Osteoporosis: Diagnosis and Conservative Treatment

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Abstract: Osteoporosis is diagnosed by identification of risk factors, diagnostic imaging modalities, and urinary or serum levels of certain bone biomarkers. Treatment goals for patients at risk for or diagnosed with low bone mineral density are achieving and maintaining peak bone mass and attenuating or reversing pathologic, age-related, and postmenopausal bone loss. To achieve these goals, medical treatment may consist of nutritional and pharmacological interventions; physical therapy treatment consists of exercise. Current research allows for tentative conclusions with regards to optimal exercise parameters.

Key Words: Osteoporosis, Diagnosis, Treatment, Exercise

In the first article of this two-part series on osteoporosis, I discussed the epidemiology of osteoporosis, the histology of bone, the influences on bone remodeling, and a classification of osteoporosis into primary and secondary forms. This second article uses that information as a basis for discussion of diagnosis and management of patients at risk for or diagnosed with decreased bone mineral density (BMD). The goal of this article is to enable the physical therapist to effectively screen patients for low BMD and to develop appropriate exercise interventions for patients at risk for or diagnosed with low BMD. This article is also meant to increase the therapist's knowledge of diagnostic modalities and interventions outside of the scope of physical therapy practice in order to facilitate and improve on patient education by the therapist. Conservative and surgical management of osteoporosis-related fractures is outside the scope of this article.

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Diagnosis

The diagnosis of osteoporosis is often first established by documenting a typical osteoporotic fracture[1]. Identifying the patient prior to such a fracture is much preferable. Identification of risk factors associated with osteoporosis, the use of appropriate diagnostic imaging modalities, and measuring the serum and urinary levels of certain bone biomarkers may allow for earlier diagnosis.

Risk Factors

As stated in the first article, the physical therapist may be confronted with a patient with undiagnosed low BMD. Forces applied normally within a therapeutic session may exceed the lowered fracture threshold and cause harm to the patient. Identifying the patient at risk for decreased BMD may help the therapist make safer choices when selecting interventions used and forces applied during these therapeutic sessions. The identification of risk factors

associated with osteoporosis (Table 1) is the only diagnostic tool available that is within the scope of practice of the physical therapist.

A structured history may reveal the dietary deficiencies, endocrine disorders, gastrointestinal diseases, bone marrow diseases, connective tissue diseases, medication use, and miscellaneous causes of secondary osteoporosis as discussed in the first article (Table 2), including the influence of caffeine, tobacco, and alcohol, and the effects of diet on calcium absorption and excretion.

Female gender is another risk factor: peak bone mass (PBM) is generally higher in men[2]. Bone mass also correlates positively with skin pigmentation[2]. Bone mass is equal in blacks and whites until adolescence, but thereafter it increases to a greater level in blacks[3]. Darker-skinned races also have slower bone turnover, higher intestinal calcium absorption, and higher blood calcium levels[4,5]. Bottomley[4] hypothesized that these differences may be the result of increased vitamin D production due to less refraction of sunlight. The combination of gender and race puts black men at the lowest risk and white women at the greatest risk for developing osteoporosis[2]. Asian women are in the same risk category as white women; Hispanic women are less at risk than white women but are not as protected as black women[5].

Body build is related to bone fragility: thin women have less cortical bone and are, therefore, at greater risk for fractures[2]. Obesity may protect women from osteoporosis in multiple ways. Increased body weight will cause increased gravity-induced strain on the bone, which may result in increased PBM[2,6]. Adipose tissue is the site where androgens are converted into estrogen; it also acts as a storage unit for estrogen[5,6]. Low body fat may result in insufficient estrogen production[3], while obesity may increase the amount of biologically available estrogen[2].

Women with a family history of osteoporosis run a higher risk for developing osteoporotic fractures[2]. BMD seems at least partly genetically determined: monozygotic

Table 1. Risk factors for osteoporosis[2-6]

- Age 50 and older
- Female
- Caucasian or Asian
- Postmenopausal
- Northern European ancestry
- Small, bony body frame
- Low body fat
- Family history of osteoporosis
- Failure to gain weight normally in puberty
- Pregnancy at an early age
- Long periods of inactivity
- Medical history positive for secondary causes of osteoporosis as outlined in Table 2

Table 2. Secondary causes of osteoporosis[1-5,11,14]

Dietary deficiencies
- Insufficient intake calcium and/or vitamin D
- Excessive consumption of phosphates, oxalates, alkalis, fatty acids, dietary fibers, proteins, refined sugar, caffeine, alcohol, and sodium

Endocrine diseases
- Female hypogonadism
  Hyperprolactinemia
  Hypothalamic amenorrhea
  Anorexia nervosa
  Premature and primary ovarian failure
  Oophorectomy
- Male hypogonadism
  Idiopathic hypogonadotropic hypogonadism
  Hyperprolactinemia
  Klinefelter's syndrome
  Primary gonadal failure
  Delayed puberty
  Cystic fibrosis
- Hyperthyroidism
- Hyperparathyroidism
- Hypercortisolism (Cushing's disease)
- Growth hormone deficiencies
- Untreated diabetes mellitus

Gastrointestinal diseases
- Subtotal gastrectomy
- Malabsorption syndromes
- Chronic obstructive jaundice
- Primary biliary cirrhosis and other cirrhoses
- Alactasia

