Anorexia nervosa is a psychological disorder with extensive physical complications that require care and monitoring from a diverse team of health care professionals. Although it can affect people of various ages and both sexes, anorexia most frequently occurs in adolescent girls during a crucial period of skeletal development. This article presents an overview of anorexia nervosa, followed by focused discussions of its effects on bone development, treatment of bone loss, and the radiologic technologist’s role in caring for anorexic patients.

After completing this article, the reader should be able to:
- Discuss risk factors for and the presentation of anorexia nervosa.
- List anorexia’s effects on various body systems.
- Describe changes in normal bone development caused by anorexia.
- Compare treatment options for age-related vs anorexia-induced bone loss.
- Explain the importance of the bone densitometry technologist in anorexia treatment and monitoring.

A certain patient stereotype likely comes to mind when discussing bone density or dual-energy x-ray absorptiometry (DXA) examinations: a somewhat feeble, elderly woman, attempting to stave off the natural decline that comes with age. Although most patients receiving bone density monitoring or treatment fit this stereotype, medical professionals should be aware of and prepared for a variety of patient types. Advances in bone densitometry technology and understanding of various conditions have expanded vastly the use and application of bone density measurements. Most notable have been expansions into care of pediatric and young adult patients, diagnosing and monitoring conditions that affect physical development, and helping to identify nutritional deficits.

Bone density examinations can be used to diagnose primary osteoporosis in children due to juvenile idiopathic osteoporosis or osteogenesis imperfecta. They also can be used to monitor conditions that result in secondary bone density changes, such as Cushing syndrome, hyperthyroidism, hypogonadism, and sickle cell anemia.1 Although many conditions are seen primarily in practices that specialize in a specific age group or type of underlying condition, other conditions should be familiar to all technologists working with bone densitometry. Eating disorders fall into that group because they can affect patients in a range of ages and without connection to an underlying physical illness. All technologists should understand what is happening to a patient with an eating disorder and how to provide the best possible care. Restrictive eating disorders, especially, have unique presentations, histories, and comorbidities that must be appreciated.

Nutrition

Nutrition plays a vital role in body development throughout infancy, childhood, and adolescence, and it directly affects proper functioning throughout
life. Healthy bone development and maintenance are most directly associated with adequate intake of calcium and vitamin D, along with vitamins A and C; both insufficiency and excess can cause predictable maladies. Indirectly, nutrition’s effects on weight, hormone secretion, energy, susceptibility to illness, and other aspects of overall health influence bone development.

The importance of nutrition begins even before birth, with the mother’s nutrition directly affecting early development and setting the stage for growth and development continuing into adulthood. The mother’s consumption of dairy products and vitamin D during pregnancy has been shown to have a direct correlation with fetal bone development and bone density in children into their teens. Lack of proper nutrition can cause intrauterine growth retardation and places infants at a deficit from which it can be difficult to recover, leaving them prone to type 2 diabetes mellitus, hypertension, stroke, and coronary artery disease later in life.

As infants and children change, so do their nutritional needs and susceptibility to various conditions. Infancy and childhood are marked by steady growth and development, requiring sufficient energy sources to support these processes and the essential building blocks for normal development to occur. Required caloric intake for active children increases from approximately 1000 calories per day at 2 years old to approximately 2000 calories per day or higher as children approach adolescence. Calcium and vitamin D are vital for the skeletal system, with recommended allowances for calcium ranging from 700 mg per day for toddlers up to 1000 mg per day for older children; vitamin D recommendations stay mainly consistent at 600 IU per day until a slight increase late in life.

Growth rate further accelerates during puberty and adolescence, reaching its peak and creating the highest nutritional demands within the lifespan. Caloric intake for moderately active boys and girls is highest during this time, with recommendations of approximately 3200 calories per day for boys and young men aged 16 to 18 years and approximately 2400 calories per day for girls and women aged 14 to 30 years. The daily calcium allowance increases to 1300 mg per day for people aged 9 to 18 years, the highest level of any point in a healthy lifespan. Calcium retention is also highest during adolescence.

In adulthood, once peak development has been reached, the body shifts to maintenance. Necessary caloric intake declines slowly but consistently after its peak and into old age. Calcium retention is lower than during adolescence or childhood, and the dietary calcium allowance decreases to 1000 mg per day after adolescence. Unlike caloric intake, however, calcium requirements change again later in life, increasing to 1200 mg per day for women older than 50 years and for men older than 70 years, as the bones begin to lose density and require additional calcium and vitamin D to maintain mass. After age 70, the daily allowance of vitamin D also increases to 800 IU per day.

Malnutrition

If the necessary amounts of nutrients are not consumed, development and function are hindered. Prolonged vitamin C insufficiency leads to scurvy (abnormal bone development in infants and children and swollen, painful joints) and reduced ability to fight infection or heal following injury. The body generally eliminates excess vitamin C through the urine, although consistently high levels of the vitamin can be harmful in certain circumstances. For example, an infant can develop symptoms of scurvy when the daily dose of vitamin C after birth is lower than exposure in the womb because of the mother’s high intake of ascorbic acid during pregnancy. High levels also can exacerbate gout and kidney stone formation.

Insufficient vitamin D results in rickets (failure of bones and teeth to develop normally) in children and osteomalacia (softening of the bones due to decalcification) in adults. Because vitamin D is fat soluble, excess also can be unhealthy, leading to diarrhea, nausea, weight loss, kidney damage, and calcification of soft tissues.

Lack of adequate vitamin A interferes with normal bone growth and development, as well as causing night blindness, xerophthalmia (dryness of the conjunctiva and cornea), degeneration of certain epithelial tissues, and susceptibility to infection. Because it is also fat soluble, excess vitamin A can be detrimental, causing peeling skin, hair loss, nausea, headache, birth defects, inhibited bone growth, and increased risk of fractures.

