

**ARTERIAL OXYGEN SATURATION IN  
CHILDREN FOLLOWING GENERAL  
ANAESTHESIA FOR ENT SURGERY**

**Dissertation submitted in part fulfilment of the  
requirement of the award of the degree of Master of  
Medicine in Anaesthesiology of the University of  
Nairobi**

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MBChB (MOI)  
June 2004**



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## DECLARATION

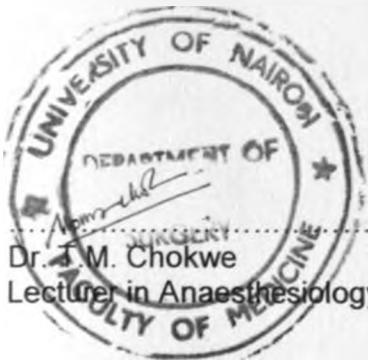
This dissertation is my own original work and has not, to my knowledge, been presented for any degree in any other university.



.....  
Dr. E.C. Bartuiyot MBChB, MOI

Date 22<sup>nd</sup> / 10<sup>th</sup> / 2004

This dissertation has been submitted for the degree of master of medicine in anaesthesia with my approval as university supervisor.



.....  
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Date 22 / 10<sup>th</sup> / 2004

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## CONTENTS

	Page
1. Title	2
2. Declaration	3
3. Acknowledgements	4
4. Contents	5
5. Abbreviations	6
6. Summary	7
7. Introduction	8
8. Literature review	10
9. Aims and objectives	26
10. Sample size	27
11. Methodology	28
12. Study design	29
13. Data collection and analysis	30
14. Results	31
15. Discussion	41
16. Conclusion	45
17. Appendixes	46
18. References	56

## ABBREVIATIONS

ABG	arterial blood gases
ASA	American society of anaesthesiologists' classification
CO <sub>2</sub>	carbon dioxide
Cms	centimetres
ENT	ear, nose and throat
FRC	functional residual capacity
GA	general anaesthesia
Gm/dl	grams per decilitre
Hb	haemoglobin
Kgs	kilograms
LED	light emitting diode
Mg	milligram
ml	millilitre
PaO <sub>2</sub>	arterial pressure of oxygen
PO <sub>2</sub>	partial pressure of oxygen
PaCO <sub>2</sub>	arterial carbon dioxide pressure
PtCO <sub>2</sub>	transcutaneous carbon dioxide pressure
SaO <sub>2</sub>	oxygen saturation

## SUMMARY

A total of 90 children aged between 9 and 144 months undergoing Ear Nose and Throat surgical procedures under general anaesthesia had continuous monitoring of oxygen saturation after their operations.

The purpose of the study was to monitor arterial oxygen saturation for the first thirty minutes in the recovery room to determine the prevalence, degree and duration of post-operative hypoxemia in ASA I or II children following general anaesthesia after ENT surgery.

The study showed that there is no significant hypoxia in these children following upper airway surgery. However there is a progressive decrease in SaO<sub>2</sub> in the first 10 to 20 minutes before it plateaus or increases.

## INTRODUCTION

The post anaesthetic recovery period is an integral part of the overall anaesthetic and surgical management of the patient. As in the peri-induction and intraoperative times, the post anaesthetic period may be marked by events ranging from a smooth uneventful recovery to a stormy emergence with major complications.<sup>1,2</sup>

It has long been accepted that air is an inappropriate breathing mixture for an anaesthetised patients.<sup>3,4</sup> The 20.9% Oxygen in ambient air is insufficient to provide a margin of safety against hypoxia, because lung function almost always deteriorates during and immediately after the course of surgery and anaesthesia.

This deterioration may be due to decreased functional residual capacity (FRC) and increased airway closure, a ventilation/perfusion imbalance, or the development of atelectasis.<sup>5</sup>

Further, carbon dioxide retention caused by hypoventilation can in turn cause hypoxia by displacing oxygen from the alveoli, but this is important only when inspired air is not oxygen-enriched.<sup>6</sup>

In light of these concerns, it has been a long-standing practice to administer no less than 30% oxygen to patients under general anaesthesia (GA) and to provide supplementary oxygen to all patients in the recovery room.<sup>2</sup>

However, healthy patients are frequently allowed to breath room air during transfer from operating room to recovery room and also when in recovery room.<sup>3</sup> The excellent cardio-respiratory status of most paediatric patients and their frequent refusal to tolerate masks or nasal catheters has made the use of supplementary oxygen in the recovery room much less common in children than in adults.



The pulse oximeter is a non-invasive monitor that utilizes spectro-photoelectric techniques to measure arterial oxygen saturation (SaO<sub>2</sub>) in a pulsating vascular bed such as that of a finger. The technique has been described and its accuracy has been documented.<sup>7</sup> Abstracts of current research indicates that the monitor is accurate in children.<sup>8,9,10</sup>

The dangers of desaturation at recovery especially after airway surgery can be a major cause of morbidity and mortality.

## LITERATURE REVIEW

### ENT SURGERY AND ANAESTHESIA

Although much of it is simple and commonplace, otorhinolaryngologic surgery has a disproportionately large potential for anaesthetic and surgical complications. It demands meticulous attention to all aspects of the patient's perioperative care.<sup>2</sup>

As many of these operations involve the airway, the anaesthesiologist must be prepared to provide good surgical access to that area while maintaining a safe ventilatory pathway for the patient.<sup>11</sup>

There are differences in the anatomy of the upper airway of neonates and infants and those of the adult. The laryngeal diameter in infant is smallest at the cricoid cartilage, whereas in the adult it is smallest at the glottic aperture and at the rima glottidis. The larynx in the infant is opposite the third cervical vertebra and has a more anterior position than in the adult. The location of the adult larynx is opposite the fourth cervical vertebra. The epiglottis of the infant is relatively larger, longer, stiffer and U-shaped by contrast to that of the adult. The infant has a macroglossia relative to the adult.<sup>12</sup>

The indication of tonsillectomy and adenoidectomy is airway obstruction and are still the most common procedures in children and should be very safe. Although the incidence of mortality from the initial tonsillectomy procedure is low, the mortality in patients requiring reoperation is very high.<sup>13</sup> Most surgical-related deaths in children are due to airway obstruction and hypovolemia secondary to postoperative bleeding.<sup>13, 14</sup>

The anaesthetist must determine preoperatively the cardiopulmonary changes in these patients and the correct airway required: oral, nasal, via tracheostomy or special apparatus.<sup>15</sup> Difficulty in inserting the original airway can be ascertained during a preoperative visit, and techniques formulated for this procedure.<sup>15, 16</sup> Pre-medication usually employs an antisialagogue. Induction of anaesthesia may be inhalational or intravenous. If inhalational is performed, an intravenous catheter is secured soon after

the patient loses consciousness. Tracheal intubation can be accomplished under deep inhalational anaesthesia with halothane, with or without facilitation of a neuromuscular blocking agent.<sup>17</sup> In addition to electrocardiogram and pre-cordial stethoscope, a pulse oximeter is of great value in monitoring during anaesthesia.<sup>2, 15</sup>

Extubation of the trachea can be safely performed once the patient has emerged from anaesthesia and displays the return of reflexes. After surgery involving the airway, skilled nursing care in the post anaesthetic room/recovery room is essential, so that signs of impending complications can be detected early and appropriate therapy instituted immediately.<sup>15</sup>

## POST ANAESTHESIA CARE

### EMERGENCE FROM GENERAL ANAESTHESIA

Recovery from general anaesthesia is a time of great physiological stress for many patients.<sup>2</sup> Emergence from general anaesthesia should ideally be a smooth and gradual awakening in a controlled environment.<sup>3, 9</sup> Following an inhalation-based anaesthetic, the speed of emergence is directly proportionate to the agent's blood solubility. As the duration of anaesthesia increases, emergence also becomes increasingly dependent on total tissue uptake, which is a function of agent solubility, the average concentration used, and the duration of exposure to the anaesthetic.<sup>2, 4</sup>

Recovery is therefore fastest from nitrous oxide and of the currently available volatile agents, emergence is fastest following desflurane anaesthesia and slowest from prolonged deep anaesthesia with halothane and enflurane.<sup>2</sup> Nonetheless, the most frequent anaesthetic-related cause of delayed emergence from inhalation anaesthesia is hypoventilation.<sup>2, 4</sup>

Emergence from an intravenous anaesthetic is a function of its pharmacokinetics. Recovery from intravenous anaesthetic agents is dependent chiefly on

redistribution rather than an elimination half-life.<sup>25</sup> As the total dose increases, however, cumulative effects become apparent in the form of prolonged emergence; the termination of action becomes increasingly dependent on the elimination or metabolic half-life. Under these conditions, advanced age, renal or hepatic disease can prolong emergence. Patients receiving propofol for induction and maintenance notably recover faster than those receiving other agents.

