COMPARISON OF THE EFFECT OF LIGNOCAINE AND GALLAMINE ON SUXAMETHONIUM-INDUCED ELEVATION OF INTRAOCULAR PRESSURE (IOP)

A DISSERTATION SUBMITTED IN PART FULFILMENT FOR THE DEGREE OF MASTER OF MEDICINE (ANAESTHESIA) OF THE UNIVERSITY OF NAIROBI.

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DECLARATION

This dissertation is my original work and has not, to my knowledge been presented for a degree in any other University.

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DEDICATION

Dedicated especially to the patients from whom data for this study was obtained; my teachers, family, friends and colleagues who were a constant source of inspiration.

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SUMMARY

This study was designed to determine the time-course, incidence as well as magnitude of suxamethonium induced elevation of intraocular pressure (IOP); and to compare the effectiveness of pretreatment with either Lignocaine or Gallamine in attenuating this adverse effect of suxamethonium. The study sample consisted of inpatients presenting for elective either opthalmological or non-opthalmological surgery at the Kenyatta National Hospital (KNH).

One hundred and fifty patients were randomly divided into three groups each consisting of 50 patients that were given pretreatment 3 minutes before induction of anaesthesia. The control group received 5mls of Normal saline and the remaining two test groups were pretreated with either Gallamine 0.5mg/kg or Lignocaine 1.5mg/kg.

In all the 3 groups there was transient elevation of intraocular pressure which was only statistically significant in the control and Gallamine groups. The incidence of intraocular hypertension following

intravenous suxamethonium and endotacheal intubation was found to be 80%. Mean increase in intraocular pressure from the resting to peak value was found to be 7.3mmHg in the Control group. Peak elevation in IOP occured 3 minutes after intravenous administration of suxamethonium and returned to normal within 6 minutes in all the 3 groups. Only in the Lignocaine group was this elevation not statistically significant from the baseline values. It was concluded for this study that Lignocaine pretreatment in a dose of 1.5mg/kg given 3 minutes prior to induction of anaesthesia with Thiopentane is more effective in attenuating post-suxamethanium intraocular hypertension than Gallamine. Subsequently Lignoicane pretreatment was recommended whenever there is risk of prolapse of intraocular contents due to suxamethonium induced elevation of IOP.

1. INTRODUCTION

Despite its serious side effects including elevation of intraocular pressure (IOP); Suxamethonium or scoline remains the drug of choice for induction of general anaesthesia particularly in emergency situations. Since the Scoline induced intraocular hypertension can be severe its use in opathalmological surgery deserves special consideration especially when the anatomical integrity of the cornea and Sclera is lost or weakened.

From the register of General Surgical emergencies in the main theatre at the Kenyatta National Hospital (KNH), in 1988 alone a total of 29 cases presented with penetrating eye injuries for emergency operation in the main theatre. Nevil (1) in a study of perforating injuries as seen and managed at the KNH found that 69.2% of the patients treated had their visual acuity either not improved; or even worsened after surgery under general anaesthesia. He mentions delay in coming to hospital by patients as probably the most important contributory factor to this poor visual prognosis.

Probably even more alarming though, was the finding in the same study that there was an average delay of six and a half hours between time of admission and operation.

Although theatre availability was given as the main reason for this delay, need for starvation of the patients in preparation for general anaesthesia was also pointed out. The latter reason is unjustifiable because these cases are surgical emergencies in which crash induction of general anaesthesia using suxamethonium is the safest standard technique even if a patient has a full stomach.

Effective precaution however is mandatory for crash induction technique using scoline in emergency cases with penetrating eye injuries. The incidence of such injuries at the Kenyatta National, Provincial as well as District hospitals, is expected to progressively increase associated with victims of road traffic accidents and assault. These patients are a big challenge to the anaesthetist whose technique should minimise the consequences during induction due to both a full stomach - aspiration pneumonia; and scoline induced elevation of IOP - prolapse of intraocular contents.

The latter could result in irreversible damage to the affected eye(2,3).

A similar situation could arise even in many other elective cases for opthalmological surgery especially if the anterior chamber of the eye is to be opened, the cornea is already weakened, the sclera is weakened or is to be excised; and surgery is to be repeated on the eyeball before complete wound healing. In all these cases if suxamethonium is to be used, an attempt should be undertaken to prevent or minimise post-suxamethonium intraocular hypertension because any increase in IOP while the eyeball is either open or its integrity lost; may cause expulsion of vitreous and subsequent irreversible loss of vision.

