

RELATIONSHIP BETWEEN LACTATE VALUES AND MORTALITY IN PATIENTS WITH HAEMORRHAGIC SHOCK IN AN EMERGENCY UNIT

by

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Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

Declaration

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I declare that a **Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit** is my own work. All sources that have been used or quoted have been indicated and acknowledged by means of complete references and that this work has not been submitted for any other degree at any other institution.

Eleonora Natali Zarrabi

Date

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

Dedication

To all the health care professionals working in emergency units, who are able to respond to situations when others cannot. To those health care professionals doing their best for their patients and always striving to be better.

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Acknowledgements

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Abstract

Introduction: Trauma is the leading cause of death in patients between the ages of 1-44 years in South Africa. Taking these statistics into consideration it is essential to better resuscitation strategies in order to improve outcome of trauma patients. Compensated and uncompensated haemorrhagic shock is frequently under diagnosed in trauma patients, which has a definitive effect on mortality. Concerns about inadequate monitoring of patients through the use of only physiological end points are raised.

Methods: A comprehensive literature review was conducted on resuscitation strategies for patients presenting with haemorrhagic shock. Physiological and metabolic end points of resuscitation were identified to guide resuscitation strategies. A quantitative, retrospective, non-experimental, descriptive, correlational and cross-sectional research design was chosen for this study. Data was collected by using biophysical measures, namely clinical audit checklists.

Results: Lactate was identified as a good indicator to predicting mortality in patients presenting with shock caused by haemorrhage. Special consideration to patients' age and physiological status should be made during resuscitation. The consequence of delayed resuscitation in haemorrhagic shock patients is associated with an increase in mortality that can be prevented. It is found that serum blood lactate levels taken over time are good predictors of patient survival rates. Patients presenting with a raised serum blood lactate level for more than 24-hours has an increase in mortality rate.

Conclusion: Emergency nurse practitioners are responsible for the monitoring of patients admitted to the emergency unit with haemorrhagic shock. The use of serum lactate levels during the first 24-hours of the resuscitation of patients with haemorrhage can assist with the implementation of strategies to reverse the effect haemorrhagic shock on cellular level in these patients.

Key words: Emergency nurse practitioner, emergency unit, end points, haemorrhagic shock, mortality rate, level I hospital, serum blood lactate levels, triage and trauma.

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List of abbreviations

ATLS	Advanced Trauma Life Support
P1	Priority one
P2	Priority two
P3	Priority three
P4	Priority four

*For the purpose of **anonymity**, the hospital in which the study was conducted will be referred to as **the hospital**, in both text and referencing.*

1 ORIENTATION TO THE STUDY

1.1 INTRODUCTION

Trauma is one of the major challenges in emergency nursing practice today (Gonzales 2008: 249; McQuillan, Makic & Whalen 2009: 2). In the view of both Kane, MacCallum and Friedrich (2007: 61) and Garner, Watts, Parry, Bird, Cooper and Kirkman (2010: 1131), trauma is responsible for more deaths than any other disease or illness in the first four decades of life. In addition, Nicol and Steyn (2004: 1) state that, in the Republic of South Africa (RSA), injury as the result of trauma causes mortality in an estimated 65 000 to 80 000 people per year. The most common cause of death among patients who have sustained injuries caused by trauma is the loss of blood, plasma or extracellular fluid which, in turn, leads to a state of shock which is known as hypovolaemic or haemorrhagic shock (Dorland 2001: 421; Nicol & Steyn 2004: 49).

Blood loss as a result of trauma may be fatal, especially in cases in which the management of haemorrhage is postponed and, consequently, the degree of shock worsens. The management of haemorrhagic shock is referred to as resuscitation and includes both the management of bleeding and the administration of fluids, depending on the severity of the shock (Morton, Fontaine, Hudak & Gallo 2005: 1256). The extent of the shock sustained during trauma may lead to end-organ failure and subsequent death as a result of a decrease in tissue oxygenation and perfusion (Cerovic, Golubovic, Spec-Marn, Kremzar & Vidmar 2003: 1300; Collins 2000: [3]).

One way in which to assess the degree of shock and the subsequent diminished tissue oxygenation on cellular level would be to monitor the end points of

resuscitation. These end points of resuscitation include metabolic markers – also referred to as metabolic end points – and haemodynamic markers – also referred to as physiological end points (Englehart & Shreiber 2006: 569). These markers will reveal whether a patient has not been optimally resuscitated during haemorrhagic shock and the tissue has remained hypoxic.

Metabolic marker that has proven to be extremely accurate in estimating damage to cells (Husain, Martin, Mullenix, Steele & Elliot 2003: 486). Lactate is produced by the body during high stress situations such as strenuous exercise or shock states. In general, lactate is merely a by-product of energy production and, in moderate quantities, is not harmful to the body. However, when the production of lactate exceeds the lactate breakdown rate of the body, then the serum lactate levels start rising and lactic acidosis ensues.

Lactic acid results from a severe build up of serum lactate as a result of tissue hypoperfusion which may, in turn, be caused by either a lack of oxygen or a diminished blood supply to cells in the body (Luft 2001: S15). One of the most common causes of the hypoperfusion of cells is blood loss due to haemorrhage. Lactic acidosis due to the hypoperfusion of cells caused by haemorrhage is classified as Type A acidosis (Luft 2001: S16). Lactic acidosis occurs at serum lactate levels of > 5 mmol/L.

Recent studies have indicated a correlation between prolonged raised levels of serum lactate and an increase in mortality rates. A raised serum lactate level for a period of longer than 24 hours correlates strongly with mortality, while a failure to decrease lactate is normally associated with 100% mortality (Husain *et al.* 2003: 486; McNelis, Marini, Jurkiewicz, Szomstein, Ritter & Nathan 2001: 481). The aim of this study is to determine the correlation between the 24-hour serum lactate levels and the 48-hour mortality rate of patients presenting with haemorrhagic shock in the emergency unit of a Level I public hospital in Gauteng province, Republic of South Africa.

1.2 BACKGROUND TO THE PROBLEM

In the emergency unit of a Level I public hospital in Gauteng, further on referred to as the hospital, as in many other emergency units countrywide and, indeed, worldwide, emergency nurse practitioners monitor vital data such as the heart rate, blood pressure and urine output of patients presenting with haemorrhagic shock. They then report any abnormalities to the attending medical doctor. These haemodynamic markers may be used as parameters in order to determine the success of the resuscitation efforts and are referred to as physiological end points (Englehart & Schreiber 2006:569).

However, according to both Porter and Ivatury (1998) and Barbee, Reynolds and Ward (2010: 113), should haemorrhagic shock be managed conventionally using physiological end points only to guide the resuscitation efforts, a significant number of patients treated in an emergency unit may still suffer from inadequate tissue perfusion, which may, in turn, lead to death (Shoemaker & Beez 2010: 5). In addition, in the opinion of Shirley (2007: 192), once these physiological end points have returned to normal, 50 to 85% of patients still manifest underlying shock which, if prolonged, may cause death.

Prevailing concerns about the inadequate monitoring of patients through the use of physiological end points only are supported by Bakker (1999: [2]), Barbee *et al.* (2010: 113) and Shoemaker and Beez (2010: 5). These writers are all of the opinion that, while initial physiological end points may indicate normal or minor abnormalities, nevertheless, the tissue perfusion and hypoxia may be severe. Englehart and Schreiber (2006: 573), Husain *et al.* (2003: 489), Porter and Ivatury (1998) and Schulman (2002: 32cc8) all suggest that the use of physiological end points is inadequate in order to make an accurate assessment of the tissue hypoxia in patients with haemorrhagic shock and that one or more metabolic end point should also be considered. Based on these facts there is now consensus that the monitoring of physiological end points alone does not result in an accurate prediction of the degree of haemorrhagic shock and that, in fact, this monitoring of

physiological end points alone may be regarded as a dangerous clinical practice. It is, thus, essential that the metabolic end points of resuscitation also be considered and incorporated into any monitoring of the success of resuscitation.

The metabolic end points of resuscitation include base deficit, bicarbonate levels and serum lactate levels. These metabolic end points all effect the serum pH (Bilkovski, Rivers & Horst 2004: 533; Tisherman, Barie, Bokhari, Bonadies, Daley, Diebel, *et al.* 2004: 898). Should a patient presenting with haemorrhagic shock not be optimally resuscitated then the tissue perfusion will remain inadequate. According to Englehart and Schreiber (2006: 569), inadequate tissue perfusion causes tissue hypoxia and may, in fact, result in significant metabolic acidosis. Metabolic acidosis, which influences the serum pH, may, in turn, cause severe cellular and organ dysfunction and this may lead to an increase in both morbidity and mortality.

Both the base deficit and bicarbonate levels may be used as indicators of tissue perfusion and tissue hypoxia, with base deficit being used to predict the severity of injury and mortality (Englehart & Schreiber 2006: 570-1; Tisherman *et al.* 2004: 906). A base is able to bind with an acid such as hydrogen ions in the body (Marieb 2007: 41). An increase in the base deficit will, thus, result in an increase in the level of free hydrogen ions and present as a decrease in both the serum pH and metabolic acidosis. As the metabolic acidosis increases, the patient's base deficit will increase and the serum pH will decrease (Englehart & Schreiber 2006: 570). In addition, bicarbonate levels serve as an indicator of worsening metabolic acidosis which, in turn, may present as a decrease in bicarbonate levels, a worsening of the metabolic acidosis and a subsequent decrease in the serum pH (Englehart & Schreiber 2006: 571).

Lactate causes metabolic acidosis as a result of the release of hydrogen ions which, in turn, is brought about by the activation of anaerobic metabolism (Englehart & Schreiber 2006: 569). Anaerobic metabolism is caused by tissue hypoxia that is left untreated. This causes the tissue hypoxia to become more severe, thus causing ongoing metabolic acidosis. The combination of an increased serum lactate level and

the presence of metabolic acidosis is associated with a higher mortality rate (Bakker 1999: [6]).

According to an article written by Porter and Ivatury (1998), at least one of the metabolic end points, namely, base deficit, bicarbonate levels, serum lactate or serum pH, should be used as an indicator with which to guide the resuscitation of trauma patients, by aiming to normalise at least one of these metabolic end points within a period of 24 hours. It is further suggested by Husain *et al.* (2003: 489) that serum lactate values are a more effective indicator of tissue perfusion and tissue hypoxia than other metabolic end points, for example base deficit. Serum lactate values that have obtained over a period of time have proven to be predictive of mortality. Englehart and Schreiber (2006: 570) indicate that, if the serum lactate levels of patients with haemorrhagic shock are not normalised within 24 hours, then the mortality rate for these patients is generally 25%. In addition, if the serum lactate levels remain elevated for more than 48 hours then the mortality rate of such patients increases 80 to 86%. Accordingly, a serum lactate value that has not normalised within 24 hours is indicative that the patient is in an ongoing shock state and there is a chance of an increasing mortality rate (Schulman 2002: 32cc4).

Despite the fact that it is possible to measure serum lactate and other metabolic end points in an emergency unit, the value of monitoring the serum lactate level remains unclear. In addition, the correlation between the serum lactate level and the mortality of patients presenting with an increased serum lactate level over a period of 24 hours following haemorrhagic shock in an emergency unit also remains unclear.

