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## Magnesium sulfate as an analgesic in the inflammatory pain – preclinical findings

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Adjuvant analgesics are commonly used in the treatment of chronic painful conditions. Magnesium is recognized to antagonize *N*-methyl-D-aspartate (NMDA) receptor channels and block calcium influx. Magnesium has been demonstrated analgesic efficacy against neuropathic pain, but data on the inflammatory pain are few and controversial.It enhances the analgesic effects of opioids, general and local anesthetics. We investigated whether magnesium sulfate affects somatic and visceral inflammatory pain and whether antinociceptive doses of magnesium sulfate cause motor impairment and/or sedation. Male Wistar rats were used. In a model of carrageenan-induced mechanical hyperalgesia magnesium sulfate had no effect when injected locally into the inflamed paw. However, subcutaneous magnesium sulfate, given in the pretreatment (before induction of inflammation) at doses of 0.5-30 mg/kg, reduced the hyperalgesia by 24.6±7 to 68±8%. Magnesium sulfate at doses of 5 and 30 mg/kg given as a treatment (2h after the induction of inflammation) produced anti-hyperalgesic effect of 43.7±6 and 27.3±4%, respectively. In acetic acid-induced writhing test magnesium sulfate given subcutaneously at doses of 1-15 mg/kg decreased the number of writhing by 49.8±10 to 77.4±2%. Higher doses of magnesium sulfate did not affect the number of writhing. In both models of pain magnesium sulfate demonstrated analgesic effect in a dose-independent manner. In a rotarod test, magnesium sulfate did not disturb motor performance in rats. Considering a therapeutic perspective, magnesium sulfate may be useful analgesics in the management of somatic and visceral inflammatory pain, at doses that do not induce motor impairment. Magnesium sulfate is effective against somatic inflammatory pain after systemic, but not after local peripheral administration. Systemic magnesium sulfate has anti-hyperalgesic effect in both the early and late phase of inflammation.

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## Pain medication, limitations and how to not get manipulated by your patient

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Pain patients come to the doctor expecting medication to relieve their pain. They have also learned to expect that their dose of medication will have to be increased with time as their body becomes "used to" the medication. Patients and disability adjusters expect the dose of medication to be sufficient for patients to be able to ignore their "problem" and function well enough to hold a job. On the other side we have learned that combinations and high doses of these medications can be lethal, and regulatory agencies have acted aggressively to protect the public from both well meaning physicians and "pill mills." Truth is known however, narcotic pain medication in reasonable doses does not work well enough to stop severe pain and allow activity for employment. Doses of medication that are high enough to work well can be fatal, and equally important, there is a reason for narcotic medication to show decreasing efficacy with time, and it is not tolerance. Usual doses of medication should not have to be increased with time to maintain efficacy. Surprisingly, the great equalizer is our patient's choices of food. Indeed there are foods that not only increase inflammatory pain; they also decrease the efficacy of narcotic pain medication. When these foods are discontinued, narcotic medication resumes its efficacy and lower doses are again effective for reducing pain. With this information, the doctor can put more of the burden of pain relief back on to the patient, expecting medication to work better, and at lower doses. Learning to teach patients that exacerbations of pain are due to food choices more than physical activity and insufficient doses of pain medication reduces patient manipulation and improves patient outcomes.

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