

Decreased blood solubility of these inhalation anesthetics in anemic patients would accelerate the approach of the arterial concentration towards a constant inspired concentration. On this basis, in the anemic patient anesthetic induction would be more rapid than in patients with normal hemato-crits. Likewise, the decrease in arterial anesthetic concentration would be more rapid, and recovery from anesthesia would be accelerated in the anemic patient. One must also consider other factors, such as cardiac output and alveolar ventilation, with respect to rate of anesthetic induction and recovery.

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Pancuronium and Intraocular Pressure

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The effect of the new nondepolarizing muscle relaxant, pancuronium, on intraocular pressure (IOP) is not well defined. Smith and Leano¹ demonstrated that the combination of an intubation dose of pancuronium with induction of anesthesia with a barbiturate and narcotic results in a decrease in IOP. Previous reports have demonstrated that most drugs used to produce anesthesia decrease IOP in a manner related to the depth of anesthesia.² In order to differentiate the effect of pancuronium from the effects of the anesthetic, IOP was measured after pancuronium was administered to unanesthetized subjects, to anesthetized subjects in a quiet state prior to surgical stimulation and

intubation, and after the administration of thiopental and pancuronium.

METHODS

Nineteen adult patients, ASA 1-3, without known intraocular abnormality, were studied. Informed consent was obtained from all patients prior to their entry into the study. All

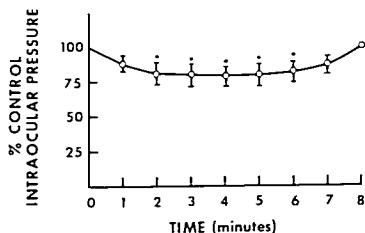


FIG. 1. Mean intraocular pressures following administration of pancuronium, 0.01 mg/kg, ± 1 SE. O = pancuronium, 0.01 mg/kg, given iv; * = statistically significant, $P < 0.01$.

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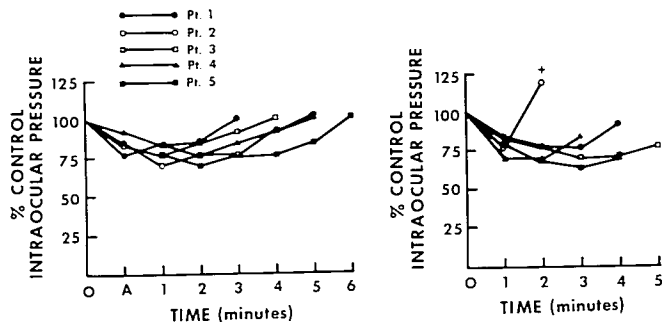


FIG. 2. Intraocular pressure responses to thiopental, 3-4 mg/kg, and nitrous oxide, 70 per cent, with oxygen, 30 per cent, followed by pancuronium, 0.08 mg/kg. *Left panel*, O = thiopental with 70 per cent N₂ and 30 per cent O₂; A = loss of lid reflex. *Right panel*, O = pancuronium, 0.08 mg/kg, given iv; + = patient coughed.

were premedicated with morphine, .1-.16 mg/kg, and scopolamine, .004-.006 mg/kg within an hour of the beginning of the study. All intraocular measurements were made with a Schiøtz tonometer with a 5.5-g weight. All measurements were made in both eyes. IOP was measured in all patients while they were awake, using 0.5 per cent proparacaine for topical anesthesia, and all pressures were normal. Ten patients received pancuronium, 0.01 mg/kg. Thereafter, bilateral IOP measurements were made at one-minute intervals until pressures returned to and remained at awake values. In five patients, anesthesia was induced with sodium thiopental, 3-4 mg/kg. Bilateral IOP measurements were made as soon as the lid reflex was lost. Nitrous oxide, 70 per cent, and oxygen, 30 per cent, were inhaled by the patient by face mask and IOP pressure readings were taken at one-minute intervals until IOP returned to awake values. The patients were then given pancuronium, 0.08 mg/kg, and IOP measurements were continued at one-minute intervals. Ventilatory assistance was given as necessary to maintain ventilation. Intraocular pressure measurements were continued until return toward baseline was noted. At this point the lungs were ventilated with pure oxygen, and additional sodium thiopental, 1-2 mg/kg, was given. The trachea was

intubated and the operation was begun. No further measurements were recorded.

