

Recommendations for Optimal Care of the Fragility Fracture Patient to Reduce the Risk of Future Fracture

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Abstract

Fragility fractures resulting from low trauma events such as a fall from standing height affect up to one half of women and one third of men after age 50 years. These fractures are frequently associated with osteoporosis. History of a fragility fracture is among the strongest risk factors for future fracture. Therefore, optimal care of the patient with a fragility fracture includes not only treatment of the presenting fracture itself but also evaluation and treatment of the underlying cause or causes to prevent future fractures. However, despite the availability of therapeutic agents that reduce fracture risk among osteoporotic patients who have had a fracture, most patients with fragility fractures are not evaluated for osteoporosis or treated adequately to reduce the risk of future fracture. Orthopaedic surgeons are the first and often the only physicians seen by fracture patients. Thus, they have the unique opportunity to serve as primary advocates to ensure that appropriate action is taken to reduce the risk of future fracture.

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Fragility fractures, defined as those resulting from a low trauma event such as a fall from standing height or less, are very common in older persons, affecting up to one half of women and one third of men after age 50 years.¹ These fractures lead to pain and disability among older individuals and represent a large and increasing financial burden on our health care system. In general, adults who have had any type of fracture are at increased risk of having additional fractures. Fragility fractures are most commonly associated with osteoporosis.² Specifically, history of osteoporotic fractures is among the strongest risk factors for further fractures. Therefore, optimal care of fragility fracture patients includes diagnosis and treatment of the underlying osteoporosis to reduce this risk. However, most patients with fragility fractures are not evaluated for osteoporosis, and often they are not

treated adequately to reduce future fracture risk, even when a diagnosis of osteoporosis has been made.^{3,4}

Most fragility fractures are managed by orthopaedic surgeons, who are usually the first and frequently the only physicians to see these patients. Therefore, orthopaedic surgeons have a unique opportunity to serve as the primary advocates for ensuring that a patient is evaluated for osteoporosis. If the patient is admitted solely under the orthopaedic surgeon's care, the surgeon should ensure that an osteoporosis evaluation is conducted and that the patient receives counseling about diet, exercise, fall prevention, and pharmacologic treatment to prevent future fractures.

In support of this view, the AAOS has adopted the following recommendations as part of its position statement on enhancing the care of patients with fractures. The AAOS encourages

the orthopaedic surgeon to do the following:

(1) Consider the likelihood that osteoporosis is a predisposing factor when a patient presents with a fragility fracture.

(2) Advise patients with fragility fractures that an osteoporosis evaluation may lead to treatment which can reduce the risk of future fractures.

(3) Initiate an investigation of whether osteoporosis is an underlying cause in patients with fragility fractures. The orthopaedic surgeon may conduct this evaluation or may refer the patient to another medical provider.

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(4) Establish partnerships within the medical and nursing community that facilitate the evaluation and treatment of patients with fragility fractures.

(5) Urge their hospitals and office practices to establish clinical pathways that ensure optimal care is provided for patients with fragility fractures.⁵

To be an optimal resource for their patients, orthopaedic surgeons are encouraged to go beyond the acute care and rehabilitation of the fracture itself. Existing guidelines for the management of osteoporosis recommend aggressive evaluation and treatment of patients who present with a fracture.⁶

Scope of the Problem

Osteoporotic fractures occur at many skeletal sites, although those of the hip, spine, wrist, and proximal humerus are the most common. Among Caucasians in the United States, the lifetime risk of fracture at age 50 years is at least 40% in women and 13% in men.⁷ Hip fractures are the major cause of morbidity and mortality associated with osteoporosis. For instance, up to one quarter of hip fracture patients die within 1 year of their fracture. As many as half of all patients with a hip fracture will have long-term disability, and 25% will require long-term nursing home care. These patients may develop additional complications, including pressure ulcers, pneumonia, urinary tract com-

plications, and depression. Although the rate of hip fracture among men is one third to one half that of women of similar age, the increased mortality associated with hip fracture is higher in men than women.⁸

Vertebral fractures are two to three times more prevalent than hip fractures, yet only about one third of vertebral deformities are diagnosed acutely.⁹ This may be because these fractures are sometimes associated with only minor symptoms or are asymptomatic and, therefore, may be overlooked by the physician. However, vertebral fractures can be extremely painful, debilitating, and frequently associated with loss of height, postural changes, kyphosis, and reduced quality of life. Like hip fractures, they are associated with increased mortality and morbidity.

