THE EFFECT OF INTRAPERITONEAL AND INTRATHECAL COBALT CHLORIDE ADMINISTRATION ON FORMALIN-INDUCED OROFACIAL PAIN

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Introduction: The exact mechanism by which cobalt chloride (CoCl2) exerts its effects is unclear. Suggested hypothesis 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/liking 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 (p0.005).

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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