Insufficient calcium leads to stunted growth and misshapen bones in children and thinning bones and
higher fracture risk in adults. Calcium deficiency also can cause tetany (involuntary muscle cramps) when nerve function is affected. However, too much calcium can lead to calcium phosphate deposits in soft tissue or kidney stones. A proper balance of other nutrients, vitamins, and minerals is required for growth and development of all tissues and structures and efficient body function throughout life. Failure to maintain a healthy diet can result in a wide range of substance-specific disorders, malnutrition, and even starvation.9

When nutrition is poor, it is important not only to identify what is out of balance but also to determine the cause. Malnutrition can result from either undernutrition (deficiency in consumption or use of nutrients) or overnutrition (overeating or overuse of vitamin supplements). Primary malnutrition occurs as a direct result of diet alone. Secondary malnutrition occurs when an otherwise healthy diet is not sufficient for a particular individual because of an underlying medical condition or lifestyle. Starvation results from prolonged lack of nutrition, causing discernable changes in body function and metabolism. Body functions slow down to conserve energy, and the body begins to digest itself to preserve essential life functions as long as possible. Unresolved starvation eventually leads to death and can result from lack of resources (eg, extreme poverty), conscious choice of deprivation (eg, a hunger strike), or an eating disorder.5

An eating disorder occurs when an individual’s failure to maintain healthy nutrition is the result of psychological, rather than physical or environmental, causes. It is not the same as malnutrition due to lack of access to nutrients or constraints imposed by an outside force and is not a choice. An eating disorder might involve unhealthy overeating, losing the ability to regulate food intake, and even eating when uncomfortably full. Alternatively, a disorder can be restrictive, for example, if the person becomes preoccupied with controlling food intake and weight to the point of interfering with normal activities and nutritional detriment. This might involve overly strict control of calorie intake, attempts to rid the body of calories (through exercise, laxatives, or vomiting), or both. Eating disorders generally are connected to distortions in body image, perception of weight, self-worth, and control. Sufferers of all types of eating disorders—even if they are aware of their condition—believe they do not have a choice or control over their actions.9

Although people of varying ages, both sexes, and all backgrounds can have an eating disorder, some groups are affected more frequently, and certain risk factors are associated with a higher susceptibility. Girls and women are more likely to experience an eating disorder in their lifetime, with most cases occurring during adolescence, a crucial period of rapid physical development.10,11 Because of this commonality of demographic factors, the majority of anorexic patients present with common complications and comorbidities. Other risk factors include having a family history of eating disorders and having depression, obsessive-compulsive disorder, or another mental condition. In addition, excessive stress and intense pressure from coaches, parents, or others can contribute to the development of eating disorders. All these factors should be taken into consideration during treatment.10,12

Eating Disorders
Symptoms and complications of eating disorders vary from person to person, and each individual must be treated as a unique case; however, the fifth edition of the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders provides guidelines for making a diagnosis. There are 3 primary groups of disorders and a fourth category for individuals who do not fully meet the criteria for a specific condition. Conditions can be classified as anorexia nervosa, bulimia nervosa, or binge eating disorder (BED).13,14 Anorexia can be purely restrictive, such as when the patient attempts to avoid calories and possibly rid the body of calories through fasting or overexercising to “justify” eating. It also can include periods of binging, when the patient feels a loss of control over his or her ability to avoid eating, and purging, when the patient attempts to undo the “damage” of eating, with an overall loss of weight. Patients with anorexia lose weight to the point of starvation while obsessing over a perceived need to lose more weight and reduce food intake. They become thin, with distorted perceptions of their bodies.

Patients diagnosed with bulimia feel a loss of control over eating, alternating periods of binge eating with attempts to purge the body of food through induced
vomiting, laxatives, or overexercising. Symptoms might be similar to anorexia; however, these patients can appear to be of normal weight or even slightly overweight.

Understanding of BED and its acknowledgment as a defined eating disorder have expanded recently. BED differs from the previous 2 types of eating disorders in that it does not involve attempts to minimize caloric intake nor does it include purging calories. Patients with BED might be normal weight or overweight and are characterized by loss of control over food and feeling embarrassed or disgusted by their own behavior. They might be in denial over the amount of food they eat or attempt to eat in secret and hide their true intake. Other patients experience some distinct symptoms of eating disorders without meeting all the criteria for diagnosis and are therefore categorized as having an eating disorder “not otherwise specified.”

**Psychological Component**

Although each diagnosis is distinct, all are within the realm of mental disorders and reflect powerful underlying psychological elements. There is no single test to diagnose an eating disorder, nor a single medication or therapy to cure one. Diagnosis might require a physical examination, laboratory tests, and a thorough psychological evaluation to determine whether a problem exists and whether that problem results from purely physical causes or psychological components. A diagnosed eating disorder depends on an individual’s attitudes toward food and nutrition and could develop as an attempt to control overwhelming emotions or other aspects of life. Therefore, effective treatment begins with identifying and addressing the core issues. Many patients have signs of more than one condition; multiple disorders should be treated simultaneously for the best outcome. A long-term approach to mental well-being and correcting the root cause is necessary, or the disorder will persist.

**Physical Component**

Eating disorders attract the attention of medical professionals well beyond psychiatry because patients struggling with these conditions are prone to numerous physical manifestations that can severely affect development, quality of life, and even life expectancy. The restrictive nature of anorexia nervosa makes it the eating disorder most often assessed by bone densitometry. Although BED and bulimia affect bone density to some degree, the effects resemble those seen in other conditions. BED is similar to general obesity in spite of its psychological component; bulimia reflects dietary deficiencies. Anorexia, which is characterized by extreme weight loss, presents unique complications and the most severe changes in body composition and function. Eating disorders result in a higher mortality rate than other mental disorders, and anorexia is associated with the highest mortality rate of all.

Some effects of anorexia are obvious to people around the patient and can be noted on a physical examination by a physician. A key differentiation between anorexia and other eating disorders is significant weight loss below what is considered a healthy body mass index and, if untreated, to the point of starvation. Because of the associated weight loss, health care professionals are more likely to suspect a patient has anorexia based on physical appearance than BED or bulimia. Even to those not trained in health care, extreme weight loss can signal poor health and raise concerns. Many effects are not so readily visible, however, and add to the underlying disorder, causing further complications. Weight loss can affect hormone production and alter or stop menses. Both weight loss and hormone dysfunction can lead to poor bone development, bone loss, and increased risk of fracture.

Hair, skin, and nails change with malnutrition. Nails become brittle and easily damaged. Hair becomes dry, easily broken, and often, as a result of the extreme weight loss, begins to thin. Skin becomes dry and might be yellowish, especially on the palms, cuticles, and soles. These easily detected symptoms signal even more significant problems within the body. The yellowing skin, for instance, indicates significant slowing of the metabolism that causes accumulation of carotene that cannot be processed efficiently. A slowed metabolism is the body’s response to receiving fewer calories than needed to function, decreasing caloric usage in an attempt to survive potential starvation. The patient’s heart rate decreases, as does digestion, causing constipation. The patient also experiences fatigue and decreased tolerance of cold.
Anorexia poses serious hazards if untreated. Protein deficiency due to malnutrition and loss of lean weight can affect numerous vital functions and systems (see Figure 1). Protein deficiency can lead to electrolyte deficiency, impairing kidney function.\textsuperscript{9,10} Signiﬁcant weight loss is associated with the depletion of heart, brain, and other organ tissue, in addition to low body temperature and decreased growth in adolescents. It promotes a slowed metabolism and the related complications. Impaired cardiac function and loss of cardiac muscle increase the risk of heart failure. Among patients with severe anorexia, 30% to 50% experience chest pain due to mitral valve prolapse from decreased heart size.

Loss of cardiac muscle results in decreased blood pressure and heart rate, which can in turn cause dizziness, fatigue, and cognitive impairment.\textsuperscript{9,10} Severe symptoms require hospitalization; left untreated, problems can become fatal.

Cerebral atrophy, especially in adolescents, creates notable deﬁcits in cognitive, emotional, and social function. In people who have experienced extreme weight loss, computed tomography and magnetic resonance imaging show a reduced amount of gray and white matter and increased ventricle size.\textsuperscript{15} These changes interfere with decision making and interpersonal relationships. Problems are worst at the peak of anorexia, improving with recovery, but persisting somewhat even years later.\textsuperscript{9,10}

Assorted signs and symptoms result directly from hormonal changes. Gonadal hormones decrease in both sexes, causing decreased libido along with the possibility of amenorrhea and decreased ovulation in adolescent girls and women.\textsuperscript{9,10} Some improvement in menstrual symptoms can be seen with the use of oral contraceptives. Other possible dysfunctions include hypothalamic–pituitary dysregulation, hypothalamic amenorrhea, hypothalamic–pituitary–adrenal axis dysregulation, and hypothyroidism. Symptoms of hormonal changes can include sensitivity to cold, hypotension, increased cholesterol level, decreased fasting glucose level, and low heart rate. Retarded growth can occur during childhood or adolescence because of decreased growth hormones.\textsuperscript{9}

Hormone dysfunction, like other nutritional deﬁciencies, can negatively affect bone

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health at all stages of life. These changes to the skeletal system are of the greatest significance to the bone densitometry technologist.

**Normal Bone Development**

To understand how eating disorders affect the bones, it is important to consider normal bone formation and maintenance, which are complex processes that begin in the womb. In the early weeks of fetal development, the skeleton begins to grow from connective tissue and cartilage. Calcification and expansion continue throughout childhood and adolescence. Two types of ossification replace cartilage or fibrous tissue with bone: endochondral and intramembranous ossification.

Intramembranous bones are flat bones, such as the skull, mandible, and clavicles. During fetal development, these bones arise from layers of undifferentiated connective tissue. The connective tissue cells surround themselves in collagen fiber and differentiate into osteoblasts, forming an ossification center (see Figure 2). Osteoblasts are the cells responsible for the production of new bone throughout life. As the osteoblasts begin to secrete osteoid, some of them become isolated in the newly calcified bony matrix; these cells are known as osteocytes, or bone cells. The osteoid is deposited between the embryonic blood vessels, creating a random network of trabeculae. The growth and differentiation continue, producing broad, flat bones with spongy interiors encased in dense compact bone.

![Figure 2. Intramembranous ossification follows 4 steps. A. Mesenchymal cells group into clusters, and ossification centers form. B. Secreted osteoid traps osteoblasts, which then become osteocytes. C. Trabecular matrix and periosteum form. D. Compact bone develops superficial to the trabecular bone, and crowded blood vessels condense into red marrow. © ASRT 2017.](image-url)
Endochondral bones make up the majority of the skeleton. Long bones are formed on or within a hyaline cartilage model (see Figure 3). As the cartilage cells grow, they develop small cavities called lacunae before degenerating and leaving space for blood vessels and connective tissue cells to invade. Like intramembranous ossification, the cells further differentiate into osteoblasts and begin to deposit bony matrix around themselves. The osteoblasts trapped in the mineralized osteoid become osteocytes.\textsuperscript{16}

In long bones, ossification begins in the shaft, or diaphysis, which becomes the primary ossification center. Secondary ossification centers appear in the epiphyses at one or both ends of the bone. Epiphyseal plates of hyaline cartilage remain between the diaphyses and epiphyses; these plates allow continued expansion of the bone through growth. The plates expand outward, and osteoblasts invade the outer regions, secreting calcium salts and calcifying the cartilaginous extracellular matrix.\textsuperscript{16}

Osteoblasts eventually die, and osteoclasts move in to break down the matrix. Osteoclasts are multinucleated cells that secrete acids to dissolve inorganic components and enzymes to digest organic compounds. The osteoclasts break down central spongy bone, creating the medullary cavity and space for marrow. After the osteoclasts phagocytize the calcified cartilage, osteoblasts return to deposit new hard cortical tissue around the spongy bone. Bones continue to lengthen in this manner until the primary and secondary ossification centers meet, and the epiphyseal plates also ossify.\textsuperscript{1,16}

Once bones are formed and well before ossification is complete, osteoblasts and osteoclasts work simultaneously to remodel bones. Bone is broken down during resorption, and new bone is formed during deposition. The balance of these 2 processes is regulated carefully to produce growth when needed and maintain homeostasis when no change is warranted. Factors influencing the remodeling process include sunlight exposure, nutrition, hormones, and physical exercise.\textsuperscript{16} Each element ensures healthy development, and a problem in any area can disrupt normal growth.

Calcium is a key component of bone, a necessary building block to strengthen the bony matrix and prevent softening and deformity under pressure. Approximately 70% of bone weight is from inorganic material, and approximately 95% of this is calcium phosphate crystalline hydroxyapatite.\textsuperscript{17} Without vitamin D, calcium absorption is poor and calcium is not used efficiently, even if an adequate amount of calcium is consumed. Most vitamin D is consumed through eggs or foods fortified with vitamin D, such as milk.
but the body can use ultraviolet light from the sun to convert dehydrocholesterol produced in the digestive tract or consumed in the diet into vitamin D in the skin. Vitamin A is required for the function of osteoblasts and osteoclasts; a deficiency can therefore retard growth by slowing osteoblast activity. Collagen synthesis requires vitamin C for proper production; poor production in the extracellular matrix results in abnormally slender, fragile bones.²

Hormones that play a significant role in bone development are secreted by the pituitary gland, thyroid gland, parathyroid gland, and ovaries or testes. Growth hormone produced in the pituitary gland promotes cartilage growth in the epiphyseal plates. Without sufficient growth hormone, the long bones do not develop properly and pituitary dwarfism results. If excess growth hormone is secreted during childhood or adolescence, pituitary gigantism results, leading to extreme height. Excess growth hormone in adulthood results in acromegaly, which causes enlargement of the hands, feet, and jaw. Thyroxine, a hormone produced by the thyroid gland, stimulates production of bone tissue to replace cartilage in the epiphyseal plates. Excess thyroxine can halt growth, causing the plates to ossify prematurely. Thyroxine also stimulates secretion of growth hormone from the pituitary gland; without it, secretion slows, stunting growth. Parathyroid hormone has an opposite effect from thyroid hormones by stimulating the number and activity of osteoclasts to break down bone, hindering bone growth and maintenance, if present in excess. Testosterone and estrogen promote bone production and ossification, although estrogen has a stronger effect. Increased levels of sex hormones during puberty accelerate growth rate as well as the ossification of epiphyseal plates that stops growth.¹⁶

Even physical activity plays a role in bone development and growth. The pull of muscles on bones creates stress, generally at levels too low to be harmful, which in turn causes the bone tissue to thicken and strengthen to better handle the stress.¹⁴ In addition to physical stress, exercise has been found to promote the release of myokine irisin in skeletal muscles, which promotes strengthening and increased density of cortical bone.¹⁸ Frequent exercise, especially with weight training, forces bones to become stronger. Athletes typically have denser, stronger bones than otherwise healthy nonathletes with typical growth. Immobilization or other lack of exercise eliminates the opportunity to strengthen the bone and can cause atrophy to the point of thinning or shortening of the bone. According to Wolff’s law, physiological or mechanical forces also influence the shape and orientation of bones during modeling to best meet the specific demands of the environment."¹⁷

**Peak Bone Mineral Density**

Bone development begins in the womb and continues throughout childhood and adolescence. Bones experience modeling with growth, adapting to physical needs and environmental influences with a net gain of bone tissue. Throughout these periods, bones do not grow at the same rate for all children and might not correlate with chronologic age. The degree to which bones are formed and epiphyses are fused can be used to determine bone age, a better reference for expected bone density throughout development than chronologic age. Because sex hormones greatly affect bone development, sexual maturation stage also can be used to establish a more accurate reference point. Evaluations of bone health and fracture risk must take into account these developmental factors and not make comparisons with adult norms.¹

Net bone growth occurs at a rate of approximately 3% to 4% per year for children and adolescents, after approximately 8 years of age.¹⁹ As the body reaches a certain point in growth and bones reach a certain length and size, signals within the body change to terminate the net gains. Bones, including growth plates, ossify to prevent further increases.

The point at which growth stops and bones are fully ossified closely correlates with the point of peak bone density. This is the point at which bone density is the highest it is expected to reach with healthy growth and should be comparable to averages for healthy adults of the same sex and size, and the reference point for determining future bone degradation. The exact age of peak bone mineral density (BMD) varies among skeletal sites and among individuals because of differing rates of development. For the average individual, peak BMD is not achieved until a few years into adulthood, around
the early 20s for women and slightly later, around the mid-20s, for men (see Figure 4).  

**Maintaining Bone Mass**

Once peak bone mass is achieved, the body’s processes shift to maintenance and protecting against structural loss. Modeling is no longer common after the age of 20, when the individual has ceased growing. After this point, remodeling occurs constantly to maintain the structural integrity of bones and sustain the skeleton’s role in regulating extracellular fluid composition. Bone resorption and bone formation occur simultaneously, with formation following resorption to provide replacement bone equal to that which is removed. Any disease process or nutritional deficit that impairs this balance results in a change in bone mass. For structural stability, a turnover rate of approximately 2% to 5% per year allows for repair and prevents fatigue damage. To maintain extracellular fluid regulation, a rate of approximately 15% to 35% per year is required, primarily in the trabecular bone of the axial skeleton adjacent to the red marrow. An increased or reduced bone turnover rate, even if no change in bone density is observed, is associated with increased risk of fractures.  

**Normal, Age-related Bone Loss**

As the body ages, certain changes are anticipated. Even in otherwise healthy individuals, the cortex of bone thins over time, changing composition and density.  

Hormonal changes, especially the decline in estrogen after menopause, changes in activity level and diet, and increased disease and injury incidence all can affect bone density and structure with a measure of predictability. For this reason, bone density is not only compared with levels in healthy young adults, but also with age-adjusted averages, similar to studies in pediatric patients. Bone loss compared with the peak BMD is considered normal to a certain extent, and age-adjusted values decline with advancing years. Even substantial bone loss might be predictable based on certain traits or lifestyle choices. Some known risks for secondary osteoporosis include long-term use of glucocorticoids, a history of alcoholism, emphysema, epilepsy, hormone therapy for prostate cancer, and long-term heparin use, among many others.  

Changes due to normal aging—or from any of the risk factors for secondary osteoporosis—affect the integrity of the bones and increase the risk of fractures and complications from fractures, increasing mortality rates.

