loon is inflated by injecting 3–5 ml air through the open female port. When the balloon stays inflated for a reasonable period, it is deflated and the apparatus is prepared for gas sterilization.

When the patient is prepared for epidural anesthesia, the needle is advanced until it is supported by the spinous ligaments. The balloon on the three-way stopcock is inflated with 3–5 ml air from a sterile syringe and the directional arm turned to close the balloon port. The stylet is removed from the epidural needle and the stopcock is attached firmly. The directional arm is then turned to close the open female port, thereby opening the balloon to the epidural needle. When the balloon deflates slowly the needle is probably too superficial. When the needle placement is proper the balloon will stay inflated. Upon entrance into the epidural space the balloon will suddenly deflate, pushing the dura away from the needle point.

We have used this technique on numerous occasions and have found it to be well accepted by the house staff. We have had no accidental subarachnoid puncture.

The balloon stopcock is easy to construct and simple to use. We believe there are several advantages to this technique. It is sensitive, the epidural space is dramatically indicated, and it allows the use of both hands for the control and slow advancement of the needle. There is no need to remove the stopcock from the needle until a continuous catheter has been placed or a single dose of local anesthetic has been injected.

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50:245–247, 1979

# "Self-taming" of Succinylcholine-induced Fasciculations and Intraocular Pressure

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Induction of anesthesia with a thiopental–succinylcholine sequence followed by endotracheal intubation is a commonly used technique. Succinylcholine (SCh) increases intraocular pressure (IOP).[1–7] This effect can be reduced by pretreatment with nondepolarizing muscle relaxants,[3,7] but such pretreatment delays onset of and increases resistance to the SCh-induced block.[8,9]

Baraka[10] reported the "self-taming" of SCh-induced muscle fasciculations by a small dose of SCh used as pretreatment before a subsequent full dose. Utilizing this technique, succinylcholine-induced changes in intraocular pressure were studied.

## MATERIALS AND METHODS

A Schöitz tonometer was used to measure intraocular pressures before and after anesthesia in 25 female patients, 16–40 years old, operated on for gynecologic problems. Lidocaine, 4 per cent, was instilled for corneal analgesia. Premedication consisted of diazepam, 10 mg, im, given 30 to 45 minutes before operation. Atropine, 0.65 mg, was given iv simultaneously with thiopental. Intraocular pressure in each eye was measured: 1) before induction of anesthesia (control); 2) 2 min after administration of thiopental (5 mg/kg); 3) 1 min after the initial dose of SCh (10 mg); 4) 2 min after the full dose of SCh (1 mg/kg); 5) after endotracheal intubation; 6) after return of spontaneous respiration.

In 15 healthy patients (Group I) measurements of intraocular pressure were made as described above. In ten healthy patients (Group II), control readings of intraocular pressure were obtained after giving an additional 10 mg diazepam iv. In addition, in three children with buphthalmos, aged 2 to 10 years, the same technique was followed but diazepam was avoided. The initial dose of SCh given to children was a fifth the total dose. Control values of intraocular pressure could not be obtained in these glaucomatous patients. Effects of this regimen on pulse, blood pressure, and fasciculations were recorded. Post-succinylcholine muscle pains were assessed postoperatively.

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Accepted for publication July 10, 1978.
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0003-3022/79/0300/0245 $00.65 

TABLE 1. Effects of "Self-taming" of Succinylcholine-induced Fasciculations on Intraocular Pressure

| | Number of Patients | Intraocular Pressure (torr) | | | | |
|---|---|---|---|---|---|---|
| | | Control | 2 Min after Thiopental | 1 Min after 10 mg Succinylcholine | 2 Min after Full Dose Succinylcholine (1 mg/kg) | After Return of Respiration |
| Group I | 15 | | | | | |
| Range | | 17–24 | 7.0–17.0 | 8.5–24.0 | 7.0–24.0 | 7.0–17.0 |
| Mean | | 19.51 | 11.27 | 13.8 | 13.6 | 13.9 |
| SD | | 2.32 | 2.42 | 3.48 | 3.67 | 3.77 |
| Group II | 10 | | | | | |
| Range | | 10.0–20.0 | 7.0–12.0 | 8.5–14.0 | 7.0–14.0 | 7.0–12.0 |
| Mean | | 13.9 | 9.0 | 11.0 | 10.8 | 9.32 |
| SD | | 3.43 | 1.54 | 1.41 | 2.45 | 1.9 |
| Group III | 3 | | | | | |
| Range | | | 19.0–34.7 | 26.5–46.0 | 24.7–39.0 | 14.5–29.5 |
| Mean | | | 28.48 | 36.9 | 33.96 | 24.0 |

## RESULTS

Changes in intraocular pressure are shown in table 1. Thiopental significantly reduced intraocular pressures in Group I and Group II, $P < 0.001$. Reductions ranged from 4.5 to 13.0 torr (mean 8.24 torr) in Group I, and from 2.5 to 9.0 torr (mean 4.75 torr) in Group II.

