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Assessment of the risk of vertebral fracture in menopausal women

FR Convery and C Lavernia
J Bone Joint Surg Am. 1987;69:952-953.

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also be demonstrated clinically in a patient with cubital tunnel syndrome by acutely flexing the elbow for one to five minutes and precipitating the patient's symptoms — the so-called elbow flexion test.

For these reasons we think that the postoperative rather than the intraoperative period plays a greater role in this condition.

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Dr. Alvine and Dr. Schurrer reply:

As was mentioned in the article, the mean time of onset for all seventeen patients, including those who had coronary artery surgery, was 3.3 days postoperatively. If the patients who had coronary artery surgery are excluded, the mean time of the onset of symptoms was 1.3 days postoperatively. While they may not have mentioned it until several days postoperatively, on close questioning these patients frequently related the onset of numbness to the period of time in the recovery room or stated that they woke up with the tingling and numbness. Subsequent to the preparation of this paper, an additional twelve patients have been referred to our office, eight of whom related the onset of symptoms to the immediate postoperative period. The other four could not recall precisely when the symptoms started, but the onset ranged from five days to six weeks postoperatively. As mentioned, many of these patients were seen many months postoperatively.

The studies of Wadsworth² and of Macnicol¹ are valid. Prolonged flexion of the elbow at any time, be it during the operation or in the postoperative period, should be avoided.

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1. MACNICOL, M. F.: Extraneural Pressure Affecting the Ulnar Nerve at the Elbow. *Hand*, 14: 5-11, 1982.
2. WADSWORTH, T. G.: The External Compression Syndrome of the Ulnar Nerve at the Cubital Tunnel. *Clin. Orthop.*, 124: 189-204, 1977.

To the Editor: We are writing regarding the recent article "Assessment of the Risk of Vertebral Fracture in Menopausal Women" (69-A: 212-218, Feb. 1987) by Buchanan et al. On the basis of this study it is recommended that women who have a vertebral density of less than seventy milligrams per milliliter be treated prophylactically for osteoporosis. There are at least two fundamental issues that are not addressed in this work. First, the "standard" for assessing fracture risk could be a measure of effect rather than cause. By having "one or more" fractures, this group of women had an activity level that should be less than that of women without fractures. A decreased activity level could result in disuse atrophy and lowered density values. The presence of fractures can affect the activity level and therefore the measured density.

The second concern is that while seventy milligrams per milliliter might be an adequate cutoff point for treatment of a certain patient, weight and size are also important and need to be considered. Ultimately the density measurements are a reflection of the ultimate strength of the vertebrae which, when exceeded, results in a fracture. The stresses in a vertebral body on a day-to-day basis are determined by the body habitus and weight of the patient. A more accurate fracture index would take into account the weight of the patient and the size of the vertebral body as well as the race of the patient. Kleeman et al.³ showed that the best predictor of fracture in an osteoporotic population was the density of a vertebral body squared divided by the area. Although in their study they also scanned patients who had fractures, their index takes geometry into account.

The use of bone densitometry to assess fracture risk is considered by

some of the developers of the technique still to be an experimental tool^{4,5}. Substantial research needs to be done before it can be used in the community. Accordingly, in our opinion, the recommendations of Buchanan et al. are premature and may be inappropriate.

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Dr. Buchanan, Ms. Myers, Dr. Greer, Dr. Lloyd, and Dr. Varano reply:

Dr. Convery and Dr. Lavernia have raised some very important issues with regard to the measurement of bone density. Their suggestion that "fractures can affect the activity level and therefore the measured density" is a well taken point. We agree with the implication that bone density could have decreased in the interim between the fracture event and the measurement by computerized tomography. Under these circumstances, the observed fracture threshold would understate the true threshold. However, the magnitude of understatement must be small since none of the twenty-four women with a bone density of greater than seventy milligrams per milliliter had a fracture. Moreover, other investigators' results are strikingly similar to ours: Rosenthal et al.⁷ observed no fractures in patients who had a bone density of greater than seventy milligrams per milliliter, but they did note an abrupt increase in the frequency of fracture in patients who had a bone density below this level.

To further address this issue, we examined the hypothesis that a decreased activity level affects the frequency of fracture independently of the effect of bone density. We compared the cases of sixty-three menopausal women who had no chronic disease with those of eighteen presumably less active subjects (patients who had pulmonary, neurological, or cardiac disease). Analysis of covariance showed that after controlling for the effect of bone density, activity level had no effect on the frequency of fracture. Thus, we doubt that activity level could have introduced much error into our data analysis.

Regarding the second concern of Convery and Lavernia, it is well known that biological factors such as height, weight, size, and age influence bone density. Whether these factors also influence fracture rates independently beyond their effect on bone density is unknown. To examine this issue, we employed multiple regression analysis, taking the frequency of fracture as the dependent variable and each biological factor as an independent variable. In every case, after controlling for the effect of bone density, the biological factors exerted no additional effect on the frequency of fracture. Thus, knowledge of these factors does not add useful information to the determination of bone density for assessing the risk of atraumatic fractures.

Assessment of fracture risk by quantitative computerized tomography has been evaluated independently at several centers. The conclusion that the threshold for atraumatic fractures lies between seventy and 100 milligrams per milliliter has been quite consistent despite the use of instruments from different manufacturers and varying methods for defining fractures^{1,4,7}. Quantitative computerized tomography discriminates between patients with and those without fractures better than other densitometry methods³. We believe that the methodology of computerized tomography is sufficiently developed to justify routine clinical use for the assessment of fracture risk and in the management of metabolic disturbances causing bone loss.

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