Succinylcholine Increases Intraocular Pressure in the Human Eye with the Extraocular Muscles Detached

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Background: The increase in intraocular pressure in the human eye that is associated with the use of intravenous succinylcholine has long been ascribed to contraction of the extraocular muscles leading to compression of the globe. This created concern that such contraction would extrude global contents in the patient with an open globe, and led clinicians to avoid the use of succinylcholine in these patients.

Methods: The authors studied 15 patients undergoing elective enucleation, and compared the intraocular pressure change after the administration of succinylcholine in the diseased eye after all the extraocular muscles had been detached to that of the normal eye that had the extraocular muscles attached.

Results: The authors found no difference in baseline intraocular pressure (mmHg) between eyes (15.1 vs. 16.1) or at peak intraocular pressure (25.2 vs. 24.7), but did observe a significant difference in pressure in both eyes when baseline was compared with peak pressure.

Conclusions: The authors concluded that extraocular muscle contraction does not contribute to the increase in intraocular pressure after succinylcholine. (Key words: Eye: aqueous humor; intraocular pressure. Muscles: extraocular muscles. Neuromuscular relaxants: succinylcholine.)

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INTRAOCULAR hypertension in the human eye after succinylcholine administration was first recognized almost 40 yr ago.1,2 The mechanism of action of this change in ocular dynamics remains unknown; however, contraction of the extraocular muscles with distortion and compression of the globe has long been accepted as the etiology of these changes.1,2 The fear that such contraction may extrude global contents3 in a patient with an open eye injury has led clinicians to asidiously avoid succinylcholine in these patients. This theory has never been substantiated and, recently, has been challenged by the reports of both an animal model4 and a series of patients5 with open eye injury in whom intravenous succinylcholine was administered without extrusion of global contents or other untoward effects observed. To explore the contribution of extraocular muscle contraction to the increase in intraocular pressure after intravenous succinylcholine administration, we measured intraocular pressure in a series of patients whose extraocular muscles had been unilaterally severed before elective enucleation, and compared these pressure changes to those of the contralateral eye with the extraocular muscles intact.

Materials and Methods

After we received approval of the Institutional Review board of the New York Hospital–Cornell University Medical College, 15 patients scheduled for elective unilateral enucleation were enrolled in the protocol.

Monitors applied to the patient before the induction of anesthesia included continuous electrocardiogram (Marquette Electronics, Milwaukee, WI) leads II and V, pulse oximeter (Nellcor, Haywood, CA), noninvasive blood pressure cuff (Critikon, Tampa, FL), and axillary temperature probe. End-tidal carbon dioxide (Novametrix, Wallingford, CT) was monitored after the induction of anesthesia.
Anesthetic management was standardized for all patients, with no premedication used. General anesthesia was induced with 3–4 mg/kg sodium thiopental administered intravenously. After loss of lid reflex, each patient’s lungs were ventilated with halothane or isoflurane at concentrations increasing from 0.35–1.5% in 60% N2O and 40% O2 over 5 min, after which tracheal intubation was performed without adjunct muscle relaxant. Mechanical ventilation was established to maintain end-tidal carbon dioxide at 35–38 mmHg. Anesthesia was titrated in response to surgical stimulation with halothane or isoflurane in 60% N2O and 40% O2. Axillary temperature remained at 35.5–35.8°C for the duration of the case.

Using standard dissecting technique, all six extraocular muscles in the diseased eye were isolated and severed, while the optic nerve, vascular, and nerve supply remained intact. The eye was returned to a neutral position and baseline intraocular pressure was then measured in each eye of a single patient using a Perkin’s applanation tonometer (Bio Rad Lab, Cambridge, MA) with strip chart recorder. After these measurements, 1.5 mg/kg succinylcholine was administered intravenously, and intraocular pressure was remeasured and recorded in each eye immediately and every 30 s thereafter for 5 min. Fasciculations were noted in all patients, but were not graded. Inspired anesthetic concentration, end-tidal carbon dioxide, and oxygen saturation were measured and remained constant throughout the study period. Heart rate and blood pressure were recorded simultaneously with intraocular pressure measurements. No medications, other than the anesthetics listed, were administered before or during the study period.

Statistical analysis of the data was performed using Student’s t test to analyze the hemodynamic data, and ANOVA for repeated measurements was used to analyze the intraocular data. A P value < 0.05 was considered to be statistically significant.

