SHOULD ETOMIDATE BE THE INDUCTION AGENT OF CHOICE IN THE EMERGENCY DEPARTMENT?

BY

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RESEARCH DISSERTATION

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DECLARATION

I declare that the research dissertation hereby submitted to the University of Limpopo, for the degree of Master of Medicine in Anaesthesiology has not previously been submitted by me for a degree at this or any other University; that it is my work in design and in execution, and that all material contained herein has been duly acknowledged.

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ACKNOWLEDGEMENTS

"**MAY YOU BE RICHLY BLESSED**"

<u>DEDICATION</u>

This work is dedicated to my family. My husband Jerry, thank you for caring, for having faith in my abilities and endless encouragement. To my daughters Phatutshedzo and Rendani, you girls rock my world.

LIST OF ABBREVIATIONS AND ACRONYMS

ASA	American Society of Anaesthesiology
Class I	A normal healthy patient
Class II	A patient with mild systemic disease
Class III	A patient with severe systemic disease
Class IV	A patient with severe systemic disease that is a constant threat to life
Class V	A moribund patient who is not expected to survive without the operation
Class VI	A declared brain-dead patient whose organs are being removed for donor purposes
Class E	Emergency
BP	Blood Pressure
$CMRO_2$	Cerebral Metabolic rate for Oxygen
CO	Cardiac Output
CPP	Cerebral Perfusion Pressure
DBP	Diastolic Blood Pressure
DGMH	Dr George Mukhari Hospital
ED	Emergency Department
EEG	Electroencephalograph
HR	Heart Rate
ICP	Intracranial Pressure
IOL	Intraocular Pressure
MAP	Mean Arterial Pressure
RSI	Rapid Sequence Intubation
SBP	Systolic Blood Pressure
SVR	Systemic Vascular Resistance

<u>LIST OF TABLES AND FIGURES</u>

1.   ABSTRACT

Purpose: The purpose of this study was to determine whether etomidate should be the induction agent of choice for Rapid Sequence Intubation in the Emergency department due to its haemodynamic stability.

Objective: To measure the haemodynamic effects of etomidate post- induction in patients undergoing Rapid sequence intubation.

Methods: This was a prospective, randomized, observational and unblinded study. The study was conducted at DR GEORGE MUKHARI HOSPITAL theatre unit on 45 patients between the ages of 11 and 65 years of age who fall under the American Society of Anaesthesiology classification (ASA) IE − IIIE. Each patient had an established intravenous line, was pre-oxygenated and then received etomidate (0.2 mg – 0.3 mg/kg). Cricoid pressure was applied immediately following loss of consciousness. Suxamethonium 1.5 mg/kg or Rocuronium 1.2 mg/kg was administered and this was followed by endotracheal intubation 60 seconds later.

Data collected included amongst others vital signs:- Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure and Mean Arterial Pressure which were measured pre-induction, immediately post-intubation, 2.5 minutes, 5 minutes and 7.5 minutes later by a non invasive automated blood pressure monitor.

Statistical Analysis: Descriptive statistical analysis was applied using the SPSS (Statistical Programme for Social Sciences) to demonstrate the demographics and ASA classifications of the patients in the study. Mean standard deviations were calculated for both males and

females in the study. Changes in vital signs immediately post-intubation were graphically demonstrated. The changes in vital signs (HR, SBP, DBP and MAP) were calculated and differences in such changes over-time were expressed as p-value. Significant differences in changes of the vital signs were noted if $p \leq 0.05$.

Results: The blood pressure increased immediately following intubation due to laryngoscopy and intubation. As anaesthesia progressed (i.e. 2.5 minutes, 5 minutes and 7.5 minutes later) the blood pressure gradually declined to levels lower than pre-induction values but at acceptable levels.

Conclusion: Etomidate is an effective anaesthetic induction agent as it is haemodynamically stable and thus should be used in an Emergency department.

2. INTRODUCTION

2.1 Problem Statement

A number of pharmacological agents are used for rapid sequence intubation (RSI) and some are associated with haemodynamic instability which is characterised by:- a drop in blood pressure (BP), elevation of heart rate (HR), depression of the myocardium and vasodilation of the capacitance vessels. These changes can be detrimental to the elderly, hypovolaemic and patients with poor cardiac reserve.

Laryngoscopy and intubation violate the patients' protective airway reflexes and lead to hypertension and tachycardia. The available evidence on etomidate suggest that it is an effective induction agent for RSI as it protects myocardial and cerebral ischaemia, decreases the risk of aspiration and has a stable haemodynamic profile [1,2].   The intention of this study is to find evidence that supports or negate the statement above.

2.2 Purpose of the Study

- To determine whether etomidate should be the induction agent of choice for RSI in the Emergency department (ED) due to its haemodynamic stability.

2.3 Objective of the Study

- To measure the haemodynamic effect of etomidate post induction in patients undergoing RSI in the ED.
- To determine changes in blood pressure following the use of etomidate.

3. LITERATURE REVIEW

3.1 Rapid Sequence Intubation

Rapid sequence intubation (RSI) is a technique used in an emergent care of patients who present in the ED and require placement of emergency airway [1]. It requires administration of a potent intravenous agent to induce unconsciousness followed immediately by rapidly acting neuromuscular blocking agent in order to facilitate tracheal intubation [2, 3].

An ideal induction agent for emergency airway management should be smooth and rapidly acting, permit optimum intubating conditions and have minimal cardiovascular, respiratory, cerebral excitatory side effects [3]. No single induction agent meets all these requirements. Prior reports suggest that etomidate with its rapid induction of sleep and associated stable haemodynamic may prove useful in many patients requiring RSI in an emergency setting [1].

3.2 Basic Pharmacology of Etomidate

Etomidate is a carboxylated imidazole compound that was introduced in Europe in 1972. It acts by modulating and mimicking gamma-aminobutyric acid type A receptors [5]. It is soluble in water and chemically unstable in a neutral solution, it is therefore formulated in a solvent called propylene glycol solution with a pH of 6.9 and an osmolality of 4640mOsm/ℓ [4]. Etomidate has been used for sedation, induction and maintenance of anaesthesia and during electroconvulsive therapy as it produces longer seizures than possible with other hypnotics [4] and has no analgesic properties [4 and 5].

Etomidate was hailed early as an ideal agent due to its many favourable properties as an induction agent including rapid onset of action (it induces loss of consciousness in 5 to 15 seconds), rapid recovery (5 to 15 minutes), cerebral protective properties by reducing intracranial pressure and decreasing cerebral oxygen consumption and it does not cause histamine release; therefore it has minimal cardiovascular and respiratory effects [2-9].

The induction dose varies from 0.2 – 0.5 mg/kg intravenously. It is highly protein bound (75% bound to albumin), highly lipid soluble (allows for rapid distribution into most organs and tissues) and exist largely in a non-ionized fraction at physiological pH [5]. It is rapidly metabolized by easterase hydrolysis in the plasma and liver forming etomidate carboxylic acid, about 75% of metabolites are excreted in urine, 13% in feaces and 10% in bile [2, 3 and 7]. The pharmacokinetics of etomidate are shown in table 1 [5].

Table 1: Etomidate Pharmacokinetics

• Distribution half – life	2.6 ± 1.3 min
• Elimination half – life	4.6 ± 2.6 hours
• Total apparent volume of distribution	4.5 ± 2.2 ℓ/kg
• Total plasma clearance	860 ± 230ml/min

Systemic Effects

Cardiovascular system

- The minimal effect of etomidate on the cardiovascular function sets it apart from other rapid-onset induction agents [4 and 5].
- Most studies done in the ED and on elective procedure have demonstrated no myocardial depression, no effects on the conduction system, no increase in myocardial oxygen consumption, no histamine release and limited sympathetic

effects and baroreceptor function [4 and 5]. This makes it ideal for patients with poor cardiac reserve and those who are volume depleted [10].

Central nervous system

- The primary action of etomidate on the central nervous system is hypnosis.
- Intracranial effects are summarized in Table 2 [5 and 10].

Table 2: Central nervous system effects of etomidate

- Reduces cerebral blood flow by up to 34%.
- Reduces cerebral metabolic rate of oxygen consumption by up to 45%.
- Intracranial pressure (ICP) is decreased by 50%.
- MAP and HR remains unchanged.
- Maintains cerebral perfusion pressure (CPP). [CPP = MAP – ICP].
- Net increase in cerebral oxygen supply - demand
- Reduce brain pathology after acute ischaemic insult in animals.
- Association with myoclonic movement.
- May cause increase in epileptic Electroencaphalograph [EEG] in patients with pre-existing epileptic foci.

Respiratory system

- Etomidate has minimal effects on ventilation.
- It does not induce histamine release therefore it can be safely used in a reactive airway disease [4].

