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# Osteoporosis of the Spine: Medical and Surgical Strategies

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**Abstract:** Osteoporosis and metabolic bone disease is a worldwide problem with far reaching health and economic consequences, especially as the population ages. Elderly people tend to have progressive loss of bone mineral, leading to significantly higher rates of fragility fractures. Osteoporosis can be classified as type I (postmenopausal) or type II (senile). The spinal vertebrae are the most at risk skeletal elements in the body to fracture. Spinal osteoporosis can be asymptomatic or present as chronic pain and/or deformity. Bone mineral density measurement is considered a prognostic objective piece of data that can assist in the management of these patients. Management is usually conservative for spinal osteoporosis. Options include the use of exercise, estrogen, and bisphosphonates. New options for vertebral insufficiency fractures include percutaneous vertebroplasty. When osteoporosis accompanies surgical degenerative spinal disease, special considerations are necessary to avoid the complications of instrumentation of osteoporotic bone. This article discusses the regulation of bone metabolism, the diagnosis and management of osteoporosis, as well as special considerations of osteoporosis of the spine.

## Epidemiology

More attention is being paid to diseases of the elderly as our population continues to age. Osteoporosis is the most prevalent bone disease in the United States and in other developed countries [1]. The world wide problems of osteoporosis and the associated fragility fractures will continue to absorb health care resources in the future. The incidence of all insufficiency fractures is known to increase with age. It is estimated that 27% of women over age 65 will suffer a vertebral insufficiency fracture [1]. Over the next 30 years, the hip fracture rate is expected to triple [2]. Current estimates predict that by the year 2040, the total cost of caring for hip fractures will be $240 *billion* dollars [3]. By using modern bone mineral density (BMD) techniques, it has been estimated that 54% of all postmenopausal Caucasian women have osteopenia and 30% have osteoporosis [4]. Not only is the incidence of concern, there are also far reaching issues regarding the quality of life of the elderly. A large number of people will suffer from nonoperative, yet painful and debilitating, vertebral fractures that greatly impair their quality of life.

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## Bone Metabolism

The basic cells that mediate bone metabolism are the osteoblasts, osteoclasts, and osteocytes. The osteoblasts produce osteoid, which mineralizes to become bone. The osteoclasts use Howship's lacunae to act as bone resorbers. Osteocytes are mature senescent osteoblasts that reside in the mineralized matrix.

Calcium is a critically important mineral and has many functions at the cellular level. It helps to regulate cell membrane potentials, acts as a cofactor for blood coagulation, plays a role in muscle cell function, and is involved with cellular signal transduction across cell membranes. It is primarily stored in the body as bone mineral, and the normal blood levels are 9–10 mg/dl. Fifty percent of the calcium in the blood is bound to albumin, 45% is present as free ions, and 5% is bound to phosphate or citrate. The bone is used as storehouse for calcium. The body tightly controls the ionized calcium concentration by stimulating calcium resorption from the bone with release of ions into the blood when calcium levels are too low.

Vitamin D is a fat-soluble steroid hormone that modulates calcium homeostasis. Vitamin D synthesis occurs when 7-dehydrocholesterol is exposed to ultraviolet light, creating the precursor $D_3$. $D_3$ then undergoes successive hydroxylation at the liver and kidney to produce the biologically active 1,25 $D_3$. Induction of the liver enzyme P450 by medication such as phenytoin will interrupt the 25 (OH) hydroxylation and will prevent the formation of active $D_3$. Currently, ergocalciferol ($D_2$) is added to milk to ensure adequate oral intake in children. 1,25 (OH) $D_3$ has multiple targets in the body. In the kidney, it increases proximal reabsorption of phosphate and in the intestine, it increases absorption of calcium by enhancing the activity of the calcium-binding protein that is necessary for the active transport of calcium across the intestinal epithelium. It also reduces the production and secretion of parathyroid hormone (PTH), which stimulates bone resorption. Bone remains the primary target tissue for vitamin D, but the exact mechanism has not been elucidated. It has been theorized that osteoclast activity may be stimulated via the osteoblast.

PTH acts closely with vitamin D to regulate calcium homeostasis to form a metabolic axis, which acts on the bone, kidney, and intestines. PTH is formed in the parathyroid gland. Its release is inversely proportional to the serum ionic calcium level. If the calcium level drops, the release of PTH is stimulated. PTH causes bone resorption to release cal-

cium, although the exact mechanism has not been elucidated. Receptors for PTH have not been identified on osteoclasts. PTH is believed to stimulate osteoblasts (which have PTH receptors) to release neutral proteases that resorb osteoid. It may also stimulate osteoblasts to release other unknown factors that directly stimulate osteoclasts to resorb bone. In the kidney, PTH decreases reabsorption of phosphate in the proximal tubule and increases the reabsorption of calcium distally.

