CHAPTER 42 Nursing Care of Clients with Musculoskeletal Disorders

LEARNING OUTCOMES

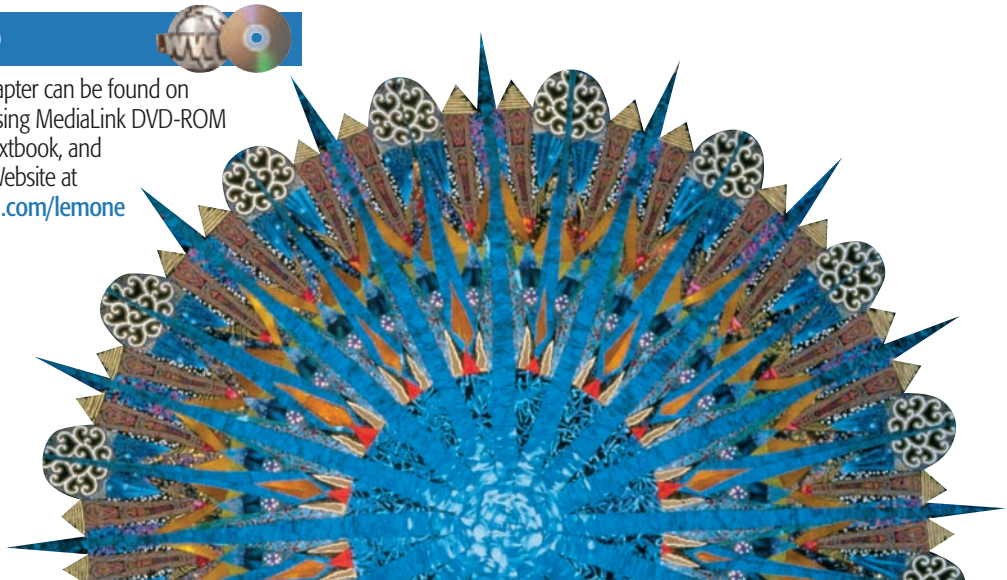
- Explain the pathophysiology, manifestations, complications, interdisciplinary care, and nursing care of metabolic, degenerative, autoimmune, inflammatory, infectious, neoplastic, connective tissue, and structural musculoskeletal disorders.
- Compare and contrast the pathophysiology, manifestations, diagnosis, and treatments for osteoporosis, osteoarthritis, Paget's disease, and rheumatoid arthritis.
- Discuss the purposes, nursing implications, and health education for the client and family for medications used to treat osteoporosis, Paget's disease, gout, osteomalacia, osteoarthritis, rheumatoid arthritis, systemic lupus erythematosus, osteomyelitis, bone tumors, scleroderma, and low back pain.
- Describe the surgical procedures used to treat clients with arthritis.

CLINICAL COMPETENCIES

- Assess functional status of clients with musculoskeletal disorders, and monitor, document, and report abnormal manifestations.
- Use evidence-based research to assess clients at risk for osteoporosis and to evaluate the effectiveness of Internet use to teach older adults with rheumatoid arthritis.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualized nursing interventions for clients with musculoskeletal disorders.
- Administer topical, oral, and injectable medications used to treat musculoskeletal disorders knowledgeably and safely.
- Provide skilled care of clients having a surgical debridement for osteomyelitis and a total joint replacement.
- Integrate interdisciplinary care into care of clients with musculoskeletal disorders.
- Provide teaching appropriate for community-based self-care of musculoskeletal disorders.
- Revise plan of care as needed to provide effective interventions to promote, maintain, or restore functional health status to clients with musculoskeletal disorders.

MEDIALINK

Resources for this chapter can be found on the Prentice Hall Nursing MediaLink DVD-ROM accompanying this textbook, and on the Companion Website at http://www.prenhall.com/lemone



KEY TERMS

ankylosing spondylitis (AS), *1469*
arthritis, *1433*
fibromyalgia, *1486*
gout, *1443*
Lyme disease, *1476*
muscular dystrophy (MD), *1458*
osteoarthritis (OA), *1449*
osteomalacia, *1447*
osteomyelitis, *1477*
osteoporosis, *1433*
Paget's disease, *1441*
polymyositis, *1476*
reactive arthritis (ReA), *1470*
rheumatic disorders, *1433*
rheumatoid arthritis (RA), *1459*
scleroderma, *1484*
septic arthritis, *1481*
Sjögren's syndrome, *1486*
systemic lupus erythematosus (SLE), *1471*
tophi, *1443*

Various metabolic, degenerative, autoimmune, inflammatory, infectious, neoplastic, connective tissue, and structural disorders may affect the musculoskeletal system. Many of these diseases have significant physical, psychosocial, and financial consequences. When these problems occur, clients experience a variety of individualized responses to their altered health status. Nursing care is directed toward meeting physiologic needs, providing education, and ensuring psychologic support for the client and family.

Arthritis refers to inflammation of the joints, while **rheumatic disorders** refer to diseases of the muscles and bones as well as the joints. These diseases affect not only the joints but also the connective tissues of the body. The various types of arthritis are discussed in this chapter in different sections, depending on the primary etiology of the disorder. Arthritis and other rheumatic disorders are widespread, affecting about 70 million people in the United States (Flynn & Johnson, 2005). Arthritic disorders are a leading cause of disability; however, their very prevalence may lead the public and healthcare professionals to treat them as normal aging processes or discount the validity of the pain and disability experienced by the person with arthritis.

There are more than 100 different types of arthritis, but the most common are osteoarthritis, rheumatoid arthritis, systemic lupus erythematosus, and gout (Moss Rehab Resource Net, 2005). The etiology of most rheumatic disorders is not clear; in many cases, the pathophysiologic processes involved are often complex and poorly understood. Many are primary disorders; others occur as secondary processes associated with another disease. The wear and tear of aging, autoimmune processes, metabolic disorders, genetic factors, and infection are also implicated as causative factors in some forms of rheumatic disease.

METABOLIC BONE DISORDERS

Metabolic bone disorders originate in the bone remodeling process, which normally involves a sequence of events of bone reabsorption and formation. In the adult, this process is primarily internal remodeling through replacement of trabecular bone. Adults replace about 25% of trabecular bone every 4 months through reabsorption of old bone by osteoclasts and formation of new bone by osteoblasts (Porth, 2005). Metabolic bone disorders may result from a variety of factors, including aging, calcium and phosphate imbalances, genetics, and changes in levels of hormones.

THE CLIENT WITH OSTEOPOROSIS

Osteoporosis, literally defined as "porous bones," is a metabolic bone disorder characterized by loss of bone mass, increased bone fragility, and an increased risk of fractures. The reduced bone mass is caused by an imbalance of the processes that influence bone growth and maintenance. Although osteoporosis may result from an endocrine disorder or malignancy, it is most often associated with aging.

The National Osteoporosis Foundation (2006) has found that osteoporosis is a health threat for an estimated 44 million Americans; 10 million people have osteoporosis and 34 million have low bone mass, increasing their risk for the disease. Although osteoporosis can occur at any age and in both men and women, 80% of those with osteoporosis are women. One in two women and one in four men over age 50 will have an osteoporosis-related fracture in his or her remaining lifetime.

Risk Factors

The risk of developing osteoporosis depends on how much bone mass is achieved between ages 25 and 35, and how much is lost later. Certain diseases, lifestyle habits, and ethnic backgrounds increase the risk of developing osteoporosis (see Focus on Cultural Diversity on page 1434). Different variables affect one's risk of osteoporosis—some can be modified and others cannot. The risk factors are summarized in Box 42–1.

BOX 42–1 Risk Factors for Osteoporosis

- A family history of osteoporosis
- Personal history of fracture after age 50
- Current low bone mass
- History of fracture in a first-degree relative
- Being female, especially Caucasian or Asian
- Being thin and/or having a small frame
- Menopause-associated low estrogen levels
- Low testosterone levels in men
- Dietary: low lifetime calcium intake, vitamin D deficiency
- Medication use: anticonvulsants, corticosteroids
- Lifestyle: inactivity, cigarette smoking, excess alcohol
- Presence of certain chronic diseases

MEDIALINK Arthritis and Osteoporosis Animations

FOCUS ON CULTURAL DIVERSITY
The Client with Osteoporosis

- Significant risk is reported for people of all ethnic backgrounds, but the highest percentage of cases is in non-Hispanic and Asian women age 50 or older, with 20% estimated to have osteoporosis, and 52% estimated to have low bone mass. Ten percent of Hispanic women age 50 or older are affected with 49% estimated to have low bone mass.
- The lowest incidence is in non-Hispanic black women over the age of 50, with an estimated 5% having osteoporosis and 35% low bone mass.
- In men, 7% of those affected are non-Hispanic white and Asian men, 4% are non-Hispanic black men, and 3% are Hispanic (all age 50 or older).

Source: National Osteoporosis Foundation. (2006). *Fast Facts*.

Unmodifiable Risk Factors

Both men and women are susceptible to osteoporosis as they age, because the osteoblasts and osteoclasts undergo alterations that diminish their activity. Women have a significantly higher risk for manifestations and complications of osteoporosis because their peak bone mass is 10% to 15% less than that of men. In addition, age-related bone loss begins earlier and proceeds more rapidly in women, beginning in their 30s and accelerating before menopause. Estrogen in women and testosterone in men appear to help prevent bone loss; decreasing levels of these hormones associated with aging contribute to bone loss. Age-related bone loss in men occurs 15 to 20 years later than in women and at a slower rate.

European Americans and Asians are at a higher risk for osteoporosis than African Americans, who have greater bone density (bone mass positively correlates with the amount of skin pigmentation). Premature osteoporosis is increasing in female athletes, who have a greater incidence of eating disorders and amenorrhea. Poor nutrition and intense physical training can result in a deficient production of estrogen. Decreased estrogen, combined with a lack of calcium and vitamin D, results in a loss of bone density (Porth, 2005).

Clients who have an endocrine disorder such as hyperthyroidism, hyperparathyroidism, Cushing's syndrome, or diabetes mellitus are at high risk for osteoporosis. These disorders affect the metabolism, in turn affecting nutritional status and bone mineralization.

Modifiable Risk Factors

Modifiable risk factors include behaviors that place a person at risk for developing osteoporosis, as well as physical changes such as menopause whose contribution to osteoporosis can be modified by preventive strategies. Calcium deficiency is an important modifiable risk factor contributing to osteoporosis. Calcium is an essential mineral in the process of bone formation and other significant body functions. When there is an insufficient intake of calcium in the diet, the body compensates by removing calcium from the skeleton, weakening bone tissue. Acidosis, which may result from a high-protein diet, contributes to osteoporosis in two ways. Calcium is withdrawn from the bone as the kidneys attempt to buffer the excess acid. Acidosis also may directly stimulate osteoclast function. A high intake of diet soda with a high phosphate content also can deplete calcium stores.

With menopause and decreasing estrogen levels, bone loss accelerates in women. Estrogen promotes the activity of osteoblasts, increasing new bone formation. In addition, estrogen enhances calcium absorption and stimulates the thyroid gland to secrete calcitonin, a hormone that suppresses osteoclast activity and increases osteoblast activity.

Both cigarette smoking and excess alcohol intake are risk factors for osteoporosis. Smoking decreases the blood supply to bones. Nicotine slows the production of osteoblasts and impairs the absorption of calcium, contributing to decreased bone density. Alcohol has a direct toxic effect on osteoblast activity, suppressing bone formation during periods of alcohol intoxication. In addition, heavy alcohol use may be associated with nutritional deficiencies that contribute to osteoporosis. Interestingly, moderate alcohol consumption in postmenopausal women actually may increase bone mineral content, possibly by increasing levels of estrogen and calcitonin.

Sedentary lifestyle is another modifiable risk factor that can cause osteoporosis. Weight-bearing exercise, such as walking, influences bone metabolism in several ways. The stress of this type of exercise causes an increase in blood flow to bones, which brings growth-producing nutrients to the cells. Walking causes an increase in osteoblast growth and activity.

Prolonged use of medications that increase calcium excretion, such as aluminum-containing antacids and anticonvulsants, increase the risk of developing osteoporosis. Heparin therapy increases bone resorption, and its prolonged use is associated with osteoporosis. Antiretroviral therapy for people with AIDS or HIV infection may cause decreased bone density and osteoporosis (Porth, 2005).

Anyone who takes a glucocorticoid medication for more than 3 months is at risk for glucocorticoid-induced osteoporosis. These medications, often prescribed to control many rheumatic diseases, include prednisone (Deltasone, Orasone), prednisolone (Prelone), dexamethasone (Decadron, Hexadrol), and cortisone (Cortisone Acetate). These medications can directly affect bone cells, slowing the rate of bone formation. They also interfere with how the body uses calcium and affect levels of sex hormones, leading to bone loss. Problems that result, such as an increased possibility of fractures, can be prevented by taking a daily regimen of calcium supplements with added vitamin D and one multivitamin (American College of Rheumatology [ACR], 2004a).

Pathophysiology

Although the exact pathophysiology of osteoporosis is unclear, it is known to involve an imbalance of the activity of osteoblasts that form new bone and osteoclasts that resorb bone. Until age 35, when peak bone mass occurs, formation occurs more rapidly than does reabsorption. After peak bone mass is achieved, slightly more is lost than is gained (about 0.3% to 0.5% per year); this loss is accelerated if the diet is deficient in

vitamin D and calcium. In women, bone loss increases after menopause (with loss of estrogen), then slows but does not stop at about age 60. Older women may have lost between 35% and 50% of their bone mass, older men may have lost between 20% and 35% (Mayo Clinic, 2002).

Osteoporosis affects the diaphysis (shaft of the bone) and the metaphysis (portion of the bone between the diaphysis and the epiphysis). The diameter of the bone increases, thinning the outer supporting cortex. As osteoporosis progresses, trabeculae are lost from cancellous bone (the spongy tissue of bone), and the outer cortex thins to the point that even minimal stress will fracture the bone (Porth, 2005).

Manifestations

The most common manifestations of osteoporosis are loss of height, progressive curvature of the spine, low back pain, and fractures of the forearm, spine, or hip. Osteoporosis is often called the "silent disease," because bone loss occurs without symptoms.

The loss of height occurs as vertebral bodies collapse. Acute episodes generally are painful, with radiation of the pain around the flank into the abdomen. Vertebral collapse can occur with little or no stress; minimal movements such as bending, lifting, or jumping may precipitate the pain. In some clients, vertebral collapse may occur slowly, accompanied by little discomfort. Along with loss of height, characteristic dorsal kyphosis and cervical lordosis develop, accounting for the "dowager's hump" often associated with aging. The abdomen tends to protrude and knees and hips flex as the body attempts to maintain its center of gravity (Figure 42–1 ■).

Complications

Fractures are the most common complication of osteoporosis, with the disease being responsible for more than 1.5 million fractures each year. These include 700,000 vertebral compression fractures, 300,000 hip fractures, 250,000 wrist fractures, and 300,000 fractures at other sites (National Osteoporosis Foundation, 2006). There may be no obvious manifestations of osteoporosis until fractures occur. Some fractures are spontaneous; others may result from everyday activities. While wrist and vertebral fractures have not been shown to increase disability or mortality, persistent pain and associated posture changes may restrict the client's activities or interfere with activities of daily living (ADLs).

INTERDISCIPLINARY CARE

Care of the client with osteoporosis focuses on stopping or slowing the process, alleviating the symptoms, and preventing complications. Proper nutrition and exercise are important components of the treatment program.

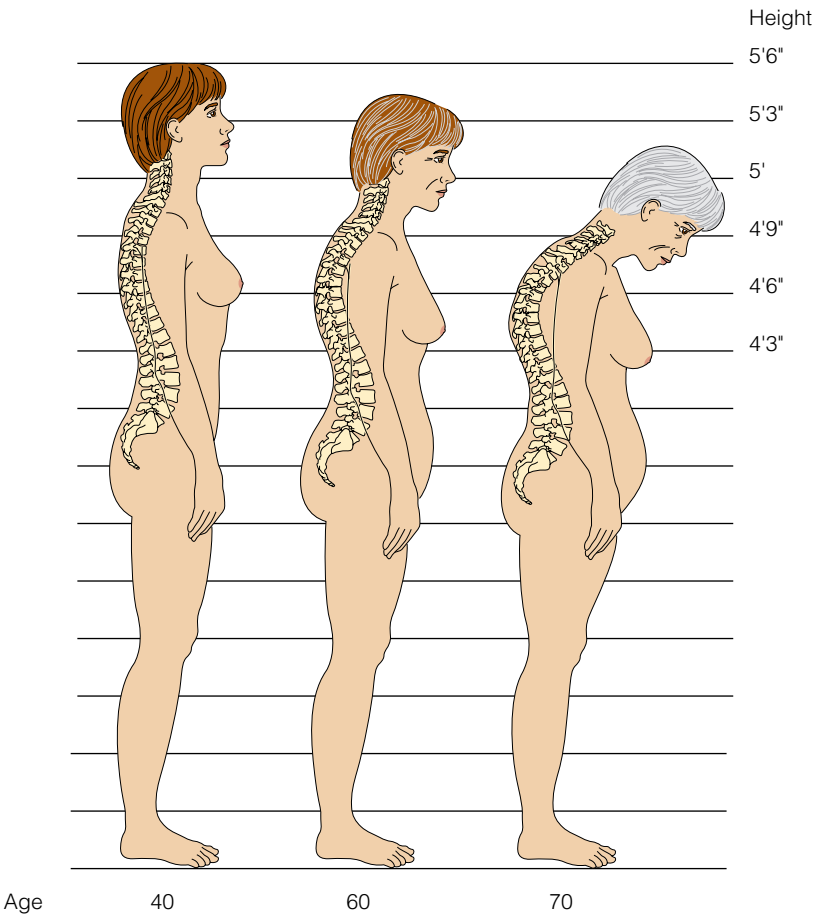


Figure 42–1 ■ Spinal changes caused by osteoporosis. As the condition progresses, height can be reduced by as much as 7 inches.

Diagnosis

The manifestations of osteoporosis can mimic those of other bone disorders, so diagnostic tests are needed to differentiate osteoporosis from other problems.

Dual-energy x-ray absorptiometry (DEXA) measures bone density in the lumbar spine or hip and is considered to be highly accurate. Ultrasound transmits painless sound waves through the heel of the foot to measure bone density. This 1-minute test is not as sensitive as DEXA, but is accurate enough for screening purposes. These tests are described in Chapter 40 ∞.

Laboratory tests include alkaline phosphatase (AST), which may be elevated following a fracture, and serum bone Gla-protein (osteocalcin), which can be used as a marker of osteoclastic activity and therefore is an indicator of the rate of bone turnover. This test is most useful to evaluate the effects of treatment, rather than as an indicator of the severity of the disease. A comparison of laboratory tests for metabolic bone diseases is outlined in Table 42–1.

Medications

Estrogen replacement therapy reduces bone loss, increases bone density in the spine and hip, and reduces the risk of fractures in postmenopausal women. It is particularly recommended for women who have undergone surgical menopause before age 50, and often is prescribed for women with other osteoporosis risk factors. Estrogen therapy alone is associated with an increased risk of endometrial cancer, so it usually is prescribed in combination with progestin (hormone replacement therapy or HRT). The choice of using HRT to prevent osteoporosis is one that must be made between the woman and her healthcare provider.

Raloxifene (Evista) is a selective estrogen receptor modulator (SERM) that appears to prevent bone loss by mimicking estrogen's beneficial effects on bone density in postmenopausal women. It does not have the risks of estrogen. Hot flashes are a common side effect, and this drug should not be taken by a woman with a history of blood clots.

Alendronate (Fosamax) and risedronate (Actonel) are from the class of drugs known as bisphosphonates. Bisphosphonates are potent inhibitors of bone resorption that may be used to prevent and treat osteoporosis. They inhibit bone breakdown, preserve bone mass, and increase bone density in the hip and vertebrae. Alendronate is especially useful for men and young adults and to prevent or treat glucocorticoid-induced osteoporosis. The nursing implications of bisphosphonates are found in the Medication Administration box on page 1437. Teriparatide (Forteo) is a synthetic parathyroid hormone, administered subcutaneously to stimulate new bone formation and mass. It is used to decrease the risk of bone fracture from osteoporosis in postmenopausal women and in men with primary or secondary hypogonadism.

Ibandronate sodium (Boniva) is the first monthly osteoporosis medication to be approved by the U.S. Food and Drug Administration (FDA). It is used for both treatment and prevention of postmenopausal osteoporosis, and reduces the number of vertebral fractures in women with osteoporosis as well as increases bone density in women who do not have the disease.

Calcitonin (Miacalcin) is a hormone that increases bone formation and decreases bone resorption. Calcitonin increases spinal bone density and reduces the risk of compression fractures; it may reduce the risk of hip fracture as well. Calcitonin usually is prescribed as a nasal spray, although it also is available in parenteral form. Because calcitonin is a protein, it can precipitate anaphylactic-type allergic responses.

Sodium fluoride stimulates osteoblast activity, increasing bone formation. When used to treat osteoporosis, bone mass of the spine increases and the risk of spinal fractures may be reduced. Fluoride therapy may, however, be associated with an increased risk of hip and other nonvertebral fractures.

Medications being investigated include vitamin D metabolites and other bisphosphonates and SERMS. See the Medication Administration Box on page 1437 for information about calcium, calcitonin, and fluoride.

TABLE 42–1 Differential Features of Osteoporosis, Osteomalacia, and Paget's Disease

DIFFERENTIATING FEATURES	OSTEOPOROSIS	OSTEOMALACIA	PAGET'S DISEASE
Pathophysiology	Resorption greater than bone formation	Inadequate mineralization of bone matrix	Excessive osteoclastic activity and formation of poor-quality bone
Calcium level (serum)	Normal	Low or normal	Normal or elevated (especially in immobilized clients)
Phosphate level (serum)	Normal	Low or normal	Normal
Parathyroid hormone level (serum)	Normal	High or normal	Normal
Alkaline phosphatase level (serum)	Normal	Elevated	Increased; not a reliable test for clients who have liver disease or are pregnant
Hydroxyproline (urine)	Not applicable	Not applicable	Increased
Radiographic findings	Osteopenia, fractures	Decreased bone density, radiolucent bands known as Looser's zones, or pseudofractures	"Punched-out" appearance of bone, increase in bone thickness, linear fractures, mosaic pattern of bone matrix

MEDICATION ADMINISTRATION The Client with Osteoporosis

CALCIUM

Postmenopausal women, regardless of whether they take replacement estrogens, are encouraged to take calcium to prevent osteoporosis.

Nursing Responsibilities

- Help clients maintain an adequate dietary intake of calcium. The best dietary source is milk and other dairy products, including yogurt.
- Postmenopausal women who take estrogens need 1000 mg of calcium daily. Those who do not take estrogens need about 1500 mg daily to minimize osteoporosis.
- Identify alternate sources, such as skim milk and low-fat yogurt, oysters, canned sardines or salmon, beans, cauliflower, and dark-green leafy vegetables.

Health Education for the Client and Family

- Take calcium carbonate in divided doses 30 to 60 minutes before meals to allow for absorption.
- Take calcium citrate with meals to minimize gastrointestinal distress.

CALCITONIN

Calcitonin-salmon injection, synthetic
Calcimar
Miacalin (injection or nasal spray)

In postmenopausal osteoporosis, calcitonin prevents further bone loss and increases bone mass if the client consumes adequate amounts of calcium and vitamin D. Calcitonin may be used in postmenopausal women who cannot or will not take estrogen.

Nursing Responsibilities

- Calcitonin is protein in nature; both the parenteral and nasal spray forms may cause an anaphylactic-type allergic response. Observe the client for 20 minutes after administration; have appropriate emergency equipment and drugs available to treat anaphylaxis.
- Alternate nostrils daily when administering calcitonin nasal spray.
- Review medical history for conditions that contraindicate use of calcitonin products: hypersensitivity to salmon calcitonin and lactation (calcitonin is secreted in breast milk and may inhibit lactation).
- Observe for side effects: nausea and vomiting, anorexia, mild transient flushing of the palms of the hands and the soles of the feet, and urinary frequency.
- Teach the client the proper technique for handling and injecting the drug at home.

Health Education for the Client and Family

- Take the medication in the evening to minimize side effects.
- Warm nasal spray to room temperature before using.
- Rhinitis (runny nose) is the most common side effect with calcitonin nasal spray. Other possible side effects include sores, itching, or other nasal symptoms. Report nosebleeds to your primary care provider.
- Nausea and vomiting may occur during initial stages of therapy; they disappear as treatment continues.
- While taking the medication, be sure to consume adequate amounts of calcium and vitamin D.

FLUORIDE

Fluoride is a mineral long recognized as essential for the normal formation of dentin and tooth enamel. Fluoride appears to decrease the solubility of bone mineral and therefore the rate of bone reabsorption. Its use in preventing and treating osteoporosis is relatively new but promising.

Nursing Responsibilities

- Monitor serum fluoride levels every 3 months.
- Have bone mineral density studies conducted at 6-month intervals to document progress of bone growth.

Health Education for the Client and Family

- Take sodium fluoride tablets after meals, and avoid milk or dairy products; these reduce gastrointestinal absorption of the medication.
- While taking fluoride, be sure to maintain an adequate calcium intake.
- Use fluoride mouth rinse immediately after brushing teeth and just before retiring at night. Do not swallow the rinse, and avoid eating or drinking for at least 30 minutes after use.
- Notify the physician if teeth become stained or mottled after repeated use of fluoride mouth rinse.

NURSING CARE

Osteoporosis is both preventable and treatable; therefore, nursing care focuses primarily on planning and implementing interventions to prevent the disease, its manifestations, and the resulting injuries. An important aspect of preventing osteoporosis is educating clients under age 35. A Nursing Care Plan for a client with osteoporosis is found on page 1438.

Health Promotion

Health promotion activities to prevent or slow osteoporosis focus on calcium intake, exercise, and health-related behaviors.

Nutrition

For clients of all ages, stress the importance of maintaining a daily calcium intake that meets National Institutes of Health (NIH) recommendations (see Chapter 10 ∞). This is particularly important for adolescent girls and young adult women who may avoid eating many high-calcium foods such as dairy products because of concerns about weight. Optimal calcium intake before ages 30 to 35 probably increases peak bone mass. Emphasize that low-fat (or nonfat) dairy products also contain calcium, although some fat in the product may enhance calcium absorption.

Milk and milk products are the best sources of calcium. The lactose in milk facilitates calcium absorption as well. Other food sources of calcium include sardines, clams, oysters, and salmon, as well as dark green, leafy vegetables such as broccoli, collard greens, bok choy, and spinach. For clients who avoid dairy products because of lactose intolerance or a vegetarian diet, suggest alternate sources.

NURSING CARE PLAN A Client with Osteoporosis

Nancy Bauer is a 53-year-old schoolteacher. She has been married for 36 years and has two children. Mrs. Bauer is 65 inches tall. She has smoked one pack of cigarettes a day for 30 years and drinks one to two glasses of wine with dinner each evening. She does not routinely exercise. Mrs. Bauer has had symptoms of menopause for 8 years, including hot flashes in the early years and mood swings of late. She has never been on hormone replacement therapy.

Mrs. Bauer is currently seeking medical advice for continuous low back pain. The pain is not relieved with an over-the-counter analgesic, and she frequently wakes up during the night because of the pain. She is diagnosed with osteoporosis.

ASSESSMENT

The nurse practitioner notes that Mrs. Bauer's vital signs are within normal limits. She has full range of motion of all extremities and is able to stand and bend over, but she reports discomfort when returning to the upright position. Mrs. Bauer has a slightly pronounced "hump" on her upper back and is 1 inch shorter than her stated height on admission. Her muscle strength is symmetric and strong.

DIAGNOSIS

- *Acute Pain* of the lower spine related to vertebral compression
- *Deficient Knowledge* related to osteoporosis and treatment to prevent further damage
- *Imbalanced Nutrition: Less than Body Requirements* related to inadequate intake of calcium
- *Risk for Injury* related to effects of change in bone structure secondary to osteoporosis

EXPECTED OUTCOMES

- Verbalize a decrease in back pain.
- Be able to describe ways to treat her osteoporosis and prevent further complications.
- Verbalize an understanding of the current research and treatment regarding osteoporosis.
- Verbalize how stopping smoking can help prevent further progression of osteoporosis.
- Seek consultation for supplements and medications to prevent further bone loss.
- Design a program of physical activity to prevent complications of osteoporosis.
- Verbalize safety precautions to prevent fractures due to falls.

PLANNING AND IMPLEMENTATION

- Teach back strengthening exercises.
- Refer to an osteoporosis support group, if available.
- Provide realistic, yet optimistic, feedback about loss of height and bone integrity and the potential outcomes of treatment.
- Assess current knowledge base, and correct misconceptions regarding treatment of osteoporosis.
- Provide current educational literature regarding treatment of osteoporosis.
- Instruct in dietary and calcium supplements that help prevent effects of osteoporosis.
- Discuss physical exercises that help prevent complications due to osteoporosis.
- Review safety and fall precautions, and provide literature regarding how to create a safe home environment.

EVALUATION

On her return visit 6 months later, Mrs. Bauer reports that she feels much better. She is no longer irritable and does not experience mood swings, because she has been taking her prescribed hormone replacements for 6 months: She is eating products rich in calcium and taking a daily supplement of calcium with vitamin D. Mrs. Bauer has reduced her wine intake to one glass in the evening and now drinks decaffeinated coffee and tea. She also states that since she stopped smoking, she has been walking 30 to 45 minutes every day.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the rationale for stopping smoking and limiting caffeine and alcohol intake in the treatment of osteoporosis?
2. What foods would you encourage for clients at high risk for osteoporosis whose serum cholesterol and LDL/HDL ratios indicate a high risk for cardiovascular disease?
3. What physical activities would you consider beneficial in helping to prevent the effects of osteoporosis in the female client who is wheelchair bound or has limited mobility?
4. Develop a care plan for Mrs. Bauer for the nursing diagnosis *Risk for Trauma*

See Evaluating Your Response in Appendix C.

Calcium supplements are available in many forms. Most supplements (including Tums) provide calcium carbonate in the range of 200 to 600 mg per tablet. Other forms of calcium, including citrate, gluconate, and lactate, generally provide a lower amount of elemental calcium per tablet. A combination of calcium with vitamin D is recommended, particularly for older adults who may have a vitamin D deficiency that impairs their ability to absorb and use calcium.

Exercise

Teach clients the importance of physical activity and weight-bearing exercises in preventing and slowing bone loss. Suggest that clients participate in regular exercise such as walking for at least 20 minutes four or more times a week. Inform clients that swimming and pool aerobic exercises are not as beneficial for maintaining bone density because of the lack of weight-bearing activity.

Healthy Behaviors

Behaviors that help prevent osteoporosis include not smoking, avoiding excessive alcohol intake, and limiting caffeine intake to two or three cups of coffee each day.

Assessment

Collect the following data through the health history and physical examination (see Chapter 40 ∞):

- *Health History:* Age, risk factors, history of fractures, smoking history, alcohol intake, medications, usual diet, menstrual

history including menopause, usual exercise/activity level (see the Nursing Research box below), low back pain.
- *Physical Examination:* Height, spinal curves.

Nursing Diagnoses and Interventions

Nursing care of clients who have osteoporosis focuses on teaching about the disease process, helping maintain physical mobility and nutrition, and solving problems associated with pain and injury.

Health-Seeking Behaviors

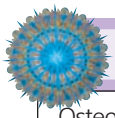
At multiple points in the client's lifetime, nurses can provide vital information that will help clients use self-care strategies to reduce their risk of developing osteoporosis:
- Assess the client's health habits, including diet, exercise, smoking, and alcohol use. *The risk of developing osteoporosis in later life is affected by such things as diet, regular participation in weight-bearing exercise, and personal habits such as smoking and alcohol consumption.*
- Teach women and men of all ages the importance of maintaining an adequate calcium intake. Provide a list of calcium-rich foods, and discuss the use of calcium supplements with clients who do not consume adequate dietary calcium. *Calcium needs vary during the course of a lifetime; however, many clients never consume adequate amounts of calcium. This affects their peak bone mass and the rate of bone loss with aging. Calcium in foods is more completely absorbed than that supplied by calcium supplements.*
- Discuss the importance of maintaining a regular schedule of weight-bearing exercise, either through an exercise program or regular physical activity. *Weight-bearing exercise promotes osteoblast activity, helping maintain bone strength and integrity.*
- Refer clients to smoking-cessation programs and alcohol treatment programs as appropriate. *Smoking interferes with estrogen's protective effects on bones, promoting bone loss. Excess alcohol intake affects the nutritional status of the client, increasing the risk of calcium and vitamin D deficiency.*
- Refer clients with significant risk factors for osteoporosis to primary care providers or clinics for bone-density evaluation. *Early identification and treatment of osteoporotic changes in bones can reduce the risk and possible long-term consequences of falls and fractures.*

Risk for Injury

Falls that would result in little or no injury in the healthy adult may cause fractures in the client with osteoporosis. Even normal movements such as twisting, bending, lifting, or rising from bed can precipitate a vertebral fracture.
- Implement safety precautions as necessary for the client who is hospitalized or in a long-term care facility. Maintain the bed in low position; use side rails if indicated to prevent the client from getting up alone; provide nighttime lighting to toilet facilities. *Most falls are preventable, particularly in hospitals and long-term care facilities.*
- Avoid using restraints (if hospitalized or a resident in a long-term care facility) if at all possible. *Restraints may actually increase the client's risk of falling and increase the risk of injury associated with a fall.*

PRACTICE ALERT
Clients may fracture osteoporotic bones when pulling against restraints.

- Teach clients who are able to participate in weight-bearing exercises to perform exercises at least three times a week for a sustained period of 30 to 40 minutes. The mechanical force of weight-bearing exercises promotes bone growth. *Bones weaken and demineralize without exercise. Walking is an*



NURSING RESEARCH Evidence-Based Practice for the Client with Osteoporosis

Osteoporosis is a major health problem in the United States. Risk factors for osteoporosis include being white, having a small body frame, not doing weight-bearing exercises, and having a family history of osteoporosis. Nursing can do much to prevent the development of osteoporosis by assessing risk factors and teaching about diet, exercise, and lifestyle. A study by Schoen (2004) was conducted to determine the validity of items on a risk assessment screening questionnaire called the Osteoporosis Risk Assessment Tool (ORAT).

IMPLICATIONS FOR NURSING

Assessment is a critical component of the nursing process, enabling nurses to identify clients at risk for diseases, to monitor ongoing interventions, and to design teaching specific to client needs. Based on review of items contained in the ORAT, the following were retained: age, gender, body mass index, previous fractures, diagnosis of thyroid disease, use of thyroid replacement medication, estrogen replacement therapy, weight-bearing exercise, family history of fractures, age at onset of menopause, use of calcium supplements, diet of calcium-rich foods, and alcohol and tobacco use. This tool is a useful means of quickly assessing clients for osteoporosis risk factors and for identifying and teaching about the prevention, diagnosis, treatment, and complications of osteoporosis.

CRITICAL THINKING IN CLIENT CARE

1. At what age do women develop maximum bone mass? (Review information in a text or on the Internet.) Based on this information, what type of teaching would be most effective?
2. Compare and contrast your teaching for a 24-year-old black woman who has a calcium-poor diet but is a nonsmoker and a thin, white 64-year-old woman who is postmenopausal and smokes.
3. While screening clients in a clinic for health risks, including osteoporosis, a man in his 70s says "Oh, I can't have bad bones . . . I'm a man!" How would you respond?

Data from Schoen, D. C. (2004). Osteoporosis. *Orthopaedic Nursing, 23*(4), 261–267.

easy, low-impact form of exercise. Swimming (including walking on the bottom of the pool) does not provide the needed weight-bearing activity.

- Encourage older adults to use assistive devices to maintain independence in ADLs. *Walking sticks, canes, and other assistive devices encourage client independence and support activities that promote bone growth.*
- Teach older clients about safety and fall precautions. *An assessment of the client's home for safety and fall risks may reduce the risk of fractures and, in turn, the cost of hospitalization and potential disability and/or death.*

Imbalanced Nutrition: Less than Body Requirements

Most Americans do not maintain their recommended daily intake of calcium. Clients must therefore be made aware of the relationship between an adequate calcium intake and maintaining strong bones.

- Teach adolescents, pregnant or lactating women, and adults through age 35 to eat foods high in calcium and to maintain a daily calcium intake of 1200 to 1500 mg. *The NIH recommends a daily calcium intake of 1200 to 1500 mg per day for adolescents and young adults, as well as for pregnant and lactating women.*
- Encourage postmenopausal women to maintain a calcium intake of 1000 to 1500 mg daily, either through diet or a calcium supplement. *Calcium needs for postmenopausal women vary, depending on age.*
- Teach clients taking calcium supplements the importance of taking the medication at the proper time and the side effects that may occur. *Free hydrochloric acid is needed for calcium absorption. Calcium carbonate supplement (e.g., Tums) should be taken 30 to 60 minutes before meals to allow adequate absorption. Calcium citrate supplements should be taken with meals to prevent gastrointestinal distress.*

PRACTICE ALERT
Calcium supplements should be taken in divided doses (two to three times daily) for improved distribution.

Acute Pain

Advanced stages of osteoporosis can result in pain and immobilization. Acute pain usually results from a complicating fracture, especially a compression fracture of the vertebrae.

- Suggest anti-inflammatory pain medications for treatment of both acute and chronic phases of pain. Clients should be instructed in the amount and frequency as noted on the manufacturer's labels. *Continuous administration of ibuprofen or other nonsteroidal anti-inflammatory, drugs (NSAIDs) can be useful to provide relief from pain, but clients must be cautioned not to exceed dosage recommendations.*

PRACTICE ALERT
Teach clients on long-term anti-inflammatory medications to watch for bright red bleeding from the stomach (in vomitus) or dark black bowel movements.

- Suggest the application of heat to relieve pain. *A heating pad may offer temporary pain relief. To avoid the "rebound effect," the heat should be removed every 20 to 30 minutes.*

Using NANDA, NIC, and NOC

Chart 42–1 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with osteoporosis.

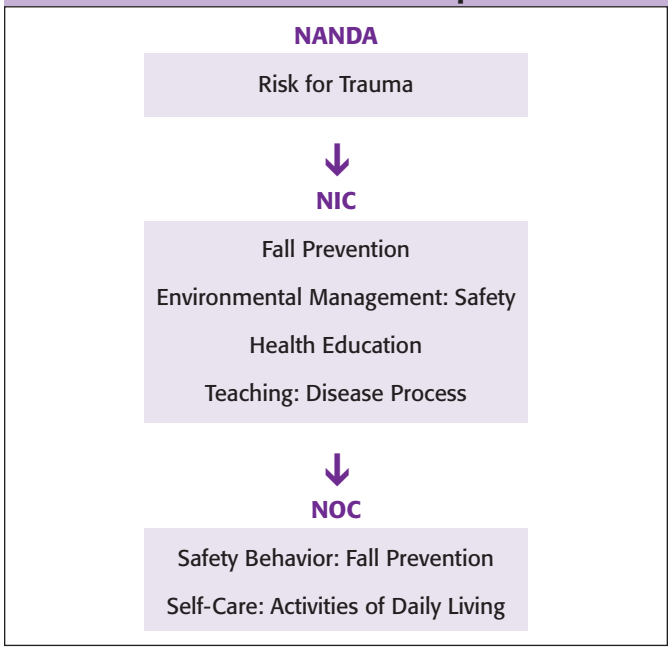
Community-Based Care

The client who has osteoporosis needs education on safety and preventing falls. In addition to home safety, outdoor safety is important, too. Clients should be taught to use assistive devices for added stability, to wear rubber-soled shoes for traction, to walk on the grass when sidewalks are slippery, and to sprinkle salt or kitty litter on icy sidewalks in the winter.

