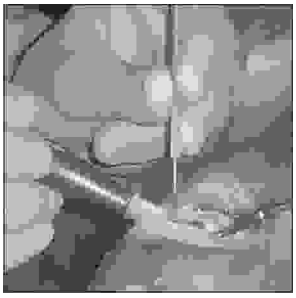
ORIGINAL PROF-1291

CATARACT SURGERY;

EFFECTS OF SPONTANEOUS VERSUS CONTROLLED VENTILATION ON INTRAOCULAR PRESSURE DURING GENERAL ANAESTHESIA



LT. COL. DR. ABDUL HAMEED BHATTI
Anaesthetist
Combined Military Hospital,
Pano Aqil Cantt

ABSTRACT... Objective: To compare the effects of spontaneous versus controlled ventilation on Intraocular Pressure (IOP) with concomitant haemodynamic changes during cataract extraction and intraocular (IOL) implant surgery under anaesthesia. **Design:** Comparative study. **Place and Duration of Study:** The study was conducted at department of Anesthesiology Combined Military Hospital Jhelum Cantt from Jan 2005 to Oct 2005. **Subjects and Methods:** 40 ASA I and II patients of both sexes aged 40-68 years, undergoing surgical cataract extraction were studies. In 20 patients ventilation was controlled while the other 20 patients breathed spontaneously during surgery. IOP was measured preoperatively in non-operated eye. **Results:** Intraocular pressure (IOP) decreased below the base line after induction of anaesthesia but it markedly increased after intubation in both group. During operation IOP decreased more in controlled ventilation group than spontaneous ventilation group. At the end of surgery before extubation, IOP increased in both groups with a greater rise in spontaneous ventilation group and extubation was followed by a further rise in IOP in both groups. Heart rate (HR) and arterial blood pressure (BP) changes followed almost the same pattern as IOP. **Conclusion:** General anaesthetics decrease IOP in general. Laryngoscopy and intubation are anaesthesia-related events, which cause rise in IOP. In appropriate patients, general anaesthesia with controlled ventilation is an acceptable technique for intraocular surgery offering advantages in terms of intraocular pressure and cardiovascular stability compared to spontaneous ventilation.

Key words: Anaesthesia, general: Ophthalmic. Cataract extaction, intraocular pressure. Spontaneous ventilation. Controlled ventilation.

INTRODUCTION

The goal of anaesthetic management during ophthalmic surgery is to provide good control of intraocular pressure (IOP), an immobile, operative field and cardiovascular stability, combined with an adequate level of anaesthesia. The choice of ventilation, whether spontaneous or controlled is controversial[1].

Regardless of the method of implantation employed, the one basic principle of IOL implant surgery, common to all methods, is the maintenance of a soft eye and orbit to reduce trauma caused by insertion. Therefore absolute

akinesia and good anaesthesia, are necessary. General Anaesthesia (GA) provides suitable operating conditions, especially in apprehensive and uncooperative patients. The Europeans almost universally use general anaesthesia to have their patients completely relaxed[2]. The eye can be considered a hollow space with a rigid. If the contents of the space increase, the IOP (normal 12-20 mm Hg) must rise. A rise in venous pressure and extreme changes in arterial blood pressure and ventilation can affect IOP. Any anaesthetic event that alter these parameters (e.g. laryngoscopy, intubation, airway obstruction, coughing, Trendelenburg position can affect IOP[3]).

So the essential features of general anaesthesia for ophthalmic surgery are a straight forward smooth anaesthetic with no hypoxia or hypercarbia, smooth recovery without coughing straining or vomiting and for intraocular surgery, particularly if lens implant is to be inserted, the tension in the eye must be as low as possible[4].

The purpose of this study was to compare the effects of controlled versus spontaneous ventilation on IOP with concomitant haemodynamic changes during cataract surgery under general anaesthesia.

PATIENTS AND METHODS

This study was carried out at Combined Military Hospital Jhelum Cantt from Jan 2005 to Oct 2005. Forty ASA I and II patients of both sexes, aged 68 years or less (range 40-68 years), undergoing surgical cataract extraction and intraocular lens implantation were studies. Any patients with respiratory or cardiac disease were excluded from the study.