The suxamethonium induced elevation of intraocular pressure (IOP) has been of particular concern to the anaethesists since the 1950s (3). Since the original report (4) that suxamethonium raised the intraocular pressure in conscious volunteers as well as anaesthetized subjects numerous investigators have studied this relationship. The original findings were soon confirmed and seen by many other workers (5,6). The normal intraocular tension lies between 15-20 mm Hg(8).

An average rise of 7.8mm Hg after suxamethonium occured in one series (7). Some cases in this study the tension rose by upto 15mm Hg. In another series rises of up to 30mm Hg occured with an average increase of 19mmHg (9). Similar rises were found by other workers in the later 1960s (10,) as well as by more recent workers (12,13). The pressure in the anterior chamber of the normal eye is some 10-22 mmHg above atmospheric pressure (11). Subsequently, any elevation in IOP due to the intraocular hypertensive effects of suxamethonium can result in prompt expulsion of Vitereous (6).

Therefore, during intraocular surgery the control of intraocular pressure assumes as vital importance as does the control of intracranial pressure in neurosurgery because the sclera just like the skull is inelastic. As a result, numerous specific methods some of which are either effective or inconvinient have been suggested to control suxamethonium induced intraocular hypertension. The following is a brief review of some of such methods particularly selected as applicable in our limited clinical set up at Kenyatta National Hospital:-

I. **PRETREATMENT WITH LIGNOCAINE**: Lignocaine given intravenously prior to the administration of suxamethonium is known to reduce the incidence and severity of both fasciculations and myalgia associated with the latter drug (14). It also significantly reduces both reflex circulatory and stress response to laryngoscopy and tracheal intubation by increasing depth of general anaesthesia (15). Recently lignocaine pretreatment has been observed to effectively attenuate the intraocular pressure response to suxamethonium (16). This efficacy of Lignocaine against IOP rise may be explained on the basis of its proposed peripheral actions (17).

Other advantages of Lignocaine pretreatment prior to general anaesthesia are that it reduces anaesthetic requirements; as well as minimising autonomic reflexes during general anaethesia (18). Intravenous lignocaine attenuates the cough reflex in light anaesthesia (19), increase of intracranial pressure even in tracheal suctioning (20); and the tachycardia as well as increase in blood pressure that occurs due to laryngoscopy and tracheal intubation (18). The main disadvantage of lignocaine pretreatment is that it prolongs the post-suxamethonium apnoea (14).

II. ***PRETREATMENT WITH NON-DEPOLARISING MUSCLE RELAXANTS:***

As will be discussed in more detail later; it is widely believed that the mechanical squeezing of the eyeball by extraocular muscles due to their scoline induced sustained contraction is probably the most important contributory factor to the resultant elevation of IOP. Non-depolarising muscles relaxants are very effective in preventing this sustained contraction. This extreme sensitivity of extraocular muscles to Non-depolarising neuromuscular blocking agents has been utilized by giving small doses of curare even to conscious spontaneously breathing patients during opthalmological operation under local anaesthesia. This effectively lowers the IOP and at one time it was widely used.

Gallamine (Flaxedil) in subparalysing doses has been advocated by many authorities including Miller (21). It is readily available, and an additional advantage associated with Gallamine use especially in opthalmogical surgery is that it prevents the vagal oculocardiac reflex that could result in bradycardia and cardiac arrest. However, like all other non-depolorising muscle relexants; Gallamine pretreatment produces significant delay in the onset of paralysis due

to suxamethonium and subsequent inadequate muscle relaxation. This results in reduced safety of the anaesthetic technique which can be disastrous especially in emergency situations that require crash induction to ensure rapid as well as smooth airway control (22).

III. **PRE-TREATMENT WITH DIAZEPAM:** Has been advocated by many workers as a remedy to suxamethonium induced intraocular hypertension (2). However more recent work has shown that the reduction of intraocular pressure due to Diazepam is not statistically significant (16, 39). Moreover Friedman et al in 1970 (23) described the interaction between Diazepam and suxamethonium which results in decreased neuromuscular block by the latter. Another disadvantage of Diazepam is the unacceptable high incidence of pain and thombosis it is likely to cause along the injected veins.