### Changes In Bone Metabolism With Aging

A number of changes occur intrinsically with aging. These underlying biochemical changes lead to osteoporosis. PTH levels are elevated in elderly people. With aging, there is also an increased risk and incidence of 1,25 $D_3$ deficiency. This is due to decreased sun exposure, as well as to decreased bioactivity of 1-α hydroxylase. This is the enzyme in the kidney that is responsible for hydroxylating 25 (OH) $D_3$ to make 1,25 (OH) $D_3$, which is the active form of vitamin D. This decrease of vitamin D leads to decreased absorption of calcium from the intestine, leading to calcium deficiency. This leads to the elevated levels of PTH seen in the elderly. These factors result in osteoclast activation, bone resorption, and progressive loss of bone mineralization.

### Other Metabolic Factors

Calcitonin is produced by the clear cells in the thyroid and is known to inhibit bone resorption. Although receptors exist on osteoclasts for calcitonin (as opposed to PTH and 1,25 $D_3$), its physiologic role in bone metabolism is still unclear.

Although receptors for estrogen have been identified on both osteoblasts and osteoclasts, the exact mechanism that estrogen has in the regulation of bone is still being determined. It is known to have a protective effect in its ability to prevent bone loss, but the mechanism is unclear. It is known that women have accelerated bone loss after menopause, and that the drop off in estrogen levels contributes to this. Estrogen replacement (ERT) is an established strategy to prevent osteoporosis, but it must be started within five years of menopause to reduce fracture risk [5]. ERT has certain risks, and each patient's medical history needs to be carefully reviewed. It is contraindicated in patients with a history of endometrial cancer or who have a family history of a first-degree relative with breast cancer.

Corticosteroids are known to cause bone loss [6]. The mechanism is believed to be due to their ability to inhibit the production of calcium-binding protein. This protein is needed for active transport of calcium in the intestine. Steroids also increase renal calcium excretion. These two actions lead to secondary hyperparathyroidism. A chronic dose of as little as 10 mg per day of prednisone is associated with bone loss. People with chronic hyperthyroidism or with chronic supplementation are also known to be at higher risk for bone loss [7].

### Spinal Osteoporosis: Clinical Presentation

Osteoporosis is characterized by decreased bone mass with an increased risk for fracture. Risk factors include age, Caucasian/Asian ethnicity, female sex, steroid use, malnourishment, calcium or vitamin D deficiency, smoking, alcohol consumption, estrogen deficiency, and chronic illness [8,9] The peak bone mass is attained by most people between the ages of 16–25. Bone loss is a relentless process, with men losing 0.3% per year and women losing 0.5% per year. After menopause, the bone loss rate accelerates 2–3% per year for approximately six to 10 years. There are classically two types of osteoporosis as described by Riggs and Melton (Table 1).

The clinical presentation of osteoporosis is one of silent progression. Frequently, the first time the diagnosis is made is with a fragility fracture that occurs with otherwise normal activity. The vertebral bodies are the skeletal elements most at risk. Another common presentation is the incidental vertebral compression fracture seen on the routine lateral chest x-ray. When the osteoporotic patient presents with a vertebral fragility fracture, the primary complaint is one of back pain. There is no radiation into the legs. The acute back pain at the site of the fracture will usually abate with fracture healing, however, some may become chronic in nature, to a lesser degree. Progressive loss of stature leads to progressive shortening of the paraspinal muscles. In order to stand more erect, prolonged active contraction is necessary to maintain posture. This leads to complaints of back pain. This generalized backache may cause patients to limit their activity and alter their quality of life. Secondary to this, patients may develop chronic pain syndrome symptoms, insomnia, and finally clinical depression. Other medical complications may include ileus, urinary retention, and rarely, spinal canal narrowing with cord compression.

Compression fractures of the vertebral bodies may present acutely after minor trauma, or insidiously with mild pain. The spine may or may not be tender to palpation at the site of fracture. Generalized backache will be paraspinal in nature. The physical examination may also reveal a kyphotic deformity of the thoracic spine, otherwise known as a dowager's hump. The mechanism of these fractures is one of flexion with axial compression, with minor events causing damage to the weak bone.

**Table 1.** Osteoporosis classification of Riggs and Melton [12]

| |
|---|
| Type I Postmenopausal: |
| within 15–20 years |
| Trabecular bone affected |
| Fractures: vertebral, distal radius, intertrochanteric femur |
| Estrogen plays primary role in treatment |
| Type II Senile osteoporosis: |
| Women *and* men >70 years |
| Trabecular *and* cortical bone affected equally |
| Multiple vertebral wedge fractures |
| Femoral neck fractures |
| Proximal humerus fractures |
| Aging, long-term calcium deficiency more important |

### Spinal Osteoporosis: Radiographic Evaluation

The standard radiographic evaluation includes an anteroposterior (AP) and lateral x-ray of the thoracic and lumbar spine. Radiographically, lack of bone mass is termed osteopenia. There are many possible causes of osteopenia, one of which is osteoporosis (Table 2) [10]. Approximately 30–50% of bone mineral loss must be present to be detectable on x-ray [11]. The vertebrae show vertical striation and biconcavity. The empty box sign is when there is an accentuated cortical outline of the vertebrae. This is due to enhanced radiolucency of the body. When osteopenia is advanced, the disc spaces may appear denser than the vertebral bodies.

