24. THE CARDIOTOXICITY OF LOCAL ANESTHETICS

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Introduction. Local anesthetic toxicity is, occasionally, the cause of severe cardiovascular complications. The long-acting local anesthetics as bupivacaine, levobupivacaine and ropivacaine have their toxicity mostly attributed to high plasma concentrations. The aim of this review is to single out the mechanisms of local anesthetic induced cardiotoxicity and treatment using lipid emulsion.

Aim of study. Long-lasting local anesthetic utilization is bounded by possible arrhythmias and contractile depression, potentially leading to cardiac arrest. One of the main mechanisms of anesthetic-induced cardiac depression is thought to be blocked Ca2+-channels in myocardial tissue.

Methods and materials. The analysis of a range of publications from PubMed and NihGov databases, selected according to the keywords: „cardiotoxicity", „anesthesia", „cardiolipin", „bupivacaine".

Results. In vivo studies have revealed bupivacaine to be a negative inotropic agent that causes significant decreases in blood pressure and heart rate through alterations in electrical excitability of the heart, dilatation of blood vessels and inhibition of the firing rate of the sinoatrial node. In vitro studies illustrated that bupivacaine more severely dysregulated calcium dynamics than ropivacaine. Calcium supplementation improved tissue contractility and restored normal beating rhythm for bupivacaine-treated tissues. Calcium channel blocker nifedipine coadministration with bupivacaine, but not ropivacaine, exacerbated cardiotoxicity, supporting the role of calcium flux in differentiating toxicity.

Conclusion. Although many anesthetics can cause cardiotoxicity, bupivacaine demonstrates a higher toxicity risk, adversely altering cardiomyocyte calcium dynamics in a functional human-derived tissue model. The same effect was not observed with ropivacaine, where toxicity risk is lower. Current cardiotoxicity treatments are restricted to cardiopulmonary support and intravenous lipid emulsion administration. Fatty acids would increase the flux of acylcarnitines into the mitochondrial matrix of cardiomyocytes and re-enable oxygen–energy coupling in cardiac tissue.