

away from ischemic areas, referred to as “countersteal.”^{2–4} On the other hand, if you decrease the partial pressure of carbon dioxide, constricting the noninvolved cerebral areas, you may not increase blood flow to the ischemic areas because they are limited already by the thrombus, which occludes the lumen, and you may cause the noninvolved areas to become relatively ischemic.^{3,5}

We still think that the overwhelming evidence from stroke management and from this paper is for maintenance of systolic blood pressures more than 140 mmHg and less than 200 mmHg as the best strategy to provide cerebral perfusion to ischemic brain through whatever collaterals may be available. Davis *et al.* did not provide any data related to end-tidal partial pressure of carbon dioxide.⁶

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Lipid Emulsion Recommendations

To the Editor:

We read with interest the work of Ruan *et al.*¹ in the February 2012 issue of *ANESTHESIOLOGY*; the article explored the effect of both triglyceride chain length and pH modulation on lipid sequestration of cardiotoxic local anesthetics in human serum *in vitro*. The authors are to be lauded for their efforts to expound the physicochemical interaction known widely as the “sink,” purported to be primarily responsible for the beneficial effects demonstrated in animal models and human subjects suffering local anesthetic systemic toxicity. Their

results in many respects epitomize the difficulties associated with forwarding a therapy such as lipid rescue – itself a product of a chance laboratory observation, when definitive knowledge of mechanistic action for lipid remains to be fully elucidated.

Ruan *et al.* demonstrated the superiority of mixed medium and long-chain triglyceride preparations in sequestration of lipophilic local anesthetics, seemingly independent of pH, when compared with long-chain triglyceride *in vitro* human serum. These results, nevertheless, conflict with the findings of Li *et al.*² (from the December 2011 issue of *ANESTHESIOLOGY*), who demonstrated advantages with long-chain triglycerides in an intact animal model. The observed disparity in outcomes between such bench-top and whole animal experiments was discussed expertly in an accompanying editorial.³

Although the advancement of any therapy is necessarily paved with conjecture and discourse, such as evidenced in microcosm with these two papers, conclusions drawn from such work must be tempered against the findings of prior investigators’ and the associated relevant (and, in the case of lipid therapy, substantial) bodies of work. It is therefore concerning that in their concluding remarks Ruan *et al.* “call into question the current advanced cardiac life support guidelines specifying use of a long-chain triglyceride emulsion” in local anesthetic systemic toxicity on the basis of their findings alone, before the existence of a body of work supporting alternative lipid emulsion preparations as clearly superior. The work of Ruan *et al.* clearly represents one step of many in the evolution of lipid emulsion therapy. Their results, however, are insufficient to alter current recommendations⁴ for lipid infusion in local anesthetic systemic toxicity.

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