**Anorexia’s Effects on Bone Development and Maintenance**

For patients with eating disorders, especially anorexia nervosa, the normal progression of bone development and maintenance is hindered. The severity of the disorder, duration of symptoms, and stage in life at which it occurs influence the extent and reparability of the damage. During childhood, eating disorders are less likely to present with drastic loss of bone density than with failure to
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gain at a typical rate. The decreased rate might be most evident in changes in height or weight. Longitudinal growth is stunted under the unfavorable conditions imposed by the eating disorder. Future growth might not fully compensate for these delays, although it is possible for the patient to still reach his or her growth potential. Malnutrition increases the risk of fractures and deformities during modeling.

The population most at risk for anorexia is also the group that experiences the highest rate of bone growth and development, namely, adolescents 13 to 18 years of age. This correlation has drastic consequences for bone health, not only while the individual struggles with anorexia, but also throughout his or her life. Although bone mass is expected to be lower than adult norms while growth is still occurring, anorexia in adolescents is closely tied to BMD below expected values for individuals of the same sex at comparable developmental stages, with decreased rates of bone accrual. Patients could have up to a 0.3% decrease in bone density per year, as opposed to the approximate 3% to 4% increase experienced by healthy individuals of the same age. Up to 50% of adolescent girls and 70% of adolescent boys with anorexia are found to have an age-adjusted Z-score of less than –1 for at least one measured site. Approximately 11% of adolescent girls have a Z-score of less than –2. Because of the typical mass increase seen in healthy peers, the gap between bone density of teens with anorexia and those without anorexia continually increases throughout the duration of the illness.

In addition to adverse effects on overall bone density, the very structure and physiology of bone is altered. Patients with anorexia experience higher levels of preadipocyte factor-1, an epidermal growth factor-like protein involved in regulating the differentiation of mesenchymal stem cells into either osteoblasts or adipocytes. These higher levels result in higher marrow fat content as opposed to bone tissue. Further changes in cell differentiation yield higher fatty yellow marrow content and lower red marrow. Tests for bone formation and bone resorption markers show declines for both, decreasing bone turnover rate. Slower turnover results in increased fracture risk, independent of bone density.

Additional changes occur in bone structure at the microscopic level. Compared with healthy bone, trabecular volume and trabecular thickness are lower, whereas trabecular separation is higher, even when no variation is identified in BMD. These changes also increase fracture risk. Improvements in weight and overall health can repair a great deal of damage due to anorexia; however, the lost opportunity for bone growth during this period cannot be recovered fully. Patients might never reach their potential peak BMD and maintain lower bone density throughout life. Women who struggled with anorexia during adolescence exhibit lower BMD in adulthood than do women who had anorexia for the same length of time with onset in adulthood.

With onset in adulthood, an individual already has attained peak bone mass; however, risks to bone health persist because maintenance is an ongoing process. When bones would otherwise be preserved at peak BMD, patients with anorexia exhibit changes similar to bone loss following menopause. The inner bone appears similar to postmenopausal patients, with changes in structure, decreased trabecular thickness and increased spacing, and a decreased number of trabeculae per area. Overall bone density and cortical thickness also are lower than in comparable healthy patients, although not quite as low as expected after menopause. Bone formation markers decrease but, unlike in adolescence, turnover as a whole is not slowed; bone resorption markers increase, compounding the rate of bone loss. In adult anorexic patients, 92% have osteopenia, and approximately 40% have osteoporosis. A related increase in fracture risk is assured with changes in both bone density and physiology. Even if patients recover from anorexia, it can be difficult to recover the lost bone mass, and they likely are left at a deficit that will, at best, not worsen.

If anorexia occurs or persists into advanced age, or past menopause for women, bone loss is further complicated by age-related changes. Even healthy women older than age 50 and healthy men older than age 70 are expected to demonstrate thinning of cortical bone, decreased overall density, and need for targeted exercise and supplementation to protect bone health. An eating disorder speeds up these degenerative processes and

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clots are still a concern; however, risk of certain types of breast cancer actually are reduced.25

Other treatments target either bone production or bone resorption. Teriparatide (Forteo) acts similarly to parathyroid hormone to stimulate new bone growth in patients with severe osteoporosis (see Figure 5). A secondary medication is required to maintain the new bone. Teriparatide is administered by daily subcutaneous injections.25,26 Denosumab (Prolia) inhibits osteoclasts to reduce bone resorption. It is given only twice per year, also as a subcutaneous injection. Bisphosphonates, which also inhibit osteoclasts, are the most commonly prescribed group of medications for treating age-related osteoporosis. Common examples include alendronate (Fosamax), risedronate (Actonel), ibandronate (Boniva), and zoledronic acid (Reclast). These can be taken orally on a weekly or monthly basis, or intravenously on a yearly or quarterly schedule.25 Oral alendronate and risedronate are associated with compliance issues, even though pills are not required on a daily basis and improvements are expected in density and fracture risk. Absorption is poor, and these medications might bind to food, requiring the patient to take them with water before eating; afterward, the patient must remain upright for 30 minutes to minimize the risk of esophagitis.26