Bone marrow disorders
- Multiple myeloma
- Lymphoma
- Leukemia
- Hemolytic anemias
- Systemic mastocytosis
- Disseminated carcinoma

Connective tissue diseases
- Osteogenesis imperfecta
- Ehlers-Danlos syndrome
- Marfan's syndrome
- Homocystinuria

Medication
- Heparin
- Glucocorticoids
- Thyroxine
- Anticonvulsants
- GnRH agonists
- Cyclosporine
- Chemotherapy

Miscellaneous causes
- Immobilization
- Rheumatoid arthritis
- Smoking

twins have been shown to have a significantly greater similarity in bone density of the radius than do dizygotic twins[6]. A failure to gain weight normally during puberty predisposes the individual to decreased PBM and osteoporosis in later life[4]. Pregnancy at an early age, when the skeletons of both the mother and the fetus are maturing simultaneously, may also result in lower BMD and increased risk for perimenopausal bone loss[5].

Diagnostic Imaging

Ordering diagnostic imaging modalities is usually outside the scope of practice of the physical therapist. Knowledge of the modalities available may, however, facilitate suggestions to primary care providers, especially when multiple risk factors have been identified during a structured history taking. It may also improve patient education regarding the diagnostic imaging modalities used. Correct interpretation of BMD measurements can guide physical therapy treatment decisions[7].

Mass screening for osteoporosis is not warranted[8]. However, Seeger[8] noted that bone densitometry may be indicated for the following select patient groups:

- Perimenopausal women with several identified risk factors
- Young premenopausal women with prolonged amenorrhea (of more than six month's duration)
- Patients with diseases or on medications, that may adversely affect BMD
- Premenopausal women with a high number of risk factors, for whom a low BMD value is expected to assist in positive lifestyle changes, thus reducing the risk of fracture in later life

Multiple imaging methods are available to determine bone density. Nuclear scanning techniques were among the earliest imaging techniques used for bone densitometry. *Single-photon absorptiometry* (SPA) uses iodine-125 as a gamma ray source[8] and operates on the principle that density of cortical bone is inversely proportional to the quantity of photons passing through. The radio-isotope emits a single-energy beam of photons that passes through the bony structures of the forearm, while a sodium-iodide scintillation counter is moved across the opposite side of the forearm to detect photons transmitted through the bone[3]. SPA is limited to use in appendicular areas with minimal soft tissue, such as the radius and calcaneus. A water bath may be used to correct for overlying soft tissues[8]. *Dual-photon absorptiometry* (DPA) uses an isotope source that emits two discrete energy photons. This allows for correction for overlying soft tissues; a water bath as with SPA is not necessary. DPA can be used to determine BMD in the spine and proximal femur[8]. Scanning time with SPA and DPA are long and nuclear scanning has been largely replaced by other methods[8].

Plain radiography has been used to determine the presence of osteopenia. However, sensitivity is low: a bone mass decrease of at least 30% is needed before it can be detected on plain radiographs[2,3,9]. Overexposure and underexposure alter apparent radiodensity; this decreases the reliability of densitometry by plain radiographs[9]. The *Singh index* grades osteoporosis based on the finding that the five major trabecular groups of the proximal femur are resorbed in a predictable, sequential manner as the disease progresses[9]; recent studies, however, have shown no correlation between the Singh index and the more widely used dual-energy X-ray absorptiometry (DEXA) measurements[8]. *Radiographic absorptiometry* assesses compact and cancellous bone of the second to fourth metacarpal bones measured against an aluminum reference wedge[8]. X-ray absorptiometry uses an X-ray rather than the isotope-based projection system of SPA and DPA[3]. *Single-energy X-ray absorptiometry* uses a water bath as does SPA to correct for overlying soft tissue; as with SPA, it can only be used at the radius and the calcaneus[8]. As discussed in the first part of this series, the World Health Organization (WHO) has adopted *dual-energy X-ray absorptiometry* as its standard imaging technique for the classification of osteopenia and osteoporosis[10]. Compared to DPA, DEXA has superior precision, lower radiation doses, shorter examination times, higher image resolution; and it is technically easier to do[3]. Traditionally, evaluation of the spine with DEXA has been performed in an anteroposterior direction: prevertebral vascular calcifications and osteophytosis may result in an artificially high BMD[8]. Lateral DEXA scans avoid these imaging pitfalls[1,8]. DEXA converts a three-dimensional body into a two-dimensional image: the measurement is calculated by dividing total bone mineral content (BMC) by the projected area of a specific region. BMD measurements using DEXA, therefore, do not measure true volumetric density, but rather integrated areal density of both compact and cancellous bone[7]. DEXA scans are widely used. The therapist needs to be able to interpret these scans as they can guide physical therapy intervention. There are three ways for reporting BMD from DEXA measurements: an unadjusted score in grams per centimer-squared, a T-score, or a Z-score. To get the T-score, the deviation of the patient's BMD score from the mean BMD of the young adult reference population, used by the WHO to define osteoporosis, is divided by the standard deviation of this same reference group. (See the discussion of the WHO classification in the first article.) The Z-score uses the same mathematical formula but compares the patient to an age- and sex-matched reference population[7].