Endocrine system

- Etomidate inhibits steroid synthesis by inhibiting the mitochondrial 11 β hydroxylase enzyme of adrenal steroid synthesis pathway [4 and 11]. This occurs 30 min after a single induction dose and can last for up to 24 hours [4].
- Concerns have been raised about the possible adrenal suppression and subsequent adverse effects [11]. In 1983 a trend towards increased mortality was reported in

critically ill patients receiving continuous infusion of etomidate [5, 8 and 10], as a result the use of infusion for critically ill patients was abandoned [3, 5, 11 and 12].

- Controversy exists regarding adrenocortical effects after a single dose of etomidate. Clinically significant adrenocortical suppression has yet to be reported, as there are no known reports of any negative outcome associated with single induction dose [12]. Single induction dose are still used as the effect is transient but should be avoided in critically ill patients [4 and 11].

Side effects [10]

1. Pain on injection

    This can be eliminated by:-

    - Pre-induction dose of Lidocaine and Fentanyl.

    - Using central venous access.

    - Pre-medicating with opiod or benzodiazepines

2. Myoclonus

    Reported in up to 35% of cases. These movements may be violent and random they are not associated with seizure activity. This can be eliminated by:-

    - Pre-induction dose of 1.5mg./kg of Lidocaine or 1.5 µg/kg Fentanyl.

    - Aborted by rapidly injecting the drug.

    - Using a rapidly acting muscle relaxant after etomidate injection.

3. Thrombophlebitis/venous irritation
4. Nausea and vomiting

3.3 Pharmacology of other induction agents

Propofol[13]

- Commonly used in theatre

- Is (2.6 diisopropylphenol) an alkylphenolcompound.

- Uses include:-   Induction of anaesthesia 1.5 – 2.5 mg/kg IV

    Maintenance of anaesthesia 50 – 150mg/kg/min IV

    Sedation 25 – 75mg.kg/min IV

    Antiemetic 10 – 20mg IV

Advantages:

- Rapid induction of anaesthesia (15 – 45 sec)

- Rapid emergence from anaesthesia (5 – 10 min)

- Does not trigger malignant hyperthermia

Disadvantages:

- Pain on injection

Systemic effects

Central nervous system

- Decrease in $CMRO_2$, CBF as well as ICP.

Cardiovascular system

- Produces a more profound cardiovascular depression than Thiopentone by causing myocardial depression and a decrease in SVR.

- Alters the baroreflex mechanism, resulting in a smaller increase in HR for a given decrease in arterial pressure.

Respiratory System

- Produce a dose dependent respiratory depression.

- Produce bronchodilation.

Thiopentone[14]

- Is an ultrashort acting barbiturate that produces general anaesthesia by acting on $GABA_A$ receptors in the brain.

- Induction dose is 3–7mg/kg.

- It acts rapidly by producing unconsciousness within 30–45 sec and consciousness returns in 5–10 min.

Systemic Effects

Central Nervous System [13]

- Produces decrease in $CMR0_2$ and CBF, thereby lowering IOP.

- It possesses potent anticonvulsant activity.

- It is neuroprotective secondary to its ability to decrease oxygen demand.

Cardiovascular system

- Decreases CO, systemic arterial pressure and peripheral vascular resistance. These effects can be profound in patients with pre-existing hypovolaemia or cardiac disease.

Respiratory System

- Produces a dose dependent respiratory depression

Ketamine[15]

- Is a phencyclidine derivative that produces dissociative anaesthesia (i.e. patient is detached from surrounding).

    Induction doses:-   1 -2mg IV

    5 – 10mg IM

    8mg rectally

- Has hypnotic, analgesic and amnestic effects

- Has a slower onset of anaesthesia when given intravenously (1 – 5min) and alteration depends on the rate of administration.

Systemic Effects

Central nervous system

- Patients may be agitated on recovery and may experience unpleasant dreams for up to 24hrs after the drug was given.

- Ketamine causes a rise in ICP and should not be used in head injured patients.

Cardiovascular System

- Causes mild stimulation of CVS mediated through SNS, BP rises by about 25% and HR by about 20%.

Respiratory System

- Does not cause significant ventilatory depression.

- Airway reflexes are maintained.

- Produces bronchodilation

3.4 Incidence of post intubation changes of different agents

Different studies have been done previously looking at different anaesthetic induction agents and their impact on haemodynamics.