In addition to causing pain and loss of function, fractures in the elderly also represent an enormous financial burden in terms of both direct (hospital acute care, rehabilitation services, long-term care) and indirect (loss of work days, postfracture morbidity) health care costs. In 1995, osteoporotic fractures were the presumed cause of 432,000 hospitalizations, approximately 2.5 million physician visits, and 180,000 nursing home admissions. As such, osteoporosis is among the most expensive chronic diseases; estimated annual direct costs associated with osteoporotic fractures are more than \$17 billion in the United States alone.⁶

However, the most compelling reason for the orthopaedic surgeon to

determine the etiology of a fracture and provide appropriate treatment of the fragility fracture patient is the fact that a previous fracture is among the strongest risk factors for new fractures.^{2,10} Patients with a nonspine fracture have approximately a two-fold greater risk for future fractures than do individuals who have not had a fracture. Furthermore, up to half of patients with a prior vertebral fracture will experience additional vertebral fractures within 3 years, many within the first year. Compared with individuals with no history of vertebral fracture, a patient with a prior vertebral fracture has nearly a five-fold increased risk of future vertebral fractures and a two- to threefold increased risk of hip and other nonvertebral fractures.¹⁰ Altogether, patients with a history of any type of prior fragility fracture have a two- to fourfold increased risk of subsequent fractures compared with those without a previous fracture.

Unfortunately, most fracture patients are discharged from the hospital or from a physician's care without adequate evaluation and treatment of the osteoporosis (the cause of the fracture). Few patients with recent fragility fractures are evaluated for low bone density or prescribed medications for osteoporosis, despite the availability of therapeutic agents that effectively reduce the risk of fracture in patients who have suffered a fracture.⁴ For example, in a study of women aged 65 years and older who had recently suffered a hip fracture, only 13% were receiving ad-

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equate treatment of osteoporosis.¹¹ It is of great concern that osteoporosis remains undiagnosed and untreated in so many patients with fractures. The key to correcting this is implementing clinical pathways for diagnosis and treatment of osteoporosis in fragility fracture patients. The potential success of such programs has been shown in several studies. For example, 6 months after implementing such a clinical pathway, two thirds of 385 fracture patients were taking antiresorptive agents and more than 80% were taking calcium and vitamin D.¹² A protocol developed in two hospitals in Glasgow, Scotland, was so successful that it was implemented in all other trauma center hospitals in the city. Now, all patients admitted for fragility fractures in Glasgow (population, nearly 1 million) are offered evaluation and treatment of the underlying osteoporosis.¹³ Barriers encountered in setting up these clinical pathways, and proven solutions to overcome these barriers, were recently reviewed.¹⁴

Etiology of Age-Related Fractures

Osteoporosis is a systemic skeletal disease characterized by progressive age-related loss of bone strength that leads to increased fracture risk. With increased age come a marked reduction in bone mass and destruction of bone architecture, leading to a considerable decrease in bone strength. Results from biomechanical testing of human cadavers indicate that the strength of the proximal femur and lumbar vertebral bodies is approximately two to eight times greater in young versus elderly individuals. Thus, in persons with osteoporosis, fractures can occur from events and activities that typically do not cause fractures in younger people, such as a minor fall from standing height. In severe cases of osteoporosis, some fractures (particularly vertebral) may result from everyday

activities such as bending over or picking up a small child. The combined effects of age-related declines in bone strength and increases in the incidence and severity of traumatic loading lead to a dramatic increase in fracture risk in older adults.

Bone Mineral Density and Fracture Risk

Although bone strength cannot be determined directly *in vivo*, bone density explains a large proportion of the variation in the mechanical properties of bone tissue and correlates strongly with whole bone strength. Therefore, bone mineral density (BMD) measurements are used in clinical evaluations as an indicator of bone strength and fracture risk. In fact, a BMD measurement predicts fracture risk better than hypertension predicts the risk of stroke or hypercholesterolemia predicts the risk of myocardial infarction.¹⁵

BMD at any age reflects the peak bone mass attained and the amount of subsequent bone loss. BMD generally peaks by age 20 to 30 years and declines rapidly around menopause and thereafter. On average, a postmenopausal woman loses one third to one half of her peak BMD over her remaining lifetime. The relative risk of fracture increases progressively with decreases in BMD, approximately doubling with each standard deviation decrease in BMD.¹⁵ Importantly, the increased risk of future fracture associated with the presence of a previous fracture is independent of a patient's BMD value. Thus, the presence of both low BMD and a previous fracture dramatically increases the risk of fracture more than does the presence of either risk factor alone.