Address the following topics when discussing home care:

- Resources for medical supplies and assistive devices
- Diet, exercise, and medications
- Pain management
- Maintaining good posture to help prevent stress on the spine
- Helpful resources:
  - National Osteoporosis Foundation
  - Osteoporosis and Related Bone Diseases National Resource Center (NIH)
  - National Women's Health Resource Center
  - Older Women's League
  - American College of Rheumatology

NANDA, NIC, AND NOC LINKAGES
CHART 42–1 **The Client with Osteoporosis**



Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

THE CLIENT WITH PAGET'S DISEASE

Paget's disease, also called *osteitis deformans*, is a progressive metabolic skeletal disorder that results from excessive metabolic activity in bone, with excessive bone resorption followed by excessive bone formation. This chronic remodeling results in the affected bones being larger and softer, with manifestations of bone pain, arthritis, obvious skeletal deformities, and fractures. The disorder affects bones of the axial skeleton, especially the femur, pelvis, vertebrae, and skull. The disease may affect one bone or multiple bones. The cause is unknown; however, several theories have been proposed, including hormonal imbalance, vascular disorder, neoplasm, autoimmune disorder, and inborn error of connective tissue. It is the second most common bone disease in the United States.

Paget's disease occurs in both men and women, affecting 1.5% to 8% of the population over the age of 50 in many countries. It is less common in people of Asian, Indian, and Scandinavian descent. It has a familial tendency as a result of mutations in several genes. The measles virus has been found in bone lesions, and the relevance of that finding is under investigation (Paget Foundation, 2004a).

Pathophysiology

Paget's disease progresses slowly. It usually follows a two-stage process: an excessive amount of osteoclastic bone resorption, followed by excessive osteoblastic bone formation. The initial phase presents with an abnormal increase in osteoclasts. The bones increase in size and thickness because of the acceleration in bone resorption and regeneration, resulting in a thick layer of coarse bone with a rough and pitted outer surface (Porth, 2005). Resorption of cancellous bone occurs rapidly. As new bone tissue tries to replace the loss, fibrous tissue forms in the bone marrow. The bone is at first hyperemic and soft, and bowing occurs. When this excessive bone cell activity decreases, the result is a gain in bone mass, but the newly formed bone becomes hard and brittle. This brittleness may lead to fractures.

Manifestations

Most clients with Paget's disease are asymptomatic for years, and the disease often is discovered when typical changes are seen on an incidental x-ray. Manifestations are often vague and depend on the specific area involved (see the box on this page). The most common manifestation is localized pain of the long bones, spine, pelvis, and cranium. The pain is described as a mild to moderate deep ache that is aggravated by pressure and weight bearing. It is more noticeable at night or when the client is resting. The pain usually is due to metabolic bone activity, secondary degenerative osteoarthritis, fractures, or nerve impingement. Because of the increase in blood flow to pagetic bone, flushing and warmth of the overlying skin may be apparent.

MANIFESTATIONS of Paget's Disease

MUSCULOSKELETAL EFFECTS
- Pain (in the long bones of lower extremities or joints)
- Deformity (enlargement of skull, bowing of lower extremities, and deformity of elbows and knees)
- Fractures of lower extremities
- Pathologic fractures (especially of the tibia)
- Compression fractures
- Collapse of the vertebrae, resulting in kyphosis and loss of height
- Muscle weakness

NEUROLOGIC EFFECTS
- Hearing loss
- Spinal cord injuries
- Dementia
- Pain from spinal stenosis
- Bladder and/or bowel dysfunction

CARDIOVASCULAR EFFECTS
- Congestive heart failure

METABOLIC EFFECTS
- Symptoms of hypercalcemia in immobilized clients
- Hypercalciuria and renal calculi
- Increased skin temperature over affected extremities

Complications

Complications of Paget's disease are as follows:

- Nerve palsy syndromes from involvement of the upper extremities
- Pathologic fractures from loss of bone structure
- Mental deterioration from compression of the brain when the skull is involved
- Compression of the spinal cord from affected cervical vertebrae causing quadraplegia
- Cardiovascular disease, resulting from vasodilation of the vessels in the skin and subcutaneous tissues overlying the affected bones
- Osteogenic sarcoma, seen in 5% to 10% of people with severe disease (Porth, 2005).

INTERDISCIPLINARY CARE

Care of the client with Paget's disease focuses on relieving pain, suppressing bone cell activity if necessary, and preventing or minimizing the effects of complications. Many clients with Paget's disease are asymptomatic and do not require treatment. For more severely affected clients, pharmacologic agents are usually effective. Occasionally, surgery may be required.

Diagnosis

Many of the diagnostic tests that are useful for the diagnosis of osteoporosis are equally useful for clients with Paget's disease (see Table 42–1). These include x-rays and bone scans to illustrate localized areas of demineralization in the early stages, seen as "punched-out" areas that lend a coarse, irregular appearance to the bone. In the later phase, x-rays show enlargement of the bones, tiny cracks in the long bones, and/or bowing of the weight-bearing bones. Computed tomography (CT)

scans and magnetic resonance imaging (MRI) help identify possible causes of pain, including degenerative problems, spinal stenosis, or nerve root impingement. Diagnostic tests are described in Chapter 40 ∞.

Laboratory tests used in diagnosis include a serum alkaline phosphatase, which will show a steady rise as the disease progresses; the normal level (30 to 115 international units/L) may be elevated from high normal to over 3000 international units/L. A urinary collagen pyridinoline test is a sensitive indicator of the rate of bone resorption.

Medications

Clients who have mild symptoms often find relief using aspirin or NSAIDs, such as ibuprofen (Motrin) and indomethacin (Indocin). Clients who are experiencing manifestations and whose diagnostic test results are elevated are usually treated with an agent that retards bone resorption, such as calcitonin or a bisphosphonate.

Bisphosphonates such as alendronate (Fosamax), pamidronate (Aredia), and tiludronate (Skelid) are the primary treatments used for severe Paget's disease. These drugs inhibit bone resorption, possibly by attaching to the surface of the calcium/phosphate phase of bone and inhibiting osteoclast activity. They are safe and usually are well tolerated by the client. Alendronate is available as an oral preparation, and pamidronate is available for intravenous administration. Oral preparations are poorly absorbed from the GI tract, and may cause gastric or esophageal irritation. Alendronate should be given with a full glass of water on an empty stomach, at least 30 minutes before other medications or food. Pamidronate is given as an intravenous infusion in D_5W or normal saline. It is given for 3 successive days, generally promoting a rapid response with reduced urinary excretion of hydroxyproline and pyridinium and a fall in alkaline phosphatase. Intravenous pamidronate may cause flulike symptoms, but these generally are brief. Calcium supplements also are prescribed for clients receiving bisphosphonates. After bisphosphonate treatment, clients often experience remission of symptoms for a year or more. See the Medication Administration box below for nursing implications.

Calcitonin inhibits osteoclastic resorption of bone. It also works as an analgesic for bone pain. The two derivatives of this medication are salmon (fish) and human. Salmon calcitonin (Calcimar) is generally preferred because it is inexpensive and widely available. Human calcitonin (Cibacalcin) is derived from human thyroid glands, which makes it more expensive and difficult to obtain. The parenteral form of calcitonin is used in treating Paget's disease. (Refer to the Medication Administration Box on page 1437 for nursing implications.)

Surgery

Different surgical interventions may be used to treat clients with Paget's disease, such as repairing a complete fracture through pagetic bone, realigning a knee through tibial osteotomy to decrease pain, or replacing a hip and/or knee for osteoarthritis. Because increased bleeding is a manifestation of Paget's disease, it is important to administer a potent bisphosphonate prior to surgery to decrease hypervascularity and reduce the risk of increased operative blood loss (Paget Foundation, 2004a).

MEDICATION ADMINISTRATION The Client with Paget's Disease

BISPHOSPHONATES

Alendronate (Fosamax) **Etidronate (Didronel)**
Pamidronate (Aredia) **Risedronate (Actonel)**
Tiludronate (Skelid)

The bisphosphonates inhibit bone resorption, increasing the mineral density of bones and reducing the incidence of fractures. They are also used both in the prevention and treatment of osteoporosis: When used for Paget's disease, bisphosphonates slow the accelerated bone turnover associated with this disease. Bone pain is relieved, and the incidence of pathologic fractures is reduced. Cardiac and vascular manifestations of the disease also improve.

Nursing Responsibilities

- Administer alendronate with water 30 minutes before food or other medications.
- Do not give foods high in calcium, vitamins with mineral supplements, or antacids within 2 hours of administering alendronate.
- Instruct the client to avoid lying down for 30 minutes after taking the drug.
- Assess renal function studies before initiating therapy; alendronate is not recommended for use in clients with renal insuffiency.
- Dilute the prescribed dose of pamidronate in 1000 mL of D_5W or normal saline; infuse over at least 4 hours. Do not add to calcium-containing solutions such as Ringer's or lactated Ringer's solutions.
- Monitor the IV site for signs of thrombophlebitis.
- Assess the client for signs of electrolyte imbalance or other adverse responses such as a drug fever.

Health Education for the Client and Family

- Take the medication as directed with clear water only. Consuming other beverages or food within 30 minutes of taking alendronate may interfere with its absorption and effectiveness.
- Do not lie down until after you have eaten. Alendronate can irritate the esophagus.
- Report symptoms such as new or worsening heartburn, difficulty swallowing, or painful swallowing.
- Fever with or without chills may occur while receiving intravenous pamidronate; this will subside without treatment. Flulike symptoms also may occur; these will subside within a week or so.
- Report any abnormal symptoms such as tingling around the mouth or numbness and tingling of the fingers or toes, which may indicate an imbalance of electrolytes in the blood.
- Take calcium and vitamin D supplements as instructed by your primary care provider.
- Response to these medications is gradual, and continues for months after the drug is stopped.

NURSING CARE

Nursing Diagnoses and Interventions

The nursing interventions for the client with symptomatic Paget's disease focus on pain control, prevention of injury or fractures, and education regarding the disease process and prescribed therapies.

Chronic Pain

The most common manifestation of Paget's disease is bone pain. This usually is the manifestation that prompts the client to seek health care.

- Assess the location and extent of the pain to determine the bone areas involved. *Bone pain in Paget's disease is poorly localized and is frequently described as "aching and deep."*
- Teach the client to take NSAIDs or aspirin on a regular basis as prescribed. *Pain is most noticeable at night or when the client is resting. The pain can become evident when it is aggravated by pressure and weight bearing.*
- Ensure correct placement of prescribed brace or corset. The client may be required to wear a light brace or corset to relieve back pain and provide support when assuming an upright position. *The client may need instruction in the correct application of the device and in the evaluation of pressure areas that may result from wearing the device.*
- Suggest referral for heat therapy and massage. *Heat therapy and massage can alleviate mild discomfort. Care should be taken when applying massage over areas prone to pathologic fractures.*

Impaired Physical Mobility

Clients with Paget's disease need to maintain or improve mobility so that they can perform necessary self-care activities and prevent complications of immobility.

- Provide an assistive device for use when ambulating. *During the active phase of Paget's disease, the client is prone to fractures. Bone deformities, activity intolerance, fear of falling, and pain are all factors that may make the client more prone to falls. An assistive device can provide both physical and psychologic support during ambulation, permit the client to ambulate further, and provide a device for resting during ambulation.*
- Teach good body mechanics. *The client with bone deformities should avoid activities that require lifting and twisting.*

PRACTICE ALERT

Activities as seemingly simple as lifting a heavy box may result in a fracture in the client with Paget's disease.

- Reinforce information about exercise protocols and activity regimens. *Exercise and activity protocols should be planned carefully to prevent injury and to minimize fatigue.*

Community-Based Care

A diagnosis of Paget's disease can be frightening for the client and family. It is important that they understand that this is a treatable disease, and that many manifestations of the disease will be relieved with treatment. Inform the client that remissions of the disease often last for a year or more after effective treatment. The Paget Foundation should be suggested as a resource. Discuss the following topics:

- The importance of following the prescribed treatment regimen and keeping scheduled follow-up appointments
- Because it may take several weeks to notice a response to treatment, the importance of continuing therapy during this time and after a response is obtained
- If bisphosphonates such as alendronate or pamidronate are ordered, the importance of taking supplemental calcium to prevent low blood calcium levels
- The importance of remaining active
- Safety in the home and outdoor environment to prevent falls
- The need to report to the primary care provider any sudden pain or disability, even if no trauma has occurred, because pathologic fractures are possible.

THE CLIENT WITH GOUT

Gout is a metabolic disease that occurs from an inflammatory response to the production or excretion of uric acid resulting in high levels of uric acid in the blood (hyperuricemia) and in other body fluids, including synovial fluid. The disorder is characterized by deposits of urates (insoluble precipitates) in the connective tissues of the body. Gout has an acute onset, usually at night, and often involves the first metatarsophalangeal joint (great toe). The initial acute attack is usually followed by a period of months or years without manifestations. As the disease progresses, urates are deposited in various other connective tissues. Deposits in the synovial fluids cause acute inflammation of the joint (*gouty arthritis*). Over time, urate deposits in subcutaneous tissues cause the formation of small white nodules (called **tophi**). Deposits of crystals in the kidneys can form urate kidney stones and result in kidney failure.

Gout may occur as either a primary or secondary disorder. Primary (inherited) gout is characterized by elevated serum uric acid levels resulting from either an inborn error of purine metabolism or a decrease in renal uric acid excretion due to an unknown cause. Purines are part of the structure of the nuclear compounds DNA and RNA; they also may be synthesized by the body. Impaired uric acid excretion leads to hyperuricemia in the majority of people with primary gout. In secondary gout, hyperuricemia occurs as a result of another disorder or treatment with certain medications. Disorders associated with rapid cell turnover, such as some malignancies (leukemia in particular), hemolytic anemia, and polycythemia, can increase purine metabolism. Chronic renal disease, hypertension, starvation, and diabetic ketoacidosis can interfere with uric acid excretion, as can certain drugs, including some diuretics (such as furosemide, ethacrynic acid, and chlorothiazide), pyrazinamide, cyclosporin, ethambutol, and low-dose salicylates. Alcohol ingestion appears to interfere with uric acid excretion and to accelerate its synthesis. In addition, hospitalized clients with gout are at risk for an acute attack from changes in their diet, abdominal surgery, or medications (Tierney et al., 2004).

Gout affects more than 84% of all Americans (ACR, 2005). Gout occurs more often in men, usually after age 30 years. In women, attacks of gout are rarely seen until after menopause. Obesity increases the risk of gout; about half of those with the disorder are 15% or more above their ideal weight (Flynn & Johnson, 2005).

Pathophysiology

Uric acid is the breakdown product of purine metabolism. Normally, a balance exists between its production and excretion, with approximately two-thirds of the amount produced each day excreted by the kidneys and the rest in the feces. The serum uric acid level is normally maintained between 3.4 and 7.0 mg/dL in men and 2.4 and 6.0 mg/dL in women. At levels greater than 7.0 mg/dL, the serum is saturated, and monosodium urate crystals may form. It is not known exactly how crystals of monosodium urate crystals are deposited in joints. Several mechanisms may be involved:

- Crystals tend to form in peripheral tissues of the body, where lower temperatures reduce the solubility of the uric acid.
- A decrease in extracellular fluid pH and reduced plasma protein binding of urate crystals are evident.
- Tissue trauma and a rapid change in uric acid levels may also lead to crystal deposition. A rapid increase in uric acid may occur with tissue trauma and release of cellular components.

The monosodium urate crystals may form in the synovial fluid or in the synovial membrane, cartilage, or other joint connective tissues. They may also form in the heart, earlobes, and kidneys. These crystals stimulate and continue the inflammatory process, during which neutrophils respond by ingesting the crystals. The neutrophils release their phagolysosomes, causing tissue damage, which perpetuates the inflammation.

Manifestations

The manifestations of gout are hyperuricemia, recurrent attacks of inflammation of a single joint, tophi, renal disease, and renal stones. Unless treated, the manifestations of gout appear in three stages: asymptomatic hyperuricemia, acute gouty arthritis, and tophaceous gout. See the Manifestations box on this page.

Asymptomatic Hyperuricemia

The first stage is asymptomatic hyperuricemia, with serum levels averaging 9 to 10 mg/dL. Most people with hyperuricemia do not progress to further stages of the disease.

Acute Gouty Arthritis

The second state is acute gouty arthritis. The acute attack (called a "flare"), usually affecting a single joint, occurs unexpectedly, often beginning at night. It may be triggered by trauma, alcohol ingestion, dietary excess, or a stressor such as surgery. It is often precipitated by an abrupt or sustained increase in uric acid levels. The affected joint becomes red, hot, swollen, and exquisitely painful and tender.

Approximately 50% of initial attacks of acute gouty arthritis occur in the metatarsophalangeal joint of the great toe. Other sites for acute attacks include the instep of the foot, ankles, heels, knees, wrists, fingers, and elbows. The pain, often intense, peaks within several hours and may be accompanied by fever and an elevated white blood cell (WBC) count and sedimentation rate. The affected joints are swollen, and the skin over the joint is warm and dusky red.

Acute attacks of gouty arthritis last from several hours up to several weeks and typically subside spontaneously. There are no long-lasting sequelae, and the client enters an asymptomatic period called the intercritical period. The intercritical period may last up to 10 years; however, approximately 60% of people experience a recurrent attack within 1 year. Successive attacks tend to last longer, occur with increasing frequency, and resolve less completely than the initial attack.

Tophaceous (Chronic) Gout

Tophaceous or chronic gout occurs when hyperuricemia is not treated. The urate pool expands, and monosodium urate crystal deposits (tophi) develop in cartilage, synovial membranes, tendons, and soft tissues. They are seen most often in the helix of the ear; in tissues surrounding joints and bursae (especially around the elbows and knees); along tendons of the finger, toes, ankles, and wrists; on ulnar surfaces of the forearms; along the shins of the legs; and on other pressure points. The skin over tophi may ulcerate, exuding chalky material containing inflammatory cells and urate crystals. Tophi can also develop in the tissues of the heart and spinal epidura. Although tophi themselves are not painful, they may restrict joint movement and cause pain and deformities of the affected joints. Tophi may also compress nerves and erode and drain through the skin.

Complications

Kidney disease may occur in clients with untreated gout, particularly when hypertension is also present. Urate crystals are deposited in renal interstitial tissue. Uric acid crystals also form in the collecting tubules, renal pelvis, and ureter, forming stones. The stones can range in size from a grain of sand to a massive structure filling the spaces of the kidney. Uric acid stones can potentially obstruct urine flow and lead to acute renal failure.

MANIFESTATIONS of Gout

ACUTE GOUTY ARTHRITIS

- Usually monoarticular, affecting metatarsophalangeal joint of great toe, instep, ankle, knee, wrist, or elbow
- Acute pain
- Red, hot, swollen, and tender joint
- Fever, chills, malaise
- Elevated WBC and sedimentation rate

TOPHACEOUS (CHRONIC) GOUT

- Tophi evident on joints, bursae, tendon sheaths, pressure points, helix of ear
- Joint stiffness, limited ROM, and deformity
- Ulceration of tophi with chalky discharge

MEDIALINK Case Study: Diet and Gout

INTERDISCIPLINARY CARE

The classic presentation of acute gouty arthritis is so distinctive that the diagnosis can often be based on the client's history and physical examination. Treatment is directed toward terminating an acute attack, preventing recurrent attacks, and reversing or preventing complications resulting from crystal deposition in tissues and formation of uric acid kidney stones.

Diagnosis

Diagnostic testing is performed to establish an accurate diagnosis and direct long-term therapy. Diagnostic tests are described in Chapter 40 ∞.

Serum uric acid is nearly always elevated (usually above 7.5 mg/dL). The WBC count shows significant elevation, reaching levels as high as 20,000/mm^3 during an acute attack. Eosinophil sedimentation rate (ESR or sed rate) is elevated during an acute attack from the acute inflammatory process that accompanies deposits of urate crystals in a joint. In addition, a 24-hour urine specimen is analyzed to determine uric acid production and excretion, and analysis of fluid aspirated from the acutely inflamed joint or material aspirated from a tophus shows typical needle-shaped urate crystals, providing the definitive diagnosis of gout.

Medications

Medications are used to terminate an acute attack, prevent further attacks, and reduce serum uric acid levels to prevent long-term sequelae of the disease. It is important to treat the acute attack of gouty arthritis before initiating treatment to reduce serum uric acid levels, because an abrupt decrease in serum uric acid may lead to further acute manifestations. Pharmacologic therapy is a mainstay of treatment in achieving these goals.

ACUTE ATTACK NSAIDs are the treatment of choice for an acute attack of gout. Indomethacin (Indocin) is the most frequently used NSAID for gout, although others are equally effective. Other NSAIDs that may be prescribed include ibuprofen (Motrin), naproxen (Naprosyn, Anaprox), tolmetin sodium (Tolectin), piroxicam (Feldene), and sulindac (Clinoril). Although extremely effective, NSAIDs are contraindicated for clients with active peptic ulcer disease, impaired renal function, or a history of hypersensitivity reactions to the drugs. As with other anti-inflammatory drugs, clients should be aware of possible risks and follow recommended doses carefully.

Colchicine can dramatically affect the course of an acute attack. Joint pain begins to diminish within 12 hours of the initiation of treatment and disappears within 2 days. Colchicine apparently acts by interrupting the cycle of urate crystal deposition and inflammation in an acute attack of gout. It has no anti-inflammatory effect in other forms of arthritis, and its use is limited to gout. The use of colchicine is limited by significant side effects. When administered orally, many clients develop abdominal cramping, diarrhea, nausea, or vomiting. Intravenous administration is limited by potential toxic effects including local pain, tissue damage if extravasation occurs during injection, bone marrow suppression, and disseminated intravascular coagulation (DIC). It is contraindicated for clients who have significant gastrointestinal, renal, hepatic, or cardiac disease.

Corticosteroids may also be prescribed for the client with acute gouty arthritis. If possible, the intra-articular route is preferred for monoarticular arthritis to avoid the multiple systemic effects of steroid therapy. When gout is polyarticular, corticosteroids may be administered either orally or intravenously.

Analgesics may also be prescribed during an acute episode of gouty arthritis. Either codeine or meperidine (Demerol) may be administered orally to manage the client's pain. Aspirin is avoided because it may interfere with uric acid excretion.

PROPHYLACTIC THERAPY In clients at high risk for future attacks of acute gout, prophylactic therapy with daily colchicine may be initiated. Prophylaxis is particularly useful during the first 1 to 2 years of treatment with antihyperuricemic agents. Although colchicine does not affect the serum uric acid directly, it reduces the frequency of attacks by preventing crystal deposition within the joint. The doses required to achieve this effect are small, and few side effects are associated with therapy.

Treatment to reduce serum uric acid levels is typically initiated for clients with recurring gout, tophi, or renal damage. Asymptomatic hyperuricemic clients require no treatment. Uricosuric agents are used for clients who do not eliminate uric acid adequately; allopurinol is prescribed for clients who produce excessive amounts of uric acid. Uricosuric drugs block the tubular reabsorption of uric acid, promoting its excretion and reducing serum levels. These drugs reduce the frequency of acute attacks, particularly when administered with colchicine. Probenecid (Benemid) and sulfinpyrazone (Aprazone, Anturane, Zynol) are the primary uricosuric drugs employed.

Allopurinol (Zyloprim) is a xanthine oxidase inhibitor that lowers plasma uric acid levels and facilitates the mobilization of tophi. Because of its effectiveness in lowering serum uric acid levels, it may trigger an attack of acute gout. The nursing implications for medications used to treat gout are included in the Medication Administration box on page 1446.

Complementary and Alternative Therapy

A variety of nutritional and herbal supplements may be used to help prevent gout or decrease the onset of manifestations. These include:

- Vitamin E and selenium may decrease tissue inflammation.
- Amino acids (alanine, aspartic acid, glutamic acid, and glycine) increase the ability of the kidneys to excrete uric acid.
- Dark reddish-blue berries (such as cherries and blackberries) are good sources of flavonoids, which help lower uric acid levels, decrease inflammation, and prevent or repair joint tissue damage.
- Acupuncture can provide pain relief.

Treatments

Treatments for gout, in addition to medications, include dietary management and rest.

NUTRITION Dietary purines contribute only slightly to uric acid levels in the body, and no specific diet may be recommended. If a low-purine diet is recommended, the client should be taught that

MEDICATION ADMINISTRATION The Client with Gout

COLCHICINE

Colchicine is used to terminate an acute attack of gouty arthritis and to prevent recurrent episodes of the disease. Colchicine does not alter serum uric acid levels, but appears to interrupt the cycle of urate crystal deposition and inflammatory response. It may be administered either by mouth or intravenously. Colchicine is also available as a fixed-dose combination with a uricosuric agent, probenecid (Benemid). Only plain colchicine is used to treat an acute attack of gout; combination therapy is employed to prevent further attacks.

Nursing Responsibilities

- Assess for possible contraindications to colchicines therapy, including serious gastrointestinal, renal, hepatic, or cardiac disease.
- Administer the following as ordered:
  - *Intravenous doses*: Give undiluted or diluted in up to 20 mL sterile normal saline for injection. Administer over a period of 2 to 5 minutes.
  - *Oral doses:* Give on an empty stomach to facilitate absorption.
  - Evaluate for adverse effects, including abdominal cramping, nausea, vomiting, and diarrhea, and report promptly, because these side effects may necessitate discontinuation of the drug.

Health Education for the Client and Family

- Drink 3 to 4 quarts of liquid per day.
- Report adverse responses, including gastrointestinal problems, fatigue, bleeding, easy bruising, or recurrent infections, to the physician.
- Do not drink alcohol.

URICOSURIC DRUGS

Probenecid (Benemid)
Sulfinpyrazone (Anturane)

Probenecid is a uricosuric drug that inhibits the tubular reabsorption of urate, promoting the excretion of uric acid and decreasing serum uric acid levels. Sulfinpyrazone is a uricosuric drug that potentiates the renal excretion of uric acid, reducing serum uric acid levels. It is used to prevent recurrent attacks of acute gouty arthritis and treat chronic gout.

Nursing Responsibilities

- Assess for prior hypersensitivity responses to this drug.
- Administer after meals or with milk to minimize gastric distress.
- Increase fluid intake to at least 3 L/day to prevent the formation of uric acid kidney calculi.
- Administer sodium bicarbonate or potassium citrate as ordered to maintain an alkaline urine.
- Do not administer aspirin to clients receiving probenecid because salicylates interfere with the action of the drug.
- Monitor clients receiving the following drugs concurrently with probenecid for increased or toxic effects: penicillin and related antibiotics, indomethacin, acetaminophen, naproxen, ketoprofen, meclofenamate, lorazepam, and rifampin.
- Monitor for possible adverse effects of probenecid, including headache, dizziness, hepatic necrosis, nausea and vomiting, renal colic, bone marrow depression, anaphylaxis, fever, hives, and pruritus.
- Administer sulfinpyrazone with meals or antacid to minimize gastric distress.
- Monitor clients taking sulfinpyrazone with other sulfa drugs for increased or toxic effects; monitor for hypoglycemia in clients receiving insulin or oral hypoglycemics concurrently, and monitor for bleeding or increased anticoagulant effect in clients receiving warfarin concurrently.
- Assess for contraindications to therapy with sulfinpyrazone, including active peptic ulcer disease, a history of hypersensitivity to phenylbutazone or other pyrazoles, or blood dyscrasias.

Health Education for the Client and Family

- Do not take aspirin or products containing aspirin while taking probenecid. Use acetaminophen for relief of mild pain.
- Drink at least 3 quarts of fluids per day to minimize the risk of kidney stone formation.
- Take sulfinpyrazone with meals to minimize gastric distress, and report epigastric pain, nausea, or black stools to the physician promptly

ALLOPURINOL (ZYLOPRIM)

Allopurinol acts on purine metabolism, reducing the production of uric acid and decreasing serum and urinary concentrations of uric acid. It is used for clients with manifestations of primary or secondary gout, including acute attacks, tophi, joint destruction, urinary stones, and nephropathy. It is not indicated for use in the treatment of asymptomatic hyperuricemia.

Nursing Responsibilities

- Monitor intake and output and increase fluid intake to approximately 3 L/day.
- Monitor for desired effect of decreased serum uric acid levels, and for adverse effects such as nausea, diarrhea, and rash.
- Assess BUN and creatinine levels prior to the initiation of and during treatment with allopurinol. Report signs of impaired renal function such as an elevated BUN and creatinine, decreased urine output, and dilute or frothy urine to the physician.
- Administer with meals to minimize gastric distress.
- Monitor CBC periodically because allopurinol therapy may cause bone marrow depression.
- In clients receiving warfarin concurrently, monitor prothrombin times and be alert to evidence of bleeding, because allopurinol prolongs the half-life of warfarin.
- Monitor clients receiving chlorpropamide, cyclophosphamide, hydantoin, theophylline, vidarabine, or ACE inhibitors concurrently for increased drug effects.
- Discontinue the drug and notify the physician immediately if the client develops a rash. Rash and hypersensitivity responses occur more frequently in clients receiving ampicillin, amoxicillin, or thiazide diuretics.

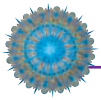
Health Education for the Client and Family

- Stop taking the drug and report any skin rash, painful urination, blood in the urine, eye irritation, or swelling of the lips or mouth to the physician immediately.
- Take the medication after meals to minimize gastric distress.
- Drink 3 to 4 quarts of fluid daily to maintain a urinary output greater than 2 L/day.
- Acute gouty attacks may occur during the initial stages of allopurinol therapy; continue therapy prescribed for attacks (such as colchicine) to minimize acute episodes.
- Do not take a double dose of medication if you miss a dose.

high purine foods include all meats and seafood, yeast, beans, peas, lentils, oatmeal, spinach, asparagus, cauliflower, and mushrooms. The obese client is advised to lose weight, but fasting is contraindicated for clients with gout. Alcohol intake and specific foods that tend to precipitate attacks are avoided.

A liberal fluid intake to maintain a daily urinary output of 2000 mL or more is recommended to increase urate excretion and reduce the risk of urinary stone formation. Urinary alkalinizing agents, such as sodium bicarbonate or potassium citrate, may be prescribed as well to minimize the risk of uric acid stones. It is important to monitor clients receiving these preparations carefully for signs of fluid and electrolyte or acid–base imbalances.

REST During an acute attack of gouty arthritis, bed rest is prescribed. It is continued for approximately 24 hours after the attack has subsided, because early ambulation may bring about recurrence of acute manifestations (Tierney et al., 2004). The affected joint may be elevated and hot or cold compresses may be applied for comfort.



NURSING CARE

Clients with gout provide self-care at home. Teaching focuses on self-management of pain and altered mobility.

Nursing Diagnoses and Interventions

Pain is a primary focus for nursing interventions in the client experiencing an acute attack of gout. The client's mobility is also impaired during an acute attack, both because of pain and prescribed activity limitations.

Acute Pain

The pain associated with an attack of acute gouty arthritis is intense and accompanied by exquisite tenderness of the affected joint. Measures to alleviate the pain are vital in the initial period until anti-inflammatory medications become effective and the acute inflammatory response is relieved. The following are important in teaching about pain relief:

- Position the affected joint for comfort. Elevate the joint or extremity (usually the foot) on a pillow, maintaining alignment. *Elevation and normal body alignment facilitate blood return from the affected joint, alleviating some of the edema.*
- Protect the affected joint from pressure, placing a foot cradle on the bed to keep bed covers off the foot. *A foot cradle keeps bed linens from applying pressure on the affected joint.*
- Take anti-inflammatory and antigout medications as prescribed. In the initial period, colchicine may be given hourly. *These medications reduce the acute inflammatory response, gradually relieving discomfort.*

PRACTICE ALERT

The affected joints are so painful that even the weight of a sheet can be unbearable.

- Take analgesics as prescribed. *Supplemental analgesia may be necessary in the acute period until the inflammatory response is mediated.*
- Maintain bed rest. *It is important to immobilize the affected joint and promote rest to prevent exacerbation of joint inflammation.*

Community-Based Care

Discuss the following topics with the client:

- *The disease and its manifestations.* Tell the client that initial attacks cause no permanent damage but that recurrent attacks can lead to permanent damage and joint destruction. Discuss other potential effects of continued hyperuricemia, including tophaceous deposits in subcutaneous and other connective tissues. Discuss the potential for kidney damage and kidney stones.
- *The rationale for and use of prescribed medication.* Stress the need to continue the medication until the physician discontinues it, even though the client is free of manifestations of gout. Tell the client to avoid drugs that increase uric acid blood levels: hydrochlorothiazide (HydroDIURIL), cyclosporine (Neoral, Sandimmune), furosemide (Lasix), and high dose of aspirin. Clients who need to reduce their risk of heart attacks may safely take one baby aspirin each day (Flynn & Johnson, 2005).
- The importance of a high intake of fluids each day and avoiding the use of alcohol.

THE CLIENT WITH OSTEOMALACIA

Osteomalacia, often referred to as *adult rickets,* is a metabolic bone disorder characterized by inadequate or delayed mineralization of bone matrix in mature compact and spongy bone, resulting in softening of bones. Bone mineralization requires adequate calcium and phosphate ions in extracellular fluid. When either of these ions is insufficient due to (1) inadequate calcium intake or decreased calcium absorption from the intestines because of insufficient vitamin D, or (2) increased renal losses or decreased intestinal absorption of phosphate, the bone matrix is not mineralized and cannot sustain weight bearing. Marked deformities of weight-bearing bone and pathologic fractures occur. The primary causes of osteomalacia are vitamin D deficiency and hypophosphatemia. Osteomalacia can be corrected with treatment.

Osteomalacia has been almost nonexistent in the United States because many foods are fortified with vitamin D, but its incidence is increasing among older adults, and people who adhere to strict vegetarian diets. It is a significant health problem in cultures whose diets tend to be deficient in calcium and vitamin D. Women in northern China, Japan, and northern India have a higher incidence of the disorder (Porth, 2005).

The major risk factors for vitamin D deficiency are a diet low in vitamin D, decreased endogenous production of vitamin D because of inadequate sun exposure, impaired intestinal absorption of fats (vitamin D is a fat-soluble vitamin), and disorders that interfere with the metabolism of vitamin D to its active forms. Gastrectomy and small-bowel disorders may reduce the absorptive surface of the bowel to the extent that nutrients are not completely or adequately absorbed.

Both vitamin D and calcium absorption may be affected. Hepatobiliary disorders that interfere with bile production and release, and chronic pancreatic insufficiency with inadequate pancreatic enzyme production can affect the absorption of fats and vitamin D from the bowel. Once absorbed, vitamin D is metabolized in the liver and the kidney to its active form; therefore, liver disorders such as cirrhosis and renal disorders can affect this activation. Certain drugs, such as isoniazid, rifampin, and anticonvulsants, accelerate vitamin D metabolism, resulting in less availability to the tissues. Renal excretion of vitamin D is increased in some kidney disorders such as nephrotic syndrome (Box 42–2).

Hypophosphatemia can be the result of insufficient dietary intake, excessive losses through the urine or stool, or a shift into the cells. Alcohol abuse is the most common cause of hypophosphatemia, because of related dietary deficiencies, vomiting, antacid use, and increased renal excretion of phosphate. Ingesting large amounts of nonabsorbable antacids causes increased phosphate losses in the stool. Several acquired and genetic disorders cause increased losses of phosphate in the urine.

Pathophysiology

The two main causes of osteomalacia are insufficient calcium absorption in the intestine due to a lack of calcium or resistance to the action of vitamin D and increased losses of phosphorus through the urine (Porth, 2005). In its natural form, vitamin D is obtained from certain foods and ultraviolet radiation of the sun. Vitamin D maintains adequate serum levels of calcium and phosphate for normal mineralization of the bone. Vitamin D deficiency or resistance to its action disrupts the normal mineralization of the bone, causing softening of the bone.

Vitamin D is inactive when it is absorbed from the intestine or synthesized from exposure to ultraviolet light. For vitamin D to become active, a two-step process must occur. Vitamin D (and its metabolites) is transported in the blood to the liver, where it is converted to calcidiol. Calcidiol is then transported to the kidney and transformed to an active form, calcitriol.

The active form of vitamin D is needed for optimal absorption of calcium and phosphorus from the intestine. Calcium and phosphorus are transported in the blood to the bones for normal mineralization. If there is a lack of vitamin D, calcium and phosphorus are not absorbed from the intestine, and serum calcium and phosphorus levels therefore fall. A deficiency in these minerals in turn activates the parathyroid glands, with loss of calcium and phosphorus from bone. The continued loss of calcium and phosphate in the bone disrupts bone mineralization.

Impaired bone mineralization causes abnormalities in both spongy and compact bone. The osteoid (the soft, noncalcified part of the matrix) continues to be produced but is not mineralized. This abnormal buildup of demineralized bone leads to gross deformities of the long bones, spine, pelvis, and skull, because the bone is soft and unable to bear the weight and stress of body movement.

Manifestations

The manifestations of osteomalacia include bone pain and tenderness (see the box below). As the disease progresses, fractures occur. In contrast to osteoporosis, osteomalacia is not associated with a significant occurrence of hip fractures. Instead, pathologic fractures occur in the commonly weakened areas (e.g., distal radius and proximal femur).

BOX 42–2 Causes of Osteomalacia

Vitamin D Deficiency
- Inadequate dietary intake
- Lack of sun exposure
- Malabsorption from intestines: gastrectomy, small-bowel disorders, gallbladder disease, chronic pancreatic insufficiency
- Renal or liver disorders
- Drug effects: isoniazid, rifampin, anticonvulsants

Phosphate Depletion
- Inadequate intake
- Impaired absorption due to chronic antacid use
- Impaired renal tubular reabsorption due to either acquired or genetic disorders

Systemic Acidosis
- Renal tubular acidosis
- Ureterosigmoidostomy
- Fanconi's syndrome

Bone Mineralization Inhibitors
- Hypophosphatasia
- Sodium fluoride or disodium etidronate (Didronel)
- Aluminum intoxication

Chronic Renal Failure

Calcium Malabsorption

INTERDISCIPLINARY CARE

Osteomalacia may be difficult to differentiate from osteoporosis because the manifestations are very similar; however, once the specific cause is determined, appropriate therapy will correct the disorder.

