DEPOLARIZING NEUROMUSCULAR BLOCKING AGENTS AND INTRAOCULAR PRESSURE IN VIVO

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In the course of an investigation of the effects of depolarizing neuromuscular blocking agents on extraocular muscle in vitro (1), the effects of two of these agents were studied in vivo in the cat to confirm the in vitro results and the opinion of Lineoff et al. (2) in respect to the part played by venous pressure in the elevation of intraocular pressure by succinylcholine.

METHOD

Cats were anesthetized with pentobarbital, tracheotomized, and placed in a Horsley-Clark frame. An internal jugular vein was cannulated, a pneumograph attached about the thorax, and one of the inferior oblique muscles of an eye dissected from its insertion. The anterior chamber of the opposite eye was cannulated to record intraocular pressure. Statham strain gauges were used to record pressures simultaneously on a type M Offner dynograph.

In order to determine if the rise of intraocular pressure produced by depolarizing neuromuscular blocking agents was due to a rise in jugular venous pressure, a dose of succinylcholine, previously determined to produce no effect upon respiration, was administered to a cat.

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Fig. 1. Effect of nonparalyzing dose of depolarizing relaxant on the intraocular pressure in the cat.
Fig. 2. Effect of apnea on cat previously paralyzed with decamethonium.
Fig. 3. Effect of respiratory paralyzing dose of depolarizing relaxant complicated by hypoventilation and apnea.
Figure 1 demonstrates an increase in tension of the inferior oblique muscle, with a rise in intraocular pressure in approximately 10 seconds without effect upon respiration or upon internal jugular venous pressure. This observation, which was repeated at will, tends to prove that the elevation in intraocular pressure is a primary action of succinylcholine. The elevation in intraocular pressure noted was not accompanied by fasciculation of other skeletal muscles.

In order to show the effect of venous pressure on intraocular pressure, a cat was prepared as previously, rendered apneic with a dose of decamethonium (Cl0), and connected to a constant volume respirator having the same tidal air as the cat under pentobarbital alone. After suitable control period, the respirator was disconnected. Figure 2 demonstrates the effect of acute apnea on intraocular pressure related to internal jugular venous pressure. It will be noted that there is a delay of approximately thirty seconds before either the internal jugular or intraocular pressure is elevated. The tension exerted by the inferior oblique muscle is not altered.

The effect of the combined application of a depolarizing neuromuscular blocking agent and apnea is demonstrated in figure 3. The cat may be more sensitive to decamethonium (Cl0) than to succinylcholine, since a dose of decamethonium produced apnea while a comparable dose of succinylcholine did not affect respiration (fig. 1). We have not studied a sufficient number of cats to determine the quantitative relationship between these drugs or to draw a specific conclusion. It will be noted that the effects of the two factors, that is the depolarizing neuromuscular blocking agent and the diminished ventilation in producing a rise in intraocular pressure, appear to be supplementary. The effect of the depolarizing neuromuscular blocking agent acts first in approximately 10 seconds and then approximately 30 seconds thereafter, the effect of increased jugular pressure exerts its effect.

**Summary and Conclusions**

Depolarizing neuromuscular blocking agents produce an increase in intraocular pressure independent of venous pressure. It occurs from ten to fifteen seconds following the administration of the drugs.

Apnea or reduced ventilation tends to increase internal jugular pressure after a short delay of approximately thirty seconds and reflexly increases intraocular pressure.

The effects of depolarizing neuromuscular blocking agents and respiratory depression on intraocular pressure appears to be supplementary.

**References**