The patients were randomly selected to receive controlled (group I, n=20) or spontaneous ventilation (group II, n=20). The patients were admitted to the hospital 1 or 2 days before the scheduled surgery. Routine clinical chemistry tests, ECG and X-ray chest were performed. Written informed consent was obtained from all patients. Surgery was performed between 0800 hours and 1300 hours. In the operation theatre an intravenous (I.V) cannula was inserted to each patient for I.V infusion (Lactated Ringers solution) and administration of drugs.

Before induction of anaesthesia, intraocular pressure (IOP) was measured in the non-operated eye using Schiotz tonometer after instillation of 0.4% benoxinate hydrochloride drops. Data were recorded at the times shown in Table I. Peri-operative monitoring consisted of continuous ECG, non-invasive arterial pressure, end-tidal carbon dioxide partial pressure and pulse oximetry.

After determination of baseline data, midazolam 2.5 mg[5] was administered 1. V. and 1 minute later, anaesthesia was induced with slow 1.V. injection of thiopentone[6] (sufficient to abolish the eyelash reflex). Post-induction data were recorded. Suxamethonium 1mg/kg was given[6,7,8] were the lungs ventilated, using a face mask with 60% nitrous oxide in oxygen and 1% Isoflurane. When fasciculations ceased and jaw became relaxed, the airway was secured, using cuffed endotracheal tube (ETT). After satisfactory placement the ETT was fixed using adhesive strapping attached to the skin over both the maxilla and mandible.

The group I patients were given vecuronium[9] 0.04mg/kg 1.V. and mechanical ventilation of the lungs (Pulmovent) was commenced with a tidal volume of 10 ml/kg, at a rate sufficient to maintain the end tidal carbon dioxide pressure at 4.0 to 4.5 kPa and anaesthesia was maintained with isoflurane[10] 0.8% and 60% nitrous oxide in oxygen. Supplementary vecuronium (0.01mg/kg) was given when required to maintain the paralysis.

The group II patients were allowed to breathe spontaneously. Isoflurane concentration was increased to achieve the level of anaesthesia deep enough to avoid straining and coughing during surgery. Anaesthesia was maintained with 60% nitrous oxide in oxygen and 1.5—2.0% Isoflurane. During spontaneous breathing end-tidal carbon dioxide partial pressure varied between 5.5 to 6.0 kPa.

Anaesthetic management was designed to maintain arterial pressure and heart rate within clinically accepted limits (systolic arterial pressure greater than 80mm Hg and less than 160mm Hg). Injection metochlopramide 10mg was given 1.V. before conclusion of the surgery to prevent post-operative nausea and vomiting[3].

At the conclusion of the surgery, in Group I patients, neuromuscular block was antagonized with neostgmine 2.5mg and glycopyrolate 0.5mg. Patients then breathed 100% oxygen. Lignocaine 1.5 mg/kg 1.V. was given about 3 min before extubation to prevent postoperative coughing. Pre-extubation data were obtained and Isoflurane was turned off. Gentle pharyngeal toilet was then performed followed by extubation. After extubation, intraocular pressure and cardiovascular data were recorded and later on patients were shifted to recovery ward.

Table-I. Time sequences when measurements of intraocular pressure, cardiovascular Variables and end-tidal carbon dioxide were obtained

Variables monitored	Pre- induction	Post- induction	Post- intubation 1 min 5min	During-operation	Pre- extubation	Post extubation
Intraocular Pressure	+	+	+	+	+	+
Heart Rate	+	+	+	+	+	+
Arterial Pressure	+	+	+	+	+	+
End tidal CO_2	-	-	+	+	+	-

Key:+ =Variable monitored , -= Variable not monitored

RESULTS

Patients' characteristics are summarized in Table II. There were no significant differences between the groups. Time sequences of measurements were not significantly different between groups. Tracheal intubation was accomplished without difficulty in all patients. It was easy to ventilate the lungs of group-I patients to an end-tidal carbon dioxide partial pressure of less than 4.5kPa. The smallest oxygen saturation was 96% in group II patients. In 20% of group II patients, ventilation had to be switched over to controlled one, on surgeon's request because of problem of reposition of iris due to raised intraocular pressure (IOP). There were no complications of extubation in any patient. Recovery from anaesthesia was slightly delayed in group II patients (Table III).

