No Pain, All Gain – Spinal Capsaicin and Bupivacaine For Prolonged Sensory Selective Block In Rats

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Introduction: Although the goal of local anesthetics (LA) is to block the transmission of signals in nociceptors to prevent pain, currently available LAs not only block sensory fibers, they also block motor and sympathetic fibers. The ability to selectively block sensory fibers, without causing hypotension and/or motor blockade could clearly lead to a major breakthrough in regional anesthesia. In prior studies we demonstrated that the co-administration of capsaicin with a LA around peripheral nerves elicits a prolonged nociceptive-selective nerve block. (1) The purpose of this study is to determine if the intrathecal injection of bupivacaine and capsaicin can create a prolonged sensory block, without prolonging motor block.

Methods: Sprague-Dawley rats (weights 250-300 g) were assigned to 3 treatment groups (n= 6-8 per group). Rats underwent spinal injection with either bupivacaine alone (B), bupivacaine followed by capsaicin after 1 minute (B+C) or capsaicin alone (C). Motor function was assessed by observation of tail drop. Nociception was evaluated by the nocifensive withdrawal reflex evoked by pinch of the tail. The animals were sacrificed on day 7 and spinal cords were sent for histopathology.

Results: There is a significant prolongation of the sensory block (130 min vs 77 min; p= 0.0067) and significant shortening of the motor block (38 min vs 83 min; p= 0.003) in the B+C group when compared to the B group. (see figure) There was no sensory or motor block in the C group. All of the animals recovered without neurologic deficit. Histopathology results are pending.

Conclusion: Our data show that the intrathecal injection of the combination of bupivacaine and capsaicin produces a prolonged differential block, without causing neurologic deficit. Several potential mechanisms could explain our findings. Capsaicin selectively binds to TRPV1 which is highly expressed in pain-transmitting C-fibers. Capsaicin may facilitate the entrance of bupivacaine into nociceptive fibers through TRPV1 channels (2), however this does not interfere substantially with traditional transmembrane crossing of LA into motor fibers. Alternatively, the activation of TRPV1 cells by capsaicin renders the cytoplasm of C-fibers more acidic (3) and therefore increases the charged form of the LA, which is generally more potent than its neutral counterpart and leaves the cell more slowly.

1. Anesthesiology 2008;109:872-878