IV. **DIURETICS:** Reduction of either aqueous or vitreous volume by diuretics could be useful for control of intraocular pressure during surgery (24), but the rate of this change is too slow (25). Osmotic dehydrating agents like Mannitol or sucrose cause appreciable fall in IOP by reducing vitreous volume. To achieve this

Mannitol 20-60gm would have to be started 45 minutes prior to surgery while the subsequent diuresis would require an indwelling urethral catheter.

Pretreatment with Acetazolamide both reduces aqueous production and facilitates its drainage but is of questionable benefit during surgery as it leads to increased intrachoroidal vascular volume (26). Therefore when the eye is opened, the tendency to prolapse of intraocular contents remains. Other disadvantages of Acetazolamide include potassium loss, metabolic acidosis, alteration of the respitory centre response to carbon dioxide and depression of the central nervous system.

V. **USE OF NON-DEPOLARISING MUSCLE RELAXANTS**: To facilitate laryngoscopy and tracheal intubation has been recommended by some anaesthetists (27); even for crash induction on the assumption that these drugs unlike suxamethonium do not cause intraocular hypertension. This technique has numerous disadvantages. To provide adequate intubation conditions that merely approximate to the same as produced by suxamethonium during crash induction requires high doses of non-depolarising muscle relaxants. This carries an added risk of incomplete reversal of these drugs with subsequent post-operative respiratory failure (27).

VII. **DIGITAL COMPRESSION OF THE GLOBE**: This manouvre when done before opening the eyeball reduces IOP by enhancing flow of aqueuos humour from the anterior chamber (31). The technique has been used in cataract surgery or any other situation in which the eyeball is intact; but it is obviously contraindicated in cases where the integrity of the globe is lost before surgery especially due to trauma.

VIII. **LOCAL NERVE BLOCK**: Retrobulbar and Facial nerve block prevents contraction of extraocular muscles hence no mechanical squeezing of the eyeball occurs after administration of suxamethonium. However this requires co-operation of the patient and is therefore inapplicable to children as well as unco-coperative adults especially with a very painful injured eye. In addition most surgeons prefer general anaesthesia since it spares the patients of a trying ordeal.

On the basis of this brief literature review, the choice of drug pretreatment using Lignocaine and Gallamine for this study was mainly due to the fact that this technique is convinient, saves time and does not significantly compromise the safety during crash induction of general anaesthesia of patients at risk. Both drugs are readily available on the anaesthetic tray in most theatres at Kenyatta National Hospital.

SPECIFIC AIMS AND OBJECTIVES:-

This study was undertaken for the following reasons:-

1. To determine the incidence and severity of scoline induced intraocular hypertension in adult inpatients presenting for elective surgery at the KNH.

2. To determine the time-course of scoline induced elevation of IOP.

3. Assess the effectiveness of intravenous Lignocaine and Gallamine pre-treatment in attenuating scoline induced intraocular hypertension.

MATERIALS AND METHODS

A total of 150 adult patients of both sexes, ASA class I or II; and aged 15-60 years premedicated with 0.6mg Atropine intramuscular, starved and scheduled for elective either opthalomological or non-opthalmological surgery requiring endotacheal intubation were studied. Any of these patients was selected for the study provided there was no ocular disease at least in one eye, no liver or systemic illness including hypertension.

After obtaining informed consent including explanation on the procedure of measuring IOP from every single patient selected for the study, they were randomly allocated into three groups of 50 patients each:

GROUP I: The control group were pre-treated with 5 mls of intravenous normal saline.

GROUP II: The Lignocaine group were pre-treated with 1.5 mg/kg of 20% preservative-free Lignocaine hydrochloride intravenous diluted to 5mls with normal saline.

GROUP III: The Gallamine group were pre-treated with 0.5 mg/kg Gallamine intravenous diluted to 5 mls normal saline.

Upon arrival into the operating room, a peripher venous indwelling cannula was inserted percutaneousl for administration of drugs. Three minutes afte administration of any of the above pre-treatment; th baseline value of IOP was taken after instilling a least 2 eyedrops of 0.4% Oxybuprocaine into the selecte eye. Thereafter intravenous induction was carried ou with a sleeping dose of Thiopentone given slowly t obtund the eye lash reflex followed by Suxamethoniu 1.5mg/kg. Post-induction oxygenation was done by gentl intermittent positive pressure ventilation (IPPV) usin the Magill's anaesthetic circuit (Mapleson A) deliverin 8L/min of oxygen.

