ASSESSMENT OF FEATURES OF CARBON MONOXIDE AND CYANIDE INHALATIONAL POISONING IN PATIENTS WITH FLAME BURNS AT KENYATTA NATIONAL HOSPITAL

DISSERTATION SUBMITTED IN PARTIAL FULFILMENT OF THE AWARD OF THE DEGREE OF MASTER OF MEDICINE IN GENERAL SURGERY, UNIVERSITY OF NAIROBI

BY

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DEDICATION

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LIST OF ABBREVIATIONS

ABGs	Arterial Blood Gases
A&E	Accident and Emergency Department
ATLS	Advanced Trauma Life Support
AUC	Area under the curve
CDC	Center for Disease Control and Prevention
CN	Cyanide
CO	Carbon Monoxide
COHb	Carboxyhemoglobin
ERC	Ethics and Research Committee
FDA	Food and Drug Administration
GCS	Glasgow Coma Scale
HBO	Hyperbaric Oxygen
Hb	Haemoglobin
HCN	Hydrogen Cyanide
ICU	Intensive Care Unit
KNBS	Kenya National Bureau of Statistics
KNH	Kenyatta National Hospital
NPV	Negative Predictive Value
$PaCO_2$	Arterial Partial Pressure of Carbon Dioxide
PaO_2	Arterial Partial Pressure of Oxygen
PPV	Positive Predictive Value
ROC	Receiver Operator Characteristics
SET	Signal Extraction Technology
SpCO	Carbon Monoxide Saturation
% TBSA	Percentage of Total Body Surface Area
UoN	University of Nairobi

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ABSTRACT

Background & Justification of the Study

Patients with flame burns invariably inhale smoke but about 60% of those presenting to hospital sustain heat or smoke inhalation that is significant to contribute to their morbidity and mortality. A direct systemic effect of smoke inhalation is caused by inhalation of toxic gases during the combustion of organic and inorganic substances. The two most common gases that contribute to morbidity and mortality of flame burnt patients are carbon monoxide (CO) and cyanide (CN). These two systemic inhaled toxicants cause many of the fire and smoke related acute deaths and should be suspected in every fire victim.

The median mortality among admitted fire victims occurs at day 5 of admission. The bulk of early deaths are attributable to burn shock and inhalational injuries. It is probable that among the causes of these early deaths is inhaled smoke containing toxicants such as CO and CN whose measurement was not being done in flame-burnt patients presenting to KNH. This gap was the basis of this study.

Aim & Objectives

To assess for features of carbon monoxide and cyanide inhalational poisoning in flame burnt patients presenting to Kenyatta National Hospital.

Methodology

This was a prospective descriptive study. Eighty two consecutive non-pediatric consenting patients presenting with acute (<24 hrs) flame burns were recruited into the study between April 2017 to February 2018. Features of possible toxic smoke inhalational poisoning were assessed and carboxyhemoglobin levels determined by a *Masimo SET^{R} Radical 57^{TM}* CO-oximeter system at admission. Serum lactate levels were also determined by *Siemens RapidPoint^{R}500* blood gas analysis machine which was used as a surrogate marker for cyanide toxicity.

Data was analysed with Statistical Package for Social Sciences (SPSS) for Windows Version 21 with p value of <0.05 considered significant.

Study Shortcomings

Lactate levels were done only on 48% of the patients due to frequent unavailability of the service at the concerned laboratory.

Results

Eighty two subjects were recruited and after data cleaning, analysis was done on eighty. Most of the subjects resided in estates considered low class. Fifty six percent were males and 44% were females. Kerosene stove explosions contributed to most of the mechanisms of burns, at 28.7%. Over 53% of the material burnt by the fire was worn clothes only. Commonest complaints that could point towards toxic inhalation was Confusion (n=23) and headache (n=21). Seventy percent (n=56) of the respondents had singed nasal vibrissae and 25% had upper airway hyperemia or edema which pointed towards likelihood of significant inhalational injury. The mean TBSA was 30.9% and average SPCO% was 5.48%. Average Arterial lactate was 2.91mmol/L. Overall mortality was 38.7% out of which 22% occurred in less than 24 hours after admission. Factors found to have positive correlation with death included upper airway edema/hyperemia (p=0.024), %TBSA (p=0.01), lactate (p=0.01) and Base deficit (p=0.01); but not SPCO% (p=0.708).

Discussion

Rampant use of open flames for household cooking and lighting by most of the subjects who resided in relatively low class houses where precaution to fire outbreaks may not be taken may have contributed to the flame injuries. The, mean SPCO% of 5.48% fell below levels that would cause symptoms hence may not have accounted for the confusion and headache seen in over one third of the subjects neither could it account for the morbidity/mortality because it was below toxic levels. In those whom lactate levels were measured, only 7 had lactate levels of >5mmol/L. However, this level was far below 10mmol/L, which is the threshold to suspect cyanide poisoning in acute burns. Therefore, the 22% of all deaths that occurred before 24 hours of admission may be attributed to well-known factors that cause acute deaths of burn patients such as burn shock and inhalational injury. There may have been little or no contribution by CO and CN inhalation.

Conclusion and Recommendations

A large proportion of flame burnt patients presenting acutely to KNH have symptoms that are seen in CO or CN inhalational poisoning. However, due to low SpCO% and low lactate levels in majority of them, these symptoms are considered nonspecific for toxic inhalational poisoning. Therefore CO and CN may not be a significant factor in their poor outcomes as hypothesized in this study. Nonetheless, many of these patients have significant inhalational injury which has a strong positive correlation to their deaths and hence would still require early high flow oxygen administration and probable tracheal intubation.

1. INTRODUCTION

Burns is a type of trauma that has been around since man first invented the use of fire. However, the earliest record of burn management is by Kentish (1797) [1]. Burns induces a complex physiological stress to the victim and untold anguish. The clinical presentation varies depending on the offending agent and duration one is exposed to it, site of the body involved, host factors and the environment within which the incidence occurs. Fire is a prime cause of burn related deaths mainly through the consequences of heat and inhalation of combustion gases. In Africa, it is estimated that fire kills 6.1 per 100,000 of the population compared to 1 in every 100,000 in high income countries[2,3].

Smoke Inhalation present local and systemic effects that are detrimental to the patient immediate after a fire-incident and later on if not identified and addressed. Inhalation of carbon monoxide (CO) and CN should be suspected in every fire victim because they are among the commonest causes of immediate deaths in fire victims[3, 4].

In Kenyatta National Hospital (KNH), there are about 550 patients admitted with major burns annually out of which 36.7% are due to flames of whom 61% sustain significant smoke inhalation[5,6,7]. Ndung'u (2008) using Tobiasen's Abbreviated burn Severity Index (1982) reported that presence of inhalational injury, percentage of burn total body surface area and depth of burn are the strongest predictors of mortality. Over 43%-68% of burn patients with inhalational injury die in KNH. Majority of these deaths occur within the first 5 days of admission. Conversely, only 8-11.5% of deaths occur among patients with pure cutaneous flame burns which tend to occur much later postadmission[5,6,8]. The early deaths of patients with inhalational injuries may be attributed to inhalaled systemic toxicities and respiratory compromise[8,9]

At KNH neither serum CO-spectrophotometry nor pulse CO-oximetry is done. Therefore, the likelihood of CO poisoning in flame burnt patients could not reliably be determined hence management was not focused to this possibility. These gaps in diagnostic process and care were identified by Buni (1997), Mburu (2004), Shisanya (2007) and Mugambi (2014) who highlighted the need of carboxyhemoglobin (COHb) determination at the earliest contact with the patient to enable a prompt and more directed oxygen administration, ventilatory support and monitoring[8,10-12].

Therefore, this study sought to assess for features of carbon monoxide and cyanide inhalational intoxication in patients presenting to KNH with flame injuries. The finding was that CO and CN were unlikely to contribute to mortality and morbidity of flame burnt patients presenting to KNH.

2. LITERATURE REVIEW

2.1. BACKGROUND INFORMATION

Discovery of fire must be one of the most life transforming achievement in the history of primates. Many of the legends or myths relating to the origin of fire are vivid and dramatic. Biblically, the first mention of fire is soon after the fall of man whereby the 'Tree of Life' was guarded with a flaming sword. Later on first humans offered burnt sacrifices using fire[13]. Archaeologists generally agree that the early humans first attained control of fire around 1.9 million years ago. This was largely used for cooking which in effect impacted mental (brain) and social evolution to the modern humans[14,15]. Although indispensable, fire has been a source of untold misery to its casualties through its thermal effects and irritant and toxic smoke produced during pyrolysis.

Globally, it is estimated that 300,000 fire related deaths occur, 95% being in low to medium income countries. In Africa 6.1 in every 100,000 people die from fire related incidences versus 1 in every 100,000 in high income countries[2]. Up to 77% of fire related deaths follow smoke inhalational complications among which the majority is due to carbon monoxide poisoning[9, 16]

Effects of smoke inhalation have been recognized for many centuries beginning soon after our ancestors attempted to build fires in non ventilated shelters. However, the first accurate description of carbon monoxide (CO) poisoning was recorded by Claude Bernard in 1857. Hitherto, a lot is known about toxic smoke poisoning and it is reported to be the cause of most immediate deaths from building fires[17]. Cobb and Etzel (1991) analysed 10 year data from the year 1979 to 1988 which included all the 56,133 deaths due to carbon monoxide inhalational poisoning in the US. Of these 28% (15 523) were associated with severe burns or house fires[18]. He found that males were 2.3 times more likely to die from CO poisoning[19]. No local data is readily available.

Kenyatta National Hospital, located in Kenya's capital Nairobi, is the largest referral hospital in East and Central Africa handling over 85,000 admissions per year with a catchment population of over 3.1million in the county alone[57]. Five hundred and fifty (0.65%) of the admissions constitute major burn victims[7] out of which 36-46% are due to fires. About 60% of the fire casualties sustain inhalational injuries of whom 43-68% succumb[5,6,8, 10, 12]. Flame burns accounts for 76% of all deaths from thermal burns, 70% of these deaths occurring in the first week of admission pointing towards probable respiratory compromise and toxic gas inhalation[6,8]. However, the systemic levels of CO in these patients who definitely had exposure to potentially toxic smoke is not known not only in KNH but also in Kenya. This gap is the core of this study.

2.2. CHARACTERISTICS AND MECHANISMS OF FIRE INJURY

Patients presenting with flame burns often sustain a degree of smoke inhalation and occasionally sustain other concomitant trauma. The burns tend to be more extensive and deeper. For these reasons, they tend to have a higher mortality than those suffering burns from other causes [3,20].

Fire characteristics such as flame/scorch height, intensity and flame depth; duration of exposure, and chemical composition of the material being burnt determine severity of both cutaneous and inhalational injury. Moreover, victim's co-morbidities and age, circumstances surrounding the burn such as being in an enclosed space where concentration of combustion toxins and oxygen depletion is higher contribute to severity of the inhalation injury and systemic toxicity[21,22].

For the last 50 years there has been massive increase in use of synthetic polymers such as polyurethane and polyacrylonitrile as well as silk, nylon and rubber in fittings and furnishings of modern buildings. Smoke from their combustion contains more hydrogen cyanide and inorganic acids such that modern fires are increasingly having complex toxicological composition. These sometimes can be the principal cause of death or their addition act in synergy with much lower otherwise non-lethal levels of carboxyhemoglobin to cause death in fire victims[22]. None the less, carbon monoxide is still likely to be the major toxicant in modern fires. This was shown by toxicological analysis of fire and nonfire deaths by Alarie (2002), where animals were exposed to smoke from a variety of burning materials[22]. Therefore, the two commonest combustion toxins in modern fires attributed to most on- scene and acute fire deaths are carbon monoxide and secondly cyanide[22,23]. These two will be the highlights of the rest of the literature.

2.3. PATHOPHYSIOLOGY AND PRESENTATION OF TOXIC SMOKE INHALATION

Smaller particles and many of the toxic gases produced during the combustion of organic and inorganic substances pass to the lower airway and lung parenchyma in gas phase[24]. With respect to morbidity and mortality, the two most important gases are carbon monoxide (CO) and cyanide (CN)[23].

Carbon monoxide is an odorless, tasteless and colorless gas produced endogenously by catabolism of heme containing compounds and several other oxidative processes at a rate of 0.42mls per hour. At this physiological level it is never toxic and is in fact vital for regulation of several cellular functions and act as a neurotransmitter [25,26].