A study by Mohammed-HadiSafaee *et* al. [16] investigated haemodynamic variations of two different drugs used for induction: Thiopentone vs Propofol. This study reveals that 2 mg/kg of Propofol causes less haemodynamic variations when compared with Thiopentone 5 mg/kg. An increase in HR was seen in both groups during laryngoscopy and tracheal intubation in both groups and a slight decrease in blood pressure of short duration were also observed in both groups. This study did not indicate to what degree were these differences and what the general outcome were [16].

Lebowitz *et al.* [17]compared the cardiovascular effects of Midazolam and Thiopentone for induction of anaesthesia in America Society of Anaesthesia (ASA) III surgical patients and it was concluded that cardiac output (CO), mean arterial pressure (MAP), HR and Systemic Vascular Resistance (SVR) all decreased significantly over 10 minutes, the mean right atrial pressure rose slightly. There were no significant difference between the two groups. The maximum decrease in MAP from control was 15.7% in both groups. The maximum decline in CO was 8.1 % in Thiopentone group and 9.6% in Midazolam group. Heart rate decreased 4.8% and 8% respectively. The mean right atrial pressure rose by 33.3% and 42.8% respectively. Reduction in SVR was 18.1% and 16.7% respectively [17].

Choi *et al.* [18] compared the haemodynamic effects of low dose Midazolam and Etomidate in an ED, they observed a 10% decrease in the mean systolic blood pressure (SBP) in patients who received Midazolam and no change in haemodynamics in those who received etomidate. Of all patients who suffered hypotensive episode 33.3% received fluid bolus to achieve reasonable perfusion pressure and none needed vasopressors [18].

These studies indicate that haemodynamic variations are inevitable during induction of anaesthesia therefore the quest to find the induction agent with least haemodynamic effects continues.

4. METHODOLOGY

4.1 Study Design

This was a prospective, randomized, observational and unblinded study.

4.2 Study Population and Site

The sample was drawn from patients scheduled for emergency surgery at DR GEORGE MUKHARI HOSPITAL (DGMH) theatre unit. An informed consent was obtained in 45 patients between the ages of 11 and 65 years of age who fall under the classification of ASA IE – IIIE scheduled for emergency surgery. The following patients were excluded:- Elderly patients above 65 years, those less than 11 years of age and pregnant patients booked for caesarean section.

4.3 Procedure

Each patient in the study group had an intravenous line, was pre-oxygenated and then received etomidate 0.2 – 0.3 mg/kg intravenously until loss of eyelash reflex. This was followed immediately by neuromuscular blockade suxamethonium 1.5 mg/kg or Rocuronium 1.2 mg/kg, tracheal intubation followed 60 seconds later. Intubation was performed by the Researcher and anaesthesia was maintained with Isoflurane/Air/Oxygen (60%).

4.4 Materials and Instruments

Monitors used included:

o   Noninvasive automated blood pressure cuff.

o   Electrocardiogram (ECG)

o   Pulse Oxymetry

o   Capnometer

4.5 Outcome Measures

The demographics of the subjects in this study were noted for age, gender and for ASA classifications. The following vitals were measured: HR, SBP, Diastolic blood pressure (DBP) and MAP. The vitals were measured pre-induction, immediately following intubation, 2, 5 minutes, 5 minutes and 7, 5 minutes post intubation.

4.6 Data Collection

Data collected was recorded on the data sheet, see attached copy of data sheet [Appendix 1].

4.7 Data Analysis

Data generated from the study was subjected to descriptive analyses to reflect the demographics of the patients and to illustrate the ASA rating at the time of induction. Changes in vital signs over-time after induction and post-intubation were calculated and also demonstrated graphically. Changes in the vital signs (HR, SBP, DBP and MAP) were noted as being significant if p-value for any of such changes was ≤ 0.05.

4.8 Reliability and Validity of the Study

The design of the study as a prospective, randomized, observational study provided an opportunity for the Researcher to maintain total control of all data generated and all information required from the study. The Researcher was solely responsible for the selection of patients, administration of etomidate and the periodic measurements of the vital signs. Induction medications and their doses were prepared and administered only by the Researcher. All the equipment used in the study was similar and serviced at appropriate intervals. Data generated from the study were constantly and continuously

reviewed with my supervisor and any case that may have had procedure deviation was excluded from the results.

4.9 Bias

No bias was anticipated for this study, as areas that could have introduced bias such as: patient selection, administration of induction agents and the recording of data were carried out only by the Researcher. This provided safeguards against inter-observer variations and possible inconsistency in data gathering. The Statistician who performed data analysis was not involved with any of the patients or the processes of data collection.

