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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52:77–78, 1980

Hypoglycemia-induced Seizures in an Infant during Anesthesia

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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

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Received from the Department of Anesthesia, Stanford University School of Medicine, Stanford, California 94305. Accepted for publication June 17, 1979.
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muscle movements[2]; a literature search has failed to record any tonic–clonic seizure due to this agent. Wilder demonstrated activation of temporal lobe epileptic foci by the use of intracarotid and intravenous administration of methohexital in small doses.[3] Sleep is known to enhance epileptiform activity in psychomotor epilepsy.[4] Therefore, an EEG was performed to exclude this diagnosis.

In this patient the cause of the seizure was hypoglycemia. Over the years, anesthesiologists have commented on the occurrence of hypoglycemia with clinical signs such as lethargy, sweating, pallor, and tremulousness, which accompany the adrenergic response to a rapid decrease in blood glucose concentration.[5,6] Thomas[7] studied blood glucose levels after induction of anesthesia in two groups of children: one group was starved for as long as eight hours and another group was allowed to drink milk until four hours before anesthesia. The study, using 40 mg/dl as the level for hypoglycemia, showed that 28 per cent of children less than 47 months of age and weighing less than 15.5 kg, who had been starved, were hypoglycemic.[8,9] There was no patient with hypoglycemia in the group that had been fed until four hours prior to anesthesia. In neither group was there any sign of regurgitation or vomiting. Also found in the study was the fact that none of the children with confirmed hypoglycemia had clinical signs or symptoms of the condition. Of interest is the case report of a 5-year-old girl who underwent adenotonsillectomy and who convulsed postoperatively. At that time "no glucose was found in the blood."[10]

Once the diagnosis of a hypoglycemic seizure has been made, or even contemplated, speed is of the essence, as repeated seizures can lead to brain injury. Studies of paralyzed animals subjected to repeated seizures have demonstrated that a point is reached when the compensatory factors that increase substrate supply to a convulsing brain cannot compensate, leading to a decrease in ATP.[11] When a pediatric patient has a seizure during or after anesthesia a sample of blood for glucose determination should be obtained and an intravenous infusion of glucose started. The use of a Dextrostix is invaluable for an immediate and relatively accurate estimation. When the patient is suspected to be hypoglycemic, dextrose, 25 per cent, 2–4 ml/kg (0.5–1.0 mg/kg), is given intravenously.[12] Thereafter, one should maintain dextrose infusion at a rate of 0.5 g/kg/hr until the child can maintain an adequate blood glucose value. If, after treatment, one is still unsure of the diagnosis, a full evaluation, including measurements of blood levels of calcium, magnesium, and ketone, lumbar puncture, and EEG, should be performed.

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52:78–80, 1980

V-Lead Adapter

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Tektronix® Models 408, 412, and 414 patient monitoring oscilloscopes were designed to record from conventional limb leads of the electrocardiogram (ECG). One can record a precordial ECG with these monitors by one of several methods that include: 1) a Tektronix 408 or 412 with modification 735D, or type 414 with option 4; 2)modified limb-lead placement[1]; 3) a V-lead adapter (013-0180-01) recently introduced by Tektronix. The modified oscilloscopes have full-lead selectors and will display precordial and limb-lead ECGs. However, one loses the option to record from limb leads when the modified limb-lead and V-lead adapter methods are used.

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Received from the Bureau of Medicine and Surgery, Navy Department. Accepted for publication June 17, 1979.

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