Osteoporosis and Fracture Prevention in the Long-Term Care Setting

PALTmed
POST-ACUTE AND LONG-TERM CARE MEDICAL ASSOCIATION

Original Panel Members:
David O. Staats, MD, Chair
Manju Beier, PharmD, FASCP
Lisa Cantrell, RNC
Jonathan Evans, MD, Facilitator
Fred Feinsod, MD, CMD
Joy Hamilton, RN, CCRN, MSN
* Carolyn Harris, RN
Cynthia Pastorino, MSN, GNP
Gretchen Robinson, MS, RD, FADA
Lisa Silverman, CTRS
Susan Stephen, PT
Stanley Wallach, MD
* Reg Warren, PhD

Contributors to 2003 update:
Naushira Pandya, MD, CMD-Chair
Consuelo Alvarez, MD, CMD
Manju T. Beier, Pharm D
Barbara Brigandi, MS, APRN
Charles Cefalu, MD, MS
Jacob Dimant, MD, CMD
Paul Drinka, MD, CMD
Jerry Earll, MD, CMD
Steve Evans, MD
Sandra Fitzler, RN
Lisa Goetz, PharmD
Joseph Gruber, RPh, FASCP
Hosam K. Kamel, MD, CMD
Charlene Kawchak, RN
Michelle Khoury, MD, CMD
Susan Levy, MD, CMD
Geri Mendelson, RN
Barbara Resnick, PhD, CRNP
Harriet Shapiro, RN
William Simonson, Pharm D, RPh
Archie Dan Smith, MD, CMD
David A. Smith, MD, CMD
David Thomas, MD
Tom Wachtel, MD
David Wilcox, MD, CMD
John Yates, MD

Contributors to 2009 update:
Naushira Pandya, MD, CMD, Chair
Norda A. Bellantoni, RN, CNHA, CAS, CALA
* Charles Cefalu, MD, MS
Paul Drinka, MD, CMD
Bassem M. Elsawy, MD CMD
Murthy Gokula, MD, CMD
Hosam K Kamel, MD, MPH, CMD
Meenakshi Patel, MD, CMD
Barney Spivack, MD, CMD
Karl Steinberg, MD, CMD
Laura Tubbs, RN, BSN, MS
Peter Winn, MD, CMD
Barbara J. Zarowitz, Pharm.D., FCCP, BCPS, CGP

***Steering Committee Member**

PALTmed Staff:
Jacqueline Vance, RN, C. CDONA/LTC, Director of Clinical Affairs
Nancy Robinson, MS, RN, Associate Director, Clinical Affairs

Technical Writers—Eleanor Mayfield and Jennifer Holmes

This clinical practice guideline is provided for discussion and educational purposes only and should not be used or in any way relied upon without consultation with and supervision of a qualified physician based on the case history and medical condition of a particular patient. The Post-Acute and Long-Term Care Medical Association (PALTmed), its heirs, executors, administrators, successors, and assigns hereby disclaim any and all liability for damages of whatever kind resulting from the use, negligent or otherwise, of this clinical practice guideline.

The utilization of the Post-Acute and Long-Term Care Medical's Clinical Practice Guideline does not preclude compliance with State and Federal regulation as well as facility policies and procedures. They are not substitutes for the experience and judgment of clinicians and caregivers. The Clinical Practice Guidelines are not to be considered as standards of care but are developed to enhance the clinicians' ability to practice.

The corporate supporters of this guideline provided funding without condition of product use, formulary status or purchasing commitment.

For more information about the PALTmed guidelines or to order copies of these clinical practice guide-lines, call 800/876-2632 or 410/740-9743 or visit our web site at https://paltmed.org.
For guideline updates visit www.CPGNews.org.

To cite this guideline use: Post-Acute and Long-Term Care Medical Association. Osteoporosis and Fracture Prevention in the Long-Term Care Setting Clinical Practice Guideline. Columbia, MD: PALTmed 2009

Preface

This clinical practice guideline (CPG) has been developed under a project conducted by the Post-Acute and Long-Term Care Medical Association (PALTmed) the national professional organization rep-resenting medical directors, attending physicians, and other practitioners who care for patients in the long-term care setting. This is one of a number of guidelines undertaken as part of the associ-ation's mission to improve the quality of care delivered to patients in these settings.

Original guidelines are developed by interdisciplinary workgroups, using a process that combines evidence and consensus-based approaches. Workgroups include practitioners and others involved in patient care in long-term care facilities. Beginning with a general guideline developed by an agency, association, or organization such as the Agency for Healthcare Research and Quality (AHRQ), pertinent articles and information, and a draft outline, each group works to make a concise, usable guideline that is tailored to the long-term care setting. Because scientific research in the long-term care population is limited, many recommendations are based on the expert opinion of practitioners in the field. A bibliography is provided for individuals who desire more detailed information.

Guideline revisions are completed under the direction of the Clinical Practice Guideline Steering Committee. The committee incorporates information published in peer-reviewed journals after the original guidelines appeared as well as comments and recommendations not only from experts in the field addressed by the guideline but also from "hands-on" long-term care practitioners and staff.

Purpose

PALTmed seeks to develop and revise guidelines that focus on specific concerns and common problems in the long-term care setting. Although AHRQ and other agencies, organizations, and associations have developed a number of guidelines for conditions that occur in elderly and chronically ill indi-viduals, many of these guidelines limit or omit considerations that are unique to the long-term care population.

PALTmed guidelines emphasize key care processes and are organized for ready incorporation into facility-specific policies and procedures to guide staff and practitioner practices and performance. They are meant to be used in a manner appropriate to the population and practice of a particular facility. Guideline implementation will be affected by resources available in the facility, including staffing, and will require the involvement of all those in the facility who have a role in patient care.

Audience

This guideline is intended for the members of the interdisciplinary team in long-term care facilities, including the medical director, director of nursing, practitioners, nursing staff, consultant pharmacist, and other professionals such as therapists, social workers, dietitians, and nursing assistants who care for residents of long-term care facilities.

PALTmed CPGs include many functions and tasks related to recognizing, clarifying, managing, and monitoring various conditions and situations. But the guidelines only sometimes specify who should do these tasks. For example, many disciplines including nursing assistants, licensed nurses, dieti-cians, and social workers may m[illegible]and document observations (e.g., that someone does not sleep at night, is more withdrawn, or has a change in usual eating patterns). But only some of them may

be qualified to determine the significance of those observations (for example, what is causing the sleeplessness or change in eating patterns). In contrast, physicians and nurse practitioners may not be present to make observations, but are trained to analyze the significance and causes of symptoms. Thus, each facility should ensure that tasks are done correctly and by appropriate interdisciplinary team members. It is important for observers to make and document findings effectively, but they should get appropriate support for interpreting the findings when this is not within the scope of their training or practice.

Assumptions

Guidelines in the long-term care setting should be consistent with fundamental goals of desirable long-term care practice. Operationally, this requirement means that the nursing facility care team systematically addresses (1) each individual's risk factors for a number of diseases and conditions and (2) the adverse consequences of the diseases and conditions on the patient's functioning and quality of life.

However, when nursing facility patients are at or near the end of life, care goals will shift from functional improvement or physical stability to palliation or comfort care. PALTmed guidelines address this transition and provide suggestions for appropriate modification of the patient's care plan.

Long-term care facilities care for a variety of individuals, including younger patients with chronic diseases and disabilities, short-stay patients needing postacute care, and very old and frail individuals suffering from multiple comorbidities. When a workup or treatment is suggested, it is crucial to consider if such a step is appropriate for a specific individual. A workup may not be indicated if the patient has a terminal or end-stage condition, if it would not change the management course, if the burden of the workup is greater than the potential benefit, or if the patient or his or her proxy would refuse treatment. It is important to carefully document in the patient's medical record the rea-sons for decisions not to treat or perform a workup or for choosing one treatment approach over another.

How to Use These Guidelines

Each guideline includes a narrative portion that covers definition, recognition, assessment, treatment, and monitoring of the condition being addressed. "Recognition" means identifying the presence of a risk or condition. "Assessment" means clarifying the nature and causes of a condition or situation and identifying its impact on the individual. "Treatment" means selecting and providing appropri-ate interventions for that individual. "Monitoring" means reviewing the course of a condition or sit-uation as the basis for deciding to continue, change, or stop interventions.

Each guideline also includes an algorithm that summarizes the steps involved in addressing the condition. In the algorithm, rectangles signify points where action is to be taken; diamonds indicate points where a decision must be made.

Terminology

We recognize that people who reside in long-term care facilities are "residents". However, we have used the term "patient(s)" throughout these guidelines because we are addressing individuals within the context of treating a medical condition. In addition, these guidelines apply substantially to individuals who come to long-term care facilities for short-term care. When referring to pharmaceutical products, we have avoided the use of brand names and refer to classes of drugs whenever possible.

TABLE OF CONTENTS

Definition 1

INTRODUCTION 1
General Prevalence of Osteoporosis 1
Consequences of Osteoporosis 1
Osteoporosis in the Long-Term Care Setting 3

RECOGNITION 3
TABLE 1. Risk Factors for Osteoporosis 4
TABLE 2. Medications Associated With Adverse Effects on Bone Metabolism 4
TABLE 3. Secondary Causes of Osteoporosis 5
Osteoporosis in Men 5
TABLE 4. Guidelines for Measuring Bone Mineral Density in Men 5

ASSESSMENT 6
Diagnostic Criteria 6
TABLE 5. Diagnostic Categories for Osteoporosis in Postmenopausal Women (Based on World Health Organization Criteria) 7
TABLE 6. FRAX: WHO Fracture Risk Assessment Tool 7
Secondary Causes of Osteoporosis 8
TABLE 7. Recommended Tests in Selected Patients for the Evaluation of Secondary Causes of Osteoporosis 9

TREATMENT 10
TABLE 8. When to Treat Osteoporosis 10
TABLE 9. When Not to Treat Osteoporosis 10
Exercise and Other Lifestyle Interventions 11
TABLE 10. Vitamin D and Calcium Supplementation in the Long-Term Care Setting 11
Fall Prevention and Hip Protection 12
TABLE 11. Treatment Options for Osteoporosis 12
TABLE 12. Medications for the Treatment of Osteoporosis 13-14
Bisphosphonates 15
Calcitonin 16
Raloxifene 17
Teriparatide 17
Hormone Replacement Therapy 17

MONITORING 19
TABLE 13. Sample Performance and Measurement Indicators 20

SUMMARY 21

REFERENCES 21
APPENDIX 1. Does This Woman Have Osteoporosis? 26
APPENDIX 2. Osteoporosis Testing and Treatment Pocket Card 27
APPENDIX 3. FRACTURE Index Questions and Scoring 28

Osteoporosis and Fracture Prevention
in the Long-Term Care Setting

Definition

Osteoporosis, or porous bone, is a disease characterized by low bone mass (**osteopenia**) and structural deterioration of bone tissue, leading to bone fragility and an increased susceptibility to fractures. Fractures are especially common in the hip, spine, or wrist, although any bone can be affected.[1]

INTRODUCTION

General Prevalence of Osteoporosis

Osteoporosis is a major public health threat for an estimated 44 million Americans and is associated with significant disability, mortality, and cost. One in two women and one in five men older than 65 years will sustain bone fractures caused by osteoporosis.[2] Despite the high risk, however, osteoporosis is accurately diagnosed in less than one third of patients, and only one in seven get appropriate treatment.[3,4]

Although osteoporosis is regarded primarily as a disease of older women, it also affects up to 2 million American men. An additional 8 million men are considered to be at risk.[5] Men account for one in four cases of both osteoporosis and hip fractures, and the death rate within 1 year of a hip fracture is twice as high in men as in women.[6] In addition, different patterns of bone loss are seen in men compared with women. Whereas women experience bone loss of 1% to 3% per year for 7 to 10 years beginning around the onset of menopause, men have a higher peak bone mass and tend to have a slower but steadier rate of bone loss of 1% per year with advancing age.[7]

