ful even though small shunts involving less than 5 per cent of cardiac output may not be detected. The procedure is simple and safe. It is particularly useful in critically ill patients to assess circulation time, cardiac output, and the presence or absence of intracardiac shunts. Our patient had pulmonary embolism followed by development of a right-to-left shunt through the foramen ovale as demonstrated by an indicator dye-dilution curve. Even though the results of hemodynamic measurements were within normal limits at the time of cardiac catheterization, the shunt at the atrial level was confirmed by the indicator dye-dilution curves simultaneously performed. This emphasizes that the difference in pressure between the right and left atria, rather than the increases in the absolute values, is important in causing for shunt.

The authors thank Dr. R. King, Professor of Surgery, for permitting them to report this case, and Dr. Harvey Feigenbaum, Director of Haemodynamic Laboratory and Professor of Cardiology, for the cardiac catheterization and indicator dye-dilution curve studies.

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Prolongation of a Pancuronium-induced Neuromuscular Blockade by Clindamycin

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Several antibiotics augment the neuromuscular blockade induced by nondepolarizing muscle relaxants.[1] There are no reports which suggest that Clindamycin is one of these antibiotics. This case report suggests that a pancuronium-induced blockade was prolonged as a result of intravenous administration of Clindamycin phosphate. Neostigmine was an ineffective antagonist of this blockade, and indeed may have prolonged it.

Report of a Case

A 54-year-old Caucasian woman, 50 kg in weight, was scheduled for an emergency incision and drainage of an abscess of the sigmoid colon. Two months previously, she had received general anesthesia with morphine and nitrous oxide for renal transplantation. Muscle relaxation was provided by pancuronium bromide, 3 mg, which was antagonized completely as judged by the response to peripheral-nerve stimulation by neostigmine, 3 mg, and atropine, 1.2 mg, intravenously. During the operation the patient received chloramphenicol, but not Clindamycin.

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Received from the Department of Anesthesia, University of California, San Francisco, California 94143. Accepted for publication May 9, 1974. Supported in part by USPHS Anesthesia Research Center Grant 5PO1 6M 15571-06 and USPHS Research Training Grant 5T1 GM 00063-16.
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Six weeks later, she was anesthetized with enflurane and nitrous oxide for a sigmoid colostomy. Muscle relaxation during this procedure was produced by pancuronium bromide, 1 mg. Since no neuromuscular blockade was present at the conclusion of this 90-minute anesthesia, as judged again by peripheral-nerve stimulation, no antagonist was given.

The patient's medications before the present procedure were acetaminophen, prednisone, vitamin A, aluminum hydroxide, propoxyphene hydrochloride, chloramphenicol, and cephalexin monohydrate. Hematocrit was 23 per cent, hemoglobin 7.2 g/100 ml, blood urea nitrogen and serum creatinine 9 and 0.5 mg/100 ml, and serum sodium, potassium, and chloride 133, 4.6, and 98 mEq/l, respectively. Serum calcium and magnesium were 10 and 2.1 mg/100 ml, respectively.

Arriving in the operating suite unpremedicated, the patient received oxygen, 6 l/min, and *d*-tubocurarine chloride, 2 mg, intravenously. Eight minutes later she received thiopental sodium, 150 mg, and succinylcholine chloride, 100 mg, intravenously, and the trachea was intubated. Five minutes later, spontaneous respiration began. Anesthesia was maintained with nitrous oxide, 60 per cent, and halothane, 0.9 per cent, inspired concentrations with controlled ventilation. Neuromuscular function was monitored with a peripheral-nerve stimulator, utilizing subcutaneous ulnar nerve stimulation via 22-gauge thin-wall needle electrodes.

Ten minutes after recovery of spontaneous respiration, pancuronium bromide, 2 mg, was administered intravenously; the operation proceeded without difficulty. Fifteen minutes after administration of pancuronium, Clindamycin phosphate, 600 mg, and chloramphenicol, 500 mg, were given intravenously. Over the next 20 minutes halothane was gradually decreased in concentration and then discontinued. Twenty-five minutes after Clindamycin administration (40 minutes after administration of pancuronium), neostigmine, 3 mg, and atropine, 1.2 mg, were given intravenously although no twitch occurred in response to ulnar-nerve stimulation. No calcium or additional anticholinesterase agent was given. Nitrous oxide was discontinued and the patient transferred to the intensive care unit with the trachea still intubated and ventilation controlled.

Eighty-five minutes after pancuronium (45 minutes after administration of neostigmine), the blood pressure was 170/140 torr, pulse 136 beats/min, temperature 37.5 C urinary output 100 ml/hour, and ventilatory rate assisted with a Bird respirator at 20 respirations/min. She was able to nod her head slightly and appropriately in response to questions, but was unable to lift her head off the bed or grip a pencil. A small twitch, fade in response to tetanic stimuli of 30 Hz and post-tetanic facilitation were present. Vital capacity was 0.25 l. During controlled ventilation, Pa_{O_2}, Pa_{CO_2}, pH_a, and base excess were 310 torr, 31 torr, 7.44 units, and minus 1.5 respectively ($Fi_{O_2} = 0.7$).

Four and a half hours after pancuronium, the patient continued to show no significant improvement in her muscle strength as measured by clinical signs and peripheral-nerve stimulation. Vital capacity was 0.35 l. Postoperative blood urea nitrogen and creatinine were 3 and 0.5 mg/100 ml, respectively, with serum potassium, 4.2 mEq/l. Ventilation was controlled for an additional 15 hours.

By 20 hours after administration of pancuronium the patient had regained full muscle strength. A strong twitch with no fade in response to tetanic stimuli of 30 Hz and the absence of post-tetanic facilitation were observed. Her grip was equal to that of the observer, and she was able to lift her head off the bed for 20 seconds. Vital capacity was 1.4 l. After extubation of the trachea, the patient was transferred to the ward, where her recovery was uneventful.

Discussion

This patient received Clindamycin phosphate intravenously and experienced a pancuronium-induced neuromuscular blockade that was prolonged approximately 20 hours. Previously, her response to pancuronium-induced neuromuscular blockade had not been prolonged. Chloramphenicol and cephalexin monohydrate are not a likely cause of the prolonged block because they are devoid of neuromuscular blocking properties.[1-3] Furthermore, chloramphenicol and cephalexin and not Clindamycin were given with the two previous anesthetics, when a prolonged block from pancuronium did not occur.

Clindamycin phosphate is a semi-synthetic antibiotic produced by a halogenated substitution of a hydroxyl group of the parent compound, lincomycin. No study of the neuromuscular properties of Clindamycin has been reported. However, lincomycin has been shown to have neuromuscular blocking properties in experimental animals.[1,2] These properties are additive to those of *d*-tubocurarine. There are no studies in man of the neuromuscular blocking properties of lincomycin. Since clindamycin is related chemically to lincomycin, perhaps it has similar neuromuscular blocking properties.

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