The fracture's morphologic appearance differs based on whether it is located in the thoracic or lumbar spine. The thoracic spine compression fractures occur on the anterior aspect of the bone. This shortening appears as an anterior wedge. The resultant loss of anterior height will lead to a dorsal kyphotic deformity. In the lumbar spine, the load of the compression is distributed equally throughout the body, therefore there is no asymmetric anterior wedging, but instead, the T12-L4 vertebrae assume a codfish appearance.

### Spinal Osteoporosis: Clinical Evaluation

Once an osteoporotic compression fracture is diagnosed, it is important to ensure that the underlying diagnosis is type I or II osteoporosis [12] (Table 1). Type I, or postmenopausal osteoporosis, occurs in women about 15–20 years after the onset of menopause. It affects trabecular bone preferentially, with the resultant compression fractures occuring in the vertebrae, distal radius, and intertrochanteric femur. ERT is the mainstay of treatment. Type II, or senile osteoporosis, is more common in women than in men, but at only a 2:1 ratio. Both trabecular and cortical bone are affected, with vertebral wedge fractures, humerus fractures, and femoral neck fractures being more characteristic. In type II, calcium and vitamin D are more central in the overall treatment regimen. Other causes of osteopenia must be ruled out. A careful history and physical examination must be performed as a first diagnostic step. Questions about constitutional symptoms, previous malignant disease, nutritional status, social habits, and family history will all help rule out other causes. A routine laboratory evaluation consists of the following serum tests: complete blood count, full chemistry panel, thyroid, and testosterone levels. A 24-hour urine collection should be done to check for calcium levels as well N-telopeptide, which is a marker of bone turnover. If hypercalcemia is detected, a workup for primary or secondary hyperparathyroidism should start with checking serum PTH and 1,25 $D_3$ levels. If hypocalcemia, hypophosphatemia, or decreased renal function is present, both 1,25 (OH) $D_3$ and 25 (OH) $D_3$ levels should be checked for underlying vitamin D deficiency. If multiple myeloma is suspected, urine and serum protein electrophoresis should be done. In addition to standard spinal x-rays, BMD is a standard part of any osteoporosis assessment [13].

**Table 2.** Causes of osteopenia seen on x-ray

| |
|---|
| Osteoporosis |
| Type I |
| Type II |
| Endocrine |
| Hyperparathyroidism |
| Hyperthyroidism |
| Diabetes mellitus |
| Cushing's disease |
| Oncologic |
| Multiple myeloma |
| Leukemia |
| Metastatic disease |
| Deficiency states |
| Vitamin D |
| Calcium |
| Vitamin C |
| Malnourishment |
| Chronic disease |
| Chronic renal insufficiency |
| Chronic hepatic insufficiency |
| Malabsorption diseases |
| Inflammatory polyarthritides |
| Drugs |
| Corticosteroids |
| Anticonvulsants |
| Immunosuppressants |
| Social |
| Tobacco use |
| Alcohol use |

### BMD Measurement

BMD is a widely accepted quantitative technique to assess skeletal mass. It is used quantitatively for osteoporosis as a predicative factor for fragility fracture as serum cholesterol is used as a predictive factor for myocardial infarction and as hypertension is used for stroke [14]. The use of BMD is now recognized as a very valuable tool to not only measure mass, but to also define normal and abnormal levels of mass for populations as well as to predict fracture risk. The Bone Mass Measurement Act, passed in 1988, provided medicare reimbursement for BMD testing. It is known that the decreased density of any measurement site in the body correlates with the future global fracture risk of a patient. For example, each standard deviation (SD) reduction of bone mass carries any increased relative fracture risk of 1.5–3.0. The World Health Organization has developed criteria for the diagnosis of osteoporosis (Table 3) [14]. BMD measurements are used to diagnose osteoporosis, to predict fracture risk, and as a measure to quantitate the response to medical treatment.

There are various ways to measure BMD, each technique having its own unique advantages. They are radiographic absorptiometry (RA), single photon absorptiometry (SPA), dual photon absorptiometry (DPA), dual-energy x-ray absorptiometry (DXA), quantitative computed tomography

**Table 3.** World Health Organization criteria for diagnosis of osteoporosis

| Category | Criteria |
|---|---|
| Normal | BMD ≤1 SD below average peak young adult |
| Osteopenia | BMD >1 SD and <2.5 SD below average peak young adult |
| Osteoporosis | BMD ≥2.5 SD below average peak young adult |
| Severe osteoporosis | BMD ≥2.5 SD below average peak young adult and fragility fracture |

