Lidocaine Alleviates Remifentanil-Induced Hyperalgesia by Inhibiting CaMKII Phosphorylation of Primary Somatosensory Cortical Rat Neurons

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Background: Previous clinical studies have shown that lidocaine alleviates severe postoperative pain after remifentanil-based anesthesia. Experimental studies have demonstrated that lidocaine may inhibit remifentanil-induced hyperalgesia, yet the mechanism is still unknown. The present study tested whether lidocaine inhibits CaMKII phosphorylation in rat neurons in the setting of remifentanil-induced hyperalgesia.

Methods: Male Sprague-Dawley rats were allocated into 3 groups randomly: remifentanil only (R), lidocaine only (L), and remifentanil+lidocaine (RL). Via intravenous tail vein (24G trocar), animals received remifentanil at 2.4 μg/kg/min (R), lidocaine at 200 μg/kg/min (L) and remifentanil at 2.4 μg/kg/min plus lidocaine at 200μg/kg/min (RL) for 2 hours. Withdrawal threshold of both mechanical and thermal hyperalgesia, immunoblotting, and immunofluorescence were used to measure remifentanil-induced hyperalgesia and CaMKII phosphorylation.

Results: There was a significant decrease in withdrawal threshold of mechanical stimulation at 0.5 and 2 hours post-infusion in Group R (P<0.05), but not in thermal withdrawal threshold. There was a significant decrease in CaMKII phosphorylation in groups L and LR compared to group R (p<0.05), as determined by immunoblot and immunofluorescence microscopy.

Conclusion: These results indicate that CaMKII phosphorylation may in part contribute to remifentanil-induced hyperalgesia, an effect that was inhibited by lidocaine. In conclusion, application of lidocaine may reduce remifentanil-induced hyperalgesia in rats by inhibiting CaMKII phosphorylation of primary somatosensory cortical rat neurons.

Reference: