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Magnesium Sulfate Prevents Succinylcholine-induced Fasciculations in Toxemic Parturients

JAY S. DEVORE, M.D.,* AND RAJKUMARI ASRANI, M.D.†

Toxemia of pregnancy is a common condition and one of the major causes of maternal mortality. Since many toxemic patients need emergency cesarean section, the anesthesiologist must recognize interactions between drugs used to treat toxemia and those used to provide anesthesia. Magnesium sulfate, a compound commonly given to toxemic patients, would be expected to interreact with muscle relaxant drugs.

When emergency cesarean section is done, the patient is considered to have a full stomach. Patients are usually given a 3-mg dose of *d*-tubocurarine before succinylcholine to prevent fasciculations. When this practice is extended to patients who have been treated with magnesium sulfate for toxemia, exaggerated responses have occurred to the point of apnea. This suggests that these patients already have had a partial nonpolarizing type of neuromuscular blockade, and would not have experienced fasciculations as a result of succinylcholine administration even if the *d*-tubocurarine had been omitted. Therefore, we observed magnesium sulfate-treated toxemic parturients to determine whether succinylcholine-induced fasciculations would occur.

METHODS

Subjects of the study were ten toxemic patients who were treated with magnesium sulfate and subsequently underwent cesarean section during general anesthesia. Therapy was instituted with magnesium sulfate, 4 g, iv, during the first hour, and was then maintained by infusing 1 g/hr thereafter. The mean total dose of magnesium sulfate was 13 g, given over a mean period of eight hours. In most cases, infusion of magnesium sulfate was continued until induction of the anesthesia, and in no case was it discontinued more than 30 min prior to induction. Serum magnesium concentrations in blood samples drawn after the induction of anesthesia were determined by the clinical laboratory. All patients were adequately treated with magnesium sulfate by clinical criteria. Anesthesia was induced in all patients by use of thiopental, 4 mg/kg, followed immediately by succinylcholine, 1.5 mg/kg. Cricoesophageal compression was applied, a cuffed endotracheal tube inserted, and anesthesia maintained with nitrous oxide and oxygen. The presence or absence of muscle fasciculations following succinylcholine administration was determined by visual inspection of the patient's extremities and face for approximately 1 min following succinylcholine administration.

TABLE 1. Serum Magnesium Concentrations

	mg/dl	mEq/l
Normal	1.9–2.5	1.5–2.0
Therapeutic	5–7	4–6
Areflexia	10–12	8–10
Respiratory arrest	12–15	10–12

RESULTS

No patient showed any sign of muscle fasciculation following administration of succinylcholine. Anesthetic and obstetric management was in each case uneventful. The mean serum magnesium concentration was 4.3 mg/dl, with a range of 2.9–6.4 mg/dl (see table 1 for equivalent dosages in mEq/l). The serum magnesium concentrations were all above normal, and correlated significantly with the total dose of magnesium sulfate and the duration of magnesium therapy.

DISCUSSION

Magnesium sulfate is still considered the standard primary treatment of toxemia of pregnancy, although it has not been shown to cause central nervous system depression in the clinical dosage.[1] A primary action of magnesium sulfate is its potential to cause blockade at the neuromuscular junction. The normal therapeutic and toxic serum concentrations of magnesium are shown in table 1, demonstrating a low therapeutic

* Assistant Professor, Anesthesia, Obstetrics and Gynecology; Director, Obstetrical Anesthesia.

† Assistant Clinical Professor of Anesthesia, University of California, Irvine, Medical Center, Orange, California.

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Address reprint requests to Dr. DeVore: Department of Anesthesiology, UCI Medical Center, Mail Route 81, 101 City Drive South, Orange, California 92668.

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index. The lower limit of the therapeutic range is 5 mg/dl, and although eight of ten patients in this study had magnesium concentrations below this minimum therapeutic level, none of the levels was so low that fasciculations occurred.

