Inhibition of Succinylcholine-Induced Increased Intraocular Pressure by Non-Depolarizing Muscle Relaxants

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The effect of prior administration of a non-depolarizing muscle relaxant on the increased intraocular pressure associated with the use of succinylcholine was studied in 30 human subjects and four cats. When succinylcholine was given alone, ten healthy human subjects had a mean increase in intraocular pressure of 8.5 mm. Hg, and four cats had a mean increase of 11.3 mm. Hg. When given gallamine, 20 mg., or d-tubocurarine, 3 mg., three minutes prior to the administration of succinylcholine, 16 healthy human subjects and four glaucoma patients had no significant increase in intraocular pressure. Four cats, given a small non-twitch-depressing dose of gallamine three minutes prior to the administration of succinylcholine, also had no significant increase in intraocular pressure. This simple, convenient method prevents the increase in intraocular pressure associated with the use of succinylcholine.

Succinylcholine (SCh) administered intravenously in man increases intraocular pressure (IOP), probably in part by contracture of the extracocular muscles.1-3 As a result, the advisability of its use in patients with glaucoma or penetrating wounds of the eye is questionable.4,5 It has been demonstrated in animals that prior administration of d-tubocurarine (dTC) will prevent the contracture of extracocular muscles and subsequent increase in intraocular pressure.4,6 Similarly, Dillon has shown that contraction of human extracocular muscles by SCh can be prevented by dTC.1 The purpose of our study was to determine whether the increased intraocular pressure asso-

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wide-angle type of glaucoma and were treated with pilocarpine. A third patient had congenital glaucoma and a fourth traumatic glaucoma secondary to hemorrhage into the anterior chamber; neither of these were treated prior to surgery. The control IOP of these patients ranged from 18.5 to 80 mm. Hg.

All drugs were given intravenously. The drugs used were gallamine triethiodide, succinylcholine chloride, d-tubocurarine chloride and thiopental sodium.

**Cats:** Four cats weighing 2.0 to 3.0 kg. were anesthetized with chloralose, 60 mg./kg., and urethane, 250 mg./kg., intraperitoneally. A plastic catheter affixed to a 30-gauge luer-lok needle was inserted into the anterior chamber at the limbus. The IOP was recorded on a Grass recorder with a Statham strain gauge (P23AA). A standard peroneal nerve-anterior tibialis muscle preparation described by Bowman was set up to determine the degree of paralysis occurring with each dose of muscle relaxant. Each cat was given a dose of SCh sufficient to decrease isometric twitch tension by 25 to 70 per cent (0.03 to 0.07 mg./kg.). This dose of SCh was repeated at least once to check the consistency of the preparation. In one hour, a nonparalyzing dose (determined by isometric twitch tension) of gallamine, 0.2 mg./kg., was given three minutes prior to administration of the control dose of SCh. Arterial blood gases, carotid arterial blood pressure and temperature were monitored and maintained within normal limits. All injections were given into the jugular vein.

**Results**

**Humans** (Table 1): Five patients had a mean IOP of 9.2 mm. Hg. After thiopental, 25 to 75 mg., and gallamine, 20 mg., the mean IOP was 8.9 mm. Hg. This change was not significant. Ten healthy patients given SCh had a mean increase in IOP of 8.5 mm. Hg, 45 seconds after the beginning of the fasciculations ($P < 0.01$). When SCh was preceded by gallamine, IOP increased 0.9 mm. Hg in ten healthy patients and 1.6 mm. Hg in the four glaucoma patients, neither result being significantly different from control pressures. The mean increase in IOP was 1.0 mm. Hg when dTC was used instead of gallamine. The small doses of gallamine and dTC produced no clinical evidence of antagonism of SCh neuromuscular blockade.

**Cats:** SCh increased the mean IOP from 9.1 to 20.4 mm. Hg ($P < 0.01$). Prior administration of gallamine prevented this change in IOP produced by SCh (fig. 1). No antagonism of SCh neuromuscular blockade by gallamine, 0.2 mg./kg., was observed according to isometric twitch tension.