**Results**

Fifteen patients were enrolled in the protocol, five adults (age 26–77 yr) and ten children (0.53–3.5 yr). Four patients were diagnosed with ocular melanoma, the remainder with retinoblastoma. Patient demographics are summarized in table 1.

The intraocular pressure at baseline was comparable in the two eyes, whether the extraocular muscles were intact or severed. Intraocular pressure had increased by the first measurement, which was taken seconds after the administration of intravenous succinylcholine, peaked after 90 s in both eyes, and returned to baseline within 5 min. Figure 1 demonstrates these changes in intraocular pressure over time in the eye with the extraocular muscles detached. There was no significant difference in the peak intraocular pressure between eyes, whether the extraocular muscles were intact or severed. There was a significant (F[1,14] = 127.7, P < 0.001) difference in intraocular pressure in both eyes when baseline pressure was compared with peak pressure after succinylcholine administration. There was no difference in the intraocular pressure increase in the adult patients when compared with the pediatric patients; therefore, the intraocular pressure data were pooled and are summarized in table 2.

We observed no significant difference in either the systolic blood pressure or diastolic blood pressure when baseline was compared with the pressure measured at peak intraocular pressure (90 s after succinylcholine administration). The heart rate increased from baseline to 90 s after succinylcholine administration before returning to baseline at 5 min. When the adult and pediatric patients were grouped separately, we found that the heart rate in the pediatric patients showed a significant increase at 90 s; however, in the adult population, the increase was not significant during the same time period. The hemodynamic data are summarized in table 3.

**Discussion**

We observed a significant increase in intraocular pressure after the administration of intravenous succinylcholine that is consistent with measurements reported by others.3,4,6 The change in intraocular pressure that we report was similar, regardless of whether all attachments of the extraocular muscles were intact or

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**Table 1. Patient Demographics**

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>15</td>
<td>19.2 (0.03–77)</td>
<td>29.8 (5.6–106)</td>
</tr>
<tr>
<td>Adult patients</td>
<td>5</td>
<td>53.6 (28–77)</td>
<td>65.86 (55.2–106)</td>
</tr>
<tr>
<td>Pediatric patients</td>
<td>10</td>
<td>1.9 (0.33–3.5)</td>
<td>11.5 (5.6–15.9)</td>
</tr>
</tbody>
</table>

Data are mean; ranges are given in parentheses.
severed. This observation calls into question what contribution, if any, contraction of the extraocular muscles may have on increased intraocular pressure in the human eye after the administration of succinyldicholine.

As in the human eye,1,5 original work in animals had demonstrated that severing extraocular muscles prevented a rise in intraocular pressure. An observation similar to ours in the animal model7 launched extensive exploration to define a mechanism of action for the intraocular pressure increase associated with the use of succinyldicholine after all extraocular muscles had been severed. It was concluded that, when the original work was performed, the dissection technique compromised the blood supply to the eye and prevented succinyldicholine from reaching the eye.7 Advancement of technique allowed the demonstration of an uncoupling of extraocular attachment and intraocular pressure elevation after succinyldicholine administration. This change in intraocular pressure appears to be multifactorial in the cat, but contraction of the nictitating membrane is felt by many to constitute a major factor.7

The human eye has no such nictitating membrane; therefore, another etiology must account for the observed phenomena. The major determinants of intraocular pressure in the human eye include extraocular muscle tone, choroidal blood volume, vitreous volume, and aqueous humor fluid dynamics.6 Vitreous volume is determined by the osmotic environment of the globe. Although we did not measure osmotic pressure directly, we did avoid the use of any hyper- or hypotonic solutions during the procedure; therefore, it is unlikely that a significant osmotic flux could occur during the study period.

Table 2. Intraocular Pressures (mmHg) with Extraocular Muscles Intact or Severed

<table>
<thead>
<tr>
<th>Extraocular Muscles</th>
<th>Baseline</th>
<th>After Succinyldicholine</th>
<th>P</th>
</tr>
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<tbody>
<tr>
<td>Intact</td>
<td>15.1 ± 1.0</td>
<td>25.2 ± 1.6</td>
<td>0.001</td>
</tr>
<tr>
<td>Severed</td>
<td>16.1 ± 1.0</td>
<td>24.7 ± 1.6</td>
<td>0.001</td>
</tr>
<tr>
<td>P</td>
<td>NS</td>
<td>NS</td>
<td></td>
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</tbody>
</table>

Data are mean ± SEM.