In four patients, anesthesia was induced with sodium thiopental, 3-4 mg/kg. Immediately after loss of lid reflex, IOP was measured, after which pancuronium, 0.08 mg/kg, was given and respiration was assisted with pure oxygen as necessary to maintain normal end-expiratory carbon dioxide as measured by the Beckman infrared CO₂ analyzer. Three minutes after pancuronium was given, bilateral IOP measurements were made and the trachea was intubated. The patient was then mechanically ventilated with 70 per cent nitrous oxide and 30 per cent oxygen as necessary to maintain normal end-tidal CO₂ values. IOP's 3 and 8 minutes after intubation were recorded.

RESULTS

In all ten unanesthetized patients receiving pancuronium, 0.01 mg/kg, IOP was observed to decrease by 1 minute after administration of the drug, and 2 minutes after drug administration IOP had decreased to approximately 80 per cent of control level. This reduction persisted for the next 4 minutes, after which a gradual return was noted, with return to 100 per cent of control IOP by 8 minutes (fig. 1). No patient showed an in-

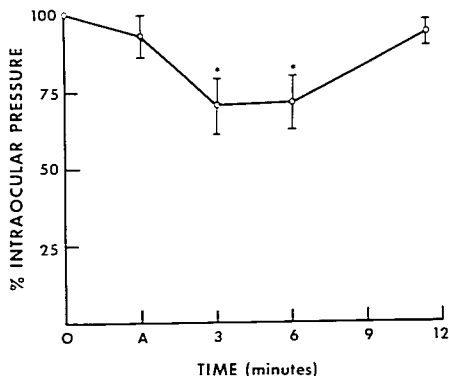


FIG. 3. Mean intraocular pressure responses to thiopental, 3-4 mg/kg, with oxygen, 100 per cent, followed by pancuronium, 0.08 mg/kg, ± 1 SE. O = thiopental and 100 per cent O₂; A = loss of lid reflex; * = statistically significant, $P < 0.01$.

crease in IOP following administration of pancuronium, and none had any objective or subjective effect of the drug. All were able to focus on a spot on the ceiling of the induction room during IOP reading. Analysis of the results by *t* test for paired data showed that the decrease in IOP was significant at the .01 level for readings taken in the 2-6-minute periods following drug administration.

In the second group of five patients, IOP was observed to decrease following the administration of sodium thiopental (fig. 2). In all patients, IOP's returned to baseline values within 6 minutes. IOP's were observed to decrease in all of these patients following subsequent administration of pancuronium, 0.08 mg/kg. By 1 minute after drug administration, IOP's decreased to 70-84 per cent of control values. The periods during which IOP's continued to decrease ranged from 2 to 4 minutes. Of interest is one patient who, approximately 2 minutes after the administration of pancuronium, was inadvertently stimulated. This resulted in airway obstruction and coughing. Intraocular pressure in this patient was observed to increase to 120 per cent of control, confirming that other factors under the control of the anesthesiologist influence IOP. The *t* test for paired data demonstrated that the decrease in IOP 1 minute after administration of pancuronium was again significant at the 0.01 level.

In the group of patients given pan-

curonium immediately after induction (fig. 3), IOP's decreased to an average of approximately 70 per cent of control values by 3 minutes after thiopental and pancuronium administration. Intraocular pressures were still approximately 70 per cent of control 3 minutes after tracheal intubation, and 8 minutes after intubation, they had increased to approximately 92 per cent of control. The *t* test for paired data showed the decrease in IOP to be significant at the 0.01 level for 3 minutes after pancuronium administration and 3 minutes after intubation.

In summary, 19 patients received doses of pancuronium while awake, after receiving a thiobarbiturate, or after being anesthetized to a steady state. A brief, consistent, significant reduction of IOP was seen after pancuronium with all conditions. The effect on IOP was neither dose-related nor increased by prior administration of a thiobarbiturate or nitrous oxide. The decrease in IOP must be a primary effect of pancuronium and is not related to the duration of action of the drug as a neuromuscular blocker.

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