Preliminary evaluation of intralipid vs. amiodarone for the prevention of bupivacaine induced ventricular arrhythmias in an experimental porcine model

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Background and Goal of Study

The current management of local anaesthetics (LA) induced arrhythmias remains uncertain. A survey of the practice among anaesthesiologist showed that they would choose amiodarone for the treatment LA induced ventricular arrhythmias (VA). Currently, lipid emulsions are considered the main treatment for bupivacaine (B) toxicity, including VA. However there is limited information regarding their efficacy on B induced VA. Our aim was to develop a model to study VA in the setting of a B intoxication, and evaluate if either intralipid or amiodarone have protective effect on these arrhythmias.

Material and methods:

Sixteen Large-White pigs were premedicated with ketamine and anesthetized with intravenous sodium thiopental 5mg/kg and sevoflurane (2.6%). Femoral vessels were canalized for invasive monitoring, analytical blood gas samples and B levels determinations. Two quadripolar catheters were used for stimulation and intracardiac recordings of the high right atrium and the right ventricular apex. After instrumentation, the animals were randomized into three treatment groups that received saline (n=6), intralipid (n=5) or amiodarone (n=5) three minutes after B administration (4 mg.kg-1 followed by an infusion of 100 ᵅg.kg-1.min-1). A modified programmed ventricular stimulation protocol (PVSP) was performed (at baseline and after 15 min of B perfusion). Ventricular pacing was set on maximal current strength at three basic cycle lengths (350, 400 and 600 ms). After an 8-beat pacing train, PVSP was initiated with coupling intervals of 290, 280, 270, and 260 ms for the first through fourth extrastimuli.

Results:

At baseline, only one animal developed VA after the PVSP. Following B administration, sustained ventricular tachydysrhythmias occurred with PVSP in 5 out of 6 animals in the saline group. In the intralipid group, one animal developed asystole, and the rest (4) presented paced-induced VA during B infusion. In amiodarone group, one animal was excluded due to technical problems; the rest of the animals (4) developed B-paced-induced VA. The arrhythmias observed were mainly ventricular flutter, ventricular fibrillation and polymorphic ventricular tachycardia.

Conclusion:
This study shows a novel and reliable experimental model useful to evaluate B-pace-induced VA. Our preliminary data shows that neither intralipid nor amiodarone prevented paced-induced VA in the context of a porcine experimental model of B intoxication.