Anesthesiology
66:419–421, 1987

Intraocular Pressure Changes during Muscular Hyperactivity after General Anesthesia

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Marked changes in intraocular pressure (IOP) probably should be avoided in the postoperative period following intraocular surgery. A sudden increase in IOP during this period may re-open the incision or wound and induce prolapse of intraocular contents. Thus, a smooth emergence without airway obstruction, coughing, or emesis is desired.[1]

Muscular hyperactivity during recovery from general anesthesia termed "spasticity," "shivering," or "shakes" is a well-known phenomenon.[2] We determined the effect of this hyperactivity on IOP.

METHODS

For the purpose of study, muscular hyperactivity was subdivided into spasticity and shivering.[3] Spasticity was defined as sustained muscular hypertonicity, most easily observed in jaw, neck, and pectoral muscles, flexors of upper limbs, and extensors and adducters of lower limbs.[3] Shivering, on the other hand, was defined as rhythmic contractions of muscle groups, with irregular, intermittent periods of relaxation.[3] Hypothermia was defined as rectal temperature less than 36° C.[4]

Thirty patients were studied. All were scheduled for minor surgical procedures of less than 30 min duration. They were aged 17–50 yr, and were ASA physical status I and had no eye ailments. Informed consent was obtained. Premedication was with morphine 0.1 mg/kg and promethazine 0.4 mg/kg im 1 h before anesthesia. Anesthesia was induced with thiopental (4–6 mg/kg) iv, and was maintained with halothane (2–3% inspired) and nitrous oxide (66%) in oxygen. All patients breathed spontaneously through a Mapleson A circuit. Neuromuscular relaxants and endotracheal intubation were avoided.

In the postoperative period, the patients were observed for 30 min for muscular hyperactivity and were then entered into one of three groups. Group 1 had no detectable muscular activity, group 2 had spasticity alone (spasticity was assessed by jaw tone, and also by the resistance offered to passively moving each forearm through 90° range of motion), and group 3 had obvious shivering.

IOP was measured in all patients by an ophthalmologist who was not aware of the purpose and details of the study. Measurements were made with a hand-held applanation tonometer, at the following intervals: (1) before induction, (2) just before discontinuing anesthesia, and (3) every 2 min after discontinuing anesthesia for 10 min, and then every 5 min for another 20 min. Care was taken to measure IOP when the patients were supine, and there was no tilt to the operating table or bed. Rectal temperatures were noted (1) immediately after induction, (2) before discontinuing anesthesia, and (3) at 5, 15, and 30 min intervals postoperatively, using a thermister probe. Heart rate and systemic arterial blood pressures were monitored by conventional non-invasive methods at every 5–10 min interval during intra- and postoperative period.

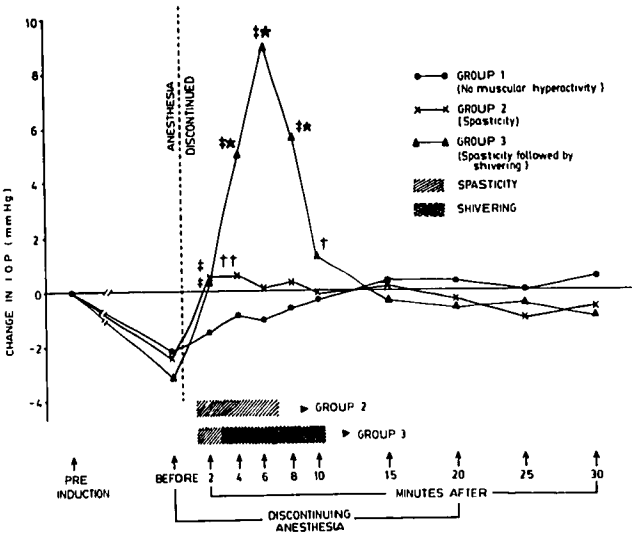

FIG. 1. Changes in IOP from the pre-induction value at various intervals in different groups, as related to the postanesthetic muscular hyperactivity. *$P < 0.001$ differ from group 2; between-group analysis. †$P < 0.05$ differ from group 1(Control); between-group analysis. ††$P < 0.01$ differ from group 1(Control); between-group analysis. ‡$P < 0.001$ differ from group 1(Control); between-group analysis.

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Received from the Department of Anesthesia, Postgraduate Institute of Medical Education and Research, Chandigarh, India. Accepted for publication October 6, 1986.

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Key words: Complications: shivering. Eyes: intraocular pressure. Temperature.

