear (Figure 20-5c), the

Starting position

Ending position

Figure 20-4
Beginner-level corrective exercise for retraining the gluteals—clamshell exercise

a.

b.

c.

Figure 20-5
Advanced-level corrective exercise for retraining the gluteals—single-leg squat matrix

abdominals are braced, the lumbar spine is neutral, and the mental focus of the individual should be on the development of hip torque.

Stage 2: Build Whole-body and Joint Stability—The "Big Three"

McGill recommends that after individuals successfully learn how to groove motion and motor patterns, they should begin focusing on developing whole-body and joint stability. An exercise program consisting of the "big three" (modified curl-ups, side bridge, and birddog) is an excellent choice for spine stabilization during the early stages of training or rehabilitation and for simply enhancing low-back health.

The modified curl-up is an exercise for the rectus abdominis. In a supine position, the client places the hands or a rolled towel under the lumbar spine and flexes one knee. He or she then raises the head and shoulders off the floor while maintaining a neutral spine, pauses, and then returns to the starting position (Figure 20-6). Clients should not flatten the back to the floor, as flattening the back flexes the lumbar spine, eliminates the neutral spine, and increases the loads on the discs and ligaments. By flexing one knee and keeping the other one straight, a neutral lumbar spine is maintained throughout the exercise movement. The bent leg should be alternated midway through each set of repetitions.

The lateral muscles of the torso (quadratus lumborum and abdominal obliques) are important for optimum spinal stability, and are targeted with the side bridge exercise. The beginner level of this exercise involves bridging the torso between the elbow and the knees (Figure 20-7). Once this is mastered and well tolerated, the challenge can be increased by bridging between the elbow and the feet (Figure 20-8). An even more advanced variation involves placing the upper leg and foot in front of the lower leg and foot to facilitate longitudinal "rolling" of the torso (Figures 20-9). This variation is far superior to exercises such as performing a sit-up with a twist because it produces greater levels of muscle activation with lower tissue loads.

Figure 20-7
Modified side bridge

Figure 20-8
Side bridge

Figure 20-6
Curl-up

The birddog is a safe and effective exercise for developing the spinal extensors. It is performed by extending one leg and the opposite arm, from an all-fours position, so that they are parallel to the floor (Figure 20-10). The extended position should be held for seven to eight seconds, and then repeated with the opposite arm and leg.

More challenging exercise progressions for developing whole-body and joint stability include performing the conventional push-up with a staggered hand placement or with labile balls under the hands (Figures 20-11 and 20-12). These advanced exercises facilitate torso stabilization. As with any exercise, abdominal bracing and good torsional control should be maintained throughout the activity.

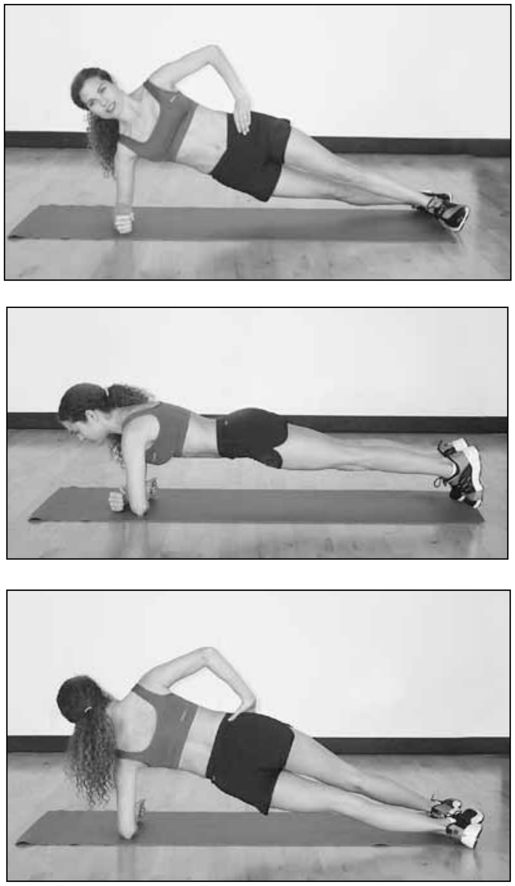


Figure 20-9
Rolling bridge

Figure 20-10
Birddog

Figure 20-11
Performing the push-up with staggered hand placement (one hand forward of the shoulder and one hand beside the lower ribs) enhances shoulder and abdominal muscle activation, thereby promoting the stabilizing functions of these muscles.