Falls and Fracture Risk

Nearly all wrist fractures, 90% of hip fractures, and up to 50% of vertebral

fractures are associated with a fall.^{16,17} Falls in the elderly are the consequence of many pathophysiological processes associated with aging, including various combinations of intrinsic, activity-related, and environmental factors. For example, poor performance on different gait assessments and difficulty rising from a chair are among the physical traits of individuals at risk for recurrent falls. Use of sedative hypnotics and impaired neuromuscular and visual function are a few of the medical conditions that contribute to falls. Identifying persons prone to recurrent and injurious falls may be important for reducing future fracture risk.

Patient Evaluation

Existing osteoporosis guidelines universally recommend that all postmenopausal women who present with fractures be evaluated for osteoporosis, including measurement of BMD.⁶ Also, certain patients who have not yet fractured but in whom there is a clinical suspicion of osteoporosis should be evaluated. These include patients on chronic glucocorticoid therapy because they are at high risk for fracture. Patients scheduled for spinal surgery or total hip replacement also should be assessed because the presence of osteoporosis in these individuals may adversely affect surgical outcome. The evaluation of a patient for osteoporosis involves conducting a thorough medical history and physical examination, measuring BMD, evaluating routine laboratory tests, and performing a radiographic evaluation (Table 1).

Medical History and Physical Examination

A standard medical history should be obtained, with particular attention paid to age, weight, personal and family history of fracture, and other risk factors (Table 2), as well as to chronic diseases or drug therapies

Table 1
Routine Diagnostic Procedures in Osteoporosis Evaluation ¹⁸

Procedure	Objective
History and physical examination	Assess established risk factors for osteoporosis (Tables 2 and 3). Assess height loss as indication of presence of vertebral fractures.
Bone density measurement	Confirm presumptive diagnosis, assess severity of osteoporosis and risk of future fracture, and use as baseline for monitoring treatment
Laboratory tests: CBC, ESR, serum calcium, creatinine, albumin, phosphate, alkaline phosphatase, liver transaminases, protein electrophoresis, urinalysis, 25-hydroxyvitamin D	Exclude secondary causes of low bone mass and skeletal fragility (eg, multiple myeloma). Check for vitamin D deficiency.
Radiograph of thoracic and lumbar spine, particularly among individuals with back pain or height loss	Assess presence of vertebral fractures

CBC = complete blood count, ESR = erythrocyte sedimentation rate

known to affect BMD and fracture risk (Table 3). The physical examination should be conducted with emphasis on the spine. Height should be measured and compared with the greatest known height to determine height loss, which is indicative of the presence of vertebral fractures.

Bone Mineral Density Measurement

Although several techniques are available to provide valid assessment of fracture risk, dual-energy X-ray absorptiometry (DXA) is generally the most accepted method to measure BMD at the hip or spine. A patient's BMD measurement is usually reported in terms of a T-score, defined as the number of standard deviations above or below the mean value for young, healthy individuals of the same sex. The World Health Organization has established an operational definition of osteoporosis based on BMD measurements and the presence of fragility fractures (Table 4). A final clinical diagnosis and treatment decision should, however, take into ac-

count not only a patient's BMD value but also other risk factors for fracture (Tables 2 and 3, Fig. 1). BMD values may be falsely elevated in individuals with vertebral compression fractures in the lumbar spine and in those with degenerative spine disease. In these instances, bone density measurements should be done at the hip, if possible. In elderly patients with a low trauma hip or vertebral

fracture, a BMD measurement may not be necessary before diagnosis and initiation of therapy because of the very high risk of additional fractures in these individuals.

Osteoporosis is generally the presumptive diagnosis for the underlying cause of fracture in a patient who has had a fragility fracture. The results of the bone densitometry examination therefore can be used to confirm a diagnosis of osteoporosis and gauge the severity of bone loss, to predict future fracture risk to help make treatment decisions, and, if desired, to monitor changes in BMD related to age, medical conditions, or therapeutic intervention.

Laboratory Tests

Laboratory tests other than a BMD measurement are not required for the diagnosis of osteoporosis but rather to initiate investigation of possible causes of low bone mass. At a minimum, routine tests should include a complete blood count (CBC and differential), serum chemistry profile, and urinalysis.