TABLE 1. Within-Group Changes in IOP at Various Intervals

Group	Pre-induction IOP (mmHg)	Just Before Discontinuing Anesthesia	Minutes after Discontinuing Anesthesia							
			2	4	6	8	10	15	20	25	30
1 (Control) n = 11	13.18 (0.58)	10.95* (0.38)	11.68 (0.39)	12.27 (0.32)	12.09 (0.51)	12.59 (0.39)	12.86 (0.38)	13.54 (0.52)	13.63 (0.56)	13.27 (0.77)	12.68 (0.83)
2 (Spasticity) n = 9	14.83 (0.60)	12.50† (0.79)	15.33‡ (0.49)	15.38 (0.56)	14.94 (0.46)	15.16 (0.48)	14.77 (0.53)	15.05 (0.48)	14.50 (0.63)	14.38 (0.47)	14.16 (0.64)
3 (Shivering) n = 8	14.75 (0.57)	11.56 (0.89)	15.18‡ (0.68)	19.75* (0.75)	23.93§ (1.91)	20.31† (1.95)	16.12 (1.20)	14.31 (0.74)	14.25 (0.66)	14.43 (0.60)	14.06 (0.71)

Mean ± SEM.
* $P < 0.05$ differ from pre-induction value within same group.
† $P < 0.01$ differ from pre-induction value within same group.
‡ $P < 0.01$ differ from the preceding value within same group.
§ $P < 0.001$ differ from pre-induction value within same group.

Patients who objected to IOP measurements, or who had airway problems or persistent cough or emesis in the postoperative period, were not included in the study. Also, those shivering severely, and those posing technical difficulty to IOP measurement because of excessive shaking, could not be included.

Analysis of variance was used to determine the statistical significance of data for within-group and between-group comparisons.

RESULTS

Out of 30 patients, two were excluded from the study. One had excessive shaking, and the other patient did not cooperate in the postoperative period. Eleven were entered in group 1 (no clinical evidence of muscular hyperactivity), nine in group 2 (muscular spasticity, but no shivering), and eight in group 3 (shivering). All the patients of group 3 demonstrated muscular spasticity also, which preceded the onset of shivering. The time course of muscular spasticity in groups 2 and 3, and that of shivering in group 3, is shown in figure 1.

Pre-induction intraocular pressures in all the groups were similar. IOP decreased during anesthesia, as indicated by lower readings of IOP, in all the groups before discontinuing anesthesia (table 1, fig. 1). In group 1, postanesthetic IOP levels maintained a lower profile (as compared to pre-induction levels), rising gradually, taking about 15 min to reach pre-induction values (fig. 1).

In groups 2 and 3, in the immediate postanesthetic period, IOP increased significantly from the reduced levels during anesthesia, resulting in its return to the pre-induction levels, within 2 min (table 1, fig. 1). This rapid return of IOP to pre-induction levels coincided with the onset of muscular spasticity in both the groups (fig. 1). In group 2, after reaching the pre-induction levels, IOP did not change further. However, in group 3, IOP increased during shivering, resulting in significantly higher IOP values at 4th, 6th, and 8th postanesthetic minutes, when compared to the pre-induction values (table 1, fig. 1). Later, the IOP decreased to pre-induction values, once the shivering disappeared (at 10–15 min in the postanesthetic period).

All the patients were normothermic (more than 36° C) in the postoperative period and the means of temperature readings of all the groups, at various intervals during investigation, were not significantly different from each other ($P > 0.05$).

DISCUSSION

Our results indicate that postanesthetic muscular hyperactivity tends to increase IOP. We used halothane for the maintenance of anesthesia in our patients. However, shivering associated with the use of other anesthetics (en-

flurane,[5] isoflurane,[6] ether,[7] narcotic-N_2O[8]) should also cause a similar rise in IOP, but this needs confirmation.

The exact cause for postanesthetic muscular hyperactivity is not certain. Muscular spasticity is considered as a part of normal emergence,[3] while shivering may be due to decreases in temperature[9] and generalized increased CNS activity.[3] However, temperature readings often do not differ between the patients who shiver and those who do not.[3,5,10,¶]

Our data do not indicate the mechanism for the increase in IOP during shivering, or the reason for the lack of an increase in those with spasticity. Also, we have not examined the influence of physiological variables or possible prophylactic techniques. A number of features associated with shivering may influence IOP. Hypoxemia during shivering[10,11] may increase IOP.[1] Intense tonic contractions of various muscle groups are common during shivering. Extraocular muscles may also undergo similar contraction, causing a rise in IOP. Contraction of orbicularis oculi may be an additional factor. Also, the contraction of thoracic muscles would increase the venous pressure[12] and increase IOP indirectly.[13] While these are mere speculations, further clinical and laboratory studies may provide some answers.

Despite the initial tendency to increase IOP, muscle spasticity alone could not increase it above the basal levels. This is probably because of a lesser degree of muscular hyperactivity in patients with spasticity alone, when compared to the patients who shiver. An attempt to grade the muscular hyperactivity and to correlate the various grades with the amount of rise in IOP, and also with the changes in various physiological variables, such as arterial blood pressure, venous pressure, and arterial blood gases, may have provided some clues in this regard.

We did not attempt to correlate arterial blood pressure with IOP changes, because of the difficulty of obtaining these measurements in shivering patients.

We conclude that postanesthetic shivering should be considered one of the potential hazards in a patient scheduled for intraocular surgery.

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