1.3 PROBLEM STATEMENT

Research has confirmed that the conventional method, namely, focusing on the physiological end points in order to evaluate the resuscitation efforts in respect of patients presenting with haemorrhagic shock, may be inadequate (Bakker 1999: [6]; Porter & Ivatury 1998; Shafi & Kauder 2004: 39; Schulman 2002: 32cc2). Both the

physiological and metabolic end points of resuscitation should be taken into account by emergency nurse practitioners during the evaluation of the resuscitation efforts of all adult patients presenting with shock due to haemorrhage.

According to the findings in studies conducted by Bilkovski *et al.* (2004: 534), Englehart and Schreiber (2006: 573), Porter and Ivatury (1998) and Schulman (2002: 32cc8), the practice of not taking the metabolic end points of resuscitation into consideration during the evaluation of resuscitation efforts may lead to ineffective resuscitation with a concomitant increase in morbidity and mortality rates. It is essential that patients presenting with haemorrhage in an emergency unit be adequately monitored by evaluating both the physiological and the metabolic end points. This, in turn, would assist emergency nurse practitioners to manage these patients timeously and to prevent decreased tissue perfusion, tissue hypoxia, deterioration and possible death (Shoemaker & Beez 2010: 6). The literature further suggests that serum lactate levels represent one of the most important parameters that should be used to evaluate resuscitation and these levels may be regarded as one of the most accurate predictors of mortality (Cerovic *et al.* 2003: 1003; Husain *et al.* 2003: 489; McNelis *et al.* 2001: 484).

Emergency nurse practitioners form part of a multidisciplinary team which is responsible for the management of patients and which is accountable for the monitoring of patients who have been admitted with haemorrhagic shock. It is, thus, of the utmost importance that these emergency nurse practitioners be acquainted with the effect of serum lactate levels as an indication of the perfusion status of a patient, and the interpretation of these levels. It is unclear what the outcomes of patients presenting with haemorrhagic shock are in the emergency unit of the hospital. One method that may be used to determine the mortality of such patients is to monitor the 24-hour serum lactate of patients admitted with haemorrhagic shock and correlate this level with the 48-hour mortality of these patients.

1.4 RESEARCH QUESTION

In light of the background and the problem statement given above, the following research question was formulated:

- What is the correlation between the 24-hour serum lactate levels and the 48-hour mortality rate of adult patients presenting with haemorrhagic shock in an emergency unit?

1.5 AIM AND OBJECTIVES

The overall aim of this study is to determine the correlation between the 24-hour serum lactate levels and the 48-hour mortality rate of patients presenting with haemorrhagic shock in the emergency unit of the hospital.

In order to achieve this aim, the objectives of the research will be to

- audit **the serum lactate levels as indicted in the patient files on admission as well as during a 4 to 8 hour, 10 to 14 hour and 22 to 26 hour timeframe following** haemorrhage caused by trauma
- document the 48-hour mortality rate in patients presenting with haemorrhage caused by trauma
- determine the relationship between the serum lactate levels and the 48-hour mortality rate in patients presenting with haemorrhage caused by trauma
- make recommendations for future resuscitation strategies for patients presenting with haemorrhagic shock in order to improve the outcomes of such patients

1.6 SIGNIFICANCE AND CONTRIBUTION

Haemorrhage is one of the biggest killers in South Africa (Kane *et al.* 2007: 61) and, thus, the management of haemorrhage is crucial and should be efficient and optimal

in order to decrease the morbidity and mortality of patients suffering from haemorrhage.

Emergency nurse practitioners are responsible for the monitoring of patients admitted to an emergency unit with haemorrhagic shock and they may find the use of serum lactate levels beneficial in the prediction of the severity of tissue hypoperfusion during the management of haemorrhagic shock. The use of serum lactate levels during the first 24 hours of the resuscitation of patients with haemorrhage may assist with the implementation of strategies to reverse the effect of haemorrhagic shock on tissue level in these patients. If the value of monitoring the serum lactate level at specific time intervals were investigated and the impact of these serum lactate levels on the 48-hour mortality of patients ascertained, this would highlight the importance of such monitoring in emergency units.

Polit and Beck (2008: 439) maintain that the National Institute for Nursing Research emphasises the need for physiological nursing research. Physiological data may be collected in a clinical setup through specialised technical equipment. The outcome of this study may be used to guide clinical practice in terms of the value of the monitoring of serum lactate levels. Accordingly, the findings may be used to contribute to the compilation of protocols and/or guidelines for improving clinical practice. These, in turn, may be used to guide nursing education, specifically in the context of the emergency care setting.

The use of serum lactate levels may also aid in the prediction of mortality in patients presenting with haemorrhagic shock and, thus, aid clinical audits within the emergency unit and promote changes to the resuscitation strategies in respect of patients diagnosed with haemorrhagic shock. Clinical audits are the cornerstone of evidence-based practice and it is possible that further research could follow based on the findings of this study.

1.7 FRAME OF REFERENCE

The frame of reference or framework of a study aims to provide a logical structure or guide in terms of which to develop the study (LoBiondo-Wood & Haber 2006: 114). Both the philosophical paradigm and the research design contribute to this structure.

The frame of reference in this study is described in terms of both the paradigm and the assumptions of the research. This frame of reference also includes the role of the researcher, the setting in which the study was conducted, the paradigm, the assumptions and the conceptual definitions.

1.7.1 Role of the researcher

The role of the researcher may be described as the influences that the researcher brings to a study (Struwig & Stead 2001: 227). According to Terre Blanche and Durrheim (2004: 49), these influences should be considered before the data collection takes place, since this is the time at which these influences would have most effect. The researcher should decide whether to be involved in the data collection process and also whether the context of the research should be manipulated. These decisions are usually determined by the paradigm that the researcher chooses to adopt (Terre Blanche & Durrheim 2004: 49).