Consequences of Osteoporosis

Osteoporosis reduces bone strength, the most serious consequence of which is fracture. Each year in the United States, an estimated 2 million osteoporosis-related fractures occur in men and women aged over 50 years.[8] By 2020, one in two Americans aged 50 years or more will be at risk for fractures from osteoporosis.[9] Expenditures for the care of patients with these fractures were estimated at $19 billion in 2005; by 2025 experts predict that costs will rise to $25 billion, a figure that is expected to rise even further.[8]

One in five of those ambulatory before a hip fracture require long-term care afterward,[1] and one out of five osteoporotic women will fracture again within the next 12 months.[10] In a study of outcomes in long-term care (LTC) patients after a second hip fracture, fewer patients maintained the ability to walk independently after a second fracture than after the first (53% vs. 91%).[11]

Fractures of the spine and hip place an older adult at increased risk of death in the first year as a result of impaired mobility and loss of independence.[12] About 24% of hip-fracture patients aged 50 years or more die in the year following the fracture.[1] In relatively healthy older women, the relative risk of dying is substantially increased after clinical vertebral and hip fractures (relative risk = 8.64 and 6.68, respectively).[13]

Not surprisingly, the risk of fracture is increased after a fall. In 2005, more than 15,000 persons aged 65 years or more died secondary to injuries from falls. Prevention of falls is therefore an even more significant priority for patients with osteoporosis. (See PALTmed's clinical practice guideline *Falls and Fall Risk*.[a])

Osteoporosis in the Long-Term Care Setting

Osteoporosis is a particular concern in the LTC setting. The aging process is associated with reduced bone formation relative to bone resorption, and the prevalence of low bone mineral density (BMD) increases with age. Aging and low intake of calcium and vitamin D (which causes secondary hyperparathyroidism), together with estrogen deficiency in postmenopausal women (and possibly also in older men), result in increased bone resorption. Lack of physical activity, certain medications or chronic illnesses, genetic predisposition, vitamin D deficiency, and other risk factors predispose older adults to low bone mass (osteopenia).

The prevalence of osteoporosis and osteopenia in LTC patients may be as high as 97%. In a Maryland study of 1475 patients residing in LTC facilities, the prevalence of osteoporosis (as assessed by composite forearm BMD) in white women ranged from 63.5% among those aged 65 to 74 years to 85.8% among those aged over 85 years. In men, prevalence ranged from 34% among those aged 65 to 74 years to 79.6% among those aged 85 years or more.[14]

In a nationwide study that used heel bone density to screen patients in skilled nursing facilities, 42.5% of those screened were determined to be at high risk of osteoporosis and related fracture.[15] This large-scale study suggests a considerable prevalence of undiagnosed osteoporosis.

Osteoporosis combined with dependency, impaired mobility, impaired or loss of usual protective body reactions, and a high fall rate renders the LTC population particularly vulnerable to fractures. About one out of every five hip fractures in the United States occurs in an LTC facility.[16] The annual rate of hip fractures among LTC facility patients is from 5% to 6%.[17] Utilization of health care, especially hospitalization, by LTC patients has been shown to increase after a fracture.[18]

Identification of LTC patients at risk for fracture is important because fracture has multiple clinical complications[19], including:

- Anxiety;
- Acute pain at fracture sites;
- Changes in self-image and loss of self-esteem;
- Chronic pain;
- Decline in function and mobility (making falls, incontinence, and worsening osteoporosis more likely);
- Decreased quality of life;

[a] Post-Acute and Long-Term Care Medical's Association. Falls and Fall Risk. Clinical Practice Guideline. Columbia, MD.

- Deep venous thrombosis and pulmonary embolism;
- Deformity;
- Delirium;
- Depression;
- Fear of falling;
- Infections (urinary tract infections, skin infections, pneumonia);
- Loss of social role;
- Neurological impingement syndromes (e.g., spinal cord damage with possible paralysis);
- Physical deconditioning caused by inactivity;
- Postural changes;
- Pressure ulcers;
- Restrictive lung disease (as a result of the lower ribs impinging on the iliac crest);
- Social isolation; and
- Worsening of existing structural problems, such as scoliosis.

Given the availability and range of effective treatments for osteoporosis, more LTC patients may benefit from identification of and evaluation for underlying osteoporosis and from pharmacologic and nonpharmacologic treatment when this condition is diagnosed. This guideline is intended to guide the practitioner in the recognition, assessment, treatment, and monitoring of patients with osteoporosis or at risk for osteoporosis in the LTC setting.

RECOGNITION

Identification of patients with osteoporosis in the LTC setting may depend largely on clinical assessment. Patients with a history of fractures of the hip, wrist, vertebrae, or long bones (humerus or femur); who report significant loss of height; or who have kyphosis are likely to have osteoporosis.

STEP 1

Does the patient have osteoporosis or evidence of its complications? Admission or transfer information, discharge summaries, patient and caregiver histories, previous X-ray reports, previous bone density study results by dual energy X-ray absorptiometry (DXA), and medication histories can help to identify patients with osteoporosis. Although the DXA scan is helpful in identifying osteoporosis and low bone mass (osteopenia), it is only a part of the assessment. The Study of Osteoporotic Fractures showed that 54% of hip fractures are seen in patients who did not meet the World Health Organization (WHO) criteria for the definition of osteoporosis.[20]

The presence of kyphosis, a history of fractures with minimal or no trauma, a loss of height associated with back pain (indicating vertebral compression), or a loss of height relative to the individual's height at age 30 strongly suggest the presence of osteoporosis. Confirm pre-existing diagnoses with current findings and document these in the patient's record. A DXA scan may be considered to assess risk for fracture, confirm the diagnosis of osteoporosis, assess the severity of the condition, or monitor the adequacy of ongoing treatment.

STEP 2

Is the patient at risk for loss of bone mass? Identification of patients who have low bone mass (osteopenia) or who are at risk for losing bone mass enables timely intervention, where clinically

appropriate, to try to prevent progression to osteoporosis.

The Study of Osteoporotic Fractures identified several factors that, in addition to low BMD, place women aged 65 years or more at risk of fracture: advanced age, lack of exercise in the past year, poor vision, recent falls, and vertebral fracture.[20] In addition, among white female residents of LTC facilities, low bone density and ability to transfer independently have also been found to be significant predictors of fracture.[21] Other risk factors include maternal history of hip fracture, previous fractures of any type after age 50 years, self-rated poor health, previous hyperparathyroidism, and the use of drugs such as long-acting benzodiazepines or anticonvulsants.[22] Some risk factors for developing osteoporosis are listed in Table 1.

TABLE 1

Risk Factors for Osteoporosis

- Female gender
- Age
- Family history of osteoporosis or fracture
- Small body frame and low body weight
- Caucasian, Asian, or Hispanic/Latino (although African Americans may also be at risk)
- History of fracture
- Low levels of sex hormones
  - Low estrogen levels in women, including menopause
  - Low levels of testosterone and estrogen in men
- Diet
  - Low calcium intake
  - Low vitamin D intake
  - Excessive intake of protein, sodium, and caffeine
- Inactive lifestyle
- Smoking
- Excess alcohol use
- Certain medications (listed in Table 2)
- Certain diseases and conditions (listed in Table 3)

Recent evidence also suggests that proton pump inhibitors, selective serotonin reuptake inhibitors, and thiazolidinediones may be associated with osteoporosis. Other medications associated with osteoporosis are listed in Table 2.

TABLE 2

Medications Associated With Adverse Effects on Bone Metabolism

- Aromatase inhibitors
- Anticonvulsants (phenytoin, phenobarbital, carbamazepine)
- Chemotherapeutic drugs (methotrexate, cyclosporine A)
- Excess thyroid hormone replacement (TSH below the normal range)
- Excess vitamin A
- Glucocorticoids
- Gonadotropin-releasing hormone agonists and antagonists
- Heparin
- Lithium

TSH, thyroid-stimulating hormone

The practitioner should consider evaluating the patient for other medical conditions that contribute to bone loss (i.e., secondary causes of osteoporosis) (Table 3). Findings that suggest such secondary causes may be noted at the time of the physical examination or upon review of general or specific laboratory tests.

TABLE 3
Secondary Causes of Osteoporosis

- Chronic liver failure
- Chronic kidney disease
- Chronic obstructive lung disease
- Cushing's syndrome
- Disuse, immobilization, or paralysis
- Hyperthyroidism or excess thyroid hormone replacement
- Hypogonadism (in both men and women)
- Malabsorption syndrome, cirrhosis, inflammatory bowel disease
- Multiple myeloma, leukemia, lymphoma
- Primary hyperparathyroidism
- Rheumatoid arthritis
- Type 1 and type 2 diabetes
- Vitamin D deficiency

Osteoporosis in Men

Men with a history of fractures or other known risk factors should be considered for osteoporosis treatment and may be offered BMD testing and treatment if indicated (Table 4). Risk factors for osteoporosis in men include alcohol, androgen deficiency or hypogonadism, chronic obstructive pulmonary disease, cigarette smoking, diabetes, use of anti-androgenic agents for prostate cancer, and weight loss.[23] Abrupt onset of hypogonadism, such as occurs after chemical or surgical orchiectomy (e.g., for the treatment of prostate cancer), causes more rapid bone loss comparable to that seen after menopause in women and is associated with osteoporosis.

TABLE 4
Guidelines for Measuring Bone Mineral Density in Men

- Age ≥70 y
- Age 50 to 69 y with clinical risk-factor profile
- Fracture after age 50 y
- Condition (e.g., rheumatoid arthritis) or medication (e.g., glucocorticoids in a daily dose ≥5 mg prednisone or equivalent for ≥3 mo) associated with low bone mass or bone loss
- Being considered for pharmacologic therapy for osteoporosis
- Being treated for osteoporosis (to monitor treatment effect)
- Not receiving therapy (in whom evidence of bone loss would lead to treatment)

Source: National Osteoporosis Foundation[28]

Risk factors for osteoporotic hip fractures in men include falls in the preceding year, history of fractures in the last 5 years, low femoral neck BMD, and quadriceps weakness.[24] Also, in the large, population-based Rancho Bernardo Study of elderly men, a low estradiol level was found to be associated with vertebral fractures.[25]

Thirty percent to 60% of all cases of osteoporosis in men result from secondary causes.[26] In a large study of alendronate for the treatment of osteoporosis in men, one third of subjects had low testosterone levels.[27] For this reason, it is important to look for the presence of potentially treatable conditions in men with risk factors for osteoporosis.

ASSESSMENT

Osteoporosis should be diagnosed on the basis of the patient's personal and family history, physical examination findings, laboratory values, and results of BMD testing, keeping in mind the probability that most women already have osteoporosis. It is currently recommended that persons at risk for osteoporosis be formally evaluated by central DXA of the spine and hip (but these recommendations are targeted to the population at large and not specifically at patients in the LTC setting). Other, more portable technologies such as heel ultrasound have been shown to play an independent and complementary role in identifying patients with osteoporosis.[29]

Although BMD testing is part of quality assessment in the management of osteoporosis, it is not necessary for older adults who have sustained a low-trauma fracture (i.e., caused by a fall from standing height or less). It may not be feasible to do BMD testing in frail elderly patients because of the inconvenience of testing (i.e., inability to lie flat, requirement for nonemergency transfers to a hospital or imaging center, cost). Thus, if BMD is not feasible, a physical exam can be used to predict osteoporosis. As shown in Appendix 1, the presence of an exaggerated cervical lordosis, a transverse abdominal crease, an increase in the wall-occiput distance, and reduction of the rib-pelvis distance may reliably predict osteoporosis.[30] Risk factors should be assessed annually.

Diagnostic Criteria

The relationship between the patient's BMD and the expected BMD for the patient's age and sex is referred to as the **Z score**. The difference between the patient's score and the expected norm is expressed in standard deviations (SDs) above and below the mean (i.e., the **T score**). The WHO criterion for osteoporosis is a BMD that is 2.5 SDs below the average for young, healthy white women (i.e., T score of –2.5 or less; Table 5). Low bone mass, or osteopenia, refers to a lesser degree of bone mass reduction (1 to 2.5 SDs below the mean for young adult white females). However, patients with a previous or current fragility fracture (e.g., of the spine or hip) may be diagnosed with osteoporosis even if their BMD is above the WHO threshold. A useful algorithm for testing and treatment of institutionalized patients with a prior fracture is shown in Appendix 2.