Quantitative computed tomography (QCT) is the only imaging modality that generates a three-dimensional measurement representing a true density measurement rather than reflecting a surface area as do the other modalities[8]. QCT allows for specific visualization of the metabolically more active cancellous bone of the vertebral bodies mainly affected in type-I osteoporosis; this may make it more sensitive for diagnosing this type of

osteoporosis[1,8]. It simultaneously scans a phantom composed of tubes containing standard solutions of bone mineral equivalent; comparison of the phantom scan with the vertebral body scan allows for determination of BMD[3]. Radiation dose of QCT is higher than that of DEXA and difficulty in relocating the site of the initial measurement, especially in severely kyphotic or osteoporotic individuals, greatly affects its reliability and usefulness for serial measurements[3,8].

Quantitative ultrasound is a relatively new method that uses ultrasound transducers placed opposite each other, usually on the calcaneus. The most commonly used clinical parameter is broadband ultrasound attenuation. This parameter reflects trabecular orientation and structure rather than BMD[8].

Andreoli et al[1] reported that large prospective studies have demonstrated that BMD measurements of the distal and proximal radius, calcaneus, proximal femur, and spine can all predict the development of all major types of osteoporotic fractures. Seeger[8] noted that it may be true in the population as a whole that one site reflects the other, but that this is probably not so in the individual; measuring the site of greatest interest, whenever possible, may be preferable for determining site-specific BMD and fracture risk. Riggs and Melton[6] agreed; they reported that the correlation between BMD of the lumbar spine and radius or calcaneus is too low (r=0.5-0.8) to accurately predict vertebral density from these appendicular measurements in individual patients.

Bone Biomarkers

Osteoporosis can be categorized further as a high- or a low-turnover disorder. High-turnover osteoporosis is an osteoclast-mediated disease, characterized by greatly increased bone resorption. It occurs predominantly around menopause but may also occur in the elderly. Diagnosis of a high-turnover variant of osteoporosis puts the patient at greater risk for problems associated with rapidly decreasing BMD and has therapeutic implications, which will be discussed below[11]. Bone densitometry provides a measurement of how much bone is present at a certain point in time; it does not provide us with information regarding the rate of bone turnover. Serum levels of the osteoblastic enzyme bone-specific alkaline phosphatase, osteocalcin, and the *N- and C-peptides,* extracellularly removed from type-I pro-collagen, are used as biochemical markers of bone formation[1,12]. Urinary levels of the collagen degradation product *hydroxyproline*, the bone collagen degradation product *hydroxylysine-glycoside*, collagen cross-linking amino acids, collagen telopeptides (peptides involved in collagen cross-linking), and *pyridinolines* (peptide degradation products) are used as markers of bone resorption[12]. Bone-specific alkaline phosphatase and osteocalcin levels appear to be the best markers for bone formation; telopeptide markers seem to be the most specific and responsive markers of bone resorption[12]. Specific clinical utility of bone biomarkers has, however, yet to be established[1].

Medical Treatment

The goals of any conservative treatment for patients at risk of or diagnosed with decreased BMD are to achieve and maintain maximum PBM, as well as to attenuate or reverse pathologic, age-related, and postmenopausal loss of BMD. To achieve these goals, medical management can consist of nutritional and pharmacological interventions. Diagnosis and pharmacological management are the responsibility of the physician. If a secondary cause of osteoporosis can be identified, specific medical treatment should be aimed at correcting the underlying disorder[1]. A dietitian can assist the physician by providing nutritional education and counseling. The goal of this section is not to provide the therapist with complete and exhaustive information needed to make a well-educated choice as to which nutritional or pharmacological intervention is indicated for a patient; this is clearly outside our scope of practice. Rather, the goal here is to provide the therapist with general information to improve patient education regarding nutritional interventions and medications prescribed most commonly by the physician.

Nutrition

In the first article, I discussed the negative effects that diets high in phosphates, oxalates, alkalis, fatty acids, and dietary fibers may have on intestinal calcium absorption. I also discussed how excessive dietary intake of protein, refined sugar, caffeine, alcohol, and sodium increase urinary excretion of calcium. A consult with a dietitian may help the patient make the necessary dietary modifications to improve calcium homeostasis.

Nutritional supplementation with calcium and vitamin D is sometimes considered one of the three categories of pharmacological intervention in the conservative treatment of osteoporosis[11]. In the first article, I discussed the recommended daily allowance (RDA) for calcium and vitamin D, and I discussed the negative effect of a calcium-deficient diet during adolescence on attaining maximum PBM and the role of calcium in preventing secondary hyperparathyroidism in the elderly. A physiologic intake of calcium and vitamin D should be considered the baseline of care on which further therapies can be implemented to decrease loss of bone mass[11].

Medication

Not including nutritional supplementation, the medications used in osteoporosis fall in two categories: anti-resorptive agents and bone-stimulating agents[11]. Estrogen, biphosphonates, selective estrogen receptor

modulators (SERMs), and calcitonin are anti-resorptive drugs[11,13]. Use of estrogen supplementation may convert a bone loss in the spine to a 1 to 2% bone gain per year; benefits in the hip are present but will be less pronounced[11]. Because bone loss is most rapid during the first five years of menopause, benefits of estrogen therapy are greater if it is started before a substantial amount of bone loss has occurred[1]. Estrogen has been associated with a ten-fold increase in the rate of uterine cancer; co-administration of progestational agents has lowered this risk to below that of the general population[11]. Estrogen has also been associated with an increased risk for breast cancer: research has shown that 11 women of 100 in the general population will have breast cancer versus 14 in 100 using estrogen. Overall mortality in women using estrogen is reduced due to improved cardiolipid profiles and a lower incidence of cardiac disease[11]. Other benefits of estrogen include retention of teeth, improved urogenital function, and decreased psychological symptoms associated with estrogen deficiency; it may also positively affect cognitive functioning[11,13].