However, toxicity results from exogenous sources mainly due to incomplete combustion of carbon-containing compounds such as coal, petroleum products and peat, common fuels in furnaces,

generators, gas heaters, and motor vehicles at homes or work environments. It is a major component of the smoke produced in virtually all open fires. Inhaled CO is rapidly and extensively absorbed into blood and distributes throughout the body. However, it tends to concentrate more in blood, heart, skeletal muscle and spleen according to human autopsies following CO-intoxication [19,25,26].

Its toxicodynamics involve both hypoxic and non-hypoxic mechanisms. First, its affinity for hemoglobin is more than 200-times higher than that of oxygen hence hemoglobin preferably combines with CO therefore decreasing oxygen carrying capacity of blood. In addition there is a left shift of the oxygen–hemoglobin dissociation curve which reduces the ability of hemoglobin to unload oxygen impairing tissue oxygen availability. Moreover, CO binds and inhibits cytochromes a3, leading to mitochondrial oxidative stress. After CO exposure has ceased, reperfusion (re-oxygenation) occurs with formation of free radical oxygen species, resulting in lipid peroxidation, cell membrane damage and caspase mediated apoptosis which are particularly harmful to the brain. This causes hypoxic encephalopathy manifesting with neuropsychological symptoms.[17,27,28].

Carbon monoxide too binds the heme molecules in myoglobin (COMb). This particularly affects the heart by decreasing facilitated diffusion of oxygen into muscle causing myocardial ischemia and decreased myocardial contractility [17].

Inhaled CO is eliminated from the body in two kinetic phases; first is the fast phase which is exhalation and second is a slower phase involving distribution to tissues, subsequent metabolism and slow release that continues after cessation of exposure. The slow phase accounts for the 300 minute half life at ambient conditions [25].

Hydrogen cyanide (HCN) represents the gaseous form of cyanide (CN), which is a colorless gas with the odor of bitter almonds resulting from incomplete combustion of polyurethanes, plastics, wool, silk, nylon, and rubber. It is 20 times more toxic than CO and can cause immediate respiratory arrest. However its half life is about one hour.[23]

CN directly stimulates chemoreceptors of carotid and aortic bodies, leading to a brief period of hyperpnea causing further inhalation of the toxic gas. It interferes with cellular metabolism, as CO does, by binding to the ferric ion on cytochrome oxidase enzyme, which is the terminal oxidase of the respiratory chain. This prevents transport of electrons in the electron chain transfer process thereby halting cellular respiration. Cells convert to anaerobic metabolism, and lactic acidosis ensues[23, 27].

Although the toxicodynamics of CO and CN may be different, both gases are asphyxiants and cause histotoxic hypoxia in almost every tissue in the body due to extensive distribution upon inhalation. Among all tissues, the most sensitive and hence worst affected are those with high metabolic rate; the brain and the heart. Consequently, both acute and delayed clinical presentation commonly seen after CO and CN poisoning are neuropsychiatric and cardiovascular[25,29].

Clinical presentation of inhalational toxicity: Fire survivors often present with upper respiratory tract symptoms including lacrimation, rhinitis, epistaxis, pharyngitis, cough, retching and shortness of breath. Patients may also have dysphagia[24].

Headache, dizziness, fatigue, shortness of breath, chest pains (due to myocardial ischemia), nausea and emesis and confusion are the commonest presentation of systemic poisons, such as CN, CO or hydrogen sulfide. These symptoms are nonspecific and may easily be misdiagnosed or ignored. Victims with reduced sensorium or those found unconscious in confined spaces are thought to have received longer inhalation exposures than conscious ones because of the unprotected airways and concentrated exposures[24,30].

Examination of patients suspected to have smoke inhalation may reveal altered mental status, presence of other physical injuries, cutaneous burns of various extend and depth. Inspection of upper airway may reveal singed nasal hair, soot in the oropharynx, facial or oropharyngeal burns, erythema or parching of mucous membranes. Edema may also be seen in the nose, posterior pharynx and larynx. These are usually due to direct thermal injury[24].

Conversely, systemic toxicity and injury to the lower respiratory tract is normally not due to heat but irritant or toxic gases and smoke particles. Clinical features include stridor, hoarseness, cough with a carbonaceous sputum marker, tachypnea, retractions, accessory muscle use, wheezing, rales/crackles, reduced air entry, diaphoresis, or cyanosis[24].

Severity of clinical manifestations of CO poisoning correlates imprecisely with the observed level of COHb. Carbon monoxide levels of 0.4-1.0% reflect endogenous production found in non-smokers living in rural areas. Five percent may be considered normal in people living in busy urban areas and smokers can tolerate levels approaching 15%. Most studies peg 10% or more or presence of clinical signs after known exposure to be indicative of CO poisoning and threshold for treatment [31]. In a healthy individual, the following symptoms may be seen at the levels indicated: [25,32,].

- 0-10% - Usually no symptoms
- 10-20% - Mild headache, atypical dyspnea, dilatation of cutaneous blood vessels

- 20-30% - Headache throbbing at the temples, impaired concentration
- 30-40% - Severe headache, weakness, blurred vision, nausea, emesis, impaired thinking
- 40-50% - Confusion, lethargy, syncope, tachycardia, tachypnea
- 50-60% - Respiratory failure, seizures, high risk of death
- 60-70%- Coma, intermittent convulsions, depressed cardiorespiratory function, death
- 70-80% Death within 1 hour
- >90% - Death within minutes

Cherry red discoloration of mucous membranes is unreliable and a late sign seen only ante mortem in 2-3% of patients. It is usually seen in autopsies where COHb levels are 30% or more. Almost a similar discoloration can be seen in victims of cyanide toxicity [33,34,35]

From any standpoint, it is logical to consider CN toxicity alongside CO toxicity in all smoke inhalation incidents because their presentations may be difficult to tell apart. However, CN tends to have predominant central nervous system and cardiovascular clinical findings. Moreover, even mild degrees of CN poisoning can cause delayed neurological sequelae in survivors and permanent disability including seizures, extrapyramidal syndromes, dystonia and post-anoxic coma [23,27].

2.4. DIAGNOSIS OF TOXIC SMOKE INHALATIONAL POISONING

History and high index of suspicion are key to clinically diagnose smoke inhalation intoxication. Although poisoning can be confirmed by detecting elevated levels of carboxyhemoglobin in the blood, the presence of clinical signs and symptoms however non-specific, should not be ignored. [30,31]. Hart et al (1985) reported a series of five comatose patients with smoke inhalation from house fires. They all had significantly elevated blood COHb at 32%$\pm$6 and cyanide. Four recovered without sequelae after hyberbaric oxygen therapy, one who had the highest cyanide levels of 3.9ug/ml (the average was 1.62$\pm$1.44) died. [36].

Reportedly, the incidence of smoke inhalation increases almost point by point with increase in Total Burn Surface Area (TBSA). It ranges from 10% at TBSA of 5%, 67% for 70% TBSA and 80% for TBSA of $\geq$85% [3,37]. However, there may be no significant correlation between extent of burns and blood concentration of CO and CN[23]. Endorf et al (2007) studying inhalational injury in 80 patients with an average of 25.4% ($\pm$2.9) TBSA found a serum COHb level of 13.7% ($\pm$1.8) [38]. At KNH, average %TBSA is 20-23%[10]. Evaluation of toxic smoke inhalation includes the following:

Pulse Oximetry

Standard pulse oximeters utilize two wavelength technology and can be misleading in the setting of carbon monoxide exposure. However, CO-oximeters such as *Masimo SET with rainbow technology* uses at least 4 wavelengths of light and are capable of detecting and differentiate among carboxyhemoglobin, methemoglobin, deoxyhemoglobin and oxyhemoglobin. Therefore, the resulting percentage of oxyhemoglobin measured by CO-oximetry is an accurate measure of the arterial oxygen saturation[3, 20,27].

Serum Carboxyhemoglobin (%COHb) and Pulse CO-Oximetry (%SpCO)

Carboxyhemoglobin levels/ saturation expressed as a percentage of total hemoglobin is the most frequently used biomarker of CO exposure[31]. It can reliably be measured by a laboratory CO-spectrophotometry or by digital CO-Oximeters such as Masimo *SET* Radical device.

The increasing preference to the use of Masimo *SET* CO-oximetry with rainbow technology is because it is non-invasive, rapid and still nearly as accurate as laboratory CO-spectrophotometry in the measurement of levels of COHb. It is able to concurrently detect oxyhemoglobin, deoxyhemoglobin and other dyshemoglobins hence unlike regular pulse oximeters, gives the true picture of arterial oxygen content in carbon monoxide poisoned patients[3,27].

Masimo pulse *CO-oximeters* were made commercially available by Masimo Corp, Irvine, California since the year 2005 and approved by the US- Food and Drug Administration in the year 2008 for non-invasive use across the whole manufacturer-specified spectrum[39]. Several studies have shown acceptable specifity and sensitivity of these CO-oximeters in measuring %SpCO in non-pediatric patients. For instance, Kot J et al (2008) compared laboratory CO-spectrophotometry of 49 patient blood samples and their *Masimo SET Rad57pulse CO-oximeter* bedside values and found a sensitivity of the pulse CO-oximeter to be 77.8%, PPV 82.4%, specificity 90.3%, and NPV 87.5% for COHb above 20%.[40] Its uncertainty in accuracy for COHb measurement compared to laboratory CO-oximetry is reported to be $\pm$ 2% at 0-15% COHb levels[41]. Moreover, it facilitates more rapid diagnosis and earlier initiation of oxygen therapy by up to an hour in CO-poisoned patients compared to laboratory CO-spectrophotometry. It also accurately detects hypoxemia in both normal and elevated levels of COHb[42,43]. This is why it was chosen for this study. However, larger trials are ongoing to determine its diagnostic dependability under different conditions.

Arterial blood gases (ABGs)

Arterial oxygen tension (PaO_2) does not accurately reflect the degree of CO poisoning or cellular hypoxia. Hart et al in a series of 5 CO and CN poisoned patients reported the following grossly deranged ABG parameters: pH 7.16 ± 0.06; PO_2 353 mm Hg ± 149; PCO_2 35 mmHG ± 10.5; HCO_3 12.6mEq/L ± 0.07; base excess −16 mEq/L ± 1.58; Oxygen saturation 66% ± 5.5[36].

PaO_2 level reflects the oxygen dissolved in blood and may not be altered by the hemoglobin-bound CO. A PaO_2 level within the reference range and for above case markedly elevated, may lead to serious underestimation of the decrement in tissue oxygen delivery and the degree of hypoxia at the cellular level that occurs in CO poisoning yet most blood gas analysis machines calculate oxygen saturation based on the PaO_2 level. They cannot distinguish COHb from oxyhemoglobin. Therefore in CO intoxications, $\%SpO_2$ must be gotten from CO-oximetry[3].

None the less, the presence of a low PaO_2 (< 60 mm Hg in room air) or hypercarbia ($PaCO_2$) level of 55 mm Hg) indicate significant respiratory insufficiency. Metabolic acidosis suggests inadequate oxygen delivery to the tissues from whatever cause including severe burns and hypotension[3].

Lactate

Elevated lactate levels may result from metabolic acidosis secondary to hypoxia, CO and CN poisoning, unrecognized trauma or inadequate resuscitation. Baud et al (1991) reported that plasma lactate concentrations measured at time of admission in patients with smoke inhalation correlated more closely with blood concentration of cyanide than with those of carbon monoxide. The concentration of lactate increases proportionally with the degree of CN poisoning such that lactate levels higher than 10 mmol/L are a sensitive indicator of CN toxicity. He found that in the context of smoke inhalation without severe burns (in ≤15%TBSA), plasma lactate of more than 10mmol/L had a sensitivity of 87% in diagnosing CN toxicity [23,27].

In most institutions, CN levels take hours to days for results; thus one must rely on clinical and indirect laboratory data such as: persistent neurologic dysfunction unresponsive to supplemental oxygen, cardiac dysfunction, severe lactic acidosis and "arterialization" of the venous blood gas, with PO_2 values similar to arterial levels.

2.5. GRADING SEVERITY OF CARBON MONOXIDE INTOXICATION

The relationship between severity of clinical presentation of acute carbon monoxide poisoning and COHb levels is not well correlated. However, factors affecting severity of CO poisoning

include concentration of CO gas and duration of exposure. Victim factors such as heart diseases, anaemia; pregnant women and their fetuses, infants and the elderly tend to be prone to CO toxicity[25,31].

