

In contrast, sodium fluoride, an agent that has yet to be approved for treatment of osteoporosis, induces a linear increase in bone formation and skeletal density that is noted even up to 4 years.<sup>10</sup> It is hoped that etidronate can produce a linear increase in density over time without the toxic effects that fluoride may produce.

Although etidronate has been used to treat established osteoporosis, it may have greater application in the prevention of early menopausal bone loss and development of osteoporosis. With the high rates of bone turnover in early menopause following the loss of estrogenic activity, osteoclastic activity is increased. An agent that suppresses this enhanced osteoclastic activity, such as etidronate, calcitonin, or estrogen, will prevent skeletal deterioration.

Long-term use of etidronate or any diphosphonate must be evaluated carefully. The complications of long-term estrogen therapy are well known. Extended use of calcitonin may incur an element of tachyphylaxis and loss of efficacy. Long-term use of diphosphonates could bring unexpected complications, although this may be

quite unlikely with low doses. Among the patients who underwent skeletal biopsies in the recent trials, there was no evidence of toxicity.

The regimen for etidronate therapy is simple. The patient takes 400 mg/d etidronate disodium for 14 days every 3 months. Because of low absorption, the drug must be taken on an empty stomach and no food should be consumed for several hours thereafter. Pre- and post-treatment bone density measurements are used to assess efficacy. The drug is not yet approved by the Food and Drug Administration for treatment of osteoporosis. As noted, long-term toxicity is unknown but short-term skeletal toxicity is not a problem. With minimal gastrointestinal side effects, the drug is highly acceptable to patients.

ANGELO A. LICATA, MD, PHD  
Department of Endocrinology  
The Cleveland Clinic Foundation  
One Clinic Center  
9500 Euclid Avenue  
Cleveland, Ohio 44195

## REFERENCES

1. Watts N, Harris S, Genant H, et al. Intermittent cyclical etidronate treatment of postmenopausal osteoporosis. *N Engl J Med* 1990; 323:73-79.
2. Storm T, Thamsborg G, Steiniche T, et al. Effect of intermittent cyclical etidronate therapy on bone mass and fracture rate in women with postmenopausal osteoporosis. *N Engl J Med* 1990; 322:1265-1271.
3. Krane SM. Etidronate disodium in the treatment of Paget's disease of bone. *Ann Intern Med* 1982; 96:619-625.
4. Russell RGG, Fleisch H. Pyrophosphate and diphosphonate in skeletal metabolism. *Clin Orthop* 1975; 108:241-262.
5. Bonnekamp PM, van der Wee-Pals LJA, van Wyk-van Lennep MML, et al. Two modes of action of bisphosphonates on osteoclastic resorption of mineralized matrix. *Bone Miner* 1986; 1:27-39.
6. Delaisse J-M, Eeckhout Y, Vaes G. Bisphosphonates and bone resorption: effects on collagenase and lysosomal enzyme excretion. *Life Sci* 1985; 37:2291-2296.
7. Gibbs CJ, Aaron JE, Peacock M. Osteomalacia in Paget's disease treated with short-term, high-dose sodium etidronate. *Br Med J* 1986; 292:1227-1229.
8. Ralston SH, Boyce BF, Corvan RA, et al. The effect of 1 alpha-hydroxyvitamin D3 on the mineralization defect in disodium etidronate-treated Paget's disease—a double-blind, randomized clinical study. *J Bone Miner Res* 1987; 2:5-12.
9. Gennari C, Chierichetti SM, Bigazzi S, et al. Comparative effects on bone mineral content of calcium and calcium plus salmon calcitonin given in two different regimens in postmenopausal osteoporosis. *Curr Ther Res* 1985; 38:455-463.
10. Murray TM, Harrison JE, Bayley TA, et al. Fluoride treatment of postmenopausal osteoporosis: age, renal function, and other clinical factors in the estrogenic response. *J Bone Miner Res* 1990; 5(suppl):S27-S36.

## Erratum

There were errors in the article, "Quality assessment in the medical intensive care unit: evolution of a data model," by Edward D. Sivak, MD, and Alejandro Perez-Trepichio, published in the May 1990 issue, Volume 57, Number 3. In Table 2, page 276, the median age of patients on the gastroenterology service was 60.8 years.

The age range of survivors on the thoracic cardiovascular surgery service was 15 to 79 years, and the age range of nonsurvivors was 52 to 69 years. The median number of pre-ICU days for nonsurvivors on the thoracic cardiovascular surgery service was 66.