Diagnosis

A history of inadequate dietary intake, renal failure, or some malabsorption states may suggest osteomalacia. Diagnostic

MANIFESTATIONS of Osteomalacia

- Bone pain: May be vague and generalized at first, becoming more intense with activity as the disease progresses; occurs most frequently in the pelvis; long bones of the extremities, spine, and ribs.
- Difficulty changing from lying to sitting position and sitting to standing position.
- Muscle weakness: Frequently an early sign in severe cases.
- Waddling gait: May be due to pain and muscle weakness.
- Dorsal kyphosis: May occur in severe cases.
- Pathologic fractures.

tests are described in Chapter 40. Table 42–1 compares the diagnostic findings of osteomalacia with those of osteoporosis and Paget's disease. X-rays demonstrate the effects of generalized bone demineralization: trabecular bone loss, cyst formation, compression fractures, bowing and bending deformities of the long bones, and osteoid deposits, particularly in the vertebral bodies and pelvis.

Laboratory tests include serum calcium, parathyroid hormone, and alkaline phosphatase levels. Calcium may be normal or low, depending on the cause of the disease. Calcium levels may be reduced when calcium absorption is impaired or in severe vitamin D deficiency. Secondary hypoparathyroidism may shift calcium from the bone into extracellular fluid, maintaining a normal serum calcium level. Parathyroid hormone is frequently elevated as a compensatory response to hypocalcemia in renal failure or vitamin D deficiency. Alkaline phosphatase usually is elevated.

Medications

Therapeutic management of osteomalacia depends on the cause of the disease. Because the causes are so diverse, it is difficult to generalize treatment. Most clients are placed on vitamin D therapy. Calcium and phosphate supplements may be indicated. Radiologic evidence of healing often is apparent within weeks of initiating therapy.

NURSING CARE

Managing the client with osteomalacia includes assessing the client's current dietary intake of vitamin D, calcium, and phosphorus and exposure to ultraviolet light. It also includes managing client responses to bone pain and tenderness, fractures, and muscle weakness.

Teaching is important not only for the client with osteomalacia, but also for people at risk for developing the disease. When milk and other dairy products began to be fortified with vitamin D, the incidence of childhood rickets decreased dramatically. Now many clients are unaware of the importance of vitamin D, calcium, and phosphorus to bone health.

Older adults as a group are at high risk for osteomalacia because of dietary deficiencies, age-related intestinal malabsorption, and possible physical mobility limitations that restrict their exposure to sunlight. Teach older adults about the importance of maintaining an adequate intake of milk and other dairy products that are not only rich in calcium and phosphorus, but also are fortified with vitamin D. Few other food sources provide enough vitamin D to meet recommended levels. Cod liver oil may be used as a supplement, because it contains significant amounts of vitamin D. Supplements are not recommended, however, for clients who get adequate vitamin D through dietary sources and sun exposure, because this fat-soluble vitamin may become toxic at high levels. Instruct clients who are taking supplements to report to their primary care provider symptoms such as anorexia, nausea and vomiting, frequent urination, muscle weakness, and constipation that may be indicative of hypervitaminosis D.

Teach the client with osteomalacia about safety measures to prevent falls. Discuss the importance of eliminating scatter rugs and clutter from living areas to prevent tripping. Teach the client to place a night-light in hallways and the bathroom to prevent falls associated with nighttime toileting. Suggest installing grab bars in the shower and tub and next to the toilet for safety.

Teach clients with bone pain and muscle weakness to use assistive devices such as walkers, canes, or crutches when ambulating. Provide referrals to physical therapy for teaching clients how to safely use these devices. Encourage clients to participate in a supervised exercise program such as water aerobics or t'ai chi to improve muscle strength and balance.

DEGENERATIVE DISORDERS

Degenerative disorders, especially degenerative joint disease, are the most common form of arthritis in the older adult. Both primary and secondary forms are seen in adults of all ages. Primary or idiopathic osteoarthritis, the most common type, occurs without a clear precipitating factor. Secondary osteoarthritis is associated with an identifiable cause. For instance, it may be related to trauma to a joint, inflammation, skeletal disorders such as congenital hip dysplasia, or metabolic disorders. Regardless of cause, degenerative disorders of the joints and muscles can lead to impaired mobility and chronic pain. These problems may in turn cause disability, especially in the performance of ADLs by older adults.

THE CLIENT WITH OSTEOARTHRITIS

Osteoarthritis (OA) (also labeled *degenerative joint disease*) is the most commonly occurring of all forms of arthritis, and a leading cause of pain and disability in older adults (Porth, 2005). This disease is characterized by loss of articular cartilage in articulating joints and hypertrophy of the bones at the articular margins. OA may be idiopathic (without known cause) or secondary (associated with known risk factors). OA affects more than 12% of Americans between the ages of 25 and 74 years, with about 90% of people having x-ray evidence of OA in the weight-bearing joints by age 40 (ACR, 2005; Flynn & Johnson, 2005). Men are affected more than women at an earlier age, but the rate of OA in women exceeds men by the middle adult years. The joints most affected are in the hand, wrist, neck, lower back, hip, knee, ankle, and feet. Men are more likely than women to have hip OA, whereas postmenopausal women more often have hand OA. Racial and ethnic effects on the development of OA are outlined in the box below.

FOCUS ON DIVERSITY
The Client with Osteoarthritis

- White women are more likely to have hand OA.
- Black women are more likely to have knee OA.
- Hip OA incidence is less in Chinese people.

Localized OA affects only one or two joints. Generalized OA affects three or more joints. Generalized OA may also be classified as nodal (involving the hand) or nonnodal (no hand involvement). Nodal OA may also affect the knees, hips, cervical spine, and lumbar spine. Idiopathic OA most commonly affects the terminal interphalangeal joints (*Heberden's nodes*), and less often the proximal interphalangeal joints (*Bouchard's nodes*) (Figure 42–2 ■), the joints of the thumb, the hip, the knee, the metatarsophalangeal joint of the big toe, and the cervical and lumbar spine. Secondary OA may occur in any joint from an articular injury.

Risk Factors

Idiopathic OA is associated with increasing age. It has been suggested that OA may be inherited as an autosomal recessive trait, with genetic defects causing premature destruction of the joint cartilage. The causes of secondary OA include trauma, mechanical stress, and inflammation of joint structures, joint instability, neurologic disorders, endocrine disorders, and selected medications.

Excessive weight contributes to the development of OA, especially in the hip and knee. Excess fat may have a direct metabolic effect in the development of the disease. Primary OA of the knee is almost four times more common in obese women and five times more common in obese men (Flynn & Johnson, 2005). Inactivity is another risk factor. Moderate recreational exercise has been shown to both decrease the chance of developing OA and the progression of manifestations when OA is present. People involved in strenuous, repetitive exercise (such as participating in sports) have an increased risk of developing secondary OA.

Other risk factors that are linked to OA are hormonal factors such as decreased estrogen in menopausal women, excessive growth hormone, and increased parathyroid hormone.

Pathophysiology

The cartilage that lines joints provides a smooth surface, so that the bones of the joint glide over one another without friction, and it distributes the load from one bone to the next, dissipating the mechanical stress that occurs with joint loading. This cartilage normally contains more than 70% water. More than 90% of its dry weight is collagen, which provides strength, and proteoglycans, which provide elasticity and stiffness to compression. Cartilage cells, the chondrocytes, nest in this meshwork of collagen and proteoglycans. Normal articular cartilage exudes some of its water with compression, providing lubrication for joint surfaces. This water is reabsorbed during relaxation of the joint.

In OA, proteoglycans and collagen are lost from the cartilage as a result of enzymatic degradation. The water content of the cartilage increases as the collagen matrix is destroyed. With the loss of proteoglycans and collagen fibers, the cartilage becomes yellow or brownish gray and loses its tensile strength. Surface ulcerations occur, and fissures develop in deeper layers of the cartilage. Eventually, large areas of articular cartilage are lost, and underlying bone is exposed. The bone thickens in exposed areas, reducing its ability to absorb energy in joint loading. Cysts can also develop in the bone. Cartilage-coated *osteophytes* (bony outgrowths often called "joint mice") change the anatomy of the joint. As these spurs or projections enlarge, small pieces may break off, leading to mild synovitis (inflammation of the synovial membrane).

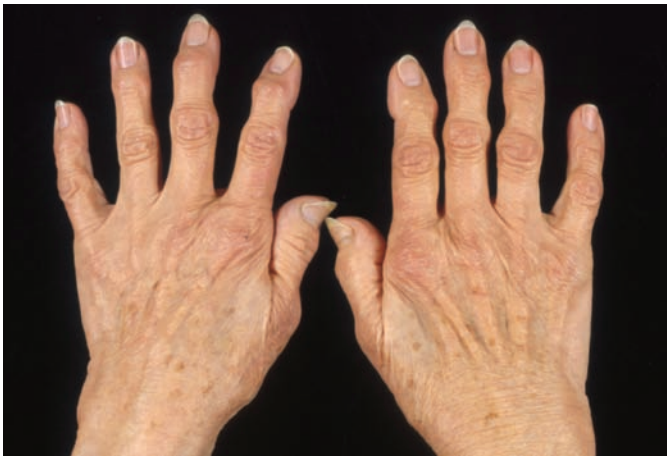


Figure 42–2 ■ Typical interphalangeal joint changes associated with osteoarthritis.

Source: L. Samsuri/Custom Medical Stock Photo.

Manifestations

The onset of OA is usually gradual and insidious, and the course slowly progressive. Pain and stiffness in one or more joints (usually weight bearing) are the first manifestations of OA. The pain is localized to the affected joints and may be described as a deep ache. It typically is aggravated by use or motion of the joint and relieved by rest, although it may become persistent as the disease progresses. Pain at night may be accompanied by paresthesias (numbness, tingling). Pain may also be referred to other parts of the body; for example, OA of the lumbosacral spine may cause severe pain along the path of the sciatic nerve. Following periods of immobility, such as sleeping all night or after a long automobile ride, involved joints may stiffen. Usually only a few minutes of activity are necessary to relieve the stiffness. Range of motion (ROM) of the joint decreases as the disease progresses, and grating or crepitus may be noted during movement. Bony overgrowth may cause joint enlargement, and flexion contractures may occur because of joint instability. In OA, enlarged joints are characteristically bony-hard and cool on palpation. Manifestations specific to affected joints are outlined in the box on the next page.

Complications

OA of the spine may involve the vertebral bodies and intervertebral disks, the diarthrodial joints, or both. *Spondylosis* is degenerative disk disease. As the intervertebral disks degenerate, disk space between the vertebrae is lost. Degenerative disk disease may be complicated by herniated disk, the protrusion of the nucleus pulposus of the disk. Herniation usually occurs in a lateral direction, potentially compressing nerve roots and causing radicular (distributed along the nerve) pain and muscle weakness. See Chapter 45 ∞ for further discussion of disk disorders.

MANIFESTATIONS of Osteoarthritis

Affected Site	Manifestations
Interphalangeal joints	■ *Heberden's nodes*—bony enlargements of distal joints; may cause pain, redness, swelling ■ *Bouchard's nodes*—bony enlargement of proximal joints
First carpometacarpal	■ Swelling, tenderness at base of thumb ■ Crepitus with movement ■ "Squared" appearance of joint
Spine	■ Localized pain and stiffness ■ Muscle spasm ■ Limited range of motion ■ Nerve root compression with radicular pain and motor weakness
Hips	■ Pain referred to inguinal area, buttock, thigh, or knee ■ Loss of internal rotation ■ Limited extension, adduction, and flexion
Knees	■ Pain and bony enlargement ■ Effusions ■ Crepitus ■ Instability and deformity with advanced disease

Disk degeneration and joint space narrowing alter the mechanics of the spinal column, promoting osteoarthritic changes in the articular processes (the facet joints) of the vertebrae. The cartilage covering the inferior and superior articular processes degenerates, causing localized pain, stiffness, muscle spasm, and limited range of motion. Osteophytes may form on articular processes, further contributing to pain and muscle spasm.

The presentation of OA in older clients is similar to that in younger adults. However, in this population, the risk of debilitation because of OA is greater, and the disease may progress faster. In addition, pain, stiffness, and limited ROM increase the risk of falls and fractures in the older adult.

INTERDISCIPLINARY CARE

At this time, no treatment is available to arrest the process of joint degeneration. Appropriate management, however, is important to relieve pain and maintain the client's function and mobility. Research is also ongoing on a new class of medications called disease-modifying osteoarthritis drugs (DMOADs) and gene therapy.

Diagnosis

The diagnosis of OA is generally based on the client's history, physical examination, and x-rays of affected joints. Diagnostic tests are described in Chapter 40 ∞.

Characteristic changes of OA are visible in x-ray studies of affected joints. Initially, irregular joint space narrowing is seen. Progressive changes include increased density of subchondral (under cartilage) bone, osteophyte formation at the joint periphery, and the formation of cysts in the bone. Examination of synovial fluid from involved joints can identify the type of arthritis. In addition, research of the blood level of hyaluronic acid (HA) (a lubricating substance in cartilage and joint synovial fluid) suggests that HA may be a useful biochemical marker indicating the presence and severity of OA (National Institute of Arthritis Musculoskeletal and Skin Diseases, 2005c).

Medications

The pain of OA often can be managed through the use of analgesics such as aspirin or acetaminophen. Acetaminophen (Tylenol) is generally preferred for use in older clients because it has fewer toxic side effects. NSAIDs such as ibuprofen (Motrin), naproxen (Aleve), or ketoprofen (Orudis KT) may also be prescribed. See information with medications for rheumatoid arthritis for FDA actions concerning these drugs. These medications are discussed in more detail in Chapter 9 ∞.

Topical medications include counterirritants, salicylates, and capsaicin, sold without prescription as creams, gels, sprays, patches, or ointments to relieve pain. Counterirritants include Flexall 454 Maximum Strength Gel, ArthriCare, Bengay, and Icy Hot; salicylates include Aspercreme and Sportscreme, and capsaicin is included in Capzasin and Zostrix. The client should be taught to keep the medications away from their eyes, nose, mouth, or any open skin, and not to bandage or apply heat to the treated area. The products should be used no more than three or four times a day and discontinued immediately if severe irritation occurs.

Medications that are effective in decreasing the pain and stiffness of OA are the NSAID COX-2 inhibitors. However, because of the increased risk of adverse cardiovascular (heart attack and strokes) and gastrointestinal (bleeding) effects of most drugs in this category, several have been recalled by the FDA and the only COX-2 inhibitor being prescribed as of 2006 was celecoxib (Celebrex).

Potent anti-inflammatory medications, such as systemic corticosteroids, are seldom prescribed for clients with OA, although intra-articular corticosteroid injections may be used. With intra-articular injections, a long-acting corticosteroid medication, often mixed with a local anesthetic such as lidocaine, is injected directly into the joint space of the affected joints. Although this procedure may provide marked pain relief, it can hasten the rate of cartilage breakdown if performed more frequently than every 4 to 6 months.

Treatments

OA is initially treated conservatively, but as pain increases and joint function decreases, surgery often becomes necessary.

CONSERVATIVE TREATMENT The goals of OA treatment are to relieve pain and maintain as much normal joint function as possible. Conservative treatment may include any or all of the following:

- ROM exercises, muscle strengthening exercises, aerobic exercises
- Heat and ice
- A balance between exercise and rest

- Use of a cane, crutches, or a walker
- Weight loss, if indicated
- Analgesic and anti-inflammatory medications.

VISCOSUPPLEMENTATION Viscosupplementation is a new treatment for OA of the knee. Hyaluronan, a natural component of synovial fluid, is injected directly into the knee joint. Four hyaluronan derivatives have been approved for use: Hyalgan, Supartz, Orthovisc, and Synvisc. The injection may provide pain relief and improvement in knee function for up to 1 year, but its long-term effects are unknown (Flynn & Johnson, 2005).

SURGERY Surgical procedures can provide dramatic results for clients with significant chronic pain and loss of joint function. Although elective surgical procedures are frequently avoided in the older adult, even older clients can benefit significantly if they do not have a chronic medical condition that contraindicates surgery.

Arthroscopy An *arthroscopy* is a surgical procedure in which an arthroscope (a thin tube that is lighted and has a camera in one end) is inserted into a joint. It may be done to diagnose the type of arthritis or to perform debridement by smoothing rough cartilage and flushing out the joint to remove debris. Although arthroscopic debridement and lavage of involved joints have been used, arthroscopy has not proven effective in the treatment of knee OA. It may be useful to remove large pieces of debris or repair a torn cartilage (Flynn & Johnson, 2005).

Osteotomy An *osteotomy*, an incision into or transection of the bone, may be performed to realign an affected joint, particularly when significant bony overgrowth or osteophyte formation has occurred. This procedure may also be used to shift the joint load toward areas of less severely damaged cartilage. Although osteotomy does not halt the process of OA, it may have a beneficial effect on joint function and pain, delaying the need for a joint replacement by several years.

Joint Arthroplasty A *joint arthroplasty* is the reconstruction or replacement of a joint. Arthroplasty is usually indicated when the client has severely restricted joint mobility and pain at rest. Pain is virtually eliminated, and the function of the joint is generally improved. Arthroplasty may involve partial joint replacement or reshaping of the bones of a joint. For most clients with OA, both surfaces of the affected joint are replaced with prosthetic parts in a procedure known as a *total joint replacement*. Joints that may be replaced include the hip, knee, shoulder, elbow, ankle, wrist, and joints of the fingers and toes.

In a total joint replacement, some or all of the synovium, cartilage, and bone on both sides of the joint are removed. A metallic prosthesis is inserted to replace one joint surface (generally the load-end or distal portion of a weight-bearing joint). The other joint surface is replaced by a silicone-lined ceramic or plastic prosthesis.

Most prosthetic joints are uncemented, that is, made of porous ceramic and metal components inserted so that they fit tightly into existing bone. The implant is secured by new bone growth into the prosthesis, a process that requires approximately 6 weeks. Although a longer non-weight-bearing period is necessary initially until the prosthesis is fixed in place by the bony growth, the implant appears to have a longer useful life span than cemented prostheses. In a cemented joint replacement, methyl methacrylate (a pliable polymer that hardens to hold the prosthesis in place) is used to secure the prosthesis to existing bone. Although the client is able to resume normal activities more rapidly following a cemented joint replacement, methyl methacrylate initiates an inflammatory response, and the joint eventually loosens.

- In a *total hip replacement,* the articular surfaces of the acetabulum and femoral head are replaced. The entire head of the femur and part of the femoral neck are removed and replaced with a prosthesis (Figure 42–3 ■). The acetabulum is remodeled, and a prosthesis of high-molecular-weight polyethylene is inserted. The success rate for total hip replacement is reported to be greater than 90%. Approximately 150,000 total hip replacements are done each year in the United States; most are for treatment of OA (Boston Total Joint Association, 2004). Most hip replacements last 10 to 15 years, after which a second joint replacement, called a revision, can be performed. Potential problems associated with a total hip replacement include blood clots in leg veins, dislocation within the prosthesis, loosening of joint components from surrounding bone, and infection. If recurrent or ineffectively treated, these complications may necessitate removal of the prosthesis, resulting in severe shortening of the extremity and an unstable hip joint.
- *Total knee replacement* is performed if the client has intractable pain and x-ray films show evidence of arthritis of the knee. More than 350,000 knee replacements are performed in the United States each year (Flynn & Johnson, 2005). Several prosthetic devices involving removal of varying amounts of bone are available for knee joint replacement (Figure 42–4 ■). The femoral side of the joint is replaced with a metallic surface, and the tibial side with polyethylene. More than 80% of clients obtain significant or total relief of pain with a total knee replacement. They must, however, engage in a vigorous program of rehabili-

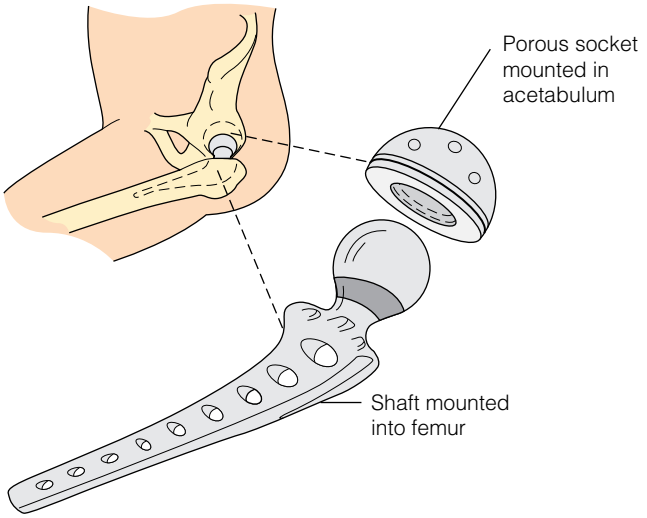
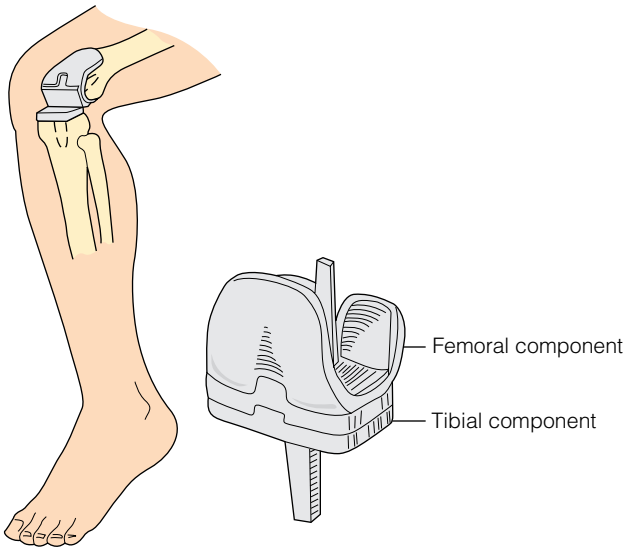


Figure 42–3 ■ Total hip prosthesis.



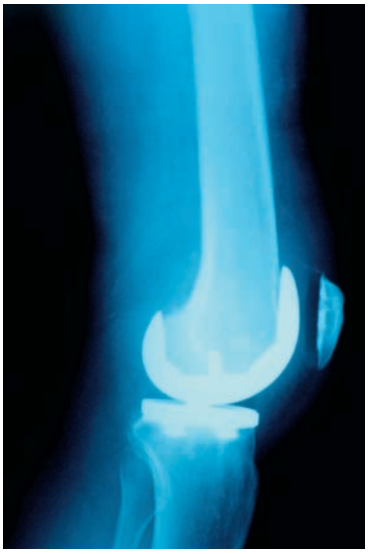


Figure 42–4 ■ Total knee replacement.

tation to achieve the best results. Joint failure is more common with knee replacement than with a total hip replacement. Loosened joint components, often on the tibial side, are the most common cause of failure. The possible complications following a total knee replacement are the same as for a total hip replacement.

- *Total shoulder replacement* is indicated for unremitting pain and marked limitation of range of motion because of arthritic involvement of both the humeral and glenoid joint surfaces of the shoulder. The joint is immobilized in a sling or abduction splint for 2 to 3 weeks following arthroplasty. Dislocation, loosening of the prosthesis, and infection are potential problems associated with total shoulder replacement.
- *Total elbow replacement* involves replacement of the humeral and ulnar surfaces of the elbow joint with a metal and polyethylene prosthesis. Pain and disabling stiffness of the joint are indications for an elbow arthroplasty. Complications, including dislocation, fracture, tricep weakness, loosening, and infection, occur frequently.

Infection is the major complication associated with total joint replacement. Not only does infection interfere with healing and prolong recovery, but it may also necessitate removal of the prosthesis and may lead to loss of joint function. Other potential complications include circulatory impairment to the affected limb, thromboembolism, nerve damage, and dislocation of the joint.

Nursing care for the client undergoing total joint replacement is outlined in the box below. Refer to Chapter 4 ∞ for further discussion of care for the client undergoing surgery.

NURSING CARE OF THE CLIENT HAVING **Total Joint Replacement**

PREOPERATIVE CARE

- Assess the client knowledge and understanding of the planned operative procedure. Provide further explanations and clarification as needed. *It is important that the client have a clear and realistic understanding of the surgical procedure and expected results. Knowledge decreases anxiety and increases the client's ability to assist with postoperative care procedures.*
- Obtain a health history and physical assessment, including range of motion of the affected joints. *This information not only allows nurses to tailor care to the needs of the individual but also serves as a baseline for comparison of postoperative assessment data.*
- Explain necessary postoperative activity restrictions. Teach how to use the overhead trapeze for changing positions. *The client who learns and practices moving techniques before surgery can use them more effectively in the postoperative period.*
- Provide or reinforce teaching of postoperative exercises specific to the joint on which surgery is to be performed. *Exercises are prescribed postoperatively to (a) strengthen muscles providing joint stability and support, (b) prevent muscle atrophy and joint contractures; and (c) prevent venous stasis and possible thromboembolism.*
- Teach respiratory hygiene procedures such as the use of incentive spirometry, coughing, and deep breathing. *Adequate respiratory hygiene is imperative for all clients undergoing joint replacement to prevent respiratory complications associated with immobility and the effects of anesthesia. In addition, many clients undergoing total joint replacement are elderly and may have reduced mucociliary clearance.*

(continued)

NURSING CARE OF THE CLIENT HAVING **Total Joint Replacement (continued)**

- Discuss postoperative pain control measures, including use of patient-controlled analgesia (PCA) or epidural infusion as appropriate. *It is important for the client to understand the purpose and use of postoperative pain control measures to allow early mobility and reduce complications associated with immobility*.
- Teach or provide prescribed preoperative skin preparation such as shower, shampoo, and skin scrub with antibacterial solution. *These measures help reduce transient bacteria that may be introduced into the surgical site*.
- Administer intravenous antibiotic as ordered. *Antibiotic therapy is initiated before or during surgery and continued postoperatively to further reduce the risk of infection*.

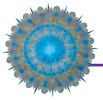
POSTOPERATIVE CARE

- Monitor vital signs, including temperature and level of consciousness, every 4 hours or more frequently as indicated. Report significant changes to the physician. *These routine assessments provide information about the client's cardiovascular status and can give early indications of complications such as excessive bleeding, fluid volume deficit, and infection*.
- Perform neurovascular checks (color, temperature, pulses and capillary refill, movement, and sensation) on the affected limb hourly for the first 12 to 24 hours, then every 2 to 4 hours. Report abnormal findings to the physician immediately. *Surgery can disrupt the blood supply to or innervation of the affected extremity. If so, rapid intervention is important to preserve the function of the extremity*.
- Monitor incisional bleeding by emptying and recording suction drainage every 4 hours and assessing the dressing frequently. *Significant blood loss can occur with a total joint replacement, particularly a total hip replacement*.
- Reinforce the dressing as needed. *The dressing is usually changed 24 to 48 hours after surgery but may need reinforcement if excess bleeding occurs*.
- Maintain intravenous infusion and accurate intake and output records during the initial postoperative period. *The client is at risk for fluid volume deficit in the initial postoperative period because of blood and fluid loss during surgery, as well as the effects of the anesthetic*.
- Maintain bed rest and prescribed position of the affected extremity using a sling, abduction splint, brace, immobilizer, or other prescribed device. *Proper positioning of the affected extremity is vital in the initial postoperative period so that the joint prosthesis does not become dislocated or displaced*.
- Help the client shift position at least every 2 hours while on bed rest. *Shifting of position helps prevent pressure ulcers and other complications of immobility*.
- Remind the client to use the incentive spirometer, to cough, and to breathe deeply at least every 2 hours. *These measures are important to prevent respiratory complications such as pneumonia*.
- Assess the client's level of comfort frequently. Maintain PCA, epidural infusion, or other prescribed analgesia to promote comfort. *Adequate pain management promotes healing and mobility*.
- Help the client get out of bed as soon as allowed. Teach and reinforce the use of techniques to prevent weight bearing on the affected extremity, such as the overhead trapeze, pivot turning, and toe touch. *Early mobility prevents complications such as pneumonia and thromboembolism, but appropriate techniques must be used to prevent injury to the operative site*.
- Initiate physical therapy and exercises as prescribed for the specific joint replaced, such as quadriceps setting, leg raising, and passive and active ROM exercises. *These exercises help prevent muscle atrophy and thromboembolism and strengthen the muscles of the affected extremity so that it can support the prosthetic joint*.
- Use sequential compression devices, or antiembolism stockings, as prescribed. *These help prevent thromboembolism and pulmonary embolus for the client who must remain immobile following surgery*.
- For the client with a total hip replacement, prevent hip flexion of greater than 90 degrees or adduction of the affected leg. Provide a seat riser for the toilet or commode. *These measures prevent dislocation of the joint*.
- Assess the client with a total hip replacement for signs of prosthesis dislocation, including pain in the affected hip or shortening and internal rotation of the affected leg.
- For the client with a total knee replacement, use a continuous passive range-of-motion (CPM) device or ROM exercises as prescribed. *Dislocation is not a problem with a knee replacement, and more emphasis is placed on range-of-motion exercises in the early postoperative period*.
- Maintain fluid intake and encourage a high-fiber diet. Administer stool softeners or rectal suppositories as needed. *Immobility contributes to the potential problem of constipation; these measures help maintain regular fecal elimination*.
- Encourage consumption of a well-balanced diet with adequate protein. *Adequate nutrition promotes tissue healing*.
- Teach or reinforce postdischarge exercises and activity restrictions. Emphasize the importance of scheduled follow-up physician visits. *Clients are discharged from the acute care facility before healing is complete. Exercises are prescribed and activities are resumed gradually to protect the integrity of the joint replacement and prevent contractures*.
- For those clients needing additional direct care after discharge, arrange placement in a long-term care or rehabilitation facility. *Activity restrictions may preclude discharge to home for some clients*.
- Make referrals as needed to home health agencies and physical therapy. *Clients often require home health care for both nursing care needs and continued physical therapy following discharge from acute or long-term care*.

PHYSICAL THERAPY AND REHABILITATION Recovery from all types of joint replacement requires postoperative physical therapy, focusing on building strength and regaining joint flexibility. Rehabilitation begins in the hospital, most often the day following surgery, and may be continued during home care. Recovery from a hip replacement is 80% complete in 4 weeks and 100% complete in 6 months. Recovery from a knee replacement is 80% complete in 4 weeks and 100% complete after 1 year. During rehabilitation, the client must follow a regimen of exercise, rest, and medication (Flynn & Johnson, 2005).

COMPLEMENTARY AND ALTERNATIVE THERAPIES The following complementary therapies are examples of those that may be used by people with OA to relieve pain and stiffness. These same therapies are also used by people with rheumatoid arthritis.

- Biomagnetic therapy
- Acupuncture
- Eliminating nightshade foods such as potatoes, tomatoes, peppers, eggplant, tobacco
- Taking nutritional supplements, such as glucosamine, chondroitin, boron, zinc, copper, selenium, manganese, flavonoids, and/or SAM-e
- Herbal therapy
- Massage therapy
- Osteopathic manipulation
- Vitamin therapy
- Yoga.



NURSING CARE

OA is a chronic process for which there is no cure. The focus of nursing care for the client with OA is providing comfort, helping maintain mobility and ADLs, and assisting with adaptations to maintain life roles. A Nursing Care Plan for a client with OA is found on the next page.

Health Promotion

Although OA cannot be prevented, maintaining a normal weight and having a program of regular, moderate exercise will reduce risk factors. Glucosamine and chondroitin are nutritional supplements for OA that are increasingly popular and have been found to be of benefit in reducing manifestations. Clients should discuss these supplements with their healthcare provider before using them.

Assessment

Collect the following data through the health history and physical examination (see Chapter 40 ∞):

- *Health History:* Family history of OA, occupation, recreational activities, joint pain and stiffness, ability to carry out ADLs and self-care activities.
- *Physical Assessment:* Height/weight; gait; joints: symmetry, size, shape, color, appearance, temperature, pain, crepitus, range of motion, Heberden's nodes, Bouchard's nodes.

Nursing Diagnoses and Interventions

The priority nursing interventions for clients with OA are directed toward managing chronic pain, facilitating physical mobility, and improving ability to provide self-care.

Chronic Pain

Pain is a primary manifestation of OA. As joint tissues degenerate and changes in joint structure occur, the amount of discomfort generally increases. The pain associated with OA increases with activity and tends to be relieved with rest. Nonpharmacologic comfort measures are appropriate, with mild analgesics used to supplement these as needed.

- Monitor the level of pain, including intensity, location, quality, and aggravating and relieving factors. *Accurate assessment of pain provides a basis for evaluation of the effect of interventions.*
- Teach clients to take prescribed analgesic or anti-inflammatory medication as needed. *Analgesics reduce the perception of pain and may decrease muscle spasm as well. Anti-inflammatory medication may be ordered to decrease local inflammatory response in affected joints.*
- Encourage rest of painful joints. *The pain of OA is often relieved by joint rest.*
- Suggest applying heat to painful joints using the shower, a tub or sitz bath, warm packs, hot wax baths, heated gloves, or diathermy, which uses high-frequency electrical currents to generate heat. *Heat application reduces accompanying muscle spasm, relieving pain. Moist heat penetrates deeper than dry heat; diathermy delivers heat directly to lesions in deeper body tissues.*
- Emphasize the importance of proper posture and good body mechanics for walking, sitting, lifting, and moving. *Good body mechanics and posture reduce stress on affected joints.*
- Encourage the overweight client to reduce weight. *Excess weight places abnormal stress on joints, particularly the knees.*
- Encourage the use of nonpharmacologic pain relief measures such as progressive relaxation, meditation, visualization, and distraction. *These adjunctive pain relief measures can reduce the client's reliance on analgesics and increase comfort.*

Impaired Physical Mobility

As intra-articular cartilage degenerates and joint structures are altered, the client with OA experiences pain, stiffness, and decreased range of motion in affected joints. When the spine, large weight-bearing joints of the hips and knees, or the ankles and feet are affected, physical mobility can be significantly reduced.

- Assess the range of motion of affected joints. *Assessing joint mobility is important as a basis for planning appropriate interventions.*
- Perform a functional mobility assessment, evaluating gait, ability to sit and rise from sitting position, ability to step into and out of the tub or shower, and negotiation of stairs. *The functional assessment provides vital data about the client's ability to maintain ADLs.*

NURSING CARE PLAN **A Client with Osteoarthritis**

Robert Cerulli is a 72-year-old retired commercial fisherman who has experienced arthritic pain in his hips for the past 10 to 15 years. During the past year, the pain in his right hip has become severe, prompting him to seek medical attention. Significant degenerative changes in both hip joints are noted on x-ray films. The physician recommends a total replacement of the right hip, and total replacement of the left hip to follow in 6 to 12 months. Mr. Cerulli has preoperative teaching and tests the afternoon prior to his surgery, scheduled for 0800 the following morning.

ASSESSMENT

Christie Phlaugh, RN, completes a health history and examination of Mr. Cerulli on admission. Reviewing his medical record, she notes that Mr. Cerulli has mild Parkinson's disease and is taking carbidopa/levodopa (Sinemet 25-100) four times a day to control his symptoms. No other chronic medical conditions have been reported. Mr. Cerulli says he has been essentially healthy his entire life. He has no known allergies to medications, has never smoked, and consumes only small amounts of alcohol.

On examination of Mr. Cerulli, Ms. Phlaugh notes that he is alert and oriented. His vital signs are BP 116/64, P 68 regular, R 18, T 97.4°F (36.3°C) PO. Peripheral pulses are strong and equal in the upper extremities, and slightly weaker but equal in the lower extremities. His feet are cool to touch but have immediate capillary refill. He has full ROM of his shoulders, elbows, and wrists. The ROM of both hips is significantly restricted. Hip flexion beyond 90 degrees prompts pain on both sides. Both flexion and extension of the knees are limited slightly. Mr. Cerulli walks with a limp, favoring his right hip, and has a shuffling gait.

Preoperative laboratory studies including CBC, coagulation studies, chemistry panel, and urinalysis show a serum creatinine of 1.7 mg/dL and BUN of 30 mg/dL, with no other abnormal values noted. His ECG and chest x-ray show no apparent pathologies. Cefazolin (Ancef) 500 mg is to be administered intravenously at 0600 prior to surgery, and Mr. Cerulli is to shower and shampoo with antibacterial soap at bedtime. The physical therapist meets with Mr. Cerulli to evaluate his mobility and begin teaching him about postoperative weight-bearing restrictions.

DIAGNOSES (POSTOPERATIVE)

- *Acute Pain* related to surgical incision
- *Impaired Physical Mobility* related to activity and weight-bearing restrictions
- *Risk for Infection* related to disruption in skin integrity
- *Risk for Ineffective Tissue Perfusion, Right Leg* related to vascular disruption and edema

EXPECTED OUTCOMES

- Maintain an adequate level of comfort postoperatively as demonstrated by:
  - The ability to move easily within restrictions
  - Compliance with instructions to cough and breathe deeply
  - Verbal expressions of comfort.
- Remain free of adverse consequences of immobility such as pneumonia, pressure areas, thromboembolism, or contracture.
- Remain free of infection.
- Maintain adequate perfusion of affected leg.
- Remain free of injury postoperatively.

PLANNING AND IMPLEMENTATION

- Assess pain at least hourly during first 24 to 48 hours postoperatively, and as needed thereafter.
- Instruct in the use of PCA and monitor its effectiveness.
- Help change position at least every 2 hours; encourage the use of the overhead trapeze to shift positions frequently.
- Maintain sequential compression device and antiembolic stocking as ordered; remove for 1 hour daily.
- Encourage the use of the incentive spirometer hourly for first 24 hours, then at least every 2 hours while awake.
- Assist out of bed three times a day after the first 24 hours.
- Maintain abduction of the right hip with pillows.
- Perform passive ROM exercises of unaffected extremities every shift.
- Encourage frequent quadriceps-setting exercises and plantar and dorsiflexion of feet.
- Assess the surgical site frequently; report signs of excess bleeding or inflammation.
- Monitor temperature every 4 hours.
- Assess pulses, color, movement, and sensation of right foot hourly for the first 24 hours, then every 2 hours for 24 hours, then every 4 hours.

EVALUATION

Mr. Cerulli returns to the orthopedic unit from the postanesthesia care unit. He becomes confused and disoriented during the first 36 hours after surgery, but his orientation and thought processes gradually clear. His family has stayed with him, and he has not experienced injury or other adverse consequences from his confusion. Otherwise, Mr. Cerulli has had an uneventful postoperative recovery. Six days after surgery, he is transferred to an extended care rehabilitation facility for further therapy until he is able to ambulate with partial weight bearing on his affected leg. He returns home 5 weeks after surgery, able to use a walker for ambulation. Arrangements are made for an overbed trapeze, elevated toilet seat, and shower chair in his home. A home health nurse and physical therapist visit Mr. and Mrs. Cerulli weekly for a month following his discharge. During this time he gradually resumes full weight bearing. Mr. Cerulli expresses pleasure with the relief of his hip pain and says he has no fear of having his left hip replaced in the future.

CRITICAL THINKING IN THE NURSING PROCESS

1. Mr. Cerulli's preoperative laboratory work showed a modest elevation in his serum creatinine and BUN. What do these studies indicate? How might these changes affect nursing responsibilities related to medication administration for Mr. Cerulli?
2. Mr. Cerulli became confused postoperatively. What factors in his history might have alerted the nurses to this possibility? How might anesthesia and postoperative analgesics have contributed to his confusion?
3. Develop a care plan for Mr. Cerulli using the nursing diagnosis *Acute Confusion*.

