Anesthesiology
63:225, 1985

Drug Security for Emergency Surgical Procedures

To The Editor:—A hospital that serves the needs of trauma patients must be prepared to provide anesthesia at very short notice. In order to do this, invasive monitoring lines are preassembled, and emergency drugs and anesthetic agents are drawn into prelabeled syringes. These intravenous drugs are placed on the anesthesia cart in the operating room. Drugs may be left unattended for a period of hours and are discarded by the anesthesia personnel the following morning. While this has proved satisfactory clinically, it does not comply with security standards as outlined by state and federal authorities (HCFA* and the Texas Department of Health).

During a recent accreditation survey, the Texas Department of Health referees cited the potential for theft and/or substitution of these drugs with the potential for danger to the patient and medicolegal liability of anesthesia personnel and the hospital itself.† Our dilemma was to meet the needs of the unpredictable emergency patient within the security and infection control guidelines of the JCAH,‡ HCFA, and the Texas Department of Health.

Prevention of drug substitution can be provided by a minor change in our customary routine. The drugs are drawn from freshly opened vials into labeled syringes as usual and are then sealed in a nonresealable transparent plastic bag. A suitable bagging appliance is the SEAL-A-MEAL II® (Dazey Products Co., Industrial Airport, Kansas; approximately $25 with a supply of bags), available at discount and department stores. Filled bags are inserted into the machine, and a heated metal bar melts the plastic and seals the bag. This bag then can be identified with time, date, and signature of the preparer. Syringes are visible through the plastic, the bag is easy to open when the emergency patient arrives, and cursory inspection will reveal any attempt to open the bag.

Although theft of the entire bag of drugs has not been prevented, this solution has proved acceptable to our Director of Pharmacy, who believes that it should prevent any substitution of drugs and increase patient safety.

LOIS L. BREADY, M.D.
Assistant Professor of Anesthesiology

MALCOLM D. ORR, M.D.
Professor of Anesthesiology

MARY ANN MOTE, R. PH.
Director of Pharmacy, Medical Center Hospital

The University of Texas
Health Science Center at San Antonio
7703 Floyd Curl Drive
San Antonio, Texas 78284

(Accepted for publication March 14, 1985.)

* Medicare Program Regulation Subpart J. 405.1027(b)(3)—9/79.

† Statement of Deficiencies and Plan of Correction, 45-0213, Bexar County Hospital District, December 12, 1984.

‡ Joint Commission on Accreditation of Hospitals. Administration of Drugs, p. 120. Accreditation Manual for Hospitals, 1985 edition.

Anesthesiology
63:225–226, 1985

Succinylcholine and Polyneuropathy

To the Editor:—We would like to draw your attention to what we feel is an important inaccuracy in Azar's recent review article, "The response of patients with neuromuscular disorders to muscle relaxants."[1] The article states there are no reports of abnormal responses to succinylcholine with peripheral neuropathy.

Fergusson *et al.*[2] has reported four patients with polyneuropathy who had ventricular tachycardia develop after intravenous succinylcholine. Although serum potassium concentrations were not measured, he thought the most likely explanation for the events was transient hyperkalemia. This hypothesis is supported by the fact that all patients had had muscle weakness for longer than 5 months and that in his, and our, experience, no untoward effects with succinylcholine have been observed in the early phase of polyneuritis. Both these observations support the theory that the arrhythmias in his patients were due to hyperkalemia from muscular denervation, as discussed in the otherwise excellent review.

STEPHEN R. HAGLEY, F.F.A.R.A.C.S.
WILLIAM M. GRIGGS M.B.B.S.
Department of Anesthesia and Intensive Care
Royal Adelaide Hospital
Adelaide, South Australia

REFERENCES

1. Azar I: The response of patients with neuromuscular disorders to muscle relaxants: A review. ANESTHESIOLOGY 61:173–187, 1984

2. Fergusson RJ, Wright DJ, Willey RF, Crompton GK, Grant IWB: Suxamethonium is dangerous in polyneuropathy. Br Med J 282:298–299, 1981

(Accepted for publication March 14, 1985.)

Anesthesiology
63:226–227, 1985

Pharmacokinetic Basis for the Dose-dependent Decline in the Neuromuscular Blocking Effect of Gallamine

To the Editor:—Gallamine triethiodide is a neuromuscular blocking drug that has been used clinically for more than three decades. Direct experimental evidence for a dose-dependent decline in the neuromuscular blocking effect (skeletal muscle paralysis) of gallamine in humans is available from the data of Walts and Dillon,[1] as shown in figure 1. It can be seen that muscle paralysis declines at an essentially constant rate, but the rate of decline (or recovery from paralysis) decreases with increasing gallamine bolus dose.