4.10 Ethical Consideration

An informed consent was obtained in all patients participating in the study. Permission to conduct the study was sought from Chief Executive Officer (CEO) and management of DGMH as well as Research Ethics and Publications Committee (REPC) of the University of Limpopo. Confidentiality of data and anonymity of the participants was maintained.

5.   RESULTS

For this series, a total of forty-five (45) patients, consisting of 10 males and 35 females were analysed. Table 3 below shows the demographic profiles as well as the American Society of Anaesthesiology (ASA) rating of the patients at the time of their surgical procedures.

There were predominantly female patients (77.8%) in this study with males making up only 22.2%. Both groups also demonstrated a spread of the ASA ratings but the majority of the patients in each group were rated as ASA IE.

Table 3:   **Demographic profiles and ASA ratings of the patients**

Baseline Features	Males [N = 10]	Females [N = 35]
Age: Range	19 – 45 years	21 – 58 years
Mean (± SD)	34.6 years (± 6.85)	31.9 years (± 9.6)
ASA Rating		
1	6 (60.0%)	25 (71.4%)
2	3 (30.0%)	6 (17.1%)
3	1 (10.0%)	4 (11.4%)

Among the patients who underwent various surgical operations vitals were noted pre-induction and immediately post-intubation. The same vitals were monitored at 2.5 minutes, 5.0 and 7.5 minutes post intubation. Table 4 and Figure 1 below show the heart rate readings at each of the various times of monitoring the vitals.

Table 4 :   **Pre- induction and Post-intubation Heart rate Readings [N=45]**

Heart Rate	Range	Mean	SD	Change from Pre-intubation	P
Pre-induction	66 - 141	97.67	21.0	-	-
0 minute Immediately post intubation	49 - 153	112.33	21.0	14.67	<0.0001∗
2.5 minutes Post intubation	66 - 144	102.78	19.1	5.11	0.0928
5.0 minutes Post intubation	66 - 134	98.49	16.8	0.82	0.7489
7.5 minutes Post intubation	61 - 139	94.64	19.1	- 3.02	0.2363

KEY: SD = Standard Deviation from the mean.   P∗ = Level of Significant difference

The pre-induction heart rate ranged between 66 and 141, with a mean ($\pm$ SD) of 97.67 ($\pm$ 21.0). Immediately post-intubation (0 min), there was a sharp rise in the heart rate to 112 $\pm$ 21.0 (mean, $\pm$ SD) and this was followed by a steady decline in heart rate from 2.5 minutes, right up to 7.5 minutes post-intubation. The changes in the heart rate between the pre- and post-intubation readings was significantly different ($p < 0.001$) immediately (0 minute) following intubation. None of the other readings at 2.5, 5.0 or 7.5 minutes was significantly different from the pre-intubation reading.

