Ketamine and Intraocular Pressure

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The effect of intravenously administered ketamine (Ketalar) on intraocular pressure has been studied by Corssen and Hoy.[1] They found a statistically significant increase in intraocular pressure 3 minutes after ketamine administration. However, in nearly 50 per cent of the cases in which measurements were made preoperatively, intraocular pressures either did not change or decreased in one or both eyes. It is possible that a few values could have influenced the significance of the results. Also, there was no standardization of preoperative medication, which is known to affect intraocular pressure.

METHODS

Twenty patients, ranging in age from 16 to 61 years, who were scheduled for various elective surgical procedures, were included in the study. No patient who had a history or evidence of ocular disease, cardiovascular or hypertensive disease, or a neuropsychiatric disorder was included.

All intraocular pressure measurements were made with a Schiøtz tonometer using a 5.5-g weight. All measurements were made by the same examiner using the same instrument. The corneas were anesthetized with topical proparacaine hydrochloride.

In all patients control measurements of intraocular pressure were obtained the evening before operation. The next morning all patients received a standard premedication with meperidine (0.8–1.0 mg/kg), diazepam (0.08–0.1 mg/kg) and atropine (0.006–0.008 mg/kg), following which intraocular pressure was again measured.

Ketamine was then administered intravenously in a dose of 2 mg/kg. Subsequent measurements were made 1, 2, 3, 4, 5, and 10 minutes after ketamine administration. All measurements were made in both eyes. Pulse, respiration, and blood pressure were simultaneously monitored. No surgical stimulation occurred during the study period, and a patent airway was maintained at all times.

RESULTS

The mean values for all 20 patients, as well as the standard deviation for each measurement, are shown in figure 1. All preoperative values were within normal limits. After premedication intraocular pressures dropped from a mean of 18 mm Hg to a mean of 17.3 mm Hg. One minute after administration of ketamine, intraocular pressures decreased from a mean of 17.3 mm Hg to 16.3 mm Hg. Thereafter, intraocular pressures remained below control values, with the lowest value being 15.9 mm Hg at 2 minutes, after which time it began increasing, returning to a value of 16.6 mm Hg at 10 minutes. This corresponded to the beginning of emergence from the initial dose of drug. None of these values is significant using the t test for paired data. Blood pressure and pulse rate increased in all patients, but these increases did not correlate with intraocular pressures.

Preinduction values showed a 3.9 per cent decrease from control. All values were below control, the lowest value being −11.6 per cent, 2 minutes after induction. The data are presented in table 1.

Measurements had to be discontinued in three patients after 5 minutes because of movement. Two of these three patients manifested increases in intraocular pressures in both eyes.

In ten patients intraocular pressure increased at some point in the study. In ten

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Received from the Department of Anesthesiology, University of Mississippi Medical Center, Jackson, Mississippi 39216. Accepted for publication May 15, 1975.

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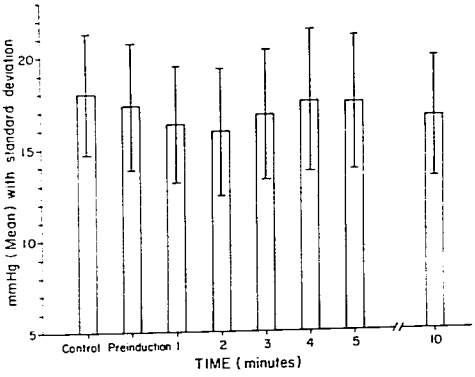

FIG. 1. Effect of ketamine on intraocular pressure, expressed as mm Hg versus time in minutes.

patients there was no change or a decrease in pressures in both eyes.

DISCUSSION

In the study of Corssen and Hoy there apparently was no standardization of premedication, and no mention of dosage is made except that a narcotic, a barbiturate, and an anticholinergic were given. Both narcotics and barbiturates are known to lower intraocular pressure by increasing the facility of outflow[2]; the larger the dose of such drugs, the greater the decrease in intraocular pressure.[3] Anticholinergic drugs, *i.e.*, atropine and scopolamine, when administered intramuscularly in the dosages usually given, do not alter intraocular pressure,[4] and usual premedicant doses of narcotics plus atropine lower intraocular pressure less than 10 per cent. The data presented here show no significant lowering of intraocular pressure following premedication. The dosages of drugs given were such that all patients were awake and conversant without stimulation. These doses may account for the lack of any significant effect of premedication. Intraocular pressure shows a slight diurnal variation, being highest in the early morning and lowest about midnight. The change is small, about 3–5 mm Hg.[6] This could account for a seemingly smaller change after premedication than was expected, since control readings were made in the evening and preinduction readings in the morning. The data also fail to show any significant alteration of preoperative intraocular pressure by ketamine at any point in the study.

Yoshikawa[5] studied the effects of comparable intramuscular doses of ketamine (5 mg/kg) in children. He found a 37 per cent increase in intraocular pressure 15 minutes following injection, with return to baseline levels after 30 minutes. These patients were all unpremedicated. The present study included no pediatric patients. Corssen's study, however, included 12 pediatric patients for whom he had preoperative values. Eight of the 12 showed either no change or a decrease in intraocular pressure in both eyes at 3 minutes. These received premedication. It would seem reasonable to attribute the large increases in pressure in Yoshikawa's study to a lack of premedication.

Ketamine is said to produce its cardiovascular effects via the sympathetic nervous system,[9,10] and release of norepinephrine.[11] The effect of the drug on intraocular pressure may be mediated similarly. It is possible that in the hypothalamus there is a coordination capable of influencing intraocular pressure which is subject to afferent impulses carried by the long ciliary nerves. Stimulation of many points causes increases

TABLE 1. Intraocular Pressures in Both Eyes* before and after Ketamine Administration

	Control		Preinduction		1 Minute		2 Minutes		3 Minutes		4 Minutes		5 Minutes		10 Minutes	
	OS	OD	OS	OD	OS	OD	OS	OD	OS	OD	OS	OD	OS	OD	OS	OD
Patient 1	20.6	20.6	24.4	20.6	20.6	20.6	15.9	17.3	15.9	15.9	20.6	20.6	20.6	17.3	15.9	14.6
Patient 2	22.4	17.3	15.9	17.3	17.3	18.9	12.2	12.2	10.2	12.2	10.2	12.2	11.2	13.4	15.9	15.9
Patient 3	20.6	20.6	24.4	20.6	13.4	12.2	13.4	13.4	14.6	13.4	12.2	15.9	17.3	15.9	15.9	15.9
Patient 4	17.3	20.6	17.3	18.9	18.9	20.6	20.6	17.3	17.3	20.6	26.6	24.4	26.6	20.6	—	—
Patient 5	20.6	22.4	20.6	22.4	14.6	14.6	12.2	12.2	11.2	10.2	11.2	10.2	12.2	13.4	20.6	18.9
Patient 6	17.3	17.3	14.6	14.6	12.2	12.2	12.2	12.2	17.3	17.3	15.9	17.3	20.6	20.6	20.6	17.3
Patient 7	17.3	20.6	17.3	14.6	14.6	17.3	12.2	14.6	15.9	17.3	17.3	17.3	20.6	17.3	—	—
Patient 8	15.9	15.9	14.6	17.3	14.6	17.3	17.3	17.3	15.9	15.9	14.9	17.3	15.9	15.9	—	—
Patient 9	24.4	20.6	20.6	17.3	24.4	20.6	24.4	20.6	24.4	24.4	20.6	24.4	24.4	20.6	24.4	24.4
Patient 10	20.6	20.0	22.4	22.4	20.6	22.4	18.9	20.6	20.6	20.6	22.4	22.4	20.6	22.4	22.4	20.6
Patient 11	12.2	17.3	12.2	17.3	13.4	17.3	14.6	20.6	17.3	20.6	17.3	17.3	13.4	18.9	13.4	14.6
Patient 12	15.9	14.6	15.9	14.6	14.6	13.4	14.6	14.6	15.9	14.6	14.6	14.6	15.9	14.6	17.3	15.9
Patient 13	15.9	14.6	15.9	14.6	17.3	17.3	15.9	14.6	15.9	18.9	17.3	14.6	17.3	15.9	14.6	18.9
Patient 14	17.3	17.3	14.6	15.9	17.3	17.3	18.9	22.4	17.3	20.6	18.9	18.9	17.3	20.6	20.6	17.3
Patient 15	17.3	17.3	20.6	20.6	17.3	18.9	17.3	18.9	18.9	20.6	18.9	20.6	17.3	18.9	13.4	13.4
Patient 16	14.6	17.3	10.2	12.2	9.4	10.2	9.4	10.2	10.2	12.2	13.4	14.6	12.2	14.6	10.2	10.2
Patient 17	14.6	17.3	14.6	14.6	13.4	14.6	14.6	15.9	15.9	15.9	18.9	20.6	18.9	20.6	14.6	15.9
Patient 18	14.6	13.4	17.3	17.3	14.6	17.3	20.6	17.3	20.6	20.6	20.6	20.6	20.6	18.9	17.3	15.9
Patient 19	13.4	15.9	20.6	12.2	14.6	13.4	14.6	12.2	15.9	12.2	14.6	15.9	14.6	15.9	15.9	15.9
Patient 20	17.3	17.3	15.9	15.9	15.9	17.3	15.9	17.3	15.9	17.3	15.9	17.3	14.6	15.9	13.4	13.4