The speed of emergence can also be influenced by preoperative medication.<sup>26</sup> Pre-medication with agents that outlast the procedure may be expected to prolong emergence. The short duration of action of midazolam makes it a suitable pre-medication agent for short procedures. The effects of preoperative sleep deprivation or drug ingestion (alcohol, sedatives) can also be additive to those of anaesthetic agents and can prolong emergence period.<sup>1</sup>

Unfortunately, hypoxemia often begins in the operating room or during transport to the recovery room and is frequently characterized by airway obstruction, shivering, agitation, delirium, pain, nausea and vomiting, hypothermia, and autonomic lability.<sup>27</sup>

## POST-OPERATIVE COMPLICATIONS

### RESPIRATORY COMPLICATIONS

Respiratory problems are the most frequently encountered complications in the recovery rooms.<sup>18</sup> The overwhelming majority are related to airway obstruction, hypoventilation, or hypoxemia.<sup>28</sup> Because hypoxemia is the final common pathway to serious morbidity and mortality, routine monitoring of pulse oximetry in the recovery room leads to earlier recognition of these complications and fewer adverse outcomes.<sup>27</sup>

#### Airway Obstruction

Airway obstruction in unconscious patients is most commonly due to the tongue falling back against the posterior pharynx.<sup>29</sup> Other causes include laryngospasm, glottic oedema, secretions, vomitus, or blood in the airway; or external pressure on the trachea (most commonly from a neck haematoma).<sup>30,31,32</sup>

Partial airway obstruction presents as sonorous respiration, while total obstruction causes cessation of airflow, an absence of breath sounds, and marked paradoxical (rocking) movement of the chest. The abdomen and chest should normally rise together during inspiration; however, with airway obstruction, the chest descends as the abdomen rises during each inspiration (paradoxical chest movement).<sup>33</sup> Patients with airway obstruction should receive supplemental oxygen while corrective measures are undertaken.<sup>31</sup>

A combined jaw-thrust and head tilt manoeuvre pulls the tongue forward and opens the airway. Insertion of an oral or nasal airway also often alleviates the problem. Nasal airways may be better tolerated than oral airways by patients during emergence and lessen the likelihood of trauma to the teeth when the patient bites down.<sup>33</sup>

If these manoeuvres fail, laryngospasm should be considered. Laryngospasm is usually characterized by high-pitched crowing noises but may be silent, with complete glottic

closure. Spasm of the vocal cords is more likely to occur following airway trauma or repeated instrumentation or stimulation from secretions or blood in the airway.<sup>33</sup> The jaw-thrust manoeuvre, especially when combined with gentle positive airway pressure via a tight fitting facemask, usually breaks laryngospasm. Insertion of an oral or nasal airway is useful in ensuring a patent airway down to the level of the vocal cords. Any secretions or blood in the hypo-pharynx should be suctioned to prevent recurrence.

Refractory laryngospasm respond to aggressive treatment with a small dose of succinylcholine (0.1-0.2mg/kg) and temporary positive-pressure ventilation with 100% oxygen to prevent severe hypoxemia.<sup>33, 34</sup> Endotracheal intubations may occasionally be necessary to re-establish ventilation, while cricothyrotomy or transtracheal jet ventilation are indicated if intubation is unsuccessful in such circumstances.<sup>34</sup>

Glottic oedema following airway instrumentation is an important cause of airway obstruction in infants and young children.<sup>35</sup> Intravenous glucocorticoids (dexamethasone 0.25mg/kg) or aerosolised racemic epinephrine (0.5ml of a 2.25% solution with 3ml of normal saline) may be useful in such cases.<sup>36</sup>

Postoperative wound haematomas following head and neck, thyroid and carotid procedures can quickly compromise the airway; opening the wound immediately relieves tracheal compression.<sup>31, 32</sup> Rarely, gauze packing may be unintentionally left in the hypo pharynx following oral surgery and can cause immediate or delayed complete airway obstruction.<sup>34</sup>

### Hypoventilation

Hypoventilation, which is generally defined as a PaCO<sub>2</sub> greater than 45mmHg, is a common occurrence following general anesthesia.<sup>24</sup> Significant hypoventilation is usually clinically apparent when the PaCO<sub>2</sub> is greater than 60mmHg or arterial blood pH is less than 7.25. Signs are varied and include excessive or prolonged somnolence, airway obstruction, slow respiratory rate, and tachypnea with shallow breathing or

laboured breathing. Mild to moderate respiratory acidosis causes tachycardia and hypertension or cardiac irritability via sympathetic stimulation, but a more severe acidosis produces circulatory depression.<sup>37</sup>

Hypoventilation in the recovery room is most commonly due to the residual depressant effects of anaesthetic agents on respiratory drive.<sup>22</sup> Opioid-induced respiratory depression characteristically produces a low respiratory rate, often with large tidal volumes.<sup>38</sup> Biphasic or recurring patterns of respiratory depression have been reported with all opioids. Proposed mechanisms include variations in intensity of stimulation during recovery and delayed release of the opioids from peripheral compartments such as skeletal muscle or possibly the lungs with fentanyl as the patient re-warms or begins to move.<sup>39, 40</sup>

Inadequate neuro-muscular blockade reversal or overdose, hypothermia, pharmacological interactions such as with amino-glycoside antibiotics or magnesium therapy, altered volumes of distribution, renal or hepatic dysfunction, or metabolic factors such as hypokalaemia or respiratory acidosis can be responsible for residual muscle paralysis in the recovery room.<sup>41</sup> Regardless of the cause, uncoordinated breathing movements with shallow tidal volumes and tachypnea are usually apparent. The diagnosis can be made with nerve stimulator in unconscious patients; fully awake patients can be asked to lift their head.<sup>41, 42</sup> The ability to sustain a head-lift for 5 seconds may be the most sensitive test for assessing the adequacy of neuro-muscular blockage reversal.<sup>41</sup>

Splinting due to incision pain and diaphragmatic dysfunction following upper abdominal or thoracic surgery, abdominal distension, or tight abdominal dressings are other factors that can contribute to hypoventilation. Increased CO<sub>2</sub> production from shivering, hyperthermia or sepsis can also increase PaCO<sub>2</sub> even in normal patients recovering from general anesthesia.<sup>43, 44, 45</sup> Marked hypoventilation and respiratory acidosis can result when these factors are superimposed on an impaired ventilatory reserve due to underlying pulmonary, neuromuscular or neurological disease. Treatment is generally

directed at the underlying cause, but marked hypoventilation always requires supported/controlled ventilation until contributory factors are identified and corrected.<sup>42</sup>

## Hypoxemia

Mild hypoxemia is common in patients recovering from anaesthetic unless supplemental oxygen is given during emergence.<sup>3</sup> Mild to moderate hypoxemia ( $\text{PaO}_2$  50mmHg) in young healthy patients may be well tolerated initially, but with increasing duration or severity the initial sympathetic stimulation often seen is replaced with progressive acidosis and circulatory depression.<sup>46, 47, 48</sup> Cyanosis may be absent if the haemoglobin concentration is reduced.

Clinically, hypoxemia may also be suspected from restlessness, tachycardia or cardiac irritability (ventricular or atrial arrhythmias).<sup>48</sup> Obtundation, bradycardia, hypotension and cardiac arrest are late signs.

Hypoventilation and increased right-to-left intrapulmonary shunting or both usually cause hypoxemia in the recovery room. A decrease in cardiac output or an increase in oxygen consumption as with shivering will accentuate the hypoxemia.<sup>48</sup> Diffusion hypoxia is an uncommon cause of hypoxemia when recovering patients are given supplemental oxygen.

Increased intrapulmonary shunting from a decreased functional residual capacity (FRC) relative to closing capacity is the most common cause of hypoxemia following general anaesthesia.<sup>11</sup> The greatest reductions in FRC occur following upper abdominal and thoracic surgery. The loss of lung volume is often attributed to micro-atelectasis; since visible atelectasis is often not evident on a chest x-ray.<sup>28</sup> A semi-upright position helps maintain FRC. Causes of atelectasis include prolonged intra-operative hypoventilation with low tidal volumes, unintentional endo-bronchial intubation or obstruction by secretions or blood, pulmonary aspiration, or pulmonary oedema.<sup>11, 34</sup>



## **CIRCULATORY COMPLICATIONS**

### **Hypotension**

Hypotension is usually either due to decreased venous return to the heart or left ventricular dysfunction. Absolute hypovolemia can result from inadequate intraoperative fluid replacement, continuing fluid sequestration by tissues or wound drainage, or postoperative bleeding. Vasoconstriction during hypothermia may mask the hypovolemia until the patient's temperature begins to rise again; subsequent vasodilatation results in delayed hypotension.<sup>49</sup>

Mild hypotension during recovery from anaesthesia is common and usually reflects the decrease in sympathetic tone normally associated with sleep or residual effects of anaesthetic agents: it typically does not require treatment. Significant hypotension is defined as a 20%-30% reduction of blood pressure<sup>49</sup> below the patient's baseline level and indicates a serious derangement requiring treatment. An increase in blood pressure following a fluid bolus (crystalloid or colloid) confirms hypovolemia. Severe hypotension will require a vasopressor or inotrope such as dopamine or epinephrine to increase arterial blood pressure until the intra-vascular volume deficit is corrected.

### **Hypertension**

Noxious stimulation from incisional pain, endo-tracheal instrumentation/intubation or bladder distension are usually responsible.<sup>50</sup> Post-operative hypertension may also reflect sympathetic activation, which may be part of the neuro-endocrine response to surgery or secondary to hypoxemia, hypercapnia or metabolic acidosis. Uncontrolled or undetected endocrine dysfunction may be revealed by surgery.

## Arrhythmias

Residual effects from anaesthetic agents, increased sympathetic nervous system activity, other metabolic abnormalities and pre-existing cardiac or pulmonary disease predispose to arrhythmias in the recovery room.<sup>20, 21, 51</sup> Bradycardia often represents the residual effects of a cholinesterase inhibitor (neostigmine), a potent opioid or a beta-adrenergic blocker. Tachycardia may represent the effect of an anticholinergic agent (atropine), a vagolytic drug (pancuronium or meperidine), a beta-agonist, reflex tachycardia (hydrallazine), in addition to more common causes such as pain, fever, hypovolemia and anaemia.<sup>20</sup>

Arrhythmias may result in inadequate cardiac output, insufficient tissue perfusion and if prolonged may result in malignant fatal cardiac dysrhythmias such as ventricular fibrillations.