Intraocular pressure (IOP) decreased below the baseline after induction but it increased after endotracheal intubation. IOP decreased to near pre-induction value at 5 min after intubation.

In group I, IOP decreased further during surgery. On the other hand, IOP remained higher in group II than group I despite increasing the concentration of Isoflurane (Table IV).

In 20% of these patients, spontaneous ventilation (SV) had to be switched over to controlled ventilation (IPPV) due to problems in reposition of the iris because of raised IOP and IOP the increased.

At the end of surgery, IOP increased before extubation. This rise in IOP was more in group II (VS) and extubation was followed by a further increase in IOP in both groups (Table IV).

The increase in heart rate with intubation, from pre-induction values was not significantly different between the groups. This effect was brief and the rise in heart rate did not persist at 5 min after intubation.

Table-II. Patients' characteristics		
Characteristics	**Group I (IPPV)**	**Group II (SV)**
Age (year)	54(42-68)	44 (29-62)
Weight (Kg)	74(9.5)	70(15.6)
Sex (Male: female)	4:6	6:4
Systolic arterial pressure (mm Hg)	145(11.2)	140 (8.6)
Diastolic arterial pressure(mm Hg)	85(8.0)	82(5.0)
Heart Rate (beat/min)	73(4.7)	74(5.2)
Intraoral pressure (mm Hg)	16.1 (2.1)	15.4(2.4)
Key: IPPV= Intermittent positive pressure ventilation SV= Spontaneous ventilation [Note: All the values are Mean (SD)]		

Table-III. Peri-operative problems related to anaesthesia		
Problems	**SV (n=20)**	**IPPV (n=20)**
Intraoperative interventions (change of ventilatory mode)	n=4 (20%)	n=0
Hypotension	n=5 (25%)	n=1 (5%)
Delayed recovery	n=10 (50%)	n=2 (10%)
Post-operative nausea and vomiting	n=2 (10%)	n=2 (10%)
Key: IPPV = Intermittent positive pressure ventilation SV = Spontaneous ventilation		

Table-IV. Peri-operative intraocular pressure (mmHg)		
Time sequence	**Group I (IPPV)**	**Group II (SV)**
Pre-induction	15.2 (2.7)	16.4 (2.9)
Post-induction	14.1 (2.5)	14.0 (2.1)
Post-intubation 1min 5 min	 22.4 (3.6) 13.6 (2.9)	 23.2 (3.7) 15.8 (2.8)
During operation	12.1 (2.4)	15.0 (2.5)
Pre-extubation	15.8 (3.2)	16.4 (3.1)
Post-extubation	22.6 (3.3)	21.8 (2.9)
Key: IPPV= Intermittent positive pressure ventilation SV= Spontaneous ventilation [Note: All the values are Mean (SD)]		

Later on, during surgery heart rate decreased in group-I (Table V), but remained at slightly higher level in group II. However, immediately before and after extubation, there was increase in heart rate in both groups.

Table-V. Peri-operative heart rate (beats/min)		
Time sequence	**Group I (IPPV)**	**Group II (SV)**
Pre-induction	74 (5.2)	73 (5.0)
Post-induction	91 (6.0)	94 (7.7)
Post-intubation 1min 5 min	 110 (7.8) 88 (5.7)	 112 (7.6) 92 (7.3)
During operation	74 (5.3)	88 (5.9)
Pre-extubation	82 (5.4)	94 (7.6)
Post-extubation	112 (7.9)	114 (7.8)
Key: IPPV= Intermittent positive pressure ventilation SV= Spontaneous ventilation [Note: All the values are Mean (SD)]		

Systolic arterial pressure slightly decreased in both groups at induction but increased immediately after intubation; and although arterial pressure decreased towards baseline at 5 minutes in both groups, it decreased more in group II. Later on during surgery systolic pressure decreased further in both groups was significant in group II. Systolic pressure increased in both groups before and after extubation (Table VI). Diastolic arterial pressure also followed almost the similar changes (Table VII).

