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A Controlled Study of the Effect of Succinylcholine Self-taming on Intraocular Pressure

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The management of patients in whom the integrity of an eye is lost or threatened has been the subject of controversy in the medical literature. Researchers using double-blind controlled studies and applanation tonometry instruments have concluded that succinylcholine (SCh) may cause an increase in intraocular pressure (IOP) even though pretreatment with nondepolarizing muscle relaxants has been given.[1,2]

In 1977, Baraka[3] used pretreatment with small doses of SCh to "self-tame" or decrease SCh-induced fasciculations. In an unmasked study using Schiötz indentation tonometry, Verma[4] found that SCh pretreatment prevented SCh-induced fasciculations and increased IOP. The present study of the effect of "tamed" SCh on IOP was blind and controlled; applanation tonometry was used to measure IOP.

METHODS

Twenty patients were anesthetized for a variety of intraocular and extraocular surgical procedures. Premedication consisted of droperidol, 0.1 mg/kg, im, with a maximum dose of 5 mg, and atropine, 0.02 mg/kg, im, with a maximum dose of 0.4 mg, given one and a half to two hours preoperatively. Inhalational induction of anesthesia with halothane–N_2O–O_2 was used for patients less than 14 years old. Older patients received thiopental, 4–7 mg/kg, iv, for induction.

All patients were given halothane, 1.5 per cent, with N_2O–O_2 1:1 during the tonometry preceding tracheal intubation. Ventilation was assisted until administration of the intubating dose of SCh, then controlled. A semiclosed circle system with a CO_2 absorber was employed for patients weighing more than 25 kg. A nonrebreathing system with a gas flow equal to 2.5 times minute volume was used for patients weighing less than 25 kg.

Baseline IOP measurements were made on one eye when the eyelid reflex was absent and the eye was in the primary position. No patient studied had glaucoma or corneal disease. The eye not affected by the surgical procedure was studied in unilateral operations. All patients were in the supine position. Measurements of IOP were performed by one ophthalmologist using a portable Perkins applanation tonometer and topical application of fluorescein with physiologic saline solution.

The study was randomized; ten patients received *d*-tubocurarine (*d*Tc), 0.09 mg/kg, iv, for pretreatment; ten patients received SCh, 0.2 mg/kg, iv., for pretreatment. The ophthalmologist did not know which pretreatment drug was being given. Immediately following the baseline IOP measurement, the pretreatment drug was given iv. One minute later, tonometry was repeated. Then the intubating dose of SCh (1–1.5 mg/kg) was given iv, followed by the final tonometry measurement a minute later.

Statistical analyses were performed utilizing the t test for paired data for comparisons within treatment groups and the Student *t* test for comparison between groups.

RESULTS

Baseline IOP values were similar in the two groups of patients. The value for the SCh-pretreatment group was 12.6 ± 3.3 torr (mean ± SE); for *d*Tc-pretreatment group, 10.7 ± 6.4 torr ($P > 0.4$). Pretreatment with *d*Tc resulted in no significant change in IOP, 9.9 ± 5.5 torr, but SCh pretreatment caused a significant increase, 21 ± 6.6 torr ($P < 0.007$). The IOP values after intubating doses of SCh were similar, for the *d*Tc- pretreatment groups, 22 ± 6.6 torr, and for the SCh-pretreatment group, 21.4 ± 6.2 torr ($P > 0.8$).

DISCUSSION

The present study has confirmed that the "self-taming" dose of SCh in itself may increase IOP. Also, doses of SCh needed for endotracheal intubation (1–1.5 mg/kg) may cause a similar increase in IOP after *d*Tc pretreatment.