A new clinical prediction tool, the **FRAX tool**, has been developed by the WHO to evaluate the fracture risk of patients from various ethnic groups. It is based on individual patient models that integrate the risks associated with clinical risk factors as well as BMD at the femoral neck. The tool is available online.[32] The elements included in the FRAX tool are listed in Table 6. This tool may be utilized even if BMD is not available, but the prediction of fracture risk in that case is less precise. Another clinical fracture risk tool is provided in Appendix 3.

TABLE 5

Diagnostic Categories for Osteoporosis in Postmenopausal Women (Based on World Health Organization Criteria)

Category	Definition by Bone Mineral Density T Score
Normal	BMD <1 SD below mean (T score of –1)
Low bone mass	BMD 1–2.5 SDs below mean (T score of –1 to –2.5)
Osteoporosis	BMD >2.5 SDs below mean (T score of ≤–2.5)
Severe or established osteoporosis	BMD <2.5 SDs below mean with one or more fragility fractures (T score of ≤–2.5 with one or more fragility fractures)

SD, standard deviation

Source: Kanis et al, 1994[31]

Current definitions of low bone mass (osteopenia) and osteoporosis by T score are the same for men and women and for all racial and ethnic groups. It is important to emphasize that there is no "safe" T score. Each decrease of 1 SD in hip BMD from age-matched controls is thought to be associated with a twofold increase in vertebral fracture risk and a 2.6-fold increase in hip fracture risk.[33] In addition, the same T score is associated with a higher fracture risk in an older individual than in a younger individual (i.e., age affects fracture risk independently of bone density).[34]

STEP 3

Decide if a work-up is appropriate and likely to be clinically helpful. The extent of bone loss in patients who are determined to be appropriate candidates for evaluation can be determined by BMD measurement of the hip and spine by central DXA, which is more reliable than clinical assessment as a predictor of fracture risk.

However, central DXA testing presents certain problems in older patients. In those aged over 70 years, spine BMD may appear high because of the presence of aortic calcification, degenerative

TABLE 6

FRAX: WHO Fracture Risk Assessment Tool

Input (variables included in the FRAX tool):

- Age
- Gender
- Weight
- Height
- Previous fracture (fragility in adult life)
- Parent fractured hip
- Current smoking
- Glucocorticoids (oral dose of ≥5 mg/d for ≥3 mo)
- Rheumatoid arthritis
- Secondary osteoporosis
- Alcohol ≥3 units daily
- BMD at femoral neck (T or Z score)

Output: 10-year risk for total fracture and hip fracture

Source: World Health Organization[32]

arthritis, degenerative disc disease, or sclerosis. In this group of patients, hip BMD measurement is more reliable. Quantitative computed tomography of the spine may be used as the primary diagnostic modality in these patients, but this technique is not widely available.

Osteoporosis may be diagnosed on X-ray without DXA testing in patients with a previous fragility fracture (i.e., in the spine, hip, humerus, wrist, or ankle) or those with evidence of low bone mass (osteopenia). Testing can be helpful, however, in assessing severity and monitoring response to treatment. Any decision to obtain BMD evaluation should take into consideration the patient's wishes, his or her overall clinical condition, predisposing risk factors, the goals of treatment, expected functional mobility, prognosis, and life expectancy. Extreme old age should not, however, exclude a patient who has risk factors for osteoporosis from being offered BMD evaluation if such evaluation is available and feasible. The results of DXA testing will help clinical decision making.

Frail elderly patients may have difficulties with the positioning and motion control necessary for central DXA testing. Newer, portable technologies such as ultrasonography and peripheral DXA (pDXA) offer simpler alternatives for BMD testing in this population. For example, in a heel-screening study of 34,486 residents of skilled nursing facilities, pDXA was used to group residents into osteoporosis risk categories according to BMD T scores. In that study, 22.4% of those screened were in the low-risk group, 35.1% in the moderate-risk group, and 42.5% in the high-risk group.[15] Use of these alternative diagnostic methods may be considered in patients who may lack access to DXA and in whom the information obtained from testing would affect clinical management.

Many patients have radiological reports that indicate low bone mass (osteopenia). One study suggested that radiological osteopenia of the spine is a strong predictor of osteoporosis by BMD. In that study, only 12.7% of those with radiological osteopenia had normal BMD, whereas 49.2% had low bone mass and 38.1% had osteoporosis.[35] Hence, radiological reports of osteopenia may assist in treatment decisions in frail elderly patients who have significant risk factors for osteoporosis and falls.[36]

Because Vitamin D insufficiency is common, it may be advisable to measure 25-hydroxyvitamin D levels.

Secondary Causes of Osteoporosis

All patients in whom osteoporosis is diagnosed should be assessed for possible secondary causes (see Table 3). This is particularly important when there is an extreme variance in BMD from age-matched means. The assessment should include a thorough history and bedside evaluation to identify conditions such as chronic obstructive pulmonary disease, Cushing's syndrome, hyperthyroidism, and hypogonadism. Endocrine conditions require confirmatory laboratory testing, as would other suspected conditions such as hyperparathyroidism, malabsorption, multiple myeloma, and vitamin D deficiency. If the secondary cause of osteoporosis is treatable, therapy may be directed toward it concurrently with primary treatment for bone loss, if clinically appropriate for the patient.

Men are more likely than women to have osteoporosis that results from a secondary cause. In one study, a secondary cause of osteoporosis was identified in most men with osteoporotic fractures.[26] Table 7 lists recommended tests for the evaluation of secondary causes of osteoporosis. (Note that not all tests will be appropriate for patients residing in LTC facilities.)

TABLE 7

Recommended Tests in Selected Patients for the Evaluation of Secondary Causes of Osteoporosis

Potential Cause	Related Test
Cushing's syndrome	24-h urine for free cortisol, salivary cortisol
Hyperthyroidism or excess thyroid hormone replacement	Free T4, TSH
Secondary hyperthyroidism	PTH, calcium, phosphate
Hypogonadism (men)	Bioavailable (free) testosterone
Chronic liver disease	Liver function tests, PT/INR
Malabsorption syndrome	Stool for fat or xylose breath test
◆ Celiac disease	◆ Anti tissue trans glutaminase antibody ◆ IgA endomysial antibody ◆ IgA and IgG antigliadin antibody
Multiple myeloma	Serum and urine protein electrophoresis and immunofixation
Osteomalacia	Alkaline phosphatase, 25-hydroxyvitamin D
Primary hyperparathyroidism	Ionized calcium, intact PTH (if calcium elevated)
Renal failure (secondary hyperparathyroidism)	Serum creatinine, serum urea nitrogen, GFR , PTH

GFR, glomerular filtration rate; PT/INR, prothrombin time and international normalized ratio; PTH, parathyroid hormone; T4, thyroxine

STEP 4

Assess the patient's function and osteoporosis-related disabilities. Patients with obvious skeletal deformity should be assessed to determine functional capabilities and limitations, including pain and risk of falling. Evaluate patients with pain to determine the nature, severity, location, and cause of the pain. Pain may be muscular or joint-related rather than skeletal in origin, or it may be totally unrelated to osteoporosis. In fact, in many patients, osteoporosis may not cause pain.

Note that spontaneous fractures are not always detected by standard X-rays, especially early after onset. Spontaneous fractures may be related to osteomalacia. They may also be seen in the feet of patients with long-standing diabetes mellitus from diabetic angiopathy and neuropathy. Odontoid fractures may be seen in rheumatoid arthritis (caused by rheumatoid synovitis, age, and steroid therapy).

Also consider whether the patient has coexisting depression or is at risk for undernutrition (requires feeding assistance; has dysphagia; suffers from dental or oral problems; is on appetite-suppressing medications; or has physical barriers, such as arthritis, hemiplegia, lack of hand-eye coordination, or poor eyesight). Such barriers may also affect mobility and ability to participate in exercise programs. Poor vision is an important cause of falls in the elderly and may be correctible. Document function in appropriate parts of the patient's record such as the Minimum Data Set (MDS), practitioner's notes, and nursing notes. (See PALTmed's clinical practice guidelines *Altered Nutritional Status*[b] and *Falls and Fall Risk*.[c]

b Post-Acute and Long-Term Care Medical Association. Altered Nutritional Status. Clinical Practice Guideline. Columbia, MD.

c Post-Acute and Long-Term Care Medical Association. Falls and Fall Risk. Clinical Practice Guideline. Columbia, MD.

TREATMENT

STEP 5

Determine whether treatment of existing osteoporosis is indicated. The choice of interventions is influenced by the patient's risk factors, comorbidities, overall condition and prognosis, general care preferences, values, and advance directives. The National Osteoporosis Foundation's recommended approach to osteoporosis treatment in both men and women is presented in Table 8.

TABLE 8
When to Treat Osteoporosis

Calcium and Vitamin D

- Postmenopausal women and men aged ≥50 y
  - Calcium: at least 1200 mg/d, including supplements if necessary
  - 457 of Vitamin D: 800–1000 IU/d of vitamin D_3 for individuals at risk of insufficiency

Pharmacologic Treatment

- Postmenopausal women or men aged >50 y with
  - a prior hip or spine fracture
  - a BMD T score of ≤–2.5 at the hip or spine
  - a T score between –1.0 and –2.5 at the femoral neck, total hip, or spine if
    - 10-year probability (from FRAX) of hip fracture is ≥3%
    - 10-year probability of a major osteoporosis-related fracture is ≥20%

Source: National Osteoporosis Foundation[28]

More studies are required to evaluate drug efficacy and effectiveness specifically in the institutionalized elderly. Drug selection may be difficult because of the high incidence in LTC patients of other coexisting chronic diseases, use of multiple medications, and other risk factors affecting bone health. In addition, some patients may be unable to comply with instructions for taking certain medications or may be unable to tolerate a medication's side effects. Other patients may have end-stage conditions.

The situations in which osteoporosis treatment is not advisable in LTC patients are listed in Table 9.

TABLE 9
When Not to Treat Osteoporosis

- The patient is in a terminal condition or is receiving palliative care
- The patient is unable to tolerate pharmacologic treatment
- The patient is unwilling to accept treatment
- The patient has been treated with bisphosphonates for the past 5 years
- The patient is non-weight-bearing and requires maximal care
- There is no evidence of treatment efficacy

STEP 6

Initiate general interventions to treat osteoporosis. Consider calcium and vitamin D supplementation for all institutionalized patients who are able to tolerate it. Supplementation is a fundamental adjunct to pharmacologic therapy for both prevention and treatment of osteoporosis. Supplementation should be initiated early in an untreated patient and should be optimized in a patient who is already receiving pharmacologic treatment.

The National Health and Nutrition Examination Survey (NHANES) and the Continuing Survey of Food Intakes by Individuals showed that about 70% of women aged 51 to 70 years do not meet the criteria for the recommended intake of vitamin D.[37] A serum level of 25-hydroxyvitamin D of at least 80 nmol/L (32 ng/mL) is optimal for persons with normal hepatic and renal function.[38] Most institutionalized elders cannot meet their calcium and vitamin D requirements through diet alone. Some patients have low dietary calcium intake because of lactose intolerance. In addition, aging reduces the ability to absorb calcium.

Decreased exposure to sunlight, decreased hydroxylation, and renal insufficiency reduce vitamin D synthesis (vitamin D regulates the absorption of calcium from the intestine). Patients who require anticonvulsant medications such as carbamazepine, phenobarbital, and phenytoin may also have vitamin D insufficiency or deficiency caused by accelerated hepatic breakdown of vitamin D.

The role of vitamin D not only for bone health but for general health has been increasingly appreciated. Low vitamin D levels are associated not only with hip fracture but also with increased mortality, and vitamin D supplementation has been shown to decrease mortality rates.[39,40] Data have shown that vitamin D plus calcium, but not vitamin D alone, prevents osteoporotic fractures in older adults.[41] For these reasons, all patients in the LTC setting should receive dietary supplementation with both calcium and vitamin D regardless of selection of other osteoporosis therapy.

