Guidelines or a plea for help?

The US Preventive Services Task Force (USPSTF) recently published a clinical guideline on the use of calcium and vitamin D supplements to prevent fractures in adults.¹ This agency "strives to make accurate, up-to-

date, and relevant recommendations about preventive services in primary care,"² and within those parameters they generally succeed. But I am confused about the value of this specific guideline, and apparently I am not alone.

The task force came to several major conclusions about calcium and vitamin D supplementation to prevent fractures:

- There is insufficient evidence to offer guidance on supplementation in premenopausal women or in men
- One should not prescribe supplementation of 400 IU or less of vitamin D_3 or 1 g or less of calcium in postmenopausal women
- The data are insufficient to assess the harm and benefit of higher doses of supplemental vitamin D or calcium.

The task force stuck to their rules and weighed the data within the constraints of the specific question they were charged to address.

A challenge to clinicians attempting to apply rigidly defined, evidence-based conclusions is that the more precisely a question is addressed, the more limited is the answer's applicability in clinical practice. Thus, Dr. Robin Dore, on page 341 of this issue of the *Journal*, says that she believes there are benefits of vitamin D and calcium supplementation beyond primary prevention of fractures, and the benefits are not negated by the magnitude of potential harm (stated to be "small" by the USPSTF).

We are bombarded by clinical practice guidelines, and we don't know which will be externally imposed as a measure of quality by which our practice performance will be assessed. In the clinic, we encounter a series of individual patients with whom we make individual treatment decisions. Like the inhabitants of Lake Wobegon, few of our patients are the "average patient" as derived from cross-sectional studies. Some have occult celiac disease, others are on proton pump inhibitors, some are lactose-intolerant, and some are on intermittent prednisone. For these patients, should the USPSTF guidelines warrant the extra effort and time to individually document why the guidelines don't fit and why we made the clinical judgment to not follow them? Additionally, how many patients in the clinical studies used by the USPSTF fit into these or other unique categories and may have thus contaminated the data? I don't see in these guidelines recommendations on how best to assess calcium and vitamin D intake and absorption in our patients in a practical manner. After all, supplementation is in addition to the actual intake of dietary sources.

For me, further confusion stems from trying to clinically couple the logic of such carefully analyzed, accurately stated, and tightly focused guidelines with what we already know (and apparently don't know). We know that severe vitamin D deficiency clearly causes low bone density and fractures from osteomalacia, and the Institute of Medicine has previously stated that adequate vitamin D is beneficial and so should be supplemented.³ Vitamin D deficiency is a continuum and is very unlikely to be defined by the quantity of supplementation. Additionally, the USPSTF has previously published guidelines on supplementing vitamin D

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intake to prevent falls—falls being a major preventable cause of primary fractures. There seems to be some conceptual incongruence between these guidelines.

While epidemiologic studies have incorporated estimates of dietary and supplemental intake of calcium and vitamin D, what likely really matters is the absorption and the achieved blood levels and tissue incorporation. As shown in the examples above, many variables influence these in individual patients. And most troublesome is that there is no agreement as to the appropriate target level for circulating vitamin D. I agree with two-thirds of the task force's conclusions—we have insufficient evidence. Are these really guidelines, or a plea for the gathering of appropriate outcome data?

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LETTER TO THE EDITOR

Sleep-disordered breathing and resistant hypertension (FEBRUARY 2013)

TO THE EDITOR: We recently read the article by Dr. Emmanuel Bravo.¹ In his comprehensive paper, he defined a road map for the workup of resistant hypertension. Resistant hypertension is a challenging problem in everyday practice, with multiple pitfalls at each step from diagnosis to treatment.

Although not mentioned in the paper, obstructive sleep apnea is strongly associated with hypertension, and its prevalence in patients with resistant hypertension can be as high as 83%.² The upper airway resistance syndrome is another form of sleep-disordered breathing in which transient increases in upper airway resistance result in repetitive electroencephalographic arousals. Unlike obstructive sleep apnea, upper airway resistance syndrome is not associated with apnea or diminished airflow, although snoring and excessive davtime somnolence are common. Repeated arousals, desaturations, or both during sleep lead to recurrent sympathetic surges with resultant nocturnal hypertension. There are a number of reports in the literature of large blood-pressure reductions after continuous positive airway pressure treatment.³

In conclusion, sleep-disordered breathing syndromes should be sought vigorously in cases of resistant hypertension, and every effort should be taken for proper management.

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