During patient-controlled demands for analgesia cannot be discounted. Further studies, we believe, should concentrate on this period and perhaps in addition measure serum drug concentrations in an attempt to achieve a correlation and produce a definitive answer. In the meantime, our study suggests barbiturate hyperalgesia to have little clinical relevance.

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References

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Difficulties Encountered in a Comparative Study of Orally Administered Midazolam and Ketamine

To the Editor:—In children, psychological preparation along with effective premedication may help alleviate psychobehavioral sequelae resulting from the surgical experience. Many premedications administered via various routes exist. Of these, the oral route appears the simplest and least traumatic in children for whom there is no intravenous access. Recent studies have established the efficacy of and have suggested dosing for oral midazolam and oral ketamine in children.1,2 We planned a randomized, prospective, double-blind study to determine if one presented a significant benefit when compared with the other. The study was aborted after undesirable side effects attributable to ketamine were consistently noted.

Our children were between the ages of 3 and 6 yr; five were boys, and one was a girl. All were ASA physical status 1 scheduled for herniorrhaphy or circumcision. No child had a history of psychiatric disorders. Four children received 6 mg/kg oral ketamine, and two children received 0.75 mg/kg oral midazolam in 0.2 ml/kg sweetened fruit-flavored drink. Monitoring included noninvasive blood pressure, pulse oximetry, continuous electrocardiogram, and respiratory rate.

After peak premedicant effect had been attained, the children were taken to the operating room, and inhalation induction was accomplished with 70% nitrous oxide in oxygen, with halothane in 0.2% increments after each four breaths to 1.5% and then 0.5% increments after each four breaths to 3%.

Of the four children premedicated with ketamine, significantly increased secretions during induction leading to desaturation (oxygen saturation by pulse oximetry < 95%) developed in three, who required pharyngeal suctioning, positive-pressure ventilation, and (in two) a small dose of succinylcholine to relieve laryngospasm. All children recovered with no further difficulties. In the fourth child, an acute hallucinatory experience developed approximately 10–15 min after premedication so severe as to require intramuscular midazolam. He had neither recall nor nightmares immediately postoperatively and at follow-up 1 week later.

Oral ketamine at this dose was used by several attending anesthesiologists before the study. One other child had a dysphoric experience treated with rapid uncomplicated inhalation induction, and several reported secretions and airway difficulties similar to those reported above. In addition, in several children an adequate level of sedation was not achieved with 6 mg/kg oral ketamine.

Although ketamine is known to have these side effects,4,5 in previous pediatric studies6,7 with oral premedication they have not been reported. Finally, we realize that the appearance of children premedicated with ketamine was obviously different from those medicated with midazolam, such that a blind study was impossible.

As a result of the issues raised in terms of safety and study logistics, we discontinued the study. The search for an ideal pediatric premedicant continues!

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To the Editor:—The furor about the revised label for succinylcholine (SCh) in part includes amazement at the high mortality rate in these pediatric patients with hyperkalemia.1–4 Most expect successful resuscitation as the K+ is redistributed, even during arrest and cardiopulmonary resuscitation. However, the clinicians caring for these patients with acute cardiac arrest may not have realized that hyperkalemia could have been the proximate cause and therefore followed American Heart Association guidelines that emphasize the lack of improved outcome with the use of calcium salts.3 This choice, to not use calcium during resuscitation, may be the case when there is asystole. Further, the picture is sometimes confused with malignant hyperthermia, and many may then be reluctant to treat with calcium because of a possible triggering action.4 However, this approach is historical and is without a solid scientific basis.

The gradient between extracellular and intracellular calcium concentrations is about 10,000-fold during relaxation and close to 1,000-fold during active contractile activity. Administration of exogenous calcium will do little to alter this gradient, as plasma calcium concentration will still be in the millimole range. Further, several studies document the inability of exogenous calcium to trigger malignant hyperthermia in the genetically pure porcine model, even with extracellular calcium concentrations to 7.5 mm (during cardiopulmonary bypass, because cardiac function is impossible in this situation).7,8 In addition, calcium given during human cardiac arrest and during an episode of malignant hyperthermia can be therapeutic and life-saving.5

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