**Table 1.** Inhibition of Succinylcholine-induced Increased Intraocular Pressure by Non-depolarizing Relaxants

<table>
<thead>
<tr>
<th>Sequence of Relaxant Administration</th>
<th>No. of Patients</th>
<th>Control</th>
<th>2-3 Minutes after Thiopental</th>
<th>Succinylcholine Fasciculations</th>
<th>45 Sec.</th>
<th>2 Min.</th>
<th>4 Min.</th>
<th>5 Min.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinylcholine alone</td>
<td>10</td>
<td>14.7†</td>
<td>(8.5–18.9)</td>
<td>18.6</td>
<td>23.2</td>
<td>21.7</td>
<td>15.4</td>
<td>13.8</td>
</tr>
<tr>
<td>Gallamine plus succinylcholine</td>
<td>10</td>
<td>11.1</td>
<td>(7.5–17.3)</td>
<td>11.0</td>
<td>12.0</td>
<td>10.8</td>
<td>9.7</td>
<td>9.8</td>
</tr>
<tr>
<td>Gallamine plus succinylcholine*</td>
<td>4</td>
<td>31.2</td>
<td>(18.5–50.0)</td>
<td>27.8</td>
<td>29.6</td>
<td>27.4</td>
<td>27.5</td>
<td>26.3</td>
</tr>
<tr>
<td>D-tubocurarine plus succinylcholine</td>
<td>6</td>
<td>12.5</td>
<td>(7.8–17.3)</td>
<td>11.8</td>
<td>13.5</td>
<td>12.5</td>
<td>11.2</td>
<td>10.8</td>
</tr>
</tbody>
</table>

* Patients with glaucoma.
† Mean intraocular pressure in mm. Hg. The numbers in parenthesis represent the range.
INHIBITION OF INCREASED INTRAOCULAR PRESSURE

Discussion

Except for the work of Wretlind and Wåhlin, previous studies demonstrate prevention of the increased IOP associated with the use of SCH by prior administration of dTC in animals. Our studies indicate that gallamine will perform the same function as dTC in cats. In humans, gallamine, 20 mg., or dTC, 3 mg., given three or more minutes prior to the administration of SCH, prevented any significant increase in IOP with SCH. Despite the wide range of control IOP, none of the glaucoma patients had an increase in IOP. The possibility that gallamine or dTC alone lowered IOP is not likely. We were able to demonstrate that gallamine, 20 mg., in combination with thiopental, 25 to 75 mg., had an insignificant effect on IOP. d-tubocurarine has been demonstrated by others to have little effect on IOP.9

Several methods have been recommended for avoiding the increase in IOP accompanying the use of SCH. These include deep anesthesia, slow intravenous infusion of SCH, and prior administration of acetazolamide.11 Using deep anesthesia is feasible but often is inconvenient or undesirable. It is doubtful that slow intravenous infusion of SCH will prevent this increase in IOP.12 Acetazolamide possesses several relative contraindications.11

One of the criticisms of this technique is that gallamine or dTC could antagonize SCH.12 According to clinical impressions in humans and isometric-twitch tension in cats, little antagonism between the small dose of gallamine or dTC and SCH was found.

Although it was not our intent to investigate mechanisms, our data support the findings of those who feel that contracture of the extracocular muscles is the main reason for the increased IOP associated with the use of SCH.1-3 However, recent evidence indicates that the sympathetic nervous system also may be involved.12

We were unable to study patients with penetrating wounds of the eye because of an inability to measure IOP satisfactorily.

Summary

We found that dTC, 3 mg., or gallamine, 20 mg., infused intravenously in patients; or a small non-twitch-depressing dose in the cat, given three or more minutes prior to the use of SCH, will prevent an increase in intraocular pressure. The merits of this method are that it is convenient and simple, and that it also decreases the incidence and severity of muscle pains associated with the administration of SCH.

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References


Surgery

BACTERIAL PERITONITIS Metabolic and hemodynamic changes in acute, diffuse bacterial endocarditis were studied in ten critically-ill patients. The patients were arbitrarily divided into two groups according to arterial pressure. Five patients had systolic pressures below 75 mm. Hg; the other five had pressures above 90 mm. Hg. Cardiac output values in the two groups were similar, and in normal range pulse pressures in the hypotensive group were markedly narrowed whereas the normotensive group had increased pulse pressures, confirming earlier observations that there is poor correlation between pulse pressure and cardiac output. Total peripheral resistance was decreased in the hypotensive group as was urinary output, whereas these were normal in the normotensive group. Total blood volume and plasma volumes were normal in both groups. Four of five hypotensive patients had low pH and elevated $P_{CO_2}$ indicating respiratory acidosis, but there was no significant alteration in pH and $P_{CO_2}$ in the other group. $P_{O_2}$ and $O_2$ saturations were decreased in all patients. All patients had excessive lactate levels, with greatly increased excess lactate in the hypotensive patients indicating tissue perfusion incompatible with life. Normal cardiac output, normal velocity of blood flow, and low or normal total peripheral resistance associated with metabolic evidence of inadequate tissue perfusion suggested a pathological distribution of flow such as arteriovenous shunting. This may have been associated with the diffuse inflammatory response of the peritonitis. The hypotensive group had alveolar hypoventilation. Both groups showed evidence of pulmonary shunting of a significant degree which contributed to the lethality of the disease (eight of ten died; the two survivors were in the normotensive group). The hemodynamic changes differed widely from those associated with gram-negative septicemias where low cardiac outputs, reduced blood flows and increased peripheral resistance usually were found, indicating basic pathophysiological differences in the two entities. (Rosoff, L., and others: Hemodynamic and Metabolic Changes Associated with Bacterial Peritonitis, Amer. J. Surg. 114: 180 (Aug.) 1967.)