(QCT), and quantitative ultrasound (QUS). Taking an x-ray of the hand with an aluminum reference metallic piece with a known density is known as RA. An optical densitometer is then used to measure BMD. Although this technique is inexpensive and does not need special equipment, it is used for appendicular measurement only. SPA and DPA both use a radioactive isotope to measure the bone and soft tissue absorption of a photon beam. Although these two techniques are good at predicting fracture risk, they require radioactivity and suffer from problems related to isotope degradation. QCT uses a standard CT scanner with a special software package. The technique uses a known density phantom and compares that to the density of trabecular bone and cortical bone, which are measured separately. The advantage of QCT is that it performs direct volume measurements of BMD at the anatomic site in question, either the hip or the spine. The disadvantage is the much higher radiation dose required compared to other modalities. A newer technology is the QUS device, which measures BMD at the calcaneus by using sound waves. It is noninvasive and cheap and has been shown to be good for hip fracture prediction. However, it is a peripheral test, and suffers the problem of all peripheral tests: discordance. Bone density decreases in the spine first, as there is a high turnover in the trabecular bone. BMD becomes reliably concordant (i.e., there are similar values in the appendicular and axial skeleton) by the age of 65 [15]. Up to the age of 65, axial BMD of the spine is the most accurate site. Currently, the gold standard of BMD measurement is DXA. It measures BMD both of the hip and spine, covering the peripheral and axial skeleton. An x-ray tube emits a radiation beam and attenuation through the skeleton is measured by a photon counter. The scan times are short, the radiation dose is low, and the technique is precise.

### Indications for BMD Testing

Currently, patients who suffer from a fragility fracture (distal radius, vertebral compression, hip, or proximal humerus) with significant osteopenia should have BMD testing done. Other indications are listed in Table 4 [16].

### BMD Results

When reading the results of BMD tests, it is important to understand what values are being measured and what they mean for the patient's treatment. The most important data reported are the T- and Z-scores. The T-score is the number of SD that the bone density is above or below the young adult mean. For every SD below normal, the fracture risk doubles. For example, a T-score of −1 has a two times risk of fracture than a person with a normal BMD [17]. The DXA test measures density at both the proximal femur and the lumbar vertebrae. In the spine, the most accurate T-score is the average of L1–L4. A 50-year-old woman with a T-score of −1 has a 30% chance of sustaining a fragility fracture. With a T-score of −2.5, the chance of sustaining a fracture increases to 60%. The Z-score is the number of SD the BMD is above or below the value expected for the patient's age. Thus, a Z-score compares the BMD to a patient's peers, as opposed to the T-score, which uses the young adult mean for comparison. A Z-score of lower than −1.5 is quite severe, and should stimulate a workup for secondary osteoporosis. It should be noted that periarticular sclerotic conditions such as osteoarthritis could cause a falsely elevated bone density reading. Overall, however, the authors prefer DXA for BMD testing due to its preciseness, low cost, and minimal radiation.

### Osteoporosis Prevention and Treatment Strategies

The goal of any osteoporosis treatment plan is to prevent further bone loss. Currently, there is no treatment that can restore bone mass to normal. Prevention starts with discouraging social risk factors such as smoking or drinking alcohol. An exercise program is both an excellent treatment and prevention modality. These exercises should consist of spine extension exercises (avoid flexion), abdominal strengthening, and walking. This program can begin supervised by a physical therapist, then continued at home. One hour of exercise two to three times per week can increase bone mineral content in the lumbar spine and total body calcium. Physical and occupational therapists can also assist in instituting a fall prevention program.

Prevention and treatment in women begin with ERT [18]. It will prevent bone loss, decrease fragility fractures, and carries with it cardioprotective benefits. However there are contraindications. There is an increased risk of breast cancer with greater than five years ERT use during the early postmenopausal period. Also, older patients who have been on

**Table 4.** Indications for BMD testing

| |
|---|
| Postmenopausal women who are not on ERT and who would consider treatment |
| Maternal history of hip fracture |
| Smokers |
| Thin habitus (>5 ft 7 in., <125 lb) |
| Medications associated with bone loss |
| Predisposing medical conditions (hyperthyroidism, s/p transplant) |
| High levels of urinary collagen cross-links (suggestive of high bone turnover) |
| History of previous fragility fractures |

ERT for more than 10 years have an increased risk of breast cancer [19]. Yet, the cardioprotective effects of ERT are believed to outweigh the other associated risks [20]. Patients who have a history of breast cancer or a first-degree relative (sister or mother) with breast cancer should not be started on ERT. There is a three to fourfold increase risk of deep venous thrombosis in postmenopausal women. Previous thromboembolic or endometrial diseases are relative contraindications. There is also an increased risk of uterine cancer, which is decreased with adjunct progestin use. Any patient who is started on ERT needs to have an initial gynecologic and endometrial biopsy at 12 months. Finally, an annual mammogram is recommended once ERT is begun.

Raloxifene is a selective estrogen receptor modulator (SERM) that represents a new class of drug. A recent study has demonstrated a 40% reduction in new spine fractures among women who have had a previous vertebral fracture and a 60% decrease in new vertebral fractures in women who have never had one previously [21]. There is no effect on the breast tissue or the uterine lining, but it does seem to have the cardioprotective effect of estrogen. It is another promising alternative to estrogen, and it has been shown to increase BMD by 2% [22].

