That parathyroid hormone (PTH) plays a role in patient morbidity or mortality cannot be disputed. Thus, measurement of PTH is essential for patient care. We agree with Dr. Garrett and colleagues that PTH is not an ideal assay (1). We further agree that the reliance on the assessment of PTH concentrations alone is a dangerous substitute in the search for a more reliable biomarker for the complications of CKD-MBD (1). But at the present time there are no better diagnostic tools with which to climb this “mountain” of CKD-MBD. And the mountain is indeed burdensome in terms of causing adverse cardiovascular outcomes, fracture, and morbidity (2). Not using the best tool available is not a good option, and we must remind ourselves that the lack of data does not equate to negative data. As clinicians, especially in the field of nephrology, we are often forced to make decisions on the basis of less than optimal evidence. The use of PTH is not an ideal assay (1). We further agree that the reliance on measurement of PTH is essential for patient care. We agree with Dr. Garrett and colleagues that PTH is not an ideal assay (1). We further agree that the reliance on measurement of PTH is essential for patient care. We also agree the assays have shortcomings and that international standardization is needed. However, with careful attention to specimen handling and collection, and the use of the same laboratory (which should use the same manufacturer’s assay), any measured difference in PTH concentration represents a true difference (3). In reality, measuring PTH more often, rather than less, is the way to deal with the possible imprecision of the assay. The Kidney Disease Improving Global Outcomes (KDIGO) guidelines recommended following trends rather than individual results for this very reason. To identify patients at risk and adjust therapies, more measurements of PTH, not fewer, should be performed to evaluate trends. Unfortunately, this recommendation to evaluate trends does not lend itself to an easily implemented treatment algorithm. But just because it cannot be easily implemented does not mean we should not measure PTH. In addition, bone alkaline or total alkaline phosphatase provides information only on skeletal function, whereas PTH also provides a measurement of a systemic uremic toxin (5). We agree with the KDIGO recommendation that alkaline phosphatase, a test far more clinically available, could be used to help guide decisions as a “blended approach” (1) when the PTH is two to nine times the upper limit of normal (2).

Garrett and colleagues further note that PTH should not be measured because there is inadequate evidence to link PTH to skeletal and cardiovascular endpoints in CKD. Does this mean that ignorance is bliss? We do believe that sufficient evidence links PTH with adverse outcomes, especially when levels are toward the extremes of the KDIGO recommendations (2), and therefore that monitoring PTH on a regular (and preferable more frequent) basis and treatment of elevated PTH levels are important. Unfortunately, much of the clinical practice of nephrology is extrapolated from the general population. There is equally limited evidence for the monthly assessment of adequacy of dialysis, the treatment of hypertension, and the management of dyslipidemia and hyperglycemia, specifically in dialysis patients. Unfortunately, there is no CKD-MBD in patients without kidney disease with which to extrapolate clinical practice. However, hyperparathyroidism is a real disease, and our real call to arms is not to avoid measurement but to conduct more research.

References

Published online ahead of print. Publication date available at www.cjasn.org.

See related article, “PTH—A Particularly Tricky Hormone: Why Measure It at All in Kidney Patients?,” on pages 299–312.