

## Generalized Extensor Spasm in Infants Following Ketamine Anesthesia

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A thus-far-unreported complication was observed in two infants anesthetized with ketamine for eye examination and right inguinal hernia repair, respectively.

### REPORT OF TWO CASES

**Case 1.** A 2½-month-old infant weighing 4.5 kg was scheduled for eye examination under anesthesia. Pentobarbital sodium, 10 mg, and atropine, 0.05 mg, were given in 45 minutes before the start of anesthesia. Induction of anesthesia was accomplished with ketamine, 10 mg, iv. During the 50-minute procedure additional 5-mg increments of ketamine were given, for a total of 65 mg. In the recovery room the baby was warm, dry, and pink. About 35 minutes after the administration of the last dose of ketamine the baby manifested generalized extensor spasm with opisthotonus. Consciousness had not yet returned. The attacks recurred every 2-3 minutes and lasted for about 10 seconds. Five minutes after administration of pentobarbital sodium, 10 mg, iv, the attacks subsided. The remainder of the recovery was uneventful.

**Case 2.** A 6-month-old infant weighing 6.8 kg was brought to the operating room for the repair of a right inguinal hernia. Atropine, 0.1 mg, was given 45 minutes before induction of anesthesia. The first dose of ketamine was 2 mg/kg, iv. Subsequently 1-mg/kg increments were given as needed. In 65 minutes a total of 130 mg of ketamine was given. The anesthetic course was uneventful. Twenty-five minutes after the last dose of ketamine, the baby developed generalized extensor spasm with marked opisthotonus recurring every 3-4 minutes. The attacks lasted 10-15 seconds. The baby remained warm, dry, and pink during this time. Following the fifth attack, pentobarbital, 15 mg, was given iv. The attacks ceased in about 6 minutes. The baby was sufficiently awake to respond to verbal stimulation in about 45 minutes.

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### DISCUSSION

In a recent editorial Winters<sup>1</sup> pointed out that most agents used for immobilization of patients and production of amnesia for surgical procedures initially produce excitation (stage I) followed by catalepsy and delirium (stage II). Originally, ketamine was thought to belong to those agents, the effects of which did not progress beyond the cataleptic state. The recent description by Thompson<sup>2</sup> of the development of grand mal seizure after administration of ketamine in a young, healthy woman with no previous history of any convulsive disorder indicates that occasionally ketamine may also cause convulsions. The likelihood of this possibility is supported by the work of Mori *et al.*,<sup>3</sup> who found that in cats the ketamine-induced catatonic "anesthetic" state was followed by electrographic seizure without clinical manifestations. It is possible that the generalized extensor spasm and opisthotonus observed in the two infants were accompanied by electrographic seizure activity. Unfortunately, we did not have opportunity to record the EEG at the time of muscular hyperactivity. Similar complications, consisting of increased muscle tone and opisthotonus, were reported by Corsen *et al.*<sup>4</sup> to occur in adults.

The purpose of the presentation of these two case reports is to draw attention to the possibility of similar occurrences in infants who receive relatively large doses of ketamine. Our patients received 14.4 and 19.1 mg/kg, iv, in 50 and 65 minutes, respectively. It is of interest that the electrographic seizure activity found by Mori *et al.*<sup>3</sup> in cats occurs after comparable doses of ketamine.

That relatively small doses of pentobarbital sodium promptly terminated the muscular hyperactivity indicates that stimulation of the cerebral cortex may have been the predominant or at least a contributing factor.

It is important that recovery room personnel be aware of the possibility of this complication of ketamine anesthesia, and that oxygen and short-acting barbiturates be immediately available for its treatment.

## REFERENCES

1. Winters WD: Epilepsy or anesthesia with ketamine (editorial). *ANESTHESIOLOGY* 36:309-312, 1972
2. Thompson GE: Ketamine-induced convulsions. *ANESTHESIOLOGY* 37:662-663, 1972
3. Mori K, Kawamata M, Mitani H, et al: A neurophysiologic study of ketamine anesthesia in the cat. *ANESTHESIOLOGY* 35:373-382, 1971
4. Corssen G, Miyasaka M, Domino EF: Changing concepts in pain control during surgery. Dissociative anesthesia with C-581. *Anesthetic Analg (Cleve)* 47:746-759, 1968

## Correspondence

## Anesthetics and Mitochondrial Respiration

*To the Editor:*—We wish to point out an error in our paper entitled "The Effects of Forane and Fluoroxene on Mitochondrial Respiration: Correlation with Lipid Solubility and *In Vito* Potency" (*ANESTHESIOLOGY* 38:437-444, 1973). The last sentence of paragraph 3 in the discussion (page 441) should read "Succinate oxidation was not inhibited after treatment with high doses, but there was resultant irreversible loss of respiratory control."

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## A Ketamine Trip

*To the Editor:*—

When logic and proportion have fallen starkly dead  
And the White Knight is talking backwards  
And the Red Queen's off with her head,  
Remember what the dormouse said—  
Feed your head. Feed your head.

—Grace Slick, *White Rabbit*, 1967 \*

"Bad trips" are well known to abusers of hallucinogens and are occasionally observed in our anesthetized patients. Since I could find

no detailed description of a "bad trip" after ketamine in the anesthesia literature, I wish to report one which followed 3 mg/kg intravenously, given as part of a respiratory drug study.

I was lying supine on a contour-flexed operating table, a pillow beneath my head. The research laboratory was dimly lit; light classical music played in the background. I was breathing 5 per cent CO<sub>2</sub>, which was not unpleasant, but the mouthpiece did tire my jaw muscles. The investigators were friends, and all-in-all I was comfortable.

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