Olson and Smollin (2008) in a systematic review categorized CO poisoning into 3 grades[25, 31]

- *Mild poisoning:* COHb of > 10% without clinical signs or symptoms of CO poisoning;
- *Moderate poisoning:* a COHb level of> 10%, but < 20-25%, with minor clinical signs and symptoms of poisoning, such as headache, lethargy, or fatigue.
- *Severe poisoning:* a COHb level of > 20-25%, loss of consciousness, and confusion or signs of cardiac ischaemia, or both.

Kathleen Meert et al (1998) described two additional grades of CO levels below 10% [44]:

- *Suspected toxic:* COHb level of $\leq$10% with acute neurologic signs.
- *Non-toxic: COHb* $\leq$10% without acute neurologic signs.

2.6. TREATMENT OF CARBON MONOXIDE POISONING

Any flame burn patient should be assumed to have inhalant intoxications whether they have obvious inhalational injuries or not. Being a form of trauma such patients should initially be treated along principles of ATLS right from the scene of incidence. Rapid evacuation to a health facility, preferably a burn centre is essential. Oxygen administration should begin as soon as is practicable.

Early empirical treatment for both CO and CN poisoning should be started soon after drawing laboratory samples. Normobaric 100% supplemental oxygen reduces half life of CO from 3-4 hours to 30-90 minutes such that on average COHb levels may reach <3% in 3.6 hours[44].

On the other hand 100% oxygen administered at 2.5-3.0 atm reduces CO half-life to 15-23 minutes[26,45,46] Although there is debate whether or not Hyperbaric Oxygen (HBO) therapy has any important role in CO poisoning, many centers in the US institute it at COHb of >25% (considered severe CO poisoning), in pregnancy and in comatose patients. Reportedly it is associated with better neurocognitive outcomes[47]. However, systematic reviews have not revealed a clear clinical benefit of HBO over normobaric Oxygen, so no clear guidelines for its use have been determined[48,49]. In fact it may be associated with increased complications especially in children with coexisting cutaneous burns[44]. Therefore prompt 100% high flow normobaric oxygen via a well

fitting non- breather face mask may suffice for majority of patients until symptoms improve and/or COHb drops to <10%. There is no evidence that continued elevation of COHb (>5%) in absence of continued symptoms necessitates further treatment [47].

Some degree of cyanide poisoning should also be assumed in almost all cases of smoke inhalation. Its treatment is by 100% oxygen plus empirical hydroxocobalamin administration which is currently recommended as a safe CN antidote in cases of smoke inhalation[45,50,44].

Being a pressing public health issue, control and prevention is key in dealing with smoke inhalation toxicities. It involves avoidance of exposure, adequate ventilation if smoke is likely to be present, and use of smoke and carbon monoxide detection systems in areas handling fire and chemicals[45].

2.7. OUTCOMES OF CARBON MONOXIDE POISONING

A poor outcome is predicted by lengthy CO exposure, loss of consciousness and extremes of age; 64 years or older and 5 years or younger. Persons having physical or cognitive disability have a higher mortality rate than matched controls, as do persons under the influence of alcohol or other drugs. Coexisting injury and cutaneous burn also increases the risk of death. The overall case-fatality rate for CO poisoning ranges from 0 to 31%.[9, 20, 51, 52]

Although deaths have been reported at COHb levels ranging from 3-70% depending on other coexisting factors, levels >50% in otherwise healthy individual are frequently fatal[25]. Pure CO intoxication accounts for as much as 75% of fatalities from inhalation injury[4,53]. Besides, inhalation of other products of combustion can worsen the effect of CO. For instance CN and CO have additive effect such that death can occur at the presence of sub-lethal levels of both[23].

Myocardial injury occur in upto 37% of moderate to severe CO poisoning with 5% in hospital mortality and a 2.1 hazards ratio of mortality in 12 years[29]. Neuropsychiatric sequelae of CO poisoning occur between 10- 30% but has been reported in up to 68% of all patients who sustain toxic levels of CO often developing within 2-28 days typically after a period of lucidity. It is commoner in the elderly and includes features of Parkinsonism, changes in personality such as Obsessive compulsive disorder, cognition and memory [25,26,**47**,54].

In conclusion, there is good reason to assume that every fire victim has some degree of toxic smoke poisoning especially for CO and CN which impact outcomes. This study set out to assess acute flame-burnt patients at KNH for CO and lactate levels to assess for possible smoke toxicity.

3. AIM AND OBJECTIVES

3.1. RESEARCH QUESTION

What are the pointers to carbon monoxide and cyanide intoxication in patients with flame burns presenting to Accident and Emergency (A&E) of Kenyatta National Hospital (KNH)?

3.2. STUDY OBJECTIVES

Aim

To assess features of carbon monoxide and cyanide inhalational intoxication in flame injured patients presenting to A&E of KNH.

Objectives

a) To record clinical features likely to be associated with carbon monoxide and cyanide poisoning in flame burnt patients presenting to KNH.
b) To determine CO saturation levels (SpCO %) in flame burnt patients as soon as they arrive to KNH.
c) To determine lactate levels as a surrogate to cyanide poisoning in patients with flame burns as soon as they arrive at A&E of KNH.

4. JUSTIFICATION OF THE STUDY

Inhalational Injury is present in 22% of all burn patients presenting to KNH out of which 43-68% end up dying mostly within the first week post-incident. Some of the attributes of smoke inhalation that are known to cause early mortality have not fully been investigated in our set up as pin pointed by Buni (1997), Mburu (2004), Shisanya (2007) and Mugambi (2014). This study aimed at shedding light on a few of these attributes such as determining systemic CO saturation (SpCO%) and lactate levels that may infer to severity of toxic smoke inhalational poisoning.

To the best of my knowledge and enquiry no study attempting to establish toxic smoke inhalational poisoning among flame burnt patients had been done in Kenya hence a gap in this knowledge in the local set up.

5. METHODOLOGY

5.1. STUDY DESIGN

The study was a prospective observational study carried out for a period of 11 months from April 2017.

5.2. STUDY SETTING AND POPULATION

The study was carried out at the Accident and Emergency, Burns unit, ICU and Burn ward (4D) at KNH.

Target population were patients were victims of flame burns who met inclusion criteria.

5.3. SAMPLE SIZE DETERMINATION

The sample size was determined using Daniel's (1999) formula for sample size calculation for finite population:

$$n' = \frac{N Z^2 P (1-P)}{d^2 (N-1) + Z^2 P (1-P)}.$$

Where

n' = sample size with finite population correction.

N = size of the target population in 6 months = 101 (36.7% of 275) [7]

Z = Z statistic for 95% level of confidence = 1.96

P = Estimated prevalence of significant smoke inhalation among flame burnt patients= 61% [8]

d = margin of error = 5%

Hence sample size = $\frac{101 \times 1.96^2 \times 0.61(1-0.61)}{0.05^2 (101-1) + 1.96^2 \times 0.61(1-0.61)}$.

= 79.30 rounded up to **80 participants**

5.4. SAMPLING METHOD

Subjects were selected in a sequential non-randomised manner. All consecutive flame burn patients who met the inclusion criteria were recruited until the desired sample size (80) was achieved. However, two additional subjects were recruited to replace two earlier ones who were excluded due to extensive burns that involved all potential sites for attaching the Co-oximeter probe besides having poor peripheral perfusion that precluded SpCO% reading by the CO-oximeter.

5.5. INCLUSION CRITERIA

a) Flame burnt patients
b) Patients who consent to the study.
c) Patients presenting within 24 hours of post-burn period.
d) Patients from 13 years of age and above.

5.6. EXCLUSION CRITERIA

a) Patients with burns from other causes other than flames.
b) Pediatric patients of 0- 12 years of age.
c) Those who declined to consent to the study.
d) Patients presenting more than 24 hour after the burn incident.
e) Patients whose SpCO% reading could not reliably be obtained for any reason.

5.7. VARIABLES

5.7.1. Independent variables

a) Demographic data
b) Circumstance of burn (indoor/outdoor)
c) Duration before rescue
d) Percentage total burn surface area (%TBSA)
e) Location of burn including inhalational

5.7.2. Dependent variables

a) Clinical signs (per data collection tool: presence of inhalational injury vital signs, GCS)
b) Biochemical parameters (per data collection tool: SpCO, SpO2, $PaCO_2$, $PaO_2 \pm PvO_2$, pH, lactate, Base deficit)
c) Short term neuropsychiatric and cardiovascular complications and death within 28 days of admission.

5.8. DATA MANAGEMENT

5.8.1. Data Collection (Tools and personnel)

a) Structured interviewer administered questionnaire (appendix C)

Demographic data and Initial clinical data was collected using structured questionnaire.

Lund Browder chart (appendix B) was used to determine %TBSA.

b) Laboratory Measurements and Targeted Outcome Indicators (appendix D)

The COHb levels was determined at the earliest contact with the patient using *Masimo SET^{R} Rad 57^{TM}* CO-oximeter.

Selected clinical and laboratory parameters (as per 5.7.2 (b) vide supra) were recorded at arrival at the A&E. Arterial blood samples were drawn and submitted for blood gas analysis

Relevant sequelae (neurological and cardiac) was noted at day 0 then weekly for four weeks post burn.

c) Research Personnel

One paramedic was recruited as the research assistant. She had emergency care training and had experience in burns management. She was trained and competence tested by the principal researcher on how to use standardized clinical interogation and data collection tools and drawing of ABG samples from the radial and femoral arteries.

d) Quality assurance in Obtainment of the Targeted parameters

The SpCO% was determined at the earliest contact with the patient using a the same *Masimo SET^{R} Rad 57^{TM}* CO-oximeter. Sensor probes were affixed on fingers or toes of the non burnt areas until a steady reading was established for at least 90 seconds. The probe was covered using the dark hood (one of its accessories) to prevent interference from the ambient light.

A suitable site for ABG sample (either radial artery or the femoral artery) was selected, cleaned with alcohol swab, allowed to dry for 1 minute then arterial pulsation felt with a gloved non dominant index finder . A sample of 1ml of blood was drawn into a sterile disposable 2.5ml heparinized syringe. This sample was submitted to the acid-base laboratory within 20 minutes where ABG parameters were measured using the *Siemens RapidPoint^{R} 500* machine.The radial arterial sample was taken from the hand that demonstrated a positive Allen test.

5.8.2. Data storage and disposal

Raw data and hard copies of data sheets have been shredded. However, electronic copies of spread sheets, clean dataset and exploratory data analysis sheets have been encrypted and deposited into the UoN's research repository for future reference as necessary.

5.9. STATISTICAL ANALYSIS

Raw data was checked for completeness, entered into excel spread sheet, cleaned, categorised and coded then fed into the Statistical Package for Social Sciences (SPSS) for Windows Version 21 for analysis. Quantitative data was analysed by student t-test describing mean, mode and standard deviations as appropriate.

Differences and relationships of attributes of categorical variables were analysed using Spearman's rank correlation tests and proportions by chi square.

Association of Variables with outcomes whether singly or in combination was assessed by chi square, Fisher-Freeman-Halton exact test and Kendal Tau correlation tests. Receiver operator characteristics (ROC) of TBSA%, arterial lactate levels and base deficit levels in predicting risk of death were used to arrive at cut off values.

Level of significance was 5% at 95% confidence interval hence a *p*-value of <0.05 was considered significant.

5.10. RESULTS PRESENTATION AND PUBLICATION

This paper has been presented in both hard and electronic forms as bound booklets and as online resource at the UoN online repository. It has been disseminated to the Department of Surgery and Office of Postgraduate studies of University of Nairobi. It has also been presented to the Department of Research and Programs of Kenyatta National Hospital.

Secondarily, it will be published in a reputable journal.

Finally it will be presented in academic forums in and out of the country as appropriate.

5.11. ETHICAL CONSIDERATION

5.11.1. Ethical approval

Approval of this study was sought from the Kenyatta National Hospital-University of Nairobi Research and Ethics committee and only proceeded after due approval (appendix E).

5.11.2. Confidentiality

a) Neither participant's name nor hospital number was recorded on the data tools, but each participant was assigned a research tracking number.
b) An inventory of participants tracking numbers was safely kept by the principal researcher.
c) Confidentiality of the clinical information of the participants was ensured at all stages of research.
d) Collected data was kept safely at all times by the principal researcher.

5.11.3. Participants' Autonomy, Beneficence and non-Maleficence

a) Written consent was sought from the participants who had attained 18 years and assent for those below 18 years and/ or their parents/guardian.
b) Participants were allowed to freely withdraw from the study at any stage if they chose to.
c) No extra cost unrelated to the management of the participant was incurred purely for the purposes of the study
d) No coercion or persuasion was resorted to in those who declined to give consent.
e) No material gain was extended to participants.
f) Any finding during the study that deemed important in treatment of the patient was passed to the attending surgical team.