Biphosphonates become incorporated within the hydroxy-apatite crystals and thus prevent osteoclastic resorption. Second-generation biphosphonates have a greater anti-resorptive function with minimal anti-formation properties[11]. Fossamax is probably the most familiar second-generation biphosphonate for physical therapists[13]. Average patients, especially in the early stages of osteoporosis, may experience a 5-7% increase in spinal bone mass and a 2-3% increase in hip bone mass in two years of therapy. Esophagitis is the main complication of biphosphonate therapy[11].

SERMs are drugs that exhibit specific actions at the estrogen-receptor sites spread throughout the body: they have the same positive effects as estrogen on bone and cardiovascular tissue but lack the negative effect on breast and uterine tissue[13]. I discussed the chemotherapeutic agent tamoxifen in the first article; other SERMs are raloxifene, droloxifene, and idoxifene. The latter two were in Phase 1 or 2 FDA trials in 1998[13].

Calcitonin is effective in increasing bone mass in the vertebral body in those patients with high-turnover osteoporosis; it is less effective in the appendicular skeleton[11]. In addition to its effect on BMD, calcitonin has an analgesic effect on pain associated with acute vertebral fractures; the mechanism behind this pain reduction is unknown[11,13]. Calcitonin is most commonly used as a nasal spray; the major disadvantage of this spray is nasal irritation in a small percentage of patients[11]. The calcitonin used is often salmon calcitonin. Salmon calcitonin is ten times more potent than human calcitonin. This is probably the result of increased circulating half-life, increased affinity for calcitonin receptors, and/or an increased duration of binding to these receptors[14].

The drugs discussed above primarily function by decreasing osteoclastic activity[11]. Sodium fluoride stimulates osteoblast precursors resulting in new bone formation on existing trabeculae. Treatment of postmenopausal osteoporosis with sodium fluoride has been hypothesized to disturb cortical bone, thus weakening existing bone[13]. High doses of fluorides have been shown to increase vertebral body bone mass, but they have also been associated with loss of bone mass in the hip and increased incidence of hip fractures. Low doses of sodium fluoride have lead to increased BMD and a 50% decrease in hip and vertebral fractures. Fluoride stimulates osteoblastic function and needs to be given with high levels of calcium to prevent stealing from the skeleton[13].

Physical Therapy Treatment

The goals of physical therapy treatment are virtually the same as those of conservative medical treatment: achieve and maintain maximal PBM and attenuate or reverse age-related and postmenopausal loss of BMD[15]. These goals are essentially similar to the treatment goals mentioned in the preferred practice pattern for skeletal demineralization in the *Guide for Physical Therapist Practice*[16]: helping the patient at risk for low BMD maintain a density above the fracture threshold and helping the patient diagnosed with low density achieve BMD above this threshold. Exercise is the physical therapy intervention of choice to achieve these goals. Both cross-sectional studies[17] and most longitudinal research clearly show a positive effect of exercise on BMD.

Of course, physical therapy has many other treatment goals for its interventions with patients diagnosed with decreased BMD. Friedlander et al[15] mentioned reduction of risk for falling as a goal, especially in the elderly. Some fall risk factors are decreased strength in knee flexors and extensors, ankle dorsiflexors, hip abductors, and decreased single-leg stance time[18]. Nelson et al[19] found that strength-training significantly increased strength and dynamic balance in postmenopausal women. Pain modulation is often a treatment goal. Bennell et al[7] suggested the use of therapeutic modalities and gentle manual mobilization. Ernst[20] reported the positive effects of exercise interventions on pain and other complaints related to osteoporosis. Educational goals aimed at lifestyle changes in the young , middle-aged, and elderly population and fall risk reduction in the older population are also indicated[7,18]. However, these goals are outside the scope of this article. I will only discuss the role of exercise in affecting PBM and BMD.

Effect of Age

In the first article, I described how bone mass peaks somewhere in the third decade, yet nearly maximal BMD is already achieved before the end of the second decade of life. For this reason, Gleeson[5] viewed osteoporosis as a pediatric disease rather than as a disease of middle to

old age. Achieving and maintaining a maximal PBM is a goal of conservative treatment for low BMD. This goal combined with the time at which PBM occurs defines our target population as premenarcheal and young premenopausal women. After attaining PBM, bone mass shows a physiologic decline, accelerated in women by menopause. The second goal for conservative treatment of patients with or at risk for low BMD is attenuating and reversing age-related and postmenopausal bone density loss. This defines our population as older premenopausal and postmenopausal women. Older men are also at risk, but the effect of exercise on BMD in men will be discussed in a later section. Two questions need to be answered. Does exercise affect BMD in premenarcheal, premenopausal, and postmenopausal women? Is there a difference in the effect of exercise in these different age groups?

Morris et al[21] researched the effect on BMD during a 10-month strength-training program in 71 premenarcheal girls, aged 9-10 years old. Thirty-eight girls were nonrandomly assigned to the exercise group; 33 girls acted as controls. The program consisted of high-impact aerobic workouts three times a week for 30 minutes. It also included a 10-week, 20-station weight-bearing strength-training circuit addressing all body parts. The exercise group showed a significantly greater increase than the control group in BMD measured by DEXA of the total body, lumbar spine, proximal femur, and femoral neck. According to the authors, this study provided direct evidence that exercise enhanced bone accrual in the premenarcheal skeleton.