Options seem numerous, yet for patients with anorexia, standard treatments for improving bone density or impeding bone loss can be ineffective or less effective than for otherwise healthy patients. In addition, treatments not typically used for osteoporosis might be necessary to address underlying causes. Multiple therapies can be used concurrently to treat multiple factors influencing bone loss. Supplemental calcium and vitamin D are provided to address nutritional deficits because both are necessary for bone health; however, simply meeting the required intake of these nutrients has not been shown to increase BMD in patients with anorexia. Supplementation, as part of overall nutritional improvement, can promote healthy weight gain, which is directly correlated with improvements in bone formation. Yet, levels remain below those of healthy individuals. Correcting the weight and nutritional elements of anorexia is vital but only part of the solution for bone health. Because of the domino...
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Effect of many symptoms of the disease, further targeted therapy is required to optimize bone gains. Exercise, which is expected to improve general bone strength and density, is associated with a decrease in bone density in patients still struggling with anorexia, although it remains beneficial for patients who have recovered in regaining some of the density lost during illness. In adult patients who have increased bone resorption markers, bisphosphonates effectively aid in increasing BMD. In adolescents, however, who have decreased markers for both resorption and production, bisphosphonates are ineffective in improving bone density. Teriparatide is effective in increasing bone density; however, it is reserved for patients with a high risk of fracture. Some, but not all, hormone replacements can be beneficial if given at the appropriate stage of life. Oral estrogen–progesterone birth control pills, for instance, might help a patient resume menstruation but are not effective in increasing BMD. A small oral dose of estrogen given to girls with immature bone age or a transdermal patch administered to adolescents with mature bone age have produced improvements in bone density. A transdermal patch of testosterone administered to adult women failed to improve BMD, although testosterone patches did improve bone formation marker levels. Treatment with dehydroepiandrosterone, a steroid used to make androgens and estrogens, has proven to improve patient weight and mood, but it does not improve BMD. Short-term use of replacement insulin-like growth factor-1 produced improved BMD in adolescents, and replacement paired with estrogen improved BMD in adults. For some adult anorexic patients who have been unresponsive to other therapies, deep brain stimulation has been successful in helping patients reach a higher body mass index than achieved previously in treatment, while also improving other symptoms associated with anorexia.

Unlike age-related bone density loss, which can be managed with lifestyle changes and nutrition or a single pharmacologic treatment, deficits due to anorexia require a multifaceted approach. No single treatment plan has been deemed ideal standard treatment for all bone loss due to anorexia, and further research is needed to determine the specific effect of many therapies on anorexic patients.

Success requires treatment targeted at the various agents affecting bone development, as well as comprehensive treatment for the underlying eating disorder and psychological motivators. A team of professionals that includes the patient’s primary care physician, psychiatrist, and dietician usually collaborate to provide an extensive care plan on an outpatient or inpatient basis that fits the patient’s needs. Even in overcoming anorexia and receiving optimum treatment for symptoms, patients do not catch up to healthy peers and are likely to continue life with somewhat diminished skeletal integrity. All treatment options require ongoing monitoring of bone density and follow-up throughout life.
Bone Densitometry’s Role

Many methods of measuring bone density have been established throughout the years, and a few remain in use today to varying degrees. Quantitative ultrasound bone densitometry uses sound waves to evaluate both the density and quality of bone to determine fracture risk without the use of radiation. The ultrasound equipment is typically a peripheral device used primarily on the heel, with some units capable of measuring at the radius, finger, and tibia. Quantitative computed tomography (CT) can provide a 3-D image, allowing the isolation of a specific region of bone for measurement. The modality also takes into account total volume, not just area, to determine density. This ability can be especially desirable in pediatric patients whose bones are changing in size and shape. Quantitative CT can be single or dual energy and used for the spine, hip, tibia, forearm, or mandible. High-resolution quantitative CT is available for the radius and tibia and allows analysis of structural parameters along with density measurements.

DXA is the primary clinical tool for measuring bone density and the only method appropriate for official diagnosis of osteoporosis and osteopenia. It is the most frequently used technology to assess fracture risk and monitor the progress of these diseases and their treatments. As with quantitative sonography, the primary objective is not the creation of an image. DXA uses 2 distinct energy peaks of radiation passing through the patient to differentiate between bone and soft tissue and provide data on bone density and fracture risk in the spine, hips, forearm, calcaneus, and phalanges, along with additional measurements, such as total body composition.

The sites measured in DXA examinations are selected largely because of their high trabecular content, which demonstrate the effects of most pathologies and treatments faster than cortical bone because of trabecular bone’s higher turnover rates. Axial trabecular bone also exhibits a greater rate of change than appendicular bone. As a result, the spine and hips are most commonly used for standard examinations and monitoring. Choosing sites expected to have the greatest rate of change allows for more rapid follow-up and decisions regarding diagnosis and treatment planning (see Figure 6).

The expected precision of the scan also influences the timing of follow-up scans. Better precision produces less fluctuation in measurements, indicating true biologic change and providing greater confidence that the change indicated has truly taken place. With poor precision, the interval between scans is longer to allow for greater change and avoid unreliable results. Precision is affected by equipment variations and sites selected, as well as by the staff performing examinations. The greater the variability in positioning and analysis, the poorer the precision. For many adult patients, follow-up examinations require a wait of 1 to 2 years or more for reliability, depending on the therapy being monitored. Pediatric patients might have sufficient changes to allow follow-up scanning in 6 to 12 months.

Whatever the age of the patient, it is crucial to use the appropriate software and reference databases for each examination. A standard adult bone density report provides the following information:

- The bone density at the measured sites.
- A Z-score comparing the patient’s results to individuals of the same age, sex, and ethnicity.
- A T-score comparing the patient’s measurements to the average peak bone density for a healthy young adult.
- A fracture risk assessment.

The T- and Z-scores are given as standard deviations from a norm, indicating a relative greater or lesser risk of fracture. The fracture risk assessment is expressed as a percentage and represents the predicted risk of fractures within a specified time frame.

Because the bones of pediatric patients are still developing, it is inappropriate to compare these patients to adult norms. Bone densitometry software includes pediatric-specific databases that allow comparison of a particular patient with the demographic data for a healthy pediatric population. A pediatric bone density report does not include a T-score, but instead consists of data on bone size, density, and a Z-score adjusted for age, sex, and ethnicity. Failure to use an appropriate database for comparison can misrepresent bone density and the risk of fracture.

Understanding the normal development of the skeleton throughout the lifespan and how assorted disease processes present in densitometry can help the...
technologist identify a potential problem before the examination is even completed, recognizing potential pathology as opposed to software or equipment malfunction. Changes might be required in study protocol, or additional information might need to be collected. Recognizing scoliosis in the posteroanterior lumbar spine, for instance, might require selecting the proximal hip on the side of convexity, as opposed to an otherwise arbitrary selection of right or left hip. Inaccurate bone mapping could reveal that the correct software was not properly used to detect edges in low-density bone. For some equipment, the technologist must make that selection; others optimize settings based on height and weight data, which should be verified for accuracy.¹

The technologist should ensure the correct reference database is used for age, sex, and ethnicity. In pediatric populations, technologists also might need to obtain and document information on bone age and sexual maturation because that information provides more accurate references for healthy growth and might

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not correlate with chronological age. This information can aid the physician in more accurately determining whether intervention is necessary. In addition, when low bone density is found in pediatric patients, it is important to provide information on the size and shape of bones along with the density. A reportedly low BMD could be the result of abnormally low density, or it might reflect bones that are narrow or short for the patient’s age; knowing which is the likely cause is valuable to the pediatrician.