6. RESULTS

This was a prospective descriptive research done at Kenyatta National Hospital Accident and Emergency, Burns unit and Burns ward between April 2017 and February 2018.

A total of 82 fire victims were recruited consecutively, but two were excluded from analysis due to incompleteness of their data. Nonetheless, the target sample size of 80 was achieved and the results are presented in this chapter.

6.1. Biodata

There were 45 males and 35 females studied the youngest aged 13 years and oldest 80 years. Those aged between 18-35 years constituted over 68% of the burn victims. About 80% resided in Nairobi County and its immediate environs in areas generally considered as low income areas. The findings of demographic characteristics of the participants are as shown on table 1.

Table 1: Demographic characteristics

	Frequency n (%)
Gender	
Male	45 (56.3)
Female	35 (43.8)
Age	
<18	4 (5.0)
18 – 25	28 (35.0)
26 – 35	27 (33.8)
36 – 45	12 (15.0)
>45	9 (11.3)

6.2. Scene of Fire Incidence

Thirty percent of the incidents happened between 6pm and midnight and 27.5% from midnight to 6am. Over 75% of the flame burns occurred indoors and at home as shown below:

Table 2: Scene of Fire Incidence

Scene	**Frequency n (%)**
Home indoors	60 (75.0)
Work place	11 (13.8)
Outdoors	8 (10.0)
Vehicular	1 (1.3)
Total	**80 (100.0)**

6.3. Fire Characteristics

6.3.1 Mechanisms/ Causes of Fire

The commonest cause of fire was Kerosene stove at 28.7% followed by candle/lamp at 16.3%.

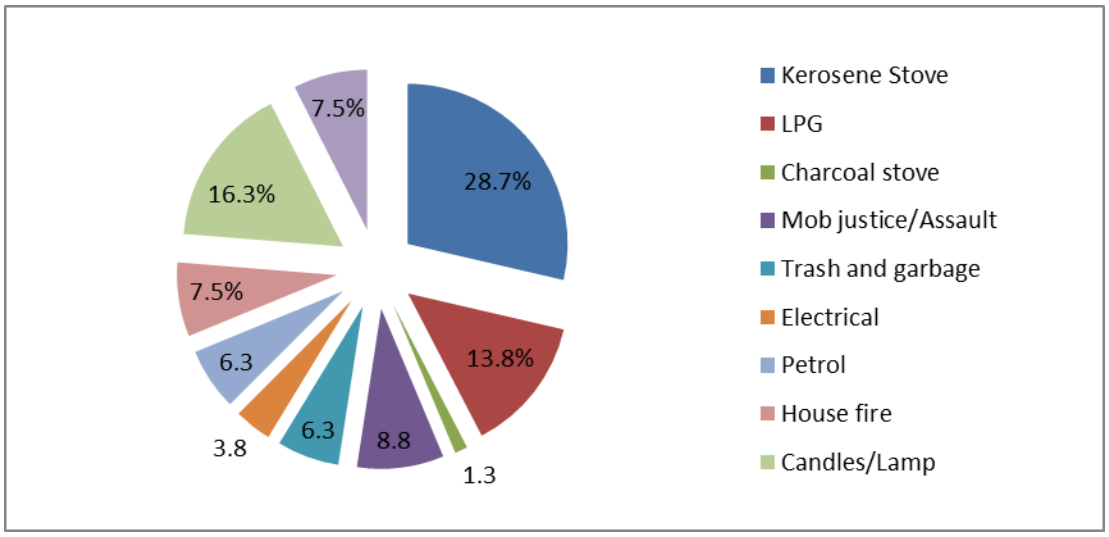

Figure 1: Pie chart showing Mechanism/Causes of Fire

6.3.2. **Predominant Burning Material**

Most of the fire incidents had the worn clothes as the only burning material at 53.8% and those whose bedding was the predominant burning material (16.3%) had candle/lamp as the cause of fire as shown in figure 2. Average time of external rescue was 16.33 minutes, 50% were taken straight to KNH, 27.5% having no first aid administered prior. Mean arrival time to KNH was 8.84hours (530minutes).

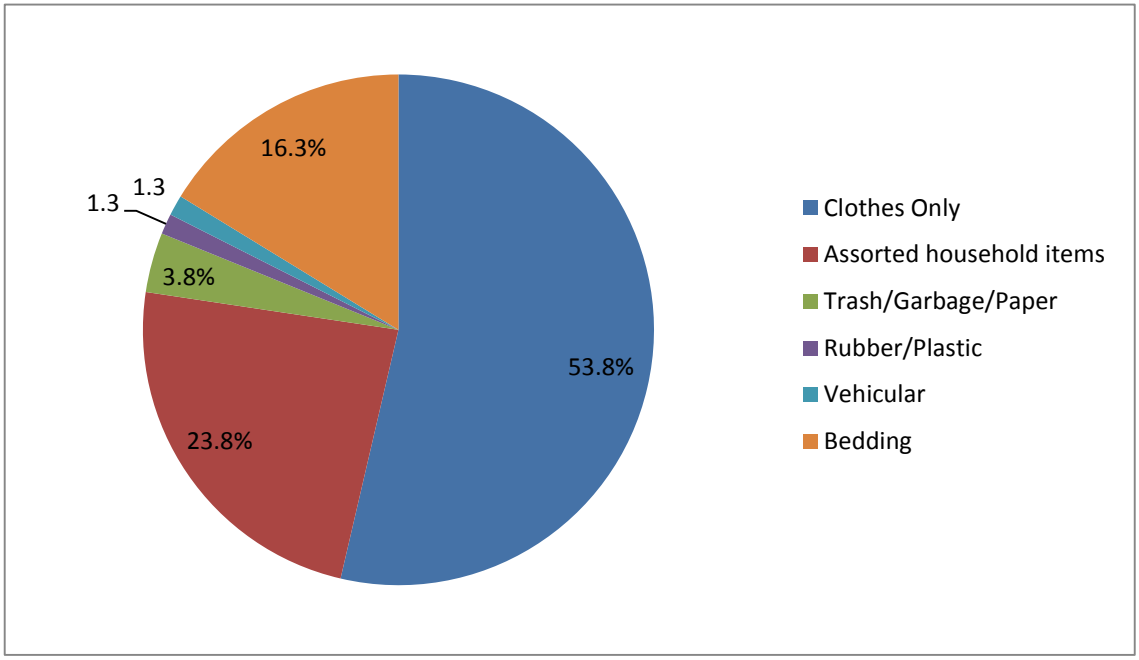

Figure 2: Pie chart showing predominant burning material

6.4. Clinical features relevant to CO/CN Inhalational

6.4.1: Mental State at the Burn-Incident

Factors influencing state of mind of the fire victims at time of incident is shown on table 3 with 31 (38%) participants either being under substance influence, convulsive disorders or were asleep.

Table 3: Mental State at the Time of Burn Incident

	Frequency n (%)
Awake & alert	49 (61.3)
Asleep	16 (20.0)
Convulsive/neuropsychiatric disorder	8 (10.0)
Drugs	4 (5.0)
Alcohol	3 (3.8)
Total	**80 (100.0)**

6.4.2. Presenting Complaints Relevant to CO/CN toxicity

Complaints that may be related to inhalation of toxic gases including CO and CN are presented in table 4.

Table 4: Presenting Complaints

	Frequency (n=80)	
	Yes	**No**
Confusion/loss of consciousness	23	57
Headache	21	59
Cough	17	63
Dizziness	12	78
Shortness of breath	10	70
Nausea/Vomiting	10	70
Dark sputum	9	71
Hoarse/Husky voice	3	77
Blurred vision	2	78
No Complaint	16	64

The mean SpCO% for those that had reported confusion/loss of consciousness and headache was 6.24 (SD4.24).

6.4.3. Upper Airway Examination Findings

Seventy percent of the flame burn victims had singed nasal vibrissae. Hyperemia or edema of the upper airway was seen in 25% of them.

Table 5: Upper Airway Examination findings

	Frequency n (%)	
	Yes	**No**
Singed nasal vibrissae	56	24
Hyperemia/Edema in upper airway	20	60
Soot	15	65
Carbonaceous sputum	6	74
Edematous lips	1	79

6.4.4. Glasgow Coma Scale (GCS) of the Subjects

Over twenty seven percent of the respondents had reduced GCS at arrival.

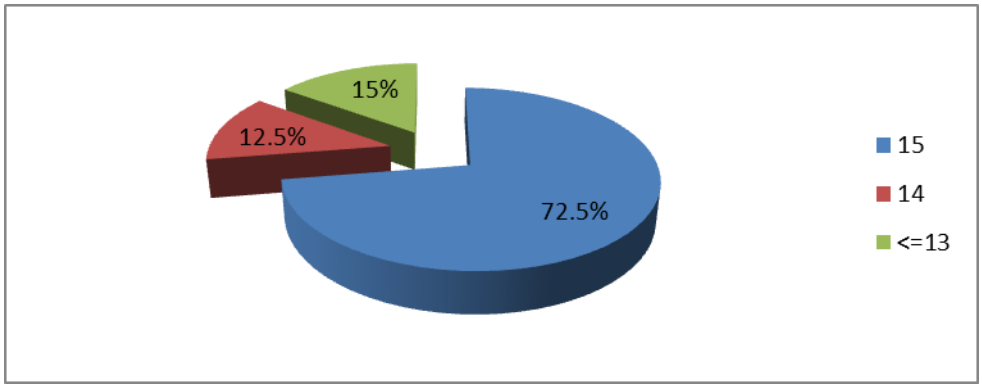

Figure 3: Pie chart showing Glasgow Coma Scale of fire victims

6.4.5. Cutaneous Total Burn Surface Area (%)

TBSA was estimated using the Lund Browder chart. The mean cutaneous TBSA was 30.9% (SD of 19.4)

Table 6: Cutaneous Total Burn Surface Area (TBSA %)

	Frequency n (%)
<10	3 (3.8)
10-19	21 (26.3)
20-29	22 (27.5)
30-39	18 (22.5)
40-49	5 (6.3)
50+	11 (13.8)
Total	**80 (100.0)**

6.5. Selected Laboratory Values

6.5.1. SpCO%

The mean SpCO% was 5.48 (SD 3.89), the highest value recorded was 21%.

Table 7: SpCO% Measurement

SpCO%	**Frequency n (%)**
<5	32 (40.0)
5-9	41 (51.2)
10+	7 (8.8)
Total	**80 (100.0)**

6.5.2. Arterial Blood pH

More than 50% of the study subjects had deranged blood pH which was predominantly acidic.

Table 8: Blood pH

	Frequency n (%)
<7.15	10 (12.5)
7.15-7.24	7 (8.8)
7.25-7.34	21 (26.3)
7.35-7.44	39 (48.8)
7.45+	3 (3.8)
Total	**80 (100.0)**

The mean blood PH was 7.30 (0.13)

6.5.3. Arterial Lactate

Only 39 of the 80 study subjects had their lactate levels measured. Their mean arterial lactate was 2.91 (2.12) mmol/L. Lactate of ≥ 5 mmol/L was seen in 7 patients, with a mean of 6.62 (1.5) mmol/L in this subgroup.

Table 9: Arterial Lactate Levels

Lactate (mmol/L)	Frequency n (%)
<5	32 (82.1)
5-9	7 (17.9)
Total	**39 (100.0)**

6.5.4. Base Excess

Over 93% of the victims had a base deficit of less than -2 with a mean of -7.87 mmol/L

Table 10: Arterial Base Excess

Base Excess (mmol/L)	Frequency n (%)
>-2.00	5 (6.3)
-6.00 to -2.00	26 (32.5)
-10.00 to -6.01	23 (28.7)
<= -10.01	26 (32.5)
Total	**80 (100.0)**

6.6. Twenty Eight Day Targeted Sequelae

6.6.1. Neuropsychiatric Sequelae

About 25% of the participants had some form of neuropsychiatric manifestation within 28 days of admission. Confusion and agitation tended to occur earlier whereas low mood and depression tended to be seen later in the admission period.

Table 11: Neuropsychiatric Sequelae

Neuropsychiatric Sequelae	Frequency n (%)
None	61 (76.3)
Confusion	5 (6.3)
Depression/low mood	9 (11.3)
Agitation/Delirium	5 (6.3)
Total	**80 (100.0)**

6.6.2. Mortality

Over 38% of the studied fire victims died within 28 days of admission, median mortality occurring on day 5.