## NON-INVASIVE MONITORING OF OXYGEN IN BLOOD.

### PULSE OXIMETRY

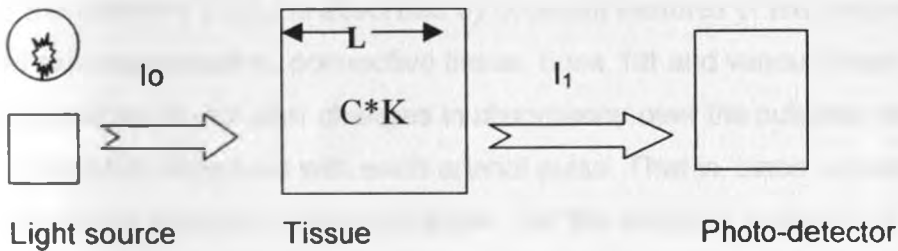
Pulse oximetry has become a standard for clinical monitoring in ICUs, emergency rooms, and operating rooms.<sup>52, 53, 54</sup> However, its development and evolution took place over many years. The first oximeter was described in 1935. However the technology was not developed further because of the cumbersome equipment involved and the contemporaneous development of the Clark electrode, which made blood oxygen partial pressures ( $PO_2$ ) measurement accurate and convenient. It was not until many years later with the development of light-emitting diodes (LEDs) and microprocessors that pulse oximetry became practical.<sup>55</sup> With proper use, pulse oximetry is an efficient, accurate and cost-effective monitoring device that is simple to use and non-invasive.<sup>56,</sup>

57, 58, 59

## THEORY OF OPERATION

Current pulse oximeters function simultaneously as optical plethysmographs and two-wavelength spectrophotometers.<sup>60, 61</sup> As a plethysmograph, the pulse oximeter detects the pulsatile increase in blood volume as increased optical density. As a spectrophotometer, the pulse oximeter compares light absorbance at two wavelengths during the pulsatile increase in the optical density to absorption characteristics that occur during the trough or diastolic portion of the cycle to quantify haemoglobin saturation.

Pulse oximetry is based on Beer-Lambert law that relates the concentration of a solute to the amount of light it absorbs at a particular wavelength.<sup>61</sup>



**Fig 1**

$I_0$  is the intensity of incident light,

$I_1$  is the intensity of the transmitted light at the photo-detector,

$L$  is the distance, through which light travels through the substance,

$C$  is the concentration of the absorbing species, and

$K$  is the extinction coefficient or absorptivity of the species of interest.

In tissue, light scatters and diffuses, and  $L$  is not equal to the distance from the light source and the detector.

The probe of the pulse oximeter is composed of two light sources and a photodiode (detector), which are placed across the pulsatile bed. The two light sources, LEDs with maximal light emission at 660nm (red) and 940nm (infrared), are alternately turned on at kilohertz frequencies. The frequencies are chosen because oxyhaemoglobin absorbs

relatively more light in the infrared region and reflects red light. Conversely, deoxyhaemoglobin absorbs, substantially more red light than oxyhaemoglobin and reflects infrared light. The pulsatile waveform is usually well defined at these two wavelengths.<sup>61</sup>

The microprocessor alters the intensity of the LEDs according to the thickness and the optical density of the monitored tissue to keep the intensity of the light reaching the photodiode in a narrow range. Thus, variations in skin pigmentation, finger size, and fat content are compensated for by the self-adjusting nature of pulse oximeters.<sup>60</sup> In addition oximeters turn off the LEDs during a portion of the cycle to compensate for ambient light interference.

Most of the oximeter's light is absorbed by constant features of the anatomy, including skin and skin pigmentation, connective tissue, bone, fat and venous blood. Thus, these constant features do not alter changes in absorbance over the pulsatile wave. The total optical absorption increases with each arterial pulse. That is, blood volume in the monitored tissue increases with each pulse, and the increase is assumed to be due solely to arterial blood. The oximeter's microprocessor compares the absorption patterns of LED during the pulsatile wave (systolic to diastolic region of the waveform) and then compares the two ratios to derive a third and final ratio.

This final ratio is not necessarily linked to particular oxygen saturation by Beer-Lambert law. Commercially available pulse oximeters have been calibrated over a wide range of haemoglobin saturation from normal volunteers. The ratio of absorbance is referenced to simultaneous in vitro oxygen saturation measurements performed with a spectrophotometric oximeter.<sup>59, 60</sup> The calibration curve obtained is then incorporated into the pulse oximeter's microprocessor algorithms.

## LIMITATIONS

The limitations are related to accuracy, alterations in absorbing species, motion or venous pulse artefacts, low perfusion states, external light sources, and interpretation errors.

A). Accuracy: The accuracy of pulse oximetry has been compared to in vitro spectrophotometric oximeters by several investigators. In general, most pulse oximeters have an excellent correlation with in vitro methods at saturations between 70% and 100%.<sup>62</sup> However, pulse oximeters may be inaccurate in the lower saturation ranges.<sup>63</sup>

B) Rapid transient haemoglobin desaturations that occur in patients may not be detected by pulse oximeters.<sup>63</sup> This is because pulse oximeters are designed to average saturation values over time to produce stable results with minimal beat-to-beat variability, which could complicate oximetry readings; it also decreases the response time of the oximeter. Newer instruments have adjustable time constants to help in preventing this shortcoming.<sup>63 64</sup>

C) Alterations in absorbing species: Because the pulse oximeter uses only two wavelengths, oxyhaemoglobin and deoxyhaemoglobin are the only moieties that can be assessed. Thus, other haemoglobin species such as met haemoglobin, carboxyhemoglobin and sulfhaemoglobin are not measured with a pulse oximeter.<sup>65</sup>  
<sup>66 57</sup>

Like oxyhaemoglobin, carboxyhemoglobin absorbs relatively less red light than does deoxyhaemoglobin. The oximeter will report falsely elevated oxygen saturation in the presence of an elevated carboxyhemoglobin. Methaemoglobin and sulfhaemoglobin have similar absorption patterns. Both absorb more red light than deoxyhaemoglobin. The net effect of increased absorption at both wavelengths is a gradual decrease of saturation reported by the pulse oximeter as the methaemoglobin fraction increases. This occurs regardless of actual oxyhaemoglobin fraction, with saturation of approximately 85% around a methaemoglobin level of 30%.<sup>65 66</sup>

Methylthionine blue artifactually lowers the oximeter value.<sup>67</sup>

D) Motion or venous artifact: Motion of either the probe or the monitored portion of the body will abruptly alter the path length of the light in the tissue,<sup>60</sup> erratic readings result. Noting the probe motion and failure of the pulse signal to correlate to the patient's actual pulse can identify the problem.

Strong venous pulsation can mimic an arterial pulse, particularly when the probe is placed in a central location such as the bridge of the nose or the ear lobe.<sup>58</sup> Thus, venous pulsations from severe tricuspid regurgitation or vigorous chest physiotherapy may be averaged with the arterial pulse by the oximeter, artifactually depressing the saturation value displayed by the oximeter. Some manufacturers offer an option to synchronize the pulse oximeter with the subject's electrocardiogram (ECG) as one strategy to improve signal fidelity and decrease the effect of motion or venous pulsations.<sup>60</sup>

E) Low Perfusion States: Artifactually depressed oximeter values occur in a variety of low perfusion states. This may result from one of two factors: The amplitude of the pulse signal, or the optical density of each pulse may be diminished to the point that background interference is counted as pulse.<sup>60</sup>

Vasoconstriction or vascular obstruction from any cause diminishes pulse amplitude. Efforts to prevent these problems include: Local warming, application of topical nitrates, digital nerve block of the monitored digit,<sup>68</sup> moving the probe to a more central location (nasal bridge, ear lobe) and synchronization of the oximeter to the subject's ECG. Severe forms of anaemia are associated with artifactual depressions in the oximeter value, particularly when oxyhaemoglobin saturations are less than 90%.

F) External Light Sources: If the photo-detector is overwhelmed with light, a pulse signal will not be accurately detected. Sources of interfering external light include surgical lights, bilirubin lights, infrared warming lamps, and sunlight.<sup>70, 71</sup>

G) Interpretation errors: The most common mistake in using pulse oximetry is to be overconfident in the reported values. A pulse oximeter is a trend indicator and does not report absolute measurements.<sup>72</sup>

According to the normal oxyhaemoglobin dissociation curve, which relates oxygen saturation of haemoglobin to partial pressures of oxygen ( $PO_2$ ), a saturation of 90% corresponds to a  $PO_2$  of 60mmHg.<sup>18</sup> This is adequate for oxygen transport, given sufficient haemoglobin and cardiac output. The oxyhaemoglobin dissociation curve and therefore haemoglobin's affinity for oxygen is shifted by several variables including acidosis, alkalosis, hypercarbia, and temperature. Thus, the assumption of an adequate arterial  $PO_2$  based on "acceptable" pulse oximeter saturation is unreliable in patients with abnormal temperature or certain metabolic derangements. Adequate arterial  $PO_2$  should be verified, particularly when oximeter saturation values are marginal (e.g. 90% to 93%). Furthermore, adequacy of tissue oxygenation is not assessed by a pulse oximeter. A patient may appear "well saturated" in the presence of hypotension, anaemia, lactic acidosis, and inadequate cardiac output.<sup>58, 63</sup>

H) Finally, the presence of carboxyhaemoglobin cannot be excluded by pulse oximetry or routine ABGs, two circumstances in which falsely normal oxyhaemoglobin saturations are seen.<sup>66</sup>

### TRANSCUTANEOUS OXIMETRY

Indirect estimates of arterial and tissue oxygen tension can be made by transcutaneous oximetry. The miniaturized heated Clark electrode was developed in the early 1970's and is now used primarily for assessing tissue oxygenation in extremities.<sup>73-74</sup>

Careful studies document that transcutaneous oxygen measurements ( $PtCO_2$ ) correlate well with  $PaCO_2$ .<sup>75</sup> In neonates the correlation is excellent, but  $PtCO_2$  is less reliable in children and adults because of the thicker skin and the pathologic states that can affect

the monitored tissue.<sup>67, 68</sup> In critically ill adults, the correlation between transcutaneous oximetry and PaO<sub>2</sub> are unreliable.