Other medications include the bisphosphonates. These compounds bind to bone mineral and inhibit the dissolution on calcium phosphate. They decrease osteoclast activity as well as decrease osteoclast recruitment. Alendronate is the most common bisphosphonate prescribed today. Typically, 5–10 mg per day are taken orally on an empty stomach, then a delay of 30 minutes before eating is advised. This is due to its the low bioavailability (0.7%). Additionally, alendronate may be poorly tolerated due to its propensity to cause gastroesophageal reflux. It is recommended that patients avoid a reclining position after taking the drug, to help better tolerate the possible reflux. Alendronate prevents bone loss, and studies have shown gains in bone mass of up to 10%. A 3% decrease in vertebral fractures at three years has been demonstrated. Overall, a 50% reduction in spine and hip fractures has been observed. Alendronate is an excellent alternative to ERT in patients in whom it is contraindicated [23].

Calcitonin is available in two forms. The injectable salmon product dose is 100 units per day and the nasal spray's dose is 200 units per day. Calcitonin has been shown to increase spinal bone mass and to decrease vertebral fractures by 37%. It is another alternative for patients who cannot take ERT. An additional benefit of the nasal preparation is the analgesic properties in the setting of an acute vertebral compression fracture. The exact mechanism of this effect is not known. Formation of antibodies to salmon calcitonin (sCT) is common and occurs in 40–70% of the patients treated for more than 4 months. Not all of these patients, however, develop a secondary resistance to sCT; therefore, the clinical significance of sCT antibodies is unclear. It does explain the known resistance to calcitonin that occurs in 25–45% of the patients after treatment periods of 6 months and longer [24].

Dietary supplementation of both calcium and vitamin D is important to help prevent decreased bone mass. In premenopausal women, calcium oral intake will not prevent osteoporosis, but will ensure achieving peak bone mass during the formative years. The importance of adequate oral intake of calcium during the growing years cannot be overemphasized. In older postmenopausal women (greater than 6 years), oral intake of calcium can slow bone loss and help prevent osteoporosis [25]. Adequate oral calcium intake is necessary in all patients, especially the elderly, to prevent secondary hyperparathyroidism. In a recent study, it was demonstrated that vitamin D deficiency is associated with hip fracture in postmenopausal women. In osteoporotic patients or in patients at risk, the recommended daily dose of elemental calcium is 1.5 g and 800 units of vitamin D.

### Vertebral Compression Fracture Treatment

The vast majority of these patients are treated nonoperatively and symptomatically. This consists of a corset for the lumbar spine or a hyperextension-type brace for the thoracic spine. Although bracing may help with pain control, it is unlikely to prevent further collapse or new fractures. Early mobilization is encouraged with physical therapy. Admission to the hospital occurs occasionally for initial pain management and for supervised mobilization. As the acute episode passes with nonoperative treatment, more problems can occur with sequelae. These issues include chronic pain, kyphosis, and depression. Medical management needs to be optimized, often using multiple medications including calcitonin for analgesia. Deformity may be static or progressive, and this needs to be closely followed. Kaneda et al. [26] have reported on late neurologic deficit due to spinal canal narrowing with delayed collapse. For these 22 patients, Kaneda performed an anterior corpectomy, ceramic replacement, instrumentation, and used a postoperative thoracolumbosacral orthosis (TLSO). With an average 34-month follow-up, he gained significant kyphosis correction and improved neurologic status. Of note, avascular necrosis was found in the corpectomy bone.

A new procedure that has been reported on extensively in the radiology literature is percutaneous vertebroplasty performed with fluoroscopic or CT guidance. Although still considered experimental, it has been gaining increased attention in the orthopaedic and spine community, with the procedure becoming more common. The procedure involves percutaneously placing a trocar into the kyphotic compressed vertebral body and restoring vertebral height and alleviating pain with the introduction of polymethylmethacrylate (PMMA) or other composite material [27–30]. In the thoracic spine, the route is typically transpedicular. In the lumbar spine, a posterolateral approach may be used. Pioneered in France, this procedure was originally used for metastatic tumors of the spine. Reports described good to excellent results regarding pain relief, albeit with a small complication rate of leakage of materials. In 1996, Cotten et al. [31] reported a series of 37 patients. They all underwent percutaneous fluoroscopically guided vertebroplasty for metastatic lesions or myeloma. Of 37 patients, 36 had sig-