Exactly one minute after administration o suxamethonium the post-induction measurement of IOP wa taken, then IPPV with a face mask continued unti another reading of IOP was taken and thereafter direc laryngoscopy and tracheal intubation performed. Onl patients with excellent intubation conditions viz-voca cords easily visible, mid-position and fixed; wer included in the study. If there was buckling o coughing while endotracheal tube was being inserte into the trachea, such cases were excluded as thi causes exegerrated intraocular response to intubatic

(2). Endotracheal intubation for all patients was achieved between 2 and 3 minutes after the administration of suxamethonium.

Inhalational supplements were avoided during the induction sequence. After securing the cuffed endotracheal tube gentle manual inflation of lungs was maintained until resumption of spontaneous ventilation. Steady state general anaesthesia stage 3 plane II was maintained with 2% Halothane, 3L/minute of oxygen and 5L/minute of Nitrous oxide on Mapleson A circuit by spontaneous breathing.

All measurements of IOP were monitored by the author, who was unaware of pretreatment given, from a single selected eye using the schiotz tonometer with 5.5gm weight. The tonometer scale readings were converted into mmHg with the help of a chart provided with the instrument. These were taken at the following intervals:-

(i) Three minutes after pretreatment but before induction - the resting value;

(ii) One and two minutes after administration of suxamethonium but before endotracheal intubation;

(iii) Thereafter at one minute interval for a total of 10 minutes after intravenous administration of suxamethonium.

STATISTICAL ANALYSIS

To determine whether the mean rise of suxamethonium induced IOP from baseline to peak value was statistically significant, the statistical test using comparison of 2 means was used (32). The deductive statistics used for this test are illustrated in table V.

Using this test, the increase in IOP for both the Control and Gallamine was found to be statistically significant at the 5% level of significance ($p>0.05$). For the Lignocaine group, the rise was not statistically significant ($p<0.05$).

RESULTS

(i) RESTING VALVES:: All the 3 groups of patients studied were comparable on the basis of age, resting both IOP and systolic blood pressure as well as dose of Thiopentone used; since the mean values for all these factors did not differ significantly (Table I).

(ii) TIME COURSE OF SUXAMETHONIUM INDUCED INCREASE IN IOP: Is illustrated by table II and displayed in figure I. There was a steep and immediate rise followed by a steep fall; and thereafter a more gradual decline of IOP. The peak rise in IOP occured at 3 minutes after administration of suxamethonium and endotracheal intubation. Thereafter there was progressive decline and within 6 minutes the IOP had returned to baseline values, but continued to drop well below these values as illustrated by table IV and displayed in figure 2.

(iii) INCIDENCE AND SEVERITY OF RAISED IOP: These were worked out only in the control group and are shown in table III. Forty patients had their IOP elevated above the baseline values giving an incidence of 80%. Only 10 patients had their IOP either unchanged or fall from the baseline value 3 minutes after suxamethonium

and induction of general anaethesia. The majority (24%) of patients had elevation of IOP between 10.6-15.5mmHg.

The maximum rise obtained in this series was 20.6mmHg. The mean rise for the entire control group was 7.3 mmHg.

(iv) COMPARISON OF THE EFFECTIVENESS OF LIGNOCAINE AND GALLAMINE: The changes in IOP in all the 3 groups from baseline values at all time intervals are shown in table IV and displayed in figure 2. IOP rose significantly ($p<0.05$) from the baseline to peak value in both the Control and Gallamine group. In the Lignocaine the rise was not statistically significant.

D I S C U S S I O N

From this study it was found that 80% of adult in-patients at the KNH induced with intravenous Thiopentane followed by suxamethonium and smooth endotracheal intubation develop detectable elevation of intraocular pressure. Elsie F. Mayers (33) found an incidence of 70% while Craythorne et al (9) got 50% Goldsmith demonstrated an incidence of 45% (10), and D.J. Bowen et al (13) demonstrated an incidence of 82% in their series. These figures emphasize the fact that the incidence of post-suxamethonium intraocular hypertension is high.