It might be necessary for the technologist to ask additional questions to help reveal the underlying cause of an abnormal presentation. Questions might cover conditions not listed on an initial questionnaire, possible past abuses (eg, physical trauma or lack of nutrition or medical care), as well as possible anomalies in development. Technologists should obtain as thorough and accurate a history as possible without bias toward one condition. Although slowed growth in an adolescent could indicate a potential eating disorder, it also could be associated with various endocrine and gastrointestinal disorders, along with other strictly physiologic causes (see Table). An imprudent technologist can cause undue stress to the patient or impede an accurate diagnosis through misdirection.

**Patient Care**

Just as much as technical competence is required to decide how best to execute the examination, effective patient care is required to ensure the examination is carried out successfully and with the most benefit to the patient. Even in the more typical bone densitometry patient, osteoporosis and bone loss can have numerous underlying causes or can even result from the combined effects of multiple agents. The caring technologist acknowledges the person as a whole and the complexity of bone health, without finding or implying fault on the part of the individual as to the cause of bone loss.

Various aspects of the patient’s health and life are considered when establishing the best treatment plan. Multiple factors—from diet and exercise to medication and supplementation—are considered, and a combined approach might be implemented to meet individual needs. Patients with eating disorders are treated in the same manner as other patients with bone loss, with the important addition of mental health care. Patients who have anorexia nervosa are dealing with multiple physical symptoms at once, while waging an internal war to

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### Table

**Differential Diagnoses for Unintentional Weight Loss**

<table>
<thead>
<tr>
<th>Area</th>
<th>Disorder/Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocrine</td>
<td>Hyperthyroidism, hypothyroidism</td>
</tr>
<tr>
<td></td>
<td>Hyperparathyroidism</td>
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<tr>
<td></td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td></td>
<td>Adrenal insufficiency (Addison disease)</td>
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<tr>
<td>Rheumatic</td>
<td>Rheumatoid arthritis, juvenile rheumatoid arthritis</td>
</tr>
<tr>
<td></td>
<td>Lupus</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Celiac disease</td>
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<td></td>
<td>Inflammatory bowel disease</td>
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<tr>
<td></td>
<td>Ulcerative colitis</td>
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<td></td>
<td>Crohn disease</td>
</tr>
<tr>
<td></td>
<td>Parasites</td>
</tr>
<tr>
<td></td>
<td>Superior mesenteric artery syndrome</td>
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<tr>
<td>Neurologic</td>
<td>Stroke</td>
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<td></td>
<td>Parkinson disease</td>
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<tr>
<td></td>
<td>Dementia</td>
</tr>
<tr>
<td>Psychiatric</td>
<td>Depression</td>
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<tr>
<td></td>
<td>Stress, anxiety</td>
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<tr>
<td></td>
<td>Obsessive-compulsive disorder</td>
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<tr>
<td>Immune system</td>
<td>HIV, AIDS</td>
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<tr>
<td></td>
<td>Hepatitis C</td>
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<tr>
<td></td>
<td>Tuberculosis</td>
</tr>
<tr>
<td>Pharmaceutical</td>
<td>Over-the-counter medications, herbal remedies</td>
</tr>
<tr>
<td></td>
<td>Prescription drugs</td>
</tr>
<tr>
<td></td>
<td>(eg, anticonvulsants, antidepressants, thyroid medication)</td>
</tr>
<tr>
<td></td>
<td>Prescription drug cessation</td>
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<tr>
<td></td>
<td>(eg, antipsychotics)</td>
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<tr>
<td></td>
<td>Substance abuse</td>
</tr>
<tr>
<td>Other</td>
<td>Chronic obstructive pulmonary disease</td>
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<tr>
<td></td>
<td>Congestive heart failure</td>
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<tr>
<td></td>
<td>End-stage renal disease</td>
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<tr>
<td></td>
<td>Malignancy</td>
</tr>
<tr>
<td></td>
<td>Excessive exercise</td>
</tr>
</tbody>
</table>
Anorexia Nervosa and Bone Densitometry

regain control of their own bodies. Health care professionals, no matter how brief their interactions with these patients, can either bolster the patient’s desire to conquer this disorder or add to the existing obstacles.

Whether performing a scan to diagnose a possible eating disorder, to monitor disease progression, or to evaluate the effectiveness of a therapeutic intervention, the bone densitometry technologist must use tact and empathy during the patient interview and throughout the examination. Although clinical history questions might seem routine for most patients, in the case of patients with anorexia, they can provide meaningful details and help guide appropriate treatment. The technologist should try to collect as much information as possible about the patient’s diet, any dietary supplements, exercise habits, and a female patient’s menstrual history. An accurate report about symptom duration and the time since initiating treatment, if applicable, provides the context for assessing the severity of bone damage and the efficacy of therapy. Unlike a primary care physician or other health care professional who sees the patient frequently, the technologist does not have the opportunity to build the same rapport over time, and these questions can be especially delicate if a diagnosis has not been made or treatment has not been initiated. In addition, the patient might be struggling with feelings of guilt or denial.

Care also should be taken in providing education to the patient recovering from or suspected of having an eating disorder. Standard advice given to osteoporosis patients regarding diet and treatment options can be inaccurate for patients whose bone loss is due to anorexia. The technologist must be aware of how treatment options change when dealing with patients at various stages of development and with various comorbid conditions to avoid misleading the patient or promoting unrealistic expectations of treatment.