The transcutaneous oximeter may measure local tissue PO<sub>2</sub> and not track arterial oxygen tension. The fact that transcutaneous oximetry may reflect the local balance between oxygen delivery and consumption could potentially be used to detect the onset of vascular compromise or shock. However, the heating element applied to the skin overrides the usual vasoconstrictive reflexes and thus decreases the method's sensitivity to the onset of shock or vascular compromise.<sup>69, 73, 75</sup> Furthermore, individuals with compromised circulation or atrophic skin may sustain burns from the device.<sup>73</sup>

Other limitations of the device include the requirement for frequent calibration, relatively slow response time in detecting hypoxemia, and a relatively long calibration time once the sensor is placed or moved.<sup>73, 74</sup>

### TRANSCONJUNCTIVAL PARTIAL PRESSURE OF OXYGEN

This method measures conjunctival PO<sub>2</sub> with a miniature polarographic electrode embedded in polymethyl methacrylate, placed against the lateral aspect of the superior palpebral conjunctiva. Transconjunctival electrodes were developed, as an alternative to PtCO<sub>2</sub> electrode because heating of the conjunctiva is not required.

Therefore, there are fewer measuring artifacts with this method, unless there is a decrease in conjunctival temperature.<sup>76</sup> Transconjunctival PO<sub>2</sub> seems to have the same limitations as PtCO<sub>2</sub> measurements because it depends on local and systematic changes in circulation. Therefore, although it can be a sensitive but non-specific indicator of local hypo perfusion in the initial stages of resuscitation, this method has not gained wide acceptance.<sup>77</sup>



## FUTURE DEVELOPMENTS

A prototype reflectance mode device that can be placed on the trunk has been developed and may possibly be less prone to artifact.<sup>74</sup>

There is also the development of in vivo near-infrared spectroscopy capable of non-invasively monitoring the state of oxygenation of deep tissues such as skeletal muscle or brain.<sup>74</sup>

Reflectance pulse oximetry combined with laser Doppler flow measurements may simultaneously assess cutaneous or mucosal perfusion and haemoglobin saturation.<sup>75</sup> However these latter four monitoring devices are not available in Kenya at present.

## AIMS AND OBJECTIVES

### Main objective

- ◆ To investigate post-operative hypoxia after ENT surgery.

### Specific objectives

- ◆ Investigate the prevalence of post-operative hypoxia,
- ◆ Investigate the degree of post-operative hypoxia,
- ◆ Investigate the duration of post anaesthetic hypoxia in otherwise healthy infants and children who are not given supplemental oxygen in the post anaesthetic recovery room.

## STUDY JUSTIFICATION

Hypoxemia is a potentially serious post-operative complication. A study done at Kenyatta National Hospital in adult patients after abdominal surgery showed oxygen desaturation incidence of 31%.<sup>78</sup> There are no studies in post-operative hypoxemia in paediatric patients that has been carried out in the Kenyan set up.

The problems found in paediatric patients may be considerably different from those in adults. Previous reports on post-operative hypoxia experience and problems associated with recovery from anaesthesia have been heavily biased towards an adult population. Several studies conducted to determine post-operative hypoxemia and possible associated factors have resulted in varied conclusions.<sup>3, 79, 80, 81</sup>

In one study only 25 (16%) out of the 152 children in the study had undergone ENT surgery and there was an occurrence of hypoxemia in 8 (5.26%) of them.<sup>82</sup> No such study has been done in Kenya. The study aims to elucidate post-operative hypoxia in children after ENT procedures and to use the information gathered to enhance post-operative care.

## SAMPLE SIZE

Data from previous results used to calculate the sample size.<sup>82</sup> To obtain a 95% confidence interval using a precision of 5% the required sample size  $n$  is calculated using the formula:

$$n = \frac{(Z_{\alpha/2})^2 (p)(1-p)}{d^2}$$

Where

$Z_{\alpha/2}$  is the critical value, the positive  $z$  value that is at the vertical boundary for the area of  $\alpha/2$  in the right tail of the standard normal distribution.<sup>83, 84</sup>

$p$  is the prevalence

$n$  is the sample size.

$d$  is the margin of error.

A 95% degree confidence corresponds to  $\alpha = 0.05$ . In the Table of the standard normal distribution this corresponds to a  $z$  value of 1.96. The critical value is therefore  $Z_{\alpha/2} = 1.96$ .

Using the formula for sample size, we can calculate:

$$n = \frac{(Z_{\alpha/2})^2 (p)(1-p)}{d^2} = \frac{(1.96)^2 (0.0526)(0.9474)}{(0.05)^2} = 76.5757$$

The sample size required is 77.

## METHODOLOGY

Patients were recruited from the ENT paediatric clients' scheduled for elective surgical ENT procedures. After carefully explaining to the guardians (see consent explanation) the nature of investigation being carried out, informed consent was obtained.

Pre-operative assessment was carried out on the child in the waiting area/receiving area 15 minutes before anaesthesia. The pre-operative review also involved measurement of preoperative arterial oxygen saturation (SaO<sub>2</sub>), Weight, and review of haematological and biochemical profiles, that are routinely done pre-operatively.

After the operation further observations were taken in the recovery room after the attending anaesthesiologists released the patient to the recovery area. Immediate pulse oximeter measurements were taken and at intervals of 10 minutes for the next 30 minutes. The position of the patient, degree of airway obstruction, level of consciousness and presence of postoperative complications was recorded.

Adequacy of ventilation was assessed clinically by observing the expiratory airflow from the nose or mouth and movement of the thorax, with or without auscultation with a stethoscope.

Degree of airway obstruction was scored as: absent, mild (light snoring with adequate ventilation), moderate (necessitating a position change or a naso-pharyngeal airway) and severe (necessitating re-intubation's).<sup>3</sup>

The technique of anaesthesia was noted whether it was spontaneous or assisted ventilation and the duration of surgery.

Supplementary oxygen in the recovery room was only to be administered in case of significant desaturation/airway compromise.

## **STUDY AREA**

The study was done at Kenyatta National Hospital ENT theatre from August 2003 to February 2004 after obtaining approval from the ethical and research committee (see appendix).

## **STUDY DESIGN**

The study was prospective descriptive design.

## **Inclusion criteria**

The following was the inclusion criteria used:

1. Paediatric patients scheduled for ENT surgery
2. ASA I and II patients
3. No haemoglobinopathies
4. Haemoglobin >10gm/dl
5. No cardiovascular anomaly
6. Consent

## **Exclusion Criteria**

The following was the exclusion criteria used:

1. Cardiovascular disease with cyanosis
2. Pulmonary dysfunction.
3. Vascular disease
4. Emergency cases
5. Lack of consent
6. ASA class III and above

## **DATA COLLECTION AND ANALYSIS**

Data collection was done through a coded questionnaire (Appendix). This was then entered in computer and stored in both hard disk and floppy diskettes. Data was analysed using Statistical Package for Social Sciences (SPSS Version 11) programmes and presented in Histograms and pie charts. P value of less than 0.05 was considered significant.

**Materials:** The following were the tools used to carry out the study; Weighing scale, Pulse oximeter and ECG monitor

## **ETHICAL CONSIDERATIONS**

Pulse oximetry is non-invasive clinical examination that poses no risk to patients.

Patients were not unduly exposed to hypoxic phases.

The study was carried out with consent from the parents / guardians after careful explanation.

All information was treated in medical confidence.

Consent was given from the ethics and research committee before embarking on the study.

## RESULTS

A total of 90 children were seen with ages of between 9 months and 144 months with a mean age of 38 months. (Fig1). Of these 55 were male and 35 female.

### PATIENTS CHARACTERISTICS

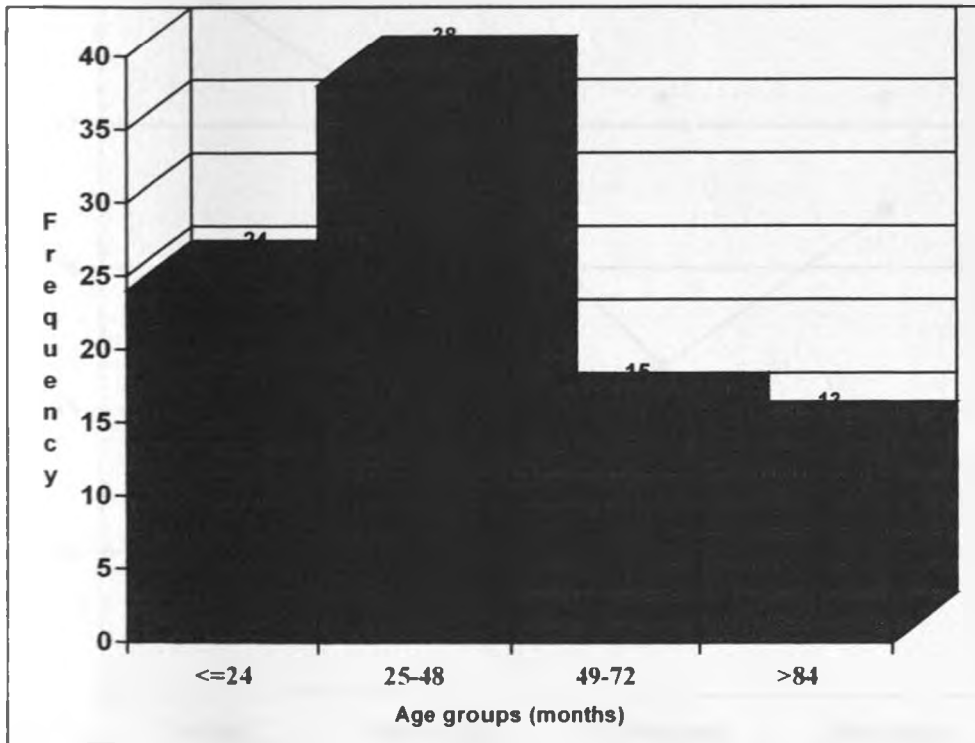


Figure 1: Age distribution

57% of the patients were ASA I while 43% were ASA II. There was no significant difference in pre operative oxygen saturation between the two groups ( $p=0.346$ ) with majority (63.9%) having saturations  $>97\%$ .

The weights of the patients ranged from 6.6Kgs to 30Kgs with a mean of 13.3Kgs while the haemoglobin levels ranged between 10.0 and 15.7g/dl with a mean of 12g/dl.

## ANAESTHETIC TECHNIQUE

All the patients were premedicated with atropine and none received opioids pre-operatively.

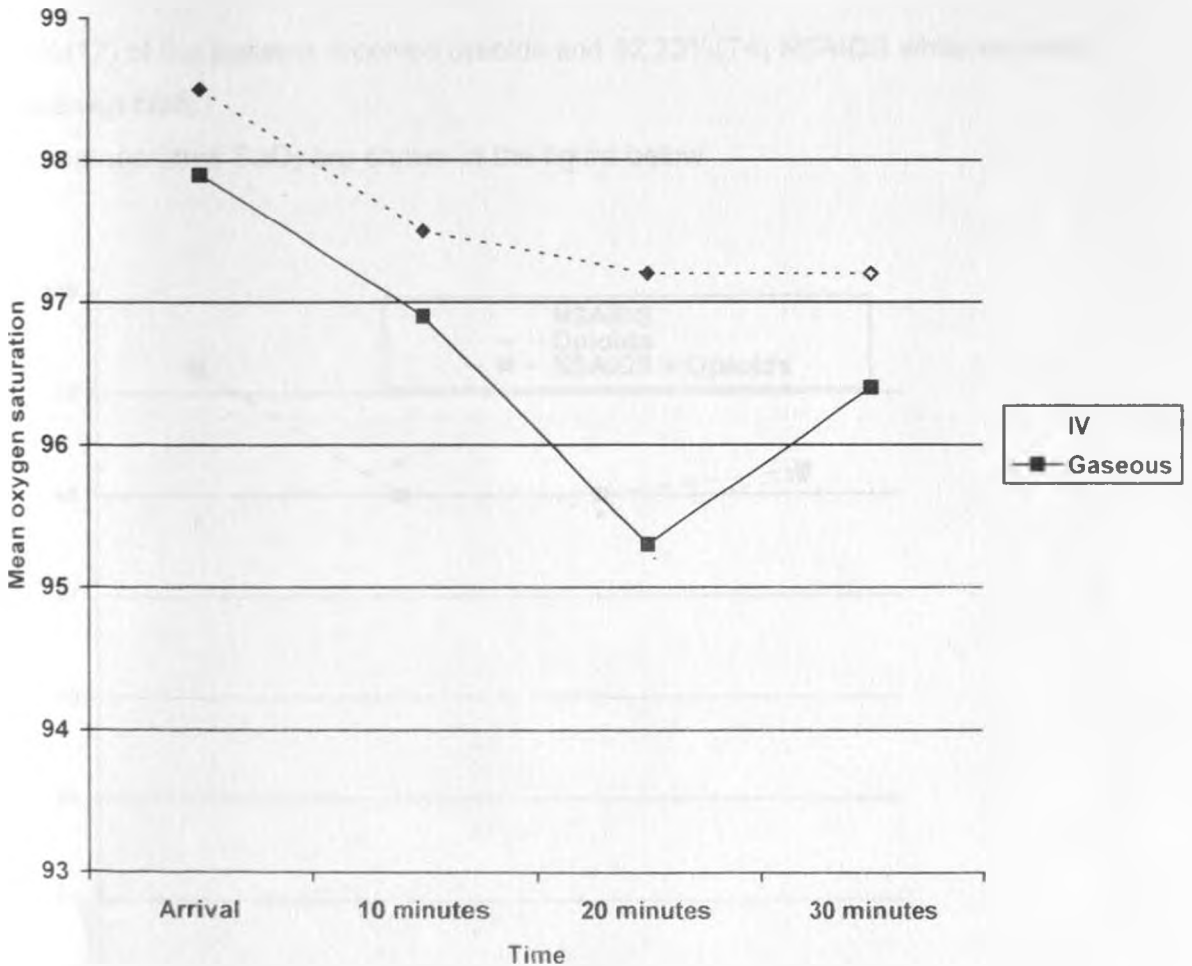


Figure 2 Mean oxygen saturation by induction agents

Most patients 90.9% (82) had inhalation agents used for induction while 1.8% (2) had intravenous agents used and 7.3% (6) had both intravenous and inhalational agents used. All patients received nitrous oxide and halothane for maintenance of anaesthesia during the procedure and all of them had tracheal intubation.



The postoperative mean SaO<sub>2</sub> was slightly lower for those children who were induced with gaseous agents as compared with those induced using intravenous agents (Fig 2).

## ANALGESIA

13.33%(12) of the patients received opioids and 82.23%(74) NSAIDS while 4(4.44%) received both.

Their postoperative SaO<sub>2</sub> are shown in the figure below.

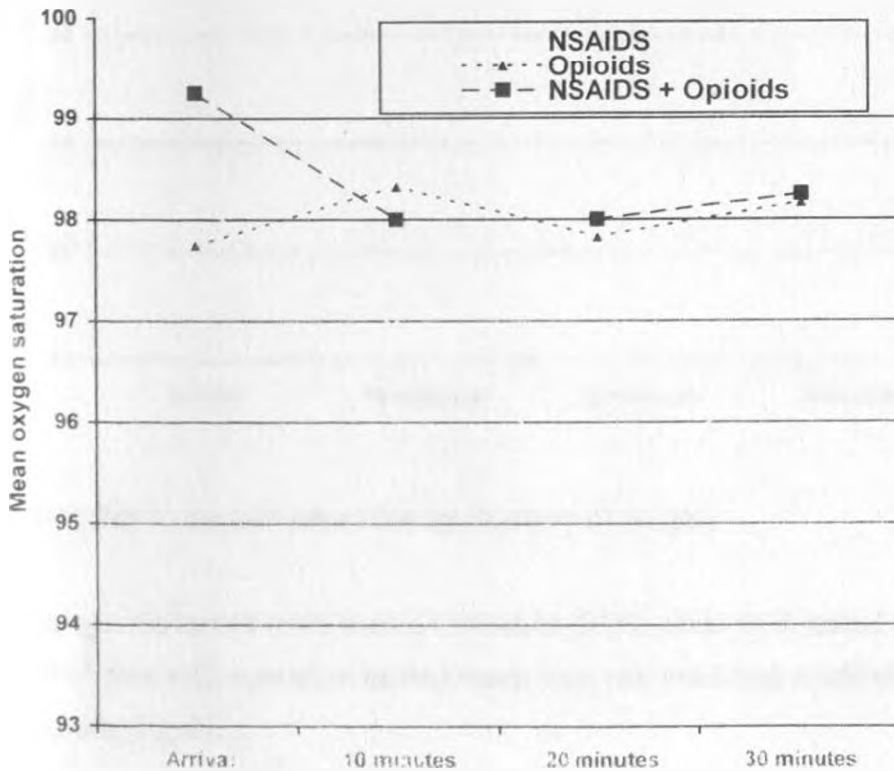


Figure 3 SaO<sub>2</sub> trend in relation to analgesic agent used

One patient who received pethidine intraoperatively had SaO<sub>2</sub> of 90% on arrival at the recovery room but this picked up after 10 minutes and remained above 97% for the rest of the observation period.