Suggested doses of vitamin D and calcium are listed in Table 10. LTC patients should receive a total of 800 to 1000 IU of vitamin D and up to 1500 mg of elemental calcium daily.[42-44] The use of calcitriol (1,25-dihydroxyvitamin D) may be appropriate in certain patients with end-stage renal disease but not in others because it has been linked to an increased risk of symptomatic hypercalcemia, nephrolithiasis, and deterioration of renal function.[45] Calcium supplementation is commonly given in the form of calcium carbonate or calcium citrate. Calcium citrate does not need gastric acid and may be indicated in patients on H_2 blockers or proton pump inhibitors or those with achlorhydria.[46] Some data suggest that calcium citrate is more bioavailable than calcium carbonate.[46] Constipation is a common side effect of calcium supplementation.

Exercise and Other Lifestyle Interventions

For patients with osteoporosis, exercise is most beneficial for increasing mobility, muscle mass, and strength and balance and for preventing falls. In one study, hip BMD measurements were higher in

TABLE 10
Vitamin D and Calcium Supplementation in the Long-Term Care Setting

Vitamin and Form	Dose
Vitamin D_3 (cholecalciferol)	800–1000 IU/d or 50,000 IU once monthly
Calcium citrate*	Two 950-mg tablets 3 times daily
Calcium carbonate*	500 mg 3 times daily

*Note that the recommended daily dose of *elemental calcium* is 1200 mg/d plus normal dietary intake for adults aged >51 y. The *elemental calcium* content of calcium citrate is 211 mg/g; the *elemental calcium* content of calcium carbonate is 400 mg/g.

older adults who reported participating in moderate or strenuous exercise and those who went through high-intensity strength training.[47]

Patients may be assessed by physical or occupational therapy to customize activities and exercise, including weight-bearing activities (usually walking) as tolerated. The program should be individualized and the interdisciplinary care team should develop a comprehensive plan of care to optimize bone health and functional ability. Where feasible, stretching, use of the extremities, and gradual muscle conditioning should also be part of the patient's exercise program. The goal of increasing activity should be facilitated through the use of restorative programs when appropriate.

Other lifestyle interventions that may help to prevent the onset or progression of osteoporosis include encouraging smoking cessation and reducing or eliminating alcohol and caffeine intake. Record the impact of interventions on function, pain, and mobility in appropriate sections of the patient record (e.g., progress notes, MDS, care plan).

Fall Prevention and Hip Protection

Because impact to the greater trochanter is a major cause of hip fracture, hip protectors (external padding systems with hard exteriors, usually sewn into undergarments) may be used to absorb and shunt energy away from the proximal femur. Accumulating evidence, however, casts some doubt on the effectiveness of hip protectors in reducing the incidence of hip fractures. In a Cochrane review, pooled data from 11 studies in LTC and residential facilities (N = 15,000) showed a marginally statistically significant decrease in hip fracture incidence with hip protector use.[48] In that same review, pooled data from three trials of community dwellers (N = 5,135) failed to show a decrease in hip fracture incidence, and no evidence was found of any significant effect of hip protectors on the incidence of pelvic or other fractures, mortality, or impact on falls.

STEP 7

Is pharmacologic therapy indicated for osteoporosis? Treatment options for osteoporosis fall into three categories: vitamin D and calcium supplementation, antiresorptive medications, and anabolic medications[49,50] (Table 11).

TABLE 11
Treatment Options for Osteoporosis

Calcium Balance	Antiresorptive Medications	Anabolic Medications
Calcium ◆ Calcium carbonate ◆ Calcium citrate	Calcitonin	Parathyroid hormone
Vitamin D analogues ◆ Cholecalciferol ◆ Ergocalciferol ◆ 1,25-Dihydroxyvitamin D	Raloxifene (selective estrogen receptor modulator)	
	Bisphosphonates ◆ Alendronate ◆ Etidronate ◆ Ibandronate ◆ Pamidronate ◆ Risedronate ◆ Zoledronic acid	

TABLE 12

Medications for the Treatment of Osteoporosis

Medication	Trade Name	Approved Indication	Vertebral Fracture Risk Reduction	Non-Vertebral Fracture Risk Reduction	Dosage	Concerns/Precautions in Long Term Care Setting
Bisphosphonates						
Alendronate	Fosamax	◆ Treatment and prevention of postmenopausal osteoporosis (PMO) ◆ Treatment of glucocorticoid-induced osteoporosis (GIO) ◆ Treatment of osteoporosis in men	**yes**	**yes**	Treatment: 10 mg/d; 70 mg/wk Prevention: 5 mg/d; 35 mg/wk	Precautions: ◆ Active gastritis, duodenitis, or ulcer ◆ CrCl <35 mL/min ◆ Esophageal stricture or motility dysfunction ◆ Hypocalcemia ◆ Poor pill-swallowing ability Give in the morning on an empty stomach with 6–8 oz water. Factors influencing administration: ◆ Ability to drink 6–8 oz. of water ◆ Ability to sit or stand upright for ≥30 min after ingestion ◆ Ability to take alendronate 30–60 min before meals and morning dosing of other medications ◆ Patients with severe dementia or kyphosis may be unable to sit or stand upright for 30 min after ingestion May cause esophageal or gastric inflammation or ulcers
Risedronate	Actonel	◆ Treatment and prevention of PMO ◆ Treatment of GIO ◆ Treatment of osteoporosis in men	**yes**	**yes**	5 mg/d; 35 mg/wk; 75 mg 2 d/mo; 150 mg/mo	Do not use if any of the following is present: ◆ Hypersensitivity to any product component ◆ Hypocalcemia ◆ Inability to sit or stand upright for 30 min Administration requirements as for alendronate May cause upper gastrointestinal irritation Not recommended in patients with CrCl <30 mL/min
Ibandronate	Boniva	Treatment and prevention of PMO	**yes**	**no**	2.5 mg/d; 150 mg/mo; 3 mg IV every 3 mo	Should be taken first thing in the morning Patient should not take anything else by mouth for 60 min Patient should not lie down for 60 min following ingestion Daily oral administration may cause gastrointestinal side effects Not recommended in patients with CrCl <30 mL/min

Medication	Trade Name	Approved Indication	Vertebral Fracture Risk Reduction	Non-Vertebral Fracture Risk Reduction	Dosage	Concerns/Precautions in Long Term Care Setting
Zoledronic Acid	Reclast	◆ Treatment and prevention of PMO ◆ Increase bone mass in men	**yes**	**yes**	Yearly infusion of 5 mg	Contraindicated in patients with hypocalcemia or hypersensitivity to zoledronic acid Adequate supplementation with calcium and vitamin D is recommended Contraindicated in patients with CrCl <35 mL/min*
Calcitonin (nasal spray)	Miacalcin (Fortical)	◆ Treatment of PMO in women >5 y post menopause	**yes**	**no**	One intranasal spray daily (200 IU), alternating nostrils	Improves bone pain (e.g., caused by acute fracture) May cause rhinitis Do not use if hypersensitivity to any product component is present Use with caution in patients with sinus problems
Raloxifene (selective estrogen receptor modulator)	Evista	◆ Prevention and treatment of osteoporosis	**yes**	**no**	60 mg/d	Precautions: ◆ Do not use in patients with active or past history of venous thromboembolism, or retinal vein thrombosis ◆ Discontinue 72 h before prolonged immobilization ◆ Increased risk of death caused by stroke ◆ May cause hot flashes Inadequate information available on use in LTC setting.
Teriparatide (parathyroid hormone)	Forteo	Treatment of severe osteoporosis in men and women with PMO who are at high risk for fracture and intolerant to other osteoporosis therapy	**yes**	**yes**	20 mcg/d subcutaneously into thigh or abdominal wall	Boxed warning for those at increased risk of osteosarcoma (patients with Paget's disease, increased alkaline phosphatase, open epiphyses or prior external beam or implant radiation therapy of the skeleton) Contraindications: ◆ Hyperparathyroidism or hypercalcemia ◆ Bone metastases or skeletal malignancies

CrCl, creatinine clearance

*On September 1, 2011, the U.S. Food and Drug Administration (FDA) notified healthcare professionals and patients of an update to the drug label for Reclast (zoledronic acid) regarding the risk of kidney failure. The revised label states that Reclast is contraindicated in patients with creatinine clearance less than 35 mL/min or in patients with evidence of acute renal impairment. The label also recommends that healthcare professionals screen patients prior to administering Reclast in order to identify at-risk patients.

Several pharmacologic treatment options are now available both to prevent the development of osteoporosis and to treat established osteoporosis (Table 12). Even in the very old, treatment with risedronate was shown to reduce the risk of new vertebral fractures.[51] Calcitonin has been beneficial for the treatment of vertebral fractures and raloxifene has been beneficial for the treatment and prevention of vertebral fractures. However, data on nonvertebral fractures are not available for either agent and the magnitude of the protective effect is less than that achieved with bisphosphonates. Teriparatide may be useful in selected patients. Pharmacologic interventions should be individualized, taking into consideration the clinical situation and patient or family preference.

Bisphosphonates

Bisphosphonates are widely used both to prevent and to treat osteoporosis. These agents are potent inhibitors of bone resorption and provide clinically proven fracture protection. Currently approved bisphosphonates include alendronate (daily and weekly oral dosing); risedronate (daily, weekly, and monthly oral dosing); ibandronate (daily and monthly oral dosing and intravenously every 3 months); and zoledronic acid (yearly intravenous dosing).

To ensure adequate absorption, bisphosphonates must be taken with a glass of plain water on an empty stomach after an overnight fast, with no food, drink, medications, or supplements to follow for at least one-half hour (alendronate, risedronate) or 1 hour (ibandronate). Absorption is very poor, typically less than 1% of the oral dose. Bioavailability may be seriously impaired by ingestion with liquids other than plain water, such as mineral water, coffee, or juice, or by retained gastric contents, as with insufficient fasting time or gastroparesis, or by eating or drinking too soon after taking the bisphosphonate. An upright posture is recommended after taking the medication to avoid esophageal irritation and gastroesophageal reflux.

Osteonecrosis of the jaw is a rare complication associated with the use of bisphosphonates in patients receiving chemotherapy or radiation therapy.[52,53] It is thus important to optimize dental health in patients receiving bisphosphonates.

The specific directions for the administration of bisphosphonates have made their use challenging in the LTC setting. However, the availability of efficacious dosage forms that can be given once weekly, once monthly, on two consecutive days each month, every 3 months, and yearly has made it easier to administer these medications to patients in LTC facilities. Bisphosphonate therapy should be given under appropriate supervision.

Alendronate. In the first Fracture Intervention Trial (FIT I), which studied 2,027 women aged 55 to 81 years with low BMD and at least one vertebral fracture, subjects treated with alendronate (5 mg/d for 2 years, then 10 mg/d) had a 47% decrease in the rate of new radiographic vertebral fractures, a 51% reduction in hip fractures, and a 48% reduction in wrist fractures.[54] Alendronate reduces clinical vertebral fracture risk within 1 year and nonvertebral fracture risk within 2 years after the initiation of treatment. The reduction in fracture risk persists for up to 3 years of follow-up.[55]

In the second trial (FIT II), which studied women who had low BMD but no vertebral fractures, a 50% reduction in the risk of new radiographic vertebral fractures was observed in patients treated with alendronate. For women with femoral-neck T scores of –2.5 or less, the reduction in risk of clinical fractures was 36%; the reduction in risk of hip fractures was 56%.[56] In a study of 327 women aged between 62 and 87 years who resided in LTC facilities, alendronate significantly improved bone density in both hip and spine and was well tolerated.[57]

Oral alendronate is also recommended in men if clinically appropriate. This agent was shown to increase BMD in a study of 241 men aged 31 to 87 years whose femoral neck BMD was less than 2 SDs below that of young men; one third of the men in this study also had low testosterone as a secondary cause of BMD loss.[27]

Because of alendronate's possible irritant effects on the upper gastrointestinal mucosa and its potential to cause worsening of underlying disease, the drug should be given with caution to patients who have active upper gastrointestinal problems such as dysphagia, duodenitis, esophageal disease, gastritis, or ulcers. Careful attention to dosing instructions will reduce the risk of esophageal irritation.