See Evaluating Your Response in Appendix C.

- Teach active and passive ROM exercises as well as isometric, progressive resistance, and low-impact aerobic exercises. *Active ROM exercises help maintain muscle tone and mobility of affected joints and prevent contractures. Isometric and progressive resistance exercises improve muscle tone and strength; aerobic exercise improves endurance and cardiovascular fitness.*

PRACTICE ALERT
The older woman with OA may be more willing to take part in weight-bearing exercises if she does so as part of a group or organized activity.

Self-Care Deficit

Just as OA of the lower extremities can reduce the client's mobility, OA of the upper extremities (the wrist, hand, and finger joints in particular) can significantly interfere with performance of ADLs such as cooking and brushing the hair. When the lower extremities are affected, bathing and toileting can be difficult.

- Perform a functional assessment of the upper and lower extremities. For upper extremities, assess the ability to touch the back of the head, and to hold and use small items such as eating utensils. *The functional assessment provides important data about the client's ability to provide self-care.*
- Assess the home setting to determine the need for assistive devices such as handrails, grab bars, walk-in shower stall, or shower chair and handheld showerhead. *Many assistive devices are relatively easy and inexpensive to obtain and can significantly improve the client's independence in performing ADLs.*
- Assist in obtaining other assistive devices such as long-handled shoehorns, zipper grabbers, long-handled tongs or grippers for retrieving items from the floor, jar openers, and special eating utensils. *These devices can prolong independence in performing ADLs.*

Using NANDA, NIC, and NOC

Chart 42–2 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with OA.

Community-Based Care

Because of the chronicity of OA, clients and their families need appropriate teaching to manage the disease and its consequences effectively. Much of the teaching focus is on preservation of joint function and mobility. Discuss the following topics:

- Safeguard against hazards to safe mobility, such as scatter rugs. Encourage installation of safety devices such as handrails and grab bars.
- Understand the disease process and its chronic degenerative nature.
- Learn exercise techniques, including ROM, isometric, postural, stretching, and strengthening to maintain healthy cartilage, preserve range of motion, and develop supportive muscles and tendons. A walking program is beneficial for clients with OA of the knee.

NANDA, NIC, and NOC Linkages
CHART 42–2 The Client with Osteoarthritis

NANDA

Chronic Pain

↓

NIC

Pain Management

Medication Administration

Heat/Cold Application

↓

NOC

Pain: Disruptive Effects

Comfort Level

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

- Do not overuse or stress affected joints with heavy lifting, excessive stair climbing or bending, or other repetitive actions.
- Sit in a straight chair without slumping; avoid soft chairs or recliners and sleep on a firm mattress or use a bed board.
- Use pain relief measures including prescribed or over-the-counter analgesic medications, and nonpharmacologic pain relief measures such as heat, rest, massage, relaxation, and meditation.

For the client who has had a total joint replacement, discuss the following:

- Use and weight bearing of the affected limb
- Appropriate environmental modifications, such as an overhead trapeze for getting out of bed, elevated toilet seats, and types of chairs to use and avoid when sitting
- Prescribed exercises
- Use of assistive devices for ambulation, such as crutches or a walker
- Possible complications, including signs of infection or dislocation, and the need to notify the physician promptly if these occur.

Make referrals to home care, physical or occupational therapy, or other community agencies as indicated, and suggest the following resources:

- National Institute of Arthritis and Musculoskeletal and Skin Diseases
- Arthritis Foundation
- American College of Rheumatology
- Moss Rehab Resource Net.

THE CLIENT WITH MUSCULAR DYSTROPHY

Muscular dystrophy (MD) is a group of inherited muscle diseases that cause progressive muscle degeneration and wasting. The differences in the types of MD relate to the age at onset, the gender affected by the disorder, the muscles involved, and the rate at which the disease progresses. These factors are summarized in Table 42–2. In the majority of cases of MD, there is a positive family history.

The most common form of MD, Duchenne's muscular dystrophy, is inherited as a recessive single gene defect on the X chromosome (a sex-linked recessive disorder), and is transmitted from the mother to male children. This disorder affects males exclusively and occurs in 1 of 3500 live male births. It can be recognized early in pregnancy in about 95% of cases by genetic studies; or in late pregnancy through amniocentesis. Genetic counseling cannot be reliably used to prevent this disease because there is no way to determine if the woman carries the defective gene. The manifestations appear in early childhood, with the average life span being about 15 years after onset (Porth, 2005).

Other types of MD have an onset at any age, and a slow progression with a normal life span.

Pathophysiology

The basic defect in MD is unknown; however, three theories have been proposed. The *vascular* and *neurogenic theories* suggest that the cause is a lack of blood supply to the muscle or a disturbance in the interaction between the nerve and muscle. The *membrane theory* suggests that an alteration in the cell membranes of the muscle causes them to degenerate. Recent genetic studies have shown a deficiency in the amount of dystrophin, a muscle membrane protein, in clients with Duchenne's MD. Dystrophin plays an important role in protecting the muscle against mechanical stresses.

Manifestations

All forms of MD exhibit manifestations of muscle weakness. The specific muscles involved depend on the type of MD. As the disease progresses, the person develops difficulty with ambulation and eventually becomes wheelchair-bound and finally bed-bound. Cardiac abnormalities, endocrine abnormalities, and mental retardation may also occur.

INTERDISCIPLINARY CARE

Because there is no cure or specific treatment for MD, care focuses on preserving and promoting mobility. An interdisciplinary approach, involving many members of the healthcare team, is necessary to meet the physical and psychologic needs of these clients and their families. Diagnosis and classification of the muscular dystrophies are most often based on the manifestations and the pattern of muscle involvement. Biochemical examination, muscle biopsy, and electromyography confirm the diagnosis. Diagnostic tests are described in Chapter 40 ∞.

Tests include measuring creatine kinase (CK-MM, the isoenzyme found in skeletal muscle) which is elevated in the client with suspected MD; performing a muscle biopsy to identify fibrous connective tissue and fatty deposits that displace functional muscle fibers, and conducting an electromyogram (EMG), which will show a decrease in amplitude in MD.

NURSING CARE

Nursing care for a client with MD focuses on promoting independence and mobility and providing psychologic support for both the client and family. A holistic approach is essential in planning and implementing care.

TABLE 42–2 **Types of Muscular Dystrophy**

TYPE	SEX AND AGE AT ONSET	CLINICAL MANIFESTATIONS	PROGRESSION
Duchenne's	Males Ages 3 to 5	Weakness of pelvic and shoulder girdles Waddling gait Toe walking Lordosis Cardiac abnormalities Low IQ in 50% of cases	Rapid; client usually confined to wheelchair by age 15; death occurs by age 20
Myotonic	Males and females Any age	Myotonia of hand muscles Muscular weakness of arms and legs Cardiac abnormalities Endocrine abnormalities Mental retardation (common)	Slow; death usually occurs in early 50s
Becker's	Males Ages 5 to 20	Weakness of pelvic and shoulder girdles	Slow; client usually confined to wheelchair at 25 years after onset; normal life span
Facioscapulohumeral	Males and females Ages 10 to 20	Weakness of face and shoulder girdles	Slow; normal life span
Limb-girdle	Males and females Ages 20 to 40	Weakness of shoulder and pelvic girdles	Extremely variable; usually slow

MEDIALINK Muscular Dystrophy Video

Nursing Diagnoses and Interventions

Self-Care Deficit

The progressive muscle weakness that is associated with MD impairs the client's ability to perform self-care.

- Provide clients and family with supportive care during the progress of the disease. *The goal of treatment is to prolong each functional stage and delay or prevent deformity. When transition from ambulation to a wheelchair occurs, depression and grief may occur.*
- Promote independence. Encourage tasks that can be accomplished rather than letting the client struggle with tasks that may prove frustrating. *All forms of MD result in progressive muscle weakness. Management of the disease is directed toward keeping the client as functional as possible while preventing any deformities.*

Community-Based Care

Teaching the client with MD focuses on maintaining function and independence and preventing deformities. Teach prescribed exercises such as stretching and counterposturing exercises. For the client with braces, discuss skin care and ways to prevent irritation under the brace. Because the client may have weakness involving muscles of respiration, teach the client how to prevent respiratory infections, such as avoiding crowds during flu season and being immunized against pneumococcal pneumonia and influenza. Provide information about support services and organizations such as the Muscular Dystrophy Association.

AUTOIMMUNE AND INFLAMMATORY DISORDERS

Autoimmune and inflammatory disorders of the musculoskeletal system are chronic systemic rheumatic disorders, characterized by diffuse inflammatory lesions and degenerative changes in connective tissues. The disorders have similar clinical features and may affect many of the same structures and organs.

THE CLIENT WITH RHEUMATOID ARTHRITIS

Rheumatoid arthritis (RA) is a chronic systemic autoimmune disease that causes inflammation of connective tissue, primarily in the joints. Its course and severity are variable, and the range of manifestations is broad. Manifestations of RA may be minimal, with mild inflammation of only a few joints and little structural damage, or relentlessly progressive, with multiple inflamed joints and marked deformity. Most clients exhibit a pattern of symmetric involvement of multiple peripheral joints and periods of remission and exacerbation.

FAST FACTS

RA

- RA is found worldwide, affecting 1% to 2% of the total population and all races.
- RA affects 3 times as many women as men.
- The onset of RA occurs most frequently between the ages of 20 and 40 years.

The cause of RA is unknown. A combination of genetic, environmental, hormonal, and reproductive factors is thought to play a role in its development. It is speculated that infectious agents, such as bacteria, mycoplasmas, and viruses (especially Epstein-Barr virus), may play a role in initiating the autoimmune processes present in RA. Several studies have found that heavy smokers are at increased risk for developing RA. It is known that the incidence of RA has decreased during the past 40 years, supporting the theory that environmental factors may change and either promote or protect against RA (Flynn & Johnson, 2005).

The course of RA is variable and fluctuating. Remissions are most likely to occur in the first year of the disease. The rate at which joint deformities develop is not constant. Disease progression is fastest during the first 6 years, slowing thereafter. RA contributes to disability and a tendency to shorten life expectancy. About 10% of people with RA go into long-term remission within 1 year; and another 50% to 60% go into remission within 2 years (Flynn & Johnson, 2005).

RA is less common than OA, with RA affecting 1% to 2% of the population (about 2.1 million people) (Flynn & Johnson, 2005). The incidence of RA increases with age up to about 70 years. Although the onset and manifestations of RA are much the same in older and younger clients, differentiating between RA and OA in the older adult may be difficult at times. It is important to establish an accurate diagnosis, however, because the management of these disorders differs significantly. Clinical features distinguishing RA from OA are listed in Table 42–3.

For older clients, RA is managed much as it is for younger people. However, prolonged bed rest or inactivity is not prescribed for acute episodes, because it may result in irreversible immobility in the older adult. Also, medications are used with greater caution because of the increased risk of toxicity. In many cases, less emphasis is placed on preventing joint deformity and more emphasis on maintaining functional status for the older client with RA.

Pathophysiology

It is believed that long-term exposure to an unidentified antigen causes an aberrant immune response in a genetically susceptible host. As a result, normal antibodies (immunoglobulins) become autoantibodies and attack host tissues. These transformed antibodies, usually present in people with RA, are called *rheumatoid factors (RFs)*. The self-produced antibodies bind with their target antigens in blood and synovial membranes, forming immune complexes (see Chapter 13 ∞ for further information about autoimmune processes).

The damage to cartilage that occurs in RA is the result of at least three processes:

- Neutrophils, T cells, and other synovial fluid cells are activated and degrade the surface layer of the articular cartilage.
- Cytokines, especially interleukin-1 (IL-1) and tumor necrosis factor alpha (TNF-α), cause the chondrocytes to attack the cartilage.

MEDIALINK Case Study: Rheumatoid Arthritis

TABLE 42–3 **Comparison of the Manifestations of Rheumatoid Arthritis and Osteoarthritis**

FEATURE	RHEUMATOID ARTHRITIS	OSTEOARTHRITIS
Onset	Usually insidious, may be abrupt	Insidious
Course	Generally progressive, characterized by remissions and exacerbations	Slowly progressive
Pain and stiffness	Predominant on arising, lasting >1 hour; also occurs after prolonged inactivity	Pain with activity; stiffness following periods of immobility generally relieved within minutes
Affected joints	■ Appear red, hot, swollen; "boggy" and tender to palpation; decreased ROM, weakness ■ Multiple joints affected in symmetric pattern; PIP, MCP, wrists, knees, ankles, and toes often involved	■ Affected joints may appear swollen; cool and bony hard on palpation; decreased ROM ■ One or several joints affected including hips, knees, lumbar and cervical spine, PIP and DIP, wrist, and 1st MTP joint
Systemic manifestations	Fatigue, weakness, anorexia, weight loss, fever; rheumatoid nodules; anemia	Fatigue

■ The synovium digests nearby cartilage, releasing inflammatory molecules containing IL-1 and TNF-α.

Leukocytes are attracted to the synovial membrane from the circulation, where neutrophils and macrophages ingest the immune complexes and release enzymes that degrade synovial tissue and articular cartilage. Activation of B and T lymphocytes results in increased production of rheumatoid factors and enzymes that increase and continue the inflammatory process.

The synovial membrane is damaged by the inflammatory and immune processes. It swells from infiltration of the leukocytes and thickens as cells proliferate and abnormally enlarge. The inflammation spreads and involves synovial blood vessels. Small venules are occluded, and vascular flow to the synovial tissue decreases. As blood flow decreases and metabolic needs increase (from the increased number and size of cells), hypoxia and metabolic acidosis occur. Acidosis stimulates synovial cells to release hydrolytic enzymes into surrounding tissues, starting erosion of the articular cartilage and inflammation of the supporting ligaments and tendons.

The inflammation also causes hemorrhage, coagulation, and deposits of fibrin on the synovial membrane, in the intracellular matrix, and in the synovial fluid. Fibrin develops into granulation tissue (*pannus*) over denuded areas of the synovial membrane. The formation of pannus leads to scar tissue formation that immobilizes the joint (Figure 42–5 ■).

Joint Manifestations

The onset of RA is typically insidious, although it may be acute (precipitated by a stressor such as infection, surgery, or trauma). Joint manifestations are often preceded by systemic manifestations of inflammation, including fatigue, anorexia, weight loss, and nonspecific aching and stiffness. Clients report joint swelling with associated stiffness, warmth, tenderness, and pain. The pattern of joint involvement is typically polyarticular (involving multiple joints) and symmetric. The proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints of the fingers, the wrists, the knees, the ankles, and the toes are most frequently involved, although RA can affect any joint. Stiffness is most pronounced in the morning, lasting more than 1 hour. It may also occur with prolonged rest during the day and may be more severe following strenuous activity. Swollen, inflamed joints feel "boggy" or spongelike on palpation because of synovial edema. Range of motion is limited in affected joints, and weakness may be evident.

The persistent inflammation of RA causes deformities of the joint itself and supporting structures such as ligaments, tendons, and muscles. As the joint is destroyed, ligaments, tendons, and the joint capsule are weakened or destroyed. Joint cartilage and bone are also destroyed. Weakening or destruction of these supporting structures results in lack of opposition to muscle pull, causing deformity.

Characteristic changes in the hands and fingers include ulnar deviation of the fingers and subluxation at the MCP joints. Swan-neck deformity is characterized by hyperextension of the PIP joint with compensatory flexion of the distal interpha-

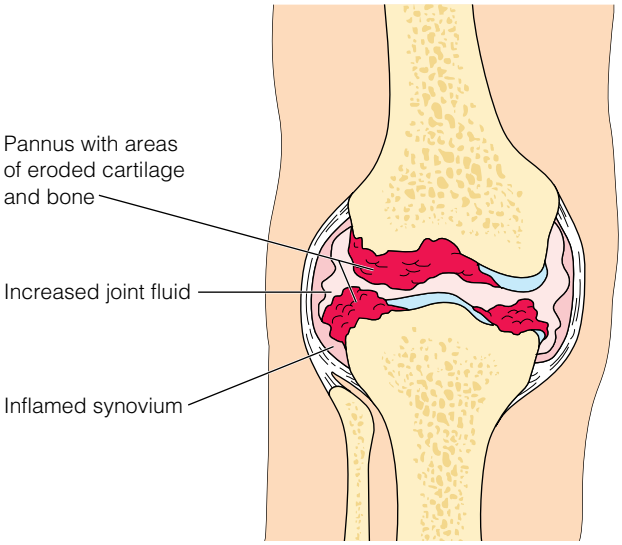


Figure 42–5 ■ Joint inflammation and destruction in rheumatoid arthritis. Note synovial inflammation with pannus formation and the erosion of cartilage and underlying bone.

langeal (DIP) joints. A flexion deformity of the PIP joints with extension of the DIP joint is called a boutonniére deformity (Figure 42–6 ■). The ability to effect a pinch is limited by hyperextension of the interphalangeal joint and flexion of the MCP joint of the thumb.

Wrist involvement is nearly universal, leading to limited movement, deformity, and carpal tunnel syndrome. Inflammation of the elbows often causes flexion contracture.

The knees are frequently affected in RA, with visible swelling often obliterating normal contours. Instability of the knee joint along with quadriceps atrophy, contractures, and valgus (knock-knee) deformities can lead to significant disability. Ambulation may be limited by pain and deformities when the ankles and feet are involved. Typical deformities of the feet and toes include subluxation, hallux valgus (deviation of the great toe toward the other digits of the foot), lateral deviation of the toes, and cock-up toes (turned-up toes).

Spinal involvement is usually limited to the cervical vertebrae. Neck pain is common, and neurologic complications can occur.

Extra-Articular Manifestations

RA is a systemic disease with a variety of extra-articular manifestations. These are seen particularly in clients with high levels of circulating rheumatoid factor. Fatigue, weakness, anorexia, weight loss, and low-grade fever are common when the disease is active. Anemia resistant to iron therapy frequently affects clients with RA. Skeletal muscle atrophy is common, usually most apparent in the musculature around affected joints.

Rheumatoid nodules may develop, usually in subcutaneous tissue in areas subject to pressure: on the forearm, olecranon bursa, over the MCP joints, and on the toes. Rheumatoid nodules are granulomatous lesions that are firm and either movable or fixed. They may also be found in viscera, including the heart, lungs, intestinal tract, and dura.

Other possible extra-articular manifestations of RA include subcutaneous nodules, pleural effusion, vasculitis, pericarditis, and splenomegly (enlargement of the spleen). The *Multisystem Effects of RA* are illustrated on page 1462.

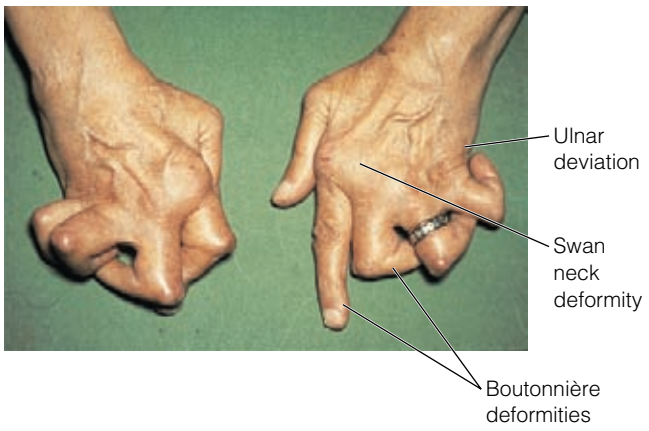


Figure 42–6 ■ Typical hand deformities associated with rheumatoid arthritis.

Source: Biophoto Associates/Photo Researchers, Inc.

Increased Risk of Coronary Heart Disease

People with rheumatoid arthritis have an increased risk of developing coronary heart disease (CHD). In turn, CHD increases the risk of myocardial infarction and death; in fact, RA is associated with a shortened life expectancy (Flynn & Johnson, 2005). RA affects the heart by:

- Direct effects on the blood vessels, with measures of C-reactive proteins (inflammatory markers) being more predictive of future cardiovascular disease than are low-density lipoprotein (LDL) levels.
- Increased risk of having low high-density lipoprotein, high cholesterol and triglyceride levels, high blood pressure, and high levels of homocysteine—all of which increase the risk for CHD.
- The damaging side effects that many medications, such as methotrexate and steroids, often have on coronary vessels.

INTERDISCIPLINARY CARE

The diagnosis of RA is based on the client's history, physical assessment, and diagnostic tests. Diagnostic criteria developed by the American Rheumatism Association are used as well (Box 42–3). At least four of seven criteria must be present to establish the diagnosis.

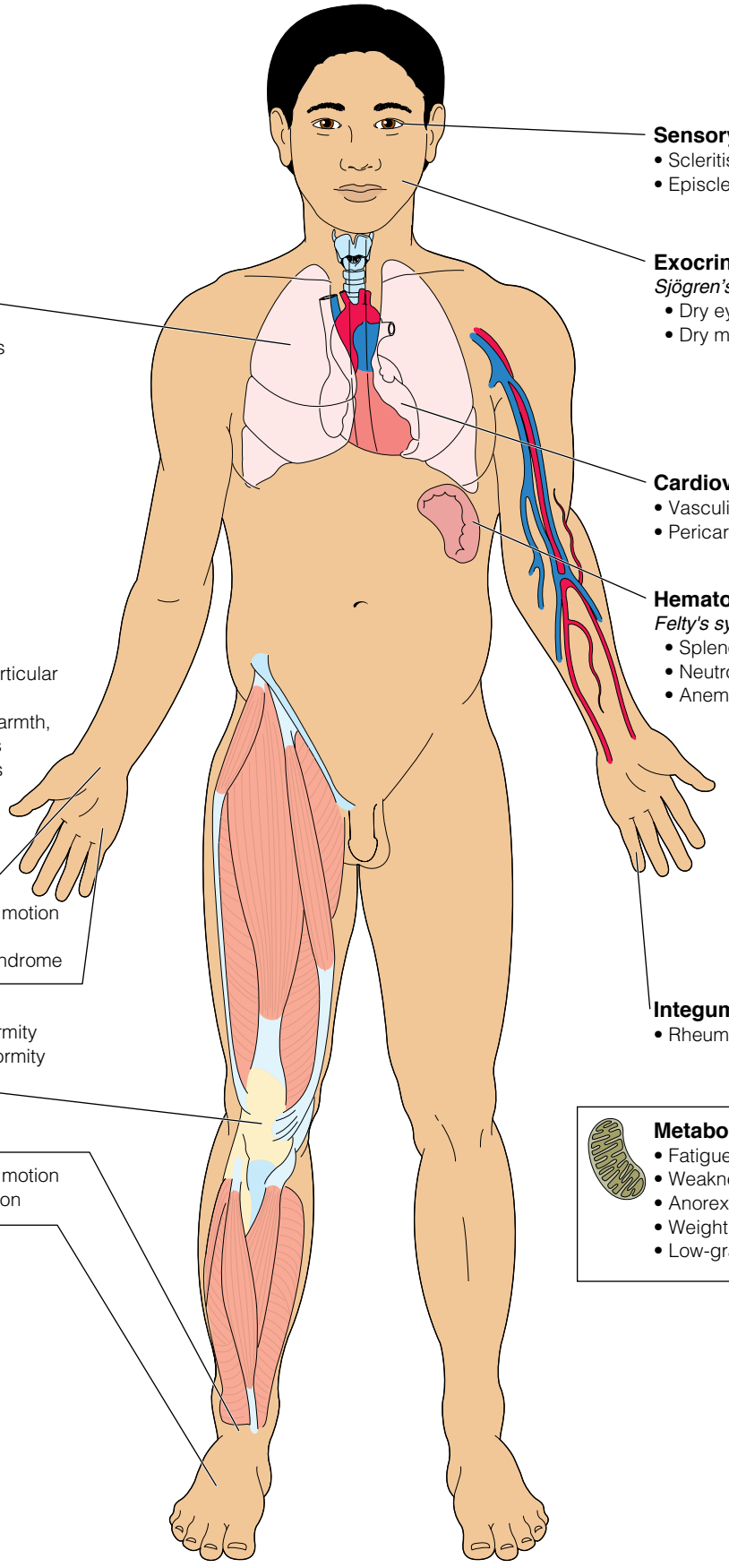
Once the diagnosis of RA has been established, the goals of therapy are to relieve pain, reduce inflammation, slow or stop joint damage, and improve well-being and ability to function. No cure currently exists for RA; the goal of treatment is to relieve its manifestations. An interdisciplinary approach is used, with a balance of rest, exercise, physical therapy, and suppression of the inflammatory processes.

Because a cure is not available and traditional therapies are not always fully effective, the client with RA is vulnerable to quackery. Many nontraditional treatments, including diets, topical preparations, vaccines, hormones, plant extracts, and copper bracelets, have been put forth. These treatments are often costly, and none has been shown to be effective.

BOX 42–3 Diagnostic Criteria for Rheumatoid Arthritis

- Morning stiffness lasting for at least 1 hour and persisting for at least 6 weeks
- Arthritis with swelling or effusion of three or more joints persisting for at least 6 weeks
- Arthritis of wrist, MCP, or PIP joints persisting for at least 6 weeks
- Symmetric arthritis with simultaneous involvement of corresponding joints on both sides of the body
- Rheumatoid nodules
- Positive serum rheumatoid factor
- Characteristic radiologic changes of rheumatoid arthritis noted in hands and wrists

MULTISYSTEM EFFECTS OF **Rheumatoid Arthritis**



Sensory
- Scleritis
- Episcleritis

Exocrine glands
Sjögren's syndrome
- Dry eyes
- Dry mouth

Respiratory
- Pleural disease
- Interstitial fibrosis
- Pneumonitis

Cardiovascular
- Vasculitis
- Pericarditis

Hematologic
Felty's syndrome
- Splenomegaly
- Neutropenia
- Anemia

Musculoskeletal
General
- Symmetric polyarticular joint swelling
- Joint redness, warmth, pain, tenderness
- Morning stiffness

Spine
- Cervical pain
- Neurologic manifestations

Wrists
- Limited range of motion
- Deformity
- Carpal tunnel syndrome

Hands
- Ulnar deviation
- Swan-neck deformity
- Boutonnière deformity

Knees
- Joint effusion
- Instability

Ankles
- Limited range of motion
- Pain on ambulation

Feet
- Subluxation
- Hallux valgus
- Lateral toe deviation
- Cock-up toe

Integumentary
- Rheumatoid nodules

Metabolic Processes
- Fatigue
- Weakness
- Anorexia
- Weight loss
- Low-grade fever

Diagnosis

Diagnostic tests are used to help establish the diagnosis of RA. Testing is also used to rule out other forms of arthritis and connective tissue disorders. Diagnostic tests are described in Chapter 40.

Laboratory tests are used to measure rheumatoid factors and the ESR, which is typically elevated. A complete blood count (CBC) is done to identify anemia. Diagnosing RA in the early stages is often difficult, but a new test is highly effective. In this blood test, clients are tested for antibodies to cyclic citrullinated peptide (CCP) with accurate detection of early RA.

Synovial fluid examination will demonstrate changes associated with inflammation, including increased turbidity (cloudiness), decreased viscosity, and increased protein and WBC levels. X-rays of affected joints are the most specific test for diagnosis of RA. Early in the disease, few changes may be evident other than soft tissue swelling and joint effusions. As the disease progresses, joint space narrowing and erosions are seen.

Medications

Four general approaches are used in the pharmacologic management of clients with RA:

- Aspirin and other NSAIDs and mild analgesics are used to reduce the inflammatory process and manage the manifestation of the disease. Although these drugs may relieve manifestations of RA, they appear to have little effect on disease progression.
- The second approach uses low-dose oral corticosteroids to reduce pain and inflammation. Recent studies suggest that low-dose oral corticosteroids also may slow the development and progression of bone erosions associated with RA.
- A diverse group of drugs classified as disease-modifying or slow-acting antirheumatic drugs are employed in the third approach to treating RA. These drugs, which include gold compounds, D-penicillamine, antimalarial agents, infliximab (Remicade), and sulfasalazine, appear to alter the course of the disease, reducing its destruction of joints. Immunosuppressive and cytotoxic drugs are included in this category as well.
- Intra-articular corticosteroids may be used to provide temporary relief in clients for whom other therapies have failed to control inflammation.

ASPIRIN Aspirin is often the first drug prescribed in the treatment of RA unless its use is contraindicated for the client. Aspirin is an inexpensive and effective anti-inflammatory and analgesic agent. The dose of aspirin required to achieve a therapeutic blood level of 15 to 30 mg/dL and its full anti-inflammatory effect is approximately 4 g per day in divided doses (three or four 5-g [325-mg] tablets qid). This effective dose is just under the toxic dose, which produces tinnitus and hearing loss. The client may be instructed to increase the dose of aspirin gradually until either maximal improvement or toxicity occurs. If tinnitus develops, the client reduces the dose by two to three tablets per day until the tinnitus stops.

Gastrointestinal side effects and interference with platelet function are the greatest hazards of aspirin therapy. Clients are instructed to take aspirin with meals, milk, or antacids to minimize gastrointestinal distress and reduce the risk of GI bleeding. Enteric-coated forms of aspirin and nonacetylated salicylate compounds produce less gastric distress than plain or buffered aspirin and reduce the risk of gastric ulceration, but they are more expensive. Salsalate (Disalcid, Mono-Gesic, Salflex) and choline magnesium trisalicylate (Trilisate, Tricosal) are examples of nonacetylated salicylate products. All salicylate products are contraindicated for clients with a history of aspirin allergy.

OTHER NONSTEROIDAL ANTI-INFLAMMATORY DRUGS A number of other NSAIDs are available for use in the management of RA if aspirin is not tolerated or effective. All NSAIDs act by inhibiting prostaglandin synthesis. Although the efficacy of all NSAIDs, including aspirin, is equivalent, client responses are individual. Several trials of different NSAIDs may be necessary to find the most effective drug.

Some NSAIDs are considerably more expensive than aspirin but may cause less gastrointestinal distress and require fewer doses per day. Gastric irritation, ulceration, and bleeding remain the most common toxic effects of NSAIDs. They can also affect the lower intestinal tract, leading to perforation or aggravation of inflammatory bowel disorders. All NSAIDs can also be toxic to the kidneys.

NSAIDs commonly prescribed for clients with RA are listed in Table 42–4. The FDA (2005) has issued planned regulatory actions for both prescription and over-the-counter (OTC) nonselective NSAIDs. These actions include increased label warnings about the potential serious adverse cardiovascular and gastrointestinal effects of these drugs; the nonprescription drugs include those containing ibuprofen, naproxen, and ketoprofen. Nursing implications for the administration of NSAIDs are described in Chapter 12 ∞.

CORTICOSTEROIDS Systemic corticosteroids can dramatically relieve the symptoms of RA and appear to slow the progression of joint destruction. The long-term use of corticosteroids is associated with multiple side effects, such as poor wound healing, increased risk of infection, osteoporosis, and gastrointestinal bleeding. Severe rebound manifestations can occur when these medications are discontinued. For these reasons, the use of systemic corticosteroids is limited to low dosages daily. The nursing implications for corticosteroid therapy are discussed in Chapter 13 ∞.

DISEASE-MODIFYING DRUGS Disease-modifying drugs are a diverse group of medications including drugs that modify immune and inflammatory responses, gold salts, antimalarial agents, sulfasalazine, and D-penicillamine (Table 42–5). They share characteristics that make them useful in the treatment of RA. Although beneficial effects are not apparent for several weeks or months following the initiation of therapy, they can produce not only clinical improvement but also evidence of decreased disease activity. Because their anti-inflammatory effect is minimal, NSAIDs are continued during therapy. As many as two-thirds of clients taking disease-modifying drugs show improvement, although these drugs have not been shown to slow bone erosion or facilitate healing. All of these drugs are fairly toxic, and close monitoring is necessary during the course of therapy.

Drugs that modify the autoimmune and inflammatory responses in clients with RA include leflunomide (Arava) and

TABLE 42–4 **Examples of Nonsteroidal Anti-Inflammatory Drugs Used to Treat Rheumatoid Arthritis**

DRUG	AVERAGE DOSE	COMMENTS AND PRECAUTIONS
Aspirin	600–900 mg 4 to 6 times daily	Least expensive NSAID; associated with risk of GI ulceration, bleeding, and possible hemorrhage; may cause hepatotoxicity
Diclofenac (Voltaren)	50 mg tid or qid; or 75 mg bid	Expensive; risk of hepatotoxicity
Etodolac (Lodine)	200-400 mg q6h	Expensive; may have less gastrointestinal toxicity
Fenoprofen (Nalfon)	300-600 mg tid or qid	Should not be administered to clients with impaired renal function; risk of GU effects such as dysuria, cystitis, hematuria, acute interstitial nephritis, and nephrotic syndrome
Flurbiprofen (Ansaid)	50-100 mg tid or qid, not to exceed 300 mg/day	Expensive
Ibuprofen (Motrin, Advil, others)	300 mg qid; 400-800 mg tid or qid	Available in prescription and OTC forms; less gastric distress reported than with aspirin or indomethacin; discontinue if visual disturbances develop
Indomethacin (Indocin)	25-50 mg bid or tid	A potent NSAID used for moderate to severe RA and acute episodes of chronic disease; higher incidence of adverse GI effects and CNS effects such as headache, dizziness, and depression
Ketoprofen (Orudis)	50-75 mg tid or qid	Expensive; older adults and clients with renal insufficiency require lower doses
Meclofenamate sodium (Meclomen)	100 mg bid to qid	Increased risk of adverse effects in older adults; GI effects include diarrhea and abdominal pain; anemia may develop during therapy
Nabumetone (Relafen)	1000-2000 mg per day	Most common adverse effects include diarrhea, dyspepsia, and abdominal pain
Naproxen (Aleve, Anaprox, Naprosyn)	250-500 mg bid	Available in prescription and OTC preparations
Oxaprozin (Daypro)	1200 mg daily	Expensive; risk of severe hepatotoxicity; rash may occur
Piroxicam (Feldene)	20 mg daily in a single or divided dose	Expensive; GI side effects including stomatitis, anorexia, and gastric distress may occur more frequently than with other NSAIDs
Sulindac (Clinoril)	150-200 mg bid	May be safer for use than other NSAIDs in clients with chronic renal disease; rare fatal hypersensitivity reaction with fever, liver function abnormalities, and severe skin reaction
Tolmetin (Tolectin)	200-600 mg tid	Expensive; may have higher rate of side effects including GI distress, headache, dizziness, elevated blood pressure, edema, and weight gain

etanercept (Enbrel). Leflunomide reversibly inhibits an enzyme involved in the autoimmune process, and etanercept inhibits the binding of tumor necrosis factor to receptor sites. Infliximab (Remicade) is a biologic response modifier and TNF-α receptor antagonist. Given by intravenous infusion, the drug is administered to reduce infiltration of inflammatory cells and TNF-α production. Adalimumab (Humira) is a biologic response modifier that is given to people with RA to reduce the inflammatory events of polyarthritis and slow the progression of joint damage. Given by subcutaneous injection, the drug cannot be administered if the person has an acute or chronic infection in any part of the body. Prior to initiating the drug, the person should be tested for tuberculosis.

Gold salts may be administered by mouth, but the intramuscular route is preferred because it is more effective. The mode of action of gold is unknown, but it may produce clinical remission in some clients and decrease new bony erosions. Weekly therapy is continued until significant improvement is noted unless toxic reactions occur. Clients experiencing benefit from gold therapy may be continued on monthly injections for several years. About one-third of clients on gold therapy experience toxic reactions, including dermatitis, stomatitis, bone marrow depression, and proteinuria. Mild skin reactions do not always necessitate discontinuation of therapy. CBC and urinalysis are monitored throughout treatment with gold to assess for more severe toxic responses.

Hydroxychloroquine (Plaquenil) is an antimalarial agent sometimes employed in the treatment of RA. Three to 6 months of therapy is required to achieve the desired response, and many clients do not experience significant benefit. Although hydroxychloroquine has a relatively low toxicity, it can cause pigmentary retinitis and vision loss. Clients receiving this drug require a thorough vision examination every 6 months.

Sulfasalazine, a drug regularly prescribed for chronic inflammatory bowel disease, may also be prescribed for RA. See Chapter 26 ∞ for further discussion of this drug and its nursing implications. For clients not responding to the above preparations, penicillamine may be prescribed. Although this agent may be effective in the management of RA, toxic reactions are common and can be severe, including bone marrow suppression, proteinuria, and nephrosis.

TABLE 42–5 **Disease-Modifying Drugs Used to Treat Rheumatoid Arthritis**

CLASS/MEDICATIONS	USUAL DOSE	ADVERSE EFFECTS	COMMENTS/NURSING RESPONSIBILITIES
Gold salts: Gold sodium thiomalate (Myochrysine) Aurothioglucose (Solganal) Auranofin (Ridaura Capsules)	Parenteral: 1st dose 10 mg; 2nd dose 25 mg, then 50 mg weekly IM Oral: 6 mg daily	■ Pruritus, dermatitis ■ Stomatitis, metallic taste ■ Renal toxicity ■ Blood dyscrasias ■ Gastrointestinal distress	■ Frequent UA and CBC ■ Monitor client after injection for flushing, fainting, dizziness, sweating, possible anaphylactic reaction
Antimalarial: Hydroxychloroquine (Plaquenil)	200-600 mg daily with meals	■ CNS reactions including irritability, nightmares, psychoses ■ Retinopathy ■ Alopecia, pruritus ■ Blood dyscrasias ■ GI disturbances	■ Should not be used during pregnancy ■ Regular ophthalmologic examination required
Sulfasalazine (Azulfidine)	2 g/day in divided doses with meals	■ Anorexia, nausea, vomiting, gastric distress ■ Decreased sperm count ■ Headache ■ Rash ■ Blood dyscrasias ■ Hypersensitivity responses including Stevens-Johnson syndrome ■ CNS, liver, and renal toxicity	■ Administer in evenly divided doses ■ Maintain high fluid intake ■ May cause yellow-orange skin or urine discoloration ■ Regular CBCs necessary
Penicillamine (Cuprimine, Depen Titratable)	125-250 mg/day initially, slowly increased to a total of 1000-1500 mg/day	■ Skin rashes ■ Fever ■ Gastrointestinal distress ■ Oral ulcers, loss of taste ■ Fever ■ Bone marrow depression with thrombocytopenia, leukopenia, anemia ■ Renal toxicity ■ May induce immune complex disorders such as Goodpasture's syndrome and myasthenia gravis	■ Regular CBC and UA necessary ■ Administer on an empty stomach ■ Discontinue during pregnancy ■ May require 2 to 3 months of therapy before benefit is seen

IMMUNOSUPPRESSIVE THERAPY Immunosuppressive or cytotoxic drugs are increasingly employed in the management of RA. Indeed, many now consider methotrexate the treatment of choice for clients with aggressive RA. Methotrexate may be used along with NSAIDs in the initial treatment plan. A weekly dose can produce a beneficial effect in as few as 2 to 4 weeks. Gastric irritation and stomatitis are the most frequent side effects associated with methotrexate, but side effects may be better controlled if folic acid is taken at the same time. Alcoholism, diabetes, obesity, advanced age, and renal disease increase the risk of toxic effects (hepatotoxicity, bone marrow suppression, interstitial pneumonitis).