The rather wide variation in the incidence of post-suxamethonium intraocular hypertension may be due to numerous factors. The most important is probably the corresponding wide variation in anaesthetic techniques and inhalatinal agents used (34). For example, vocal cords spraying with local anaesthetic agents was carried out in some of these studies while in other this was not done. Another contributory factor could be the different drugs used for premedication (3). In addition there are other numerous factors that affect IOP during anaesthesia as will be discussed later.

The time course of post-suxamethonium induced hypertension compares well with the pattern obtained by other workers (35,16). However Cummingham et al (2) and other workers using similar methodology obtained peak rise immediately after suxamethonium and before intubation. This difference can be explained by the fact that in their study the IOP measurements were taken at two minute intervals after suxamethonium and tracheal intubation, while it has been shown that the increase in IOP due to intravenous suxamethonium, maintains its peak for one minute (31) only, and decreases immediately, thereafter. Therefore measurement of IOP two minutes after intubation will miss the peak values. The universal agreement in all these studies is that intraocular hypertension effect of suxamethonium is transient as well as self-limiting and becomes insignificant by 6 minutes after administration of the drug.

Intraocular pressure increases from baseline to peak values in the control group of this study ranged between 1-20.6mmHg with a mean value of 7.3mmHg. This was in agreement with results obtained by some other workers (4,16); but differs significantly from others (9,10,12,13) who obtained higher values ranging between 15-30mmHg. This wide variation may be partly explained

by the different techniques of tonometry used. As yet no absolutely satisfactory method of measuring IOP in clinical practice has been developed. All measurements of IOP in this study were made by the author using a schiotz tonometer, a technique accurate to within +-2mm Hg. Its main advantages are that it is simpler to use, more tolerable by patients especially in taking baseline values when they are still awake; and has been the most commonly used in similar studies especially when large samples were being studied.

The portable applanation tonometer is reputed to be more accurate but could not be used in this study mainly because it is a lot more complicated and therefore requires more skill to use than the Schiotz type. Subsequently the services of an opthalmologist would be required which was not possible. In addition to tonometery; the variation in the severity as well as incidence of the intraocular hypertensive action of suxamethonium can also be attributed to other factors which could not be standardized in this study.

Numerous factors influence changes in intraocular pressure. Thiopentone causes a dose dependent reduction in intraocular pressure (34) but the dosage for all the 3 groups of patients was comparable (table I). Atropine causes a slight, transient and insignificant fall in IOP

of normal eyes (36). Opioid analgesics and inhalation anaesthetic agents on the whole cause a decrease in IOP but the magnitude is insignificant (34). Increased venous pressure is immediately transmitted to the eyeball and the choriocapillaries therein distend producing back pressure on the ~~aqueons~~ veins draining the canal of schlem; and this causes corresponding rise in IOP.

During anaesthesia venous pressure is influenced mainly by posture and transmitted intrathoracic pressure. Effects of the former were avoided by having all patients in a horizontal supine position and the latter by selecting only the cases in which intubation was smooth. Central venous pressure (CVP) was not monitored and insertion of a CVP indwelling catheter was not only considered to be unjustifiable ethically, but expensive as well.

Hypercapnia and hypoxia cause elevation of IOP (37). This was avoided in this study by ensuring adequate post-induction intermittent positive pressure ventilation. Moreover changes in arterial blood gases for such a short period of this study are most probably unimportant especially as all patients were healthy with no chest disease.

Changes in systolic blood pressure due to endotracheal intubation are relatively unimportant since any rise in arterial blood pressure leads to the displacement of aqueous humour from the anterior chamber and blood from the choroidal vessels (38). It has been shown that in normal eyes intraocular pressure remains constant over a fairly wide range of normal blood pressure (38). However, when the systolic arterial pressure decreases below 85-90 mmHg, IOP diminishes progressively and profoundly towards atmospheric pressure; which is reached at a systolic arterial pressure of 50-60mmHg. For this reason patients whose systolic blood pressure fell below 90mmHg were excuded in this study. Suxamethonium induced rise in IOP is independent of fasciculations due to the same drug (39, 16). Therefore no attempt was made to monitor the incidence and intensity of fasciculations in this study.

The exact underlying mechanism of the elevation of IOP due to suxamethonium still remains unclear. From the nature of the response, a steep and relatively immediate rise, its principal cause is most likely to be mechanical. It is unlikely that any degree in osmotic relationship or secretory activity can manifest so quickly. Moreover, the relatively quick recovery also supports the concept that mechanical forces are the most likely requisite for the elevation of IOP.

