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Preliminary evaluation of lipid emulsion on electrophysiologic effects of ropivacaine. Study in an experimental porcine model of ropivacaine intoxication

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Background and Goal of Study

Ropivacaine is considered a less cardiotoxic agent in comparison with other long duration amide local anesthetics. There have, however, several case reports in which significant cardiac side effects have occurred after ropivacaine administration. Recently, experimental studies suggest that lipid emulsions are effective in reversing local anesthetic cardiac toxicity. The so-called â€œlipid sinkâ€ effect, is suggested to be the basic mechanism of this treatment and its efficacy varies depending on the physico-chemical properties of the local anesthetics. In our knowledge no studies have evaluated the efficacy of intralipid in reversion the electrophysiologic effects of ropivacaine.

Material and methods:

Eleven Large-White pigs were premedicated with ketamine and anesthetized with intravenous sodium thiopental 5 mg/kg. The anesthetic maintenance was performed with sevoflurane (2.6%). Femoral artery and vein were canalized for invasive monitoring, analytical blood gas samples and ropivacaine levels determinations. After instrumentation and monitorization, a ropivacaine bolus of 5 mg.kg-1 was administered. We aim to induce an intense ventricular conduction slowing and myocardial contractility depression without provoking asystole. The animals were randomly assigned to two groups: control group (C-group, n=5) and intralipid group (IL-Group, n=6). Ventricular conduction (evaluated by QRS duration) was measured in sinusal rhythm and after ventricular pacing at cycle length of 400 ms on the baseline, 3 min after ropivacaine administration and 1, 5,10 and 30 minutes after Intralipid infusion (1.5 mL/kg over 1 minute followed by an infusion of 0.25 mL/kg/min). In C-group a saline infusion was administered instead of Intralipid. Statistical analysis: Mann-Whitney test and Wilcoxon test as appropriate.

Results: Ropivacaine induced an intense slowing of ventricular conduction and use-dependence effect, manifested in sinusal and in stimulated QRS. QRS duration in sinusal rhythm increased from 67Â±9 ms to 101Â±18ms (p=0.003) and stimulated QRS increased from 95Â±7 ms to 359Â±65 ms after ropivacaine administration (p=0.003). Lipid infusion increased the recovery of the enlargement of the QRS in sinusal rhythm in the first minute: 16% in IL-Group vs. 5% in C-group (p=0.05); Figure 1. We did not detect significant differences in the sinusal QRS interval or in the stimulated QRS interval at 5, 10 and 30 minutes of IL administration between the two groups.
Conclusion: Ropivacaine infusion was associated with an intense slowing of ventricular conduction and a market use-dependence block. There was a transient rapid recovery in sinusal QRS interval seen in the intralipid group at 1 min of its administration. However, this difference was no sustained on the rest of timepoint of the experiment. More research is warranted to define the role of intralipid on ropivacaine cardiac toxicity.