literature about special meanings of the phase angle as compared to Xc are flawed by inconsistent comparisons among groups with different vector lengths.

Body impedance (from either whole-body or body segments, measured in either single or multiple frequency) is described by a complex number, or vector Z. The bivariate vector Z can be represented in the real-imaginary plane (that is, rectangular coordinates) as a combination of R (the opposition to flow of an alternating current through intra- and extracellular ionic solutions, representing the real part of Z) and Xc (the capacitance produced by tissue interfaces and cell membranes, representing the imaginary part of Z at every current frequency) across soft tissues (fat plus fat-free mass without bone). The impedance vector Z can alternatively be represented in the polar coordinates with two other components, magnitude [$|Z| = \sqrt{(R^2 + Xc^2)}$] and direction (that is, phase angle = arctan Xc/R). In statistical analysis, the bell-shaped distribution of both vector components and their mutual correlation allow easy handling of Z as a bivariate, normal, random vector in the rectangular coordinate system (such as a probability RXc graph [2–3]).

In the literature and in practice, in subjects with a comparable R value (comparable vector length), greater Xc values (greater phase angles) are observed in subjects with greater soft tissue mass (such as obese people and athletes) as compared to subjects with lower Xc values (smaller phase angles) and reduced soft tissue mass (leanness, malnutrition, cachexia). However, great phase angles of long vectors (high Xc and R values) are observed in dehydration (for example, cholera, post-dialysis), even with malnutrition and poor prognosis, and reduced phase angles of short vectors (reduced Xc and R values) are observed in fluid overload (edema, pre-dialysis) even with good nutrition and prognosis [1, 2, 4]. Indeed, the smaller inter-subject variability of the phase angle as compared to Xc readings, due to the correlation between R and Xc, does not reduce the inverse, strong effect of tissue hydration on phase angle. Thus, only considering the phase angle and neglecting vector length necessarily finds a better (worse) nutrition and prognosis at the end (start) of a dialysis session (Fig. 4 in [2]).

In conclusion, trivial errors can be avoided by plotting vectors at appropriate, bivariate reference intervals (tolerance ellipses) [3], and looking carefully at either the R reading or vector length before stating the patient's prognosis in terms of Xc or phase angle, respectively.

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Statistical error in pamidronate study?

To the Editor: The paper by Fan et al, recently published in Kidney International [1], addresses a very important question, as discussed in an Editorial in the same issue [2].

The authors conclude: *"This study has shown that the early rapid bone loss that occurs in men during the first 12 months after renal transplantation can be prevented by two intravenous doses of pamidronate given at transplantation and one month later. The regimen was simple to administer, well tolerated and potentially applicable to other clinical groups of glucocorticoid treatment patients."*

It might be so, but it is doubtful whether the authors have shown this to be the case. The authors randomized 26 male patients undergoing renal transplantation to receive either placebo (500 mL 0.9% saline, $N = 12$) or intravenous pamidronate (0.5 mg/kg wt in 500 mL 0.9% saline, $N = 14$) at the time of transplantation and again one month later. The primary effect parameters were changes in bone mineral density (BMD) at the lumbar spine and at the femoral neck after 12 months. There was a 6 to 9% decrease in BMD after 12 months compared to baseline in the placebo group, whereas there was no significant decrease after 12 months in the pamidronate group. However, when performing a clinical randomized placebo-controlled study, the changes in the effect parameters in the intervention group have to be compared to the changes in the placebo group with an appropriate statistical method before any conclusion on the effect of the intervention can be made. This was not reported in the paper by Fan et al [1]. The authors did not present numbers that allow the readers to do the proper analysis on their own, but from the figures given, it seems unlikely that there were statistically significant differences in the changes in BMD at the lumbar spine or at the femoral neck between the pamidronate group and the placebo group. This might be due to a type II error, that is, a lack of statistical power due to the relatively small number of subjects in the study.

It would be appropriate if the authors report on the comparisons between the changes in BMD between the pamidronate and the placebo group, and if statistically insignificant, to report the type II error given the observed differences and the number of subjects, and then to make a more appropriate conclusion.

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Reply from the authors

Dr. Svendsen makes the valid points that intergroup analysis of the outcomes after therapy or placebo should be performed and that as the numbers of patients in this study is small, the risk of a type II error cannot be discounted, at least with respect to the lumbar spine data.

Using both nonparametric (Mann-Whitney U) test and unpaired Student's *t*-test, we have compared the changes in bone mineral density (BMD) in the control group with those in the pamidronate-treated group 12 months after renal transplantation. At the femoral neck, patients who were treated with pamidronate lost less bone compared with control ($P < 0.002$ for both tests). However, at the lumbar spine, there were no intergroup differences, although as stated in the paper [1], within group comparison showed significant loss in control but no such loss in the pamidronate treatment group.

The baseline study of regional BMD in our patients reveals that the mean T score at the femoral neck was significantly lower than at the lumbar spine (-0.89 ± 0.32 vs. -0.20 ± 0.37, $P < 0.05$). The finding that cortical osteopenia is greater than cancellous osteopenia in dialysis patients has also been documented by other investigators [2]. Accelerated cortical bone loss driven by secondary hyperparathyroidism [3, 4] may explain the distribution of bone loss at different sites in dialysis patients. Our previously published work also documented that after transplantation in men, but not in women, bone loss was greater at the femoral neck than at the lumbar spine [5]. These findings suggest that protection of the skeleton from the consequences of hyperparathyroidism may explain the apparent greater benefit of pamidronate at this predominantly cortical site.

We believe, therefore, that the data conclusively point to beneficial effects of pamidronate at the femoral neck, and probably at the lumbar spine as well, albeit with less certainty.

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INSTRUCTIONS FOR LETTERS TO THE EDITOR