The interactions of magnesium and neuromuscular blocking drugs have been previously studied. Morris and Giesecke[2,3] showed that the effects of *d*-tubocurarine and magnesium sulfate are additive and that *d*-tubocurarine is approximately a thousand times as potent as magnesium sulfate as a neuromuscular blocking agent. Aldrete *et al.*[4] gave healthy male surgical patients 1 g magnesium sulfate intravenously and found that this dose decreased the frequency and intensity of muscle fasciculations following the injection of succinylcholine, as well as preventing the rise in serum potassium that otherwise occurred.

The present study has demonstrated that succinylcholine-induced muscle fasciculations are extremely unlikely to occur in the toxemic patient who has received sufficient magnesium sulfate to increase her serum concentration of magnesium significantly above the upper limit of normal. These patients do not need pretreatment with *d*-tubocurarine before succinylcholine administration to prevent fasciculations.

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Hypoglycemia-induced Seizures in an Infant during Anesthesia

Aubrey Maze, M.B., Ch.B.,* and Stanley I. Samuels, M.B., B.Ch.†

Ketamine hydrochloride is a popular drug for producing immobility during radiation therapy in young children. In certain patients methohexital sodium, 5 per cent, given by deep intramuscular injection has also proven to be a useful agent for this purpose. We present a case in which convulsions occurred following treatment with methohexital sodium.

Report of a Case

A 4-month-old, 6.5-kg male infant had been diagnosed at 8 weeks of age as having a retinoblastoma. He underwent enucleation of the left eye, and radiation therapy to his right eye was begun. His first seven treatments were uneventful. For his eighth treatment an intramuscular injection of methohexital, 65 mg, was given into the anterior aspect of the thigh. Three minutes later the infant fell asleep and was placed on the radiation table for treatment. At that time his blood pressure was 80 torr and heart rate 120/min. Ten minutes after the injection of methohexital and at the end of the treatment, the child had a bilateral tonic clonic seizure, and it was noticed that his eyes rolled backwards. Heart rate was 120/min and blood pressure was 90 torr by palpation. Mild respiratory obstruction and central cyanosis were treated by the use of an oral airway and administration of oxygen by mask. A new bottle of Dextrostix® in which the Dextrostix strips all matched the "O" color block was obtained. The bottle had been stored at approximately 28–30 C. The Dextrostix analysis showed a blood glucose level of 25 mg/dl. The child was given dextrose, 25 per cent, 20 ml. The seizures, which had lasted about 5 min, stopped shortly after the infusion. Analysis of blood drawn at this time showed a calcium concentration of 10.2 mg/dl, normal electrolyte values, and no ketonemia. The temperature was 37 C. A lumbar puncture showed three cells, protein 27 mg/dl, and glucose 48 mg/dl. A blood glucose test performed an hour later showed 109 mg/dl. Within 30 min of the seizure the child was active and behaved normally. Two hours after the seizure the child ate without incident. On further inquiry, it was determined that the mother usually fed the child at about 2 AM each night, but on the evening before therapy the child had slept from 6 PM until just prior to their arrival at the hospital. Hence, the mother had not fed the child for nearly 13 hours. The child subsequently underwent further radiation therapy without problems. Blood glucose levels were periodically checked and found to be normal. To rule out latent epilepsy, an electroencephalogram (EEG) was performed; it disclosed no abnormality. Methohexital sodium, 65 mg, given intramuscularly, did not provoke an epileptiform EEG.

Discussion

Convulsions during anesthesia are extremely dangerous and, unless promptly treated, may lead to a vegetative state.[1] Hence, rapid treatment is of primary consideration, and should be followed by an attempt to reach a diagnosis of the cause of the convulsions. This patient had a seizure following prolonged starvation and the use of methohexital sodium. Clinically, methohexital sodium is associated with involuntary

* Assistant Professor of Anesthesia and Pediatrics.
† Assistant Professor of Anesthesia.

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Address reprint requests to Dr. Maze.