That the dose-dependent decline in the pharmacologic effect of gallamine is related to its pharmacokinetic properties is evident from an examination of the relationship between the rate of decline of neuromuscular paralysis and the apparent rate of decline of (log) gallamine plasma concentration over the same effect range. Walts and Dillon[1] provided pharmacodynamic (effect–time) data for different doses of gallamine from which the average rate of decline of effect can be calculated by linear regression (fig. 1). These authors, however, did not measure gallamine plasma concentrations. The pharmacokinetics (plasma concentration–time profiles) of gallamine have only recently been fully characterized in surgical patients[2] at doses comparable to that used to generate the pharmacodynamic data. Using average pharmacokinetic parameters for gallamine,[2] plasma concentrations of the drug corresponding to the various degrees of muscle paralysis reported by Walts and Dillon[1] can be predicted. These concentrations allow the estimation of the apparent rate of decline (k_{app}) of (log) plasma gallamine concentration over the paralysis range observed with each dose of gallamine. A plot of the average rate of decline of gallamine-induced muscle paralysis as a function of the calculated k_{app} for three doses of gallamine is presented in figure 2. There

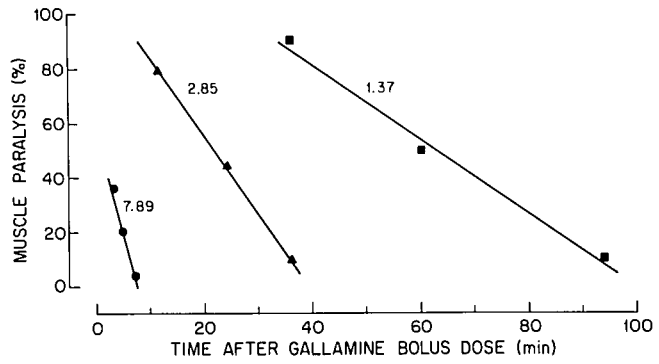

FIG. 1. Pharmacologic effect–time data reported by Walts and Dillon,[1] showing the dose dependence of the rate of decline of the neuromuscular blocking effect (muscle paralysis) of gallamine in humans following three (●—32.2 mg or 18 mg/m^2; ▲—56.5 mg or 36 mg/m^2; ■—128 mg or 72 mg/m^2) different bolus doses of the drug. Each point represents the median value from 20 patients undergoing general anesthesia, while the solid lines represent the linear regression lines. The numbers next to each set of data are the average rates of decline of the neuromuscular paralysis (per cent per min), obtained by linear regression.

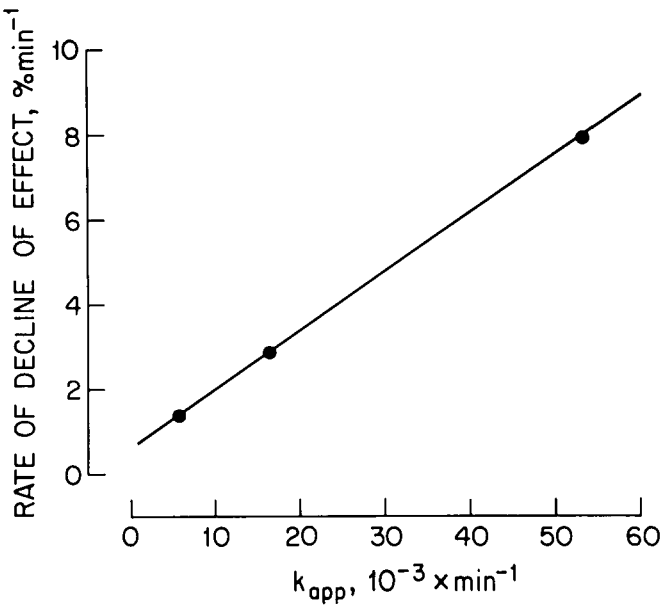

FIG. 2. Relationship between the rate of decline of the neuromuscular blocking effect of gallamine and the apparent rate of decline of (log) plasma gallamine concentration over the same effect range (k_{app}). Individual data points represent data from each dose of gallamine used, while the solid line represents the linear regression line ($r^2 > 0.99$, $P < 0.001$). $k_{app} = 2.3 \log Cp_{max} - \log Cp_{rec}/t$ where Cp_{max} and Cp_{rec} represent gallamine plasma concentrations at maximum (peak) paralysis and at recovery, respectively, and t represents the time interval between these two degrees of paralysis.