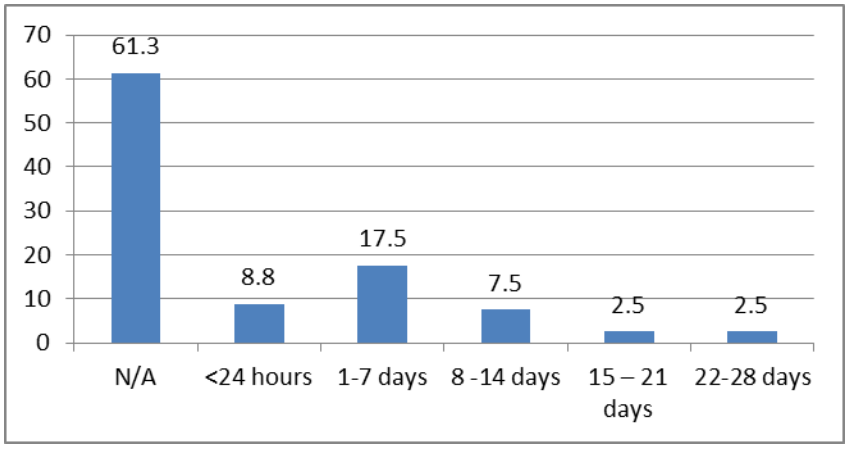

Figure 4: Twenty-eight day mortality among flame burnt patients

6.7. Receiver Operator Characteristics of selected Parameters

This section presents the cut off values as predictors of risk of death in flame injured patients based on sensitivity and specificity derived from ROC curve for the TBSA%, arterial lactate and base excess. A p-value of <0.05 was considered statistically significant.

6.7.1 Cut off Values for Arterial Lactate Levels

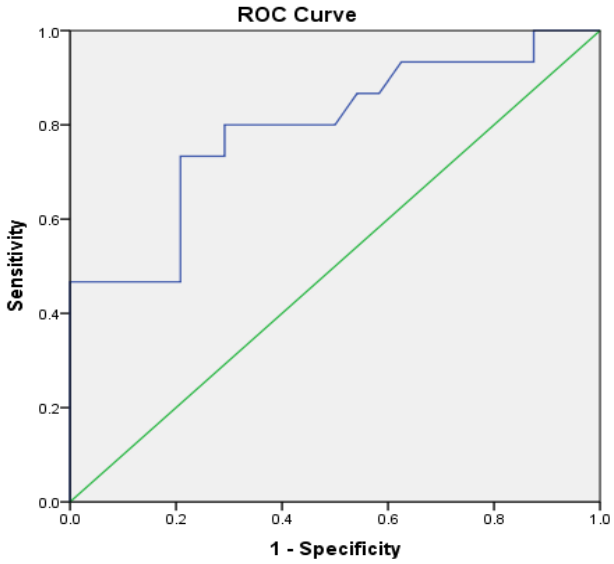

Figure 5: ROC curve for arterial lactate as a predictor of death in flame burnt patients

Test Variable	AUC	Std. Error	Asymptotic Sig.	Cut off	Sensitivity	Specificity
Arterial lactate	0.792	0.078	.002	**2.3600**	73.3%	20.8%

The value of the area under the curve (AUC) in the figure above has achieved statistical significance with p-value = 0.002, which means it has a favourable sensitivity and specificity characteristics.

6.7.2 Cut off Values for TBSA%

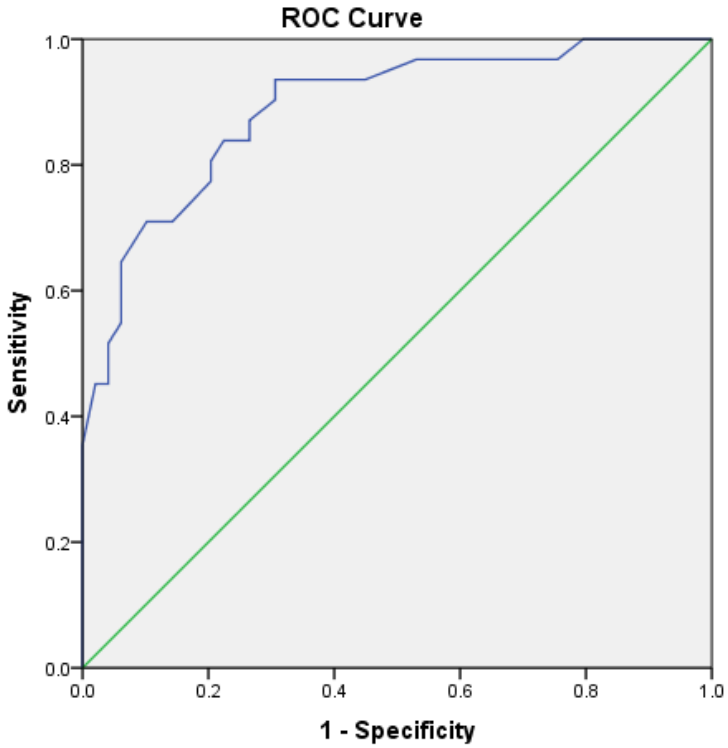

Figure 6: ROC curve for TBSA% as a predictor of death in flame burnt patients

	Area	Std. Error	Asymptotic Sig.	Cut off	Sensitivity	Specificity
TBSA%	.890	.037	.000	**32.500**	71.0%	12.2%

The value of the area under the curve (AUC) in figure 6 above has achieved statistical significance with p-value of < 0.001 meaning it has a favourable sensitivity and specificity characteristics.

6.7.3 Cut off Values for Base Excess

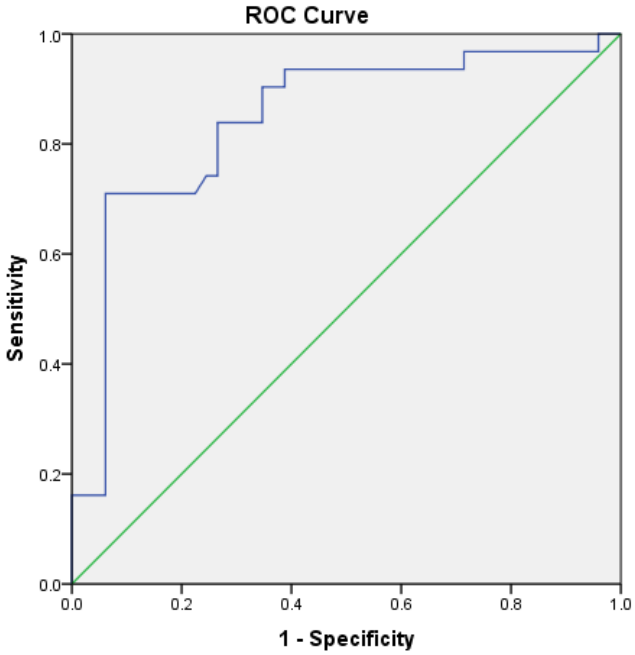

Figure 7: ROC curve for Base excess as a predictor of death in flame burnt patients

	Area	Std. Error	Asymptotic Sig.	Cut off	Sensitivity	Specificity
Arterial Base Excess	.844	.048	.000	-10.05	71.0%	8.2%

The value for area under the curve (AUC) in figure 7 for arterial base excess is excellent hence the cut off value is good for evaluation of patients at risk of death. It has a p-value of < 0.001, therefore favourable sensitivity and specificity characteristics.

7. DISCUSSION

Demographics and Risks to fire Outbreaks

Eighty fire victims were studied. About 56% (n=45) were males and 44% (n=35) females, a similar finding by Mugambi and Muriithi[8, 55]. Over 68% were aged between 18-35 years, the youngest being 13 years and oldest 80 years. The study revealed that the sample population resided in places generally considered of lower socioeconomic status, 80% of who resided in Nairobi County such as Kibera, Mathare, Kayole and Kawangware; and its immediate environs such as Mwiki and Kinoo. This infers to the kind of houses they dwelt in and probable non-modern simple materials used in their construction. This is important because most of the fire incidents (75%) happened indoors and at home. The high incidents of fires happening at home may be because precaution and prevention of fire outbreaks was not observed. Furthermore, the dwelling places may have been be so small and crammed with household items which may be so near the fire place that fire easily broke out. Rampant use of accelerants such as kerosene and open flames for household cooking and lighting added to the risk.

Mechanisms and Characteristics of Fire

The vast majority of the flame burn incidents happened in enclosed spaces at home, 30% happening around dinner time between 6pm to midnight. According to Kenya Bureu of Statistics (2017) kerosene, was the commonest source of energy in most Nairobi households, whereby 55.9% of them routinely depended on it for cooking needs[58]. This explains it being the commonest accelerant of fires in this study, whereby kerosene stove explosions and mishaps, caused 28.7% of the fires, which is similar to Ndiritu's (2005) finding. This was followed by candles and lamps which fell onto seats and bedding or set a loosely hanging curtain on fire, contributing to 16.3% of the mechanisms. This may explain most of the 27% fire incidents that occurred after midnight when most subjects fell asleep probably with a candle or a lamp on. Cooking gas leaks and explosions contributed to 13.8% of the mechanisms. Charcoal and electrical were among the least of causes.

Over 53% of all the mechanisms of fire resulted in burning of clothes only worn by the victims. About 40% of the incidences involved bedding and assorted household items mainly caused by housefires and candles/lamps falling onto bedding or seats.

Rescue and First Aid Administered Following Flame burns

Most of the victims managed to rescue themselves form the fire scene. External rescue arrived on average 16 minutes later. Fifty percent checked in a peripheral clinic/hospital and treatment was started before referral to KNH. This mirrors Ndiritu's finding of 48.2% in the year 2005[56]. The other 50% were brought straight to KNH; 55% (n=22) of them had a form of first aid administered. Arrival at the Kenyatta National Hospital averaged 8 hours 50 minutes after the incident. This is a great improvement from 20 hours and 25 minutes reported by Ndiritu G.S. (2005)[56].

All these factors may have contributed to the relatively low value of SpCO% measured among the subjects. However; these same factors seem not to have impacted on the outcomes of the studied patients.

Presenting Features Significant to Inhalational Injury

The most common complaint the victims presented with that would raise suspicion of inhaled smoke intoxication was confusion (n=23) and headache (n=21) followed by cough and dizziness. Hoarse voice and production of dark sputum that pointed towards significant smoke inhalation were recorded only in 12 cases. When examined, 70% of the patients presented with singed nasal hairs and 25% hyperemia and edema of the upper airway and 27% had reduced GCS. These indicated a likelihood of significant inhalational injuries and is close to Nthumba's and Ndung'u's finding who reported inhalational injury to be present in 60% of flame burnt patients [5, 6]. There was a significant correlation between altered GCS and upper airway hyperemia/ edema ($p < 0.01$)

Twenty percent of the victims were asleep when the fire broke out and 18.8% either had a seizure or were under the influence of drugs and alcohol. However, these state of altered mental state and wakefulness did not have significant correlation with %TBSA as assessed by Kendal Tau's test ($p=0.280$).

The overall mean of cutaneous TBSA was 30.9% with a median of 25%.

SpCO % Findings and its Correlations

The Mean SpCO in this study was 5.48%. This would fall into the category of no intoxication[25,31]. Factors that may explain the relatively low SpCO reading include the long interval of presentation at KNH's A&E, averaging 8hours 50 minutes, which is almost 2 half-lives of CO. Additionally, 50% of the patients had treatment commenced before arrival. More than 42% used volatile

hydrocarbons (Kerosene/LPG) whose carbon content may be less dense and combustion better than other heavier hydrocarbons hence producing less CO. Also time of exposure to the smoke was relatively short (16 minutes) before being rescued.

However, a few associations here below may be revealing:

Association of SpCO% with presenting complaints and fire characteristics: the mean SpCO of those presenting with headache, which is the most common CO poisoning presentation was 6.24%. Also those whose measurements were taken 4 hours or less from time incidence had a mean SpCO% of 6.9%.

Further analysis revealed no correlation of the SpCO% and the following: Scene of fire (p=0.960), Mechanism/source of fire (p=0.725), predominant burning materials (p=0.241), substance influence at time of incidence (p=0.214) and sleep at time of incidence (p=0.595)

Association of SpCO% with TBSA and Upper airway injury: since flame burn victims were at the scene of fire and smoke, it is expected that the higher the %TBSA the longer was the exposure to the fire and smoke hence the more the severity of inhalation injury. This is according to Lafferty and Monafo[3, 37]. In our study, significant inhalational injury that presented with upper airway hyperemia and edema had a strong correlation with %TBSA (p=0.016). Additionally, SpCO% had a significant and moderate association with upper airway hyperemia/edema (p=0.05). However, SpCO% had no correlation with singing of nasal hairs (p=0.271) nor %TBSA (p=0.144). This is similar to Baud's finding [23]. In comparison to Endorf's (2007) study which reported a mean COHb level of 13.7% versus a mean TBSA of 25.4%[38], this study revealed a more discordant finding of a mean SpCO of 5.48% versus a mean TBSA of 30.9%. Therefore, the extent of %TBSA is not a reliable indicator of severity of CO inhalational poisoning[23].

Association of SpCO with other laboratory parameters: Although the SpCO levels were low, analysis for any association with other laboratory parameters was done. The correlation revealed no association with pH levels (p=0.233), no correlation with arterial lactate levels (p= 0.356), neither was there any association with arterial base deficit (p=0.525). This lack of association with markers of tissue anaerobic respiration is expected at low levels of SpCO such as above which may cause no significant derangements in cellular/tissue metabolic processes.

Arterial lactate Measurement

Arterial lactate was obtained only on 39 subjects due to frequent unavailability of the service at the concerned laboratory. Only 7 subjects recorded lactate levels above 5mmol/L, but none had a value ≥10mmol/L. Overall mean arterial lactate was 2.91mmol/L. Baud et al (1991) reported that arterial lactate above 10mmol/L in patients without severe burns (TBSA of ≤ 15%) had a sensitivity of 87% in diagnosing cyanide intoxication[23,27.] In our study, no sample measured above 10mmol/L hence there may have been no significant cyanide exposure in any of the patients studied. Only one subject who was exposed to burning plastic tanks while asleep may have had cyanide exposure with a lactate of 9.17mmol/L. He died within 12 hours of admission.

Twenty Eight Day Sequelae

No neuropsychiatric sequela whatsoever was seen in 76.3% of the subjects. Agitation and confusion occurred in 12.6% of the participants, commonly within the first week of admission. Over 11% exhibited low mood and depression which was more common after the first week of admission. However, these neuropsychiatric manifestations did not have association with SpCO levels (p=0.298). This may be due to generally low levels of SpCO% recorded where only 7 subjects had SpCO of ≥10% which falls in mild CO poisoning category[31]. According to Blumenthal (2001), it is among those with CO levels above 10% that neuropsychiatric sequelae tend to occur 10-30% of times especially among the elderly patients.

Important Contributors of Death as an Outcome

Death being an important and ultimate outcome, it was assessed in light of various clinical parameters.

The subjects who died within 24 hours had a mean pH of 7.07, lactate of 5.74mmol/L, base deficit of -15.1mmol/L, TBSA of 63% and SpCO of 7.87%.

All the subjects with severe lactic acidosis (>5mmol/L) died within 28 days. Muriithi EK (2017) found that majority of such patients, died within seven days[55]. The correlation between lactate levels and death among flame burn patients was found to be strong and significant (p<0.001)

Other than lactate, base deficit also points towards anaerobiosis. It was found to have a strong and significant association with death (p <0.001).

Low arterial pH too was found to have a strong and significant correlation with death ($p<0.001$).

Cutaneous TBSA was also found to have a strong and significant correlation with death ($p<0.001$). Inhalational injury as manifested by upper airway hyperemia and edema in our study had a significant association with death as assessed by chi square test ($p=0.024$). All these findings echo the findings of Ndung'u and Muriithi[5, 55].

Additionally, death was found to have significant association with upper airway hyperemia and or edema ($p=0.024$) as well as reduced GCS ($p=0.01$).

However, SpCO levels was not found to have significant association with death ($p=0.708$).

Receiver Operator Characteristics of Selected Predictors of Death

The ROC was drawn up for three parameters: Lactate, TBSA and Base deficit.

The ROC for the arterial lactate showed an excellent area under the curve (AUC) of 0.792 achieving a statistical significance of 0.002 at a cut off value of 2.36mmol/L. This attained a sensitivity of 73.3% and specificity of 20.8% in predicting risk of death among flame burnt patients. A similar value was arrived at by Muriithi E.K (2017)[55].

The value for the AUC for TBSA% was found to be excellent (0.890) with a statistical significance of $p < 0.001$ at a cut off value of 32.5%. This TBSA had a sensitivity of 71% and specificity of 12.2% in predicting risk of death among flame-burnt patients.

The value for the area under the curve for arterial base excess was excellent (0.844) and achieved a p value of <0.001 indicating that the cut off value of -10.05mmol/L had a sensitivity of 71% and specificity of 8.2% in predicting risk of death. A similar value was arrived at by Muriithi E.K (2017)[55].

8. STUDY LIMITATION

a) Pre-hospital treatment and time lapse after incidence which averaged 8 hours 50 minutes may have precluded obtainment of the peak Carbon monoxide intoxication levels.
b) CO/CN toxicity symptoms are nonspecific and their symptoms may have been due to other inhaled toxicants and several other causes such as burn shock, effects of cutaneous burns among others.
c) Second commonest inhalational toxic gas, cyanide which could affect patient's outcome could not directly be measured by equipment available at the research centre.
d) Furthermore, lactate measurement was frequently unavailable at the research centre and only 39 of the participants had their samples tested for it.

9. CONCLUSION & RECOMMENDATION

9.1. Conclusion:

A large proportion of flame burnt patients presenting acutely to KNH have symptoms and signs that may raise suspicion of CO or CN inhalational poisoning. However, due to a relatively low mean SpCO% of 5.48% and low lactate levels averaging 2.91mmol/L in majority of them, these symptoms , being nonspecific in themselves may not be due CO or CN inhalational poisoning within the local context. Therefore CO and CN are unlikely to be significant factors in contributing to poor outcomes of flame-burnt patients in local context as hypothesized in this study.

The 28 day case fatality of 38.7% among the flame-burnt patients is as a result of factors well known to significantly contribute to mortality in these patients including inhalational injury, the extent and depth of TBSA leading to burn shock as depicted by low base deficit, blood pH and high lactate, among other factors.

9.2. Recommendations:

Despite of low SpCO% finding, many of the flame burnt patients have inhalational injury which has a strong positive correlation with their deaths and hence would still require early high flow oxygen administration and tracheal intubation.

Aggressive early treatment of the acid base derangements which have very strong correlation with death in flame burnt patients should be instituted accordingly.

A follow up study to determine CO levels among the flame burnt patients within 3 hours of burn incident may be done to find out the peak levels that may be found to be significant in contribution to morbidity and mortality outcome.

Finally, fire risk awareness campaigns should be relentless to stem most of the flame burns which are preventable.

10. REFERENCES

1. Kentis J., *An Essay on Burns Which Happen to Men in Mines,* 1797, Edinburg G&J Robinson.
2. Mock C., et al (eds); *WHO Plan for Burn Prevention and Care;* WHO 2008, Geneva.
3. Lafferty K.A. et al, *Smoke Inhalation Injury,* Medscape Sept 2015. (www.medscape.com).
4. Heimbach D.M., Waeckerle, J.F., *Inhalation Injuries,* Ann Emerg Med, 1988 Dec; 17(12): 1316-20.
5. Ndung'u, W.A., *The Value of Using Abbreviated Burns Severity Index Score in Predicting Outcome of Patients with Moderate to Severe Thermal Injuries at KNH;* MMed Postgraduate Dissertation, 2008, University of Nairobi, Kenya.
6. Nthumba P., Oliech J.S., *Outcome of Moderate to Severe Burns at KNH;* East and Central African Journal of Surgery 2005 Dec;10(2): 37-42.
7. Central Health Records and Statistics, Kenyatta National Hospital (2015).
8. Mugambi, J.W., *Management of Inhalation Injury and its Effect on Patients' outcome in Burns Unit KNH;* MSc Postgraduate Dissertation, 2014, University of Nairobi, Kenya.
9. Darling G.E, et al, *Pulmonary Complications in Inhalation Injuries with Associated Cutaneous burns,*J Trauma, 1996, Jan;40(1):83-9.
10. Buni, M.K., *Pattern of Burns as Seen at Kenyatta National Hospital,* MMed Postgraduate Dissertation, 2006, University of Nairobi, Kenya.
11. Mburu M.G., *Study of Paediatric Thermal Burns; Presentation, Causes and Management at KNH,* MMed Postgraduate Dissertation, 2014, University of Nairobi, Kenya.
12. Shisanya K.S.A., *Occurrence and Early Outcomes of Inhalation Burns Injury Patients Admitted to Kenyatta National Hospital,* Postgraduate Dissertation, 2007, University of Nairobi, Kenya.
13. Nelson T., *The Holy Bible, New King James Version,* Genesis Chap 3 and 4, Nashville 1982.
14. Gowlet J.A.J.,*The Discovery of Fire by Humans; A Long and Convoluted Process,* Phil Trans. R. Soc B 371: 20150164.
15. Townsend E., *The Cooking Ape: An Interview with Richard Wrangman,* Gastronomica, vol 5 No 1 (winter 2005) pp29-37.
16. Ryan C.M., et al, *Objective estimates of Probability of Death From Burn Injuries,* NEJM, 1998, 338:362.
17. Varon J.M., Marik P.E., *Carbon Monoxide Poisoning,* Emerg and Intensive Care Med, 1997.
18. Cobb N, Etzel R.A., *Unintentional Carbon Monoxide related Deaths in the US,* 1979-1988, JAMA 1991;266((5): 659-663.

19. Centers for Disease Control and Prevention, *Unintentional non-fire related Carbon Monoxide Exposures;* US, 2001-2003.
20. Hettiaratchy S., Dziewulski P., *Pathophysiology and Types of Burns;* BMJ 2004, June 12; 328 (7453): 1427-1429.
21. Edlich R.F. et al, *Thermal Burns*; Medscape Sept 2015. (www.medscape.com).
22. Alarie Y., *Toxicity of Fire Smoke; Critical Review,* Toxicology; Jul 2002 32(4), 259-289.
23. Baud F.J., et al, *Elevated Blood Cyanide Concentratioj in Victims of Smoke Inhalation,* NEJM, Dec 9 1991, 325(25);1761-66.
24. Gorguner M., Akgun M., *Acute Inhalation Injury,* J Med, 2010, Apr; 42(1): 28-35.
25. *www.atsdr.cdc.gov/ToxProfiles/tp201-c2.pdf*
26. Blumenthal I., *Carbon Monoxide poisoning,* JRSOC Med., 2001 Jun;94(6):270-272.
27. Rehberg S., et al, *Pathophysiology, Management and Treatment of Smoke Inhalation Injury;* Expert Review, Resp Med 2009 Jun; 3(3): 283-297.
28. Thom S.R., *Carbon Monoxide Mediated Brain Lipid Peroxidation in the Rat,* J Appl Physiol 1990; 68:997-1003. (PMID 2341364).
29. Christopher R.H., et al, *Myocardial Injury and Long Term Mortality Following Moderate to Severe Carbon Monoxide Poisoning,* JAMA, 2006 Jan; 295(4): 398-402.
30. Hampson G.B., et al, *Practice, recommendation in Diagnosis, Management and Prevention of Carbon Monoxide Poisoning,* Amj Respir Crit Care Med, 2012 Dec; 186(11):1095-1101.
31. Olson K., Smolling C., *Carbon Monoxide (acute),* BMJ Clin Evid 2008 Jul; 2008(2103).
32. Einhorn *IN. Physiological and toxicological aspects of smoke produced during the combustion of polymeric materials.* Environ Health Perspect *1975; 11:163.*
33. Hatlestad D, *Carbon Monoxide Poisoning, Incidence, diagnosis and Treatment,* EMS World; 2005 Oct. (www.emsworld.com).
34. Harper A., Croft-Barker J, *Carbon Monoxide Poisoning, Undetected by Both Patiens and Their Doctors,* Age, Ageing 2004 Mar; 33(2):105-9.
35. Simini B., *Cherry Red Discoloration in Carbon Monoxide Poisoning,* Lancet 1998 Oct; 352(9134) (PMID 9798630).
36. Hart G.B., et al, *Treatment of Smoke Inhalation by Hyperbaric Oxygen;* Journ of Emerg Med, 1985; 3(3): 211-215.
37. Monafo, W.W., *Initial Management of Burns,* NEJM 1996 Nov; 335(21): 1581-6.

38. Endorf F.W., Gamelli R.L., *Inhalation Injury, Pulmonary Perturbations and Fluid Resuscitation,* Jour of Burn Care and Research, 2007 Jan/Feb; 28(1):80-83.
39. www.accessdata.fda.gov
40. Kot J., et al, *Carbon Monoxide Pulse Oximetry Versus Direct Spectrophotometry for Early Detection of Carbon Monoxide Poisoning,* Anestez Jol inters Ter, 2008 Apr-Jun;40(2);75-8.
41. Feiner J.R., et al; *Accuracy of Carboxyhemoglobin Detection by Pulse Oximetry During Hypoxemia,* Anesth Analg 2013 Mar 11.
42. Barker S.J., et al; *Measurement of carboxyhemoglobin by Pulse Oximetry: a Human Volunteer study,*Anesthesiol., 2006 Nov; 105(5); 892-7.
43. Hampson N.B., *Noninvasive Pulse Oximetry Expedites evaluation and Management of Patients with Carbon monoxide Poisoning,* AMJ Emerg Med 2012 Nov; 30(9); 2021-4
44. Kathleen M., et al, *Outcome of children with Carbon Monoxide Poisoning Treated Normobaric oxygen,* Journal of Trauma, Injury, Infection and Critical Care; 1998 Jan; 44(1):149-154.
45. Cope R, *Overview of Smoke Inhalation,* The Merck Veterinary Manual (online) Dec 2014.
46. Shochat G.N., *Carbon Monoxide Toxicity;* Medscape , May 2016. (www.medscape.com).
47. Quinn D.K., et al, *Complications of Carbon Monoxide Poisoning: A Case Discussion and Review of Literature; Primary Care Comparison,* J Clin Psychiatry 2009;11 (2):74-79.
48. Buckley N.A. et al, *Hyperbaric Oxygen for Carbon Monoxide Poisoning: a systematic Review and Critical Analysis of Evidence;* Toxicol Per 2005; 24 (2):75-92.
49. Buckey N.A., et al, *Hyperbaric Oxygen for Carbon Monoxide Poisoning, Cochrane Database*; *Systematic Review*, 2011; (4):CD002041.
50. Hall A.H., et al, *Sodium Thiosulphate or Hydrocobalamin for the Empiric Treatment of Cyanide Poisoning*; Ann Emerg Med 2007 Jun;49(6):806-13.
51. Mashall S.W., et al, *Fatal Residential Fires, Who Dies and Who Survives?* JAMA, 1998 May; 279(20): 1633-7.
52. Hampson N.B., Hauff N.M., *Risk Factors for Short Term Mortality From Carbon Monoxide Poisoning Treated with Hyperbaric Oxygen,* Crit Care Med 2008; 36(9): 2523.
53. Bizovi K.E., Leikin J.D., *Smoke Inhalation Among Firefighters,* Occup Med, 1995 Oct-Dec; 10(4): 721-33.
54. Raub J.A., et al, *Carbon Monoxide Poisoning- A Public Health Perspective*, Toxicology 2000; 145:1-14. (PMID 10771127).

55. Muriithi E.K, *Correlation of Initial Serum Lactic Acid and Base Deficit with Early Mortality in Patients Admitted With Major Thermal Burns at Burns Unit in Kenyatta National Hospital*, MMed postgraduate Dissertation, 2017, University of Nairobi, Kenya.
56. Ndiritu S.G, *Epidemiological Pattern, Risk and Safety Awareness and Prehospital Care of Burns patients; A Cross Sectional Survey at Kenyatta National Hospital,* MMed postgraduate Dissertation, (2005), University of Nairobi, Kenya.
57. KNBS, '*Kenya Population and Housing Census, 2009*' (2010)
58. KNBS, '*Exploring Kenya's Inequality, "pulling Apart or Pooling Together"'- Nairobi County*, (2017)

APPENDIX A: CONSENT

RESEARCH TOPIC: ASSESSMENT OF CARBON MONOXIDE AND CYANIDE INHALATIONAL POISONING IN PATIENTS WITH FLAME BURNS AT KENYATTA NATIONAL HOSPITAL

ENGLISH VERSION

This form is to ask for Consent from patients and/or their kin who present to KNH with flame burns and who would be assessed for possible toxic smoke poisoning.

Principal investigator: DR. MACKUTWA, Edward Nandi

Institution: School of Medicine, Department of surgery- University of Nairobi

Supervisors: PROF. KHAINGA S.O., DR. NDUNG'U J.M. and DR ANANGWE C.

This informed consent has three parts:

- Information sheet (to share information about the research with you)
- Certificate of Consent (for signatures if you agree to take part)
- Statement by the researcher

Part I: Information sheet

1. ***Introduction***

My name is Dr. Mackutwa Edward, a surgical post graduate student at the University of Nairobi's School of Medicine. I am carrying out a study entitled "**ASSESSMENT OF CARBON MONOXIDE AND CYANIDE INHALATIONAL POISONING IN PATIENTS WITH FLAME BURNS AT KENYATTA NATIONAL HOSPITAL"**

This will be carried out through participants answering specific questions only related to the patient through interviewer administered questionnaire, patient examination and laboratory tests which are

necessary and form part of patient management process. I am inviting you to willingly take part in this study

2. Benefits of the Study

Important finding on the participant will be passed to the attending team for treatment decisions. The results of the study may inform management decisions of similar patients in the future to better their care. It will shed light to information hither to not known for instance, the incidence and levels of carbon monoxide poisoning in fire victims presenting to KNH, which is one of the findings this study seeks to determine.

3. Costs and Potential Harm

If you refuse to participate in the study be assured that your decision will not jeopardize the required care for the patient. Furthermore, this study poses no harm to the patient and there will be no extra cost incurred for participating in the study. There will be no financial grant to the participants.

4. Your Obligation

If you agree to participate, you will be asked to provide a few personal information of the patient, other details related to the burn incidence and patient's condition or symptoms before, during and after the fire incidence. Afterwards we will examine the patient and do some laboratory tests which also will be needed for his/her treatment.

5. Confidentiality

All the information gathered will be taken in confidence and no one will see it except my assistant, my supervisors and I, all who are duty-bound to ensure confidentiality.

The patient's name or identity will not appear in any research document. The information about the patient will be identified by a unique research number and only the researchers can relate the number to you/your patient as a person. Other than for (2) above, your information will only be used for this study and will not be shared with anyone else unless authorized by the Kenyatta National Hospital/University of Nairobi - Ethics and Research Committee (KNH/UoN-ERC).

6. Study Credibility and Legitimacy

This study was approved by my three supervisors. It was appraised and approved by the Chairman of the Department of Surgery, School of Medicine at the University of Nairobi. It was then submitted to KNH/UoN-ERC which reviewed and approved it to be done for a duration of six months. KNH/UoN-ERC is the regulatory body in the hospital whose work is to make sure research process is safe for the participants and that you are protected from harm.

7. Who to Contact

You can ask questions or seek clarifications about the study any time you wish to. If need be, you may also talk to anyone you are comfortable with about the research before making a decision.

If you have any query about the research you want addressed by another person other than the researchers, please feel free to contact the following who will address your concerns:

a) Secretary, KNH/UoN-ERC
   P.O. Box 20723 -00202
   KNH, Nairobi
   Tel: +254-020-2726300-9 ext. 44355
   Email: KNHplan@Ken.Healthnet.org Or uonknh_erc@uonbi.ac.ke
   Twitter: @UONKNH_ERC https://twitter.com/UONKNH_ERC
   Facebook: https://www.facebook.com/uonknh.erc

b) Research Supervisor from Kenyatta National Hospital
   DR. ANANGWE Charles.
   Department of Anesthesia and Critical care
   P.O. Box 20723 -00202
   KNH, Nairobi
   Tel: 254-020-2726300
   Cell: 0733 617 677
   Email: charlesanangwe@gmail.com

c) Research Supervisors from University of Nairobi

- PROF. KHAINGA Stanley Ominde
  Department of Surgery, School of Medicine, University of Nairobi
  P.O. Box 19676 -00202, KNH, Nairobi
  Tel: 0202726300
  Cell: 0723 436408
  Email: skhainga@yahoo.com

- DR. NDUNG'U James M.
  Department of Surgery, School of Medicine, University of Nairobi
  P.O. Box 19676-00202, KNH, Nairobi
  Tel: 0202726300
  Cell: 0722 522253
  Email: jndungu49.jm@gmail.com

d) Principal Researcher:

DR. MACKUTWA, Edward Nandi.
Department of Surgery, School of Medicine, University of Nairobi
P.O. Box 19676-00202, KNH, Nairobi
Mobile phone: 0721 249119 (reachable any time)
Email: eddynandi@gmail.com

Part II: Consent Certificate (confidential once signed) ***Research Track Number _____***

I …….....…..freely give consent to take part in the study conducted by Dr. Mackutwa Edward N, the nature of which has been explained to me by him/his research assistant. I have been informed and have understood that my participation is voluntary and understand that I am free to withdraw from it any time I wish so and this will not in any way alter the care given to me/my patient. The results of the study may or may not benefit me/my patient directly, but may benefit similar future patients. Furthermore, it will help Medical professionals to better understand "**ASSESSMENT OF CARBON MONOXIDE AND CYANIDE INHALATIONAL POISONING IN PATIENTS WITH FLAME BURNS**"

SIGNED CONSENT……

(Patient/Kin)

Date.....

DD/MM/YY

SIGNED ASSENT …….....……

(13-18 Year olds)

Date……

DD/MM/YY

Thumb print of participant if Unable to sign due to illiteracy

Statement by a witness if participant is illiterate

I have witnessed the accurate reading of the consent form to the participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Name of witness……

Signature of witness……..

Date…….....……

Part III: Statement by the researcher

I have clearly read out the information sheet to the participant, and to the best of my ability made sure that the participant understood the following:

- All information gathered will be treated with confidentiality.
- Refusal to participate or withdrawal from the study will not compromise the quality of care and treatment given to the patient.

The results of this study might be published in a reputable journal to enhance the knowledge of the "**ASSESSMENT OF CARBON MONOXIDE AND CYANIDE INHALATIONAL POISONING IN PATIENTS WITH FLAME BURNS**"

Also I confirm that the participant was given opportunity to seek clarification about his concerns in the study, and all the queries clarified to the best of my ability.

I confirm that the individual has not been coerced into giving consent, and the consent has been given freely and voluntarily.

A copy of this Informed Consent Form has been provided to the participant and duly signed by the participant.

Name of researcher taking consent……

Signature of researcher taking the consent……

Date……

UTAFITI: ATHARI ZA GESI YA SUMU KWENYE MOSHI ULIOVUTWA NA MAJERUHI WA MOTO WANAOTIBIWA KATIKA HOSPITALI KUU YA KENYATTA

TAFSIRI LA KISWAHILI

Fomu hii ni ya kuuliza kibali kujumuishwa kwenye utafiti wa majeruhi wa moto au jamaa zao wanaofika Hospitali kuu ya Kenyatta na wanadhaniwa kuathirika na moshi walioivuta.

Mtafiti mkuu: DKT. MACKUTWA, Edward Nandi

Wahadhiri wasimamizi: PROF. KHAINGA S.O., DKT. NDUNG'U J.M. na DKT ANANGWE C.

Wote wa kitengo cha upasuaji cha Chuo Kikuu cha Nairobi na hospitali kuu ya Kenyatta.

Makubaliano haya yana sehemu tatu:

- Maelezo kuhusu utafiti huu.
- Cheti cha Kibali (kitakachotiwa sahihi na wahusika wanaokubali kujumuishwa utafitini)
- Ithibati ya mtafiti

Sehemu ya kwanza: Maelezo

1. ***Utangulizi***

Jina langu ni Dkt Mackutwa Edward, mwanagenzi wa upasuaji katika chuo kikuu cha Nairobi. Ninafanya utafiti "**ATHARI ZA GESI YA SUMU KWENYE MOSHI ULIOVUTWA NA MAJERUHI WA MOTO WANAOTIBIWA KATIKA HOSPITALI KUU YA KENYATTA**"

Utafiti utajumuisha wahusika kuulizwa maswali kuhusu muathiriwa, uchunguzi wa mwili na wa damu ambazo pia zitatumika kwa kusaidia matibabu ya mgonjwa. Ninakualika kujumuika kwenye utafiti huu kwa hiari.