nificant pain relief, but there were 33 leaks. Of the eight foraminal leaks, two patients required surgical decompression. The other leaks were clinically insignificant. Other authors have reported on the compression fracture experience and have found very few complications [27–30]. Regarding pain relief, the success rates have approached 90–100%. It appears that the leak problem may be due to the underlying bony destruction associated with erosive tumors as compared to the compression phenomena of fracture. Other authors have noted that controlling the amount of PMMA injected with help keep leaking to a minimum. A modification to this technique is the balloon vertebroplasty (Figs. 1–3). Two balloons are introduced via bilateral trocars. Inflation of the balloons restores vertebral height. One balloon is deflated and PMMA is injected through that trocar. Then the second balloon is deflated and PMMA is injected into the created defect. In addition to the recovery of vertebral height and the correction of a segmental kyphosis, a third advantage to the balloon technique is the relatively low pressure that is required for the injection, as compared to the original vertebroplasty technique. This procedure is still in its infancy and there are few studies in the orthopaedic literature that detail the results of vertebroplasty in the osteoporotic compression fracture. Most of the published studies are in the radiology literature. These vertebroplasties were done for the pathologic and tumorous vertebral fractures. Although the indications for this procedure are still not clearly defined, the compression fracture that remains recalcitrant to medical management and is associated with marked kyphosis may be an excellent candidate for this procedure.

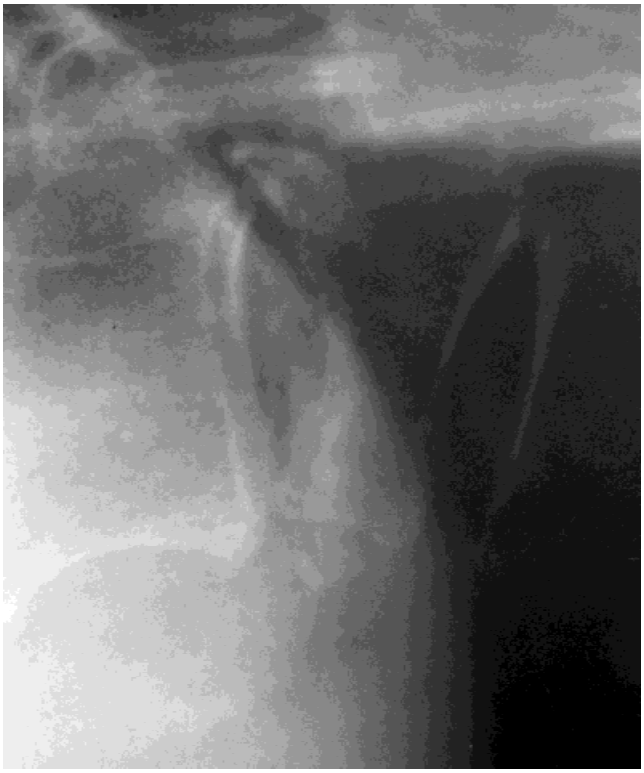

**Fig. 1.** Thoracic anterior wedge compression fracture. Note the loss of anterior body height.

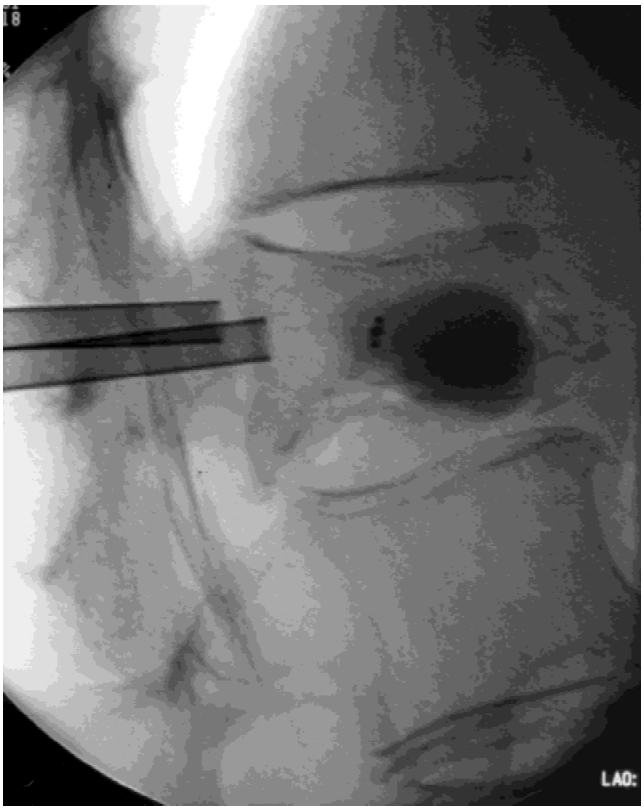


**Fig. 2.** Lateral fluoroscopy with transpedicular trocars and balloon vertebroplasty device in the vertebral body. Note the restored anterior height.