Risedronate. In the North American and multinational arms of the Vertebral Efficacy with Risedronate Therapy study, risedronate significantly reduced new vertebral fractures in 1 year by 65% and 61%, respectively. In the North American arm, the cumulative incidence of new vertebral and nonvertebral fractures decreased by 41% and 30%, respectively. In the multinational arm, risedronate decreased the risk of vertebral fractures by 49% but did not significantly reduce risk of nonvertebral fractures.[58,59]

In the Hip Intervention Program, which studied 5,445 women aged 70 to 79 years with osteoporosis indicated by low BMD at the hip, risedronate reduced the risk of hip fracture by 40%. In a subgroup of patients who had osteoporosis and a baseline vertebral fracture, a 60% reduction in hip fracture was observed. In a separate arm of the study involving 3,886 women aged over 80 years who had nonskeletal risk factors for osteoporosis (but who did not necessarily have osteoporosis), risedronate did not reduce fracture rates significantly.[60] The Cochrane Database of Systematic Reviews has confirmed that risedronate and etidronate are beneficial for the primary and secondary prevention of osteoporotic fractures in postmenopausal women.[61,62]

Ibandronate. Ibandronate is approved for the treatment and prevention of osteoporosis in postmenopausal women and is available for oral administration or in parenteral solution for intravenous use. Pivotal trial data have indicated that ibandronate has no effect on the incidence of nonvertebral fractures. Daily, cyclic, and monthly regimens showed significant gains in BMD with reduction in turnover by 50% to 60% and significantly less height loss.[63] There was equivalency (by BMD response) for monthly dosing. In that study, significant reduction in nonvertebral fractures was shown only in post-hoc subgroup analysis.

Zoledronic acid. Zoledronic acid is the second intravenous bisphosphonate approved for osteoporosis. In the HORIZON (Health Outcomes and Reduced Incidence with Zoledronic Acid Once Yearly) trial, a once-yearly infusion over a 3-year period reduced the risk of vertebral, hip, and other fractures.[64] Nonvertebral fractures were reduced by 25%, clinical fractures by 33%, and clinical vertebral fractures by 77%.

Calcitonin

Calcitonin, an endogenous hormone secreted by the parafollicular C cells of the thyroid, is marginally effective at increasing BMD and reducing the risk of vertebral fractures. It has also been found to improve bone pain caused by acute compression fractures. The medication is administered in a nasal spray formulation of salmon-derived calcitonin, one spray (200 IU) daily, alternating nostrils.

Adverse effects of calcitonin are rare and usually consist of nasal irritation, nausea, and rhinitis.

In the Prevent Recurrence of Osteoporotic Fractures study, a 5-year, double-blind, placebo-controlled study of the effect of nasal calcitonin on vertebral fracture rates in postmenopausal women with osteoporosis, calcitonin resulted in a 33% to 36% reduction in the risk of new vertebral fractures and a nonsignificant reduction in the risk of nonvertebral fractures.[65]

A study comparing alendronate and nasal calcitonin concluded that after 12 months of therapy, alendronate produced significantly greater increases in BMD of the hip (2.78% vs. 0.58% in the femoral neck) and lumbar spine (5.16% vs. 1.18%) and greater decreases in bone turnover than nasal calcitonin.[66] Calcitonin is recommended for long-term use only if contraindications to all other drugs are present.

Raloxifene

The selective estrogen receptor modulator raloxifene is indicated for osteoporosis prevention and treatment. In the Multiple Outcomes of Raloxifene Evaluation (MORE) trial, women with low BMD and no prior vertebral fractures who took raloxifene had a 55% reduction in incidence of new vertebral fractures. In women with previous vertebral fractures, raloxifene at a dose of 60 mg/d resulted in a 30% reduction in the incidence of new fractures.[67] Raloxifene has not been shown to reduce nonvertebral fractures.

Raloxifene does not adversely affect breast or uterine tissue; it does not cause breast tenderness or vaginal bleeding. However, raloxifene causes more frequent hot flashes and, like hormone replacement therapy (HRT), increases the risk of venous thromboembolism and cataracts. Raloxifene can be used for the prevention of breast cancer in osteoporotic women as well as in postmenopausal women at high risk for breast cancer.[68]

Teriparatide

Teriparatide, a form of parathyroid hormone, was approved in 2002 for the treatment of osteoporosis in both men and women. This drug stimulates new bone formation in excess of resorption and significantly increases BMD. Studies have shown that after 9 months of therapy, teriparatide increases lumbar spine BMD by as much as 9.7% in women[69] and 5.9% in men.[70] In women, the drug also reduces the risk of new vertebral fractures by 65% and that of nonvertebral fractures by 53%. It does not affect the risk of hip fracture.

Teriparatide is given by daily subcutaneous injection. Its use is currently recommended for up to 2 years. A bisphosphonate is often required after stopping teriparatide therapy to preserve the gains in BMD. This agent may be suitable for selected LTC patients with severe osteoporosis (T score <–3) who are unable to take bisphosphonates. However, patients with primary or secondary hyperparathyroidism, Paget's disease, bony metastases, history of bone irradiation, or multiple kidney stones should not take teriparatide. Insufficient information is available on its use in patients with severe renal insufficiency (CrCl <30 ml/min) and those on hemodialysis. Teriparatide is more costly than other osteoporosis therapies.

Hormone Replacement Therapy

In the past, HRT was recommended for the prevention of osteoporosis in postmenopausal women. However, in light of more recent studies, HRT is no longer recommended. Data suggest that continuous combined hormone therapy is associated with an increase in cardiovascular disease, stroke, venous thromboembolism, and breast cancer. These risks outweigh any benefits of reduced fracture

risk.[71] Safer and highly effective therapeutic choices for the treatment and prevention of osteoporosis exist (e.g., bisphosphonates, calcium, and vitamin D), and these should be considered for first-line use in the LTC setting.

STEP 8

Treat symptoms related to skeletal deformity. Chronic pain caused by osteoporosis or its complications may be treated by local modalities, analgesics, and calcitonin.[72]

Acute back pain caused by an osteoporotic vertebral compression fracture is best treated by short-term bed rest, analgesics, and calcitonin, followed by a program of mobilization and exercise to retain or improve muscle strength and mobility. Narcotic analgesics will usually be necessary along with cathartics to minimize constipation. A cane, wheelchair, or wheeled walker may initially be required to reduce weight bearing. The effects of prolonged immobility on this vulnerable population should always be a consideration.

Application of ice packs early after a fracture, followed by mild superficial heat and gentle range-of-motion and relaxation exercises, helps to reduce pain and contraction in adjacent muscle groups. Corsets or other immobilization devices may help to minimize pain but should be removed once the pain subsides.

Extensive use of back braces may increase discomfort and contribute to disuse of back extensor muscles. A kypho-orthosis with weights fitted below the inferior angle of the scapula, used in conjunction with posture training, enables patients to better contract back extensor muscles and reduces kyphotic posture. These devices may be poorly tolerated, however, and may cause sores from trauma to frail skin.

Two interventional procedures, **vertebroplasty** and **balloon kyphoplasty**, are increasingly being used in the treatment of patients with severe, persistent pain caused by acute vertebral compression fractures or unstable vertebral fractures. Vertebroplasty involves the percutaneous injection of polymethyl methacrylate (bone cement) into the vertebral body under fluoroscopic guidance. In balloon kyphoplasty, orthopedic balloons are inserted into both sides of the vertebral body under image guidance. The balloons are inflated to restore vertebral body height and normal shape to the collapsed vertebra, then deflated and removed. The cavity in the vertebra is filled with bone cement to prevent further collapse.[73,74] Results from a recent multicenter randomized controlled study showed balloon kyphoplasty resulted in faster and greater improvements in pain relief, back function and quality of life as early as 1 month after the procedure compared to nonsurgical care of patients with acute painful vertebral compression fractures.[75] In addition, a case series comparison of patients treated with balloon kyphoplasty vs. vertebroplasty showed balloon kyphoplasty to be associated with fewer adverse events than vertebroplasty.[76] These procedures have been quite successful, with low complication rates, and may be appropriate in carefully selected patients, especially if there is local expertise and experience in performing them.

Short T1 inversion recovery (STIR) computed tomography allows differentiation of old from new compression fractures noted in a patient who is experiencing pain and with initial routine X-ray evidence of a fracture. Studies in the LTC population are not available.

STEP 9

Institute measures to improve function and try to prevent serious complications. Falls are a major cause of complications such as fractures and other injuries in patients with osteoporosis. Measures to prevent falls or reduce the frequency of falling and the severity of injuries from falling may include:

- Compensating for neurologic and musculoskeletal deficits where possible (e.g., by encouraging exercises that improve posture; increasing muscle strength, especially in the proximal leg muscles; treating symptoms of neuropathy and arthritis; and preventing contractures);
- Implementing appropriate rehabilitative or restorative services, such as muscle-strengthening and weight-bearing exercise as tolerated by the patient, and assisted ambulation;
- Correcting treatable causes of orthostatic hypotension and dizziness (e.g., by improving fluid intake; discontinuing unnecessary restriction of dietary salt; and reducing doses of antihypertensive medications, diuretics, and other medications that can cause dizziness or lightheadedness);
- Discontinuing or reducing doses of medications that affect balance or level of consciousness;
- Ensuring that floor coverings are not loose or slippery;
- Ensuring that lighting is adequate, especially at night;
- Implementing a toileting schedule to avoid unassisted trips to the bathroom (especially at night), which often result in falls;
- Installing chair and bed alarms or placing call buttons within the patient's reach;
- Installing handrails in bathrooms and hallways;
- Optimizing sensory capacity (e.g., by encouraging the use of glasses and hearing aids, addressing peripheral neuropathy, and removing impacted cerumen from ears);
- Reducing clutter in rooms and hallways to allow cognitively impaired individuals to ambulate safely;
- Using a long shoe horn or a long-reach clamp to reduce strenuous flexion of the spine;
- Using beds that are low to the floor and floor pads that provide cushioning when a patient falls out of bed;
- Using hip protectors consistently in patients at high risk of falling; and
- Administering vitamin D and calcium supplementation.

MONITORING

STEP 10

Periodically assess, monitor, and document the patient's progress. If feasible, obtain objective measurements of the patient's symptoms and overall course at least every 6 months. Such indicators include:

- Indicators of strength and mobility,
- Measures of function and dependency in activities of daily living, and
- Objective pain scales.

The National Osteoporosis Foundation currently recommends obtaining BMD by central DXA every 2 years to monitor the effect of therapy;[28] as stated above, however, this may not be appropriate for many patients in the LTC setting. Patients who are taking high-dose steroids may require more frequent assessment of BMD because of high bone turnover. In the LTC setting, additional BMD assessments should be obtained when feasible and when they will provide information essential to treatment or monitoring that cannot be readily obtained by other means. Assessment of BMD at peripheral sites should not be used to monitor the effect of osteoporosis therapy.

Serum and urine biochemical markers of bone turnover that reflect collagen breakdown are also available. Serum markers of bone formation include bone-specific alkaline phosphatase and osteocalcin. Bone resorption may be assessed by serum C-telopeptide measurements. Definitive guidelines for the use of markers of bone turnover are not available, however.

The post-acute care patient who is in the LTC facility for short-term rehabilitation following a hip or other fracture should be closely monitored for the development of perioperative complications such as deconditioning, immobility, and thromboembolism. Many such patients experience delirium, depression, eating problems, malnutrition, and pressure sores. If a patient has not had a prior BMD assessment and performance of the test is not feasible during his or her stay at the facility, consider obtaining a central DXA measurement when the patient has returned to the community.