Lohman et al[22] randomly assigned 59 women to an exercise group and 47 women to a control group. All women were white, eumenorrheic, premenopausal, aged 28-39, and not on medications known to affect bone metabolism. The exercise program consisted of 12 weight-lifting exercises at three sets of 8-12 repetitions at a load of 70-80% of the one repetition maximum (1RM). RM loads relate the load lifted to the number of repetitions: an *n* RM load is a load a person can lift with correct form for *n* repetitions, but not *n+1* repetitions[23]. Sessions lasted an hour, three times per week for 18 months. All women took a daily 500 mg calcium supplement. BMD established by DEXA increased significantly ($P<0.05$) for the exercise group at the lumbar spine at 5 months (2.8%), 12 months (2.3%), and at 18 months (1.9%) as compared with the control group. Femoral trochanter BMD increased significantly ($P<0.05$) in the exercise versus the control group at 12 months (1.8%) and 18 months (2.0%). Despite an attrition in the exercise group of over 50% at 18 months, the authors concluded that the study results supported the hypothesis that strength-training redistributed bone mineral rather than causing an overall increase in bone mineral content.

Friedlander et al[15] randomized 127 women with an average age of 30 into an exercise and a control group. The exercise program consisted of three one-hour classes a week in a combination of moderate-load progressive resistance exercises and high-impact aerobic exercise at 70-85% of the maximal heart rate; the control group used a stretching routine. The study lasted two years. After one year, there were no significant between-group differences in BMD. After two years, DEXA measurements showed a significant ($P<0.05$) gain in BMD at the lumbar spine and femoral trochanter in exercise versus control group; measurement by SPA showed a similar significant increase in the calcaneus. Spinal trabecular bone density measured by QCT decreased significantly ($P<0.05$) from baseline in the control group, but not in the exercise group, causing another significant between-group difference at two years. Attrition at two years was 50%: only 63 subjects completed the study. The authors hypothesized that the trend for those with initially lower BMD to drop from the study and decreased compliance during the second year of the study might have resulted in an underestimation of beneficial effects of exercise on BMD. They concluded that a program of moderate-intensity weight-training and aerobics could significantly increase BMD in the spine, hip, and calcaneus of young women.

Heinonen et al[24] randomly assigned 49 premenopausal women between 35 and 45 years old to an exercise group and 49 to a control group. The exercise group performed 20 minutes of progressive high-impact exercise, 15 minutes of callisthenics, and a 15-minute warm-up and cool-down three times a week for 18 months. High-impact exercises imparted forces between 2.1 and 5.6 times bodyweight. The post-test revealed significant increases in BMD in the lumbar spine, femoral neck, distal femur, patella, proximal tibia, and calcaneus in the exercise group. The authors concluded that high-impact exercise might prevent osteoporosis and osteoporosis-related fractures later in life.

Bassey et al[25] compared the effects of five daily sets of ten vertical two-leg jumps on BMD in both pre- and postmenopausal women. The premenopausal study lasted six months: 25 women were randomized into the exercise group, 30 women served as controls. A jump height of approximately 9 cm generated ground reaction forces around three times body weight. The exercise group showed significant increase ($P<0.05$) compared to baseline in BMD of femoral neck, trochanter, and lumbar spine and significant between-group increase in BMD at the trochanter. The postmenopausal study initially lasted 12 months: 70 women were randomly assigned to the exercise group, 54 to the control group. A jump height of approximately 8.5 cm generated ground reaction forces of almost four times body weight. Despite higher compliance, higher impact forces, and a longer training duration, there were no significant differences from baseline nor between groups in the postmenopausal women. Initial BMD or use of HRT did not change the outcome. Twenty-four estrogen-deplete women continued the program for an additional six months, but again no significant differences were noted.

The authors stated that the jumping regimen described was associated with significant increases in femoral BMD in pre-, but not postmenopausal women. They, however, warned about dismissing the potentially positive effects of other exercise interventions in this age group.

In a meta-analysis, Kelley[26] pooled 11 randomized trials of aerobic and strength training programs with 370 exercising and 349 control subjects, all of them postmenopausal women. Duration of the exercise programs varied from 7-39 months, frequency varying between 2 and 7 times per week. Kelley was unable to establish percentages of VO_2max for the aerobic programs but found that parameters for the strength training interventions varied between 1-3 sets, 1-10 exercises, 7-14 repetitions, and 30-84% of a 1 RM load. BMD increased on average 0.27% in the exercise group. When both the groups that did not measure BMD, specific to the site loaded by the exercises, and the groups receiving calcium or estrogen were deleted, this effect increased to a 0.76% increase in BMD. Both aerobic and strength training increased regional BMD. Kelley could not establish a correlation between program parameters and regional BMD changes, but he noted that exercise might slow the rate of bone loss in postmenopausal women.

Martin and Notelovitz[27] studied the effect of 30 or 45 minutes of three-times-per-week treadmill walking at 70-85% of the maximal heart rate over a period of 12 months in naturally postmenopausal women. Between-group differences in lumbar and forearm BMD were not significant at 12 months. However, when comparing the subgroups of women who had entered menopause recently, i.e. within the last six years, lumbar BMD decreased significantly ($P<0.05$) in the control versus the exercise group. The authors concluded that although the exercise program did not significantly increase BMD in postmenopausal women, training did attenuate lumbar BMD loss in recently postmenopausal women.