Other immunosuppressive agents such as cyclosporine, azathioprine, and monoclonal antibodies have also been employed in the treatment of clients with severe, progressive, crippling disease who have failed to respond to other measures.

Treatments

The primary objectives in treating RA are to reduce pain and inflammation, preserve function, and prevent deformity.

REST AND EXERCISE A balanced program of rest and exercise is an important component in the management of clients with RA. During an acute exacerbation of the disease, the client may be hospitalized, or a short period of complete bed rest may be prescribed. For most clients, regular rest periods during the day are beneficial to reduce manifestations of the disease. Additionally, splinting of inflamed joints reduces unwanted motion and provides local joint rest. A variety of orthotic devices are available to reduce joint strain and help maintain function.

Rest must be balanced with a program of physical therapy and exercise to maintain muscle strength and joint mobility. ROM exercises are prescribed to maintain joint function and prevent contractures. Isometric exercises are used to improve muscle strength without increasing joint stress. Isotonic exercises also help improve muscle strength and preserve function. Low-impact aerobic exercises, such as swimming and walking, have been shown to benefit clients with RA without adversely affecting joint inflammation or prompting acute episodes.

PHYSICAL AND OCCUPATIONAL THERAPY Physical and occupational therapists can design and monitor individualized activity and rest programs.

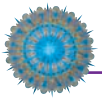
HEAT AND COLD Heat and cold are used for their analgesic and muscle-relaxing effects. Moist heat is generally the most effective, and can be provided by a tub bath. Joint pain is relieved in some clients through the application of cold.

ASSISTIVE DEVICES AND SPLINTS Assistive devices, such as a cane, walker, or raised toilet seat, are most useful for clients with significant hip or knee arthritis. Splints provide joint rest and prevent contractures. Night splints for the hands and/or wrists should maintain the extremity in a position of maximum function. The best "splint" for the hip is lying prone for several hours a day on a firm bed. In general, splints should be applied for the shortest period needed, should be made of lightweight materials, and should be easily removed to perform ROM exercises once or twice a day.

NUTRITION For most clients with RA, an ordinary, well-balanced diet is recommended. Some clients may benefit from substitution of usual dietary fat with omega-3 fatty acids found in certain fish oils.

SURGERY Surgical intervention may be employed for the client with RA at a variety of disease stages. Early in the course of the disease, synovectomy (excision of synovial membrane) can provide temporary relief of inflammation, relieve pain, and slow the destructive process, helping to preserve joint function. Arthrodesis (joint fusion), may be used to stabilize joints such as cervical vertebrae, wrists, and ankles. Arthroplasty, or total joint replacement, may be necessary in cases of gross deformity and joint destruction. Total joint replacement and nursing care of clients undergoing this surgery are discussed in the preceding section on OA.

OTHER THERAPIES Several newer treatments that are not yet in widespread use may be employed in clients with progressive RA. Plasmapheresis has been used to remove circulating antibodies, moderating the autoimmune response. Total lymphoid irradiation decreases total lymphocyte levels, although serious adverse effects are associated with this treatment, and its continued efficacy has not been established.



NURSING CARE

Clients with chronic, progressive, systemic disorders such as RA have multiple nursing care needs involving many functional health patterns. Physical manifestations of the disease often result in acute and chronic pain, fatigue, impaired mobility, and difficulty performing routine tasks. The disease also has many psychosocial effects. The client has an incurable chronic disease that may lead to severe crippling. Pain and fatigue can interfere with the client's ability to perform expected roles, such as home maintenance or job responsibilities. Even though the client's hands may appear swollen or deformed, other people may not understand the systemic nature of the disease or realize the difference between RA and OA. A Nursing Care Plan for a client with RA is found on the next page.

Health Promotion

People with RA have control of their lives by becoming arthritis self-managers. They can help prevent deformities and the effects of arthritis by following prescriptions for exercise, rest, weight management, posture, and positioning. The following suggestions are outlined by the Moss Rehab Resource Net (2005):

- Never attempt an activity that cannot be stopped immediately if it proves to be beyond your power to complete it.
- Respect pain as a warning signal. When you experience pain, change your method of doing things, use equipment or tools if necessary, and take intermittent rest periods.
- Use the strongest joints available for an activity. For example, use the palm of your hand or the crook of your elbow instead of fingers for grasping while carrying.
- Avoid stress toward a position of deformity, such as when the fingers drift toward the little finger. For example, open a jar with your right hand and close a jar with your left hand.
- Avoid activities that need a tight grip, such as writing, wringing, and unscrewing.

Assessment

Collect the following data through the health history and physical examination (see Chapter 40 ∞):

- *Health History:* Pain, stiffness, fatigue, joint problems: location, duration, onset, effect on function, fever, sleep patterns, past illnesses or surgery, ability to carry out ADLs and self-care activities.
- *Physical Assessment:* Height/weight; gait; joints: symmetry, size, shape, color, appearance, temperature, range of motion, pain; skin: nodules, purpura; respiratory: cough, crackles; cardiovascular: pericardial friction rub, apical bradycardia, S_3.

Nursing Diagnoses and Interventions

Many nursing diagnoses may be appropriate for the client with RA. This section focuses on those related to its predominant manifestations and their effect on the client's life.

Chronic Pain

Pain is a constant feature of RA when the disease is active. Pain accompanies both acute inflammation and lower levels of chronic inflammation. Some clients say the pain in joints and surrounding tissue is like a deep, constant toothache. Pain can significantly affect the client's ability to provide self-care and maintain daily activities. It also contributes to the client's fatigue.

- Monitor the level of pain and duration of morning stiffness. *Pain and morning stiffness are indicators of disease activity. Increased pain may necessitate changes in the therapeutic treatment plan.*
- Encourage the client to relate pain to activity level and adjust activities accordingly. Teach the importance of joint and whole-body rest in relieving pain. *Pain is an indicator of excess stress on inflamed joints. Increasing pain indicates a need to decrease activity levels.*
- Teach the use of heat and cold applications to provide pain relief. The client may apply heat by showering or taking tub

NURSING CARE PLAN A Client with Rheumatoid Arthritis

Janice James is a 42-year-old high school science teacher who began noticing vague joint pain, fatigue, poor appetite, and general malaise, which she initially attributed to a case of the flu. However, her symptoms continued, and she reports feeling very stiff in the mornings, often taking until 10:00 or 11:00 A.M. to begin to feel "normal." She then began to notice aching in her hands and wrists, which she attributed to the quilting she loves to do in the evenings. She made an appointment with her family physician when she noticed that her knuckles and finger joints are not just achy but also swollen and hot. Noting that Mrs. James has lost 10 lb since her last visit and has mild anemia and a significantly elevated ESR, the physician refers her to the rheumatology clinic for further evaluation. Following examination, laboratory, and radiologic testing, the rheumatologist establishes a diagnosis of rheumatoid arthritis and initiates a multidisciplinary team conference to plan the management of Mrs. James's rheumatoid arthritis.

ASSESSMENT

Cathy Greenstein, RN, completes an assessment of Mrs. James. She notes that Mrs. James is well groomed and answers questions readily but appears fatigued and ill. Mrs. James relates that her job has been extremely stressful because teacher layoffs have resulted in larger class sizes and fewer teaching assistants. Despite symptoms, she continues to teach full time, but says she feels unable to keep up with all her responsibilities due to her fatigue.

Mrs. James states that she is allergic to penicillin. Her past medical history reveals only the usual childhood diseases and three uncomplicated pregnancies, resulting in the births of her children, ages 14, 11, and 9. Physical assessment findings include BP 124/78, P 82 regular, R 18, T 100.2°F (37.8°C) PO. Hands: swelling of the proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints of both hands; second and third PIP and second MCP joints on right hand are red, shiny, hot, spongy, and tender to palpation; able to extend fingers to 180 degrees but cannot make a complete fist with either hand, with flexion limited to less than 90 degrees; grip strength is weak bilaterally; wrist ROM is limited in all directions. Knees are swollen, and flexion is slightly limited; positive bulge sign in the right knee. Diagnostic findings are an ESR of 52 mm/h, a hematocrit of 30% and positive for rheumatoid factor. Few changes other than soft tissue swelling are evident on hand and wrist x-rays.

DIAGNOSES

- *Chronic Pain* related to joint inflammation
- *Impaired Home Maintenance* related to fatigue
- *Activity Intolerance* related to the effects of inflammation
- *Deficient Knowledge: Therapeutic Regimen*

EXPECTED OUTCOMES

- Verbalize effective pain management strategies:
  - Use assistive devices to minimize joint stress with ADLs:
  - Verbalize a plan to reduce responsibilities for home maintenance.
  - Express a willingness to plan rest breaks during the day.
  - Demonstrate understanding of the prescribed therapeutic regimen and its importance for both short- and long-term benefit.

PLANNING AND IMPLEMENTATION

- Teach techniques for relieving pain and morning stiffness, including:
  - Schedule NSAIDs at equal intervals throughout the day
  - Take morning NSAID dose with milk and crackers approximately 30 minutes before rising
  - Perform ROM exercises in shower or bathtub
  - Apply local heat with paraffin dip or compress, use cold packs as needed.
  - Teach techniques to minimize joint stress while performing ADLs.
- Provide Arthritis Foundation literature and information.
- Discuss ways to delegate household tasks to other family members.
- Explore ways to incorporate 30-minute rest breaks into work schedule.
- Provide information about the disease process and its manifestations, prescribed medications with desired and adverse effects, and the importance of balancing rest and activity.

EVALUATION

The initial treatment regimen of aspirin, rest, exercise, and physical therapy succeeded in partially relieving the acute manifestations of rheumatoid arthritis in Mrs. James. However, complete remission has not been achieved. She has had difficulty scheduling rest periods at work and has had to struggle to delegate household tasks. "I don't look sick to the kids, and they seem to think housecleaning is a terrible imposition on their time. It's often easier to just do it myself than to fight about it. Besides, that way it gets done right." Mrs. James has faithfully followed the prescribed medication regimen and exercise routines, and she has kept her scheduled appointments and maintained contact with the treatment team.

CRITICAL THINKING IN THE NURSING PROCESS

1. Mrs. James is 42 years old. Would your nursing interventions differ if she were 72 years old? If so, how.
2. Rheumatoid arthritis is a chronic illness. What are the physical, emotional, and economic implications of a chronic illness that results in chronic pain and deformity?
3. Develop a nursing care plan for Mrs. James using the nursing diagnosis *Ineffective Role Performance*.

See Evaluating Your Response in Appendix C.

baths, or using warm compresses or other local applications such as paraffin dips. For clients who find that heat increases pain and swelling during periods of acute inflammation, cold packs may be more effective. *Both heat and cold have analgesic effects and can help relieve associated muscle spasms.*

- Teach about the use of prescribed anti-inflammatory medications and the relationship of pain and inflammation. *Anti-inflammatory agents reduce chemical mediators of inflammation and swelling, relieving pain.*
- Encourage using other nonpharmacologic pain relief measures such as visualization, distraction, meditation, and progressive

relaxation techniques. *These techniques can reduce muscle tension and help the client focus away from the pain, decreasing the intensity of the pain experience.*

Fatigue

The pain and chronic inflammatory processes associated with RA lead to fatigue. Other factors contribute as well. Discomfort often disrupts the client's sleep patterns. Anemia, muscle atrophy, and poor nutrition also play a role in the development of fatigue. The client with RA may experience depression or hopelessness, with associated manifestations of fatigue.

- Encourage a balance of periods of activity with periods of rest. *Both joint and whole-body rest are important to reduce the inflammatory response.*
- Stress the importance of planned rest periods during the day. *Rest is vital during acute exacerbations of the disease but also important to maintain the client in remission.*
- Help in prioritizing activities, performing the most important ones early in the day. *Assigning priorities helps the client avoid performing relatively unimportant activities at the expense of more meaningful and important ones.*
- Encourage regular physical activity in addition to prescribed ROM exercises. *Aerobic exercise promotes a sense of well-being and restful sleep patterns.*
- Refer to counseling or support groups. *Counseling and support groups can help the client develop effective coping strategies and deal with depression and hopelessness.*

Ineffective Role Performance

Fatigue, pain, and the crippling effects of RA can interfere with the client's ability to pursue a career and fill other life roles, such as parent, spouse, or homemaker. As the client's role changes, so must the roles of other family members. This can contribute to changes in family processes, increased stress in the family, and further difficulty coping with the effects of the disease.

- Discuss the effects of the disease on the client's career and other life roles. Encourage the client to identify changes brought on by the disease. *Discussion helps the client to accept the changes and begin to identify strategies for coping with them.*
- Encourage the client and family to discuss their feelings about role changes and grieve lost roles or abilities. *Verbalization allows family members to validate and accept feelings about losses and changes, thus helping them to move into new roles.*
- Listen actively to concerns expressed by the client and family members; acknowledge the validity of concerns about the disease, prescribed treatment, and the prognosis. *Demonstrating acceptance of these feelings and concerns promotes trust and validates their reality.*

PRACTICE ALERT

Remember that grief resolution takes time and that clients may respond to loss with anger.

- Help the client and family identify strengths they can use to cope with role changes. *Identifying strengths helps the client and family to consider role changes that maintain self-esteem and dignity.*
- Encourage the client to make decisions and assume personal responsibility for disease management. *Clients who assume a personal and active role in managing their disease maintain a greater sense of self-control and self-esteem.*

Disturbed Body Image

The acute and long-term effects of RA can affect the client's body image, leading to feelings of hopelessness and powerlessness, social withdrawal, and difficulty adapting to changes. When inflammation and joint deformity occur despite compliance, the client may have difficulty accepting the need to continue therapeutic measures, particularly those that have side effects or are costly or time consuming. In addition, unproven alternative treatment strategies and quackery may become increasingly attractive to the client.

- Demonstrate a caring, accepting attitude toward the client. *This attitude helps the client accept the physical changes brought on by the disease.*
- Encourage the client to talk about the effects of the disease, both physical effects and effects on life roles. *Verbalization helps the client identify feelings and gives the nurse opportunity to validate these feelings.*
- Encourage the client to maintain self-care and usual roles to the extent possible. Discuss the use of clothing and adaptive devices that promote independence. *Independence enhances the client's self-esteem.*
- Provide positive feedback for self-care activities and adaptive strategies. *Positive reinforcement encourages the client to continue adaptive measures and maintain independence.*
- Refer to self-help groups, support groups, and other agencies that provide assistive devices and literature. *These groups and agencies can help the client develop adaptive strategies to cope with the effects of RA, enhancing the client's self-concept, body image, and independence.*

Using NANDA, NIC, and NOC

Chart 42–3 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with RA.

Community-Based Care

RA is typically a chronic, progressive disease. As with most diseases of this nature, involvement of the client and family in its management is vital. Education is an important nursing role in caring for clients with RA and their families. (See the Nursing Research box on page 1469). Address the following topics for home care of the client and for family members:

- Disease process and treatments, including rest and exercise
- Medications
- Management of stiffness and pain
- Energy conservation
- Use of assistive devices to maintain independence, including self-care aids such as handheld showers, long-handled brushes and shoehorns, and eating utensils with oversized or special handles
- Clothing options such as elastic waist pants without zippers, Velcro closures, zippers with large pull-tabs, and slip-on shoes
- How to apply splints and take care of skin

NANDA, NIC, AND NOC LINKAGES

CHART 42–3 **The Client with Rheumatoid Arthritis**

NANDA

Powerlessness

↓

NIC

Self-Esteem Enhancement

Support Group

Emotional Support

Environmental Management

Self-Care Assistance

↓

NOC

Health Beliefs: Perceived Control

Health Beliefs: Perceived Resources

Social Support

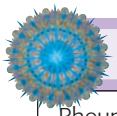
Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

- Home and equipment modifications, such as a raised toilet seat, grab bars in the bathroom, a bath chair, or adapted counter heights for clients in a wheelchair
- Physical therapy, occupational therapy, home health and homemaker services
- Helpful resources:
  - National Institute of Arthritis and Musculoskeletal and Skin Diseases
  - American College of Rheumatology
  - Arthritis Foundation.
  - The Arthritis Society
  - American Physical Therapy Foundation
  - American Chronic Pain Association.

THE CLIENT WITH ANKYLOSING SPONDYLITIS

Ankylosing spondylitis (AS) is a chronic inflammatory arthritis that primarily affects the axial skeleton, leading to pain and progressive stiffening and fusion of the spine. The typical age of onset is between 17 and 35, with at least half a million people with AS in the United States (Spondylitis Association of America, 2006). The incidence is greater in men than women and men have more severe disease. AS is difficult to diagnose in the early stages, but may be a major cause of persistent back pain in young adults.

The cause of ankylosing spondylitis is unknown. As with the other spondyloarthropathies, there is a strong genetic component. Approximately 90% of people with ankylosing spondylitis have the HLA-B27 antigen; about 8% of the general population has this antigen (Porth, 2005).



NURSING RESEARCH **Evidence-Based Practice: Teaching the Client with Rheumatoid Arthritis**

Rheumatoid arthritis (RA) is a disease that can occur at any age, but is seen most often in older adults. RA causes physical, emotional, and economic difficulties, but appropriate management can do much to reduce pain and disability, improve a sense of control, and improve quality of life. With recent advances in computer technology, the Internet has become a convenient means of providing information to people with RA. However, little is known about how many older adults use the computer to gain access to information. This study was conducted to examine the use of computers and the Internet by older adults with arthritis and to describe the characteristics of those who did use the Internet to find health information. Although one of every four older adults who participated in the study owned a computer, only slightly more than half actually used the Internet. Lack of knowledge about using the computer or about accessing the Internet were given as possible reasons.

IMPLICATIONS FOR NURSING

The Internet is a powerful method for providing health information to older adults. Although health history questions rarely contain questions about availability and use of the computer and the Internet, it may be equally as important as asking about other components of one's dwelling. If older adults have but do not use a computer, referral to community resources that provide computer learning classes can facilitate their success in using the computer and doing online searches of the Internet for health information. In addition, prior to recommending an Internet-based health resource, nurses should review the site for content, readability, navigation features, credibility, organization, and graphic appearance.

CRITICAL THINKING IN CLIENT CARE

1. You are designing an Internet site to teach older adults about RA. What topics would you include? How would your presentation be most effective for this age group?
2. You are conducting a computer-literacy course for older adults with RA at a local library. All of them have computers, but none of them have used the Internet to find out about the disease. What sites would you recommend, and why?
3. Develop a plan to include assessment about computers on an agency's health history. What would you include to convince the agency personnel that this is important?

Data from Tak, S. H., & Hong, S. H. (2005). Use of the Internet for health information by older adults with arthritis. *Orthopaedic Nursing, 24*(2), 134-139.

Pathophysiology

Early inflammatory changes often are first noted in the sacroiliac joints. As the cartilage erodes, joint margins ossify and are replaced by scar tissue. The joints of the spine are also affected, with inflammation of the cartilaginous joints, and gradual calcification and ossification that leads to ankylosis, or joint consolidation and immobility. Other organ systems may be affected as well, including the eyes, lungs, heart, and kidneys.

Manifestations

The onset of ankylosing spondylitis is usually gradual and insidious. Clients may have persistent or intermittent bouts of low back pain. The pain is worse at night, followed by morning stiffness that is relieved by activity. Pain may radiate to the buttocks, hips, or down the legs. As the disease progresses, back motion becomes limited, the lumbar curve is lost, and the thoracic curvature is accentuated. In severe cases, the entire spine becomes fused, preventing any motion. Clients with ankylosing spondylitis may also experience peripheral arthritis, primarily affecting the hip, shoulders, and knee joints. Systemic manifestations include anorexia, weight loss, fever, and fatigue. Many clients develop uveitis (inflammation of the iris and the middle, vascular layer of the eye).

For most clients with ankylosing spondylitis, the disease is intermittent with mild to moderate acute episodes. These clients have a good prognosis with little risk of severe disability.

INTERDISCIPLINARY CARE

Diagnostic testing shows an elevated ESR during periods of active disease and typically a positive HLA-B27 antigen. The diagnosis of ankylosing spondylitis is usually confirmed with x-ray examination of the sacroiliac joints and spine. The sacroiliac joint becomes blurred and gradually obliterated. As the disease progresses, vertebrae become squared, and disk spaces narrow.

As with other forms of arthritis, the management of ankylosing spondylitis is multidimensional. Physical therapy and daily exercises are important to maintain posture and joint ROM. NSAIDs relieve pain and stiffness and allow the client to perform necessary exercises. Indomethacin (Indocin) is the NSAID most commonly used to treat ankylosing spondylitis. It may, however, have many adverse effects, including headache, nausea and vomiting, depression, and psychosis. Other drugs that may be prescribed include sulfasalazine (Azulfidine) and topical or intra-articular corticosteroids. Severe hip joint arthritis may necessitate total hip arthroplasty.

NURSING CARE

The primary nursing role in ankylosing spondylitis is to provide supportive care and education. To promote mobility, teach the client to take NSAIDs at regular intervals throughout the day with food, milk, or antacid. Encourage the client to maintain a fluid intake of 2500 mL or more per day. Suggest that the client perform exercises in the shower because warm, moist heat prompts mobility. Stress the importance of following the prescribed physical therapy and exercise program to maintain mobility.

Teach the client that proper positioning and posture are important. When sleeping, a bed board may be used to provide firmness, and the person should sleep in the supine position using either no pillow or only one small pillow. Other important self-care activities include losing weight if applicable, avoiding smoking, and using muscle-strengthening exercises. Suggest occupational counseling if pain and deformity are severe enough to cause work-related problems.

THE CLIENT WITH REACTIVE ARTHRITIS

Reactive arthritis (ReA) (*Reiter's syndrome*) is an acute, nonpurulent inflammatory arthritis that is believed to be a response to an exposure or infection with certain types of bacteria, including *Chlamydia* (a bacterium contracted during sexual activity) or *Salmonella, Shigella, Yersinia, or Campylobacter* (which cause dysentery from contaminated or spoiled food). This type of arthritis most often affects young men who have an inherited HLA-B27 antigen. Reactive arthritis is often found in clients with HIV infection, although the reason for the association is not clear. Reactive arthritis is typically self-limited, although it can be recurrent or progressive. About 15% to 20% of people with ReA develop a chronic arthritis or spondylitis (Spondylitis Association of America, 2006).

Manifestations

Nonbacterial urethritis is often the initial manifestation of Reiter's syndrome. In women, urethritis and cervicitis may be asymptomatic. Conjunctivitis and inflammatory arthritis follow. The arthritis is usually asymmetric, affecting large weight-bearing joints such as the knees and ankles, the sacroiliac joints, or the spine. Mouth ulcers, inflammation of the glans penis, and skin lesions may occur. The heart and aorta may also be affected.

INTERDISCIPLINARY CARE

The diagnosis of reactive arthritis is based on the client's history and presenting symptoms. Manifestations of ReA typically occur 2 to 4 weeks after the infection, and subside in 3 to 12 months. The condition has a tendency to recur. No test is specific for the disorder. Urethral or cervical cultures are obtained to rule out gonococcal infection. When *Chlamydia* is suspected, the client and sexual partner are treated with tetracycline or erythromycin. Reactive arthritis is treated symptomatically, usually with NSAIDs.

NURSING CARE

Clients with reactive arthritis usually are seen in primary care settings such as a clinic or physician's office, making the nursing role primarily one of education. Teach the client about the association of the arthritis with the precipitating infection (if identified). Stress the importance of treating the infection effectively if it is still present. Use this opportunity to provide information about sexually transmitted infections and protective measures to prevent their transmission (see Chapter 52). Discuss the usual self-limited nature of reactive arthritis, the appropriate use of prescribed NSAID preparations, and symptomatic relief measures such as application of heat and rest.

THE CLIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS

Systemic lupus erythematosus (SLE) is a chronic inflammatory connective tissue disease. It affects almost all body systems, including the musculoskeletal system. The manifestations of SLE are widely variable and are thought to result from cell and tissue damage caused by deposition of antigen–antibody complexes in connective tissues. SLE affects multiple body systems, and it can range from a mild, episodic disorder to a rapidly fatal disease process.

FAST FACTS

SLE

- Approximately 1 person in 2000 is affected by SLE (about 500,000 in the United States), with women predominating by a ratio of 9:1 over men.
- SLE usually affects women of childbearing age (when the incidence is 30 times greater than in men) but it can occur at any age.
- SLE is more common in African Americans, Hispanics, and Asians than it is in Caucasians (Porth, 2005).
- The incidence of SLE is higher in some families.

Although the exact etiology of SLE is unknown, genetic, environmental, and hormonal factors play a role in its development. Twin studies and a familial pattern of the disease point to a genetic component, as does an increased incidence of other connective tissue diseases in relatives of people with SLE. Certain human leukocyte antigen (HLA) genes are seen more frequently in people with SLE. Environmental factors such as viruses, bacterial antigens, chemicals, drugs, or ultraviolet light may play a role in activation of the pathologic mechanisms of the disease. In addition, it is felt that sex hormones may influence the development of SLE. Women with SLE have reduced levels of several active androgens that are known to inhibit antibody responses. Estrogens have been shown to enhance antibody responses and have an adverse effect in clients with SLE.

The course of SLE is mild in most clients, with periods of remission and exacerbation. The number and severity of exacerbations tend to decrease with time. In some clients, however, SLE is a virulent disease with significant organ system involvement.

Clients with active disease have an increased risk for infections, which are often opportunistic and severe. Infections such as pneumonia and septicemia are the leading cause of death in clients with SLE, followed by the effects of renal or central nervous system (CNS) involvement. See the Multisystem Effects of SLE on the next page.

Pathophysiology

The pathophysiology of SLE involves the production of a large variety of autoantibodies against normal body components such as nucleic acids, erythrocytes, coagulation proteins, lymphocytes, and platelets. Autoantibody production results from hyperreactivity of B cells (humoral response) because of disordered T-cell function (cellular immune response). The most characteristic autoantibodies in SLE are produced in response to nucleic acids, including DNA, histones, ribonucleoproteins, and other components of the cell nucleus.

SLE autoantibodies react with their corresponding antigen to form immune complexes, which are then deposited in the connective tissue of blood vessels, lymphatic vessels, and other tissues. The deposits trigger an inflammatory response leading to local tissue damage. The kidneys are a frequent site of complex deposition and damage; other tissues affected include the musculoskeletal system, brain, heart, spleen, lung, GI tract, skin, and peritoneum. The autoantibodies produced and their target tissue determine the manifestations of SLE.

A number of drugs can cause a syndrome that mimics lupus in clients with no other risk factors for the disease. Procainamide (e.g., Procan-SR, Pronestyl) and hydralazine (Apresoline, Hydralyn) are the most common drugs implicated, along with isoniazid (INH).

Renal and CNS manifestations of SLE rarely occur with drug-induced lupus, but arthritic and other systemic symptoms are common. Manifestations of drug-induced lupus usually resolve when the medication is discontinued.

Manifestations

Typical early manifestations of SLE mimic those of rheumatoid arthritis, including systemic manifestations of fever, anorexia, malaise, and weight loss, and musculoskeletal manifestations of multiple arthralgias and symmetric polyarthritis. Joint symptoms affect more than 90% of clients with SLE. Although synovitis may be present, the arthritis associated with SLE is rarely deforming.

Most people affected by SLE have skin manifestations at some point during their disease. In fact, SLE was originally described as a skin disorder and named for the characteristic red butterfly rash across the cheeks and bridge of the nose (Figure 42–7 ■).

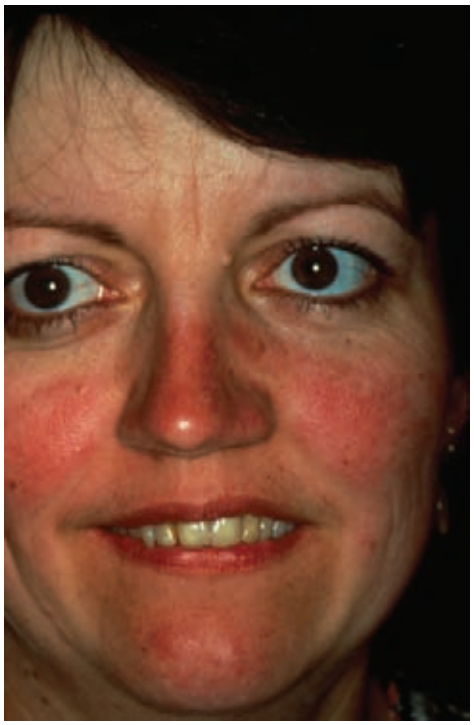
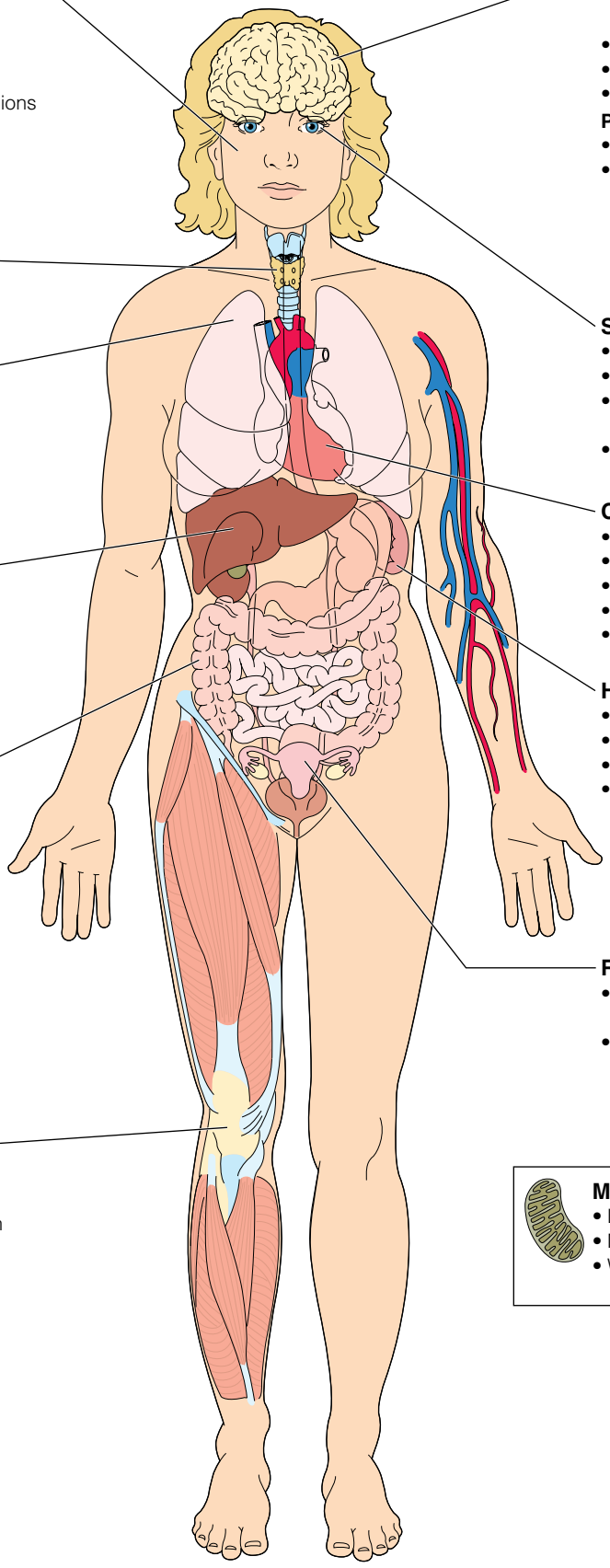


Figure 42–7 ■ The butterfly rash of systemic lupus erythematosus.

MULTISYSTEM EFFECTS **of Systemic Lupus Erythematosus**



Integumentary
- Butterfly rash on face
- Photosensitivity
- Maculopapular rash on exposed body surfaces
- Discoid lesions
- Erythematous fingertip lesions
- Splinter hemorrhages
- Alopecia
- Ulcers (lip, mouth, nose)

Endocrine
- Thyroid abnormalities
- Hyperparathyroidism
- Glucose intolerance

Respiratory
- Pleurisy
- Pleural effusion
- Pneumonitis
- Interstitial fibrosis

Urinary
- Proteinuria
- Cellular casts

Potential Complications
- Nephrotic syndrome
- Renal failure

Gastrointestinal
- Anorexia
- Nausea
- Abdominal pain
- Diarrhea
- Hepatomegaly

Musculoskeletal
- Arthralgias
- Symmetric polyarthritis
- Joint swelling and effusion
- Morning stiffness

Neurologic
- Neuropathies (peripheral and central)
- Seizures
- Depression
- Psychosis

Potential Complications
- Stroke
- Organic brain syndrome
  - Intellectual impairment
  - Memory loss
  - Personality changes
  - Disorientation

Sensory
- Conjunctivitis
- Photophobia
- Retinal vasculitis with transient blindness
- Cotton-wool spots on retina

Cardiovascular
- Pericarditis
- Myocarditis
- Endocarditis
- Vasculitis
- Venous or arterial thrombosis

Hematologic
- Anemia
- Leukopenia
- Thrombocytopenia
- Splenomegaly

Reproductive
- Pregnancy-induced hypertension, edema, and proteinuria
- Fetal loss

Metabolic Processes
- Low-grade fever
- Malaise
- Weight loss

Many clients with SLE are photosensitive; a diffuse maculopapular rash on skin exposed to the sun is common. Other cutaneous manifestations include discoid lesions (raised, scaly, circular lesions with an erythematous rim), hives, erythematous fingertip lesions, and splinter hemorrhages. Alopecia is common in clients with SLE, although the hair usually grows back. Painless mucous membrane ulcerations may occur on the lips or in the mouth or nose. Common manifestations of SLE are listed in the box below.

Approximately 50% of people with SLE experience renal manifestations of the disease, including proteinuria, cellular casts, and nephrotic syndrome. Up to 10% develop renal failure as a result of the disease.

Hematologic abnormalities such as anemia, leukopenia, and thrombocytopenia are common with SLE. Cardiovascular disorders such as pericarditis, vasculitis, and Raynaud's phenomenon often occur. Less frequently, myocarditis, endocarditis, and venous or arterial thrombosis may develop. Pleurisy, pleural effusions, and lupus pneumonitis are common pulmonary manifestations of SLE.

Many clients with SLE develop transient nervous system involvement, often within the first year of the disease. Organic brain syndrome manifestations include decline in intellect, memory loss, and disorientation. Other possible neurologic manifestations include psychosis, seizures, depression, and stroke. Ocular manifestations of SLE include conjunctivitis, photophobia, and transient blindness due to retinal vasculitis.

Gastrointestinal manifestations of SLE, such as anorexia, nausea, abdominal pain, and diarrhea, may affect up to 45% of clients with the disease. The liver may be enlarged, and liver function tests may yield abnormal results.

INTERDISCIPLINARY CARE

Because of the diversity of organ system involvement and manifestations of SLE, diagnosis can be difficult. No one specific test is available to confirm the presence of this disease in all people suspected of having it. Instead, the diagnosis is based on the client's history and physical assessment, as well as laboratory studies.

As with rheumatoid arthritis, effective management of SLE requires teamwork, with active participation by both the client and members of the healthcare team. Although there is no cure for SLE, the 10-year survival rate is greater than 70% among clients with this disease, which was once considered fatal in most cases.

MANIFESTATIONS of SLE

- Painful or swollen joints and muscle pain
- Unexplained fever
- Red rash, especially on the face
- Unusual loss of hair
- Pale, cyanotic fingers or toes
- Sensitivity to the sun
- Edema in legs and around eyes
- Ulcers in the mouth
- Enlarged glands
- Extreme fatigue

Diagnosis

The multiple autoantibodies produced in SLE cause a number of abnormalities in laboratory tests. Diagnostic tests for the musculoskeletal system are described in Chapter 40 ∞.

- *Anti-DNA antibody testing* is a more specific indicator of SLE, because these antibodies are rarely found in any other disorder.
- *ESR* is typically elevated, occasionally to >100 mm/h.
- *Serum complement levels* are usually decreased as complement is consumed or "used up" by the development of antigen-antibody complexes.
- *CBC* abnormalities include moderate to severe anemia, leukopenia and lymphocytopenia, and possible thrombocytopenia.
- *Urinalysis* shows mild proteinuria, hematuria, and blood cell casts during exacerbations of the disease when the kidneys are involved. Renal function tests including *serum creatinine* and *blood urea nitrogen (BUN)* may also be ordered to evaluate the extent of renal disease.
- *Kidney biopsy* may be performed to assess the severity of renal lesions and guide therapy (see Chapter 27 ∞).

Medications

The client with mild or remittent SLE may need little or no therapy other than supportive care. Arthralgias, arthritis, fever, and fatigue can often be managed with aspirin or other NSAIDs. Aspirin is particularly beneficial for clients with SLE because its antiplatelet effects help prevent thrombosis. It may, however, cause liver toxicity and hepatitis.

Skin and arthritic manifestations of SLE may be treated with antimalarial drugs such as hydroxychloroquine (Plaquenil). Hydroxychloroquine has also been shown to be effective in reducing the frequency of acute episodes of SLE in people with mild or inactive disease. Retinal toxicity and possibly irreversible blindness are the primary concerns with this drug. For this reason, the client taking hydroxychloroquine undergoes ophthalmologic exam every 6 months.

Clients with severe and life-threatening manifestations of SLE (such as nephritis, hemolytic anemia, myocarditis, pericarditis, or CNS lupus) require corticosteroid therapy in high doses. Such clients may require 40 to 60 mg of prednisone per day initially. The dosage is tapered as rapidly as the client's disease allows, although lowering the dosage may precipitate an acute episode. Some clients with SLE require long-term corticosteroid therapy to manage symptoms and prevent major organ damage. These clients are at increased risk for corticosteroid side effects, such as cushingoid effects, weight gain, hypertension, infection, accelerated osteoporosis, and hypokalemia.

Immunosuppressive agents such as cyclophosphamide or azathioprine may be used, alone or in combination with corticosteroids, to treat clients with active SLE or lupus nephritis (see the Medication Administration box on the next page). When these agents are used in combination, lower, less toxic doses of each drug can be used. The client receiving immunosuppressive agents is at increased risk for infection, malignancy, bone marrow depression, and toxic effects specific to the drug prescribed.

MEDICATION ADMINISTRATION **Immunosuppressive Agents for SLE**

CYTOTOXIC AGENTS
Azathioprine (Imuran)
Cyclophosphamide (Cytoxan)
Cyclosporine (Sandimmune)

Certain cytotoxic or antineoplastic drugs are effective as immunosuppressive agents. They act by decreasing the proliferation of cells within the immune system and are widely used to prevent rejection following a tissue or organ transplant. They are usually administered concurrently with corticosteroid therapy, allowing lower doses of both preparations, and resulting in fewer side effects.