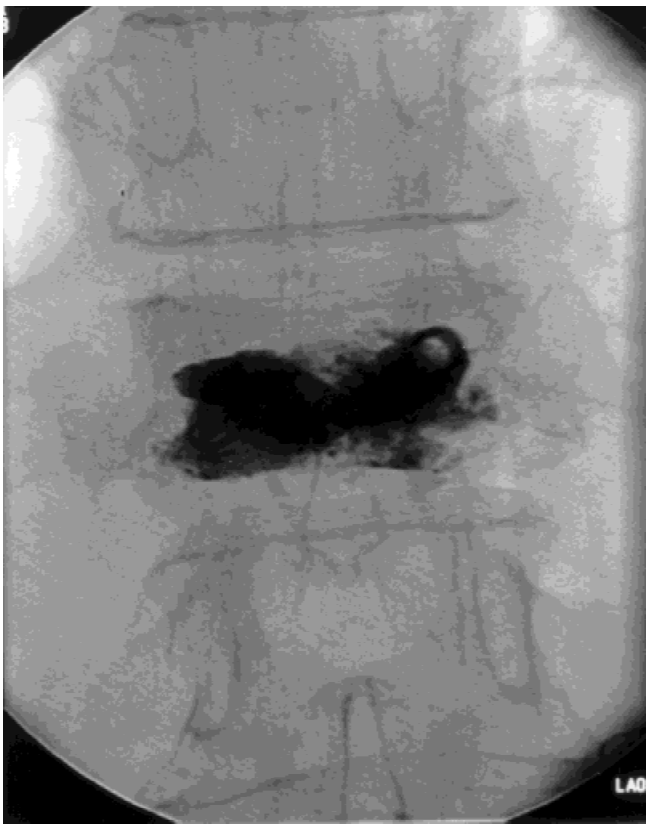

**Fig. 3.** AP fluoroscopy of completed vertebroplasty with polymerized polymethylmethacralate (PMMA) bone cement in place. The vertebral height is restored.

## Special Problems With Instrumentation of the Osteoporotic Spine

Although most spine surgeons try to avoid using instrumentation when operating on the osteoporotic spine, it is sometimes necessary. At times, there are indications such as instability, scoliosis, or kyphosis when instrumentation would improve the chance of a successful arthrodesis or otherwise help to ensure a better surgical outcome. The problem resides in the lack of mineral in the bone, leaving essentially very porous trabecular bone. This porous bone has poor pullout strength. Also, the pedicles widen analogous to the widening of the femur with advanced osteoporosis. All that is left are the cortices of the pedicles, with no trabecular bone available for gripping of the threads of the screws. Hu [32] has described some strategies that can be employed in these patients. These involve using multiple sites of fixation such as sublaminar wires around the nearly all cortical lamina or using multiple levels of hooks. These techniques can be used in lieu of pedicle screws if the posterior elements are intact (e.g., posterior spinal fusion for unstable kyphotic compression fracture). If the posterior elements are not intact (e.g., after decompression for degenerative spondylolisthesis or stenotic degenerative lumbar scoliosis), other strategies can be used to prevent pedicle screw pullout. The laminectomy can be used to directly visualize the pedicle to ensure exact placement and reduce the risk of cutout. Undertapping or avoiding tapping at all should be employed. Adjacent levels that did not require laminectomy can be used to augment the construct by placing laminar hooks to increase the lever arm and decrease the stress on the pedicle screws. In very porous bone, PMMA can be used to augment the pedicle screw holes, which can help secure stable fixation to the vertebral bodies.

## Summary

As the population ages, all physicians will be involved with patients with osteoporosis. The systemic effects of this disorder will lead to a great deal of morbidity. To effectively treat the rising percentage of elderly patients in the population, prevention must be established as the mainstay of treatment. Ensuring early and adequate intake of calcium and vitamin D will help to maximize total bone mass during the growing years. As osteoporosis affects the spine with aging, both silent and acute compression fractures may be the earliest sign of osteoporosis. There are new medications that can restore bone mass to some degree and can even help to reduce the risk of new and subsequent fractures. In the patient who fails conservative management of the vertebral insufficiency fracture, newer, although experimental, techniques are being developed to help rehabilitate patients much faster. This will help to avoid the “fracture disease” of the debilitated patient who suffers from a painful vertebral osteoporotic compression fracture. Special measures should be taken in the osteoporotic spine that requires spinal fusion, and instrumentation should be used very carefully.

## References


1. Melton LJ III: Epidemiology of vertebral fractures in women. *Am J Epidemiol* 129:1000–1011, 1989.
2. Kannus P, Niemi S, Parkkari J, et al: Hip fractures in Finland between 1970 and 1997 and predictions for the future. *Lancet* 353:802–805, 1999.
3. Cummings SR, Rubin SM, Black D: The future of hip fractures in the United States. *Clin Orthop* 252:163–166, 1990.
4. Melton LJ III: How many women have osteoporosis now? *J Bone Miner Res* 10:175–177, 1995.
5. Cauley JA, Seeley DG, Ensrud K, et al: Estrogen replacement therapy and fractures in older women. Study of Osteoporotic Fractures Research Group. *Ann Intern Med* 122:9–16, 1995.
6. Amin S, LaValley MP, Simms RW, et al: The role of vitamin D in corticosteroid-induced osteoporosis. *Arthritis Rheum* 42:1740–1751, 1999.
7. Schneider DL, Barrett-Connor EL, Morton DJ: Thyroid hormone use and bone mineral density in elderly women. Effects of estrogen. *JAMA* 271:1245–1249, 1994.
8. Wasnich RD: Epidemiology of osteoporosis. In: Favus MJ (ed). *Primer on the Metabolic Bone Diseases and Disorders of Mineral Metabolism* (4th ed.). Philadelphia: Lippincott, pp 257–259, 1999.
9. Cummings SR, Nevitt MC, Browner WS, et al: Risk factors for hip fracture in white women. *N Engl J Med* 332:767–773, 1995.
10. Jergas MD, Genant HK: Radiology of osteoporosis. In: Favus MJ (ed). *Primer on the Metabolic Bone Diseases and Disorders of Mineral Metabolism* (4th ed.). Philadelphia: Lippincott, p 60, 1999.
11. Lachmann E, Whelan M: The roentgen diagnosis of osteoporosis and its limitations. *Radiology* 26:165–177, 1936.
12. Riggs BL, Melton LJ III: Evidence for two distinct syndromes of involutional osteoporosis. *Am J Med* 75:899–901, 1983.
13. Miller PD, Bonnick SL, Rosen CJ: Clinical utility of bone mass measurements in adults: Consensus of an international panel. *Semin Arthritis Rheum* 25:361–372, 1996.
14. The WHO Study Group: *Assessment of Fracture Risk and Its Application to Screening for Postmenopausal Osteoporosis.* Geneva: World Health Organization, 1994.
15. Pouilles JM, Tremollieres F, Ribot C: Spine and femur densitometry at menopause: Are both sites necessary in the assessment of the risk of osteoporosis? *Calcif Tissue Int* 52:344–347, 1993.
16. Miller PD, Bonnick SL, Johnstons CC, et al: The challenges of peripheral bone density testing: Which patients need additional central density skeletal measurements? *J Clin Dent* 1:211–217, 1998.
17. Hui SL, Slemenda CW, Johnston CC Jr: Age and bone mass as predictors of fracture in a prospective study. *J Clin Invest* 81:1804–1809, 1988.
18. Lindsay R, Hart DM, Aitken JM, et al: Long-term prevention of postmenopausal osteoporosis by oestrogen. Evidence for an increased bone mass after delayed onset of oestrogen treatment. *Lancet* 1:1038–1041, 1976.
19. Steinberg KK, Smith SJ, Thacker SB, et al: Breast cancer risk and duration of estrogen use: The role of study design in meta-analysis. *Epidemiology* 5:415–421, 1994.
20. Grodstein F, Stampfer MJ, Colditz GA, et al: Postmenopausal hormone therapy and mortality. *N Engl J Med* 336:1769–1775, 1997.
21. Ettinger B, Black DM, et al: Reduction of vertebral fracture risk in postmenopausal women with osteoporosis treated with raloxifene. *JAMA* 282:637–645, 1999.
22. Scott JA, Da Camara CC, Early JE: Raloxifene: A selective estrogen receptor modulator. *Am Fam Physician* 60:1131–1139, 1999.
23. Liberman UA, Weiss SR, Broll J, et al: Effect of oral alendronate on bone mineral density and the incidence of fractures in postmenopausal osteoporosis. The Alendronate Phase III Osteoporosis Treatment Study Group. *N Engl J Med* 30:1437–1443, 1995.
24. Grauer A, Ziegler R, Raue F: Clinical significance of antibodies against calcitonin. *Exp Clin Endocrinol Diabetes* 103:345–351, 1995.

25. Dawson-Hughes B, Dallal GE, Krall EA, et al: A controlled trial of the effect of calcium supplementation on bone density in postmenopausal women. *N Engl J Med* 323:878–883, 1990.
26. Kaneda K, Asano S, Hashimoto T, et al: The treatment of osteoporotic-posttraumatic vertebral collapse using the Kaneda device and a bioactive ceramic vertebral prosthesis. *Spine* 17:S295–S303, 1992.
27. Martin JB, Jean B, Sugui K, et al: Vertebroplasty: Clinical experience and follow-up results. *Bone* 25:11S–15S, 1999.
28. Deramond H, Depriester C, Galibert P, et al: Percutaneous vertebroplasty with polymethylmethacrylate. Technique, indications, and results. *Radiol Clin North Am* 36:533–546, 1998.
29. Gangi A, Kastler BA, Dietemann JL: Percutaneous vertebroplasty guided by a combination of CT and fluoroscopy. *Am J Neuroradiol* 15:83–86, 1994.
30. Jensen ME, Evans AJ, Mathis JM, et al: Percutaneous polymethylmethacrylate vertebroplasty in the treatment of osteoporotic vertebral body compression fractures: Technical aspects. *Am J Neuroradiol* 18: 1897–1904, 1997.
31. Cotten A, Dewatre F, Cortet B, et al: Percutaneous vertebroplasty for osteolytic metastases and myeloma: Effects of the percentage of lesion filling and the leakage of methyl methacrylate at clinical follow-up. *Radiology* 200:525–530, 1996.
32. Hu SS: Internal fixation of the osteoporotic spine. *Spine* 22:43S–48S, 1997.