2. ***Faida ya Utafiti huu***

Ukaguzi wowote muhimu utatumika kufaidi matibabu ya muathiriwa. Matokea ya utafiti huu huenda yatatasaidia jinsi ya kuwashughulikia majeruhi kama hao siku za usoni. Kwa mfano, tutapata kujua kiwango cha gesi ya sumu iitwayo carbon monoxide kwa majeruhi wa moto wanaotibiwa KNH, ambayo hadi sasa haijulikani, ndiposa tuwe na mikakati ya kuwatibu ipasavyo.

3. Gharama na Madhara za Utafiti

Natoa hakikisho kwamba hata kama hutaki kushiriki kwenye utafiti huu, wewe au mgonjwa wako hautachukuliwa vibaya na utapata matibabu yanayostahili. Utafiti huu haupanii kuleta madhara aina yoyote kwa muathiriwa. Hautatozwa fedha za ziada kwa minajili ya utafiti huu wala hakuna fedha mhusika atapewa.

4. Jukumu Lako Katika Utafiti

Ikiwa utakubali, utaulizwa maswali machache ya kibinafsi yanayokuhusu, maelezo ya tukio, na hali ya muathiriwa kabla, wakati na baada ya tukio. Baadaye tutachunguza majeraha na kupima damu ya muathiriwa ambazo vilevile zitahitajika kuelekeza matibabu.

5. Faragha ya Habari za Mhusika

Habari zote zitakazokusanywa kwa ajili ya utafiti zitabanwa na watafiti na hazitatolewa ovyo. Jina au kitambulisho cha mgonjwa hayatanakiliwa popote ila tu atapewa nambari maalum ya utafiti. Watafiti watatumia mbinu fiche itakayokutambulisha kwao. Licha yaliyokaririwa (2), habari za mgonjwa zitatumiwa tu kwa ajili ya utafiti huu na hazitatolewa kwa yeyote pasipo na idhini ya Kamati ya Maadili ya Utafiti wa Hospitali Kuu ya Kenyatta na ile ya Chuo Kikuu Cha Nairobi (kwa ufupi KNH/UoN-ERC).

6. Uhalali wa Utafiti huu

Utafiti huu umekubaliwa na wahadhiri wasimamizi wangu, ukapigwa msasa na Mwenyekiti wa kitengo cha upasuaji wa chuo kikuu cha Nairobi ambaye aliuwasilisha kwa Kamati ya Maadili ya Utafiti wa Hospitali Kuu ya Kenyatta na ile ya Chuo Kikuu Cha Nairobi (KNH/UoN-ERC) ambayo iliudhinisha uweze kufanywa kwa muda wa miezi sita. Kamati hii ndio ihakikishayo usalama wa wanaohusishwa kwa utafiti na kwamba hawadhuriwi kwa vyovyote vile.

7. Jukwa la Malalamishi na Habari Zaidi

Waweza kutuuliza maswali yoyote wakati wowote au umuulize yeyote utakaye kuhusu mchakato wa utafiti huu kabla au hata baada ya kukubali kuhusishwa.

Iwapo una swali lolote kuhusu utafiti huu ambao waona heri lishughulikiwa na mtu mwingine isipokuwa watafiti, waweza kuwasiliana na wafuatao ambao wako tayari kukushughulikia ipasavyo:

a) Katibu, KNH/UON-ERC

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b) Msimamizi Kutoka Hospitali kuu ya Kenyatta

DKT. ANANGWE, Charles

Kitengo cha Anesthesia na Wagonjwa Mahututi

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Rununu: 0733 617 677

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c) Wahadhiri Wasimamizi Kutoka Chuo Kikuu cha Nairobi:

- PROF. KHAINGA, Stanley O.

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d) Mtafiti Mkuu (mimi)

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   KNH, Nairobi

   Rununu: 0721 249119 (wazi usiku na mchana)

   Barua pepe: eddynandi@gmail.com

Sehemu ya Pili: Cheti cha Kibali (siri baada ya kutiwa sahihi) ***Nambari Maalum_____***

Mimi …….....….ninakubali kwa hiari kuhusishwa kwa utafiti unaoendelezwa na Dkt. Mackutwa Edward N, kuambatana na maelezo yeye mwenyewe/ msaidizi wake amenipa. Ninaelewa kwamba nimehusishwa kwa hiari na kwamba niko huru kujiondoa wakati wowote nitakao hata bila sababu, na hii haitaathiri kwa namna yoyote matibabu ipasayo. Aidha naelewa kwamba matokeo ya utafiti huu huenda usinifaidi binafsi lakini huenda ukawa wa manufaa siku zijazo kwa waathiriwa wa moto na moshi kama nilivyo.Kuna uwezekano utafiti huu utaongeza maarifa kwa taaluma ya utabibu kuhusu "**ATHARI ZA GESI YA SUMU KWENYE MOSHI ULIOVUTWA NA MAJERUHI WA MOTO**"

SAHIHI (KIBALI HALISI)……

(Mgonjwa/jamaa)

Tarehe.....

Siku/mwezi/mwaka

KIBALI MAALUM …….....……

(Watoto wa miaka 13-18)

Tarehe ……

Siku/mwezi/mwaka

Chapa cha kidole gumba cha kushoto kwa wasio na elimu ya kusoma na kuandika

Taarifa ya shahidi ya makubalino na mhusika asiyejua kusoma

Nimeshuhudia mshusika akisomewa kwa njia inayoeleweka kwa rahisi, naye akapewa fursa nzuri ya kuuluza maswali. Nina dhibitisha mhusika alipeana kibali kwa hiari yake mwenyewe.

Jina la shahidi.....……

Sahihi la shahidi.....……..

Tarehe.....……

Siku/mwezi/mwaka

Sehemu ya tatu: Taarifa ya Mtafiti

Nimemusomea mhusika na kadiri ya uwezo wangu kumuelewesha yafuatayo:

- Habari zozote zitokazo kwake zitawekwa siri.
- Kukataa kupeana kibali cha kuhusishwa kwa utafiti huu haitaathiri matibabu anayostahili.
- Matokeo ya utafiti huu kwa jumla utachapishwa katika jarida la kisayansi au utabibu ama upasuaji kuweza kuchangia maarifa ya "**ATHARI ZA GESI YA SUMU KWENYE MOSHI ULIOVUTWA NA MAJERUHI WA MOTO**"

Nimehakikisha kwamba mhusika amepewa fursa kamili ya kuuliza maswali kuhusu kuhusika kwake kwa utafiti huu na kwamba kwa kadiri ya uwezo wangu nimemuelewesha ipasavyo.

Ninahakiki kwamba mhusika hajalazimishwa kupeana kibali kuhusika kwenye utafiti huu bali amekubali kwa hiari.

Nakala ya kibali hiki kimewasilishwa kwa mhusika naye akatia sahihi ipasayvo.

Jina la mtafiti aliyepewa kibali cha mhusika……

Sahihi ya mtafiti mhusika**.....**……

Tarehe……

Siku/mwezi/mwaka

APPENDIX B: LUND BROWDER CHART

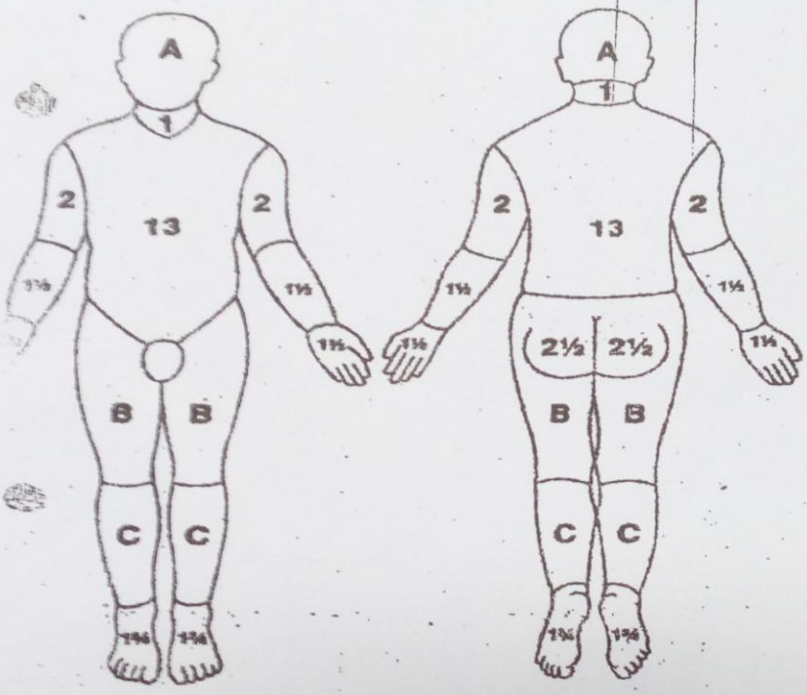
BURN RECORD

NAME
ADDRESS
AGE M/F
ADMISSION WEIGHT WARD

BURN	DATE	TIME OF ADMISSION	TYPE OF BURN
			INHALATION INJURY YES/NO
REGISTERED			PLACE AND TIME OF BURN
			INFORMANT

LUND AND BROWDER CHARTS

IGNORE
SIMPLE ERYTHEMA



Superficial
Deep

REGION	%
HEAD	
NECK	
ANT. TRUNK	
POST. TRUNK	
RIGHT ARM	
LEFT ARM	
BUTTOCKS	
GENITALIA	
RIGHT LEG	
LEFT LEG	
TOTAL BURN	

RELATIVE PERCENTAGE OF BODY SURFACE AREA AFFECTED BY GROWTH

AREA	AGE 0	1	5	10	15	ADUL
A = ½ OF HEAD	9½	8½	6½	5½	4½	3½
B = ½ OF ONE THIGH	2¾	3¼	4	4½	4½	4¾
C = ½ OF ONE LEG	2½	2½	2¾	3	3¼	3½

APPENDIX C: INTERVIEWER ADMINISTERED QUESTIONNAIRE

I. DEMOGRAPHIC DATA

Research No_____ Date_____ & Time_____ of Enrolment/Arrival

Sex_____Age (yrs) _____Residence_____

II. FOCUSED HISTORY

a. Date_____ & time of _____ Incidence

b. Scene of Incidence:

- Place of incidence: Home ☐ Work place ☐ Other(specify) ☐ _____
- Inside the House/Building ☐
- Outdoors ☐
- Vehicular ☐
- Specify burning material_____

c. Rescue Time_____Rescuer to hosp: Police ☐ EMS* ☐ Family ☐ others_____

d. First aid/Care given_____Time started_____

e. Complaints

• Confusion/ loss of consciousness	☐	Fatigue/Lethargy	☐
• Convulsions	☐	Headache	☐
• Shortness of breath	☐	Nausea/Vomiting	☐
• Cough	☐	Visual impairment	☐
• Dark sputum	☐	Dizziness	☐

- Prior substance use/ co-morbidities/asleep (specify)_____

III. INITIAL CLINICAL EXAMINATON

1. GCS_____Neurological signs_____
2. Other injuries (specify)_____
3. %TBSA Burn Per Lund and Browder: Total_____Approx 2^0_____3^0_____%
4. Site of Burn (indicate on Lund Browder Chart)_____
5. Carbonaceuos sputum Yes ☐ No ☐
6. Singed nasal Vibrissae Yes ☐ No ☐
7. Soot ☐ hyperemia ☐ Edema ☐ in the upper airway (Nostrils to oropharynx)
8. Cardiovascular findings: BP____Others_____
9. Other findings_____

NB: Tick ☑ as appropriate

*Emergency Medical Services

APPENDIX D: LABORATORY MEASUREMENTS & TARGETED OUTCOME INDICATORS

RESEARCH TRACK NO_____DATE OF ENROLMENT_____

	INITIAL (Day 0)	Week 1 (specify day of onset)	Week 2 (specify day of onset)	Week 3 (specify day of onset)	Week 4 (specify day of onset)
LABORATORY WORKS					
%SpCO		N/A			
%SpO_2					
PaO_2					
$PaCO_2$					
Arterial Blood Lactate					
PvO_2 (if lactate >5mmol/L)					
Arterial Base Excess					
Arterial Blood pH					
MANAGEMENT					
Oxygen (Method of delivery)		N /A			
Any antidote given (specify)					
OUTCOMES					
Neuropsychiatric features (parkinsonism, personality changes, memory changes, cognitive changes etc)					
Cardiovascular system (failure, ischemia)					
Death					
Others (specify)					

APPENDIX E: KENYATTA NATIONAL HOSPITAL- UNIVERSITY OF NAIROBI ETHICS AND RESEARCH COMMITTEE APPROVAL