If secondary osteoporosis (ie, diseases or conditions associated with low bone mass) is suspected on the basis of clinical findings or because the patient is relatively young, specific tests should be considered to evaluate contributing causes that may

Table 2
Major Risk Factors for Osteoporotic Fractures ⁶

Not Modifiable	Possibly Modifiable
Advanced age	Low bone mineral density
Female sex	Oral glucocorticoid use
Personal history of adult fracture	Recurrent falls
History of fracture in first-degree relative	Current cigarette use
Dementia	Alcoholism
Poor health/frailty	Estrogen deficiency, including menopause onset before age 45 years
Caucasian or Asian race	Lifelong low calcium intake
	Low body weight
	Little or no physical activity

Table 3
Diseases and Drugs Associated With an Increased Risk of Generalized Osteoporosis and/or Fractures in Adults⁶

Diseases	Drug Therapies
Rheumatoid arthritis	Oral glucocorticoids
Type I diabetes mellitus	Heparin (high doses or prolonged use)
Multiple sclerosis	Excess thyroid medication
Nutritional disorders (especially vitamin D deficiency)	Aromatase inhibitors Tamoxifen (premenopausal use)
Osteogenesis imperfecta	Anticonvulsants
Renal disease	Immunosuppressants
Ankylosing spondylitis	Testosterone antagonists
Prolonged immobilization	

require additional medical attention. These include thyroid disorders, vitamin D deficiency, hyperparathyroidism, hyperadrenalism/Cushing's syndrome, hypogonadism (in men), and malabsorption syndromes (including celiac disease). It is also important to exclude other conditions associated with fracture, such as multiple myeloma and osteomalacia.

Vitamin D deficiency is very common among older individuals but may not be apparent from laboratory reference ranges for serum calcium and phosphate. Thus, all elderly individuals presenting with a fracture should be tested for vitamin D deficiency by measuring levels of 25-hydroxy-

vitamin D. Vitamin D supplementation (800 IU/day) is encouraged in elderly fracture patients.⁶

Specific laboratory tests are available to measure markers of bone formation and bone resorption in serum and urine samples.²⁰ These assays may be helpful in assessing rate of bone turnover and in monitoring response to treatment. However, the clinical utility of biochemical markers in the postfracture patient presently is not known.

Radiographic Evaluation

In older patients with back pain, increasing kyphosis, or excessive height loss (> 4 cm), lateral lumbar and

thoracic spine radiographs should be obtained to document or exclude prevalent vertebral fractures.

Treatment Options

Criteria are generally consistent for diagnosing osteoporosis and making decisions about pharmacologic treatment of patients with and without fragility fractures. Most guidelines agree that, unless treatment is contraindicated, a patient who presents with a fragility fracture attributable to osteoporosis should be treated to reduce the risk of new fractures.⁶ Many experts concur that all patients presenting with a vertebral fracture should be considered for treatment, regardless of BMD measurement. In women without a fragility fracture, both BMD and other risk factors (Tables 2 and 3) should be used to guide treatment decisions.⁶ Although recommendations for the evaluation of men with fractures have not yet been established, an approach similar to that recommended for postmenopausal women is appropriate (Fig. 1). The approach to treatment of a patient with an osteoporotic fracture to reduce the risk of subsequent fractures can be divided into four parts: general recommendations, treatment

Table 4
Operational Definition of Osteoporosis Proposed by the World Health Organization^{18,19}

Category	BMD and Fracture Characteristics	Risk of Fracture	Action
Normal	BMD not more than 1 SD below the young adult mean	Low	No intervention
Low bone mass (osteopenia)	BMD between 1 and 2.5 SDs below the young adult mean	Medium	Consider measures to prevent bone loss in younger women. Exclude contributing causes, particularly in younger individuals. Consider treatment in elderly individuals with history of fragility fracture.
Osteoporosis	BMD >2.5 SDs below the young adult mean	High	Treatment recommended. Exclude contributing causes, particularly in younger individuals.
Severe osteoporosis	BMD >2.5 SDs below the young adult mean in the presence of one or more fragility fractures	Very high	Treatment strongly recommended. Exclude contributing causes.