In this study the researcher has been working as a nurse practitioner in emergency care for the past six years. In addition, she is involved in the management of patients presenting with haemorrhagic shock in both the hospital and the pre-hospital emergency care environment. As a nurse practitioner, she works in the emergency unit as well as in an air ambulance transport service which transports patients globally. Her work experience supports her knowledge of and experience in the field of emergency nursing.

With reference to the collection of data in this study, the researcher was involved in reviewing files, selecting the sample and documenting the data on the clinical audit checklist. The data collection was conducted in a structured way. In order to ensure that the data was not manipulated, the researcher and her supervisor agreed that the supervisor would check so as to ensure that data had not been manipulated.

1.7.2 Study setting

The study setting refers to the location in which a study is conducted (Burns & Grove 2009: 722). The milieu in which this research took place was the emergency unit of a Level I public hospital in the province of Gauteng, RSA (see Figure 1.1).



Figure 1.1: Map of the Republic of South Africa and its provinces (adapted from the South African Government Information [n.d.]

Based on the classification of health establishments in the National Health Act, Act No. 61 of 2003, (National Department of Health 2006: 10) it was decided on the following preliminary definition of a Level I public hospital, namely, that such an hospital includes a facility that provides inpatient services as well as specialist and sub-specialist care within the public sector. The emergency unit involved in this study is located in a Level I public hospital in Gauteng Province, RSA.

The hospital has 832 beds of which 92 beds are reserved for critical care and high care patients. Of these ninety-two critical care beds, forty-four are intended for adult critical care patients and 12 beds for high care adult patients. There are 29 neonatal critical care beds and seven paediatric critical care beds available in the hospital (The Hospital 2010; Van Niekerk 2007).

The emergency unit is a 40-bed division which includes seven resuscitation beds – six beds for adult patients and one paediatric resuscitation bed. As a result of the shortage of beds available for critical care and high care patients in the hospital, two additional beds for critical care patients and four for high care patients have been provided in the emergency unit of the hospital (Van Eeden 2010).

The majority of patients admitted to the emergency unit comprise either critically ill or injured patients of all ages. The condition of the patients entering the emergency unit varies in terms of the degree of urgency. Patients are classified according to severity of illness or injury by means of the Cape Triage System which classifies such patients into priority groups, namely, Priority 1 (P1), Priority 2 (P2) and Priority 3 (P3) – (see Section 1.7.5.6). With the exception of orthopaedic emergencies, Priority 3 patients are referred to the district hospital or local clinic. Injuries and illnesses typically treated in the emergency unit include life-threatening and/or life-altering conditions such as airway obstruction, chest injuries, head injuries, hip and pelvis fractures and myocardial infarction.

The staff working in the emergency unit consists of approximately 48 professional nurses of whom eight have an additional, post-basic qualification in emergency

nursing. In addition, a further eight of the professional nurses are currently enrolled for the post-basic programme in emergency nursing. Additional nursing staff includes 24 staff nurses and 10 auxiliary nurses. All nursing staff members working in the emergency unit work 12-hour shifts. These 12-hour shifts include both day and night shifts. The optimal workforce per shift consists of 24 nursing staff – this includes 12 professional nurses and 12 enrolled/auxiliary nurses. The general shortage of professional nurses and auxiliary/staff nurses in the emergency unit has meant the use of permanent nursing staff (working overtime) and agency nurses in order to optimise the staff establishment per shift (Van Eeden 2010).

Support personnel working in the emergency unit include admission clerks, cleaners and porters. There are also two or three medical doctors in the emergency unit at all times. Various specialists, such as neurosurgeons, paediatricians, radiologists and intensivists, are available 24 hours a day on a consultation basis.

1.7.3 Paradigm

There is usually more than one way in which it is possible to make sense of things. However, underlying differences in interpretation are paradigms. Paradigms refer to the fundamental models or frames of reference one may use in order to organise one's observations and reasoning (Babbie 2005: 486) in order to increase one's understanding. Polit and Beck (2010: 14) define a paradigm as a view of the real world and as a perception of the intricacies of this world. Quantitative research is conducted on the basis of a positivistic paradigm which is, in turn, characterised by rationality and science. The objective of research which is conducted on the basis of the positivistic paradigm is to explain the causes of natural phenomena (Polit & Beck 2010: 15; Sale, Lohfeld & Brazil 2002: 44). Science is characterised by empirical research in terms of which the phenomena may be reduced to empirical indicators which represent the truth (Sale *et al.* 2002: 44).

A positivistic paradigm was used for this research with the aim of explaining the correlation between 48-hour mortality in patients presenting with haemorrhagic

shock and raised serum lactate levels over a 24-hour period. A clinical audit was constructed using a quantitative design. The audit made use of specific indicators in order to measure and to understand the correlation between 48-hour mortality and serum lactate levels. This implies that the underlying phenomenon was studied objectively. As suggested by Polit and Beck (2004: 15), the methodology in this study was orderly and a disciplined process was used to come to a conclusion. (See Section 3.4. and Annexure B.)

1.7.4 Assumptions

Assumptions refer to statements or principles that are considered to be true but without any scientific backup (Polit & Beck 2004: 748). Assumptions have their foundation in universally accepted truths, theories, earlier studies, and the practical environment (Burns & Grove 2005: 39). Assumption is formed that nature exists autonomously from a human perspective (Polit & Beck 2010: 14).

The researcher in this study opted to make use of quantitative research in order to address the research question. The assumptions are embedded in the positivist paradigm (Polit & Beck 2004: 15). Assumptions in the positivistic paradigm are not influenced by the researcher's point of view, and a high degree of objectivity in respect of the data is essential. The outcomes obtained from the quantitative data are often numeric and quantifiable with the results being obtained by means of a deductive process which is, in turn, conducted on the basis of a pre-specified design (Polit & Beck 2010: 16).