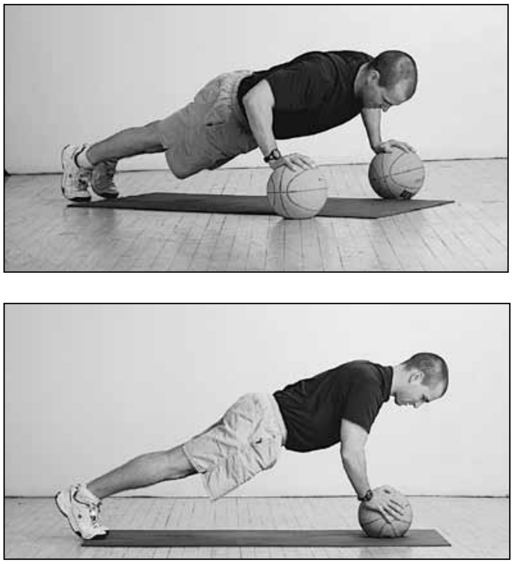


Figure 20-12
Placing both hands on a single ball or each hand on one ball while performing a push-up significantly enhances the rectus abdominis and internal and external obliques activation on both sides of the torso.

Stage 3: Increase Muscle Endurance

During stage 3, the focus is on improving muscle endurance. Endurance is typically developed first with repeated sets or repetitions of exercises. The client then progresses to longer-duration workouts for an overall increase in total work volume. Endurance progression should generally begin with isometric holds, such as curl-ups, side bridges, and birddog exercises. It is recommended that individuals continue to progress by increasing the number of repetitions completed rather than extending the "hold time."

The "reverse pyramid for endurance training" is an approach to designing endurance sets based on the Russian tradition of maintaining excellent exercise technique and form. For example, an endurance workout involving the side bridge exercise might consist of:

- Five repetitions on the right side followed by five on the left
- Rest
- Four repetitions on the right side followed by four on the left
- Rest
- Three repetitions on the right side followed by three on the left

The basic rationale behind this approach is that the exerciser does as much as possible while he or she is least fatigued. That is, it is easier to maintain proper exercise technique as the repetitions are reduced with each fatiguing set.

Stage 4: Build Strength

The fourth stage in the progression to building the ultimate back emphasizes strength development. Training for maximum back strength without injury is a difficult challenge that requires a delicate balance. A one-size-fits-all recipe for back-strength development does not exist. However, several basic considerations exist for strengthening the back:

- Develop general fitness and balancing ability to train safely and effectively
- Consider the matching of a client's fitness level and motor abilities to the skill demands of the planned training program
- Develop the foundation of proper motor and motion patterns to protect any potential weak links
- Consider the balance of strength around a joint and between adjacent joints, as well as the balance of strength to endurance
- Consider the range of motion required by the task and whether the client's motion capability is appropriately matched

Stage 5: Power

Power development represents the fifth and final stage in Dr. McGill's recommended progression to building the ultimate back. Developing spine power can potentially compromise both safety and performance. Power is the product of force and velocity.

Power = Force x Velocity

Therefore, if either force or velocity is high, then the other should generally be kept low. Power is developed in the extremities and transferred through the torso. Efficient and effective power transfer through the torso requires spine posture control, spine stiffness and stability, and strength.

For clients who have successfully progressed through the first four stages, abdominal plyometric exercises can be incorporated into the training programs to enhance their abilities to effectively and efficiently transfer power through their torsos. The medicine ball toss, performed either while standing or while supine on the floor or a stability ball, is an example of an effective abdominal plyometric exercise that provides excellent progression to power (Figure 20-13).

Figure 20-13
Medicine ball toss

Case Study

Amy, a 37-year-old overweight female with chronic low-back pain has decided to begin an exercise program to improve her overall conditioning and lose weight on the advice of her physician and physical therapist. She has no previous exercise history and has become increasingly inactive due to her low-back pain. Physical therapy reduced her symptoms to localized discomfort and she is afraid of making her back worse again by doing the wrong things at the gym. Amy was released from therapy with a home exercise program that includes supine pelvic tilts, modified curl-ups, several dynamic stabilization exercises (e.g., modified side bridge, modified birddog), wall squats, hamstring and piriformis stretches, and 10 minutes of cycling. She has hired an ACE-AHFS to help her achieve her weight-loss goal without exacerbating her low-back pain.

All the exercises on Amy's home exercise program are clearly described with illustrations, verbal cues, and intensity and frequency guidelines. Additional programming is developed by the ACE-AHFS to address Amy's conditioning and weight-loss goals. The ACE-AHFS initially tries aerobic exercise on a recumbent cycle; however, Amy reports some increased pain with prolonged sitting. Several other modes of activity are then tested, including stair climbers and elliptical trainers. Treadmill walking on a cushioned deck provides the most comfortable aerobic exercise for Amy and is chosen as the primary modality. The speed is kept below Amy's stride maximum to ensure that she can maintain postural alignment throughout her workout. Incline is introduced as tolerated by her conditioning as well as her low back.

A total-body strength program supplements the aerobic work for weight loss and promotes more complete conditioning. Initially, weights are low and repetitions are moderate to high due to Amy's deconditioned state and to promote her learning of exercise technique. Supine or standing exercises replace seated exercises whenever possible. In the event that a seated exercise is used, posture is closely monitored. Strengthening of the core muscles is emphasized, along with stability of the lower back. Back extension exercises are cautiously introduced because Amy's physical therapist did not recommend them and it is not clear if they are appropriate. Any complaints of pain, weakness, radicular symptoms, or increased low-back discomfort indicate that a given exercise should be discontinued.

Stretching concentrates on the iliopsoas, hamstrings, piriformis, gluteal complex, quadriceps, and quadratus lumborum.

As Amy progresses, the frequency, intensity, and duration will increase as tolerated. Her low-back comfort is continuously monitored. More functional exercises can be introduced as her fitness improves. Proper exercise technique and postural maintenance are always a critical concern during Amy's workouts.

Summary

LBP is a significant source of cost and disability, as more than 80% of Americans suffer from at least one episode of back pain during their lifetimes. A significant proportion of these individuals seek assistance from a healthcare professional. While the majority of acute back pain improves over time, some people develop recurrences, and still others experience continuous pain. The onset and persistence of LBP is related to individual, activity, and psychological factors.

Mechanical causes account for the majority of LBP cases. Discogenic pain can be attributed to conditions such as a herniated disc, sciatica, spinal stenosis, spondylolysis, and spondylolisthesis. Treatment options for individuals with LBP have evolved and, in some cases, become a source of much debate. However, for almost all conditions, current thinking among health professionals is one of active recovery. Exercise is a central focus in the treatment of LBP.

The back is a very unique and complex structure involving a flexible column with complex muscle and ligamentous support. The spine contains the spinal cord and lateral nerve roots, and musculature intimately involved in several other functions (e.g., breathing mechanics). Many of the traditional approaches used to train other joints in the body are not appropriate for the back—either they fail to produce positive results or they result in injury. The ACE-AHFS can play a vital role in the prevention of LBP and maintenance of back health through designing evidence-based exercise programs that address the important structures and functions of the spine.

References

Anderson, G.B.J. (1997). The epidemiology of spinal disorders. In: Froymoyer J.W. (Ed.) *The Adult Spine: Principles and Practice* (2nd ed.). Philadelphia: Lippincott Raven.

Axler, C. & McGill, S.M. (1997). Low back loads over a variety of abdominal exercises: Searching for the safest abdominal challenge. *Medicine & Science in Sports & Exercise,* 29, 6, 804–811.

Bahr, R. et al. (2004). Low back pain among endurance athletes with and without specific back loading: A cross-sectional survey of cross country skiers, rowers, orienteers, and nonathletic controls. *Spine,* 29, 449–454.

Barr, K.P., Griggs M., & Cadby, T. (2007). Lumbar stabilization: A review of core concepts and current literature, part 2. *American Journal of Physical Medicine and Rehabilitation,* 86, 72–80.

Biering-Sorensen, F. (1984). Physical measurements as risk indicators for low-back trouble over a one-year period. *Spine,* 9, 106–119.

Borenstein, D., Wiesel, S., & Boeden, S. (2004). *Low Back and Neck Pain: Comprehensive Diagnosis and Management* (3rd Ed.). Philadelphia: Elsevier.

Bressler, H.B. et al. (1999). The prevalence of low back pain in the elderly: A systematic review of the literature. *Spine*, 24, 1813–1819.

Brosseau L. et al. (2002). Efficacy of the transcutaneous electrical nerve stimulation for the treatment of chronic low back pain. *Spine,* 27, 596–603.

Carey, T.S. & Garrett, J.M. (1999). Recurrence and care seeking after acute back pain: Results of a long-term follow-up study. *Medical Care,* 37, 157–164.

Currie, S.R. & Wang, J.L. (2004). Chronic back pain and major depression in the general Canadian population. *Pain*, 107, 54–60.

Deyo, R.A. & Weinstein, J.N. (2001). Primary care: Low back pain. *New England Journal of Medicine,* 344, 363–370.

Deyo, R. et al. (2006). Back pain prevalence and visit rates. *Spine*, 31, 2724–2727.

Hayden, J., van Tulder, M., & Tomlinson, G. (2005). Systematic review: Strategies for using exercise therapy to improve outcomes in chronic low back pain. *Annals of Internal Medicine,* 142, 776–785.

Hodges, P.W. & Richardson, C.A. (1996). Inefficient muscular stabilization of the lumbar spine associated with low back pain. *Spine,* 21, 2640–2650.

Hurwitz, E.L. & Morgenstern, H. (1997). Correlates of back problems and back related disability in the United States. *Journal of Clinical Epidemiology,* 50, 669–681.

Leboeuf-Yde, C. (2000). Body weight and low back pain. *Spine,* 25, 2, 226–237.

Long, A., Donelson, R., & Fung T. (2004). Does it matter which exercise? A randomized control trial of exercise for low back pain. *Spine,* 29, 23, 2593–2602.

Luo, X. et al. (2004). Estimates and patterns of direct health care expenditures among individuals with back pain in the United States. *Spine,* 29, 79–86.

Luoto, S. et al. (1995). Static back endurance and the risk of low back pain. *Clinical Biomechanics*, 10, 323–324.

Malmivaara, A. et al. (1995). The treatment of acute low back pain: Bed rest, exercises, or ordinary activity? *New England Journal of Medicine,* 332, 351.

Manek N., & MacGregor, A.J. (2005). Epidemiology of back disorders: Prevalence, risk factors and prognosis. *Current Opinion in Rheumatology,* 17, 134–140.

McGill, S. (2008). *Ultimate Back Fitness and Performance* (3rd ed.). Waterloo, Ont.: Backfitpro Inc.

McGill, S. (2007). *Low Back Disorders* (2nd ed.). Champaign, Ill.: Human Kinetics.

Morris C. (2006). *Low Back Syndromes.* New York: McGraw-Hill.

Rubin, D. (2007). Epidemiology and risk factors for spine pain. *Neurologic Clinics,* 25, 353–371.

Saal, J.A. & Saal, J.S. (1989). Nonoperative treatment of herniated lumbar intervertebral disc with radiculopathy: An outcome study. *Journal of Biomechanics,* 14, 431–437.

Wendell, L. (2001). *Exercise Presciption and the Back*. New York: McGraw-Hill.

Wong, D & Transfeltd, E. (2007). *Macnab's Backache* (4th ed.). Philadelphia: Lippincott Williams & Wilkins.

Yu, C. & Garfin, S.R. (1994). Recognizing and managing lumbar spondylolisthesis. *Journal of Musculoskeletal Medicine,* 11, 55–63.

Suggested Reading

Barr, K.P., Griggs M., & Cadby, T. (2007). Lumbar stabilization: A review of core concepts and current literature, part 2. *American Journal of Physical Medicine and Rehabilitation,* 86, 72–80.

Barr, K.P., Griggs M., & Cadby T. (2005). Lumbar stabilization: A review of core concepts and current literature, part 1. *American Journal of Physical Medicine and Rehabilitation,* 84, 473–480

Borenstein, D., Wiesel, S., & Boeden, S. (2004). *Low Back and Neck Pain: Comprehensive Diagnosis and Management.* Philadelphia: Elsevier.

Deyo, R.A. & Weinstein, J.N. (2001). Primary care: Low back pain. *New England Journal of Medicine,* 344, 363–370.

Malmivaara, A. et al. (1995). The treatment of acute low back pain: Bed rest, exercises, or ordinary activity? *New England Journal of Medicine,* 332, 351.

Morris C. (2006). *Low Back Syndromes.* New York: McGraw-Hill.

Rubin, D. (2007). Epidemiology and risk factors for spine pain. *Neurologic Clinics,* 25, 353–371.

Saal, J.A. & Saal, J.S. (1989). Nonoperative treatment of herniated lumbar intervertebral disc with radiculopathy: An outcome study. *Journal of Biomechanics,* 14, 431–437.

Wendell, L. (2001). *Exercise Presciption and the Back.* New York: McGraw-Hill.

Wong, D & Transfeltd, E. (2007). *Macnab's Backache* (4th ed.). Philadelphia: Lippincott Williams & Wilkins.

Yu, C. & Garfin, S.R. (1994). Recognizing and managing lumbar spondylolisthesis. *Journal of Musculoskeletal Medicine,* 11, 55–63.