BMD = bone mineral density, SD = standard deviation

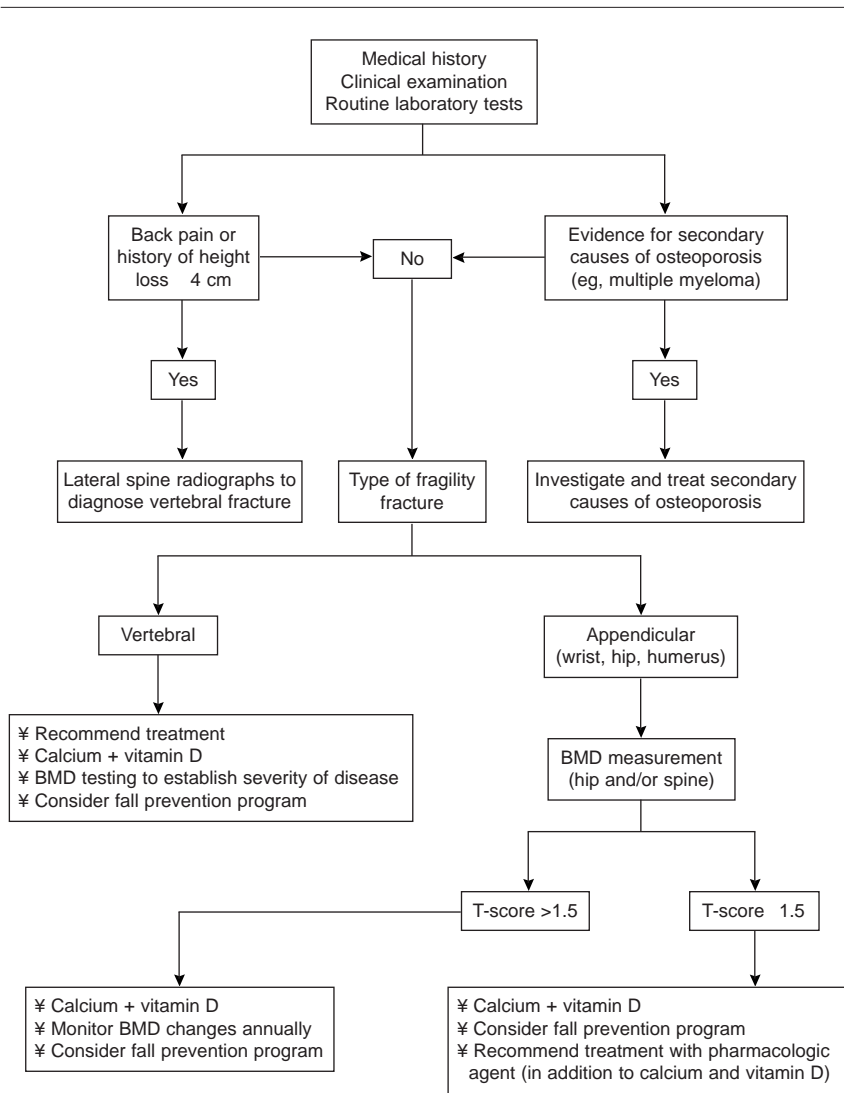


Figure 1 Algorithm for evaluation of fragility fracture patients.

of osteoporosis using pharmacologic agents, prevention of falls, and injury site protection.

General Recommendations

All fragility fracture patients should be counseled on reducing risk factors. Patients should be encouraged to participate in regular weight-bearing exercise, such as walking. The type and frequency of exercise should be appropriate to the patient's risk level, recognizing that those with severe osteoporosis may experience additional fractures of the spine, rib, or other sites from routine activities such

as turning in bed or bending over.²¹ Smokers should be strongly advised to quit. Patients with alcoholism should be treated (one or two drinks a day have not been associated with an increased risk of osteoporosis or fracture). All patients should be evaluated for their risk of falling and appropriate interventions initiated.

All fragility fracture patients with osteoporosis should be encouraged to maintain an adequate intake of calcium (1,200 mg/day) and vitamin D (800 IU/day), either in their diet and by exposure to sunlight, or through dietary supplements.⁶ When taken as

a supplement, calcium citrate is recommended over calcium carbonate because calcium citrate is more readily absorbed in most circumstances. Calcium plus vitamin D may reduce fracture risk among elderly institutionalized and community-dwelling patients, many of whom have vitamin D deficiency and low calcium intake.²²⁻²⁴ However, there are few data specifically regarding the efficacy of calcium and vitamin D supplementation in fragility fracture patients. Treatment with other pharmacologic agents can markedly reduce the risk of fracture over the effects seen with calcium and vitamin D alone. Thus, fracture patients with osteoporosis should strongly be considered for pharmacologic interventions in addition to calcium and vitamin D.

