Letter

Early venovenous haemodiafiltration for sepsis-related multiple organ failure

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In a prospective observational study that included 60 consecutive patients over a 10-year period, Page and coworkers [1] studied the effects of early continuous venovenous haemodiafiltration (CVVHDF) during sepsis-induced multiple organ failure. In two-thirds of the patients rapid metabolic improvement during CVVHDF was associated with circulatory improvement and a low mortality rate, whereas lack of metabolic improvement after 12 hours of CVVHDF (mainly based on changes in base excess) was associated with a 100% mortality rate. The authors concluded that early CVVHDF may improve the prognosis of sepsis-related multiple organ failure, and that failure to correct metabolic acidosis rapidly during the procedure is a strong predictor of mortality.

In that study, metabolic acidosis was assessed using base excess values, and the authors highlighted the influence of individual changes 6-12 hours after initiation of CVVHDF on predicted outcome. However, despite the findings reported, the usefulness of base excess is questionable. First, because of the high incidence of circulatory failure occurring after several days of hospitalization, base excess may be influenced by large volume crystalloid infusion, resulting in altered protein status. Second, the link between base excess and lactate concentration was weak ($r^2 = 0.36$ for the patients studied). We believe that lactate values may be more important in predicting outcome than base excess. Indeed, it

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is widely accepted that early lactate clearance is associated with improved outcomes in septic shock [2] and that lactate levels are not affected by CVVHDF [3]. In the study by Page and coworkers [1] one cannot exclude the possibility that the lack of improvement in base excess in the nonresponder group was linked to persistent lactate production, and so metabolic improvement during the procedure is not necessarily superior to the trend in blood lactate as a predictive tool.

Although beneficial effects of early high-volume isovolaemic haemofiltration have been reported [4,5], such benefits may not be realized with standard volume procedures [6]. Furthermore, a randomized study design would have enhanced the strength of the findings reported by Page and coworkers [1]. Moreover, we believe that a comparison between observed mortality and that predicted by Simplified Acute Physiology Score (SAPS) II does not permit one to draw conclusions regarding whether CVVHDF has a beneficial effect [7], at least for secondary shock, in view of the poor performance of physiological scores for delayed acute renal failure.

In summary, this study of standard volume CVVHDF in sepsis [1] is undoubtedly important, but its findings should be viewed with caution until further investigations have been undertaken.

Authors' response

Antoine Vieillard-Baron and François Jardin

We thank Drs Jacobs and Brivet for their remarks.

Beyond the pertinence of comparing predicted mortality by SAPS II with observed mortality, the overall mortality rate of our population was 53%, which is far from the 85% and 92% mortality rates previously reported in similar populations [8,9].

Moreover, in a previous prospective study conducted by Dr Brivet himself and his coworkers [10], the mortality rate in patients who exhibited acute renal failure related to septic shock was as high as 79.4%, whereas the mean SAPS score was 19, corresponding to a mean SAPS II score of about 50, which is much lower than the SAPS II score in our population [1].

Drs Jacobs and Brivet consider base excess to be of questionable usefulness in assessing metabolic acidosis. However, as they point out above, it appears not to perform so poorly because the change in base excess after 12 hours of CVVHDF strongly discriminated those patients with a 100% mortality rate. They also suggest that base excess could in part reflect hyperchloraemic acidosis induced by large volume crystalloid infusion. However, as reported in Table 2 of our report [1], plasma chloride concentration was in the normal range and did not differ between the two groups. On the other hand, we agree that base excess reflects not only lactic acidosis but also renal acidosis. However, in our opinion, this is better for evaluating the severity of illness in such patients because it takes into account two of the main parameters that have been reported to be associated with high mortality rates in sepsis (i.e. renal failure and persistent lactic acidosis) [11].

Drs Jacobs and Brivet point out that we did not use high-volume haemofiltration. It is true that we used a flow rate of only 2000 ml/hour, leading to an average convection exchange of 28 ml/kg per hour. However, because we performed CVVHDF, we also used 1000 ml/hour of dialysis, leading to a 'global exchange' of about 40 ml/kg per hour.

Finally, Drs Jacobs and Brivet suggest that the lack of improvement in base excess in nonresponders may have been linked to persistent lactate production. Indeed, it was caused by persistent lactate production! However, on reading the latter comment by Drs Jacobs and Brivet, we wonder whether they completely understood our report, in which CVVHDF is proposed to be an additional means to ameliorate circulatory failure, not a treatment for acidosis *per se*.

Competing interests

The authors declare that they have no competing interests.

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