

## Dr. Roll, *et al* reply

*To the Editor:*

We thank Drs. Besada and Nossent for their remarks regarding the rate of serious infections in patients with antineutrophil cytoplasmic antibody-associated vasculitis (AAV) treated with rituximab<sup>1</sup>. Their points are well taken, and more studies on this topic are required in the future. Clearly, previous therapies and the additional immunosuppression, particularly the often longterm administration of steroids, have to be considered and are likely to mean some risk. Nonetheless, our longterm data available to date could not show an increased infection risk in this refractory patient population<sup>2</sup>. The problems concerning hypogammaglobulinemia and late-onset neutropenia are well known<sup>3,4</sup>. Unfortunately, values of IgM, IgG, or neutrophils are available for only a few patients in our registry<sup>5</sup>. In contrast to patients with rheumatoid arthritis, patients with AAV showed an enhanced mortality, mostly attributable to the more intense previous and concomitant medication<sup>5</sup>. Of interest, this enhanced mortality was not seen regarding serious and overall infections in this group. In the whole group we saw a decreasing rate of infections soon after therapy. However, for definitive conclusions, longterm data for Ig levels and comedications are lacking in our registry. So we can only confirm the unenhanced infection rate in patients with AAV in our observation time, and agree that the problems of hypogammaglobulinemia, late-onset neutropenia, and T cell dysfunction must be given attention. Longer observation times are needed to draw definitive conclusions.

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