The assumptions for this study were described by both Polit and Beck (2010: 14) and Sale *et al.* (2002: 44-5) and include the following:

- **An ontological assumption:** There is only one truth and this one truth is an objective reality that exists independent of human perspective
- **An epistemological assumption:** Both the investigator and the investigated are independent entities and, thus, do not influence one another.

- **Methodological assumptions:** Based on the best ways in which to obtain the evidence, namely,
 - a deductive process is followed as the researcher develops predictions from the general principles found in the literature, namely, that both physiological and metabolic end points should guide the emergency nurse practitioner when managing a patient with haemorrhagic shock (See Section 2.5)
 - the emphasis is on discrete, specific concepts as delineated in the clinical audit checklists, a structured instrument, and may be used to collect data which is generalisable to similar settings and populations (see Annexure B)
 - the focus is on the objective and quantifiable data which is obtained from the clinical audit checklists (see Chapter 4)
 - the research design (quantitative, retrospective, non-experimental, descriptive, and cross-sectional) was pre-specified and fixed during the development of the proposal (see Table 1.1)
 - the information collected comprises empirical data and, thus, the data exists within an objective reality and is not influenced by either personal perceptions or beliefs (see Annexure B). The quantitative data were collected by means of a structured instrument, namely, a clinical audit checklist. This, in turn, renders the data generalisable to similar settings and populations.

1.7.5 Conceptual definitions

For the purposes of simplicity and consistency, key concepts such as emergency nurse practitioner, emergency unit, haemorrhagic shock, triage, trauma and mortality rate, as used during the study, will be defined below.

1.7.5.1 *Emergency nurse practitioner*

A nurse may be described as “a person trained to care for the sick, somebody caring for patients, somebody trained to look after ill and injured people, especially somebody who works in a hospital or clinic, administering the care and treatment that a doctor prescribes” (*Encarta 2003; Oxford English Dictionary 2005*).

On the other hand, a practitioner may be defined as “somebody who practises a particular profession, especially medicine” (*Encarta* 2003) and, according to the *Oxford English Dictionary* (2008), “a person engaged in an art, discipline, or profession, especially medicine”.

Accordingly, an emergency nurse practitioner may be defined as a nurse practitioner who delivers skilled nursing care for patients during the critical phase of their illnesses or injuries in an emergency unit. These nurses are experienced in the care of patients whose diagnoses are often unknown (*MercSource-Health Information. Pure and Simple. Online Dictionary* 2009).

In this study, the term emergency nurse practitioner includes all professional nurses, as well as all auxiliary and staff nurse practitioners, who are registered as nurses with the South African Nursing Council (SANC) and who are responsible for caring for patients admitted to the emergency unit of the hospital.

1.7.5.2 *Emergency unit*

The *Collins English Dictionary* (2006: 511) defines an emergency as “an unforeseen or sudden occurrence, especially of danger demanding immediate action”. The *MercSource-Health Information. Pure and Simple. Online Dictionary* (2009), further states that an emergency is “a sudden occurrence, often dangerous, such as an accident or an urgent or pressing need”.

For the purpose of this study the term “emergency unit” refers to a unit situated in the hospital. Critically ill or injured patients of all ages are admitted, managed, stabilised and referred to and by the emergency unit of the hospital. These patients are treated by appropriate specialists and then transferred to the appropriate critical care units (CCUs) or general wards in the hospital.

1.7.5.3 Haemorrhagic shock

Haemorrhage is defined by *Dorland's Pocket Medical Dictionary* (2001) as bleeding or, more specifically, blood that breaks away from a blood vessel.

Shock is defined by both the Trauma Nursing Core Course (TNCC) Provider Manual (Hoyt and Selfridge-Thomas 2007: 721) as a syndrome resulting in an abnormality of the circulatory system that results in inadequate tissue perfusion and inadequate oxygen and nutrient supply to tissue, thus causing inadequate metabolic needs supply of the cells. Shock may be classified according to its pathophysiology into cardiogenic, obstructive, distributive and hypovolaemic shock (Hoyt & Selfridge-Thomas 2007: 721). Hypovolaemic shock is the most common cause of shock among trauma patients (American College of Surgeons 2005: 73; Hoyt & Selfridge-Thomas 2007: 727).

Hypovolaemic shock results from a loss of circulating intravascular volume, which is caused by blood escaping from a blood vessel – also referred to as haemorrhage. Hypovolaemic shock may be due to an absolute or relative fluid loss (Urden *et al.* 2006: 1012).

For the purpose of this research the term “haemorrhagic shock” will refer to hypovolaemic shock caused by bleeding or haemorrhaging, where the aetiology is based on a loss of blood following trauma.

1.7.5.4 Mortality rate

Mortality is defined by the *Merriam-Webster Online Dictionary* (2007) as “the quality or state of being dead”.

Mortality rate, as it is referred to in research, is “the death rate, which reflects the number of deaths per unit of population in any specific region, age group, disease, or other classification, usually expressed as deaths per 1 000, 10 000, or 100 000”.

The researcher's use of the term "mortality rate" will include all those patients who have died as a result of shock caused by haemorrhage within 48 hours after their admission to the emergency unit.

1.7.5.5 Trauma

Trauma is derived from the Greek word that means "wound". The *Stedman's Medical Dictionary* (2005) defines trauma as "an injury, physical or mental". Trauma may also refer to an injury that occurs when some form of energy is transferred from the environment to a human host. Trauma arise when injury occur to living tissue, causing a wound (*Merriam-Webster Online Dictionary* 2007).