## DURATION OF SURGERY

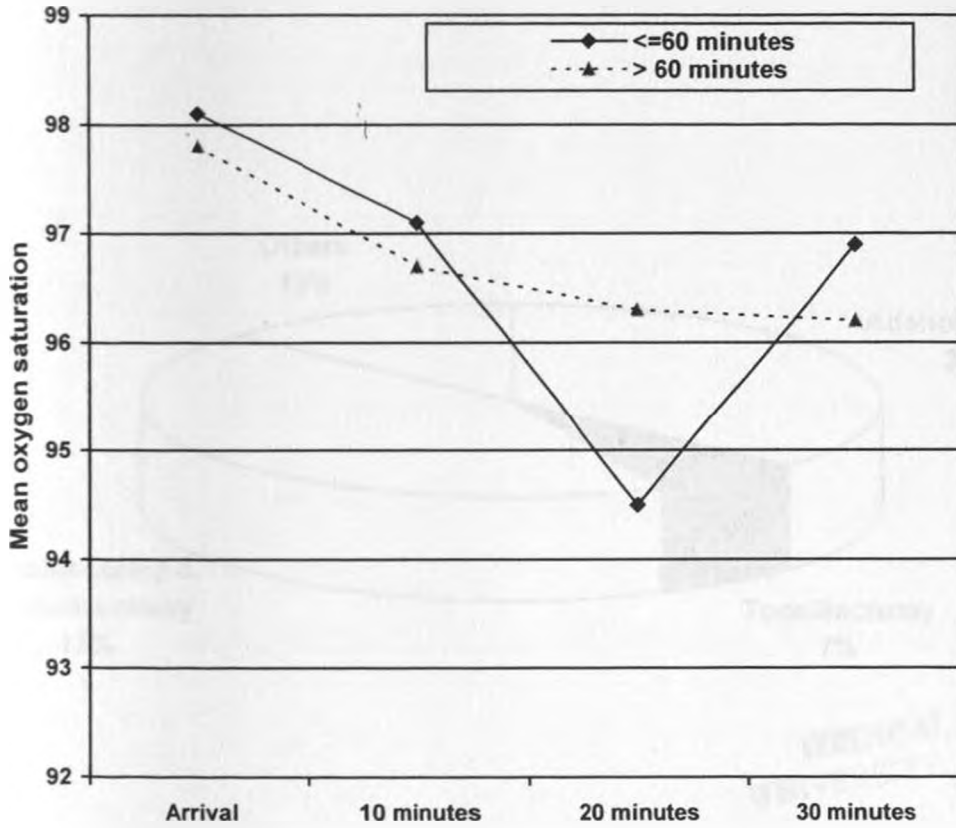
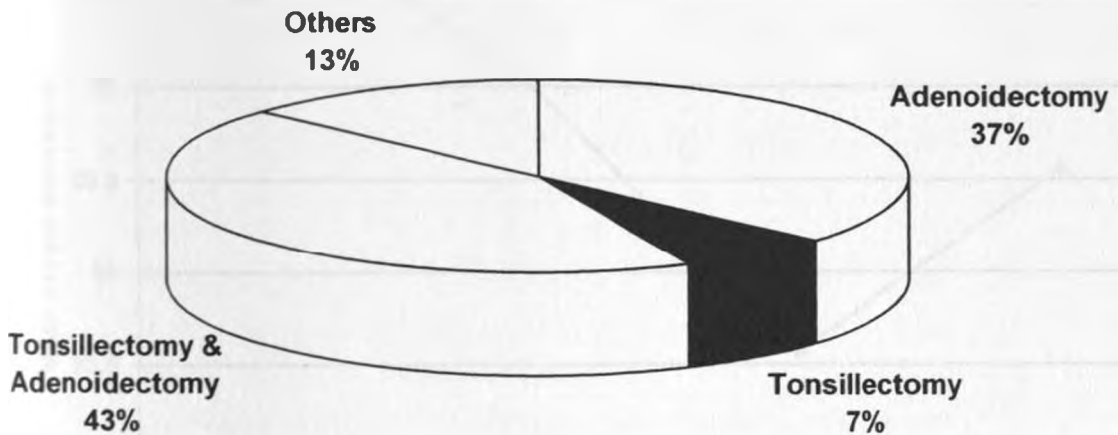


Figure 4: Mean oxygen saturation by duration of surgery

Most surgeries lasted more than 61 minutes (54%) while 46% lasted less than one hour. Patients with operation lasting more than one hour had relatively lower oxygen saturations (Fig 4).

## TYPES OF PROCEDURES



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Figure 5. Pie chart showing the types of procedures performed

The most common surgery done was both the tonsillectomy and adenoidectomy (43.3%) followed by adenoidectomy (36.7%). Other operations included sub mucosal diathermy for hypertrophy of inferior turbinates and a combination of three procedures that is tonsillectomy, adenoidectomy and sub mucosal diathermy.

POSTOPERATIVE SaO<sub>2</sub> TRENDS IN ALL CHILDREN

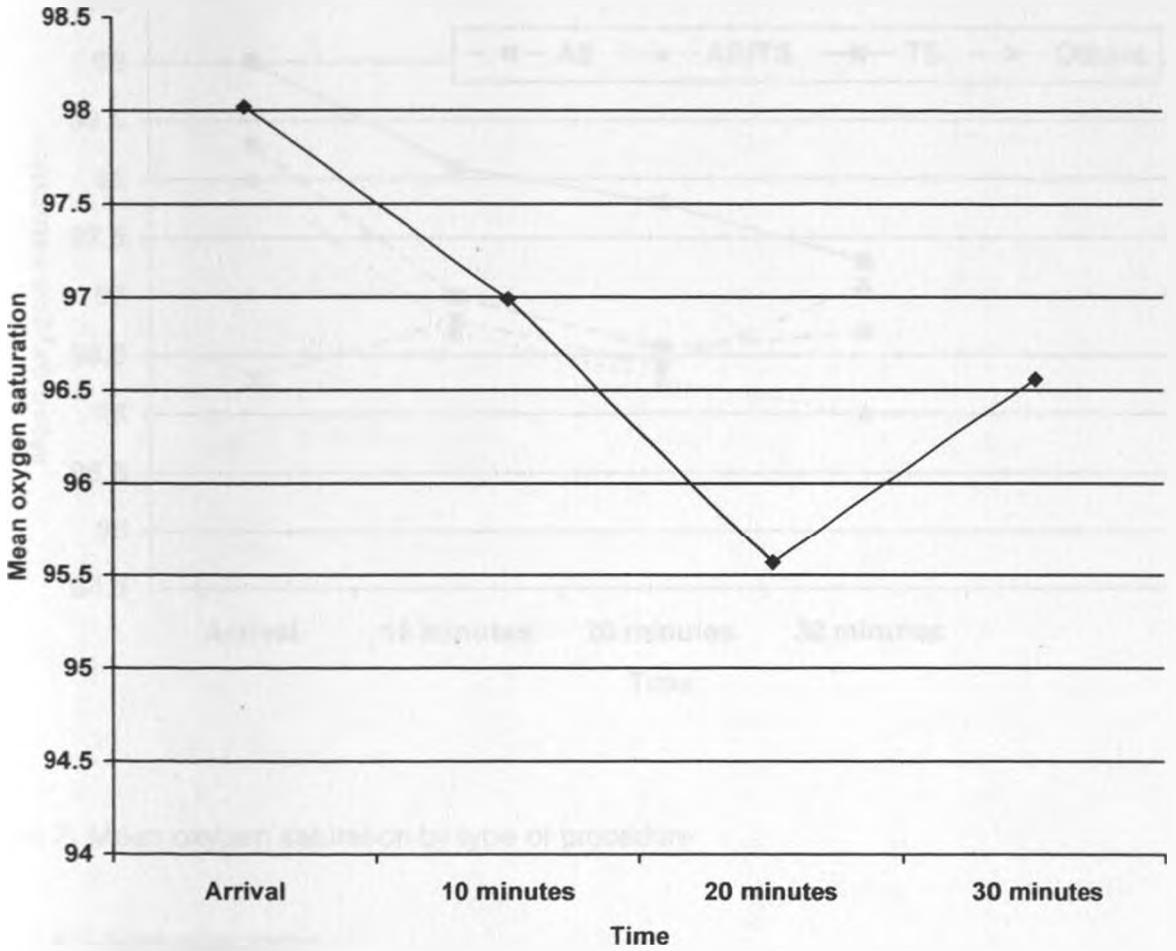


Figure 6: Mean oxygen saturation

The general trend of SaO<sub>2</sub> over the 30 minutes observation period was a gradual decrease until after 20 minutes when it began to stabilise or slightly increase (Fig 6).

## SaO<sub>2</sub> TREND BY TYPE OF PROCEDURE

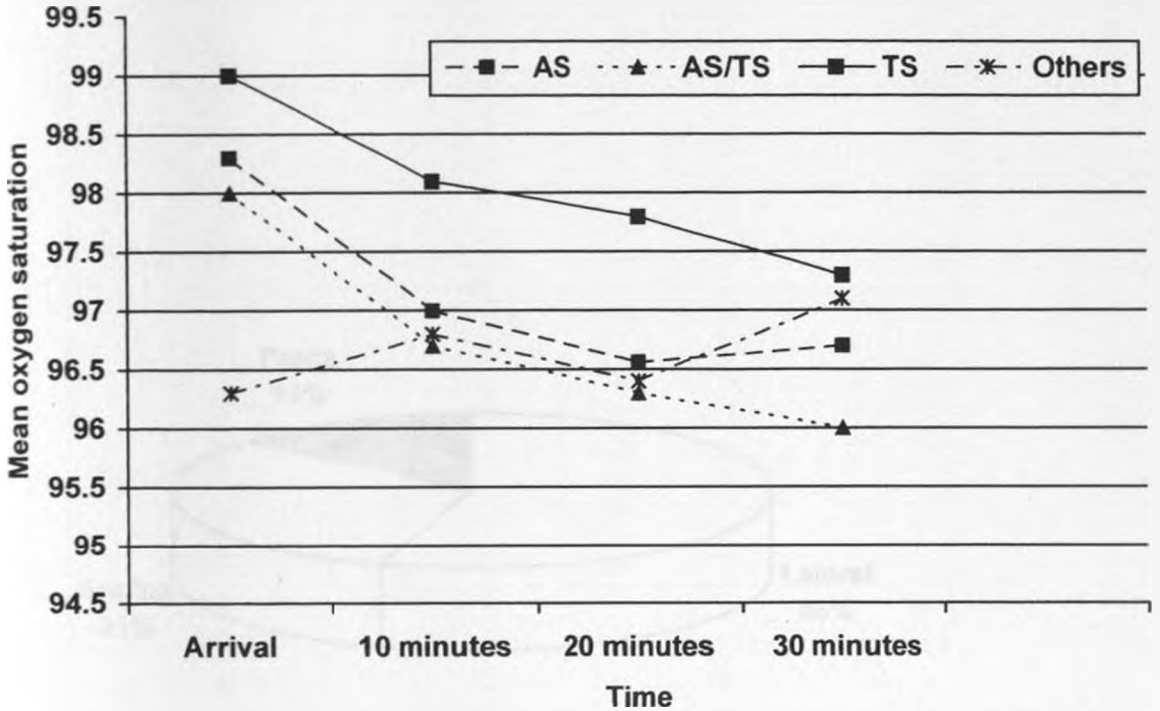


Figure 7: Mean oxygen saturation by type of procedure

Key AS-Adenoidectomy  
TS-Tonsillectomy  
AS/TS-Adenoidectomy and tonsillectomy

The mean saturations at arrival in recovery area were highest in patients who had tonsillectomy done and remained so for the period of observation. A general trend of decline was seen in these and also those having had adenoidectomy as well. Other surgeries performed had patients with relatively lower SaO<sub>2</sub> levels but these gradually improved over the 30 minutes observation period to match those of tonsillectomy patients (Figure 7).