Table-VI. Peri-operative systolic arterial pressure (mmHg)		
Time sequence	**Group I (IPPV)**	**Group II (SV)**
Pre-induction	142 (9.7)	145 (13.1)
Post-induction	136 (11.7)	132 (9.0)
Post-intubation 1min 5 min	 162 (12.9) 148 (12.1)	 168 (12.6) 141(9.7)
During operation	138 (11.7)	119 (8.6)
Pre-extubation	144 (9.7)	136 (11.6)
Post-extubation	160 (9.2)	164 (11.9)
Key: IPPV= Intermittent positive pressure ventilation SV= Spontaneous ventilation [Note: All the values are Mean (SD)]		

Table-VII. Peri-operative diastolic pressure (mmHg)		
Time sequence	**Group I (IPPV)**	**Group II (SV)**
Pre-induction	85 (8.0)	84 (6.2)
Post-induction	80 (7.6)	81 (5.4)
Post-intubation 1min 5 min	 108 (10.3) 92 (7.2)	 108 (8.4) 88(6.3)
During operation	74 (6.7)	68 (6.5)
Pre-extubation	84 (6.0)	78 (6.0)
Post-extubation	106 (8.4)	105 (8.2)
Key: IPPV= Intermittent positive pressure ventilation *SV= Spontaneous ventilation* *[Note: All the values are Mean (SD)]*		

DISCUSSION

Intraocular pressure (IOP) is determined by extra ocular muscle tone, scleral rigidity, vascularity of the orbit and production and outflow of aqueous humour[11,12]. After induction of anaesthesia with thiopentone, IOP decreased significantly in both groups, but it increased above the base line values after laryngoscopy and intubation. These changes in IOP at this time conform to those reported in a previous study[6].

The intubation induced increase in IOP was transient. Five minutes after intubation the IOP decreased below the pre-intubation value and there was no significant difference between the groups. Thiopentone reduces IOP mainly via its depressant effect on central diencephalic controlling areas for IOP, although increased aqueous drainage has also been shown to occur[1, 6]. Suxamethonium causes a transient increase in IOP for 4-6 minutes[7, 8]. Studies on the non-depolarizing neuromuscular blocking drug, vecuronium have revealed either no effect or a decrease in IOP.[1, 2, 9] The effect of nitrous oxide on IOP has received little attention[11], but it has been well established that all the volatile inhalation agents decrease IOP in a does-dependent manner[1,2,10]. Several mechanisms of action to later IOP have been suggested; effects on the central controlling areas in the midbrain, by altering aqueous outflow and by altering extraocular muscle tone[12]. However, these inhalation agents may cause excessive cardiovascular depression, particularly in elderly patients, if concentrations are increased in an attempt to improve the ocular conditions for surgery.

Later on during surgery, IOP decreased significantly in group I patients in which end-tidal carbon dioxide ($ETCO_2$) partial pressure was kept between 4.0-4.5 kPa. But ib group II, IOP remained at higher level despite increasing the depth of anaesthesia. $ETCO_2$ was also raised in this group i.e. between 5-6 kPa. These changes in IOP conform to a previous study[13]. In group I (IPPVgroup), a satisfactory operation field was reported by surgeons, but in 20 percent of group II patients, surgeon felt problem in reposition of iris due to raised IOP and patients had to be switched over to IPPV. These findings do not conform to a recent study[14]

The marked increase in IOP was seen in both groups after extubation. This exaggerated increased in IOP was probably caused by the very light anaesthesia at this time. This result implies that, where tracheal intubation is performed in patients with raised IOP, steps should be taken to control IOP during extubation. These peri-extubation changes in IOP are in conformation with the previous study[14].

Haemodynamic (arterial pressure and heart rate) changes after intubation, during surgery and at extubation were in conformation with the pervious studies[14,15]. Arterial pressure decreased in group II and heart rate in group I. These changes are probably due to increased depth of anaesthesia in group II. Older patients showed more drop in blood pressure. Volatile agents, particularly halothane, depress baroreflex function[16] and exert a direct effect on myocardial activity, by reducing both the amount of calcium release from the sarcoplasmic reticulum and the influx of calcium to activate myofibrils[17]. Contractility is depressed more by the volatile agents in a failing heart[18] and this may have some bearing on the greater depression of cardiovascular indices in older patients. Group II (VS) patients had lower mean end-operative arterial oxygen

saturation (SaO_2) than group I (IPPV) patients (SV 96%; IPPV 98%) and were extubated sooner at the end of anaesthesia, fever, sore throat and myalgia was similar.