Nursing Responsibilities

- Monitor blood count, with particular attention to the WBC and platelet counts. Notify the physician if WBCs fall below 4000 or platelets below 75,000.
- Monitor renal and liver function studies including creatinine, BUN, creatinine clearance, and liver enzyme levels. Report any abnormal levels to the physician.
- Oral preparations should be administered with food to minimize gastrointestinal effects. Antacids may be ordered.
- Increase fluids to maintain good hydration and urinary output.
- Monitor intake and output.
- Monitor for signs of abnormal bleeding: bleeding gums, bruising, petechiae, joint pain, hematuria, and black or tarry stools.
- Use meticulous hand washing and other appropriate measures to protect the client from infection. Assess for signs of infection.
- Pulmonary fibrosis is a potential adverse effect of cyclophosphamide. Therefore, monitor the results of pulmonary function studies and be alert to clinical signs of dyspnea or cough.

Health Education for the Client and Family

- Avoid large crowds and situations where you might be exposed to infections.
- Report signs of infection such as chills, fever, sore throat, fatigue, or malaise to the physician.
- Use contraceptive measures to prevent pregnancy while you are taking these drugs because they cause birth defects.
- Avoid the use of aspirin or ibuprofen while taking these drugs. Report any signs of bleeding to the physician.
- You may stop menstruating while you are taking cyclophosphamide. The menses will resume after the drug is discontinued.
- If you are taking cyclophosphamide, be sure to report difficulty breathing or cough to the physician.

Treatments

Because of the photosensitivity associated with SLE, the client should be cautioned to avoid sun exposure. Clients should use sunscreens with a sun protection factor (SPF) rating of 15 or higher when out of doors. Topical corticosteroids may be used to treat skin lesions. Some physicians recommend avoiding the use of oral contraceptives, because estrogen can trigger an acute episode.

Clients with lupus nephritis who progress to develop end-stage renal disease are treated with dialysis (hemodialysis or peritoneal dialysis) and kidney transplantation, discussed in Chapter 29 ∞.

NURSING CARE

Nursing care for the client with mild SLE may be limited to teaching. The client with severe disease, however, has many diverse nursing needs, which vary according to the organ systems involved. Because of the close link between rheumatoid arthritis and SLE, many of the nursing diagnoses and interventions identified for the client with arthritis may be appropriate for the client with lupus. The client with lupus nephritis or end-stage renal disease has the nursing care needs outlined in the sections of Chapter 29 ∞ related to glomerulonephritis and chronic renal failure. This section focuses on the needs of the client related to the dermatologic manifestations of lupus, an increased risk for infection, and health maintenance.

Nursing Diagnoses and Interventions

The priority nursing interventions for the client with SLE are focused on problems with impaired skin integrity, ineffective protection, and impaired health maintenance.

Impaired Skin Integrity

Skin lesions are a common manifestation of SLE. A rash or discoid lesion interrupts the integrity of the skin and the first line of protection against infection, increasing the client's already high risk of infection. These lesions, which usually appear on exposed parts of the skin, can also be disfiguring and cause the client emotional distress.

- Assess knowledge of SLE and its possible effects on the skin. *Assessment allows the nurse to base teaching and information on the client's existing knowledge, improving learning and retention.*
- Discuss the relationship between sun exposure and disease activity, both dermatologic and systemic. *It is important for the client to understand that sun exposure may not only cause dermatologic manifestations but also trigger an acute episode.*
- Suggest the following strategies to limit sun exposure:
  - Avoid being out of doors during hours of greatest sun intensity (10:00 A.M. to 3:00 P.M.).
  - Use sunscreen with an SPF of 15 or higher when sun exposure cannot be avoided. Apply it 30 minutes before going out into the sun.
  - Reapply sunscreen after swimming, exercising, or bathing.
  - Wear loose clothing with long sleeves and wide-brimmed hats when out of doors.

  These strategies can help the client maintain a normal lifestyle while helping to prevent acute episodes.
- Keep skin clean and dry; apply therapeutic creams or ointments to lesions as prescribed. *These measures promote healing and reduce the risk of infection.*

Ineffective Protection

Ineffective protection can be a problem for the client with SLE, who is at increased risk for infection and multiple organ system

problems because of the disease. In addition, treatment with corticosteroids or immunosuppressive agents further impairs immune responses and the ability to fight infection. The following interventions are for the client who is hospitalized.

- Wash hands before and after providing direct care. *Hand washing removes transient organisms from the skin, reducing the risk of transmission to the client.*

PRACTICE ALERT

Hands must be washed before and after providing direct care, even if gloves are worn. A decrease in this type of medical asepsis is contributing to the increasing number of hospital-acquired infections that are resistant to antibiotics.

- Use strict aseptic technique in caring for intravenous lines and indwelling urinary catheters or performing any wound care. *Aseptic technique offers protection against external and resident host microorganisms.*
- Assess frequently for infection. Monitor temperature and vital signs every 4 hours. Assess for signs of cellulitis, including tenderness, redness, swelling, and warmth. Report signs of infection to the physician promptly. *Therapy can suppress usual responses, such as elevated temperature and inflammation. The fever of infection may be mistaken for the fever commonly associated with lupus. The client receiving immunosuppressive therapy for the disease has an even higher risk for infection.*
- Monitor laboratory values, including CBC and tests of organ function; report changes to the physician. *An elevation in the WBC count with a shift to the left (increased numbers of immature leukocytes in the blood) may be an early indication of infection. Changes in liver function studies, renal function studies, myocardial enzymes, or other laboratory values may indicate organ system involvement.*
- Initiate reverse or protective isolation procedures as indicated by the client's immune status. *These procedures provide further protection from infection for the severely immunocompromised client.*
- Ensure an adequate nutrient intake, offering supplementary feedings as indicated or maintaining parenteral nutrition if necessary. *Adequate nutrition is important for healing and immune system function.*
- Teach the client the importance of good handwashing after using the bathroom and before eating. *Hand washing reduces the risk of infection with endogenous organisms.*
- Monitor for potential adverse effects of medications including thrombocytopenia and possible bleeding, fluid retention with edema and possible hypertension, loss of bone density, osteoporosis, and possible pathologic fractures, renal or hepatic toxicity, and cardiac effects, particularly in the client with fluid retention and hypervolemia. *Medications used to treat SLE have many potential adverse effects that can impair normal protective and homeostatic mechanisms.*

Impaired Health Maintenance

As with other chronic diseases, much of the responsibility for maintaining optimal health rests with the client. Disease manifestations such as fatigue, arthralgias, arthritis, and increased risk for infection can interfere with the client's ability to maintain health. Psychosocial issues can also be a significant factor in health maintenance for the client with lupus. These issues may include denial of the significance of the disease, poor coping, lack of financial and other resources, and an inadequate support system.

- Assess the ability to maintain optimal health, identifying physical and psychosocial factors that may affect health maintenance. *Before intervening to improve the client's health maintenance, the nurse must identify and understand factors affecting it.*
- Provide care and teaching in a nonjudgmental manner. *To intervene effectively, the nurse must accept the client and family as they are.*
- Encourage the client and family members to discuss the effect of the disease on their lives. *Open discussion helps the client and the nurse identify barriers to health maintenance and begin exploring alternative strategies.*
- Initiate an interdisciplinary care conference with the client and family. *In this care conference, a number of perspectives can be expressed, improving the planning of strategies for health maintenance activities.*
- Refer the client and family to counseling as needed. *Counseling may help the client and family develop the necessary coping skills to accept and deal with the disease.*
- Refer the client and family to community and social service agencies, and local support groups. *These groups and agencies are valuable resources for the client and family.*

Community-Based Care

Teaching is a critical factor in preparing clients with SLE for self-care at home. Address the following topics:

- The disease and its potential effects. Promote an optimistic outlook, stressing that the majority of clients do not require long-term corticosteroid therapy and that the disease may improve over time.
- The importance of skin care.
- The importance of avoiding exposure to infection.
- The need to follow the prescribed treatment plan, including rest and exercise, medications, and follow-up appointments. Discuss manifestations of an acute episode (often called a *flare*) and stress the importance of contacting the physician promptly if any of these manifestations occur.

PRACTICE ALERT

Warning Signs of a Flare

- Increased fatigue
- Pain, abdominal discomfort
- Rash
- Headache
- Fever
- Dizziness

- The significance of wearing a Medic-Alert tag identifying their condition and therapy such as corticosteroids or immunosuppressives.
- Family planning with the client and spouse. The use of oral contraceptives may be contraindicated for the client; if appropriate,

provide information about alternative means of birth control. Pregnancy is not contraindicated for most women with lupus. However, the pregnant client requires close monitoring because acute episodes sometimes accompany pregnancy.

- The need for preventive health care for both men and women with SLE. Women should have gynecologic and breast examinations and men should have prostate examinations each year. Both men and women should have regular screenings for cholesterol and blood pressure. Annual influenza vaccinations are important, as is pneumococcal vaccinations for older clients. If clients are taking corticosteroids or antimalarial medications, annual eye examinations should be conducted to screen for and treat any eye problems.
- Helpful resources:
  - National Institute of Arthritis and Musculoskeletal and Skin Diseases
  - Lupus Foundation of America.

THE CLIENT WITH POLYMYOSITIS

Polymyositis is a systemic connective tissue disorder characterized by inflammation of connective tissue and muscle fibers leading to muscle weakness and atrophy. When muscle fiber inflammation is accompanied by skin lesions, the disease is known as dermatomyositis. Polymyositis is an autoimmune disorder of unknown cause that affects more women than men. The onset of the disease typically occurs between the ages of 40 and 60 years, although a childhood-onset form is also seen.

The immune mechanism causing the inflammatory response in polymyositis is not clear, but autoantibodies can be identified in the majority of people with the disease. The activation of complement is thought to contribute to the inflammatory process. Inflammation leads to muscle fiber necrosis and degeneration.

Manifestations

Initial manifestations of polymyositis include muscle pain, tenderness, and weakness; rash; arthralgias; fatigue; fever; and weight loss. Skeletal muscle weakness is the predominant manifestation. Its onset may be either insidious or abrupt. Muscle weakness tends to progress over weeks to months. Muscles of the shoulder and pelvic girdles are particularly affected, making it difficult for the client to get out of chairs, climb stairs, and reach overhead. Weakness of neck flexor muscles may make it difficult to raise the head from a pillow. Affected muscles may also be tender and painful. A characteristic dusky red rash may be present on the face and upper trunk. Other manifestations include Raynaud's phenomenon, dysphagia, dyspnea, and cough (due to interstitial pneumonitis). The risk of malignancy is increased, particularly in clients with dermatomyositis.

INTERDISCIPLINARY CARE

There is no specific test to diagnose polymyositis. Autoantibodies may be identified in blood serum. Serum levels of muscle enzymes are elevated, particularly creatine kinase (CK) and aldolase levels. Biopsy of involved muscle shows patchy muscle fiber necrosis and the presence of inflammatory cells.

A combination of rest and corticosteroid therapy is prescribed for the client with polymyositis. Long-term corticosteroid therapy may be necessary to manage the disease. Immunosuppressive agents such as methotrexate, cyclophosphamide, and azathioprine may be used for clients who do not respond well to treatment with corticosteroids.

NURSING CARE

The nursing role in caring for the client with polymyositis is supportive. Measures to promote comfort are important. Muscle weakness may interfere with the client's ability to provide self-care and manage health and home. The client may have difficulty with speech because of pharyngeal muscle weakness. Provide alternate means of communication as needed, and use patience in listening. Observe closely while the client eats, because aspiration is a potential problem. Modify the client's diet as needed to maintain nutrition and safety.

Education of the client and family is an important component of care. Emphasize the need to balance periods of rest and activity. Discuss skin care to prevent dryness and infection. Teach the client about prescribed medications and their short- and long-term side effects. Provide information about safety measures while eating. Encourage family members to become trained in performance of the Heimlich maneuver and CPR. Discuss signs of respiratory infection and other possible complications of polymyositis, including renal failure and malignancy.

THE CLIENT WITH LYME DISEASE

Lyme disease is an inflammatory disorder caused by the spirochete *Borrelia burgdorferi,* which is transmitted primarily by ticks. It is the most commonly reported tick-borne illness in the United States. Geographically, Lyme disease is more prevalent in the areas where the ticks are found: in the coastal Northeast, the upper Midwest, and coastal California regions of the United States (ACR, 2004b). It has also been reported throughout Europe, Asia, and Australia. Ticks that act as vectors for Lyme disease, primarily *Ixodes dammini, I, pacificus,* and *I. scapularis* in the United States, are usually carried by mice or deer, although other animals may be infected. The most frequent time of onset is the summer months.

FAST FACTS

Lyme Disease

- Lyme disease is the most common tick-transmitted disease in the United States.
- Lyme disease occurs most often in children and young adults living in rural areas.
- All stages of Lyme disease can be cured by antibiotics, but some people with late neurologic or arthritic involvement may not improve.

Pathophysiology

Borrelia burgdorferi enters the skin at the site of the tick bite. After an incubation period of up to 30 days, it migrates outward in the skin, forming a characteristic lesion called erythema migrans. It may also spread via lymph or blood to other skin sites, nodes, or organs. The inflammatory joint changes associated

with Lyme disease closely resemble those of rheumatoid arthritis (vascular congestion, tissue infiltration by inflammatory cells, possible pannus formation, and erosion of cartilage and bone).

Manifestations

Manifestations often are seen in the skin, musculoskeletal system, and CNS. Lyme disease begins with flulike manifestations and a skin rash, followed weeks or months later by Bell's palsy or meningitis, and months to years later, by arthritis. This progression is highly individualized.

Erythema migrans is the initial manifestation of Lyme disease. This flat or slightly raised red lesion at the site of the tick bite expands over several days (up to a diameter of 50 cm), with the central area clearing as it expands. Systemic symptoms such as fatigue, malaise, fever, chills, and myalgias often accompany the initial lesion. As the disease spreads, secondary skin lesions develop, as do migratory musculoskeletal symptoms, including arthralgias, myalgias, and tendinitis. Persistent fatigue is common during this stage of the disease. Headache and stiff neck are characteristic neurologic manifestations.

Complications

With untreated infection, complications can develop months to years after the initial infection. Chronic recurrent arthritis, primarily affecting large joints (especially the knee), is common. Permanent disability may result. Other effects that may be seen weeks to months after the initial infection include meningitis, encephalitis, and neuropathies, as well as cardiac complications including myocarditis and heart block.

INTERDISCIPLINARY CARE

Both manifestations and laboratory studies are used to establish the diagnosis of Lyme disease. Culture of the organism from tissues and body fluids is difficult and slow. Antibodies to *B. burgdorferi* can be detected by either enzyme-linked immunosorbent assay (ELISA) or Western blot methods within 2 to 4 weeks of the initial skin lesion.

The early diagnosis and proper antibiotic treatment of Lyme disease are important to preventing the complications of infection. A number of antibiotics may be used to treat Lyme disease, including doxycycline (Doxy-Caps, Vibramycin), tetracycline, amoxicillin (Amoxil), cefuroxime axetil (Ceftin), or erythromycin. Therapy may be continued for up to 1 month to ensure eradication of the organism from affected tissues. The nursing implications for various classes of antibiotics are summarized in Chapter 12 ∞.

In addition to antibiotic treatment, aspirin or another NSAID may be prescribed for relief of arthritic symptoms. The affected joint may be splinted to rest the joint. When the knee is involved, weight bearing may be restricted and the use of crutches indicated.

NURSING CARE

Nursing care focuses on prevention of the disease. Many people do not protect themselves from tick bites. This protection is becoming increasingly important with a higher incidence of Lyme disease, due in part to an overpopulation of deer and the encroachment of the suburbs on once rural areas. Simple measures that can help prevent tick bites are as follows:

- Avoid tick-infested areas, especially in spring and summer, such as woods and rural areas with brush and tall weeds.
- Cover exposed skin with long-sleeved shirts and tuck pants into socks. Wearing high rubber boots may provide additional protection.
- Use insect repellents that contain DEET on clothing and exposed skin and apply permethrin to clothing prior to exposure.
- Inspect skin, especially in areas of tight-fitting clothing, after exposure.
- Remove attached ticks with fine-tipped tweezers. Grasp the tick firmly as close to the skin as possible and pull the tick's body away from the skin. If the tick's head remains in the skin, it will not cause Lyme disease (the bacteria are in the tick's midgut). Most cases occur when the tick has been feeding for at least 24 hours (Tierney et al., 2004). Clean the area with an antiseptic.

INFECTIOUS DISORDERS

Infectious disorders of bone and joints are caused by a pathogen, and are often difficult to treat. Chronic infections may result in pain, deformity, and disability.

THE CLIENT WITH OSTEOMYELITIS

Osteomyelitis is an infection of the bone. Osteomyelitis may occur as an acute, subacute, or chronic process. It occurs as a consequence of bacteremia (hematogenous osteomyelitis), invasion from a contiguous focus of infection, or skin breakdown in the presence of vascular insufficiency (Tierney et al., 2004).

Osteomyelitis can occur at any age, but adults over age 50 are more commonly affected. The older adult is at risk for osteomyelitis for several reasons. Immune function tends to decline with aging; the older adult also is more likely to have a chronic disease process that affects immune function. Circulatory status in older adults often is compromised by atherosclerotic processes, impairing blood flow to the bone. Older adults have a higher risk of pressure ulcers because of circulatory, skin, sensation, and mobility changes associated with aging. Pressure ulcers that cannot be staged and treated because of eschar formation pose a particular risk. In addition, the older adult may not demonstrate the typical signs of infection and inflammation, which allows an infectious process to become well established before it is detected.

Pathophysiology

The cause of osteomyelitis is usually bacterial; however, fungi, parasites, and viruses can also cause bone infection. *Staphylococcus aureus* is the most common infecting organism. Other organisms include *Escherichia coli, Pseudomonas, Klebsiella, Salmonella,* and *Proteus.*

Direct contamination of bone from an open wound, such as an open fracture or a gunshot or puncture wound, is the most common cause of osteomyelitis; osteomyelitis also may occur as a complication of surgery. The third mode of entry for microorganisms that invade bone tissue is the extension from adjacent soft tissue infection. Clients with venous stasis or arterial ulcers of the lower extremities or long-term complications of diabetes mellitus are at risk for this type of bacterial invasion.

After entry, bacteria lodge and multiply in the bone, resulting in the inflammatory and immune system response. Phagocytes attempt to contain the infection, releasing enzymes in the process that destroy bone tissue. Pus forms, followed by edema and vascular congestion. The Haversian canals in the medullary (marrow) cavity of the bone allow the infection to travel to other segments of the bone. If the infection reaches the outer margin of the bone (Figure 42–8 ■), it raises the periosteum of the bone, spreading along the surface. Lifting of the periosteum from the cortex disrupts the blood vessels that enter the bone. Pressure increases, further compromising the vascular supply and leading to ischemia and eventual necrosis of the bone. Blood and antibiotics cannot reach the bone tissue once the pressure compromises the vascular and arteriolar systems. In addition, bacteria adhere to damaged bone, coating the underlying bone with a protective film that further impedes host defenses.

Hematogenous Osteomyelitis

Hematogenous infections are caused by pathogens that are carried in the blood from sites of infection elsewhere in the body. Hematogenous osteomyelitis primarily affects older adults, people with sickle cell anemia, and intravenous drug users. The spine is the usual site of infection in adults. Pathogens enter the well-perfused vertebral bodies of adults via the spinal arteries. From there, the infection spreads into the disk space. The lumbar spine is involved more frequently than the thoracic or cervical spine. Urinary tract infections, soft tissue infection, endocarditis, and infected intravenous sites are sources of pathogens.

Clients with acute hematogenous osteomyelitis experience an acute onset of pain, tenderness, and fever. Soft tissue swelling over the affected bone may be noted. The course of vertebral osteomyelitis in intravenous drug users often is subacute, with vague, dull pain in the affected region and a normal or low-grade fever. The pain intensifies over 2 to 3 months, and is accompanied by tenderness, muscle spasm, and limited range of motion.

Osteomyelitis from a Contiguous Infection

Infections caused by an extension of infection from adjacent soft tissues fall into this category of osteomyelitis. The infection is a result of or complication of direct penetrating wounds, joint replacements, decubitus ulcers, and neurosurgery. This is the most common cause of osteomyelitis in adults.

The diagnosis of osteomyelitis often is not made until the infection has become chronic because the signs of acute infection may be masked by local tissue inflammation. Failure to heal a surgical wound or fracture or a developing sinus tract may be initial indicators of a bone infection.

Osteomyelitis Associated with Vascular Insufficiency

People with diabetes and peripheral vascular disease are at risk for developing osteomyelitis involving the feet. Diabetic neuropathy exposes the foot to trauma and pressure ulcers. The client may be unaware of the infection as it spreads into the bone. When tissue perfusion is poor, normal inflammatory responses and wound healing are impaired. The infection often is diagnosed when the client seeks treatment for a nonhealing sore, swollen toe, or acute cellulitis.

Manifestations

Manifestations of osteomyelitis vary according to the age of the client, the cause and site of involvement, and whether the infection is acute, subacute, or chronic (see the box on page 1479).

INTERDISCIPLINARY CARE

The care of the client with osteomyelitis focuses on relieving pain, eliminating the infection, and preventing or minimizing complications. Early diagnosis is important to prevent bone necrosis by early administration of the appropriate antibiotic.

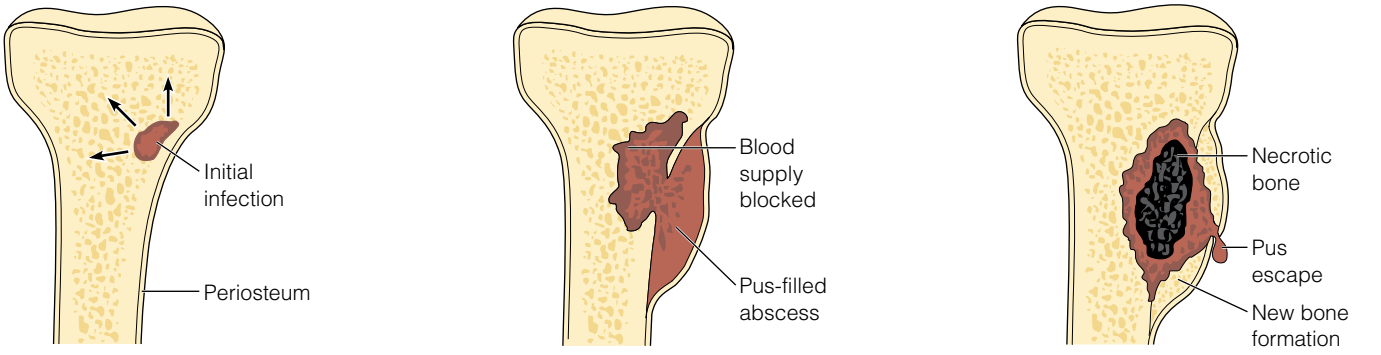


Figure 42–8 ■ Osteomyelitis. *A,* Site of initial infection. Bacteria enter and multiply in the bone, and the inflammatory response is initiated. *B,* Acute phase, in which infection spreads to other parts of the bone. Pus forms, edema occurs, and the vascular supply is compromised. If the infection reaches the outer margin of the bone, the periosteum is lifted, and ischemia and necrosis eventually occur. *C,* Chronic phase. Necrotic bone separates, a new layer of bone forms around the necrotic bone, and a sinus develops to allow the wound to drain.

MANIFESTATIONS of Osteomyelitis

CARDIOVASCULAR EFFECTS
- Tachycardia

GASTROINTESTINAL EFFECTS
- Nausea and vomiting
- Anorexia

MUSCULOSKELETAL EFFECTS
- Limp in involved extremity
- Localized tenderness, especially in epiphyseal area

INTEGUMENTARY EFFECTS
- Drainage and ulceration at involved site
- Swelling, erythema, and warmth at involved site
- Lymph node involvement, especially in the involved extremity

OTHER EFFECTS
- High temperature with chills
- Abrupt onset of pain
- Malaise

Most clients require both debridement of bone and a long period of antibiotic administration.

Diagnosis

The diagnosis of osteomyelitis is based on bone scans, MRI, blood tests, and biopsy. As described in Chapter 40 ∞, an MRI, CT scan, and bone scan may be conducted to identify abscesses, sinus tracts, and bone changes. An ultrasound can detect subperiosteal fluid collections, abscesses, and periosteal thickening and elevation associated with osteomyelitis. During an acute infection, ESR and WBC are elevated. Blood and tissue cultures (from affected bone or soft tissue) are obtained to identify the infecting organism and direct antibiotic therapy.

Medications

Antibiotic therapy is mandatory to prevent acute osteomyelitis from progressing to the chronic phase. Parenteral antibiotic therapy begins as soon as cultures (blood and/or wound) are obtained. A penicillinase-resistant semisynthetic penicillin (e.g., methicillin, oxacillin) may be given until the culture and sensitivity results are known. These antibiotics are used initially because many cases of osteomyelitis are caused by *Staphylococcus aureus*. When the detailed sensitivity report is obtained from the cultures, more definitive antibiotics are prescribed.

For the client with acute or chronic osteomyelitis, antibiotics are continued for 4 to 6 weeks. Intravenous antibiotic administration or oral therapy is common. Oral therapy with twice-daily ciprofloxacin has been shown to be as effective as parenteral therapy for treating adult clients with chronic osteomyelitis caused by susceptible organisms (Tierney et al., 2004).

Surgery

Surgical debridement is the primary treatment for the client with chronic osteomyelitis. The periosteum is excised and the cortex is drilled to release the pressure from accumulated pus. During this procedure, cultures may be obtained and sent to the laboratory for analysis. The wound holes are irrigated, and the wound is then closed. The cavity may be kept clean by inserting drainage tubes that are connected to an irrigation and suction system. Postoperatively, the nurse is responsible for instilling and removing dilute antibiotic solutions through the drainage tubes. See the box below for related nursing care.

A musculocutaneous (myocutaneous) flap is another approach used for the treatment of the dead space caused by extensive debridement of the infected site. The procedure involves moving or rotating a muscle and the section of skin fed by the arteries from that muscle into the cavity created by the surgery. A skin graft is performed later.

NURSING CARE OF THE CLIENT UNDERGOING Surgical Debridement for Osteomyelitis

PREOPERATIVE CARE
- Discuss the impending surgery, the client's concerns regarding surgery and its risks, and what steps will be taken if surgery is ineffective. *Open discussion and active listening are important means of gaining the client's trust and encouraging the client to express concerns about the outcome of the surgery. Surgery is frequently performed when 36 to 48 hours of antimicrobial therapy yields no improvement and when prolonged bacteremia and evidence of an abscess formation are present. The periosteum is excised, allowing access to the purulent material in the infected area. If pus is not apparent, several holes may be drilled into the bone. In some cases, irrigation tubes are inserted and connected to an elaborate system for postoperative antimicrobial therapy.*
- Clients may need extensive antimicrobial treatment postoperatively if an irrigation system is surgically implanted. Before the procedure, explain to the client that bed rest and an extended period of treatment in the hospital are imperative. *Clients who understand the events that may occur postoperatively may be more accepting of the required restrictions.*

POSTOPERATIVE CARE
- Provide meticulous care of the dressing and/or irrigation setup. *Frequently, the irrigation tubes are connected to a three-way stopcock, which allows irrigation and drainage of the debrided area without separating the tube from the collection device. Nurses need to be extremely coutious and adhere to strict sterile technique.*
- Assess the client for manifestations of further infection. *Although the client will receive antimicrobial agents, it is important to monitor the client continually for sudden spikes in temperature, pain at the involved site, and other indications of superinfection.*

HEALTH EDUCATION FOR THE CLIENT AND FAMILY
- While receiving antimicrobial agents, be sure to drink adequate amounts of fluid and eat a high-calorie diet to minimize the risks for damage to the kidneys, yeast infection, and adverse gastrointestinal effects.

NURSING CARE

The client with chronic osteomyelitis faces frequent and lengthy hospitalizations and/or treatment modalities. The prognosis is uncertain, and functional deficits and amputation are a constant concern. The ongoing expenses, loss of financial support, and role changes within the family are also client concerns.

Nursing Diagnoses and Interventions

Nursing diagnoses associated with acute osteomyelitis focus on preventing the transmission of infection and problems due to immobility. Providing comfort and client teaching are also very important.

Risk for Infection

Compromised immune status places the client with osteomyelitis at risk for superinfection. An inadequate kilocalorie intake is an additional factor that contributes to the risk.

- Maintain strict hand washing practices. *Meticulous hand washing helps prevent the spread of infection by minimizing the entry of organisms into susceptible clients.*

PRACTICE ALERT
Careful hand washing before and after direct care is essential even if gloves are worn.

- Administer antimicrobial therapy at specified time intervals. *Optimal blood levels of antibiotic therapy are mandatory in clients with infectious processes.*
- Maintain the client's optimal dietary kilocalorie and protein intake. *High kilocalorie and protein intake provide the client with sufficient nutritional support for the body's needs during the stressful event of the inflammatory process.*

Hyperthermia

The infection and associated inflammatory process can cause fever in the client with osteomyelitis.

- Monitor temperature every 4 hours and when client reports chills and/or fever. Blood cultures are frequently ordered when an acute elevation of temperature occurs. *A sudden rise in temperature in clients with either acute or chronic osteomyelitis may indicate inadequate antimicrobial management.*
- Maintain a cool environment and provide light clothing and bedding during temperature elevation. *Proper environmental conditions and clothing enhance the evaporative process during acute temperature elevation and promote comfort.*
- Ensure a daily fluid intake of 2000 to 3000 mL. Dehydration may result from evaporative fluid losses during acute temperature elevations. Furthermore, clients taking large doses of antibiotic therapy may experience fluid loss through excessive diarrhea as a side effect of the therapy. *Fluid replacement is necessary during this time to prevent further dehydration.*

Impaired Physical Mobility

Pain, infection, inflammation, and the use of immobilizers can all impair the mobility of the client with osteomyelitis.

- Maintain the affected limb in functional position when immobilized. *The client may hesitate to move the involved extremity because of continuous pain; therefore, the extremity must be maintained in functional position to avoid flexion contracture.*
- Maintain rest, and avoid subjecting the affected extremity to weight-bearing activities. *The involved extremity must be immobilized to avoid pathologic fractures caused by stress on the weakened bone.*
- Ensure active or passive ROM exercises every 4 hours. *Flexion contracture occurs when the client remains immobile or when there is only minimal joint movement. Consult a physical therapist for plan of exercises to avoid contracture.*

Acute Pain

The client with osteomyelitis experiences pain due to swelling.

- Use a splint or immobilizer when the client experiences acute pain from swelling. *Splinting or immobilizing the involved extremity provides support and reduces pain caused by movement.*
- Ask the physician to order scheduled administration of narcotic and nonnarcotic analgesics on a 24-hour basis rather than as needed. *The use of 24-hour administration allows blood levels of pain-relieving medications to remain constant.*

PRACTICE ALERT
Clients are often reluctant to ask for a prn pain medication, allowing the pain to reach a level that is difficult to manage.

- Use nonpharmacologic strategies (e.g., heat distraction, relaxation techniques) for pain management. *Pain of the muscles and joints may be controlled through nonpharmacologic interventions. Warm moist packs, warm baths, or heating pads to the involved extremity provide comfort due to vasodilation.*
- Avoid excessive manipulation of the involved area; handle the area gently. Carefully assess the client for guarding, limping, or unwillingness to move the affected part. Communicate to other healthcare professionals the client's preferences for assistive devices and means of manipulating the involved area. *Gentle handling and minimal manipulation help reduce pain.*

Community-Based Care

Although clients may be hospitalized for acute treatment and surgery, most care is provided at home. Home health services can provide intravenous medications, if prescribed. Discuss the following topics for home care:

- The importance of careful hand washing, especially after toileting and dressing changes.
- The importance of taking all antibiotics as prescribed. Include information about helping prevent the yeast infections (of the mouth or vagina) often associated with prolonged antibiotic therapy by eating 8 oz of live-culture yogurt each day.
- The need to take pain medications on a regular basis to prevent pain from becoming severe. Provide information about how to deal with side effects, such as constipation, by increasing fluid and fiber intake.
- How to perform wound care and sources for needed equipment and supplies.
- Rest or limited weight bearing for the affected extremity or body part. Teach how to avoid complications associated with

prolonged immobilization, such as frequently shifting position, keeping skin and linens clean and dry, and doing active ROM exercises for unaffected joints.

- The importance of maintaining good nutrition. An adequate supply of kilocalories, protein, and other nutrients is necessary for immune function and healing. Suggest frequent small meals and using nutritional supplements such as Ensure to help maintain nutritional intake.

THE CLIENT WITH SEPTIC ARTHRITIS

Septic arthritis can develop if a joint space is invaded by a pathogen. The primary risk factors for septic arthritis are persistent bacteremia (bacteria in the blood) (e.g., due to use of injectable drugs, endocarditis) and previous joint damage (e.g., due to trauma or rheumatoid arthritis). Arthroscopic surgery and total joint replacements that allow potential direct contamination of the joint are additional risk factors (Tierney et al., 2004).

Pathophysiology

The most common bacteria implicated in septic arthritis include *gonococci, S. aureus,* and streptococci. Infections by gram-negative bacteria such as *E. coli* and *Pseudomonas* are seen with increasing frequency, particularly in people who inject recreational drugs or are immunocompromised (Tierney et al., 2004).

Infection of the joint leads to inflammation with resulting synovitis and joint effusion. Abscesses may form in synovial tissues or bone underlying joint cartilage. If not treated promptly and effectively, septic arthritis can lead to destruction of the affected joint. A single joint, often the knee, is usually affected. Septic arthritis may also affect other joints such as the shoulder, wrist, hip, fingers, or elbow.

Manifestations

The onset of septic arthritis is typically abrupt, marked by pain and stiffness of the infected joint. The joint appears red and swollen, and is hot and tender to the touch. Effusion (increased fluid within the joint space) is usually present. Systemic manifestations of infection, such as chills and fever, often accompany local manifestations, although these may be muted if the client is taking anti-inflammatory medications.

INTERDISCIPLINARY CARE

Septic arthritis is a medical emergency requiring prompt treatment to preserve joint function. When it is suspected, fluid from the affected joint is aspirated and sent for Gram stain and culture. Cultures also are obtained from all likely sources of the infection, including blood, sputum, or wounds. The synovial fluid culture is always positive in nongonococcal septic arthritis but often is negative for bacteria in early gonococcal arthritis. Infected synovial fluid usually is cloudy, with a high WBC count and a low glucose level. Joint x-ray films are often normal in the initial stages, but soon show demineralization, bony erosions, and joint space narrowing.

The infected joint is treated with rest, immobilization, elevation, and systemic antibiotics. Treatment with a broad-spectrum parenteral antibiotic is initiated before the results of culture are obtained. The medication may be changed or adjusted once the organism has been identified. Antibiotic therapy is continued for at least 2 weeks after inflammatory manifestations have abated. Frequent joint aspirations may be performed to remove excess fluid and pus, and to evaluate for the continued presence of bacteria. Surgical drainage may be performed if the hip joint is involved (because of the difficulty of aspirating this joint) or when medical therapy does not rapidly eliminate bacteria from the synovial fluid. Physical therapy is implemented during the recovery period to ensure maintenance of optimal joint function.

NURSING CARE

Septic arthritis can be frightening to the client who experiences a sudden onset of joint pain and swelling and is faced with the possibility of rapid functional loss of movement. Nursing care is both supportive and educative. Clients may be hospitalized for initial treatment with intravenous antibiotics. It is important to monitor the client's response to therapy, including systemic manifestations such as fever. Position the affected joint appropriately, using pillows to elevate it as needed. Splints or traction may be used to immobilize the joint. Warm compresses may be ordered for comfort. Active ROM exercises preserve joint mobility and should be initiated as soon as the physician allows.

The client with septic arthritis needs information about the disorder, its etiology, and its treatment. Teach the client how organisms may gain entry into the joint space. Discuss the role that the use of injected drugs and sexually transmitted infections play in septic arthritis, and means to prevent infection as appropriate (e.g., using clean "works," practicing safer sex). Refer the client to a drug treatment program if necessary. Emphasize the importance of complying with all aspects of the treatment plan to prevent joint destruction and disability.

NEOPLASTIC DISORDERS

Bone tumors may be either primary (arising in the bone itself) or metastatic (seeded from a tumor elsewhere in the body). Like other tumors, bone tumors can be either benign or malignant.

THE CLIENT WITH BONE TUMORS

Benign bone tumors tend to grow slowly and do not often destroy surrounding tissues. Primary malignant tumors of the bone are rare, accounting for only about 1% of all adult cancers (Porth, 2005). Malignant tumors grow rapidly and metastasize. Virtually every malignant tumor can metastasize to bone. However, the most common metastatic bone tumors originate from primary tumors of the prostate, breast, kidney, thyroid, and lung.

Primary bone tumors arise from bone tissue itself, that is, cartilage (chondrogenic), bone (osteogenic), collagen (collagenic),

and bone marrow cells (myelogenic). The tissue type, neoplasm classification, sites, and incidence of the most common primary bone tumors are summarized in Table 42–6. The focus for discussion in this section is care of the client with a primary bone tumor.

Pathophysiology

The etiology of bone tumors is unknown, but there is a connection between increased bone activity and the development of primary bone tumors. Bone tumors frequently occur when primary bone growth is at its peak in adolescence or is overstimulated during disease, such as Paget's disease.

Primary tumors cause bone breakdown, called *osteolysis,* which weakens the bone, resulting in bone fractures. Normal bone adjacent to the tumor responds to tumor pressure by altering its normal pattern of remodeling. The bone's surface becomes altered, and the contours enlarge in the area of the tumor growth.

Malignant bone tumors invade and destroy adjacent bone tissue by producing substances that promote bone resorption or by interfering with a bone's blood supply. Benign bone tumors, unlike malignant ones, have a symmetric, controlled growth pattern. As they grow, they push against neighboring bone tissue. This weakens the bone's structure until it becomes unable to withstand the stress of ordinary use and frequently causes pathologic fracture.

Manifestations

The three main manifestations of bone tumors are pain, a mass, and impaired function. Bone pain usually comes on slowly and lasts for as long as a week, is constant or intermittent, and may be worse at night. The mass is described as a swelling or lump on the bones that is firm, slightly tender, and may be felt through the skin. The mass may interfere with normal movement and/or cause the bone to break. The manifestations of bone tumors are usually associated with a history of a fall or blow to the extremity that brings the mass to the client's attention. The injury, rather than the growth itself, usually causes the client to seek medical attention. Manifestations of bone tumors are listed in the box on the next page.

INTERDISCIPLINARY CARE

Treatment and care of the client with bone tumors focuses on prompt diagnosis, removal of the tumor, prevention of complications, and client education.

Diagnosis

The diagnosis of bone tumors is critical to the survival of the client and possible preservation of the affected limb. Diagnostic tests are described in Chapter 40.

Radiologic studies include x-rays, CT scans, and MRI. X-rays show the location of the tumors and the extent of bone involvement. Benign tumors are characterized by sharp margins that are clearly separate from the surrounding normal bone. Metastatic bone destruction has a characteristic "moth-eaten" pattern in which the growth has a less-defined margin that cannot be separated from the normal bone. CT scan and MRI are useful in evaluating the extent of tumor invasion into bone, soft tissues, and neurovascular structures. Percutaneous needle biopsy or needle biopsy at the time of surgery is used to determine the exact type of bone tumor.