Verma also reported increases in IOP following pretreatment doses of SCh, namely 1.5 to 7.5 torr in two

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groups of patients and a mean increase of 8.42 torr in three children with buphthalmos. Like us, he also found no significant change in IOP after the intubating dose of SCh following the self-taming dose. He concluded that SCh caused no significant increase in IOP when compared with IOP *before anesthetic drugs were given.*[4] However, comparisons in the present study are with control IOP obtained *after induction of anesthesia.* IOP may be lowered by central nervous system depressants, including potent inhalational agents, hypnotics, tranquilizers, narcotics, and neuroleptic drugs.[5,6] The difference in conditions under which control pressures were measured probably accounts for the contradictory conclusions reached by this study and that of Verma. Both studies indicate that even after premedication and induction of anesthesia, pretreatment with SCh in a self-taming dose may cause a sudden marked increase in IOP, which conceiveably could cause extrusion of ocular contents in an eye with a penetrating injury.

Use of the Schiötz indentation tonometer was not considered, because it is subject to errors of greater magnitude and variety than those occurring with applanation tonometry. Clinical measurement of IOP (tonometry) depends upon subjecting the eye to a force that flattens or indents it. Schiötz (indentation) tonometry depends on translation of a measured weight-induced corneal indentation into torr by a conversion table, even the most recent of which is said to be in need of improvement. Changes in scleral rigidity may cause falsely high or low readings. The force with which the Schiötz tonometer is applied by the operator affects the readings. There is no endpoint read-out. The plunger that indents the cornea may apply considerable force, which milks out aqueous humor from the anterior chamber and lowers IOP. This effect is measured in tonography, a study to determine the facility of aqueous outflow.

In contrast, applanation tonometry flattens, but does not indent, the cornea. Weight applied to the eye is very small, resulting in minimal changes in IOP. Readings are not affected by rigidity of the ocular coats. Conversion tables are not needed. An endpoint read-out is provided, utilizing corneal patterns, which must be properly centered to obtain an accurate reading. Measurements are nearly as accurate as those obtained by direct cannulation of the eye.[7–9] Repeated tonometry was avoided because of its direct IOP-reducing effect.[10]

We have not given SCh to a patient during open-eye surgery, and have, therefore, not directly observed its effect on the eye. However, we have observed the effect of ocular muscle contraction following suboptimal retrobulbar block with incomplete akinesia, which may be analogous to the SCh effect. These muscle contractions have caused the vitreous to push forward, sometimes being expelled from the eye, even though IOP had been markedly lowered by narcotics, tranquilizing drugs, hyperosmotic agents, and eye massage before the eye was incised.

We have also observed extrusion of ocular contents from open eyes with coughing or straining, even though IOP was lowered preoperatively. Therefore, we do not agree with Verma that when IOP is lowered before SCh is given it is safe to use SCh even though it causes an increase in IOP to the preoperative level. IOP in open eyes is 0 or atmospheric, but ocular contents may be extruded by a sudden force, which in the intact eye causes increased IOP.

TABLE 1. Effect of *d*-Tubocurarine (*d*Tc) Pretreatment on Intraocular Pressure (IOP)

	IOP (torr)		
	Baseline	1 Min after *d*Tc Pretreatment	1 Min after Intubating Dose of SCh
Patient 3	10	11	10
Patient 4	16	14	16
Patient 5	4	5	17*
Patient 10	10	12	30*
Patient 13	11	9	28*
Patient 14	10	8	20*
Patient 15	6	5	20*
Patient 16	9	6	25*
Patient 17	5	6	24*
Patient 19	26	23	30*
MEAN ± SE	10.7 ± 6.4	9.9 ± 5.5	22 ± 6.6†

* >6 torr increase in IOP.

† Significant difference between baseline value and value obtained after intubating dose of SCh, $P < 0.007$.