## POSITION IN RECOVERY

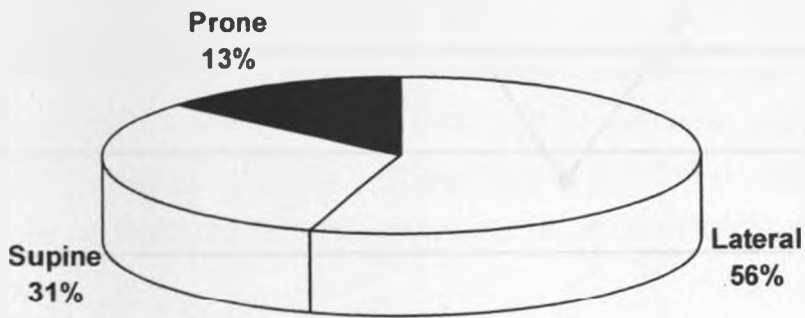


Figure 8. Pie chart showing the recovery position.

Most patients arrived at recovery in the lateral position (55.1%), 13.5% in prone and 31.5% in supine position (Figure 8).

SaO<sub>2</sub> TREND BY POSTOPERATIVE POSITION

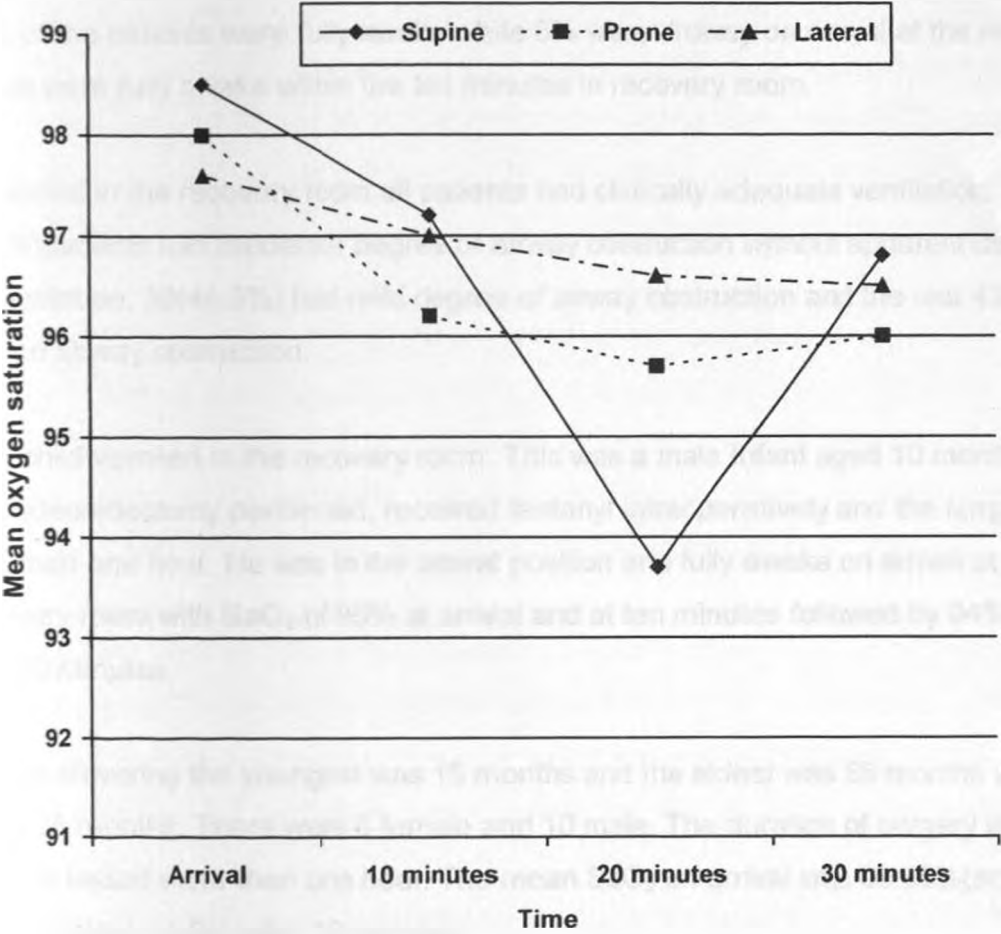


Figure 9: Mean oxygen saturation by position of patient

Children in the lateral position had minimal variation in their postoperative mean SaO<sub>2</sub> unlike those in the supine position who showed a drop in SaO<sub>2</sub> between 10 and 20 minutes of up to 4% but recovered to 97% at 30 minutes as shown in Fig 9.

## POST OPERATIVE COMPLICATIONS

92% of the patients were fully awake while 8% were drowsy on arrival at the recovery but all were fully awake within the ten minutes in recovery room.

On arrival in the recovery room all patients had clinically adequate ventilation. Two (2.3%) patients had moderate degree of airway obstruction without apparent disturbance in ventilation, 39(44.3%) had mild degree of airway obstruction and the rest 47(53.4%) had no airway obstruction.

One child vomited in the recovery room. This was a male infant aged 10 months who had adenoidectomy performed, received fentanyl intraoperatively and the surgery lasted less than one hour. He was in the lateral position and fully awake on arrival at the recovery room with SaO<sub>2</sub> of 95% at arrival and at ten minutes followed by 94% at 20 and 30 minutes.

16 had shivering the youngest was 15 months and the eldest was 55 months with mean of 39.25 months. There were 6 female and 10 male. The duration of surgery in 12(75%) of them lasted more than one hour. The mean SaO<sub>2</sub> on arrival was 96.06%(sd =1.81) and 95.86(sd =1.50) after 10 minutes.

Two children complained of pain. A girl aged 8 years who had both adenoidectomy and tonsillectomy performed on her. She was induced with intravenous agents and given only NSAIDs and the operation lasted over 61 minutes. Her oxygen saturation on arrival in the recovery room was 99%, then 97% at 10 minutes followed by 94% at 20 and 30 minutes later. The other child was a 3 year 10 months old female after tonsillectomy and adenoidectomy but her SaO<sub>2</sub> were between 97% and 99% during the period of observation.



## DISCUSSION

The study was limited to otherwise healthy children (ASA I and II). Such patients do not ordinarily receive supplementary oxygen in the recovery room. An increased tendency to airway closure has been previously demonstrated in small children.<sup>86</sup> In spite of these this study was unable to show any increased hypoxemia in children after ENT procedures.

The mean arterial oxygen saturation on arrival in the recovery room was 98.02(sd 1.84). This corresponds to a PaO<sub>2</sub> of 97.5mmHg assuming normal acid-base status and Hb-O<sub>2</sub> affinity. The saturations in most patients progressively decreased until after 20 minutes when it starts increasing. Desaturation after 10 minutes in recovery was significant (P=0.001) but not to the level of causing to hypoxemia.

The study intended to look at the incidence of postoperative hypoxemia and its duration after ENT surgeries at the Kenyatta National Hospital. Other studies done in the same institution involved abdominal operations, which showed an incidence of 30.9%,<sup>78</sup> other studies have showed great variances in incidences of postoperative hypoxemia.<sup>78, 82</sup>

Pulmonary gas exchange deteriorates during general anaesthesia. In adults the increase in P (A-a) O<sub>2</sub> continues into the early post operative period, even after minor surgical procedures, and may result in hypoxemia if patients are allowed to breathe room air.<sup>18,34,85</sup>

The pulse oximeter probe applied to a fingertip, senses changes in light absorption that occur synchronously with arterial pulsation. Thus the oximeter detects arterial rather than capillary haemoglobin saturation (SaO<sub>2</sub>), on a beat-to beat basis, without heating or arterialisation. Previous studies have established that the pulse oximeter accurately reflects SaO<sub>2</sub> in all age groups, with various hematocrits and in various positions, over a range of 70-100%.

The reliability of pulse oximetry can be affected by conditions that reduce vascular pulsation in the fingertip, such as hypothermia, hypotension and the use of vasoconstrictive drugs.<sup>62,68,69</sup> None of these conditions occurred in this study.

Because of the increased susceptibility of children to airway closure, it was expected that haemoglobin desaturation might be more severe in patients undergoing ENT procedures because of the shared airway, but the data did not indicate such a trend. There are several possible explanations; the surgery relieved the airway obstruction and oedema may not have occurred in the immediate postoperative period.

The use of opiates for intra-operative analgesia did not lead to an increased incidence of hypoxia. In the 12 children who received opiates the mean SaO<sub>2</sub> was 97.75%(sd =2.7) at arrival and 97.83%(sd =0.85) at 20 minutes. There was, however no significant difference in the extent of reduction in SaO<sub>2</sub> between the two groups at the various time intervals.

Children in prone position had lower SaO<sub>2</sub> compared with those in lateral or supine positions on arrival in recovery room. Patients in supine position had higher oxygen saturations the explanation of these may be attributed to the level of wakefulness and co-operation unlike those who were in the prone position who were fighting and/or crying. Prone position also has splinting effect preventing free abdominal excursion. Desaturation tended to occur during periods of struggling as a result of increased oxygen consumption. However those in supine position had the highest drop of SaO<sub>2</sub> up to 4% and this is significant in postoperative patients who have low SaO<sub>2</sub>.