Treatment of Osteoporosis Using Pharmacologic Agents

The ultimate goal in treating fragility fracture patients with osteoporosis is to prevent subsequent fractures. Randomized, placebo-controlled clinical trials involving large populations of women with osteoporosis, as identified by low BMD and/or the presence of a vertebral fracture, have clearly demonstrated the antifracture efficacy of several agents, including bisphosphonates (alendronate, risedronate), hormone therapy, a selective estrogen receptor modulator (raloxifene), calcitonin, and parathyroid hormone (teriparatide) (Table 5). Table 6 summarizes pharmacologic interventions considered to be first-line agents for treating fragility fracture patients with osteoporosis.

Bisphosphonates

Bisphosphonates inhibit bone resorption by osteoclasts. Oral bisphosphonates are poorly absorbed and may induce mild gastrointestinal disturbances. Alendronate (Fosamax; Merck, West Point, PA) and risedronate (Actonel; Proctor and Gamble, Cincinnati, OH), via oral administration either daily or once weekly, are

Table 5
Evidence of Antifracture Efficacy of Agents to Treat Osteoporosis* 24,25

Antiresorptive Agent	Fracture Type		
	Vertebral	Hip	Nonvertebral
Bisphosphonates			
Alendronate	A ^à	A	A
Risedronate	A	A	A
Etidronate	A	C	C
Estrogen replacement therapy/HRT	A	A	A
SERMs (Raloxifene)	A	C	C
Calcitonin, intranasal	A	C	C
Teriparatide (hPTH [1-34])	A	Ñ	A
Calcium and vitamin D preparations			
Vitamin D monotherapy and analogs (Calcitriol, alfacalcidol, etc)	C	C	C
Calcium monotherapy	B	C	C
Vitamin D plus calcium	C	A	A

* From randomized, placebo-controlled clinical trials of women with prior vertebral fractures or with osteoporosis

Nonvertebral fractures; osteoporotic fractures exclusive of the spine

^à Also seen in men

A = convincing evidence of antifracture efficacy, B = inconsistent results, C = ineffective, or insufficient evidence of efficacy

HRT=hormone replacement therapy, SERM=selective estrogen receptor modulator

the bisphosphonates most commonly used to treat osteoporosis. Other bisphosphonates with less frequent dosing regimens are currently being investigated.

Etidronate was among the first bisphosphonates to be developed for clinical use and is therefore considered a first-generation bisphosphonate. Relatively small clinical trials show that etidronate increases BMD modestly and reduces vertebral fracture risk by approximately 30%, with no effect on the risk of nonvertebral fractures.²⁶ High doses and prolonged use of etidronate may impair bone mineralization. Thus, etidronate is not considered to be a first-line agent for treatment of patients with fragility fractures.

In comparison, very large randomized clinical trials of alendronate and risedronate have shown increases in BMD (3% to 10%) accompanied by statistically significant and substantial (up to 50%) reductions in the risk of vertebral fractures, hip fractures, and other nonvertebral fractures in postmeno-

pausal women with osteoporosis (defined by low hip BMD measurement or the presence of a vertebral fracture).^{9,24,27,28} Patients in these studies received adequate calcium and vitamin D, indicating that the bisphosphonates provided additional reductions in fracture risk beyond any effects attributable to calcium and vitamin D. Bisphosphonates also are effective at reducing bone loss and fracture incidence associated with glucocorticoid-induced osteoporosis.^{29,30} Studies of alendronate in men with low spine BMD measurements have shown increases in BMD and reductions in vertebral fracture risk similar to those seen in women.³¹ Finally, despite early concern that bisphosphonate treatment might adversely affect fracture healing, there is no evidence of impaired healing with newer-generation bisphosphonates. Animal studies indicate that bisphosphonate use does not interfere with callus formation but may delay callus remodeling.

Hormone Therapy

Hormone therapy, which may consist of estrogen alone or estrogen plus progestin, increases BMD at all skeletal sites in postmenopausal women. One very large randomized prospective trial, the Women's Health Initiative (WHI), showed a 34% reduction of hip fracture risk during hormone therapy (estrogen plus progestin) in normal postmenopausal women.³² This finding also was supported by several meta-analyses of other trials of hormone therapy, which confirmed a 33% reduction in vertebral fracture risk³³ and 13% to 27% reduction in nonvertebral fracture risk^{33,34} after hormone therapy. However, the estrogen-plus-progestin arm of the WHI was terminated early because of increased incidence of breast cancer, pulmonary embolism, stroke, venous thrombosis, and heart attack in the group receiving hormone therapy.³² In February 2004, the estrogen-alone arm of the WHI study also was stopped. Although estrogen alone appeared to be beneficial for women aged 50 to 60 years, it increased the risk of dementia and stroke in women older than 65 years. Thus, use of estrogen alone may be considered in women aged 60 years or younger who do not have osteoporosis. Although hormone therapy clearly is useful for treatment of menopausal symptoms and decreases fracture risk, it is not recommended as a first-line agent for prevention of osteoporotic fractures in postmenopausal women.³⁵