The initial pretreatment dose of SCh did not elicit an increase to above the control intraocular pressure; rather, it was lower ($P < 0.001$). Examination of intraocular pressures after administration of thiopental and after the pretreatment dose of SCh showed significant increases ($P < 0.01$) in the ranges of 1.5 to 7.5 torr (mean 2.55 torr) and 1.5 to 3.25 torr (mean 2.07 torr), respectively, in both Group I and Group II. The subsequent dose of SCh did not increase intraocular pressure in either group, as there was no significant difference between intraocular pressures after the pretreatment dose and those after the subsequent dose.

Measurement of intraocular pressures in 11 patients immediately after endotracheal intubation showed no further increase. Intraocular pressures also remained the same after return of respiration.

Changes in intraocular pressure similar to those in the first two groups were found in glaucomatous children. The average increase after the initial dose of SCh was comparatively large (mean 8.42 torr). This was followed by a gradual reduction in intraocular pressure after the full dose of SCh, with reversion to near that seen after thiopental when the SCh effect waned. Intraocular pressures in Group II were low ($P < 0.001$) compared with those in Group I. This may have been due to a decrease in the tone of extraocular muscles by diazepam.

Pulse rate and blood pressure did not change significantly after initial and subsequent administrations of SCh. Respiration was not affected by the pretreatment dose, but apnea and muscular relaxation were achieved with the subsequent full dose of SCh. Fasciculations occurred after the pretreatment dose in nine patients of Group I and in four patients of Group II; thus, the overall incidence of fasciculations was 52 per cent. With the full dose of SCh only one patient experienced mild fasciculations, and the other 24 (96 per cent) had no fasciculations.

Surprisingly, review of the patients' records showed that only one patient complained of slight pain in the calf muscles in the postoperative period. Otherwise none of these patients had post-succinylcholine myalgia.

## DISCUSSION

Anesthetic management plays an important role in the successful outcome of ophthalmic operations. All anesthetics and muscle relaxants are capable of reducing intraocular pressure, except SCh, which increases it.[5,6]

Lincoff *et al.*[1,2] reported that sustained contracture of extraocular muscles increases intraocular pressure, but at slightly deeper levels of anesthesia no effect of SCh on the extraocular muscles could be produced even with 10–15 mg SCh. Electromyographic studies of human subjects revealed that such contractures occur only in unanesthetized or very lightly anesthetized patients. During deeper anesthesia SCh appears to have no effect on extraocular muscles. In the present study it was found that 10 mg SCh increased intraocular pressures in both groups even at adequate depths of anesthesia, though these increases resulted in values that were significantly below the

control values (table 1). It is probable that sustained contracture of extraocular muscles is responsible for this initial increase in intraocular pressure.

The subsequent full dose of SCh and endotracheal intubation did not increase intraocular pressure. There have been reports that intraocular pressure increases after endotracheal intubation.[11,12] Straining and coughing during endotracheal intubation, which could contribute to the increase in intraocular pressure, were not seen.

Glaucomatous patients showed comparatively large increases in intraocular pressure in response to pretreatment doses, suggesting that this technique should not be used for such patients.

Surprisingly, the problem of post-SCh myalgia did not arise. It is difficult to say whether the protective effect against SCh-induced myalgia resulted from the pretreatment dose of diazepam or other factors. It has been reported that even 5 mg SCh may produce myalgia,[13] so perhaps diazepam was responsible for its prevention.

In conclusion, pretreatment with a small subparalytic dose of SCh before administration of the full dose prevents muscle fasciculations and increases in intraocular pressure. It is concluded that this sequence of anesthesia can safely be used in intraocular or perforating ophthalmic operations.

The author is greatly indebted to Professor Stanley V. Malvea for his kind interest and suggestions during the study. Thanks are also due to Drs. Miss Usha R. Sharma, B. L. Gupta, and A. Jayaram, and to the patients who voluntarily submitted to the study.

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50:247–249, 1979

# Use of a Left Atrial Pressure Monitor to Diagnose a Malfunctioning Mitral Valve Prosthesis

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Intra- and postoperative monitoring of left atrial pressure (LAP) is routine for all cardiac surgical procedures at our center.[1] The following case demonstrates the value of left atrial pressure monitoring to detect faulty prosthetic valve performance after mitral valve replacement.

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## Report of a Case

A 61-year-old white male needed anesthesia for replacement of his mitral valve. He had had rheumatic fever at the age of 6 years, but had been asymptomatic until five months prior to admission. At that time he experienced atrial fibrillation and congestive cardiac failure that progressed despite optimal medical management with digoxin and furosemide. The patient was admitted with the diagnosis of mitral insufficiency.

The patient was anesthetized with diazepam, pancuronium, $N_2O/O_2$, and fentanyl. The anesthetic course prior to cardiopulmonary bypass was uneventful. Intravenous administration of nitroprusside was necessary to maintain mean left atrial pressure (LAP) at ≤20 torr. The mitral valve was replaced with a 31-mm Björk-Shiley prosthesis without difficulty during 38 minutes of ischemic cold cardioplegic arrest. Myocardial performance