Figure 1: Readings of Heart Rate: Pre-induction and post-intubation
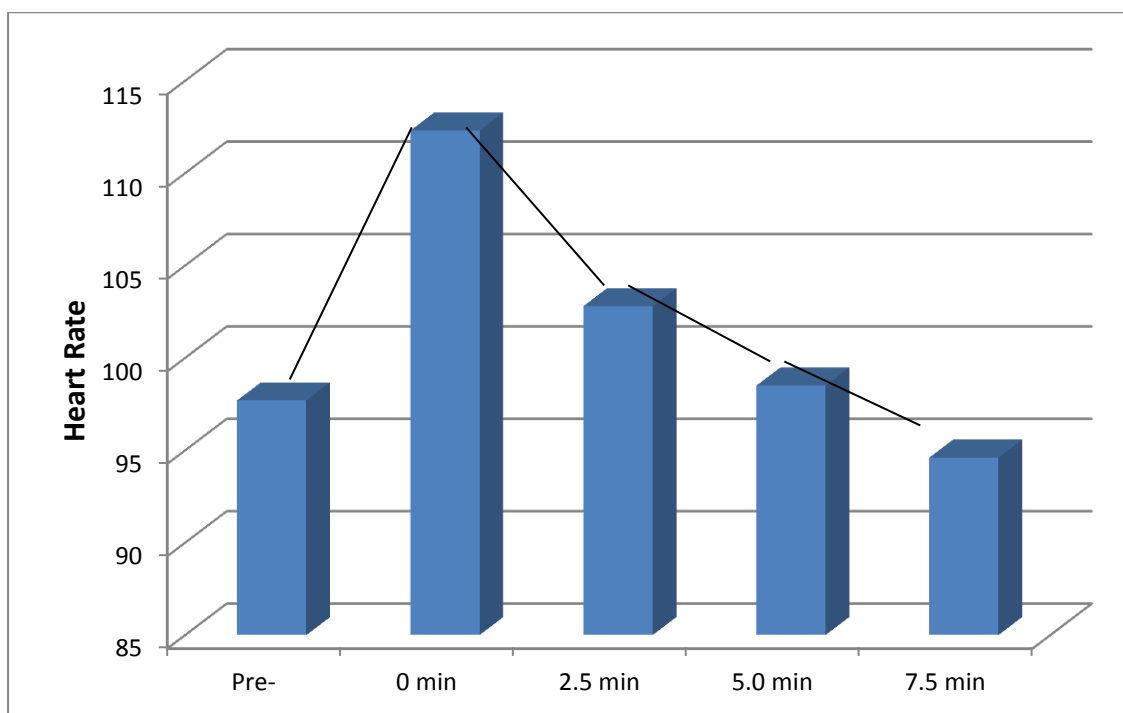
Table 5 and Figure 2 illustrate the readings of systolic, diastolic and mean arterial blood pressure readings, taken at pre-induction and post-intubation intervals. Changes in blood pressure in these patients followed the same pattern which was observed for heart rate. The mean ($\pm$ SD) of the pre-intubation systolic and diastolic blood pressure were 117 mm Hg ($\pm$ 15.1) and 70.7 mm Hg ($\pm$ 13.8) respectively, the blood pressure readings increased sharply immediately (0 minute) post-intubation to 143.4 mm Hg ($\pm$ 20.6) for systolic and

89.5 mm Hg (± 17.4) for diastolic blood pressure. These sharp increases in systolic and

diastolic pressures immediately post-intubation were statistically significant with p <

0.0001

Table 5: **Systolic, Diastolic and Mean arterial blood pressures taken at**
**pre-induction and post-intubation [N=45]**

Blood Pressure	Range	Mean	SD	Change from pre-intubation	P
Systolic:					
Pre-Induction	82 - 144	117.0	15.1	-	-
Post-intubation					
0 min	93 - 185	143.4	20.6	26.4	< 0.0001*
2.5 min	88 - 190	124.5	22.3	7.5	0.0601
5.0 min	63 - 158	109.6	18.5	- 7.4	0.0310*
7.5 min	77 - 146	109.3	16.6	- 7.8	0.0189*
Diastolic:					
Pre-Induction	43 – 98	70.7	13.8	-	-
Post-intubation					
0 min	44 – 127	89.5	17.4	18.8	< 0.0001*
2.5 min	38 – 154	72.6	19.6	1.9	0.5196
5.0 min	26 – 101	63.1	16.4	- 7.6	0.0038*
7.5 min	26 - 89	63.1	13.9	- 7.6	0.0032*
MAP:					
Pre-Induction	22 – 115	85.8	16.1	-	-
Post-intubation					
0 min	71 – 143	108.7	16.5	22.9	< 0.0001*
2.5 min	55 – 160	89.2	19.4	3.4	0.3398
5.0 min	44 – 113	78.1	16.2	- 7.6	0.0097*
7.5 min	42 - 118	79.1	17.1	- 6.6	0.0449*

MAP = Mean Arterial Pressure, SD = Standard Deviation, P* = Level of Significant
Difference

It was noted that systolic blood pressure was statistically reduced below the pre-induction level at 5.0 minutes post-intubation (p = 0.0310) and at 7.5 minutes post-intubation (p = 0.0189). Similarly, diastolic blood pressure, following an initial statistically significant increase, showed marked reduction at 5.0 minutes post-intubation (p = 0.0038) and at 7.5 minutes post-intubation (p = 0.0032) when compared with the pre-induction readings.

The Mean Arterial Pressure (MAP) demonstrated identical changes between pre-induction and post-intubation readings. It increased dramatically from pre-intubation level of 85.8 mm Hg (± 16.1) to 108.7 mm Hg (± 16.5) immediately post-intubation (0 minute), with statistical significant difference reflected as p-value < 0.0001. By 2.5 minutes post-intubation, the MAP had been reduced to a reading of 89.2 mm Hg (± 19.4). However, the MAP readings became significantly reduced after 5.0 (p = 0.0097) and 7.5 minutes (p = 0.0049) when compared with the pre-intubation MAP readings.

