has critiqued, we are not referring to a particular statistical model and consequently (but perhaps ambiguously) using "parameter" synonymously with "measurement". For the sake of clarity we would therefore be content to replace "parameter" with "measurement" in the locations Dr. Goodman has highlighted.

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Convulsions after the administration of high dose ropivacaine following an interscalenic block

To the Editor:

Mardirosoff *et al.* describe the occurrence of convulsions after the administration of high dose ropivacaine following an interscalene block.¹ This interesting case merits further documentation and comments to increase its educational value. The delay of 20 min between the end of the administration of ropivacaine and the convulsions argues against an *iv* injection. Therefore, it will be interesting to know if the authors observed premonitory signs (such as dizziness or incoherent speech) before convulsions. Indeed, it seems the sequence of the different phases of central nervous system intoxication are better individualized with ropivacaine,² which is usually not the case with bupivacaine. Furthermore, did the authors make a second blood drug measurement in order to build a pharmacokinetic model and, by extrapolation, estimate the ropivacaine blood concentration at the time of the convulsions? In a case reported previously with severe cardiac toxicity after sciatic block with ropivacaine,³ we evaluated by extrapolation the total and unbound plasma concentration of ropivacaine to be 8.8 and 1.8 mg·L⁻¹ respectively, at the time of the critical event. Indeed, a "grey" zone remains between the appearance of central nervous system toxicity and slight changes in EEG recordings with venous total plasma concentration of ropivacaine of 2.2 mg·L⁻¹ (range 0.5–3.2) as reported by Knudsen *et al.*⁴ and the development of severe cardiac toxicity. The ropivacaine plasma concentrations in Mardirosoff's case together with ours³ and those known in the literature^{4,5} could add valuable information.

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REPLY:

We thank Ekatodramis and Borgeat for their interesting comments.

We did not observe any premonitory signs before the convulsions took place; neither did we perform a second blood drug measurement.

Nevertheless, unlike the case they reported,¹ the total venous concentration at the time of convulsions was probably lower than 2.09 mg·L⁻¹ which, in our case, was the concentration measured one hour after the injection. Indeed, studies on ropivacaine pharmacodynamics suggest that 45 to 60 min is the average time to achieve maximal plasma concentrations after a plexus block or a wound infiltration.^{2,3} The 2.09 mg·L⁻¹ concentration measured in our patient is consistent with concentrations observed in the aforementioned studies, in which such severe central nervous system (CNS) toxicity was not observed.

Moreover, Scott showed that the rate of absorption was better correlated with CNS toxicity than the peak plasma concentration.⁴ This might also constitute another causal factor in our case, since a faster rate of local anesthetic absorption has been demonstrated in patients with chronic renal failure.⁵

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