The identification of quality indicators related to osteoporosis risks, complications, treatment, and prevention may assist facilities in monitoring not only the care of individual patients with osteoporosis but also their overall performance in the care of patients with osteoporosis. Table 13 suggests process and outcome indicators that a facility may wish to use to measure the success of interventions to manage osteoporosis.

TABLE 13

Sample Performance Measurement Indicators

Process indicators

- Is an evaluation process in place that identifies osteoporosis and risk for additional loss of bone mass?
- Has the underlying cause of osteoporosis been identified if clinically indicated?
- Has a diagnostic work-up been done if indicated?
- Are patients receiving treatment being monitored for side effects of medications?
- Are patients with osteoporosis assessed for pain?
- Are patients with osteoporosis who have pain treated for pain?
- Are patients with osteoporosis assessed for risk of falling and do they have a care plan in place?
- Are patients at risk for osteoporosis or who have osteoporosis receiving calcium and vitamin D supplements?

Outcome indicator

- Number of fragility fractures

STEP 11

Is the patient tolerating the medication regimen prescribed to prevent or treat osteoporosis? Monitor the patient for side effects of osteoporosis treatments. Calcium supplements may cause constipation. Calcitonin may cause nasal irritation. Alendronate, ibandronate, and risedronate may cause increased dyspepsia, esophageal irritation, musculoskeletal pain, and other symptoms. Nursing staff should document the presence of key symptoms and the practitioner should adjust treatment accordingly.

If the patient cannot tolerate the prescribed regimen, the practitioner should consider an alternative approach that may control symptoms and prevent disease progression and is acceptable to the patient.

SUMMARY

Osteoporosis is common among patients in LTC facilities. Although thought of as primarily a disease of older women, osteoporosis affects both men and women. Osteoporosis combined with dependency, impaired mobility, and a high fall rate renders the LTC population particularly vulnerable to fractures. About one in every five hip fractures in the United States occurs in an LTC facility. Effective treatments are available, but more studies are required to assess their efficacy in frail elderly patients residing in LTC facilities.

Measures to prevent osteoporosis and osteoporosis-related fractures should be implemented in LTC facilities to the extent possible (as determined by assessment and evaluation of individual residents by use of a combination of physical assessment and diagnostic testing, if feasible). A basic prevention program should focus on optimizing bone mass, ensuring adequate vitamin D and calcium supplementation, and preventing falls. The choice of interventions to treat osteoporosis will be influenced by the patient's risk factors, overall condition and prognosis, preferences, values, advance directives, and contraindications to specific therapies.

REFERENCES

1. National Osteoporosis Foundation. Fast Facts on Osteoporosis. Available at: http://www.nof.org/osteoporosis/diseasefacts.htm. Accessed 02/06/09.
2. American Academy of Orthopaedic Surgeons. Your Orthopaedic Connection: Osteoporosis, July 2007 Update. Available at: http://orthoinfo.aaos.org/topic.cfm?topic=A00232. Accessed 02/06/09.
3. Hodgson SF, Watts NB, Bilezikian JP, et al. American Association of Clinical Endocrinologists medical guidelines for clinical practice for the prevention and treatment of postmenopausal osteoporosis: 2001 edition, with selected updates for 2003. Endocr Pract 2003; 9(6): 544-564.
4. McKercher HG, Crilly RG, Kloseck M. Osteoporosis management in long-term care. Survey of Ontario physicians. Can Fam Physician 2000; 46: 2228-2235.
5. Looker AC, Orwoll ES, Johnston CC, Jr., et al. Prevalence of low femoral bone density in older U.S. adults from NHANES III. J Bone Miner Res 1997; 12(11): 1761-1768.
6. Center JR, Nguyen TV, Schneider D, et al. Mortality after all major types of osteoporotic fracture in men and women: an observational study. Lancet 1999; 353(9156): 878-882.
7. Hannan MT, Felson DT, Anderson JJ. Bone mineral density in elderly men and women: results from the Framingham osteoporosis study. J Bone Miner Res 1992; 7(5): 547-553.
8. Burge R, Dawson-Hughes B, Solomon DH, et al. Incidence and economic burden of osteoporosis-related fractures in the United States, 2005-2025. J Bone Miner Res 2007; 22(3): 465-475.
9. U.S. Department of Health and Human Services, Public Health Service. Bone Health and Osteoporosis. A Report of the Surgeon General. Updated Available at: http://www.surgeongeneral.gov/library/bonehealth/. Accessed 02/06/09.
10. Lindsay R, Silverman SL, Cooper C, et al. Risk of new vertebral fracture in the year following a fracture. JAMA 2001; 285(3): 320-323.
11. Pearse EO, Redfern DJ, Sinha M, Edge AJ. Outcome following a second hip fracture. Injury 2003; 34(7): 518-521.
12. Johnell O, Kanis JA, Oden A, et al. Mortality after osteoporotic fractures. Osteoporos Int 2004; 15(1): 38-42.
13. Cauley JA, Thompson DE, Ensrud KC, et al. Risk of mortality following clinical fractures. Osteoporos Int 2000; 11(7): 556-561.

14. Zimmerman SI, Girman CJ, Buie VC, et al. The prevalence of osteoporosis in nursing home residents. Osteoporos Int 1999; 9(2): 151-157.
15. Gloth FM, 3rd, Simonson W. Osteoporosis is underdiagnosed in skilled nursing facilities: a large-scale heel BMD screening study. J Am Med Dir Assoc 2008; 9(3): 190-193.
16. Ekman A, Michaelsson K, Ljunghall S, Mallmin H. Almost all institutionalized women are osteoporotic, when measured by heel and finger ultrasound. J Intern Med 2001; 249(2): 173-180.
17. Kamel HK. Update on osteoporosis management in long-term care: focus on bisphosphonates. J Am Med Dir Assoc 2007; 8(7): 434-440.
18. Zimmerman S, Chandler JM, Hawkes W, et al. Effect of fracture on the health care use of nursing home residents. Arch Intern Med 2002; 162(13): 1502-1508.
19. Gold DT. The nonskeletal consequences of osteoporotic fractures. Psychologic and social outcomes. Rheum Dis Clin North Am 2001; 27(1): 255-262.
20. Wainwright SA, Marshall LM, Ensrud KE, et al. Hip fracture in women without osteoporosis. J Clin Endocrinol Metab 2005; 90(5): 2787-2793.
21. Chandler JM, Zimmerman SI, Girman CJ, et al. Low bone mineral density and risk of fracture in white female nursing home residents. JAMA 2000; 284(8): 972-977.
22. Cummings SR, Nevitt MC, Browner WS, et al. Risk factors for hip fracture in white women. Study of Osteoporotic Fractures Research Group. N Engl J Med 1995; 332(12): 767-773.
23. Hannan MT, Felson DT, Dawson-Hughes B, et al. Risk factors for longitudinal bone loss in elderly men and women: the Framingham Osteoporosis Study. J Bone Miner Res 2000; 15(4): 710-720.
24. Nguyen TV, Eisman JA, Kelly PJ, Sambrook PN. Risk factors for osteoporotic fractures in elderly men. Am J Epidemiol 1996; 144(3): 255-263.
25. Barrett-Connor E, Mueller JE, von Muhlen DG, et al. Low levels of estradiol are associated with vertebral fractures in older men, but not women: the Rancho Bernardo Study. J Clin Endocrinol Metab 2000; 85(1): 219-223.
26. Orwoll ES, Klein RF. Osteoporosis in men. Endocr Rev 1995; 16(1): 87-116.
27. Orwoll E, Ettinger M, Weiss S, et al. Alendronate for the treatment of osteoporosis in men. N Engl J Med 2000; 343(9): 604-610.
28. National Osteoporosis Foundation. Clinician's Guide to Prevention and Treatment of Osteoporosis, 2008 Update. Available at: http://www.nof.org/professionals/Clinicians_Guide.htm. Accessed 02/06/09.
29. Gonnelli S, Cepollaro C, Gennari L, et al. Quantitative ultrasound and dual-energy X-ray absorptiometry in the prediction of fragility fracture in men. Osteoporos Int 2005; 16(8): 963-968.
30. Green AD, Colon-Emeric CS, Bastian L, et al. Does this woman have osteoporosis? JAMA 2004; 292(23): 2890-2900.
31. Kanis JA, Melton LJ, 3rd, Christiansen C, et al. The diagnosis of osteoporosis. J Bone Miner Res 1994; 9(8): 1137-1141.
32. World Health Organization Collaborating Centre for Metabolic Bone Diseases. FRAX WHO Fracture Risk Assessment Tool. Available at: http://www.shef.ac.uk/FRAX/. Accessed 02/06/09.
33. Melton LJ, 3rd, Atkinson EJ, O'Fallon WM, et al. Long-term fracture prediction by bone mineral assessed at different skeletal sites. J Bone Miner Res 1993; 8(10): 1227-1233.
34. Kanis JA, Johnell O, Oden A, et al. Ten year probabilities of osteoporotic fractures according to BMD and diagnostic thresholds. Osteoporos Int 2001; 12(12): 989-995.

35. McCullagh CD, McCoy K, Crawford VL, Taggart H. How reliable is a radiological report in osteoporosis in diagnosing low bone density? Ulster Med J 2003; 72(1): 34-37.
36. Canadian Task Force on the Periodic Health Examination. Canadian guide to clinical preventive health care. 1994. Ottawa: Canada Communication Group, 620-631.
37. Moore C, Murphy MM, Keast DR, Holick MF. Vitamin D intake in the United States. J Am Diet Assoc 2004; 104(6): 980-983.
38. Heaney RP. The Vitamin D requirement in health and disease. J Steroid Biochem Mol Biol 2005; 97(1-2): 13-19.
39. Simon JA. Review: vitamin D supplementation decreases all-cause mortality in adults and older individuals. ACP J Club 2008; 148(2): 30.
40. Autier P, Gandini S. Vitamin D supplementation and total mortality: a meta-analysis of randomized controlled trials. Arch Intern Med 2007; 167(16): 1730-1737.
41. Avenell A, Gillespie WJ, Gillespie LD, O'Connell DL. Vitamin D and vitamin D analogues for preventing fractures associated with involutional and post-menopausal osteoporosis. Cochrane Database Syst Rev 2005; (3): CD000227.
42. Dawson-Hughes B. Vitamin D and calcium: recommended intake for bone health. Osteoporos Int 1998; 8 Suppl 2: S30-34.
43. NIH Consensus conference. Optimal calcium intake. NIH Consensus Development Panel on Optimal Calcium Intake. JAMA 1994; 272(24): 1942-1948.
44. Drinka PJ, Krause PF, Nest LJ, Goodman BM. Determinants of vitamin D levels in nursing home residents. J Am Med Dir Assoc 2007; 8(2): 76-79.
45. Sairanen S, Karkkainen M, Tahtela R, et al. Bone mass and markers of bone and calcium metabolism in postmenopausal women treated with 1,25-dihydroxyvitamin D (Calcitriol) for four years. Calcif Tissue Int 2000; 67(2): 122-127.
46. Heller HJ, Greer LG, Haynes SD, et al. Pharmacokinetic and pharmacodynamic comparison of two calcium supplements in postmenopausal women. J Clin Pharmacol 2000; 40(11): 1237-1244.
47. Nelson ME, Fiatarone MA, Morganti CM, et al. Effects of high-intensity strength training on multiple risk factors for osteoporotic fractures. A randomized controlled trial. JAMA 1994; 272(24): 1909-1914.
48. Parker MJ, Gillespie WJ, Gillespie LD. Hip protectors for preventing hip fractures in older people. Cochrane Database Syst Rev 2005; (3): CD001255.
49. Parikh S, Avorn J, Solomon DH. Pharmacological management of osteoporosis in nursing home populations: a systematic review. J Am Geriatr Soc 2009; 57(2): 327-334.
50. Warriner AH, Outman RC, Saag KG, et al. The management of osteoporosis among home health and long term care patients with a prior fracture. [In press.]
51. Boonen S, McClung MR, Eastell R, et al. Safety and efficacy of risedronate in reducing fracture risk in osteoporotic women aged 80 and older: implications for the use of antiresorptive agents in the old and oldest old. J Am Geriatr Soc 2004; 52(11): 1832-1839.
52. Khosla S, Burr D, Cauley J, et al. Bisphosphonate-associated osteonecrosis of the jaw: report of a task force of the American Society for Bone and Mineral Research. J Bone Miner Res 2007; 22(10): 1479-1491.
53. American Dental Association. Dental Management of Patients Receiving Oral Bisphosphonate Therapy Expert Panel Recommendations. Report of the Council on Scientific Affairs, 2008 Update. Available at: http://www.ada.org/prof/resources/topics/topics_osteonecrosis_bispho-