Dillon et al (40) described investigations in both man and animals in which they demonstrated that suxamethonium produced a sustained contraction of extraocular muscles. This response differs from that of skeletal muscles because the threshold of response of the ocular muscles is higher, and their reaction is one of slow but sustained contraction, as opposed to relaxation (41). This difference has been explained on the basis of histology (10). Unlike other skeletal fibres, extraocular muscles contain a large number of slowly reacting muscle fibres which when exposed to stimulation by either acetylcholine or depolarising solution respond with slow tetanic contraction.

Wrethind and Wahlin (42) showed photographic evidence of transient dilatation of both choroidal and conjuctival blood vessels after administration of suxamethonium. This is thought to be a direct effect of suxamethonium on the smooth muscle in eyeball blood vessels. Some investigators have shown that the elevation of intraocular pressure following suxamethonium is further exaggerated by intubation (5) but not prolonged. It seems most reasonable therefore, to conclude that the IOP response to suxamethonium is mainly due to both increased tone of extraocular muscles

and transient dilatation of intraocular vessels especially in the choroidal blood vessels. The increase is significantly aggravated by a reflex response to laryngoscopy and tracheal intubation.

In this study Lignocaine was found to be more effective than Gallamine in controlling the intraocular hypertensive effect of suxamethonium. This observed efficacy of Lignocaine in reducing the post-suxamethonium IOP rise may be explained on the basis of the proposed peripheral actions of Lignocaine (17). Suxamethonium is known to cause immediate activation of fusiform systems in extraocular muscles. This can be suppressed by local anaesthetic agents acting either directly at the muscle spindles discharge from intrafusal muscle receptors without simultaneously paralysing the motor fibres. Lignocaine prevents the suxamethonium induced tonic and sustained contraction of extraocular muscles by the same mechanism. Numerous other advantages of Lignocaine pretreatment have already been pointed out in the introduction (14-21).

On the other hand the efficacy non-depolarising muscle relaxants including Gallamine, in counteracting the intraocular hypertensive effect of suxamethonium has been disputed by results of a number of studies

(16,32,43). Also pretreatment with non-depolarizing relexants significantly reduces the safety of anaesthetic technique as already pointed out (22).

Nevertheless, the sequence of pretreatment with Gallamine or d-Tubocurarine before induction of anaesthesia with adequate dose of Thiopentone and suxamethonium prior to intubation, has not been associated with loss of intraocular contents. The safety of the technique even in emergency surgery for penetrating injuries is supported by extensive practical work (44). Furthermore, some studies have shown Gallamine to be more effective in preventing Suxamethonium induced bradycardia than Atropine (45).

CONCLUSION AND RECOMMENDATIONS

Despite the high incidence and severity of intraocular hypertension caused by suxamethonium when used to facilitate endotracheal intubation; its use carries no extra hazard at least provided the anatomical integrity of the eye ball is intact. The time-course of the intraocular hpertensive action of suxamethonium is so short that if a period of at least 6 minutes is allowed after administration of this drug; the anterior chamber of the eye can be safely opened. This is a sufficient interval between induction and incision of the eye.

There is therefore, no justification in depriving patients undergoing eye operations of the advantages of this short-acting muscle relaxant which is still unsurspassed as an aid to smooth as well as atraumatic endotracheal intubation. This is essential for providing an unhampered field to the eye surgeon in almost all but the briefest of opthalmic procedures.

However, suxamethonium alone should not be used whilst the eye is being or has been opened as in penetrating eye injuries. In such cases its use should be preceeded by Gallamine or better still Lignocaine pretreatment, to minimise the risk of vitreous loss.

Since Lignocaine pretreatment does not reduce the safety of a rapid sequence induction technique using suxamethonium, there is no need to delay surgery for penetrating eye injuries merely because of the fear of consequences of a full stomach during general anaesthesia. There is need to emphasize this to both Opthalmologists and theatre staff at the Kenyatta National Hospital.

Further studies in which the outcome of surgery for perforating injuries is evaluated in terms of visual prognosis vis-a-vis anaesthetic technique used, would provide a clinically important criterion for determing the effectiveness of the various techniques to control suxamethonium induced intraocular hypertension. The need for such studies is further emphasized by the fact that as yet, none of the currently used techniques completely abolishes the hypertensive intraocular effect of suxamethonium. Ideally such technique would prevent extrusion of vitreous and subsequent loss vision by maintaining IOP at least within; or better still below normal limits, even after administration of suxamethonium especially in patients at risk.