Effect of Exercise in Men

As I discussed in the first article, osteoporosis is not limited to women. Although at a much lower risk for developing osteoporosis than women, the loss of BMD associated with type-II primary or due to secondary osteoporosis does occur in men. The treatment goals of achieving and maintaining maximal PBM and attenuating and reversing loss of BMD also apply. So how does BMD in men respond to exercise?

Blumenthal et al[28] studied the effects of prolonged aerobic exercise at 70% of the maximum heart rate in men over the age of 60. Though poorly set up methodologically, their study showed a 19% increase in bone density measured at the distal radius in men who continued in their aerobic exercise program.

Kelley et al[29] did a meta-analysis of eight randomized and non-randomized studies on the effect of exercise on BMD in men. The exercise programs were very heterogenous in type, frequency, and intensity of exercise. The populations studied were also very non-uniform varying from older sedentary males to young athletic men and heart-transplant patients. The authors found significant between-group changes of 2.6% (exercise minus control) when the sites assessed for BMD were specific to the sites loaded, but not when the sites assessed were not the sites loaded with the exercise intervention. Within-group analyses showed statistically significant increases only in the subgroup of older men but not for younger men. The authors concluded that their meta-analysis showed that site-specific exercise might improve and maintain BMD in the femur, lumbar spine, and calcaneus of older men; they related the lack of an effect in younger men to possibly pre-existing optimal levels of BMD.

Specificity

The training principle of specificity has been shown to affect the response of physiologic systems other than bone: in the case of exercise and osteoporosis, it dictates that the major impact of an activity should occur at the site where we wish to increase BMD[30].

Blumenthal et al[28] found an increase in bone density in the radius as a result of lower-extremity aerobic exercise, but as stated earlier, the methodology used in this study was not optimal. The fact that in the meta-analyses by Kelley[26] and Kelley et al[29], the effect of exercise on BMD increased when studies that did not measure density at the site being loaded were deleted indicates that the effect of exercise on BMD is site-specific. Heinonen et al[24] also found no significant effect on BMD of the non-weight bearing radius in their study of the effect of lower extremity high-impact weight bearing exercises in premenopausal women.

Type of Exercise

Exercise programs have traditionally been characterized as either endurance- or strength-training programs. Which type of exercise is effective in increasing BMD? Is one of these types of exercise superior to the other or are they equally effective?

Kelley[31] performed a meta-analysis of ten studies researching the effect of aerobic exercise on lumbar spine BMD in 330 postmenopausal women, of whom 192 exercised while 138 served as controls. Exercises consisted of walking, stationary cycling, water exercises, high- and low-impact aerobics, stair climbing, and jogging. Study length varied between 28-80 weeks, frequency between one and four times a week, duration between 15-60 minutes, and intensity between less than 60% to 90% of VO_2max. Vertebral BMD increased in the experimental group with 0.32+/-2.46% versus a 2.51+/-2.69% loss of BMD in the control group. The significant difference between the exercise

and control groups was primarily due to a loss of BMD in the control group. Kelley found no significant correlation between program parameters and vertebral BMD. He concluded that the exact dose-response relationship of aerobic exercise could not be determined from his analysis.

Dalsky et al[32] non-randomly assigned 35 postmenopausal sedentary women to short-term (9 months) and long-term (22 months) weight-bearing aerobic-training programs. The experimental groups exercised three times a week for 50-60 minutes at 70-90% VO_2max. Lumbar BMD increased 5.2% above baseline in the exercise group after nine months of training versus a decrease of 1.4+/-0.8% in the short-term controls. Eleven women exercised for 22 months; their BMD increased 6.1% above baseline versus a 1.1+/-1.1% decrease in the long-term control group. Fifteen women stopped exercising at 9 months; after 13 months of decreased activity, their bone mass returned to 1.1% above the baseline measurement. The study did not control for the use of hormone replacement therapy.

Chow et al[33] randomly assigned 48 women aged 50-62 to a control group, an aerobic exercise group, and a group combining strength and aerobic exercises. None of the women used drugs affecting bone metabolism. Aerobic training consisted of 30 minutes of weight-bearing exercise at 80% of the maximum heart rate; strength training added to the other experimental group consisted of 10-15 minutes of one set of exercises with cuff weights at a 10 RM load. The women exercised three times a week for a year. Both experimental groups had significantly higher bone mineral values of the femur and trunk than the control group at post-test, but no difference between experimental groups was found. The authors concluded that exercise could prevent postmenopausal bone loss, but that adding strength training did not increase this effect.

Hartard et al[34] non-randomly assigned 15 postmenopausal women to a control group and 15 to an exercise group. The experimental group performed one to two sets of progressive resistance exercises for shoulders, hips, and trunk at an 8-12 RM load twice a week. This intensity equals a load of 70-80% of the 1 RM load[23]. The study lasted for 6 months or at least 40 sessions. Lumbar BMD did not significantly change in either group, but femoral neck BMD remained unchanged in the exercise group and decreased significantly ($P<0.05$) in the control group.