TABLE 2. Effect of Succinylcholine (SCh) Pretreatment on Intraocular Pressure (IOP)

	IOP (torr)		
	Baseline	1 Min after SCh Pretreatment	1 Min after Intubating Dose of SCh
Patient 1	17	16.5	17.5
Patient 2	16	28.5*	25
Patient 6	12	27*	25
Patient 7	11	22*	24
Patient 8	8	23*	20
Patient 9	13	20*	20
Patient 11	15	27*	25
Patient 12	15	18	28*
Patient 18	7	6	6
Patient 20	12	22*	23
MEAN ± SE	12.6 ± 3.3	21 ± 6.6†	21.4 ± 6.2

* >6 torr increase in IOP.

† Significant difference between baseline value and value obtained after SCh pretreatment, *t* test for paired data, $P < 0.007$.

In conclusion, pretreatment with a small subparalytic dose of SCh before administration of the full paralyzing dose may increase IOP and cannot safely be used for patients in whom the integrity of an eye is lost or threatened.

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Treatment of Unilateral Pulmonary Insufficiency by Selective Administration of Continuous Positive Airway Pressure Through a Double-lumen Tube

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Continuous positive airway pressure (CPAP), when titrated to provide optimal oxygenation without hemodynamic embarrassment, has been shown to be successful in the treatment of acute respiratory insufficiency.[1] In patients with unilateral pulmonary disease and refractory hypoxemia, provision of equal levels of CPAP to both lungs may increase the dead space-to-tidal volume ratio ($\dot{V}_D/\dot{V}_T$) and increase intrapulmonary shunt ($\dot{Q}_S/\dot{Q}_t$) by hyperinflation of the compliant lung and shift of blood flow to the diseased lung by mechanical compression of pulmonary vessels.[2] The use of selective CPAP in the treatment of unilateral pulmonary disease has not been previously reported. We treated a patient with acute unilateral pulmonary insufficiency with a two-CPAP system involving the selective application of optimal CPAP to each lung with the aid of a double-lumen endobrochial tube.

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Key words: Ventilation: continuous positive airway pressure, distribution; Equipment: tubes, endobronchial, double-lumen.

Report of a Case

A 70-year-old man was admitted for treatment of unilateral (right-sided) pneumonia of three days' duration with signs of cough, shortness of breath, diarrhea, fever and chills. During the next 36 hours, his condition became worse and he was transferred to the intensive care unit. The temperature was 39.5 C blood pressure 136/76 torr, and heart rate 110 beats/min. Respiratory rate was 57–64 breaths/min, and accessory muscles of respiration were actively used. Auscultation of the lungs revealed coarse rales, rhonchi, and wheezing over the right lung field. During breathing of 70 per cent oxygen, Pa_{O_2} was 51 torr, Pa_{CO_2} 28 torr, and pH_a 7.50. Repeat roentgenogram of the chest showed right-lower-lobe infiltration and right-upper-lobe consolidation. The left lung was essentially normal except for a slight increase in pulmonary vascular markings. Permission for flexible fiberoptic bronchoscopy was denied by the patient. A nasotracheal tube was inserted and aggressive tracheobronchial toilet instituted. Repeat of the chest roentgenogram showed no improvement (fig. 1).

Because of progressive hypoxemia in the presence of marked tachypnea with the use of accessory muscles of respiration and decrease of lung volume (forced vital capacity = 700 ml), with the diagnosis of acute unilateral pulmonary insufficiency secondary to viral, mycoplasmal or Legionnaires' disease, CPAP was instituted.

A cannula was inserted into the radial artery, and a Swan-Ganz-catheter was introduced percutaneously via the internal jugular vein and was floated into the pulmonary artery. The arterial blood oxygen content (Ca_{O_2}), pulmonary arterial blood oxygen content ($C\bar{v}_{O_2}$), and pulmonary capillary blood oxygen content (Cc_{O_2}) were calculated according to the formula:

$$C_{O_2} = (\text{hemoglobin concentration} \times \text{oxygen saturation} \times 1.34) + (0.0031 \times P_{O_2})$$

The $\dot{Q}_s/\dot{Q}_t$ was calculated from Berggren's formula for shunt: