per minute. This rate change did not allow adequate expiratory time and resulted in excessive pressure within the system. Fortunately, in every instance the patient was disconnected before any adverse effects occurred.

After extensive investigation, it was determined that the erratic performance occurred every hour, lasting approximately 5 to 7 seconds each time. Interestingly, this time period coincided with the audiofrequency signals generated by the Simplex clock-setting system which recently had been installed in our institution. This system uses audiofrequency tone bursts, superimposed on the ac current, to reset the clocks in the hospital automatically.

After consultation with the Emerson and Simplex Companies, it was concluded that the most effective and inexpensive solution, in view of the wide distribution of the clock-reset signal, would be to filter the electrical power within the ventilator. Therefore, a harmonic-neutralized constant-voltage transformer of about 400 watts maximum capacity * was added to each of the Emerson units. This transformer can be safely installed by an electrician, using the instructions provided. The total cost, including installation, was approximately $160.00.

Since this modification, the potentially lethal erratic characteristics of the Emerson Postoperative Ventilator have been eliminated. In our opinion, this experience further stresses the need for all manufacturers of patient care equipment to be cognizant of the potential for electrical interactions which can produce erratic function in solid-state control circuits.

ADDENDUM

Since submission of this information for publication, the authors have been advised by the Emerson Company that the control circuitry in all new Postoperative Ventilator units has been modified to filter out high-frequency interference signals.

* Sola Electric, Elk Grove Village, Illinois 60007. Catalogue number CVS-23-22-150.

CASE REPORTS

Administration of Gallamine in the Presence of Renal Failure—Reversal of Neuromuscular Blockade by Peritoneal Dialysis

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The administration of gallamine, a nondepolarizing muscle relaxant excreted entirely by the kidney,[1] is generally contraindicated in the presence of compromised renal function. However, occasionally it becomes mandatory to administer gallamine when other muscle relaxants are contraindicated because of their pharmacologic side-effects. We present a case report of a man who had acute renal tubular necrosis, hyperkalemia, and severe hypovolemic shock, who received gallamine for the above reason. Neuromuscular blockade was reversed promptly by peritoneal dialysis. Several cases in which blockade induced by gallamine was reversed by hemodialysis have been reported.[2-4] This is the first instance in which peritoneal dialysis has been shown to be effective.

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REPORT OF A CASE

A 56-year-old man, was admitted to the hospital with a diagnosis of aortic valvular stenosis. Seven years previously he had undergone debridement of the aortic valve for aortic stenosis. About one and a half years prior to the present admission, he had developed atrial fibrillation and congestive heart failure. From that time he had been retricted to a bed–chair existence.

On February 14, 1969 the patient underwent replacement of the aortic valve, with two hours and two minutes of cardiopulmonary bypass. The pharmacologic responses to both succinylcholine and *d*-tubocurarine were clinically normal during the procedure. Postoperatively, the patient was oliguric and hypotensive, and required epineph-

rine support for approximately 24 hours. He received both cephalothin, 2 g every 6 hours, and streptomycin, 0.5 g every 12 hours, for four days.

Two days postoperatively the electrocardiogram was interpreted as consistent with an inferior myocardial infarction. The patient was still oliguric, and urinary sodium, potassium, and osmolality was 48 mEq/l, 62 mEq/l, and 300 mOsm/l, respectively.

Although the patient was somewhat obtunded, the trachea was extubated on the third postoperative day. On the following day, BUN had risen to 156 mg/100 ml, the patient was lethargic but responsive, and a diagnosis of acute tubular necrosis was established.

On the fifth postoperative day, a peritoneal dialysis catheter was placed transabdominally. As soon as the catheter was in place, dark blood issued from it, and the patient rapidly went into shock. He was brought to the operating room with an irregular carotid pulse at a rate of 120 beats/min and no detectable blood pressure. Since it was considered dangerous to administer either succinylcholine, because of potential potassium release, or *d*-tubocurarine, because of possible ganglionic blockade, a total of 120 mg gallamine in divided doses was administered. Lidocaine infiltration was accomplished prior to abdominal incision and a lacerated vein in the root of the mesentery was located and controlled. Shortly thereafter, the blood pressure rose to 100/60 mm Hg, and the patient was anesthetized with nitrous oxide.

Postoperatively the patient's cardiovascular status was stable except for ventricular irritability, but he was totally flaccid. The pupils, however, did react to light. The patient did not respond to stimuli from a Block-Aid Monitor set at the highest stimulating amplitude. That evening he had an episode of ventricular tachycardia followed by ventricular fibrillation. His circulatory status was precarious from that time until death.

Two days postoperatively peritoneal dialysis was started. There had been no discernible change in neuromuscular transmission as gauged by nerve stimulation. After two dialyses, each of which contained two liters and equilibrated for 90 minutes, stimulation of the ulnar nerve elicited a twitch and tetanic fade. After four dialyses, responses to stimulation of the nerve became normal. The patient was extremely lethargic and did not obey commands, although he responded to his name. He had facial twitching, which was thought to represent seizure activity. Weaning from the ventilator was begun gradually during the next day via a T-piece adapter with a 150-ml extension receiving 10 l/min oxygen. Pa_{O_2} was in the low seventies and Pa_{CO_2} approximately 45 mm Hg. The maximum tidal volume was 500 ml; it was not possible to determine a meaningful vital capacity. Subsequently mechanical ventilation was employed intermittently.

The next day, four days postoperatively, the patient developed a murmur of mitral regurgitation which was thought to represent papillary muscle dysfunction secondary to myocardial infarction. Ventricular irritability increased during the course of the day, and fibrillation developed. Attempts to resuscitate the patient were unsuccessful.

Blood samples before peritoneal dialysis and after four runs were analyzed for neuromuscular blocking activity by the following bioassay: The concentrations of neuromuscular blocking drug in the patient's plasma were estimated in the isolated lumbrical muscle of the guinea pig using the electrical responses of the endplate region to carbachol.[2] The carbachol-antagonizing effects of ultrafiltered plasma samples were compared with those of known concentrations of gallamine. The plasma obtained from the patient during neuromuscular blockade before peritoneal dialysis had a neuromuscular blocking effect equipotent to that of 2.1×10^{-5} M gallamine; the plasma obtained after dialysis, when the block had been reversed, contained the equivalent of 5.5×10^{-4} M gallamine.

Discussion

The patient had no spontaneous reversal of neuromuscular blockade in 48 hours, which is consistent with the known duration of action of gallamine in patients without renal function.[3, 4] The neuromuscular blockade was rapidly reversed by four peritoneal dialyses, which reduced the concentration of the neuromuscular blocking agent in the blood by a factor of 3.8. Such a decrease can easily account for the observed restoration of impulse transmission since small changes in concentration of curare-type blocking agents effect large changes in transmission.[6] The absolute concentrations found in this assay are close to those reported in the literature for transmission block.[7]

Since the patient had received both cephalothin and streptomycin before abdominal exploration, it is possible that part of the neuromuscular blockade was due to these antibiotics. However, since neuromuscular blockade was not clinically evident preoperatively, and was present following the administration of gallamine, it is reasonable to assume that it was the gallamine which was removed by the peritoneal dialysis.

These findings suggest that residual neuromuscular blockade due to administration of gallamine to a patient with impaired renal function may be effectively reversed by peritoneal dialysis.

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Cardiac Arrest Following Succinylcholine in Patients with Central Nervous System Injuries

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The danger of cardiac arrest following administration of succinylcholine to patients with burns,[1, 2] massive trauma,[3] tetanus,[4] and spinal-cord injuries[5-7] has been described. This paper reports this complication in a patient who developed spastic paralysis following severe brain injury and a patient who was quadriplegic following a posterior fossa craniotomy for a medulloblastoma of the brain stem.

REPORT OF TWO CASES

Patient 1: A 20-year-old white man was involved in an auto accident in which he sustained a head injury which resulted in fixed, dilated pupils, deviated eyes, and incoordinated movements of all extremities. On admission to the hospital, the blood pressure was 60 mm Hg systolic and the pulse rate 140 beats/min. Roentgenograms of the chest disclosed no abnormalities. Serum potassium was 3.8 mEq/l. After improvement of the patient's condition with intravenous fluids, a tracheostomy was performed.

Upon transfer to the Intensive Care Unit, the patient remained unconscious, but the vital signs were stable and breathing was spontaneous. Neurologic evaluation revealed generalized, intermittent spasms which resembled grand mal seizures. Brain-stem injury was diagnosed and the patient was treated with steroids and anticonvulsants. Three days later, he developed a left pneumothorax, which was treated with a tube thoracostomy.

Neurologically, the patient improved slowly, but remained spastic. Fourteen days after admission the spasticity was markedly reduced, his eyes opened on command, and he responded to painful stimuli by moving. Twenty-one days after admission, his arms moved with coordination, but some spasticity remained. He had recovered from intermittent respiratory infections and the tracheostomy tube was removed.

On the 26th day after accident, the patient was scheduled for an open reduction of the left humerus. Preoperative medication consisted of meperidine, 35 mg, and atropine, 0.4 mg. After preoxygenation, anesthesia was induced with 125 mg sodium thiamylal. Intravenous infusion of 0.2 per cent succinylcholine was started and intubation of the trachea was accomplished after about 125 mg had been administered. Following this, the patient suddenly became cyanotic and pulseless. An EKG monitor showed ventricular fibrillation. Ventilation with oxygen and external cardiac compression were started immediately. After six minutes of external cardiac compression and administration of intravenous bicarbonate, the heart was defibrillated successfully with the first countershock. Five minutes later, the patient moved and breathed spontaneously, and the trachea was extubated. Serum potassium was 3.8 mEq/l three and a half hours after the cardiac arrest. In the Intensive Care Unit, the patient had mild spasticity, but could move all limbs. He responded to painful stimuli by movement, but did not speak.

Thirty-five days after the accident, the patient was scheduled again for surgery of his left humerus. Premedication consisted of atropine, 0.4 mg, given 45 minutes preoperatively. Anesthesia was induced uneventfully with methoxyflurane and nitrous oxide, and intravenous 0.2 per cent succinylcholine was started. After about 50 mg of succinylcholine, cardiac arrest recurred and the EKG again showed ventricular fibrillation. After intubation of the trachea, ventilation with oxygen and external cardiac compression were instituted. Following administration of sodium bicarbonate intravenously, satisfactory cardiac function with sinus rhythm returned spontaneously within four minutes. Recovery was uneventful and the patient rapidly returned to his preoperative condition.

At this point, the patient was transferred to the

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