CONCLUSION

This study has shown that the general anaesthetics decrease the intraocular pressure (IOP) in general. Laryngoscopy and intubation are anaesthesia related events, which cause rise in IOP. The general anaesthesia using controlled ventilation is particularly suitable for intraocular lens (IOL) implant surgery because it provides a satisfactory operative field by conferring patient's immobility, decreasing IOP and ensuring cardiovascular stability, especially in the elderly patients.

Moreover, because of lighter plane of anaesthesia, there is early recovery. On the other hand, patients on spontaneous ventilation require deeper plane of anaesthesia with consequent delayed as well as cardiovascular instability and poor control of IOP.

REFERENCES

1. Nicol M. **Anaesthesia for ophthalmic surgery.** In: Aitkenhead AR, Rowbotham DJ, Smith G. Textbook of Anaesthesia. 4th ed. Harcourt Publisher Ltd. London: Churchill Livingstone, 2001: 594-596.

2. Jaffe NS. **Results of intraocular lens implant surgery.** Am J Ophthalmol 1978; 85:13?

3. Morgan, Jr. GE, Mikhail MS, Murray MJ. **Clinical Anesthesiology.** 4th ed. McGraw-Hill Companies, Inc. USA, 2006:826-830.

4. Donlon, Jr. JV, Doyle DJ, Feldman MA. **Anesthesia for eye surgery.** In: Miller RD. Miller's Anesthesia 6th ed. Elsevier, Churchill Livingstone, 2005: 2527-2533.

5. Gobeaux D, Sardnal F. **Midazolam and flumazenil in ophthalmology.** Acta Anaesthesiol Scand Supply 1990; 92: 35-8.

6. Mirakhur RK, Shepherd WFI, Darrah WC. **Propofal or thiopentone: effects on intraocular pressure associated with induction of anasesthesia and tracheal intubation (facilitated with suxamethonium).** Br J Anaesth 1987; 59:431-6.

7. Salim M. **Effect of succinylcholine on intraocular pressure.** AFMJ (Pak) 1991.

8. Kelly RE, Dinner M, Turner LS, Haik B, Abramason DH, Daines P. **Succinylcholine increases intraocular pressure in the human eye with the extraocular muscles detached Anesthesiology** 1993; 79 (5): 948-52.

9. Polarz H, Bohrer H, von-Tabouillot W, Martin E, Tetz M, Volcker HE. **Comparative effects of atracurium and vecuronium on intraocular pressure.** Ger J Ophthalmol 1995; 4 (2): 91-3.

10. Mirakhur RK, Elliott P, Shepherd WF, Mc Galliard JN. **Comparison of the effects of isoflurane and halothane on intraocular pressure.** Acta Anaesthesiol Scand 1990; 34 (4): 282-5.

11. Murphy DF. **Anaesthesia and intraocular pressure.** Anesth Analg 1985; 64: 520- 530.

12. Holloway KB. **Control of the eye during general anaesthesia for intraocular surgery.** Br J Anaesth 1980; 52: 671-679.

13. Smith RB, Aass AA, Nemoto EM. **Intraocular and intracranial pressure during respiratory alkalosis and acidosis.** Br J Anaesth 1981; 53: 96.

14. Van-den-Berg AA, Honjol NM. **Clinical comparison of spontaneous versus controlled ventilation general anaesthesia using isoflurane for intraocular surgery: intraoperative, recovery and post-operative effects.** Anaesth intensive care 1994; 22 (6): 683-90.

15. Vucevic M, Purdy GM, Ellis FR. **Esmolol hydrochloride for management of the cardiovascular stress responses to laryngoscopy and tracheal intubation.** Br J Anaesth 1992; 68: 529-30.

16. Duke PC, Fownes D, Wade JG. **Halothane depresses bareoreflex control of heart rate in man.** Anaesthesiology 1977; 46: 184-7.

17. Katsuoka M, Ohnishi ST. **Inhalation anaesthetics decrease calcium content of cardiac sarcoplasmic reticulum.** Br J Anaseth 1998; 62: 669-73.

18. Hobbhah J, Conzen P, Forst H, Peter K. **Influence of inhalation anaesthetics on the myocardium.** Anaesthetist 1989; 38: 561-86.