All patients require an empathetic approach and a focused effort adapted to their unique situations, but patients striving to recover from eating disorders present a particular challenge in this regard. Health care professionals can become accustomed to straightforward medical conditions and diagnoses with known causes, easily recognizable presentations, predictable progressions, and standard treatment options. These types of conditions are more readily understood, often include familiar elements that health care providers have personally experienced to some degree, and are therefore more relatable. The etiology of eating disorders, however, is still not fully understood, and reliable treatment plans have not been established. The very nature of eating disorders involves development of behaviors that contradict social norms (eg, taking pleasure in food, building social experiences around eating and drinking) and even basic human instincts for survival (eg, consuming sufficient nutrients to protect the body, ensure organ function, and support the normal stages of growth). Not only are behaviors often baffling to health care personnel, but the attitude of patients toward the behaviors, such as the belief that there is nothing unusual or harmful in their actions, can be nearly incomprehensible.

It can be challenging for a health care professional to avoid displaying negative emotions in this situation. He or she might disapprove of the patient’s behavior or even feel averse to providing care. These feelings can be difficult to mask and could give the impression that the patient is at fault, responsible for bad outcomes because of poor decisions, or unworthy of receiving care. Even a professional who genuinely desires to help might exhibit frustration toward the patient because of an inability to connect, feeling helpless to make a difference, or lack of patient cooperation during care. The technologist need not have personal experience with eating disorders, however, to express genuine concern for patients’ well-being or a desire to help them receive the best care possible without judgment or blame.

Technologists must try to avoid dismissing or belittling a patient’s thoughts or feelings, even if they are not readily understood. Instead, the technologist should acknowledge the patient’s feelings and respond positively or redirect the conversation to something that the patient appropriately views as positive. Understanding the extent of anorexia’s effects on both body and mind and the potentially confounding attitudes the patient might have will help eliminate negativity stemming from misconceptions or frustrations. Even if caring gestures do not receive an overt positive response, they contribute to the supportive environment necessary for the patient’s long-term recovery.
Conclusion

Understanding the normal physiology of bone and how anorexia nervosa affects a patient at different stages of development will equip technologists to provide better care. Anorexia is a complex condition requiring coordinated treatment from multiple health care professionals to address the underlying psychological causes of the disease, as well as the various physical complications and symptoms. Anorexia affects patients of different ages and both sexes, but the highest rate of incidence occurs among adolescent girls at a crucial point in skeletal development. Changes due to anorexia greatly increase the risk of osteoporosis and fractures before recovery, with lingering effects on bone health throughout life, even after effective treatment. Technologists should understand how bone density fits into the broad spectrum of anorexia complications and how they can contribute to quality care.

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21. Ivaska KK, Gerdhem P, Väänänen HK, Akeson K, Obrant KJ. Bone turnover markers and prediction of fracture: a prospective follow-up study of 1040 elderly women for a
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* Your answer sheet for this Directed Reading must be received in the ASRT office on or before this date.

Read the preceding Directed Reading and choose the answer that is *most correct* based on the article.

1. After 50 years of age for women and 70 years for men, the daily calcium allowance:
   a. decreases gradually for the remainder of the lifespan.
   b. decreases to slightly below young adult levels.
   c. increases to 1200 mg per day.
   d. exceeds the recommended allowance for people aged 9 to 18 years.

2. Insufficient vitamin D can result in _______ in children or _______ in adults.
   a. rickets; osteomalacia
   b. scurvy; swollen joints
   c. thin bones; xerophthalmia
   d. stunted growth; tetany

3. An eating disorder occurs when an individual fails to maintain healthy nutrition as a result of:
   a. lack of access to nutrients.
   b. deliberate starvation.
   c. physical causes.
   d. psychological causes.

4. What is the key differentiation between anorexia nervosa and other eating disorders?
   a. alternating bingeing and purging
   b. extreme loss of weight
   c. no attempt to minimize caloric intake
   d. normal or overweight appearance

5. Symptoms of hormonal changes associated with anorexia can include all of the following *except*:
   a. sensitivity to cold.
   b. increased heart rate.
   c. increased cholesterol level.
   d. decreased fasting glucose level.

6. In long bones, the primary ossification center is located in the center of the:
   a. epiphyseal plate.
   b. medullary cavity.
   c. epiphysis.
   d. diaphysis.

[continued on next page]
Directed Reading Quiz

7. Parathyroid hormone stimulates the number and activity of:
   a. osteoblasts to break down bone.
   b. osteoblasts to build new bone.
   c. osteoclasts to break down bone.
   d. osteoclasts to build new bone.

8. When an individual has ceased growing, remodeling occurs ______ to maintain the structural integrity of bones.
   a. approximately once a year
   b. only after an injury
   c. constantly
   d. after osteoporosis develops

9. Which of the following are considered risk factors for secondary osteoporosis?
   1. long-term heparin use
   2. emphysema
   3. history of alcoholism
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3

10. Anorexia affects bone structure at the microscopic level, resulting in lower trabecular:
    1. volume.
    2. separation.
    3. thickness.
    a. 1 and 2
    b. 1 and 3
    c. 2 and 3
    d. 1, 2, and 3

11. Most adult patients with anorexia (92%) have:
    a. osteoporosis.
    b. osteopenia.
    c. a history of stress fractures.
    d. a Z-score less than –2.

12. Which hormone-related therapy for bone loss provides benefits for bone density comparable with that of estrogen, while acting as an estrogen antagonist in other areas?
    a. raloxifene (Evista)
    b. teriparatide (Forteo)
    c. denosumab (Prolia)
    d. risedronate (Actonel)

13. Simply meeting the required intake of calcium and vitamin D has not been shown to increase ______ in patients with anorexia.
    a. body mass index
    b. self-esteem
    c. bone mineral density
    d. heart disease

14. ______ is the primary clinical tool for measuring bone density and the only method appropriate for official diagnosis of osteoporosis and osteopenia.
    a. Computed tomography
    b. Quantitative sonography
    c. Positron emission tomography
    d. Dual-energy x-ray absorptiometry (DXA)

15. In DXA, precision is affected by equipment variations, sites selected, and the staff performing examinations.
    a. true
    b. false

16. A pediatric bone density report includes all of the following except:
    a. bone size.
    b. density.
    c. T-score.
    d. Z-score.