Nelson et al[19] randomly assigned 20 postmenopausal women to an exercise group and 19 women to a control group. None of the women had used hormone replacement therapy for at least 12 months. The experimental group performed five exercises for 52 weeks twice a week using three sets at 80% of a 1 RM load (8 RM). Femoral neck BMD increased by 0.9+/- 4.5% in the experimental group; lumbar BMD increased by 1.0+/-3.6%. These values decreased with 2.5+/-3.8% and 1.8+/-3.5%, respectively, in the control group.

Intensity of Exercise

The intensity of exercise is determined by resistance and speed of movement[23]. In research studies, intensity is commonly expressed as an *n* RM load, as a percentage of an *n* RM load, as a percentage of body weight, as a percentage of VO_2max; or it can be related to a heart rate measure. In the preceding sections, I have discussed the parameters of exercise programs that have shown to be effective in increasing BMD in a variety of populations. In the interest of efficiency of our exercise intervention, however, information regarding optimal intensity will be helpful.

Grove and Londeree[35] randomly assigned 15 postmenopausal women to a control group, a low-impact exercise group, or a high-impact exercise group. Both experimental groups exercised 20 minutes three times a week with 15-minute warm-up and cool-down periods over one year. The low-impact group only used exercises that imparted less than 1.5 times body weight; the high-impact group used exercises with peak forces exceeding two times body weight. The exercise routines appear to have been aerobic, but the article is not clear on this issue. The control group showed a linear, significant ($P<0.05$) decrease in lumbar BMD measured with DPA over the one-year period. Both exercise groups maintained BMD; the high-impact group actually increased BMD from 1.17+/-0.10 to 1.19+/-0.10 g/cm^2. The difference between exercise groups was not significant. The authors concluded that 20 minutes of low-impact exercises three times per week appears effective at maintaining BMD. However, the high-impact group consumed significantly more caffeine than both other groups: the diuretic effect may have increased calcium excretion resulting in a more negative calcium status. The authors also did not control for hormone replacement therapy. Statistical power may have been too low with only five subjects per group to pick up any differences between the exercise groups.

Kerr et al[36] randomized 56 postmenopausal women not taking medications known to affect bone metabolism into two exercise groups. Both groups exercised one upper and one lower limb on alternate days. One group used three sets of 8 RM; the other group used three sets of 20 RM. The opposite limb was used as a control. This progressive resistance regimen lasted one year. Three subjects from the low-rep group and seven subjects from the high-rep group dropped from the study. At the end of the study, there were no differences in strength gain between the two groups. However, BMD measured by DEXA increased significantly ($P<0.01$-0.05) in the exercised versus control limb of the low-rep group at the trochanteric hip site, the intertrochanteric hip site, at Ward's triangle, and at the ultradistal radius site. The high-rep group increased BMD significantly ($P<0.01$) only at the radius mid-site. Between-group differences were significant with increased BMD at the ultradistal forearm and intertro-

chanteric site in the low-rep as compared to the high-rep group. The authors concluded that postmenopausal bone density at clinically important sites might be significantly increased by a low-rep high-load regimen but not by a high-rep low-load training program.

To design maximally effective and efficient exercise protocols we need to understand the effect of exercise-related mechanical stimuli on bone. Strain rate, strain magnitude, and strain distribution can potentially have differential effects on bone remodeling[37]. Strain rate would appear to be related to the speed of force development; strain magnitude, to the load used. Changes in load direction, characteristic of many high impact sports such as soccer and badminton, may alter strain distribution in bone[37]. Judex and Zernicke[37] studied the strain parameters mentioned above in the mid-diaphyseal tarsometatarsus of growing roosters. They found that drop jumps created large increases in peak strain rates (+740%), moderate increases in peak strain magnitudes (+30%), and unaltered strain distributions when compared to baseline walking. Two hundred drop jumps daily for three weeks increased bone formation rates at the periosteal surface by 40% and at the endocortical surface by 370% as compared to controls. Earlier running protocols, which negatively affected bone formation, involved much smaller maximal strain rates, similar maximal strain magnitudes, and a much greater number of loading cycles. The authors hypothesized that strain rate might be an important osteogenic stimulus. Consequently they suggested that brief exercise protocols that maximize strain rates while keeping the load magnitude at physiologic levels might be the most appropriate intervention to stimulate bone formation.

Frequency

As with intensity, the frequency of the programs shown to be effective in affecting BMD varies enormously. Again from the standpoint of efficiency, it would be helpful to have more definitive information regarding optimal frequency of exercise. The only research study that directly addressed frequency used an *in-vivo* four-point bending model in rat tibiae[38]. The authors compared the bone response between an alternate day, a Monday-Wednesday-Friday, and a daily loading regimen and found no differences among these three loading schedules. They concluded that when training human subjects, mineral apposition rates and periosteal bone formation rates would likely be similar in 3 to 4 day-a-week regimens as compared to daily programs.

Weight Bearing or Non-Weight Bearing Exercise

Exercise needs to be adapted to the impairments and functional limitations present, especially in the elderly. Exercise should not put people at an increased risk for falling, especially since osteoporotic fractures are frequently associated with falls. Exercising in a non- or partial-weight bearing environment such as using a stationary bicycle or working out in a pool may be a valuable alternative to full-weight bearing exercise. But does this type of exercise positively affect BMD?

