

## Sodium bicarbonate for severe metabolic acidaemia – Authors' reply

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## Authors' reply

We report that in a severely acidaemic critically ill population, the infusion of 4.2% sodium bicarbonate solution to target a plasma pH equal or above 7.30 is not associated with a decrease in 28-day mortality and the presence of at least one organ failure at day 7 (primary composite outcome). One possible reason, as suggested by Cristian Baicus, is the insufficient power, 28-day mortality being decreased in the sodium bicarbonate group only after multivariate analysis. Interestingly and contrary to what was suggested by Sheldon Magder and Gordan Samoukovic, our trial also shows that sodium bicarbonate infusion is associated with a better 28-day mortality in the prespecified

stratum of patients with moderate to severe acute kidney injury.

Whether the sodium bicarbonate infusion was associated with a reduction in the number of long-term major kidney events is uncertain because data on long-term kidney function were not collected. However, the composite of death and new receipt of renal-replacement therapy during the intensive care unit stay in the overall population was 136 (70%) in the control group and 119 (61%) in the bicarbonate group (p=0.0596).

In our trial, most of the patients presented with hyperlactatemia, and gastrointestinal or kidney loss of bicarbonate were considered as exclusion criteria. Thibaut Baudic and colleagues asked whether a lower chloride load in the sodium bicarbonate group could have been an important mechanism associated with the better outcome observed in the bicarbonate group. Although no definitive answer could be given by this pragmatic trial, chloremia upon enrolment and at 24 h and 48 h was not different between the two groups (appendix figure S5).1

Finally, we absolutely agree with Magder and Samoukovic that our multicentre, randomised trial suggests for the first time that, although widely accepted,2,3 severe acidaemia per se might be controlled by sodium bicarbonate infusion along with its mechanism-specific treatments, rather than being a reflex indication for dialysis. More studies certainly need to be done to better assess this hypothesis in the critically ill population presenting with moderate to severe acute kidney injury and severe acidaemia.

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