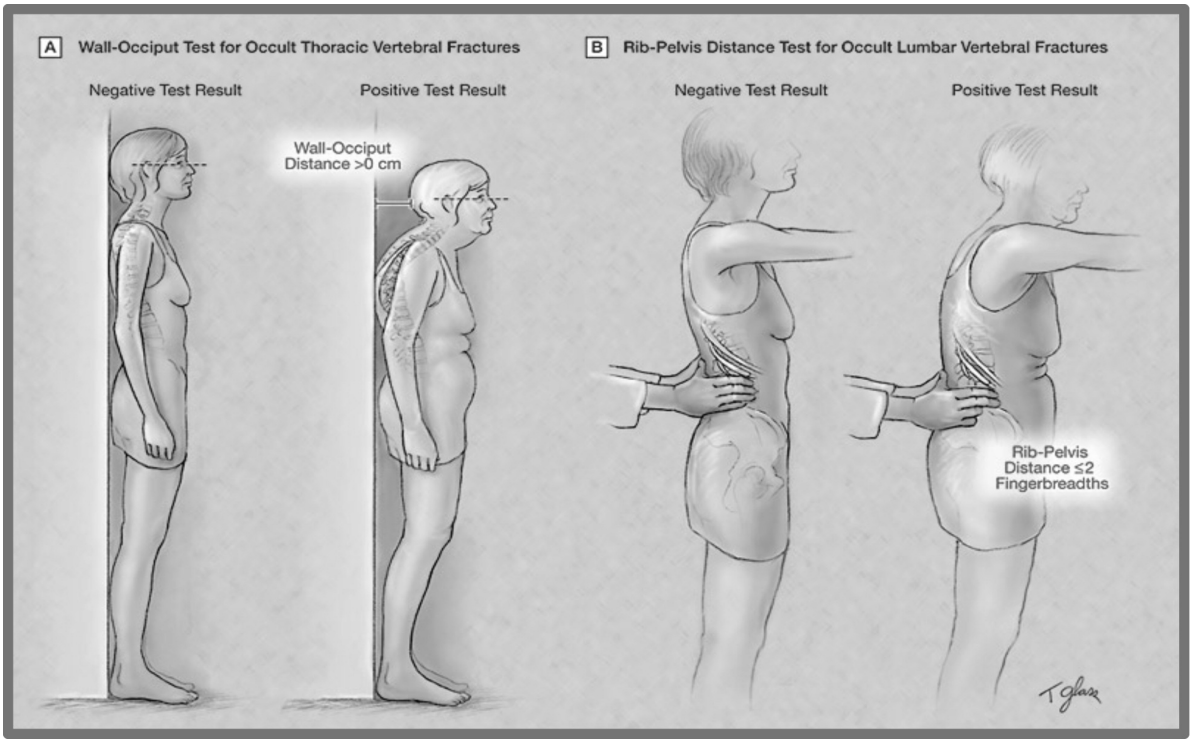
sphonate_report.pdf. Accessed 02/06/09.
54. Black DM, Cummings SR, Karpf DB, et al. Randomised trial of effect of alendronate on risk of fracture in women with existing vertebral fractures. Fracture Intervention Trial Research Group. Lancet 1996; 348(9041): 1535-1541.
55. Black DM, Thompson DE, Bauer DC, et al. Fracture risk reduction with alendronate in women with osteoporosis: the Fracture Intervention Trial. FIT Research Group. J Clin Endocrinol Metab 2000; 85(11): 4118-4124.
56. Cummings SR, Black DM, Thompson DE, et al. Effect of alendronate on risk of fracture in women with low bone density but without vertebral fractures: results from the Fracture Intervention Trial. JAMA 1998; 280(24): 2077-2082.
57. Greenspan SL, Schneider DL, McClung MR, et al. Alendronate improves bone mineral density in elderly women with osteoporosis residing in long-term care facilities. A randomized, double-blind, placebo-controlled trial. Ann Intern Med 2002; 136(10): 742-746.
58. Harris ST, Watts NB, Genant HK, et al. Effects of risedronate treatment on vertebral and nonvertebral fractures in women with postmenopausal osteoporosis: a randomized controlled trial. Vertebral Efficacy With Risedronate Therapy (VERT) Study Group. JAMA 1999; 282(14): 1344-1352.
59. Reginster J, Minne HW, Sorensen OH, et al. Randomized trial of the effects of risedronate on vertebral fractures in women with established postmenopausal osteoporosis. Vertebral Efficacy with Risedronate Therapy (VERT) Study Group. Osteoporos Int 2000; 11(1): 83-91.
60. McClung MR, Geusens P, Miller PD, et al. Effect of risedronate on the risk of hip fracture in elderly women. Hip Intervention Program Study Group. N Engl J Med 2001; 344(5): 333-340.
61. Wells G, Cranney A, Peterson J, et al. Risedronate for the primary and secondary prevention of osteoporotic fractures in postmenopausal women. Cochrane Database Syst Rev 2008; (1): CD004523.
62. Wells GA, Cranney A, Peterson J, et al. Etidronate for the primary and secondary prevention of osteoporotic fractures in postmenopausal women. Cochrane Database Syst Rev 2008; (1): CD003376.
63. Delmas PD, Recker RR, Chesnut CH, 3rd, et al. Daily and intermittent oral ibandronate normalize bone turnover and provide significant reduction in vertebral fracture risk: results from the BONE study. Osteoporos Int 2004; 15(10): 792-798.
64. Black DM, Delmas PD, Eastell R, et al. Once-yearly zoledronic acid for treatment of postmenopausal osteoporosis. N Engl J Med 2007; 356(18): 1809-1822.
65. Chesnut CH, 3rd, Silverman S, Andriano K, et al. A randomized trial of nasal spray salmon calcitonin in postmenopausal women with established osteoporosis: the prevent recurrence of osteoporotic fractures study. PROOF Study Group. Am J Med 2000; 109(4): 267-276.
66. Downs RW, Jr., Bell NH, Ettinger MP, et al. Comparison of alendronate and intranasal calcitonin for treatment of osteoporosis in postmenopausal women. J Clin Endocrinol Metab 2000; 85(5): 1783-1788.
67. Ettinger B, Black DM, Mitlak BH, et al. Reduction of vertebral fracture risk in postmenopausal women with osteoporosis treated with raloxifene: results from a 3-year randomized clinical trial. Multiple Outcomes of Raloxifene Evaluation (MORE) Investigators. JAMA 1999; 282(7): 637-645.
68. Bevers TB. The STAR trial: evidence for raloxifene as a breast cancer risk reduction agent for postmenopausal women. J Natl Compr Canc Netw 2007; 5(8): 719-724.

69. Neer RM, Arnaud CD, Zanchetta JR, et al. Effect of parathyroid hormone (1-34) on fractures and bone mineral density in postmenopausal women with osteoporosis. N Engl J Med 2001; 344(19): 1434-1441.
70. Orwoll ES, Scheele WH, Paul S, et al. The effect of teriparatide [human parathyroid hormone (1-34)] therapy on bone density in men with osteoporosis. J Bone Miner Res 2003; 18(1): 9-17.
71. Hulley S, Grady D, Bush T, et al. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. Heart and Estrogen/progestin Replacement Study (HERS) Research Group. JAMA 1998; 280(7): 605-613.
72. Gennari C, Agnusdei D, Camporeale A. Use of calcitonin in the treatment of bone pain associated with osteoporosis. Calcif Tissue Int 1991; 49 Suppl 2: S9-13.
73. Linville DA, 2nd. Vertebroplasty and kyphoplasty. South Med J 2002; 95(6): 583-587.
74. Ledlie JT, Renfro MB. Kyphoplasty treatment of vertebral fractures: 2-year outcomes show sustained benefits. Spine 2006; 31(1): 57-64.
75. Wardlaw D, Cummings SR, Van Meirhaeghe J, et al. Efficacy and safety of balloon kyphoplasty compared with non-surgical care for vertebral compression fracture (FREE): a randomised controlled trial. Lancet. 2009; 373(9668):1016-24.
76. Taylor RS, Taylor RJ, Fritzell P. Balloon kyphoplasty and vertebroplasty for vertebral compression fractures: a comparative systematic review of efficacy and safety. Spine 2006; 31(23): 2747-2755.

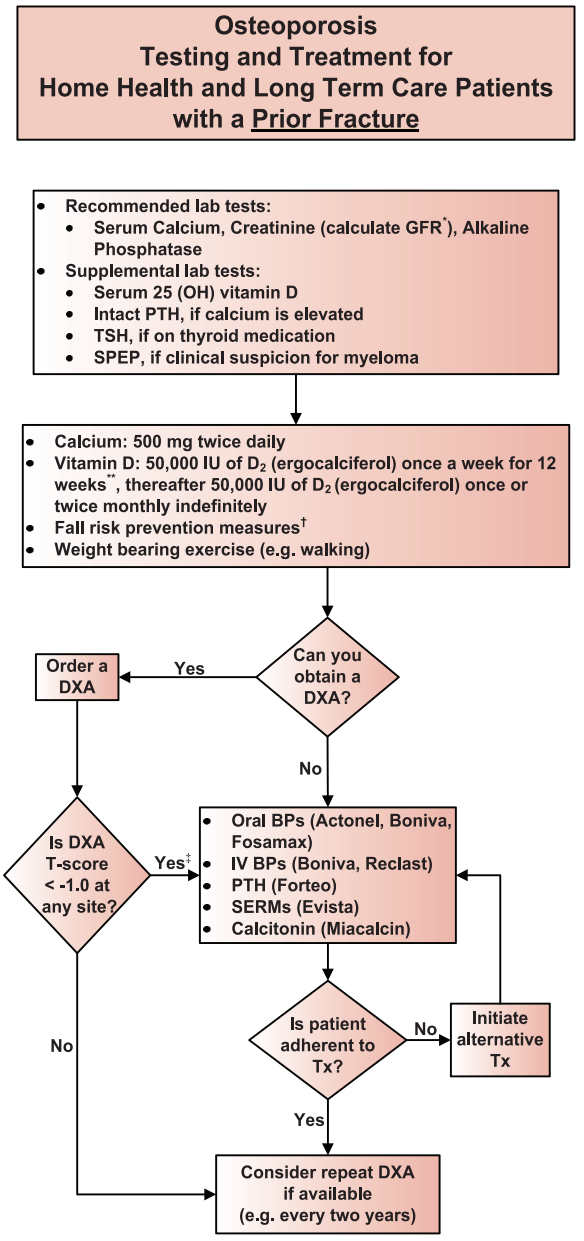
APPENDIX 1

Does This Woman Have Osteoporosis?

Reprinted with permission from JAMA 2004; 292: 2890-2900. Copyright © 2004 American Medical Association. All rights reserved.