The term "trauma", as it is used in this research, describes any incident as a result of which the patient enters the emergency unit and which causes haemorrhage and haemorrhagic shock. These incidents include motor vehicle accidents, train accidents, motorbike accidents, bicycle accidents, pedestrian–vehicle accidents, stab wounds, gunshot wounds, assaults and falls.

1.7.5.6 Triage

The *Merriam-Webster Online Dictionary* (2007) defines triage as the: "the sorting of patients according to the urgency of their need for care". In the emergency unit in a hospital all patients entering the unit are triaged and sorted according to priorities with a Priority 1 (P1) patient requiring immediate management, followed by Priority 2 (P2) and Priority 3 (P3). Priority 4 (P4) patients are regarded as unsalvageable or dead on arrival.

The definitions, as applied in the emergency unit of the hospital, are derived from the Cape Triage System which was developed by the Cape Triage Group of the Western Cape. This system has been in use in the Western Cape since January 2006, and has since been introduced to the rest of South Africa, as well as to the emergency unit of the hospital which was the focus of this study (Gottschalk, Wood, DeVries, Wallis &

Bruijns. 2006: 149). The definitions of the different priorities as used in this research are as follows:

- ⇒ **Priority 1 (P1)**: Patients admitted with life-threatening injuries or medical patients who are so severely injured or critically ill that they will die should they not receive immediate medical interventions.
- ⇒ **Priority 2 (P2)**: Patients admitted with potential life-threatening injuries or medical emergencies that are not as serious as a P1. Although their condition is stable for the moment, these patients require watching by trained personnel and frequent re-triaging as the possibility of rapid deterioration does exist.
- ⇒ **Priority 3 (P3)**: These are patients referred to as the “walking wounded” who do not require immediate management. These patients have minor injuries or medical conditions and following management will usually be discharged from the hospital.
- ⇒ **Priority 4 (P4)**: These patients are either dead on arrival or unsalvageable when they arrive at the emergency unit.

1.8 RESEARCH METHOD

Polit and Beck (2006: 504) define a research method as the “steps, procedures and strategies for gathering and analysing data in a research investigation”. The research method consists of both the research design and the methodology.

Polit and Beck (2004: 48–53) describe a structured approach to constructing a quantitative research design. This approach consists of five phases with specific steps. The approach commences with asking a question and ends with answering that question (Polit & Beck 2004: 48). Table 1.1 provides a summary of the phases followed during the development of the proposal, the conducting of the study and the writing of the report in a dissertation format.

Table 1.1: Summary of the phases followed in constructing the research method

Phase 1: Thinking <i>Conceptualise an idea through reading, creative thinking and ideas from colleagues</i>		
Steps	Criteria	Application
1. Identify a problem	Includes the development of a research problem and the formulation of a question	The problem identified in this research was the use of predominantly physiological end points to evaluate the resuscitation efforts in respect of patients presenting with haemorrhagic shock, in the emergency unit of the hospital <i>Chapter 1, Section 1.2 to 1.4</i>
2. Conduct a literature review	Quantitative research is based on previous knowledge and investigations. A literature review is regarded as extremely important in determining what is already known about the research topic.	A detailed literature review on the shock syndrome, haemorrhagic shock, physical examination of patients presenting with haemorrhagic shock, fluid resuscitation and evaluation of resuscitation efforts was conducted. <i>Chapter 2</i>
3. Conduct clinical field work	The aim of field work is to gain further knowledge on the research topic through interaction with peers in the clinical setting – also to gain access to patients' files and information relevant to the research.	Information was obtained by utilising patient files – this conformed to the inclusion criteria of the research – and by conducting a detailed literature review on the research topic. <i>Chapter 1 and Chapter 2</i>

Phase 2: Plan methods and procedures <i>Decide on the methods and procedures that will be used to address the research question and to collect the data</i>		
Steps	Criteria	Application
1. Select a design	The appropriate study design should be selected so as to minimise bias and to enhance the ability to interpret the study findings.	A quantitative, retrospective, non-experimental, descriptive, cross-sectional and correlational design was used as the research design in this study. <i>Chapter 3, Section 3.3</i>
2. Identify the study population	The entire population in which the researcher is interested and to which the researcher would like to generalise the findings of his/her research.	The population for this study included all the files of male and female trauma patients who presented with haemorrhagic shock on admission to the emergency unit of a Level I public hospital during the period of this study <i>Chapter 3, Section 3.4.1.1</i>
3. Develop a sampling plan	The methods applied to select a sample of the population which will be studied in such a manner that the sample is representative of the entire population.	The sampling plan used in this research included the identification of the study population, the development of a sampling frame, the purposive selection of patients' files, the sample selection and the identification of a sample. <i>Chapter 3, Section 3.4.1, Figure 3.1</i>

Phase 2: Plan methods and procedures <i>Decide on the methods and procedures that will be used to address the research question and to collect the data</i>		
Steps	Criteria	Application
4. Develop methods to measure research variables	The researcher must develop methods to record and measure the data as accurately as possible.	The research variables in this study were measured by means of the development of the data-collection instrument and the application of the principles of validity and reliability to the data-collection instrument. <i>Chapter 3, Section 3.4.2.1 and Section 3.5</i>
5. Plan ethical measures	Measures to ensure that the rights of subjects are protected.	The main ethical issues that were adhered to in this research included obtaining the approval of the ethics committee to conduct the research, and the attainment of informed consent and the right to privacy of the participants in the study. <i>Chapter 3, Section 3.5 and Annexure A</i>
6. Finalise research plan	Prior to collecting the data a pre-test or pilot study is conducted to ensure that the research is plausible.	A pre-test was used to ensure that the research was plausible. <i>Chapter 3, Section 3.4.2.3</i>