* OS = left eye (*oculus sinister*); OD = right eye (*oculus dexter*).

or decreases of intraocular pressure, independent of general vascular changes. Decreases in intraocular pressure are probably sympathetically mediated since they can be prevented by section of the sympathetic trunk. Stimulation of the peripheral end of cut cervical sympathetic trunk causes constriction of ciliary vessels and is associated with a marked decrease of intraocular pressure.[6]

Armaly[8] observed a decrease in intraocular pressure following stimulation of the parasympathetic root of the ciliary ganglion, probably reflecting a decrease in ciliary blood volume.

In summary, while most anesthetic agents have been shown to lower intraocular pressure, nitrous oxide being the only possible exception,[3,12] the present study shows that ketamine in clinically used doses, administered to a premedicated patient, has no significant effect on intraocular pressure.

The authors thank Doctor Don Turner, Department of Surgery, for his help in statistical preparation of this paper.

Fine-screen Filtration of Pressurized Whole Blood, Packed Cells, and Fresh-frozen Erythrocytes

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Fine-screen filtration removes particulate matter in stored blood, thereby alleviating a potential cause of respiratory failure following massive transfusion.[1] Previously, we reported[2] that fine-screen filtration: 1) need not interfere with flow rate of blood compared with the control (170-micron) filter; 2) removes twice as much debris as the control filter; 3) reduces screen filtration pressure (a measure of debris removal); 4) does not harm fresh whole blood infused by gravity. However, the clinical requirements of transfusion therapy demand that blood be pressurized to achieve rapid infusion through fine-screen filters or blood warmers, or when a massive blood loss is encountered. Concern that fine-screen filters affect erythrocytic integrity when blood is pressurized to 300 torr has been voiced. Therefore, we determined the hemolytic effect on erythrocytes of infusing whole blood, packed cells, and fresh-frozen erythrocytes at 300-torr pressure.

METHODS

Ten units each of indated whole blood, packed erythrocytes, and fresh-frozen erythrocytes (FFE) were infused at 300-torr pressure through a 40-micron woven knit filter. The erythrocyte count, hematocrit, and plasma free hemoglobin were measured before and after filtration. Screen filtration pressure (SFP) was measured in 4 units of FFE before and after filtration by the method of Swank.[3] The 20-micron screen used in the measurement of SFP was fixed in 1 per cent glutaraldehyde solution, after which electron micrographs were made. To avoid damage to the transducer during measurement of SFP before the FFE were filtered, the SFP

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Received from the laboratories of the Department of Anaesthesia, Harvard Medical School at the Massachusetts General Hospital, Boston, Massachusetts 02114. Accepted for publication May 3, 1975. Supported in part by Harvard Anaesthesia Center Grant GM15904-08.

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