Selective Estrogen Receptor Modulators

Selective estrogen receptor modulators (SERMs) are synthetic molecules that bind to the estrogen receptor and, depending on the tissue, behave as either an estrogen agonist or antagonist. A 3-year clinical trial with the SERM raloxifene (Evista; Eli Lilly, Indianapolis, IN) showed a 2% to 4% increase in spine and hip BMD that was accompanied by statistically sig-

Table 6
Recommended Pharmacologic Agents to Treat Osteoporosis and Reduce the Risk of Future Fracture in Patients With Fragility Fractures*

Generic Name	Trade Name	Approved Indication	Recommended Dose	Dosing Instructions and Contraindications
Alendronate	Fosamax	Osteoporosis in postmenopausal women and in men	10 mg po qd or 70 mg po weekly	With full glass of water, >30 minutes before food in morning Contraindications: severe renal insufficiency, esophageal motility problem, hypocalcemia, or inability to stand or sit upright for 30 minutes.
Risedronate	Actonel	Osteoporosis in postmenopausal women and in men	5 mg po qd or 35 mg po weekly	With full glass of water, >30 minutes before food in morning Contraindications: severe renal insufficiency, hypocalcemia, or inability to stand or sit upright for 30 minutes.
Raloxifene	Evista	Osteoporosis in postmenopausal women	60 mg po qd	With meals at any time of the day Contraindications: in premenopausal women and those with history of or active venous thromboembolic events
Teriparatide (hPTH [1-34])	Forteo	Postmenopausal women with osteoporosis who are at severe risk for fracture	20 µg sc injection qd	Injection into thigh or abdominal wall. Contraindications: Paget's disease, prior radiation therapy, bone metastases, history of skeletal malignancies, or hypercalcemia

* See prescribing information for each drug for additional recommendations and guidelines.

niPcant 30% and 50% reductions in the incidence of new vertebral fractures in women with and without existing vertebral fractures, respectively.^{24,36,37} Raloxifene had no effect on risk of hip or other nonvertebral fractures.

Calcitonin

The largest clinical trial of osteoporotic women (1,108 patients) treated with nasal calcitonin (Miacalcin; Novartis Pharmaceuticals, East Hanover, NJ) showed small increases in BMD and a statistically signiPcant 20% reduction in the risk of new vertebral fractures (for only one of several tested doses), with no effect on nonvertebral fractures.³⁸ However, because of concerns with the study design and conduct that compromised the results, calcitonin is not considered to be a Prst-line agent for treatment of patients with fragility fractures.

Teriparatide (Parathyroid Hormone [1-34])

Intermittent administration of recombinant human parathyroid hormone (1-34) stimulates bone formation and resorption and has recently been approved for the treatment of severe osteoporosis in the United States and European Union. Daily subcutaneous administration of hPTH (1-34) (Forteo; Eli Lilly) increases BMD and statistically signiPcantly reduces the risk of new vertebral and nonvertebral fractures by 65% and 53%, respectively, in osteoporotic women with an existing vertebral fracture.³⁹ Several studies in animals suggest that hPTH (1-34) also may enhance fracture healing itself, although this remains to be tested in humans.

Possible Future Treatments

Drugs that can be given at less frequent intervals are currently undergoing clinical trials. For instance, a re-

cent study showed that intermittent administration of oral ibandronate (ie, 12 doses every 3 months) increased spine and hip BMD (P < 0.0001) and reduced vertebral fracture risk by 50% (P = 0.0006)⁴⁰ A once-monthly oral form of ibandronate also is being evaluated, although no fracture efficacy data presently are available. Once-yearly intravenous infusion of zoledronic acid was effective in increasing BMD and suppressing bone turnover in postmenopausal women. After 1 year, BMD in the spine and femoral neck increased 5% and 3.5%, respectively, compared with placebo (P < 0.001), although no fracture efficacy data are available⁴¹

A recent study evaluated treatment of postmenopausal women with oral strontium ranelate.⁴² After 3 years, BMD was signiPcantly increased in both the spine and the hip (P < 0.001 for both), and the risk of new vertebral fractures was reduced statistical-

ly significantly by 41%, although the incidence of nonvertebral fractures was not altered. The mechanism of action of this drug is unclear.