Phase 3: Empirical data		
<i>Plan the collection of the data and the preparation of that data for analysis.</i>		
Steps	Criteria	Application
1. Collect data	Quantitative study's data collection process is according to a pre-established plan. It includes instructions on how, what, where and when the data will be collected.	The sample was purposively selected from the population using a sampling frame and the development of a data collection instrument <i>Chapter 3, Section 3.4.1 and Chapter 4, Section 4.2</i>
2. Prepare for data analysis	The data obtained from quantitative studies is typically numeric. Such data needs to be coded and, thus, the data gathered should be translated into numeric data for statistical analysis.	The data captured on the data-collection instrument was coded and processed into numeric format by a statistician for the purposes of statistical analysis. <i>Chapter 4, Section 4.2</i>
Phase 4: Data analysis		
<i>Plan how the data will be prepared, interpreted and reported</i>		
Steps	Criteria	Application
1. Data analysis	The data is processed and analysed in an orderly fashion. Quantitative data is normally analysed by means of statistical analysis.	Statistics developed by the statistician were used for data analysis and for the interpretation of the study results. <i>Chapter 4, Section 4.2</i>
2. Interpretation of results	The researcher makes sense of the data and evaluates the findings, to obtain adequate answers to the research question.	The results of the study were interpreted by identifying objectives according to the aim of the research. <i>Chapter 1, section 1.5, Chapter 5 Section 5.3.</i>

Phase 5: Dissemination <i>Develop a strategy on the way in which the research report will be communicated to others</i>		
Steps	Criteria	Application
1. Communication of findings	The findings of the study should be communicated to other health care professionals through a research report.	The research will be reported on in the five chapters of the study: <ul style="list-style-type: none"> ⇒ Chapter 1: Orientation to the study ⇒ Chapter 2: Literature review ⇒ Chapter 3: Research method ⇒ Chapter 4: Results, discussion and analysis ⇒ Chapter 5: conclusion and recommendations
2. Utilisation of the findings	A plan should be set in place to facilitate the utilisation of the research findings in other settings.	The utilisation of the study findings were summarised according to objectives identified in the aim of the research. The objective was to make recommendations for future resuscitation strategies for patients presenting with haemorrhagic shock in order to improve the outcomes of such patients <i>Chapter 1, section 1.5, Chapter 5, Section 5.3.4</i>

Source: Adapted from Polit and Beck (2004: 48–53)

The research method that was applied in this study is discussed in detail in Chapter 3.

1.9 LIMITATIONS

Limitations are defined by Burns and Grove (2005: 39) as boundaries or problems in the research conducted that limit the generalisability of the research concerned. Limitations may be theoretical or methodological in nature. Although the researcher took great care to ensure minimal limitations in respect of this research it is, nevertheless, the opinion of Polit and Hungler (1989: 22) and Babbie (2005: 254) that virtually all research studies contain some flaws or limitations.

In order to minimise the methodological limitations the researcher constructed a clinical audit checklist which was used as measuring instrument and which included relevant indicators in an effort to avoid irrelevant data. The aim is that data be clear, unambiguous and relevant. The clinical audit checklist was given to experts to evaluate the relevance of the data captured via the data collection instrument. These experts included specialists and peers working in the emergency unit of the hospital as well as a statistician. In addition, a pre-test was conducted to ensure that the researcher would be able to collect the relevant data.

However, the following limitations were identified:

- The study was conducted in a single setting, namely, in a Level I emergency unit of a public hospital in Gauteng province, RSA.
- The number of patients' files obtained during the data collection (N = 46) may be regarded as a limitation. Of the 495 patients' files that were identified and which met the inclusion criteria, 275 files only were obtainable. The remaining files were either incomplete and it was not possible to use the data or else they had been lost. This means that 9,29% only of the intended population's files were used (see Chapter 3; Section 3.4.1).
- Resource constraints impacted hugely on the sample size in the following way:
 - There was no collaboration between the administration department and the researcher despite efforts to involve the head of department of the emergency unit and top management to address this issue.

- During the data collection it was noted that the documentation involved in the collection of the serum lactate levels by nurse practitioners and other healthcare professionals had been completed haphazardly.
- Although the arterial blood gas machine was calibrated on a weekly basis, there were, nevertheless, occasions on which it was either out of order or it was not possible to measure the serum lactate level. This, in turn, resulted in a loss of data in respect of this research and this contributed to the small sample obtained from the population.

1.10 LAYOUT

This dissertation consists of the following six chapters:

- ⇒ **Chapter 1** which introduces the area of interest, identifies the research problem, delineates a research question on which the aim and objectives of the study were based and provides a summary of the method planned and used in the study. It further identifies the frame of reference on which the study was based and tabulates the phases used to plan and to implement the research. The significance and limitations of the study are presented.
- ⇒ **Chapter 2** summarises the existing literature on the topic of interest and, thus, orientates the reader in respect of previous research conducted on the topic. The literature review involves a critical discussion of the information published on the topic being studied and this, in turn, helps to place the research problem in the correct context.
- ⇒ **Chapter 3** discusses the research method in depth. The chapter includes discussions on the research design and methodology.
- ⇒ **Chapter 4** presents the results of the study as well as a detailed interpretation of the study results. The chapter also presents a literature control in order either to support or to contradict the research findings.
- ⇒ **Chapter 5** concludes the research findings and discusses recommendations for both strategies in terms of which to assess haemorrhagic shock as well as for further research that may be conducted. A critical reflection concerning personal

and methodological issues is included in order to demonstrate the researcher's personal and professional development.

1.11 CONCLUSION

Chapter 1 provided a short introduction to the study to familiarise the reader with the topic. It also described the background to the problem which the study was designed to solve and the aim and objectives of the research. The chapter also described and defined the important terms used in the study. In addition, chapter 1 also provided a summary of the research method as well as outlining the ethical considerations, significance and layout used in this dissertation. The following chapter, Chapter 2, consists of a literature review.