APPENDIX 2
Osteoporosis Testing and Treatment Pocket Card



*Glomerular Filtration Rate (GFR) = $\frac{(140 - age) \times (weight\ in\ kg) \times (0.85\ for\ women)}{(creatinine \times 72)}$

**If serum 25 (OH) vitamin D cannot be measured or is < 30 ng/mL (75 nmol/L)

† See other side of card for list of measures (Item #4)

‡Assuming no contraindications (e.g. GFR < 30-35 mL/min for BPs, male sex for Evista)

BP – bisphosphonate; DXA – dual energy X-ray absorptiometry; IU – international unit; IV – intravenous; PTH – parathyroid hormone; SERM – selective estrogen receptor modulator; SPEP – serum protein electrophoresis; TSH – thyroid-stimulating hormone; Tx – treatment

Developed by consensus: Jeffrey R. Curtis MD MPH[1], Kellie L. Flood MD[1], Ryan C. Outman MS[1], Kenneth G. Saag MD MSc[1], Amy H. Warriner MD[1], Cathleen Colón-Emeric MD[2], Kenneth W. Lyles MD[2], Sarah D. Berry MD MPH[3], Nelson B. Watts MD[4], S. Bobo Tanner MD[5]

[1]University of Alabama at Birmingham, [2]Duke University and the Durham VA Medical Center GRECC, [3]Harvard Medical School, [4]University of Cincinnati Bone Health and Osteoporosis Center, [5]Vanderbilt University

PS-17049

APPENDIX 3

FRACTURE Index Questions and Scoring

FRACTURE Index Questions and Scoring

Age

65-69	1 point	
70-74	2 points	
75-79	3 points	
80-84	4 points	
85+	5 points	**3**

Previous Fractures

Yes	1 point	
No	0 point	**0?**

Family History of Fracture

Yes	1 point	
No	0 point	**1**

Weight 125 lbs or less

Yes	1 point	
No	0 point	**0**

Currently Smoke

Yes	1 point	
No	0 point	**0**

Need Use of Arms to Assist Standing from a Sitting Position

Yes	2 points	
No	0 point	**2**

TOTAL 6

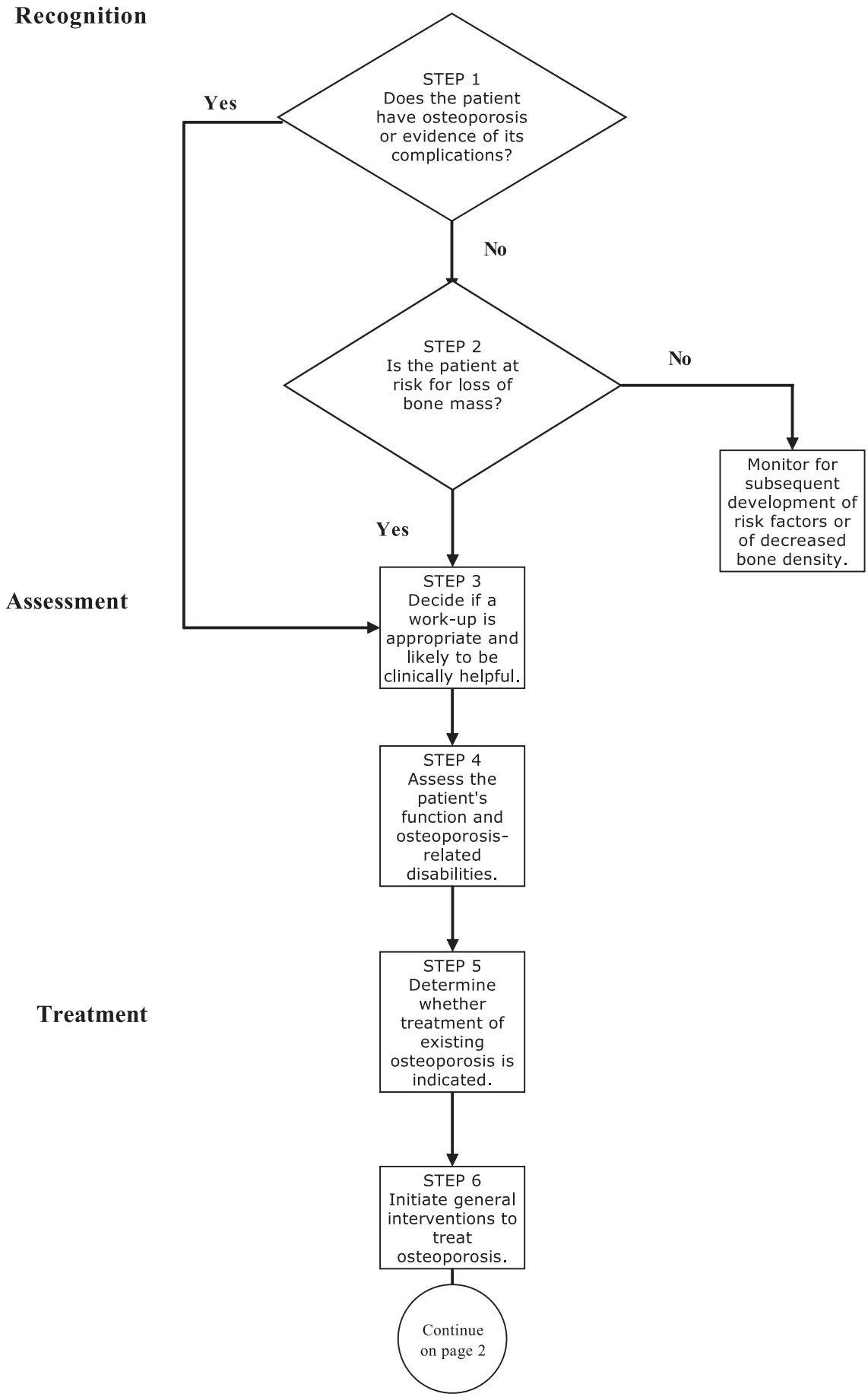
To determine the Fracture Index add the points from the above 6 questions.

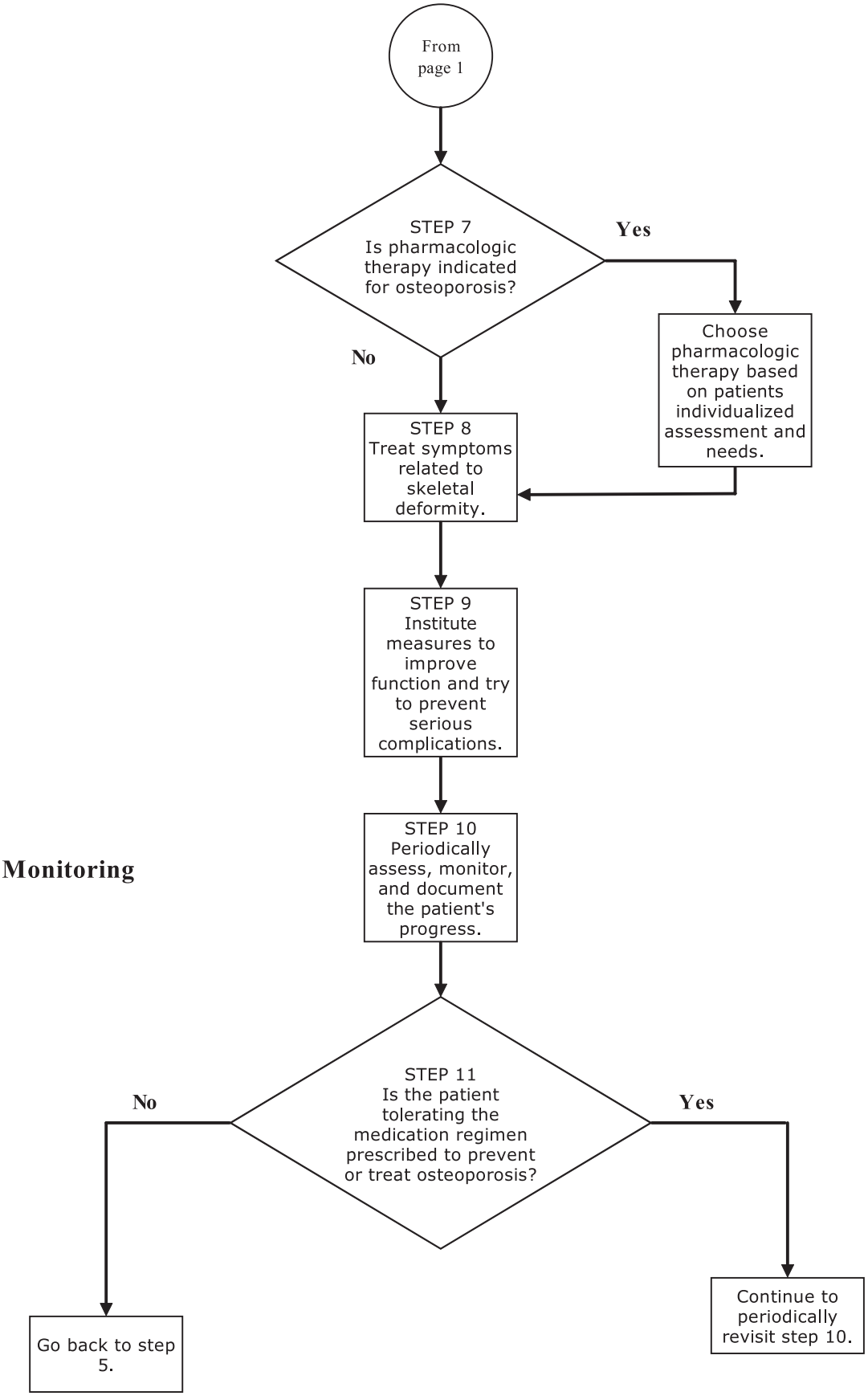
If the Fracture Index is greater than or equal to 4
there is a high risk for fracture due to osteoporosis.

Adapted from Black DM, Steinbuch M, Palermo L, et al. An assessment tool for predicting fracture risk in postmenopausal women. Osteoporosis Int. 2001; 12:519-528

NOTES

This is the osteoporosis and fracture prevention algorithm to be used in conjunction with the written text of this clinical practice guideline. The numbers next to the different components of the algorithm correspond with the steps in the text.


Recognition
STEP 1
Does the patient have osteoporosis or evidence of its complications?
Yes
No
STEP 2
Is the patient at risk for loss of bone mass?
No
Monitor for subsequent development of risk factors or of decreased bone density.
Yes
Assessment
STEP 3
Decide if a work-up is appropriate and likely to be clinically helpful.
STEP 4
Assess the patient's function and osteoporosis-related disabilities.
Treatment
STEP 5
Determine whether treatment of existing osteoporosis is indicated.
STEP 6
Initiate general interventions to treat osteoporosis.
Continue on page 2


From page 1
STEP 7
Is pharmacologic therapy indicated for osteoporosis?
Yes
No
Choose pharmacologic therapy based on patients individualized assessment and needs.
STEP 8
Treat symptoms related to skeletal deformity.
STEP 9
Institute measures to improve function and try to prevent serious complications.
STEP 10
Periodically assess, monitor, and document the patient's progress.
Monitoring
STEP 11
Is the patient tolerating the medication regimen prescribed to prevent or treat osteoporosis?
No
Yes
Go back to step 5.
Continue to periodically revisit step 10.

The Post-Acute and Long-Term Care Medical Association developed this guideline with the support and cooperation of the following individuals and companies:

Charles Cefalu, MD, MS, Clinical Practice Committee Chair
Naushira Pandya, MD, CMD, Project Chair

Steering Committee Members:

Lisa Cantrell, RN, C
Charles Cefalu, MD, MS (Chair)
Sherrie Dornberger, RNC, CDONA, FDONA
Sandra Fitzler, RN
Marianna Grachek, MSN CNHA CALA
Joseph Gruber, RPh, FASCP, CGP
Susan M. Levy, MD, CMD
Evvie F. Munley
Jonathan Musher, MD, CMD
Barbara Resnick, PhD, CRNP
William Simonson, Pharm.D., FASCP, CGP

Corporate Supporters:

Eli Lilly and Company
Medtronic Spinal and Biologics
Procter & Gamble

Organizational Participants:

American Association of Homes and Services for the Aging
American College of Health Care Administrators
American Geriatrics Society
American Health Care Association
American Society of Consultant Pharmacists
Gerontological Advanced Practice Nurses Association
National Association of Directors of Nursing Administration in Long-Term Care
National Association of Health Care Assistants

PALTmed
POST-ACUTE AND LONG-TERM CARE MEDICAL ASSOCIATION

Copyright © 2009
Post-Acute and Long-Term Care Medical Association