TABLES AND FIGURES

TABLE I

MEAN (+-SD) RESTING VALUES IN EACH GROUP

GROUPS	SALINE (CONTROL)	GALLAMINE	LIGNOCAINE
AGE (years)	28.84+-9.48	28.1+-11.14	32.62+-14.73
BODY WEIGHT (Kg)	64.08+-11.83	59.36+-11.12	64.28+-9.68
RESTING SYSTOLIC BP (MmHg)	119.6+-14.98	11.0+-15.97	115.88+-15.77
RESTING IOP (mmHg)	14.69+-3.37	14.60+-3.80	14.70+-4.23
DOSE OF THIOPENTONE (Mg)	358+-69.48	329.9+-66.12	333.5+-75.19

TABLE II

TIME COURSE OF INTRAOCULAR PRESSURE FOR ALL GROUPS (MEAN +-SD) AT EACH OBSERVATION TIME

TIME (Minutes)	0	1	2	3	4	5	6	7	8	9	10
CONTROL GROUP	14.69±3.37 -	17.54±5.17 -	19.44±6.44 -	21.99±6.68 -	16.32±4.40 -	14.40±3.73 -	13.97±3.46 -	13.60±3.40 -	13.20±3.59 -	13.23±3.52 -	13.23±3.52 -
LIGNOCAINE GROUP	14.70±4.23 -	16.05±3.98 -	16.23±4.14 -	16.30±4.20 -	14.72±3.68 -	13.87±3.87 -	13.30±3.81 -	12.99±2.65 -	12.82±2.55 -	12.45±2.40 -	12.28±2.12 -
GALLAMINE GROUP	14.60±3.80 -	16.40±4.06 -	17.80±4.78 -	19.00±4.36 -	15.50±4.13 -	13.80±4.03 -	13.50±3.65 -	13.50±3.40 -	13.44±3.44 -	12.81±3.30 -	12.49±1.79 -

TABLE III

INCIDENCE AND SEVERITY OF SUXAMETHONIUM INDUCED RAISED IOP IN THE CONTROL GROUP

MAGNITUDE OF INCREASE IN IOP (mmHg) FROM BASELINE TO PEAK VALUE	NUMBER OF PATIENTS	PERCENTAGE
No Change or Decrease	10	20%
1.0 - 5.5	11	22%
5.6 - 10.5	9	18%
10.6 - 15.5	12	24%
15.6 - 20.5	7	14%
20.6 - 25.5	1	2%
TOTAL	50	100%

TABLE IV

IOP (mmHg) CHANGE FROM RESTING VALVES AT ALL TIME INTERVALS

TIME (Minutes)	0	1	2	3	4	5	6	7	8	9	10
CONTROL GROUP	0	2.85	4.75	7.30	1.53	-0.29	-0.72	-1.09	-1.49	-1.46	-1.46
LIGNOCAINE GROUP	0	1.35	1.53	1.60	-0.02	-0.83	-1.40	-1.71	-1.88	-2.25	-2.42
GALLAMINE GROUP	0	1.80	3.2	4.40	0.9	-0.8	-1.1	-1.1	-1.16	-1.79	-2.11