The mean SaO<sub>2</sub> in the 11 children who had mild airway obstruction was 95.82%(sd =1.40) at 10 minutes and 95.64%(sd =1.12) at 20 minutes. This was lower than the group preoperative mean SaO<sub>2</sub> but the difference was not statistically significant (p=0.066). Saturations in awake and drowsy patient were not significantly different. This data correlates with studies that show there is no correlation between postoperative hypoxia and degree of consciousness in children.<sup>85</sup>

ENT surgery for example tonsillectomy and adenoidectomy carries direct injury to the upper airway but there is no effect/interference with the mechanical functions of respiration that is involved in ventilation. Hypertrophy of inferior turbinates and adenoid hypertrophy causes some degree of airflow resistance. Sub mucosal diathermy for hypertrophy of inferior turbinates and adenoidectomy relieves this resistance <sup>16</sup> In this study patients undergoing sub mucosal diathermy had higher SaO<sub>2</sub> levels post operatively as compared to the pre operative values.

Alexander et al (1973) found that after upper abdominal surgery FRC was reduced by 30% and after herniorrhaphy by 15%. <sup>86</sup>They concluded that this was due to abdominal muscle spasm, bowel distension and pneumoperitoneum. Upward shift of the abdomen tend to close airways and promote hypoxia. They also found little change in FRC after limb surgery. This suggests that at the end of anaesthesia normal muscle activity quickly returns to normal in respiratory muscles after adequate reversal of the muscle relaxants.

The routine use of 100% oxygen at the end of operation may have prevented the development of hypoxemia as noted in the drop of SaO<sub>2</sub> at 20 minutes in the recovery room (see page 28)

There were no differences in the various age groups and weights. Children have adequate compensatory mechanisms unlike in the elderly people. This correlates with other research works looking into the aspect of age in evolution of hypoxia <sup>78, 79</sup>

The patients who were induced with gaseous agents had lower saturations post operatively but the difference was not statistically significant. The low saturations at 20 minutes can be explained by the fact that most children by then had recovered fully from the effects of anaesthesia and were agitated.

Induction of anaesthesia using inhalation technique is appropriate in young children who may become distressed by repeated attempts at venous cannulation. All anaesthetics except nitrous oxide depress respiration but provided the administration is

correctly judged to give early waking, no serious under ventilation occurs postoperatively.<sup>85</sup>

Nitrous oxide is about 34 times more soluble than nitrogen, so that at the end of anaesthesia the volume of nitrous oxide coming out of blood greatly exceed the volume of nitrogen going in the other way, and this displaces oxygen from the lungs (Fink effect). The effect is severe but transient and the administration of oxygen for a few minutes prevents the main effect.

Narcotic analgesics were not routinely used in ENT procedures. This could possibly due to the fear by the anaesthetist on the side effects of the drug especially on the depression of respiratory centre, chest rigidity, postoperative nausea and vomiting as seen in one of the patients.<sup>40</sup> None of the children was premedicated with opioid.

## CONCLUSION

Hypoxemia is not a common occurrence following upper airway surgery in ASA I and II children but there is a general trend of decline in SaO<sub>2</sub>.

There was no correlation between the type of procedure and the incidence of desaturation to less than 90%.

Children in supine position during the recovery period have a greater variation in SaO<sub>2</sub> as compared to those in lateral or prone position.

Use of opiates and NSAIDs did not lead to post operative hypoxia.

There was no difference in the degree of postoperative desaturation in children who were induced with intravenous agents versus inhalation agents.

## RECOMMENDATION

Patients in the study were ASA I and II. More seriously ill patients may well show more profound changes in SaO<sub>2</sub>.

There is need for continuous monitoring in the recovery room. Standards of minimal monitoring which include the provision of a pulse oximeter for all patients in recovery room need to be strengthened.

## APPENDIXES

### APPENDIX 1

The American Society of Anaesthesiologists (ASA) classifies patients according to their general conditions as follows:

ASA I – A normal healthy patient

ASA II – A patient with mild systemic disease.

ASA III – A patient with severe systemic diseases that limit activity but is not incapacitating.

ASA IV – A patient with incapacitating systemic disease that is a constant threat to life.

ASA V – A moribund patient who is not expected to survive 24 hrs with or without operation.

ASA VI – Organ transplant patient.

E – Added as a suffix for emergency operation.<sup>2, 11</sup>

## APPENDIX 2

### CONSENT EXPLANATION

Your child has been invited/chosen to participate in this study.

### ABOUT THE STUDY

#### Why is this study carried out?

Postoperative hypoxia has been noted to occur after general anaesthesia.

### PATIENT INFORMATION

The Pulse oximeters to be used in this study have been tested before and in use for Monitoring in this hospital.

These are not new instruments used for experiment.

The process and procedures to be used in this study are aimed at getting the best outcome.

There is potential benefit to the patient in terms of close monitoring and early recognition of complications.

Choice not to participate in study will not lead to denial of surgery as planned.

There is no interference with preoperative and postoperative instructions given by the surgeon and/or the anaesthesiologist.

There is no financial gain by participating in study.

An assessment of vital signs and general condition of the patient will be done at regular intervals and staff will be available to give the necessary assistance.

#### Who is to participate in this study?

Randomly selected children scheduled for elective ENT surgery at Kenyatta National Hospital with consent from their parents/guardians.

Anybody can participate so long as they meet the study criteria regardless of race, ethnicity or gender.

#### Has this study been approved by the appropriate authorities?

Before the study begins, an approval by the Kenyatta National Hospital Ethics and Research Committee is sought.

## **PROCEDURE**

This is what will happen if you decide to participate in the study.

First someone will meet you before the day of the scheduled surgery. He will give more information about the risks and benefits of the study. He will ask questions relevant to the study and do physical examination (take weight and height) and your questions answered.

When you consent for your child to participate in the study you will be required to sign informed consent.

Fifteen minutes before surgical procedure while in the receiving area observations will be taken.

After the operation more observations will be carried out when the patient arrives in the recovery room the next 30 minutes.



APPENDIX 3

CONSENT FOR AND WILLINGNESS TO PARTICIPATE IN STUDY

Name of patient (print):.....

Surname First name Other(s)

IP No:.....

Study No:.....

Diagnosis:.....

Operation:.....

This document DOES NOT guarantee the signatories an operation.

Approved medical personnel practicing at Kenyatta National Hospital as per the standards and practices recognized by the said hospital, Kenya Medical Practitioners and Dentists Board and Nursing Council of Kenya as the case may be will conduct all medical procedures/observations.

- The physician has satisfactorily explained the proposed observation method(s) or procedure(s), the nature and purpose of the study, the risks and the benefits involved.
- I have had a chance to ask questions. I have all the information I desire, and my questions have been answered satisfactorily.
- I agree my child to participate in the study. I have been told that if I have future questions, about the study or about my rights as a subject. I can ask the investigator as listed.

Information about confidentiality

The information you provide will be held in the strictest confidence. Nothing will be published or discussed in public that can identify you.

My signature below acknowledges that I have read, understood and agree to the foregoing statements:

Signature of patient/Parent/Guardian:.....Date:.....

Signature of witness/ Translator/Reader:.....Date:.....

The nature, known risks, purpose of the study to be performed on this patient has been explained to him/her.

Signature of the Physician:.....Date:.....

APPENDIX 4

QUESTIONNAIRE

Patient No:..... Age:.....

Sex: Female  Male

Diagnosis:..... Planned Surgery:.....

Weight:..... (Kgs) Hb:.....(gm/dl) Height:.....(Cms)

Preoperative Assessment

ASA Physical Status I  II

Pre-medication

Atropine  Opioids

Others  Specify:.....

Pre-operative O<sub>2</sub> Saturation:.....%

Surgery

Induction Agents Used

IV

Gaseous

Intra-operative Analgesics

Opioids

Pethidine:.....

Fentanyl

Other:.....

NSAIDS

Specify:.....

Muscle relaxants

Yes

No

Duration of Surgery

< 60 minutes

>61 Minutes

APPENDIX 5

**OBSERVATION IN RECOVERY ROOM**

1. Position of patient on arrival

Supine

Prone

Lateral

2. Level of consciousness

Awake

Drowsy

Unconscious

3. Degree of Airway obstruction

Absent

Mild

Moderate

Severe

4. Post-Operative Complications

Vomiting

Shivering

Pain

Oxygen Saturation Monitoring Chart

	Arrival	10 minutes	20 minutes	30 minutes
SaO2				
Pulse rate				



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**Ref: KNH-ERC/01/1351**

**Date: 24 June 2003**

Dr. Bartuiyot E C  
Dept. of Surgery (Anaesthesia)  
Faculty of Medicine  
University of Nairobi

Dear Dr. Bartuiyot,

**RESEARCH PROPOSAL "ARTERIAL OXYGEN SATURATION IN CHILDREN FOLLOWING  
GENERAL ANAESTHESIA FOR ENT SURGERY"** (P57/5/2003)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** your above cited research proposal.

It may be clear cut in terms of interpretation of the results if the observations and measurements are going to be made according to defined surgical procedures since they will introduce important variables. For example, would tonsillectomy/adenoidectomy influence ventilation more than say a middle ear/sinus procedure?

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely,

PROF. A. N. GUANTAI  
SECRETARY, KNH-ERC

Cc Prof. K.M. Bhatt, Chairperson, KNH-ERC  
The Deputy Director (C/S), KNH  
The Dean, Faculty of Medicine, UON  
The Chairman, Dept. of Surgery, UON  
CMRO  
Supervisor Dr. Chokwe T.M., Dept. of Surgery (Anaesthesia) UON

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