We thank Sampimon *et al.* for their critical comments and additions to our review.[1,2] We mentioned β-blockers and chlorhexidine to provide an overview of the various factors that might be implicated in the pathogenesis of encapsulating peritoneal sclerosis (EPS). As Sampimon *et al.* correctly point out, the particular β-blocker practolol and chlorhexidine are not used anymore.

We have a number of problems with the figure concerning the incidence of EPS in the AMC Amsterdam. First, it appears that two periods of five years are compared with one 3.6-year period. Second, according to the figure there were 10 patients with EPS between 1995 and 2008, while an earlier paper from the same group reported 11 patients with EPS between 1995 and 2006.[1] More importantly, we believe that these relatively small numbers are difficult to interpret without providing data on the population at risk. Data from two other university hospitals in the Netherlands clearly indicate a rise in the incidence of EPS during recent years.[3] Ideally, a national (or international) registry should provide more insight into the epidemiology of EPS.

We agree that the prevention guideline proposed in our paper is largely opinion-based due to lack of available evidence. Although the study of CT scans in EPS by Tarzi *et al.* was published after submission of our review, we are not surprised by their finding that in 9 of 13 patients with EPS the CT abdomen was normal or near-normal at a median of 1.5 years before the time of clinical diagnosis.[4] Accordingly, we advocated to consider discontinuation of PD in patients with two or more risk factors, even in the absence of CT abnormalities.

Sampimon *et al.* object to discontinuation of PD in long-term PD patients without any sign of changes in the peritoneum, based on their finding that clinical signs of established EPS are preceded by specific trends in peritoneal transport characteristics.[2] However, there are no reassuring data that the progression of EPS can be halted once changes in peritoneal transport are overt. In fact, EPS even develops after discontinuation of peritoneal dialysis (PD) in a considerable number of cases. On the other hand, it is beyond doubt that the incidence of EPS rises substantially after prolonged PD treatment. Therefore, we are reluctant to expose our long-term PD patients to the risk of developing this devastating complication, especially when an alternative treatment modality is available.

Finally, we hope that the discussion evoked by our proposal for an EPS prevention guideline as well as the announced proposal of Sampimon *et al.* may ultimately result in a consensus guideline on how to treat our patients optimally.

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