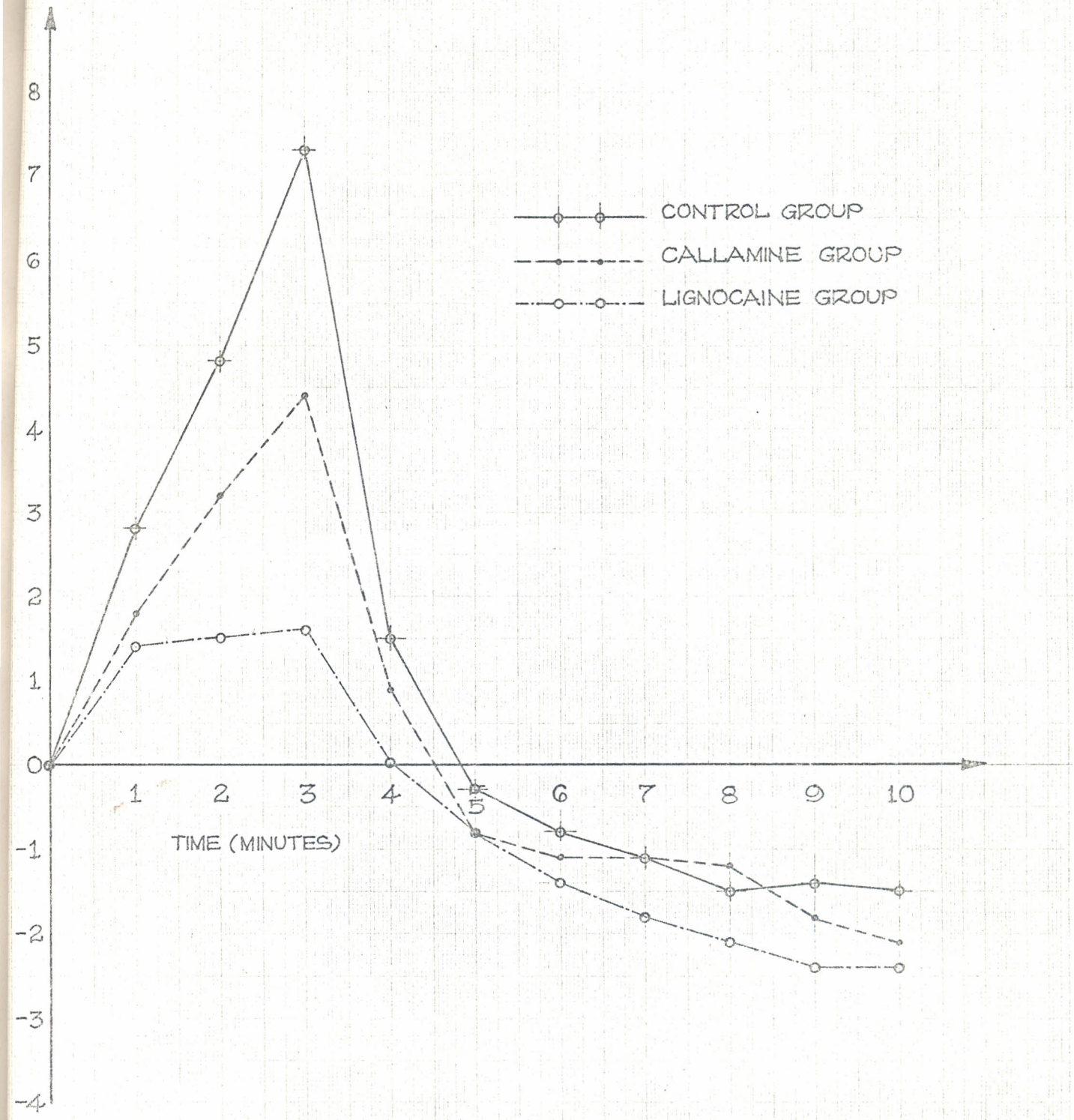
TABLE V

DEDUCTIVE STATISTICS FOR MEAN BASELINE AND PEAK VALUES (mmHg) IN ALL GROUPS:-

GROUP	SAMPLE SIZE	BASELINE VALUE +-SD(mmHg) A	PEAK VALUE +-SD (mmHg) B	DIFFERENCE (B-A)	STANDARD ERROR OF THE MEAN (S.E.M)
CONTROL	50	14.69+-3.37	21.99+-6.68	7.30	1.058
LIGNOCAINE	50	14.70+-4.23	16.30+-4.20	1.60	0.843
GALLAMINE	50	14.60+-3.80	19.00+-4.36	4.40	0.818

FIGURE 2

INTRAOCULAR PRESSURE CHANGE FROM BASELINE VALUES AT VARIOUS INTERVALS



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D A T A S H E E T

PATIENT'S NAME:

WARD: IP NO: AGE: SEX: WEIGHT:

PRETREATMENT:

THLOPENTONE: SUXAMETHONIUM:

TIME (minutes)	0	1	2	3	4	5	6	7	8	9	10
SCALE											
IOP (mmHg)											
BP (mmHg)											

PATIENT'S NAME:

WARD: IP NO: AGE: SEX:

PRETREATMENT:

THLOPENTONE: SUXAMETHONIUM:

TIME (Minutes)	0	1	2	3	4	5	6	7	8	9	10
SCALE											
IOP (mmHg)											
BP (mmHg)											