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[One of the authors responds:]

We agree with Joel Ray and David Cole regarding the relative practical value of serum total homocysteine and methylmalonic acid in elderly patients with suspected cobalamin deficiency. Although testing for serum holotranscobalamin is not routinely available in many countries, we believe that it may be appropriate in future as a routine clinical test for cobalamin deficiency. However, to date, a consensus on the definition for cobalamin deficiency, especially among elderly patients, has not been achieved.[1] Thus, in our experience, serum total homocysteine is currently a helpful, inexpensive indicator of true (tissue) cobalamin deficiency, as suggested in our article.[2]

We agree with Peter Wetterberg's comments on oral cobalamin. However, the usefulness of oral cobalamin therapy has only recently been documented, starting in 1995, with studies that meet the criteria of evidence-based medicine.[3-6]

We read with great interest Jonathan Prousky's comments, although we have no experience with the therapies he describes. Nevertheless, we believe that this information indirectly supports the concept of food-cobalamin malabsorption.[7]

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Short ACTH stimulation test for adrenal reserves of cortisol, not adrenal function

The classical description of Addison's disease in a 15-year-old girl[1] is a timely reminder of this well-known but uncommon disorder. In their description of the investigative work-up and discussion, Chantelle Barnard and associates[1] imply that the short adrenocorticotropin hormone (ACTH) stimulation test is diagnostic of primary adrenal insufficiency. This is a common misapprehension.

In the test, an intravenous (or intramuscular) injection of 250 µg of synthetic ACTH (tetracosactrin) results in release of preformed cortisol from adrenal stores, which is measured in the serum 30 (and/or 60) minutes later and compared with the baseline concentration. An abnormal response (a serum cortisol peak below 550 nmol/L or an increment of less than 200 nmol/L from baseline or both) identifies adrenal insufficiency but cannot distinguish Addison's disease (primary adrenal failure) from secondary hypoadrenalism. In pituitary disease (ACTH deficiency), for instance, the result of the test may be abnormal because of reduced stores of cortisol, even though the adrenal glands themselves have normal biosynthetic and secretory func-

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