Figure 2:   Systolic, Diastolic & MAP blood pressure readings, Pre- and Post-intubation
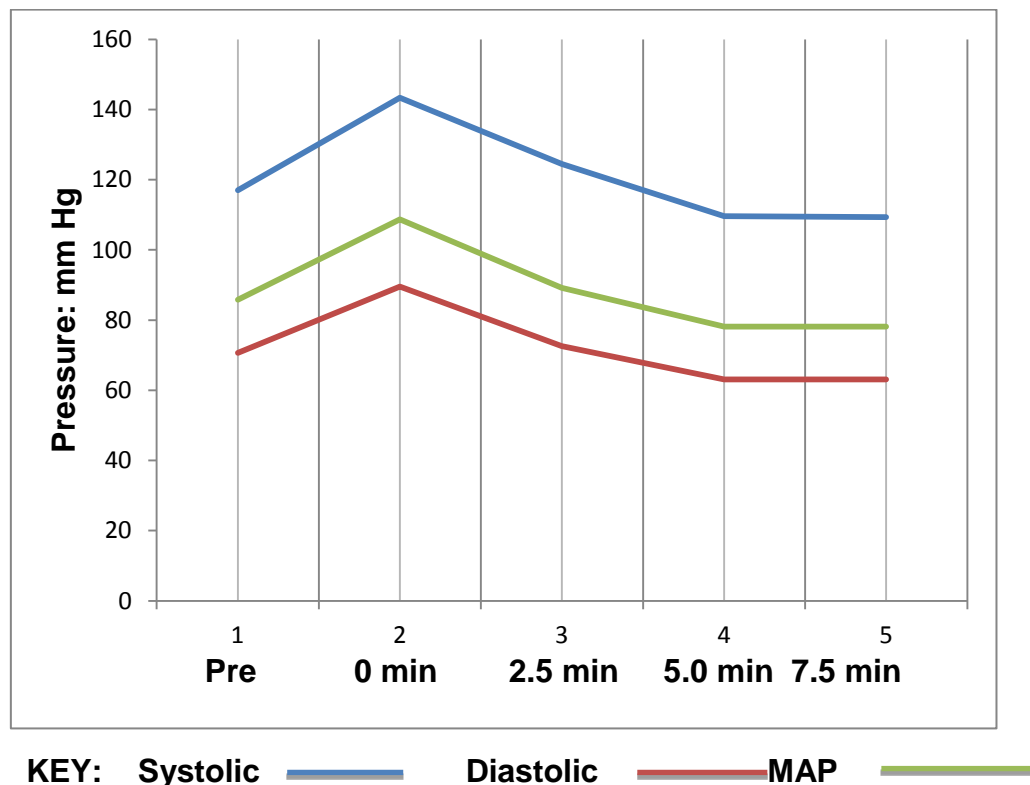
6.   DISCUSSION

The purpose of the study was to determine whether etomidate should be the induction agent of choice for RSI in the ED due to its haemodynamic stability. The findings of the study revealed the following:

6.1   Tachycardia was observed immediately post intubation (i.e. at time 0) compared to pre-induction value (Table 4).   This increase was attributed to laryngoscopy and endotracheal intubation.

A steady decline in HR at 2.5 minutes to 7.5 minutes post intubation was observed. All the values post intubation were greater than the baseline value (pre-intubation).

6.2   Blood pressure changes were similar to those of the heart rate. Systolic blood pressure, DBP and MAP increased sharply immediately post-intubation (Table 5) due to symphathetic stimulation following laryngoscopy and endotracheal intubation. The values showed a decline from 2.5 minutes to 7.5 minutes post-intubation. The blood pressure dropped as the period of anaesthesia progressed; this was due to a variety of factors, including direct effects of anaesthesia, inhibition of the sympathetic nervous system and loss of baroreceptor reflex control of arterial pressure.

Even though the drop in blood pressure was statistically significant at 5 minutes and 7.5 minutes, it was clinically not significant as it required no special management (vasopressors and/ or intravenous fluids), and that the drop in SBP, DBP and MAP seen from the fifth minutes was less than 20% of the pre-intubation values.

The following are some of the studies evaluating the use of etomidate in the ED setting. Woodard and coworkers performed a retrospective chart review to assess the haemodynamic effects of etomidate in 56 trauma patients who were given (0.2-0.4mg/kg) for RSI in the ED, of these, 51 patients received succinylcholine (1-2mg/kg) and 5 received vecuronium (0.1mg-0,3mg/kg). They found that the mean SBP change of 13mmHg was not clinically significant and that no patient required intervention because of haemodynamic problems, of the 12 patients who were hypotensive before intubation, only one had a further decrease in BP after intubation. Pre and post intubation pulse rates did not change. In summary etomidate in trauma RSI did not cause haemodynamic instability, even in patients who were hypotensive before induction. [19].

In a prospective observational study, Smith and Coworkers evaluated the use of etomidate in 34 patients undergoing RSI in ED. All patients received defasciculating dose of vecuronium (0.01mg/kg) followed by etomidate (0.3mg/kg) and succinylcholine (1.5-2.0mg/kg). Blood pressure, HR and oxygen saturation were measured every 2min before, during and after induction for 6min. Mean changes in systolic and diastolic blood pressure were 1 ± 39mmHg and 0 ± 28mmHg, respectively. For patients with an initial MAP of ≤80mmHg, MAP dropped by an average of 6%. No patient with an initial MAP of >80mmHg dropped to below 80mmHg. It was concluded that etomidate is a safe and effective agent for use in a range of patients undergoing RSI in the ED. [1].

Swanson and colleagues retrospectively reviewed records from 79 patients over the age of 10 who underwent intubation in an aeromedical setting. Overall 53 patients

(67%) received etomidate (0.2-0.4mg/kg) and 42 of these (79%) also received succinylcholine. The intubation success rate was 96% for the 53 patients who received etomidate. Two cricothyrotomies were performed in patients who could not be intubated. Of the 46 patients with haemodynamic data, mean SBP was 139.11±31.21mmHg before and 137.85 ±32mmHg after RSI. Average heart rates were 101.59±23.95 beat/min before and 97,76±23.45 beats/min after RSI. The authors concluded that etomidate is safe and effective for RSI in the aeromedical setting [20].

7.   CONCLUSION

In conclusion, the studys' results show that etomidate is an effective anaesthetic induction agent for use in RSI. The haemodynamic stability seen with the use of etomidate has been demonstrated in this study, in our setting we see a lot of volume depleted patients as most of them come from far and depend on public transport or ambulance services which might take long to arrive at the hospital. The use of etomidate is gaining popularity worldwide as an induction agent [1, 6 and 8].

8.   RECOMMENDATIONS

- Etomidate be used in an ED as most patients are haemodynamically unstable.
- Further research is needed to determine the clinical outcome of a single dose of etomidate in causing adrenal suppression.

9.   LIMITATIONS OF THE STUDY

This study was limited by the following:

o   Small sample size (n = 45).

o Etomidate was not compared with other induction agents in a blinded fashion.

o Patients were manipulated (i.e. Endotracheal tube was inserted and inhalational anaesthetic started post-intubation for maintenance which might affect the outcome).

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11.   APPENDIX

<u>APPENDIX 1</u>

DATA COLLECTION FORM

SUBJECT NO:--------------------

AGE:-------------------------------------   **Gender: M------   F-------**

ASA-------------------------------------

TYPE OF SURGERY--------------------------------

	Pre Induction	**Post Induction**			
VITALS		**Immediately post intubation** (0 min)	**2,5 min**	**5 min**	**7,5 min later**
Heart					
Systolic BP					
Diastolic BP					
MAP					
Saturation					

UNIVERSITY OF LIMPOPO (Medunsa Campus) CONSENT FORM

Statement concerning participant in a Research Project

NAME OF PROJECT: SHOULD ETOMIDATE BE THE INDUCTION AGENT OF CHOICE IN THE EMERGENCY DEPARTMENT?

I understood the aims and objections of the proposed study and was provided the opportunity to ask questions and given adequate time to rethink the issue. The aim and objectives of the study are sufficiently clear to me. I have not been pressurized to participate in any way.

I understand that participation in this study is completely voluntary and that I may withdraw from it at any time and without supplying reasons. This will have no influence on the regular treatment that holds for my condition neither will it influence the care that I receive from my regular doctor.

I know that this study has been approved by the Research, Ethics and Publications Committee of Faculty of Medicine, University of Limpopo (Medunsa Campaus) / Dr. George Mukhari Hospital. I am fully aware that the results of this study will be used for scientific purposes and may be published. I agree to this, provided my privacy is guaranteed.

I hereby give consent to participate in this study.

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Name of patient/volunteer   Signature of patient or guardian

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Place   Date   Witness