Correspondence

Epidural anesthesia and splanchnic perfusion

To the Editor:

Piper *et al.* reported that during and after aortic surgery, gastric intramucosal pH (pHi) showed a similar decrease from baseline in patients with and without epidural anesthesia. CO₂-gap was also comparable between groups.¹

These findings are in disagreement with the majority of studies on the effects of epidural anesthesia on splanchnic perfusion during abdominal surgery. Most studies reported beneficial effects of epidural anesthesia, resulting in increased regional blood flow or gastric pHi.¹⁻³

Experimental work using intravital microscopy also demonstrated that thoracic epidural anesthesia increases gut mucosal blood flow.⁴ Others reported that during lumbar epidural blockade, intestinal vasoconstriction resulted due to a compensatory increase in splanchnic sympathetic activity.⁵ Therefore, beneficial changes in gastrointestinal mucosal blood flow (and pHi, or CO₂ gap) cannot be expected if the epidural results in anesthesia *outside* the area of interest.

Usually, investigators who demonstrated evidence for improved splanchnic blood flow during epidural anesthesia performed measurements *inside* the anesthetized region.²⁻⁴ In the study by Piper *et al.*, epidural catheters were inserted at L3-4.¹ Therefore, the anesthetic block did not include those sympathetic nerves that supply the gastric wall. The site of pHi measurement and the location of epidural anesthesia were different, thus explaining why epidural anesthesia failed to produce beneficial effects on gastric intramucosal pHi, or CO₂ gap.

Obviously, the authors missed this important point. With all their patients having received lumbar epidural anesthesia only, the conclusion that epidural bupivacaine has no effect on splanchnic microvascular hemodynamics and pHi was inappropriate.

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References

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REPLY:

Sielenkämper *et al.* criticize two aspects of our study: firstly, that the results with respect to gastric pH were foreseeable and thus our study was superfluous. Secondly, they assume that we concluded that "epidural bupivacaine has no effect on splanchnic microvascular hemodynamics and pHi" and they emphasized that this conclusion is inappropriate.

Concerning their first comment, they base their argument on theoretical assumptions on the effect of lumbar vs thoracic epidural anesthesia (EA) on splanchnic perfusion and on four studies, of which two were done in sham-operated animals and two during intestinal surgery in humans. These studies are definitely different from the aortic surgery setting (where patients present with vascular pathology and undergo aortic cross-clamping) and, consequently, cannot be used to predict the results of our trial. Furthermore our study did not focus on gastric pH, but analysed regulators of circulation and hemodynamic responses as well. Publications on EA and regulators of circulation in aortic surgery are rare.¹⁻⁴ At the time the trial was designed, no study on the influence of EA on gastric pH in aortic surgery had been published. A single other article has focussed on these patients since then.⁵ Väisänen *et al.* reported similar results concerning the inability of thoracic EA to influence gastric pH.

Concerning their second criticism, Sielenkämper *et al.* misquote us. We wrote: "It is concluded that periop-