In summary, when pharmacologic intervention is indicated, a large body of evidence supports use of alendronate, risedronate, raloxifene, and teriparatide as front-line agents for prevention of new vertebral fractures. Of these, alendronate, risedronate, and teriparatide have been shown to prevent nonvertebral fractures. Only alendronate and risedronate have been shown to reduce the risk of hip fractures.

Prevention of Falls

Falls among older adults are a major risk factor for osteoporotic fractures; thus, reducing the risk of falls is an important part of fracture prevention strategies. However, because falls in the elderly are complex events, they have proved to be remarkably resistant to prevention. Nevertheless, several intervention trials have been successful in reducing falls among the elderly.⁴³ For example, an intensive multifactorial risk factor abatement strategy that included exercise, reduction of medications, and environmental modifications reduced falls by 30%. Other randomized trials have shown that balance- and strength-training regimens, including home-based, professionally prescribed programs that promote balance retraining, muscle strengthening, and walking, as well as group tai chi exercise programs, were able to reduce the older person's risk for noninjurious and injurious falls. Thus, to reduce the risk of falls, physicians should recommend regular weight-bearing exercise for their fragility fracture patients, consider prescribing physical and occupational therapy for fall prevention, and attempt to reduce the number and doses of sedative medications. To be most effective, fall-prevention programs should target both intrinsic and environmental risk factors and should be individually

tailored to each patient's risk profile.^{21,43}

Prevention of Hip Fractures Using Hip Protectors

In 80% to 90% of hip fractures, the immediate cause of the fracture is a sideways fall with direct impact on the greater trochanter of the proximal femur. Thus, there has been interest in the use of external padding, or hip protectors, to prevent hip fractures. Compared with other therapies designed to prevent fractures, an advantage of hip protectors is that they provide immediate protection. Laboratory tests showed that hip protectors exhibit a wide range of force attenuation during a sideways fall, ranging from 20% to 95% of the applied load, depending on the specific padding system. Some padding systems reduce the applied load to a level below that predicted to fracture the elderly femur.⁴⁴

Several, but not all, clinical studies indicate that hip protectors are effective in reducing hip fracture.⁴⁵⁻⁴⁷ For example, clinical trials of a specially designed external hip protector showed a statistically significant >50% reduction of hip fractures in elderly, institutionalized patients.^{45,46} An evidence-based systematic review of seven trials (a total of 3,553 participants) reported that external hip protectors reduce the risk of hip fracture after a fall among individuals at high risk for hip fracture.⁴⁸ However, in most studies, compliance was low. The fact that protection is afforded only when the pad is worn points to the major limitation of and challenge for the widespread use of hip protectors. Furthermore, effectiveness may vary from one manufacturer to another.⁴⁷ These findings are applicable to elderly individuals living in nursing homes or who require residential care or supported living at home; thus, the generalization of the findings beyond this population is unknown. Also, these devices do not protect against fractures other than those of the hip.

Changing the Paradigm

Awareness of the need for new clinical pathways and accountability in the care of patients with fragility fractures is widespread. For example, in addition to the AAOS position statement on this subject, the National Committee for Quality Assurance, a nonprofit organization that accredits and certifies health care organizations, in July 2003 released a new edition of the Health Plan Employer Data and Information Set (HEDIS), a tool used by health plans to measure and evaluate their performance. Recognizing the need for improved care of fragility fracture patients, a new HEDIS measure, "Osteoporosis Management in Women Who Have Had a Fracture," will document the percentage of women aged 67 years and older who are diagnosed with a fracture and who subsequently receive either a BMD test or are prescribed an FDA-approved treatment for osteoporosis within 6 months of the date of the fracture. It is likely that this quality-of-care measure will then be used to compare health care providers and/or provider groups.

Summary

Fracture patients with osteoporosis have a very high risk of suffering a new fracture, often within 1 year of the original fracture. Therefore, optimal care of these patients must include not only acute management of the presenting fracture but also the prevention of subsequent fractures. Therapies proved to reduce fracture risk in osteoporotic patients are available, including bisphosphonates, SERMs, and hPTH (1-34). As the health care provider most likely to manage the presenting fracture, the orthopaedic surgeon can help ensure that the patient receives optimal post-fracture care by considering the possibility of osteoporosis as the under-

lying cause of the fracture and by ensuring that evaluation and appropriate medical treatment and follow-

up of the fracture patient occur. By taking an active role in managing or referring patients with osteoporosis,

the orthopaedic surgeon can substantially improve the long-term outcome for these individuals.

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