TABLE 42–6 Description of Common Primary Bone Tumors

TISSUE TYPE	BENIGN	MALIGNANT	SITE	INCIDENCE
Chondrogenic (cartilage-forming tumors)	**Osteochondroma**—most common benign tumor		Pelvis, scapula, ribs	Higher in males
	Chondroma		Hands, feet, ribs, spine, sternum, or long bones	Ages 30 to 50 Higher in males
		Chondrosarcoma	Femur, pelvis, ribs, head (epiphysis) of long bones	13% of malignant bone tumors Middle age and older Higher in males
Osteogenic (bone-forming tumors)	**Osteoid Osteoma**		Shaft (diaphysis) of long bones (i.e., femur, tibia)	Ages 20 to 30 Higher in males
		Osteosarcoma—most common malignant tumor	Long bones, knee	38% of malignant bone tumors Predominant in adolescents and people ages 50 to 60
Collagenic (collagen-forming tumors)		**Fibrosarcoma**	Femur, tibia	4% of malignant bone tumors Wide age distribution, but usually occurs in people ages 40 to 50 Higher in females
Myelogenic (tumors of bone marrow cells)	**Giant cell tumor**		Shaft (diaphysis) of long bones (i.e., femur, tibia, radius, humerus)	4% to 5% of bone tumors Wide age distribution Higher in females

MANIFESTATIONS of Neoplasms of the Musculoskeletal System

BONY SARCOMAS

Site	Manifestations
Upper or lower extremity or pelvis	■ Worsening deep bony pain ■ Pain at night or during rest that may radiate and become severe ■ Muscular weakness or atrophy
Metaphysis of distal femur, proximal tibia, proximal humerus, and pelvis	■ Soft tissue mass extending from bone with erythematous or warm skin over tissue mass ■ Change in ability to perform ADLs ■ Fever

SOFT TISSUE SARCOMAS

Site	Manifestations
Upper or lower extremity and pelvis	■ Enlarging firm mass with irregular borders, which causes pain in surrounding soft tissue structures
Thigh; shoulder, and pelvis	■ Erythema or warmth and venous dilation over skin ■ Muscular weakness and atrophy with limited range of motion change in ability to perform ADLs and change in gait ■ Paresthesia with neurologic involvement and distal swelling ■ Palpable local lymph nodes
Pelvis	■ Altered bowel and bladder habits or pain with intercourse

Laboratory tests include an alkaline phosphatase (elevated with malignant bone tumor) and a calcium level (increased with massive bone destruction).

Treatments

As with other malignant tumors, bone tumors are treated with chemotherapy, radiation therapy, and surgery.

CHEMOTHERAPY Chemotherapeutic agents are administered to shrink the malignant tumor before surgery, to control recurrence of tumor growth after surgery, or to treat metastasis of the tumor. Chemotherapeutic agents used to treat bone tumors are listed in Box 42-4. See Chapter 14 for further discussion of chemotherapy and its nursing implications.

RADIATION THERAPY Radiation therapy may be used in combination with chemotherapy. Radiation therapy is frequently applied to metastatic bone carcinomas as a method of pain control. It is also used to eliminate bony tumors or to eliminate any remaining tumor after a surgical procedure. Radiation therapy is discussed in Chapter 14.

SURGERY The goal of surgery for the treatment of primary bone tumors is to eliminate the tumor completely. Tumors are removed either by excising the tumor itself or by amputating the affected limb. The type of procedure varies from removing the tumor only, to removing the tumor along with a small margin of normal tissue surrounding the tumor, to removing the tumor and a wide zone of normal tissue, to removing the tumor and part or all of the bone in which it lies. Cadaver allografts or metal prostheses often are used to replace missing bone, avoiding amputation. Care of the client undergoing amputation is discussed in Chapter 41.

BOX 42–4 Chemotherapeutic Agents Used for Musculoskeletal Neoplasms

Alkylating Agents
Ifosfamide
Cyclophosphamide

Antibiotics
Doxorubicin
Bleomycin

Antimetabolites
Methotrexate

Plant Alkaloids
Vincristine

Synthetic Agents
Cisplatin

NURSING CARE

Nursing care for the client with bone tumors requires innovative interventions from the time of diagnosis through the rehabilitation phase. In the acute phase, problems associated with pain, lack of knowledge, immobility, coping, and anxiety are foremost. If the client develops complications from treatment or if a malignancy metastasizes, problems related to home health maintenance management, self-concept, and prevention of further complications take priority.

Nursing Diagnoses and Interventions

The client with a bone tumor requires nursing care to meet many health problems, including prevention of injury, relief of pain, assistance with mobility, and teaching about the disease process and treatment.

Risk for Injury

In the client with a bone tumor, changes in bone tissue can cause pathologic fractures.

- Teach how to avoid falls or injury to the tumor site, such as by using assistive devices when walking and ensuring the home environment is not conducive to falling (for example, remove throw rugs and use night-lights). *Pathologic fractures may occur at the tumor site because bone destruction can weaken the area.*

- Provide referral to physical or occupational therapy for fitting of and teaching about assistive devices for ambulating, such as a cane, crutches, or a walker. *Assistive devices can reduce the risk of falling when the client has significant weakness of an extremity or when balance has been affected by treatment of the disease.*

Acute Pain, Chronic Pain

In the client with a bone tumor, pain may be related to direct invasion of the tumor or to pathologic fractures. Clients may experience both acute and chronic pain.

- Develop strategies for controlling both acute pain (from surgery, fracture, or inflammation) and chronic pain (from progression of the disease). *Analgesics combined with nonpharmacologic methods of pain control provide optimum relief of pain. Chronic pain, when mild in nature, is best managed with NSAIDs or aspirin. Moderate pain is best managed with a combination of codeine and NSAIDs. Severe pain is best relieved with long-acting or sustained-relief narcotic analgesics.*
- Provide assistive devices (e.g., canes, walkers, crutches) when the client ambulates. *Assistive devices lessen the pain by supporting weight bearing during ambulation.*

Impaired Physical Mobility

Pain, muscle wasting, or surgical procedures can impair the physical mobility of the client with a bone tumor.

- Begin muscle strengthening and active and passive ROM exercises immediately after surgery. A continuous passive motion (CPM) machine may be used after surgical procedures to either upper or lower extremities. *Muscle strengthening exercises must be encouraged as soon as possible to prevent muscle wasting and shorten the rehabilitation period.*
- Encourage exercises that help strengthen the triceps muscles. *The triceps are the major muscles in the arms and must be strengthened to assist in use of crutches or other assistive devices.*
- For the client who has undergone an amputation of a lower extremity, encourage quadriceps and gluteal setting exercises and leg raises. *These exercises will benefit the client when the rehabilitation period begins.*

Decisional Conflict

A lack of knowledge about the diagnosis and treatment regimen can impair the client's ability to make informed decisions about the treatment plan.

- Discuss issues related to diagnosis, radiologic evaluation, biopsy, surgery, chemotherapy, radiation therapy, potential complications, alternative therapies, risks, benefits, nursing management, discharge plans, home care, and long-term treatment and follow-up. *The client requires this information in order to make informed decisions about treatment.*

Community-Based Care

The client with a primary bone tumor needs information about the disease, its potential consequences, and treatment options. Present information in a matter-of-fact manner, taking time to listen to and address the client's and family's concerns. Discuss expected effects and potential side effects of surgery, chemotherapy, and radiation therapy. Provide information about how to minimize side effects. Teach the postsurgical client about wound care, demonstrating dressing changes and stump care (if amputation has occurred). Provide the client with a list of local resources for obtaining supplies. Discuss activity and weight-bearing restrictions. Refer the client to physical therapy for teaching about ambulation and appropriate muscle-group strengthening exercises. Ensure that the client who has experienced an amputation is working with or has a referral to a prosthetic specialist. For the client with metastatic disease, discuss hospice services and support groups for clients with cancer.

CONNECTIVE TISSUE DISORDERS

Connective tissue is the most abundant and widely distributed body tissue. It not only connects body parts but also provides support; forms bones, cartilage, and the walls of blood vessels; and attaches muscles to bones. Connective tissue consists of three elements: (1) long fibers embedded in a (2) noncellular ground substance, and (3) cells specific to the class of connective tissue. Fibers made up primarily of collagen, a protein, are the most abundant in connective tissue.

Connective tissue disorders, also known as collagen diseases, are a group of immune-mediated disorders. Although they appear to have a genetic component, their cause is unknown. Because connective tissue and collagen are widely distributed in many varied tissues, these are systemic diseases with diverse manifestations.

THE CLIENT WITH SYSTEMIC SCLEROSIS (SCLERODERMA)

Systemic sclerosis, also known as **scleroderma** ("hardening of the skin"), is a chronic disease characterized by the formation of excess fibrous connective tissue and diffuse fibrosis of the skin and internal organs. The cause of scleroderma is unknown, but genetic, immune, and environmental factors are thought to play a role. Although this uncommon disease is distributed worldwide, a higher incidence is noted in coal and gold miners and in people exposed to certain chemicals such as polyvinyl chloride, epoxy resins, and aromatic hydrocarbons.

FAST FACTS

Scleroderma

- Scleroderma affects approximately 300,000 people in the United States, with about one-third having the systemic form and the rest the localized form.
- Scleroderma affects women more often than men by a ratio of approximately 3:1.
- Although it can occur at any age from infants to older adults, the onset of scleroderma typically occurs between the ages of 25 and 55 years (Scleroderma Foundation, 2006).

Pathophysiology

Abnormalities in cellular immune function are believed to contribute to the development of scleroderma. Abnormal proliferation of fibrous connective tissue occurs in affected tissues, including the skin, blood vessels, lungs, kidneys, and other organs.

Scleroderma may be either localized, affecting the skin only, or generalized (systemic sclerosis), with both skin and visceral organ involvement. Localized involvement may occur as irregularly shaped patches of skin (morphea) or a line of disease on the arm, leg, or side of the face (linear scleroderma) (International Scleroderma Network, 2004). Eighty percent of people with generalized disease have limited involvement, frequently manifested by CREST syndrome, a combination of calcinosis (abnormal calcium salt deposition in the tissues), Raynaud's phenomenon, esophageal dysfunction, sclerodactyly (localized scleroderma of the fingers), and telangiectasia (dilated, superficial blood vessels). The remainder of clients with generalized systemic sclerosis have a diffuse form of the disease and a higher risk of visceral organ involvement. Infections and diseases of the cardiovascular, renal, pulmonary, and CNS are the most common causes of death in people with systemic sclerosis.

Manifestations

The initial manifestations of systemic sclerosis are usually noted in the skin, which thickens markedly. Diffuse, nonpitting swelling also is noted. As the disease progresses, the skin begins to atrophy, becoming taut, shiny, and hyperpigmented (Figure 42–9 ■). Facial skin tightening leads to loss of skin lines and a pursed-lip appearance. Skin tightness may limit mobility, particularly of the face and hands. Other skin manifestations include telangiectasias (flat, red areas caused by dilation of small blood vessels, usually noted on the face, hands, and in the mouth) and calcium deposits, usually noted around joints.

Arthralgias and Raynaud's phenomenon are common early manifestations of systemic sclerosis. Raynaud's phenomenon (intermittent attacks of small artery vasospasm) is characterized by pallor of the fingers followed by cyanosis, and then reactive hyperemia with redness. Attacks are usually triggered by cold temperatures.

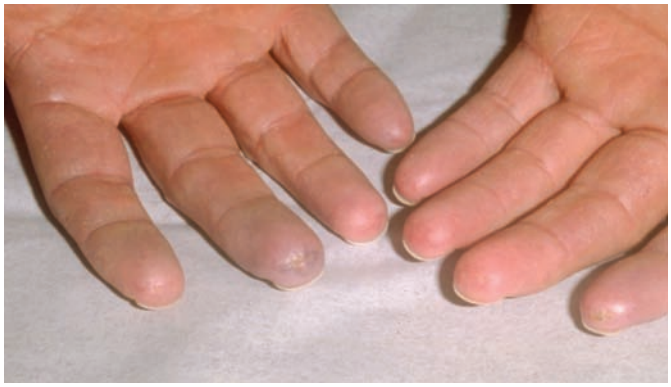


Figure 42–9 ■ Characteristic skin changes of scleroderma.

Source: Logical Images/Custom Medical Stock Photo.

The client with visceral organ involvement may have varied symptoms. Dysphagia is common, because the motility of the esophagus is affected. Pulmonary involvement can lead to exertional dyspnea due to impaired gas exchange and right-sided heart failure due to pulmonary hypertension. Involvement of the heart may cause manifestations of pericarditis and dysrhythmias. Diarrhea or constipation, abdominal cramping, and malabsorption can occur when the GI tract is affected. Renal effects can lead to proteinuria, hematuria, hypertension, and renal failure.

The prognosis for localized and limited scleroderma is good; many clients have a normal life span. The course of diffuse systemic sclerosis is highly variable. This disease is usually progressive; complete remission is rare.

INTERDISCIPLINARY CARE

The manifestations of systemic sclerosis often allow diagnosis with little or no testing. No cure is currently available; treatment is symptomatic and supportive.

Diagnosis

No single diagnostic test is specific for systemic sclerosis, although a titer of 1:40 or higher for antinuclear antibody (ANA) is the most sensitive for diagnosis. Other laboratory studies that are done include an ESR, which is typically elevated from the chronic inflammatory process and a CBC, which will demonstrate anemia. A skin biopsy may be done to confirm the diagnosis.

Medications

Medications to treat systemic sclerosis are chosen based on the client's symptoms. Immunosuppressive agents and corticosteroids are of limited benefit, but may be used to slow or prevent pulmonary fibrosis and in life-threatening disease. Penicillamine may be used to treat scleroderma and pulmonary fibrosis. Calcium channel blockers such as nifedipine (Procardia) or alpha-adrenergic blockers such as prazosin (Minipress) may be prescribed for clients with Raynaud's phenomenon. When manifestations of esophagitis accompany systemic sclerosis, H_2-receptor blockers such as cimetidine (Tagamet) or ranitidine (Zantac), antacids, or omeprazole (Prilosec), which blocks all gastric secretion, may be ordered. Tetracycline or another broad-spectrum antibiotic may be prescribed to suppress intestinal flora and relieve symptoms of malabsorption. Clients with kidney disease are usually treated with angiotensin-converting enzyme (ACE) inhibitors such as captopril (Capoten) to control hypertension and preserve renal function. End-stage kidney disease is managed with dialysis and transplantation.

Physical Therapy

Physical therapy is an important part of the management of systemic sclerosis to maintain mobility of affected tissues, the hands and face in particular. Because the mouth opening, if involved, becomes increasingly smaller as the disease progresses, stretching and strengthening of facial muscles can be vital to maintaining oral food intake.

NURSING CARE

Nursing care needs of clients with systemic sclerosis are individualized to the effects and manifestations of the disease, with interventions summarized in the following discussion.

Nursing Interventions

Skin manifestations are present to some degree in nearly all clients with scleroderma. Nursing care related to the skin focuses on maintaining skin integrity and flexibility. Measures to maintain supple skin are important, because elasticity cannot be regained once it is lost. Apply moisturizers to prevent dryness and cracking. Protect the skin where it is stretched taut over joints or bony prominences. Perform ROM exercises to help prevent joint contractures due to increasingly tight skin.

Difficulty swallowing and recurrent esophagitis may interfere with the client's nutritional status. Provide small, frequent meals. Consult with the dietitian and the client to determine which foods are easy to swallow. Keep the client in a sitting or Fowler's position after meals and elevate the head of the bed at night to minimize esophageal reflux.

The dermatologic and systemic effects of the disease may have significant psychologic effects on the client, leading to feelings of helplessness and hopelessness, and self-esteem disturbance. Establish an atmosphere of trust with the client. Listen actively and acknowledge concerns about the disease and its effects on the client's life and appearance. Encourage the client to share these concerns with family members and significant others. Provide referral to social services or counseling as appropriate.

The client with predominant pulmonary disease has nursing care needs similar to those of other clients with restrictive respiratory disorders . If the client with systemic sclerosis has impaired renal function, nursing care is similar to that for clients with chronic renal failure (see Chapter 29 ∞).

Community-Based Care

Teach the client with systemic sclerosis about the disease and introduce measures to help manage its effects. Stress the importance of good skin care and physical therapy exercises to maintain mobility, particularly of the hands and face. Discuss the need to avoid chilling (local and whole body) to prevent episodes of Raynaud's phenomenon. Teach the role of proper dress in the winter: loose, warm clothing, gloves, and warm stockings. If needed, stress the need to stop smoking because of the vasoconstrictive effect of nicotine and the respiratory effects of the disease. Provide the client with information about manifestations of disease progression and organ involvement. Teach the client to report new or worsening symptoms to the physician. In addition, suggest the following resources:

- National Institute of Arthritis and Musculoskeletal and Skin Diseases
- Scleroderma Foundation
- Scleroderma Research Foundation
- International Scleroderma Network.

THE CLIENT WITH SJÖGREN'S SYNDROME

Sjögren's syndrome is an autoimmune disorder that causes inflammation and dysfunction of exocrine glands throughout the body. Sjögren's syndrome primarily affects women, with a ratio of women to men at 9:1 The highest incidence is between the ages of 40 and 60 years. Although it can occur as a primary disorder, Sjogren's syndrome is often associated with other rheumatic disease, including rheumatoid arthritis, SLE, primary biliary cirrhosis, scleroderma, Hashimoto's thyroiditis, and interstitial pulmonary fibrosis (Tierney et al., 2004).

Pathophysiology

In this disease, exocrine glands in many areas of the body are destroyed by infiltration of lymphocytes and deposits of immune complexes. The salivary and lacrimal glands are particularly affected, leading to the characteristic manifestations of *xerophthalmia* (dry eyes) and *xerostomia* (dry mouth). Clients often experience dry, gritty-feeling eyes and may develop corneal ulcerations. Mucosal dryness affects taste, smell, chewing, and swallowing and leads to increased dental caries. Parotid gland enlargement is common. Excess dryness can also affect the nose, throat, larynx, bronchi, vagina, and skin. Systemic effects of Sjögren's syndrome include arthritis, dysphagia, pancreatitis, pleuritis, neurologic manifestations including migraine, and vasculitis. Nephritis may occur, but renal failure rarely results. Clients with Sjögren's syndrome have a greatly increased risk of developing malignant lymphoma.

INTERDISCIPLINARY CARE

The diagnosis of Sjögren's syndrome is often based on the client's history and clinical presentation. Schirmer's test, which measures the quantity of tears secreted in a 5-minute period in response to irritation, ocular staining, and slit-lamp examination of the eye, may be performed. A definitive diagnosis can be made by biopsy of either the lacrimal or salivary gland.

Treatment is supportive. Artificial tears are used to decrease eye irritation and dryness. The client can keep the mouth moist by drinking fluids, using a saliva substitute, and chewing sugarless gum. Medications that increase mouth dryness, such as atropine and decongestants, should be avoided.

NURSING CARE

Nurses caring for clients with Sjögren's syndrome need to teach measures to protect the client's eyes and oral mucosa. Instill artificial tears as needed. Encourage the client to sip fluids throughout the day. Provide frequent oral hygiene, particularly before and after meals. Ensure that the client has sufficient fluids to drink during meals, because fluids help with chewing and swallowing.

THE CLIENT WITH FIBROMYALGIA

Fibromyalgia is a common rheumatic syndrome characterized by musculoskeletal pain, stiffness, and tenderness. Fibromyalgia affects more than 6 million Americans, 90% of them women be-

tween 20 and 50 years of age (National Fibromyalgia Research Association, 2004). The cause is unknown, but possible etiologies include sleep disorders, depression, infections, and an altered perception of normal stimuli. Fibromyalgia can be a complication of hypothyroidism, rheumatoid arthritis, or (in men) sleep apnea. It closely resembles chronic fatigue syndrome, except that musculoskeletal pain is predominant in fibromyalgia, whereas fatigue is a more significant feature of chronic fatigue syndrome.

Pathophysiology

No inflammatory, structural, or physiologic muscle changes have been demonstrated in fibromyalgia. A connection between fibromyalgia and the central nervous system is being studied.

Manifestations

A gradual onset of chronic, achy muscle pain is typical, although the onset may be sudden, occasionally following a viral illness. The pain may be localized or involve the entire body. The neck, spine, shoulders, and hips are often affected. Pain is produced by palpating localized "tender points" (Figure 42–10 ■). Local tightness or muscle spasm may also occur. Systemic manifestations of fibromyalgia include fatigue, sleep disruptions, headaches, morning stiffness, painful menstrual periods, and problems with thinking and memory (called the "fibro fog"). Pain and fatigue are aggravated by exertion.

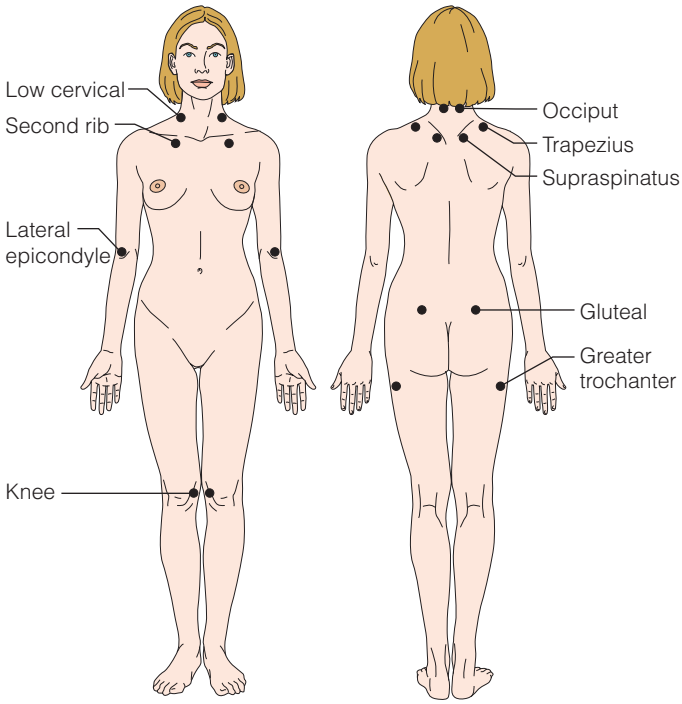


Figure 42–10 ■ Location of "tender points" in fibromyalgia.

INTERDISCIPLINARY CARE

The diagnosis of fibromyalgia is based on the history and physical assessment. The criteria developed by the American College of Rheumatology that are used for diagnosis are a history of widespread pain that has been present for at least 3 months and pain at 11 of the 18 tender points on palpation. There are no laboratory or diagnostic tests for the disorder, although tests may be performed to rule out other rheumatic disorders, such as rheumatoid arthritis or SLE. Fibromyalgia also may occur as a complication of hypothyroidism, so thyroid function studies are performed.

This disorder may resolve spontaneously or become chronic and recurrent. The client with fibromyalgia needs reassurance of the benign nature of the disorder along with validation of its reality. Therapeutic measures include local heat applications, massage, stretching exercises, and sleep improvement. Amitriptyline, a tricyclic antidepressant, has been shown to promote better sleep and relieve manifestations of fibromyalgia. NSAIDs have not been effective in its treatment.

The National Institute of Arthritis and Musculoskeletal and Skin Diseases (2005a) is conducting research to better understand why people with fibromyalgia have increased sensitivity to pain, the role of stress hormones in the body, the effect of genetics, and what medications or behavioral treatments are most effective.

NURSING CARE

Nursing care for clients with fibromyalgia is supportive and educational, provided in community settings such as clinics and other primary care settings. It is important to validate clients' concerns and reassure them that their symptoms are not "all in the head." This syndrome is recognizable and manageable; its course is not progressive. Teach clients about the disorder, and reassure them that it resolves uneventfully in most instances. Provide verbal and written instructions about the use of heat, exercise, stress-reduction techniques, and prescribed medications to relieve manifestations. In addition, suggest the following resources:

- Fibromyalgia Network
- National Fibromyalgia Research Association
- American College of Rheumatology
- National Institute of Arthritis and Musculoskeletal and Skin Diseases.

STRUCTURAL DISORDERS

Structural disorders of the musculoskeletal system most commonly affect the spine. The disorders discussed in this section are spinal deformities and low back pain.

THE CLIENT WITH SPINAL DEFORMITIES

Scoliosis and kyphosis are the two most common deformities of the spinal column. *Scoliosis* is a lateral curvature of the spine. *Kyphosis* is excessive angulation of the normal posterior curve of the thoracic spine (Figure 42–11 ■).

An estimated 500,000 adults in the United States are affected by scoliosis. It usually is diagnosed in adolescence, with girls affected more than boys by an 8:1 margin. Idiopathic scoliosis is the most common form of the disorder, accounting for approximately 75% of cases. Congenital and neuromuscular

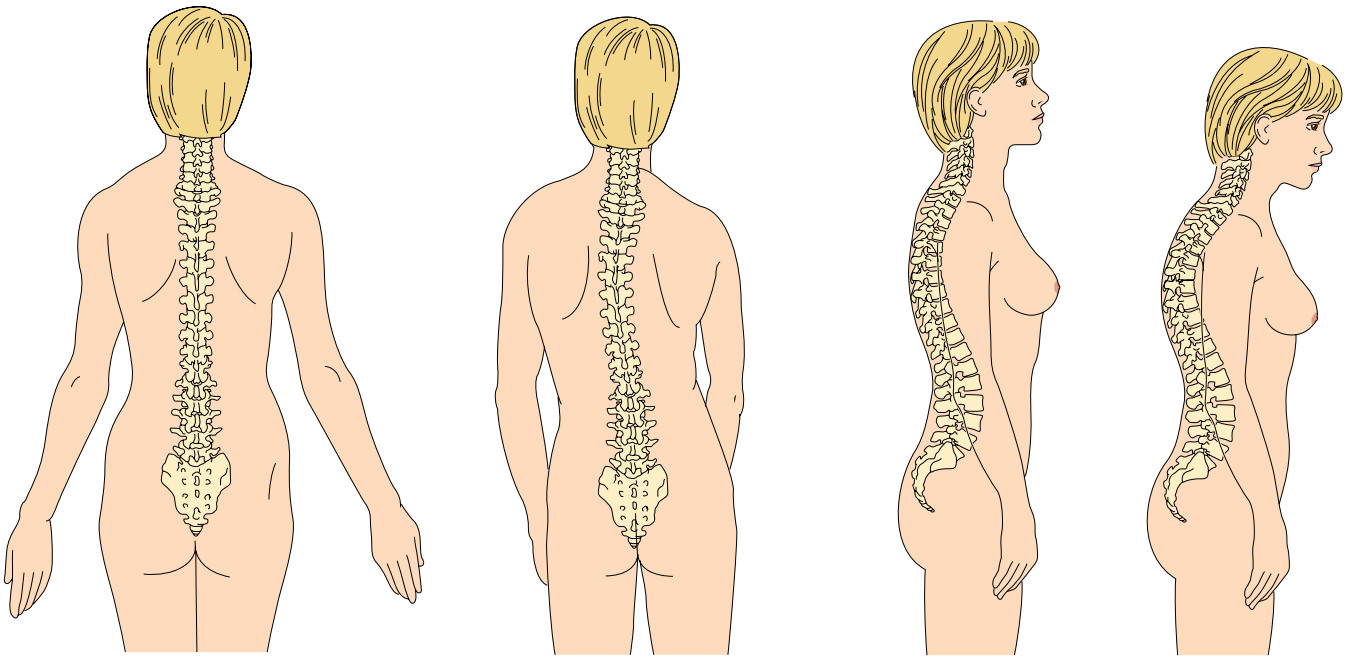


Figure 42–11 ■ Common deformities of the spinal column: *A,* Scoliosis is a lateral curvature of the spine. *B,* Kyphosis is an exaggerated posterior curvature of the thoracic spine.

disorders such as cerebral palsy, poliomyelitis, and MD account for the rest (Porth, 2005).

Pathophysiology

Detailed discussions of the causes and treatment of scoliosis and kyphosis in younger clients can be found in pediatric nursing textbooks. This discussion focuses on the nursing care of adults with these disorders. The manifestations of scoliosis and kyphosis are listed in the box on below.

Scoliosis

Scoliosis is classified as *postural* when the small curve corrects with bending, and *structural* when the curve does not correct with bending (Porth, 2005). Most clients requiring treatment have structural scoliosis, a curve caused by a fixed deformity.

The lateral curve that occurs in scoliosis is usually evident in the thoracic, lumbar, or thoracolumbar regions of the spine. The vertebral bodies in these spinal regions can be rotated as well as curved to one side or the other.

As scoliosis emerges, the soft tissues (muscles and ligaments) shorten on the concave side of the curvature. Over time, progressive deformities of the vertebral column and ribs develop, causing one-sided compression of the vertebral bodies. The degree of compression and twisting varies according to the location of each vertebra within the curved portion of the spine.

If the lateral curvature is less than 40 degrees when the client's spine reaches maturity, the risk of further progression during adult life is small. However, the spine becomes unstable if the lateral curvature is greater than 50 degrees, and curvature likely will worsen throughout the client's lifetime.

Scoliosis is usually first noted by the deformity it causes, such as one shoulder that is higher than the other, a prominent hip, or a projecting scapula. Pain is present in severe cases, usually in the lumbar region. Pain also may be caused by pressure on the ribs or the crest of the ilium. Shortness of breath may result from diminished chest expansion, and gastrointestinal disturbances may occur because of crowding of the abdominal organs.

Kyphosis

Like scoliosis, kyphosis is classified as postural or structural. Postural kyphosis is caused by a slumping posture. Structural

MANIFESTATIONS of Scoliosis and Kyphosis

SCOLIOSIS
- Asymmetry of shoulders, scapulae, waist creases
- Prominence of the thoracic ribs or paravertebral muscles on forward bend
- Lateral curvature and vertebral rotation on posteroanterior x-ray film

SEVERE SCOLIOSIS
- Back pain
- Shortness of breath
- Anorexia, nausea

KYPHOSIS
- Posterior rounding at the thoracic level
- Kyphotic curve of over 45 degrees on x-ray film

kyphosis may result from congenital malformations or pediatric disorders such as rickets or poliomyelitis. However, kyphosis also may occur during adulthood from vertebral tuberculosis and Paget's disease or from metabolic disorders such as osteoporosis and osteomalacia. The condition can also result from the surgical removal or radiation of intervertebral disks for the treatment of spinal cord tumors or cysts.

The manifestations of kyphosis include moderate back pain and increased curvature of the thoracic spine as viewed from the side ("hunchback"). Impaired mobility and respiratory problems may occur in cases of severe curvature.

INTERDISCIPLINARY CARE

Diagnosis of scoliosis and kyphosis is important to prevent severe spinal deformity in the adult. The client stands with the arms relaxed and hanging freely at the sides while the examiner evaluates the client from both the back and the front for symmetry of the shoulders, scapulae, waist creases, and the length of the arms. The client then bends forward, and the examiner observes for prominence of the thoracic ribs or vertebral muscles. The client is then viewed from the side while the screener looks for increased thoracic rounding or lumbar swayback.

A scoliometer is used to quantify the prominence of any curvatures noted during the examination. The scoliometer is placed at the apex of the curvature. A reading of greater than 10 degrees requires referral to a physician (Porth, 2005).

Diagnosis

Upright posteroanterior and lateral x-rays are used to confirm the diagnosis of curvature of the spine. For the client with scoliosis, the degree of curvature is measured by determining the amount of lateral deviation to the left or right. For the client with kyphosis, anteroposterior and lateral views typically reveal wedging of the vertebrae.

Treatments

Scoliosis and kyphosis may be treated conservatively or with surgery.

CONSERVATIVE TREATMENT Braces, electrical stimulation, and traction may be used to prevent progression of scoliosis and kyphosis in younger clients whose skeletons have not yet matured. Unfortunately, these approaches are ineffective in the adult client. Conservative treatment for adults with scoliosis and kyphosis may include weight reduction, active and passive exercises, and the use of braces for support.

SURGERY The use of surgery to correct spinal deformities depends on factors such as the degree of curvature and the client's overall physical, emotional, and neurologic status. Even with surgery, it is not possible to correct the abnormal curvature completely. The surgical procedure involves attaching metal reinforcing rods to the vertebrae, and is usually performed using an anterior approach, although more severe curvature may require both an anterior and a posterior approach. The types of straightening devices used most frequently use bilateral rods with wire hooks or screws that stabilize the spine and correct the deformity.

NURSING CARE

Nursing Diagnoses and Interventions

Nursing interventions focus on minimizing the risk for injury and neurologic impairment.

Risk for Injury

Clients with spinal deformities are at risk for injury from several sources, including structural aspects of bracing both before and after surgical intervention, dislocation of hooks and rods resulting from improper alignment or movement of the back, and changes in body position after prolonged immobilization.

- Assess the environment for safety hazards. *The client needs to learn to use handrails on stairways and take precautions when walking on slippery surfaces or areas with throw rugs.*

PRACTICE ALERT
Some braces do not allow the client to flex or hyperextend the spinal column.

- Teach the client ways to reduce irritation of skin surfaces beneath the brace: wearing a smooth cotton t-shirt or cotton tube under the brace at all times, changing undergarments at least once daily, and washing them with a mild soap. Undergarments should be changed more frequently in warmer weather. *The client wearing a brace is especially prone to skin breakdown and must take precautions to prevent it.*

PRACTICE ALERT
Teach the client to avoid lotion and body powders; they may irritate the skin.

- Teach the client to loosen the brace during meals and for the first 30 minutes after each meal. *Clients have difficulty eating if the brace is tight. Loosening the brace during and after each meal will allow adequate nutritional intake and promote comfort.*
- Teach clients how to apply the brace, and explain ambulatory restrictions. *Clients requiring a brace need to learn how to apply the brace prior to ambulating. Ambulation is frequently restricted to walking rather than sitting for long periods.*
- Turn clients who have undergone spinal surgery by using the log-rolling technique. Clients require a position change at least every 2 hours. *The use of a turn sheet and sufficient assistance allow the nurse to maintain the client's proper body alignment during the turning procedure.*
- Use a fracture bedpan following surgery. *The fracture bedpan provides minimal misalignment of spine and is more comfortable.*

Risk for Peripheral Neurovascular Dysfunction

Surgical procedures can lead to neurologic impairment in the client with a spinal deformity.

- Monitor the movement and sensation of lower extremities every 2 hours for the first 8 hours then every shift and as needed. *Neurologic assessment related to sensation and*

movement of the lower extremities is necessary because the surgical procedure is in close proximity to spinal nerves. Swelling of the surgical site can impinge on the spinal nerves and cause a loss of sensation and movement.

Community-Based Care

Clients with structural scoliosis or kyphosis need reassurance that the condition was not caused by poor posture. If a brace is prescribed to relieve pain and other symptoms associated with the disorder, provide verbal and written instructions for wearing the brace, such as the number of hours per day it is to be worn and activity restrictions to follow when wearing or not wearing the brace. Teach the client how to protect and care for skin under the brace.

Surgical clients need postoperative teaching regarding site care and activities. Clients who have spinal surgery often are allowed to ambulate fairly soon after surgery, but sitting may be restricted because of the stresses it places on the spine. Instruct the client to notify the physician if numbness, tingling, pain, or weakness of an extremity develops after surgery.

Discuss the importance of not smoking and of avoiding respiratory infections for clients with scoliosis or kyphosis that restricts respiratory excursion. Encourage these clients to obtain pneumococcal pneumonia and influenza immunizations.

THE CLIENT WITH LOW BACK PAIN

Acute or chronic low back pain involves the lumbar, lumbosacral, or sacroiliac areas of the back. In most cases, low back pain is due to strains in the muscles and tendons of the back caused by abnormal stress or overuse. Low back pain caused by degenerative disk disease and herniated vertebral disks is covered in Chapter 45 ∞.

Pathophysiology

The pathophysiology of back pain varies with its many causes (Box 42–5). In general, the five causes and types of back pain are as follows:

- Local pain is caused by compression or irritation of sensory nerves. Fractures, strains, and sprains are common causes of local pain; tumors also may press on pain-sensitive structures.
- Referred pain may originate from abdominal or pelvic viscera.
- Pain of spinal origin, that is, pain associated with pathology of the spine such as disk disease or arthritis, may be referred to other structures such as the buttocks, groin, or legs.
- Radicular back pain is sharp, radiating from the back to the leg along a nerve root. This pain may be aggravated by movements such as coughing, sneezing, or sitting.
- Muscle spasm pain is associated with many spine disorders, although its origin may be unclear. This type of back pain is dull and may be accompanied by abnormal posture and taut spinal muscles.

Manifestations

Clients with low back pain report pain ranging from mild discomfort lasting a few hours to chronic debilitating pain. Acute pain is usually caused when the client participates in an activity that is not usually pursued, such as unusual lifting or bending, playing an active sport, or shoveling snow. Manifestations are presented in the box below.

BOX 42–5 Factors Associated with Back Pain

Mechanical Injury or Trauma
- Muscle strain or spasm
- Compression fracture
- Lumbar disk disease

Degenerative Disorders
- Spondylosls
- Spinal stenosis
- Osteoarthritis

Systemic Disorders
- Osteomyelitis
- Osteoporosis or osteomalacia
- Neoplasms, primary or metastatic

Referred Pain
- Gastrointestinal disorders
- Genitourinary disorders
- Gynecologic disorders
- Abdominal aortic aneurysm
- Hip pathology

Other
- Fibromyalgia
- Psychiatric syndromes
- Chronic anxiety
- Depression

MANIFESTATIONS of Low Back Pain

ALTERATIONS IN GAIT AND FLEXION
- Walking in a stiff, flexed state
- Inability to bend at waist
- Limp, which may indicate impairment of the sciatic nerve

NEUROLOGIC INVOLVEMENT
- When tested for light and deep touch with a pin and cotton ball, may feel sensations in both limbs but experience a stronger sensation in the unaffected side
- Loss of both bowel and bladder control due to involvement of the sacral nerve

PAIN
- Pain in the affected leg when walking on heel or toes
- Continuous, knifelike localized pain in muscles close to the affected disk
- Pain that radiates down posterior of leg
- Sharp, burning pain in the posterior thigh or calf
- Pain in middle of buttock
- Tenderness when muscle close to the affected disk is palpated
- Severe pain with straight leg-raising maneuver

INTERDISCIPLINARY CARE

Care of the client with low back pain focuses on relieving pain, correcting the condition if possible, preventing complications, and educating the client.

Diagnosis

The choice of diagnostic tests for the client with low back pain depends on the suspected diagnoses, clinical findings, and history. Current guidelines for care recommend that radiography, CT scans, and MRI be used only with clinical signs of a potentially serious underlying condition. Diagnostic testing may be considered if pain and other manifestations continue to limit the client after 4 weeks of conservative treatment. Diagnostic tests are described in Chapter 40 ∞.

Medications

The medications of choice for low back pain include NSAIDs and analgesics. NSAIDs block prostaglandin production and reduce inflammation, thus relieving the pain. Muscle relaxants, such as cyclobenzaprine (Flexeril), methocarbamol (Robaxin), or carisoprodol (Soma), may be used, but little evidence supports their efficacy.

Epidural steroid injections may be used to help reduce intense, intractable pain. A steroid solution is injected into the epidural space, which helps decrease the swelling and inflammation of the spinal nerves.