Table 3. Hemodynamic Data

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Baseline</th>
<th>After Succinyldicholine</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic pressure (mmHg)</td>
<td>15</td>
<td>98.4 ± 5.5</td>
<td>99.1 ± 5.5</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic pressure (mmHg)</td>
<td>15</td>
<td>66.1 ± 4.5</td>
<td>65.7 ± 4.7</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All patients</td>
<td>15</td>
<td>99.9 ± 7.1</td>
<td>107.4 ± 7.6</td>
<td>0.05</td>
</tr>
<tr>
<td>Pediatric</td>
<td>10</td>
<td>115.0 ± 5.5</td>
<td>123.1 ± 5.5</td>
<td>0.05</td>
</tr>
<tr>
<td>Adult</td>
<td>5</td>
<td>69.3 ± 9.7</td>
<td>75.5 ± 15.3</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data are mean ± SEM.

Table 2. Intraocular Pressures (mmHg) with Extraocular Muscles Intact or Severed

Table 3. Hemodynamic Data
Arterial hypertension causing increased blood flow into the eye will increase choroidal blood volume and, perhaps, raise intraocular pressure. Our measurements of intraocular pressure were performed during steady state anesthesia, with oxygen saturation, ventilation, and arterial blood pressure all unchanged throughout the study period, effectively eliminating arterial hypertension as a possible mechanism of action of increased intraocular pressure. Succinylcholine administration has been associated with choroidal arterial vasodilation and, consequently, increased intraocular pressure. This research demonstrated that this increased flow will raise intraocular pressure only slightly. Consequently, the contribution of an increase in choroidal blood volume after succinylcholine administration to elevated intraocular pressure is minor.

Increased central venous pressure can be transmitted to the episcleral venous plexus to increase outflow resistance and raise intraocular pressure. The only hemodynamic variable that significantly changed during the study period was a slight tachycardia in the pediatric patients. Although we did not measure central venous pressure, given the stability of the other measured variables, it probably remained relatively constant during the study period. A small change in central venous pressure after the administration of succinylcholine has been demonstrated in an animal model; however, it was felt that this change in central venous pressure was of a magnitude that could make only a small contribution to the observed changes in intraocular pressure. We conclude that changes in central venous pressure, similar to those in choroidal artery blood volume, make a minor contribution to elevated intraocular pressure after the administration of succinylcholine.

The final parameter to consider is aqueous humor fluid dynamics. The balance between aqueous humor production and drainage is a primary physiologic control mechanism regulating intraocular pressure. Aqueous humor is constantly produced in the posterior chamber of the eye, and circulates to the anterior chamber to bathe the lens and cornea. It then exits through the trabecular meshwork and the canal of Schlemm to be reabsorbed into the orbital venous system. When this egress of fluid is interrupted, aqueous humor fluid volume will increase and raise intraocular pressure. Previous work has demonstrated a cycloplegic action of succinylcholine with a relaxation of accommodation that will diminish axial thickening of the lens. This action will increase both anterior chamber size and outflow resistance because of decreased tension on the scleral spur from the ciliary muscle. With production of aqueous humor constant, an increase in aqueous humor volume and increased intraocular pressure occurs in the intact globe. The time course of this cycloplegia parallels the metabolism of succinylcholine by pseudocholinesterase, as well as that of the raised intraocular pressure.

All the patients in our study had malignant ocular disease, most often retinoblastoma. Although intraocular tumors can change ocular elastance, baseline intraocular pressure was similar in both the diseased and normal eyes, and was comparable to normal values. The increase in intraocular pressure that we measured after the administration of succinylcholine was also comparable between eyes; therefore, we conclude that intraocular elastance was not significantly affected by the disease state of our patients.

In summary, we measured an increase in intraocular pressure in the human eye, after the intravenous administration of succinylcholine, that was similar, regardless of whether or not the extracocular muscles were attached. We speculate that the increase in intraocular pressure that we observed is multifactorial, but is primarily the result of the cycloplegic action of succinylcholine with a deepening of the anterior chamber and increased outflow resistance relating to decreased tension on the scleral spur. The changes in intraocular pressure that are observed after succinylcholine are of greater magnitude that those observed with pure cycloplegics; therefore, we feel that a slight increase in both central venous pressure and choroidal blood volume further contribute to the increase in intraocular pressure. We propose that changes in extracocular muscle tone do not contribute significantly to the increase in intraocular pressure noted after succinylcholine administration.

References

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