The effect of intraperitoneal and intrathecal cobalt chloride administration on formalin-induced orofacial pain

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Abstract

Introduction: The exact mechanism by which cobalt chloride (CoCl₂) exerts its effects is unclear. Suggested hypotheses include mitochondrial toxicity, ATP synthesis inhibition and reactive oxygen species production, mechanisms that are implicated in migraine and chronic pain. The aim of this study was to assess the effect of single-dose CoCl₂ administered via central (intrathecal i.t.) versus peripheral (intraperitoneal -i.p.) route on formalin-induced orofacial pain (OFP).

Materials and methods: Male BALB/c mice were divided in two groups that received CoCl₂ (i.p. - 25mg/kg b.w. or i.t. - 0.025mg/kg b.w. administration) and two groups that received saline. Three hours later, mice received 20μL formalin into the right whisker pad; the time mice spent rubbing/licking the injected area was recorded. The results are expressed as percentages of inhibition.

Results: Both routes of CoCl₂ administration induced a significant decrease in pain behavior in phase one of the OFP test, with percentages of inhibition (PCIs) of over 30%. In the second phase, the decrease remained significant for both i.t. and i.p. CoCl₂ groups, but the decrease after central administration was more important than after systemic administration (55.8% vs. 27.4% PCIs). For both administrations the CoCl₂ groups were statistically different from control groups (p<0.005).

Conclusions: Our initial hypothesis was that CoCl₂ would increase pain-related behavior. However, CoCl₂ had a pronounced central effect on orofacial pain, with anti-inflammatory-like consequences. This may be secondary to CoCl₂’s ability to induce neurotransmission inactivation by reducing Ca²⁺ pre-synaptic influx (synaptic blockade).

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