Conservative Treatment

The majority of clients with acute low back pain need only a short-term treatment regimen. Limited rest, combined with appropriate exercise and education, is often the primary method of treatment. There is no evidence that activity is harmful or aggravating to the source of pain. In fact, activity promotes bone and muscle strength and may increase endorphin levels. Therefore, active rehabilitation helps to restore function and reduce pain.

Pain may be relieved by an ice bag or hot water bottle (or heating pad) applied to the back. Exercise programs are helpful provided that the client begins gradually and increases activity gradually as the recovery process continues. Physical therapy procedures include diathermy (deep heat therapy), ultrasonography, hydrotherapy, and transcutaneous electrical nerve stimulation (TENS) units. These therapies reduce the muscle spasms and pain temporarily. They are frequently used in combination with exercise to provide early mobilization for the client.

NURSING CARE

Nursing care of the client with low back pain focuses on relieving the pain. In addition, most clients have very little understanding of the anatomy of the spine, the reasons for the pain, the choices for treatment, and the importance of self-management. Therefore, education is another essential aspect of treating low back pain.

Health Promotion

Recommendations for preventing back pain from the National Institute of Neurological Disorders and Stroke include the following:

- Have a regular exercise program.
- Stretch before working in the yard, jogging, and playing sports.
- Quit smoking.
- Lose weight, if needed.
- Maintain a correct posture.
- Use supportive seats when driving.
- Lift by bending at the knees rather than at the waist.
- Reduce emotional stress that causes muscle tension.

In industrial and work settings, nurses should be alert for situations that increase the risk of back pain and injury. Office workers should have chairs with appropriate seat height and length and back support. Modifications of work space or machinery may be necessary for industrial workers to avoid excess stresses on back muscles. Finally, it is important to remember that back pain is a leading cause of lost work time for nurses themselves. Remind coworkers to use good body mechanics and to seek help when lifting or moving clients.

Nursing Diagnoses and Interventions

Nursing interventions for the client with low back pain are based on problems with acute pain, deficient knowledge, and risk for impaired adjustment.

Acute Pain

Muscle spasms and inflammation are among the contributing factors of low back pain.

- Teach the client appropriate comfort measures. *Clients with low back pain have discomfort due to muscle spasms and/or inflammation due to nerve compression, surgery, or irritation from a brace.*
- Instruct the client to take NSAIDs or analgesics on a routine schedule rather than as needed. *Maintaining a constant blood level of the NSAIDs or analgesics reduces inflammation and provides continuous pain relief.*

Deficient Knowledge

The client with low back pain requires information regarding treatment modalities.

- Encourage clients to stay active. *There is little scientific evidence to show that bed rest is beneficial, but there is ample evidence about the adverse effects of bed rest. Staying in bed for more than one to two days can actually increase pain, and cause joint stiffness and muscle weakness.*
- Teach the client about the "rebound phenomenon" of prolonged heat or ice therapy. *Ice remaining on the skin longer than 15 minutes or heat longer than 30 minutes causes a reverse effect known as the rebound phenomenon. For example, heat produces maximum vasodilation in 20 to 30 minutes. Continuation of the application beyond 30 to 45 minutes causes tissue congestion, and the blood vessels constrict. Likewise, with cold application, maximum vasoconstriction occurs when the skin reaches a temperature of 60°F (15°C). Prolonged cold can create a drop in temperature, at which time vasodilation occurs.*
- Provide instructions about appropriate back exercises such as partial sit-ups with the knees bent and knee-chest exercises to stretch hamstrings and spinal muscles. Each exercise

should be done 5 times and gradually increased to 10 times. Advise the client to discontinue any exercise that is painful and to seek professional advice before continuing the exercise. *Repetition of prescribed back exercises, such as the pelvic tilt, partial sit-ups, and back rolls, will strengthen the muscles that protect the spine and thus prevent back strain.*

Risk for Impaired Adjustment

The need for lifestyle changes may lead to impaired adjustment in people with back pain.

- Teach the use of appropriate body mechanics in lifting and reaching. The client should be instructed to plan the lift, keep the object being lifted close to the body, and avoid twisting when lifting. Encourage the client to obtain help when lifting. *An item is considered excessively heavy if it equals 35% of the lifter's body weight.*
- Instruct the client to modify the workplace or environment to minimize stress to the lower back. *Lumbar supports in chairs, adjustment of chair or table height, and rubber floor mats help prevent back strain or injury.*
- Encourage obese clients to lose weight. *The trunk of the body must carry excess weight when the client is obese. Obese people are farther away from the objects they lift because of their greater abdominal girth. They may also have more difficulty squatting to lift. The greater the distance between an object and the client's center of gravity, the higher the risk for straining the lower back.*

Community-Based Care

Back pain is a common problem in the United States and other industrialized countries. Nurses can have an effect on this significant problem by teaching health practices to prevent back injury to clients of all ages. Teach clients how to safely lift, bend, and turn when engaging in physical activity. Stress the importance of using large muscle groups of the legs to lift rather than bending and lifting with the smaller muscles of the back. Teach other aspects of good body mechanics, including posture, sleeping on a firm mattress, and sitting in chairs that provide good support. Discuss the positive effect of maintaining optimal body weight and good physical fitness.

THE CLIENT WITH COMMON FOOT DISORDERS

Hallux valgus, hammertoe, and Morton's neuroma are common foot disorders that cause pain or difficulty in walking. All three disorders may be caused by wearing poorly fitting or confining shoes. These disorders are more prevalent among women.

Pathophysiology

Hallux Valgus

Hallux valgus, commonly called a *bunion*, is the enlargement and lateral displacement of the first metatarsal (the great toe) (Figure 42–12 ■). Hallux valgus develops when chronic pressure against the great toe causes the connective tissue in the sole of the foot to lengthen so that the stabilizing action of the great toe is gradually lost. The toe bends laterally away from the midline of the body, and the metatarsophalangeal joint (MTP) is exposed to friction during walking and becomes enlarged. As the deformity progresses, calluses form over the metatarsal head, and bursitis develops in the MTP. In severe cases, the lateral displacement of the great toe may approach 70 to 90 degrees, and the second toe may be forced upward, causing hammertoe. Although bunions may be a congenital disorder, most are caused by wearing pointed, narrow-toed shoes or high heels.

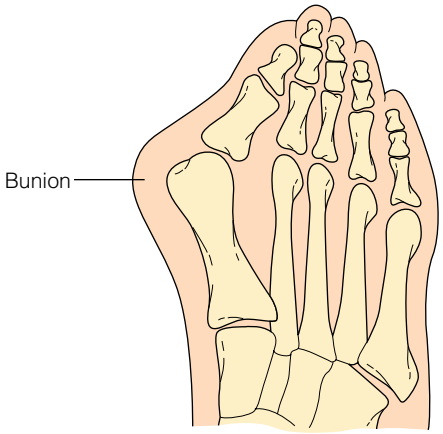


Figure 42–12 ■ Hallux valgus (bunion).

Hallux valgus is obvious on physical examination of the foot. The client may report an inability to fit into shoes. Often, the client may report joint pain or pain around calluses. In advanced or severe cases, the first metatarsal joint may have limited range of motion, particularly in dorsiflexion, and crepitus (crackling or popping) may occur during joint movement.

Hammertoe

Hammertoe (claw toe) is the dorsiflexion of the first phalanx with accompanying plantar flexion of the second and third phalanges. The condition may affect any toe, but the second toe is most commonly affected. Clients initially experience mild inflammation of the synovial membranes of the involved joints. As the deformity progresses, the dorsiflexed joint rubs against the overlying shoe, causing painful corns to develop.

Morton's Neuroma

Morton's neuroma is a tumorlike mass formed within the neurovascular bundle of the intermetatarsal spaces (Figure 42–13 ■). The neuromas usually occur in only one foot, most frequently in the third web space. Like other common foot disorders, Morton's neuroma usually is caused by wearing tight, confining shoes. The condition develops when repeated compression of the toes causes irritation and scarring of tissues surrounding the plantar digital nerve. The affected nerve becomes inflamed and swells. After repeated episodes of inflammation, the nerve fibers become fibrotic, and a neuroma forms.

Manifestations include a burning pain at the web space of the affected foot that radiates into the tips of the involved toes.

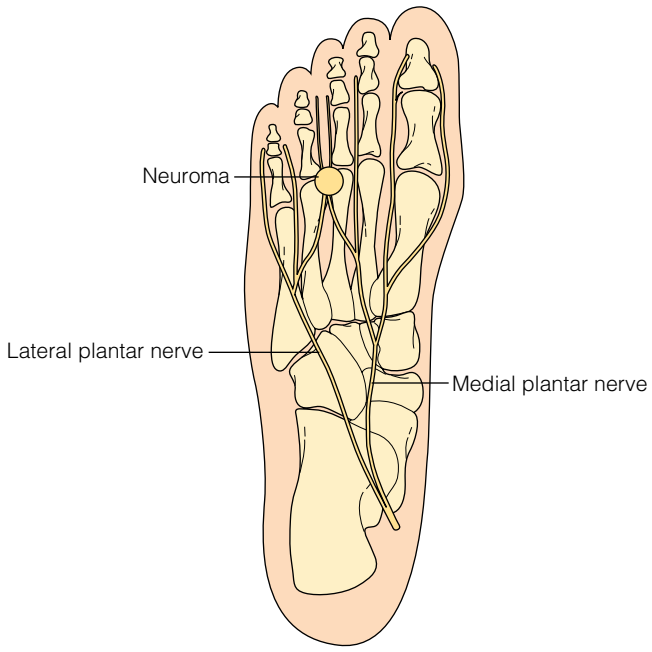


Figure 42–13 ■ Morton's neuroma.

Weight bearing usually worsens any symptoms; removing the shoe and massaging the foot often relieves the pain. The neuroma may present as a palpable mass between the affected toes. The area over the neuroma usually is tender.

INTERDISCIPLINARY CARE

Care of the client with common foot disorders such as hallux valgus, hammertoe, and Morton's neuroma focuses on relieving pain, correcting the structural deformity, and preventing reoccurrence. In most cases, all three conditions are diagnosed by inspection. X-ray films of the affected foot are taken if the need for surgery arises.

Conservative treatment for common foot disorders usually involves the use of corrective shoes. Orthotic devices that cushion and stretch the affected joints may be placed within shoes or between the client's toes. For Morton's neuroma, metatarsal pads are used to spread the client's toes and decompress the affected nerve. Analgesics may be prescribed to relieve pain and inflammation. In severe cases, corticosteroid drugs may be injected into the affected joints or surrounding tissue to relieve acute inflammation.

Surgery is reserved for clients with intractable toe deformities or pain. Hallux valgus is treated with bunionectomy; ligaments are lengthened or shortened as needed, and pins are drilled into place so the toe remains in position. Similarly, the correction of hammertoe also involves straightening the affected toe and inserting pins to retain the correction. A cast may be applied over the foot following surgery to correct toe deformities. Surgery for Morton's neuroma causes loss of sensation to a portion of the foot because removing the neuroma involves cutting out a portion of the plantar nerve.

NURSING CARE

Nursing care for clients with these foot deformities focuses on the same areas because the conservative treatment and preoperative and postoperative interventions are similar.

Nursing Diagnoses and Interventions

Pain relief, prevention of infection, and client education are important components of the nursing care of clients with foot disorders.

Chronic Pain

In the client with a foot deformity, constant pressure of footwear over the involved joint can cause pain.

- Instruct clients to wear corrective footwear to assist in the conservative treatment of foot problems. *Pain related to foot problems can result from improper footwear that does not provide proper toe room; in addition, heels higher than 1 inch can cause constant flexion and hyperextension problems. In some instances, the client must purchase special shoes or orthotics to ensure correct fit and relief of symptoms.*
- Suggest purchasing appropriate pads to wear over painful bunions, calluses/corns, and the ball of the foot. *Protective pads are manufactured for specific foot problems; these include bunion pads, corn pads, and metatarsal pads.*
- Instruct clients to remove pads and inspect the skin every other day. Clients who have difficulty reaching or observing the involved foot should ask another person to do the inspection for them. *It is especially important to emphasize the need for inspection to clients who have experienced loss of sensation of the feet due to such disorders as diabetes and chronic peripheral vascular disease.*

Risk for Infection

Like all surgeries, foot surgery carries a risk of infection. This risk may be increased because of impaired peripheral circulation and exposure of the feet to the environment.

- Teach clients proper care and cleaning of exposed pins implanted during the surgical procedure. *Pins inserted into soft tissue of the toes and bones are prone to becoming infected and can potentially result in osteomyelitis.*
- Teach clients how to keep pins and casts dry while bathing or ambulating in inclement weather. Clients must wear a plastic bag over the cast or pins when bathing or walking in rain or snow. *When casts or pins are exposed in water, infection may result.*

Community-Based Care

For clients in all age groups, teach the importance of well-fitting footwear. Discuss the long-term effects of wearing high-heeled shoes with constricting toes with women in particular. Suggest alternatives for stylish footwear, and encourage clients to wear supportive and nonrestrictive footwear at all times. Discuss the possible effects of bunions on balance, and talk about safety measures to prevent falls and injury. Teach clients techniques to relieve pressure on affected joints.

EXPLORE MEDIALINK

Prentice Hall Nursing MediaLink DVD-ROM

Audio Glossary
NCLEX-RN® Review

Animations

Arthritis
Carpal Tunnel
Muscular Dystrophy
Osteoporosis

COMPANION WEBSITE www.prenhall.com/lemone

Audio Glossary
NCLEX-RN® Review
Care Plan Activity: Lower Back Pain
Case Studies:
Diet and Gout
Hip Replacement
Rheumatoid Arthritis
MediaLink Applications
Compartment Syndrome
Osteoporosis Prevention
Links to Resources

CHAPTER HIGHLIGHTS

- Metabolic bone disorders begin in the bone remodeling process, and may result from aging, calcium and phosphate imbalances, genetics, and changes in hormone levels. The disorders include osteoporosis, Paget's disease, gout, and osteomalacia.
- Osteoporosis is a major health problem in the United States, with fractures being the most common complication. Health promotion activities to prevent development of the disease include a calcium-rich diet, weight-bearing exercise, and a healthy lifestyle.
- Gout is characterized by hyperuricemia and the deposit of tophi in the subcutaneous tissues. Attacks of the disease typically begin with an acutely painful inflammation of the first joint of the great toe.
- Degenerative musculoskeletal disorders include osteoarthritis (OA) and muscular dystrophy (MD). OA is the most commonly occurring of all forms of arthritis, and a leading cause of pain and disability in older adults. The disease is characterized by loss of cartilage in articulating joints and hypertrophy of bone at the articular margins. Pain and inflammation are most often conservatively managed with NSAIDs.
- If pain and disability are not controlled in clients with arthritis, total joint replacements may be performed.
- Autoimmune and inflammatory disorders of the musculoskeletal system include rheumatoid arthritis (RA), ankylosing spondylitis (AS), reactive arthritis (ReA), systemic lupus erythematosus (SLE), polymyositis, and Lyme disease.
- Although the cause of RA is unknown, it is believed to be a combination of genetic, environmental, hormonal, and reproductive factors. RA is a systemic disease, affecting one or many joints with the risk for severe contractures and deformity, and also causing fatigue, weakness, anorexia, weight loss, and fever. The primary objectives of treatment and care are to reduce pain and inflammation, preserve function, and prevent deformity.
- SLE is a chronic inflammatory connective tissue disease, affecting almost all body systems, including the musculoskeletal system. Skin lesions are a common manifestation, exhibited by a characteristic rash on the face. The client with SLE is at increased risk for infection.
- Lyme disease is caused by the spirochete *Borrelia burgdorferi*, carried and transmitted primarily by ticks. The disease can be treated effectively with antibiotics.
- Osteomyelitis and septic arthritis are infectious musculoskeletal disorders. Osteomyelitis may be the result of a bloodborne pathogen, a contiguous infection, or a complication of vascular insufficiency. Septic arthritis is a medical emergency, requiring immediate treatment to preserve joint function.
- Bone tumors may be benign or malignant, primary or metastatic. The primary manifestations of a bone tumor are pain, a mass, and impaired function. Nursing care is directed toward teaching to prevent injury and interventions to relieve pain.
- Scleroderma is a chronic disease characterized by the formation of excess connective tissue and diffuse fibrosis of the skin and internal organs. It may be either localized or generalized. Other connective musculoskeletal disorders are Sjögren's syndrome and fibromyalgia.
- Structural musculoskeletal disorders affecting the spine are manifested by scoliosis, kyphosis, and low back pain. Those commonly affecting the feet are hallux valgus, hammertoe, and Morton's neuroma.

TEST YOURSELF NCLEX-RN® REVIEW

1 Although all of the following nursing diagnoses are important when planning care for the client with osteoporosis, which is most significant in terms of long-term disability?
   1. *Chronic Pain*
   2. *Risk for Falls*
   3. *Activity Intolerance*
   4. *Acute Pain*

2 You are preparing a teaching plan for a woman with osteoarthritis. Which group of medications should you prepare to discuss?
   1. opioids
   2. antibiotics
   3. hormones
   4. NSAIDs

3 You are monitoring the laboratory reports for a client with an acute attack of gout. Which of the following measurements would you expect to be increased?
   1. hematocrit
   2. uric acid
   3. alkaline phosphatase
   4. creatinine

4 What is a potential complication of both osteoporosis and osteomalacia?
   1. infection
   2. blood clots
   3. fractures
   4. contractures

5 You are assessing a woman who has come to an orthopedic clinic complaining of knee pain. Which of the following assessments you made would indicate an increased risk for osteoarthritis?
   1. being overweight by 30 pounds
   2. having a history of falls
   3. eating a diet high in calcium
   4. walking 30 minutes each day

6 A postoperative nursing care plan for a client who has had a total knee replacement includes monitoring vital signs and laboratory results. The rationale for these interventions is to:
   1. teach the client the importance of these assessments.
   2. promote rapport between the client and the healthcare providers.
   3. ensure adequate circulation to the involved extremity.
   4. prevent the progression of infection.

7 When comparing osteoarthritis and rheumatoid arthritis, what assessment finding would be different in the client with rheumatoid arthritis?
   1. Health history includes weight loss and fever.
   2. Abnormal joint findings are limited to the hands.
   3. Stiffness is relieved by activity.
   4. Heberden's nodes are located on the finger joints

8 *Ineffective Protection* is an appropriate nursing diagnosis for the client with SLE. What would be your most important intervention for the hospitalized client?
   1. Monitor laboratory findings.
   2. Provide appropriate skin care.
   3. Practice careful hand washing.
   4. Administer prescribed medications.

9 How is the causative organism for Lyme disease spread?
   1. through the bite of an infected mosquito
   2. by brief contact with an infected tick
   3. primarily by droplets from infected people
   4. by an infected tick embedded for >24 hours

10 Of the different types of arthritis, which one is considered a medical emergency, requiring immediate diagnosis and treatment?
   1. osteoarthritis
   2. septic arthritis
   3. reactive arthritis
   4. gouty arthritis

See Test Yourself answers in Appendix C.

BIBLIOGRAPHY

Abdelhafiz, A., Lowles, R., Alam, N., Abebajo, A., & Philp, I. (2003). Clinical assessment of symptomatic osteoarthritis in older people. *Age and Aging, 32*(3), 359–360.

_____. (2005a). *FDA actions on COX-2 inhibitors & NSAIDs (Non-steroidal anti-inflammatory drugs)*. Retrieved from http://arthritis.about.com/od/arthritismedications/a/qufdaactions_p.htm

About Arthritis. (2005b). *FDA announces changes for all NSAIDSs; Bextra withdrawn from market*. Retrieved from http://arthritis.about.com/od/nsaids/a/fadaaction.htm

Altizer, L. (2004). Patient education for total hip or knee replacement. *Orthopedic Nursing, 23*(4), 383–388.

American College of Rheumatology. (2004a). *Glucocorticoid-induced osteoporosis*. Retrieved from http://www.rheumatology.org/public/factsheets/gi_osteopor_new.asp?aud=mem

_____. (2004b). *Lyme disease*. Retrieved from http://www.rheumatology.org/public/factsheets/lyme.asp?aud=mem

_____. (2005). *Background information on arthritis and rheumatology: Prevalence statistics*. Retrieved from http://www.rheumatology.org/press/index.asp?uad=mem

Arthritis Foundation. (2004). *Disease center: Osteoarthritis treatment*. Retrieved from http://www.arthritis.org/conditions/DiseaseCenter/OA/oa_treatment1.asp

Berarducci, A. (2004). Osteoporosis education: A health-promotion mandate for nurses. *Orthopaedic Nursing, 23*(2), 118–120.

Boston Total Joint Association. (2004). *Total hip replacement surgery.* Retrieved from http://www.bostontotaljoint.com/thr.html

Brown, S. (2005). Managing systemic lupus erythematosus. *Nurse 2 Nurse, 4*(11), 28–30.

Capriotti, T. (2004). The 'alphabet' of rheumatoid arthritis treatment. *Medsurg Nursing, 13*(6), 420–428.

Centers for Disease Control and Prevention. (2005). *Bone health*. Retrieved from www.cdc.gov/nccdphp/dnpa/bonehealth/

Cornell, T. (2004). Factfile. Ankylosing spondylitis: An overview. *Professional Nurse, 19*(8), 431–432.

Dochterman, J., & Bulechek, G. (2004). *Nursing interventions classification (NIC)* (4th ed.). St. Louis, MO: Mosby.

Easterbrook, L. (2003). Explaining about ... arthritis. *Working with older people, 7*(3), 7–9.

Flynn, J., & Johnson, T. (2005). *The Johns Hopkins white papers: Arthritis*. Baltimore, MD: Johns Hopkins Medicine.

Gill, J., Quisel, A., Rocca, P., & Walters, D. (2003). Diagnosis of systemic lupus erythematosus. *American Family Physician, 68*(11). Retrieved from http://www.aafp.org/afp/20031201/2179.html

Harvey, C. (2005). Wound healing. *Orthopaedic Nursing, 24*(2), 143–160.

International Scleroderma Network. (2004). *What in the world is scleroderma*? Retrieved from http://www.sclero.org

Kass-Wolff, J. (2004). Calcium in women: Healthy bones and much more. *Journal of Obstetrics, Gynecologic, and Neonatal Nursing, 33*(1), 21–33.

Kee, J. (2004). *Handbook of laboratory and diagnostic tests with nursing implications* (5th ed.). Upper Saddle River, NJ: Prentice Hall.

Lange, R., & Nies, M. (2004). Benefits of walking for obese women in the prevention of bone and joint disorders. *Orthopedic Nursing, 23*(3), 211–215.

Lucas, B. (2004). Does a pre-operative exercise programme improve mobility and function post-total knee replacement: A mini-review. *Journal of Orthopaedic Nursing, 8*(1), 25–33.

Mayo Clinic. (2002). *Osteoporosis*. Retrieved from www.mayocli8nic.com/invoke.cfm?id=DS00128

Moorhead, S., Johnson, M., & Maas, M. (2003). *Nursing outcomes classification (NOC).* (3rd ed.). St. Louis, MO: Mosby.

Morrow, M. (2004). Duchenne muscular dystrophy—A biopsychosocial approach. *Physiotherapy, 90*, 145–150.

Moss Rehab Resource Net. (2005). *Arthritis fact sheet*. Retrieved from http://www.mossresourcenet.org/arthritis/htm

NANDA International. (2005). *Nursing diagnoses: Definitions & classification 2005–2006*. Philadelphia: Author.

National Fibromyalgia Research Association. (2004). *Fibromyalgia syndrome*. Retrieved from http://www.nfra.net/

National Institute of Arthritis and Musculoskeletal and Skin Diseases. (2003). *Systemic lupus erythematosus; lupus and quality of life*. Retrieved from http://www.niams.nih.gov/hi/topics/lupus/slehandout/

_____. (2004a). *Questions and answers about arthritis and rheumatic diseases*. Retrieved from http://www.niams.nih.gov/hi/topics/arthritis/arthrheu.htm

_____. (2004b). *Questions and answers about knee problems*. Retrieved from http://www.niams.nih.gov/hi/topics/kneeprobs/kneeqa.htm

_____. (2005a). *Fibromyalgia*. Retrieved from http://www.niams.nih.gov/hi/topics/fibromyalgia/ffibro.htm

_____. (2005b). *Hyaluronic acid shows potential as biomarker for osteoarthritis*. Retrieved from http://www.niams.nih.gov/ne/highlights/spotlight/2005/hyaluronic_acid.htm

_____. (2005c). *What is gout?* Retrieved from http://www.niams.nih.gov/hi/topics/gout/ffgout.htm

National Osteoporosis Foundation. (2006). *Fast facts*. Retrieved from http://www.nof.org/osteoporosis/diseasefacts.htm

Nivens, A. (2004). Paget's disease: A case in point. *Orthopedic Nursing, 23*(6), 355–363.

Overstreet, M. (2005). Lyme disease: The dangerous hitchhiker. *Nursing Made Incredibly Easy, 3*(3), 38–44.

Paget Foundation. (2004a). *A health professional's guide to the management of Paget's disease of the bone*. Retrieved from http://www.Paget's.org/Information/FactSheet/mgmt_of_pdisbone.html

_____. (2004b). *A nurse's guide for assessment and management of patients diagnosed with Paget's disease of bone*. New York: The Paget Foundation.

Porth, C. M. (2005). *Pathophysiology: Concepts of altered health states* (7th ed.). Philadelphia: Lippincott.

Pullen, R., Jr. (2004). Caring for a patient on Plaquenil therapy. *Nursing, 34*(6), 32hn4, 32hn16.

_____, Cannon, J., & Rushing, J. (2003). Managing organ-threatening systemic lupus erythematosus. *Medsurg Nursing, 12*(6), 368–379.

Roberts, D. (2003). Alternative therapies for arthritis treatment: Part 1. *Orthopedic Nursing, 22*(5), 335–344.

Risley, S., Thomas, M., & Bray, V. (2004). Rheumatoid arthritis, new standards of care: Nursing implications of infliximab. *Journal of Orthopaedic Nursing, 8*(1), 41–49.

Schoen, D. C. (2004). Osteoporosis. *Orthopaedic Nursing, 23*(4), 261–267.

Scleroderma Foundation. (2006). *What is scleroderma?* Retrieved from http://scleroderma.org/medical/overview.shtm

Spondylitis Association of America. (2006). *Reactive arthritis/Reiter's syndrome, (ReA), Fast facts about ankylosing spondylitis (AS)*. Retrieved from http://www.spondylitis.org/about/reative.aspx

Taggart, H., Mincer, A., & Thompson, A. (2004). Caring for the orthopaedic patient who is obese. *Orthopaedic Nursing, 23*(3), 204–210.

Tak, S. H., & Hong, S. H. (2005). Use of the Internet for health information by older adults with arthritis. *Orthopaedic Nursing, 24*(2), 134–139.

Temple, J. (2004). Total hip replacement. *Nursing Standard, 19*(3), 44–51, 53.

Tierney, L., McPhee, S., & Papadakis, M. (Eds.). (2004). *Current medical diagnosis & treatment* (43rd ed.). Stamford, CT: Appleton & Lange.

Tretheway, P. (2004). Systemic lupus erythematosus. *DCCN: Dimensions of Critical Care Nursing, 23*(3), 111–115.

U. S. Food and Drug Administration (FDA). (2005). *COX-2 selective (includes Bextra, Celebrex, and Vioxx) and non-selective non-steroidal anti-inflammatory drugs (NSAIDs)*. Retrieved from http://www.fda.gov/cder/drug/infopage/COX2/

Vestergaard. P., Emborg, C., Stoving, R., Hagen, C., Mosekilde, L., & Briken, K. (2003). Patients with eating disorders: A high-risk group for fractures. *Orthopaedic Nursing, 22*(5), 325–331.

Wilkens, R. (2004). Making the most of antirheumatic drugs in older patients. *Journal of Musculoskeletal Medicine, 21*(6), 317–322.

Wilkinson, J. (2005). *Prentice Hall nursing diagnosis handbook with NIC interventions and NOC outcomes* (8th ed.). Upper Saddle River, NJ: Prentice Hall.

Wilson, B., Shannon, M., & Stang, C. (2005). *Prentice Hall nurse's drug guide 2005*. Upper Saddle River, NJ: Prentice Hall.

UNIT 12 BUILDING CLINICAL COMPETENCE
Responses to Altered Musculoskeletal Function

FUNCTIONAL HEALTH PATTERN: Activity-Exercise

Think about clients with altered activity-exercise patterns for whom you have cared in your clinical experiences.

- What were the clients' major medical diagnoses (e.g., sprain, joint dislocation, fracture, amputation, carpal tunnel syndrome, osteoporosis, Paget's disease, gout, osteomalacia, osteoarthritis, muscular dystrophy, rheumatoid arthritis, ankylosing spondylitis, systemic lupus erythematosus, polymyositis, Lyme disease, osteomyelitis, bone tumors, systemic sclerosis, scoliosis, low back pain, hammertoe)?
- What manifestations did each of these clients have? Were these manifestations similar or different?
- How did the clients' altered activity-exercise patterns interfere with their health status? Did pain increase with movement? Was the pain worse in the morning or did the pain increase throughout the day? Did they complain of muscle weakness or muscle cramps? Did they notice any redness or swelling of the joints? Had they ever had any muscle or bone diseases or injuries? Did they have any surgery or physical therapies for muscle or bone problems? Did they take any medications or herbal supplements for musculoskeletal disorders? Did they include foods with calcium in the diet? Did they have any family history of bone, joint, or muscle problems? Did they exercise regularly? Did they take part in strenuous activity or heavy lifting? Did they use assistive devices to move around?

The Activity-Exercise Pattern includes disorders that result in insufficient physiologic movement to carry out activities of daily living. Activity and exercise are affected by the ability of the musculoskeletal system to allow movement of the body and by perceived health status in two primary ways:

- Factors that result from trauma or surgery to tissues (e.g., contusion), tendons (e.g., strain, epicondylitis), ligaments (e.g., sprain), or bones (e.g., dislocation, fracture, amputation).
- Factors that result from medical disorders or deformities of joints (e.g., arthritis, gout, hammertoe), bones (e.g., osteoporosis, Paget's disease, osteomalacia, osteomyelitis, tumors, scoliosis), or muscles (e.g., muscular dystrophy, polymyositis).

The tissues and structures of the musculoskeletal system perform many functions, including support, protection, and movement. The bones form the body's structure and provide support for soft tissues. The bones protect vital internal organs from injury. The bones also store minerals and serve as a site for hematopoiesis (blood cell formation). The bones and joints of the skeleton and the skeletal muscles work together to allow the body to perform both gross, simple movements and fine, complex movements. Musculoskeletal disorders affect a client's perceived activity-exercise patterns, leading to manifestations such as:

- Pain (tissue damage ► stimulates sensory nerve endings ►which release chemical mediators such as bradykinin and histamine ►resulting in transmission of pain sensation to brain and nerve fibers)
- Limited mobility (disease or injury ►causes excessive loss of movement to muscles and joints ►resulting in reduction or restriction of range of motion to a body part)
- Edema (inflammation or infection ►causes impaired circulation, venous pooling, and proteins leaking into the interstitium ►resulting in edema).

Priority nursing diagnoses within the activity-exercise pattern that may be appropriate for clients include:

- *Risk for Disuse Syndrome* related to decreased range of motion, pain with movement, use of functional positioning splints
- *Risk for Falls* related to impaired balance, difficulty with gait, numbness of feet, decreased lower extremity strength
- *Impaired Physical Mobility* as evidenced by limited ability to perform gross/fine motor skills, uncoordinated or jerky movements, postural instability
- *Risk for Peripheral Neurovascular Dysfunction* related to musculoskeletal pain, pallor, diminished pulses, paralysis, and paresthesia.

Two nursing diagnoses from other functional health patterns often are of high priority for the client with altered activity-exercise patterns:

- *Impaired Skin Integrity* (Nutritional-Metabolic)
- *Risk for Disturbed Sensory Perception: Tactile (Cognitive-Perceptual)*

Directions: Read the clinical scenario below and answer the questions that follow. To complete this exercise successfully, you will use not only knowledge of the content in this unit, but also principles related to setting priorities and maintaining client safety.

CLINICAL SCENARIO

You have been assigned to work with the following four clients for the 0700 shift on an orthopedic unit. Significant data obtained during report are as follows:

- Jesse Drummond is a 70-year-old African American man with type 2 diabetes mellitus who is 3 days postoperative with bilateral below-the-knee amputations. Vital signs are T 99°F, P 88, R 24, BP 150/92. He is complaining of feeling pain in his feet.
- Joyce Stevens is an 84-year-old who is 2 days postoperative for hip replacement surgery. Her vital signs are T 99.6°F, P 100, R 30 and shallow, BP 110/86. She is confused when spoken to. Petechiae have been noted on her arms and legs. She is complaining of difficulty breathing.
- José Rivera, a 21-year-old, was admitted with osteomyelitis of the upper right leg. He has a history of a gunshot wound to the leg. Vital signs are T 102.6°F, P 98, R 22, BP 138/80. He is scheduled for surgical debridement of the wound this morning. He is complaining of pain and requesting pain medication.
- Kim Wong is a 30-year-old who was admitted with manifestations of painful and swollen joints, muscle pain, pale and cyanotic fingers and toes, and edema of the legs and periorbital areas. Her vital signs are T 100.6°F, P 78, R 16, BP 108/72. She is complaining of extreme fatigue. She is to have blood drawn for complete blood count (CBC), anti-DNA antibody testing, and serum complement levels.

Questions

1 In what order would you visit these clients after report?

1. _____
2. _____
3. _____
4. _____

2 What top two priority nursing diagnoses would you choose for each of the clients presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Jesse Drummond		
Joyce Stevens		
José Rivera		
Kim Wong		

3 After the amputation wound is dressed, what is the client taught to do to toughen the stump?

1. Dangle the stump for 20 minutes every hour while awake.
2. Push the stump into soft and then harder surfaces.
3. Elevate the stump on two pillows, keeping the knee straight.
4. Apply prosthesis over the compression dressing.

4 To prevent hip contractures in the client with an above-the-knee amputation, what does the nurse instruct the client to do?

1. Lie supine for short periods throughout the day.
2. Elevate the stump above the level of the heart.
3. Perform active range-of-motion exercises every 8 hours.
4. Avoid sitting in a chair for prolonged periods of time.

5 The nurse explains to the client with gout that a low-purine diet is recommended. The client understands a low-purine diet when which meal is ordered?

1. ham and asparagus casserole
2. chicken and dumplings
3. chili and spinach salad
4. shrimp and scallop pasta

6 The client's laboratory results are hematocrit of 28%, hemoglobin of 8 g/dL, WBC count of 4000/mm^3, platelet count of 98,000/mL, eosinophil sedimentation rate of 100 mm/h, positive anti-DNA antibodies. What medical diagnosis is supported by these lab values?

1. systemic lupus erythmatosus
2. rheumatoid arthritis
3. ankylosing spondylitis
4. polymyositis

7 Clients who have autoimmune diseases such as systemic lupus erythematosus are at increased risk for developing what disease?

1. chronic renal failure
2. hypertension
3. liver insufficiency
4. coronary heart disease

8 A prescription for ibuprofen (Advil) is given on discharge to the client with rheumatoid arthritis. Which toxic effects of the medication does the nurse instruct the client about?

1. diarrhea, nausea, and vomiting
2. blurred vision, tinnitus, and headache
3. gastric irritation, ulceration, and bleeding
4. dizziness, dry mouth, and abdominal cramps

9 When performing a neurovascular assessment, which are included in the initial and focused assessments? (Select all that apply.)

1. pain
2. paroxysm
3. pallor
4. pulses
5. paresis
6. pallesthesia
7. paresthesia

10 Which client is at greatest risk for developing osteoporosis?

1. menopausal, Caucasian woman who smokes one pack of cigarettes a day
2. menopausal, African American woman who has diabetes and hypertension
3. premenopausal, underweight Asian woman who is allergic to dairy products
4. premenopausal, obese African American woman who has a sedentary lifestyle

11 An older adult sprained an ankle after tripping on an uneven sidewalk. Which is the MOST important intervention?

1. Use a walker when ambulating.
2. Take anti-inflammatory and pain medications to reduce ankle pain.
3. Follow a regimen of rest, ice, compression, and elevation.
4. Immobilize the ankle with an air splint.

12 Which actions by the nurse need to be followed when caring for the client with osteomyelitis?

1. Place the client in a private room and use gloves and gown with wound care and good hand washing.
2. Place the client in a semiprivate room with another infected client and use isolation precautions for both clients.
3. Place the client near the nurse's station and use standard precautions when caring for the client.
4. Place the client at the end of the hall away from other clients to prevent spread of infection and teach the client to use good hand washing.

CASE STUDY

William Comfort is a 24-year-old Caucasian male admitted with a compound fracture of the left femur. He states he was riding a four-wheeler on a hill-side path and was thrown from the vehicle. He slid approximately 50 feet down the hill on his left side. His fall was stopped when his foot became tangled in some brush. On admission, his vital signs were T 99.8°F, P 100 and thready, R 24, BP 116/70. His height is 74″ and weight is 198 pounds. Assessment revealed an open fracture of the left leg with bleeding and edema around the open site and severe pain on movement of the leg. Popliteal and pedal pulses are difficult to palpate. His left leg is pale and cool to touch with a capillary refill of 4 seconds. He states his leg feels numb. Multiple lacerations and abrasions are noted on his left trunk and arm. He states he does not have any medical problems and has not seen a doctor in the past 5 years. He is employed as a computer repairman. He lives in an apartment with two friends.

Blood is drawn for a baseline compete blood count (CBC) and urine is obtained for a urinalysis. An intravenous line is started in the right arm with lactated Ringer's infusing at 150 mL/h. He is given a tetanus toxoid immunization and is medicated with morphine sulfate for pain. X-rays are taken of the left leg, left arm, and abdomen. The wounds are cleansed with an antibacterial solution and antibiotic ointment is applied. He went to surgery for an open reduction of the left leg fracture and has been placed in skeletal traction to separate the bony fragments and reduce and immobilize the left leg fracture.

The pathophysiology of a femur fracture is a large amount of force applied to the shaft of the femur, resulting in breaking of the bone. An open fracture is diagnosed when the bone is broken with bone fragments protruding through the skin. Manifestations of a femur fracture are edema, and a deformed and painful thigh. The client is unable to move the hip or knee. Popliteal and pedal pulses are difficult to palpate. Capillary refill time is increased. Pallor and coolness indicate arterial compromise. Sensations to the leg may be burning, numbness, prickly feeling, or stinging. Complications of a femur fracture include hypovolemia, fat embolism, dislocation of the hip or knee, muscle atrophy, and ligament damage.

Skeletal traction is the application of a pulling force through placement of pins into the bone. Pins are inserted under sterile conditions into the bone. One or more pulling forces may be applied to maintain alignment of the femur fracture. The disadvantages of skeletal traction are increased anxiety, increased risk of infection, and increased discomfort.

When planning nursing care for Mr. Comfort, the nursing diagnosis of *Impaired Physical Mobility* related to fracture of the left femur with skeletal traction is appropriate for implementing nursing interventions.