In a cross-sectional study, Taaffe et al[39] compared the BMD of 13 gymnasts, 26 swimmers, and 19 non-athletic controls. The average age was 19, and all women were eumenorrheic. Both athletic groups participated in weight-training programs 2-3 times a week. These programs consisted of eight exercises of three sets of ten repetitions involving the upper and lower limbs. Gymnasts were found to have a significantly ($P=0.0001$) higher femoral neck BMD than controls; BMD values of controls exceeded those of swimmers, even after normalization for body weight and bone size. Trochanteric BMD was significantly ($P=0.0002$) higher in gymnasts than in both other groups, even after correction for body mass. Arm BMD was significantly ($P<0.01$) higher in gymnasts than in both the other groups; leg BMD was higher in gymnasts than in swimmers ($P<0.05$). Whole-body BMD corrected for body mass and bone size was also highest in gymnasts. The authors concluded that long-term non-weight bearing exercise that incorporates forceful muscular contractions such as swimming conferred no beneficial effects on BMD in young women. However, the authors hypothesized that frail individuals due to their detrained state might derive skeletal benefit from even casual swimming.

Bravo et al[40] studied 86 osteopenic postmenopausal women between 50 and 70 years old who exercised in waist-deep water for one hour three times a week for a year. The exercises consisted of 40 minutes of dynamic jumping interspersed with unspecified muscle exercises. Increased length and speed of jumping were used to progressively increase the volume of exercise. The women exercised at 30-40% of their heart rate reserve. Nineteen women dropped out. Lumbar BMD decreased significantly ($P<0.001$) and femoral neck BMD remained constant as compared to baseline measurements. Flexibility, agility, strength, endurance, and cardiovascular endurance all increased. The authors concluded that this regimen provided an insufficient osteogenic stimulus.

Bloomfield et al[41] non-randomly assigned seven postmenopausal women to an exercise group and seven others to a control group. The authors controlled for estrogen, calcium, and vitamin D use. The exercise group cycled on a stationary bicycle for eight months three times a week for 30 minutes at 60-80% of maximal heart rate. At post-test, there were no significant differences between groups in femoral neck BMD, but lumbar BMD did change significantly ($P<0.01$): the exercise group increased lumbar BMD with 3.55+/-1.43% versus a 2.44+/-0.81% decrease in the control group subjects.

Exercise Summary

Research into the effects of exercise is flawed: duration of the study is often insufficient, sample sizes are small and therefore statistical power is low, studies do not always control for the effects of nutritional supplementation or medication, loads used are insufficient, there is no control for a ceiling effect, and BMD is not always measured at the site that is mechanically loaded[17,20]. Some aspects of exercise prescription have only been studied in animal models[37,38]. However, we can make some tentative conclusions based on the research reviewed here:

- High-impact aerobic and strength training can increase BMD in premenarcheal girls[21]
- High-intensity training (70-80% of the 1 RM, 70-85% of maximal heart rate, or impact forces greater than two times body weight) can increase BMD in premenopausal women[15,22,24,25]
- High-intensity aerobic training (70-90% of maximal heart rate) may reverse[32] or attenuate lumbar BMD loss, the latter especially in recently postmenopausal women[27]
- High-intensity strength training (80% of 1 RM) may maintain or increase lumbar and femoral BMD in postmenopausal women[19,34]
- Positive effects on BMD are lost when training is terminated[32]
- Site-specific exercise can increase BMD in older men[29]
- Exercise effects on BMD are specific to the site loaded[24,26,29]
- High-load low-rep routines are more effective at increasing BMD in postmenopausal women than low-load high-rep regimens[36]
- Dynamic exercises are more effective than static exercises at affecting BMD[37]
- Exercising 3-4 times per week is as effective as daily exercise[38]
- Non-or partial-weight bearing exercise is not effective in young women, but may be effective in very frail patients[39-41]

These tentative conclusions can serve as a guideline when developing exercise interventions for patients at risk for or diagnosed with low BMD. Functional limitations of the patient may force us to choose a less than optimal intervention. Bloomfield et al[41] have shown that stationary cycling may be a valuable alternative for patients with orthopaedic limitations or limitations of gait or stability. The main precaution for any exercise program for patients diagnosed with osteoporosis is trunk flexion exercises. Flexion may cause or increase anterior compression fractures and wedging of the osteoporotic vertebral bodies and should be avoided[42].

Conclusion

Knowledge of the risk factors for osteoporosis will allow the physical therapist not only to make more appropriate referrals to other primary care providers, but it will also assist the therapist in the appropriate choice and parameters of the physical therapy interventions used. Patient education will be served by an increased knowledge of diagnostic modalities and therapeutic interventions outside the scope of practice of physical therapy. Exercise is the physical therapy intervention of choice to achieve goals related to BMD. Tentative conclusions have been presented with regards to exercise program development. The American College of Sports Medicine[30] suggested using the principles that have been shown to affect the response of other physiologic systems to exercise when setting up or evaluating a study into the effects of exercise on osteoporosis. We have discussed the *principle of specificity*. The *principle of overload* dictates that to effect a change the training stimulus must exceed normal loading. The *principle of reversibility* notes that positive effects will be lost if a training stimulus is discontinued. The *principle of initial values* states that those with the lowest initial levels have the greatest potential for improvement as a result of a training stimulus. The *principle of diminishing returns* describes the individual biological ceiling that determines the maximum extent of a training stimulus. Using these principles when developing an exercise program for the patient at risk for or diagnosed with osteoporosis will likely allow the physical therapist to set up both an effective and efficient intervention.

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