2 LITERATURE REVIEW

2.1 INTRODUCTION

Chapter 1 provided an overview of the study itself while chapter 2 presents a discussion of the relevant literature. The aim of the literature review is to orientate both the researcher and the reader in respect of developments in the field pertaining to the research. Chapter 2 further enhances the theory base for the proposed study and indicates the significance of the study. These reasons for conducting a literature review is supported by are shared by Hofstee (2006: 91).

The literature review will provide an overview of the shock syndrome, the classification of shock, as well as a detailed discussion on haemorrhagic shock. The fluid resuscitation of patients suffering from haemorrhagic shock, the assessment of resuscitation, specific considerations with regard to the evaluation of resuscitation and the pathophysiology of the systemic inflammatory response caused by severe trauma will also be discussed in this chapter.

2.2 OVERVIEW OF THE SHOCK SYNDROME

Shock is perhaps one of the most complicated and deadly syndromes in healthcare and is a common occurrence in an emergency unit (Kaplow & Hardin 2007: 243; Shoemaker & Beez 2010: 5). Shock syndrome is the term used to describe the different conditions which cause both inadequate tissue perfusion and circulatory failure. Shock is classified according to causative factor. The classification include hypovolaemic/haemorrhagic shock (plasma loss or whole blood), cardiogenic shock (loss of contractility of the heart), obstructive shock (compression of the heart,

mediastinal shift or venous pooling) or distributive shock (decreased venous return or poor distribution of blood flow) (Kaplow & Hardin 2007: 244; Urden, Stacey & Lough 2006: 1009). If not managed properly, the outcome of shock may be a decrease in tissue perfusion and acute circulatory failure (Urden *et al.* 2006: 1009). Shock syndrome is described by Urden *et al.* (2006: 1009) as an acute and widespread state pertaining to an inadequate supply of oxygen to the tissue and cells in the body which, in turn, may cause metabolic, cellular and haemodynamic changes.

Shock syndrome is caused by an imbalance between oxygen delivery and oxygen demand on a cellular level. This imbalance may result in cellular dysfunction and death. In the view of Gutierrez, Reines and Wulf-Gutierrez (2004: 373), shock syndrome refers to a decrease in blood pressure which causes tissue hypoperfusion. Shock may be life threatening if it is not possible to sustain normal cellular metabolism. Any illness or injury that causes a decrease in tissue perfusion may potentially result in shock (Hoyt & Selfridge-Thomas 2007: 721). In light of these perspectives on shock syndrome, it is evident that shock is not a disease, but rather a clinical manifestation of the body's inability to perfuse its tissue adequately. This view is supported by Hoyt and Selfridge-Thomas (2007: 721).

Shock syndrome affects all the bodily systems and, once it occurs, the homeostatic mechanisms are activated in order to restore the blood flow to enable adequate tissue perfusion (Smeltzer *et al.* 2008: 356). However, if the tissue perfusion is inadequate then shock syndrome will involve all the cells in the body as well as their chemical and metabolic balance. The consequences of inadequate tissue perfusion differ from organ to organ, depending on the compensatory mechanisms and the activation of these compensatory mechanisms. If shock syndrome is left untreated it may progress into irreversible stages, causing severe organ damage and even death (Jacobs & Baker 1995: 82) – see Chapter 2, Section 2.2.2.

In addition, shock syndrome may be categorised into four stages, depending on the severity of the shock. These stages include the following: initial, compensatory,

progressive and refractory (Urden *et al.* 2006: 1009). The pathophysiology of shock syndrome and each of the four stages are discussed in Chapter 2, Sections 2.2.1 and 2.2.2.

2.2.1 Pathophysiology

Generally, shock is caused by an inadequate supply of oxygen and nutrients to the cells, which is caused by an insufficient blood supply to cells. Owing to the lack of oxygen and nutrients, the cells are forced to produce their own energy by means of anaerobic metabolism, that is, metabolism not requiring oxygen (Marieb 2007: 302). The activation of anaerobic metabolism brings about an acidotic cellular environment which, in turn, causes normal cell function to cease. When normal cell function ceases the cell membranes become more permeable with the result that fluid and electrolytes diffuse freely. This, in turn, causes the cells to swell and to burst, leading to cellular death (Smeltzer *et al.* 2008: 357).

When the body is under stress the metabolism requires more oxygen but, because of the prevailing state of stress, it is not possible to supply this oxygen naturally. Compensatory mechanisms are then activated in order to meet the increased demand for oxygen and perfusion on a cellular level. Firstly the respiratory, endocrine and circulatory compensatory mechanisms are activated to increase oxygen delivery on a cellular level (Morton, Fontaine, Hudak & Gallo 2005: 1252-3). If these compensatory mechanisms are not able to meet the oxygen requirements of the cells, the cells then switch from aerobic metabolism to anaerobic metabolism. normal cellular metabolism requiring oxygen to anaerobic metabolism (Marieb 2007: 303). However, anaerobic metabolism is not effective in the production of energy and it causes the release of lactic acid which, in turn, increases the amount of acid that must be eliminated by the body. If the cellular hypoperfusion and decreased oxygenation continue then cellular death occurs and the tissue and organs become progressively more dysfunctional (Morton *et al.* 2005: 1253). The earlier medical

interventions are introduced the better the prognosis for the patient's recovery (Smeltzer *et al.* 2008: 359; Shoemaker & Beez 2010: 5).

In order to summarise the pathophysiology of shock it is necessary to understand the determinants of blood pressure as part of the normal physiology of the body. Figure 2.1 presents a diagrammatic representation of the determinants of blood pressure according to Stone and Humphries (2008: [2]).

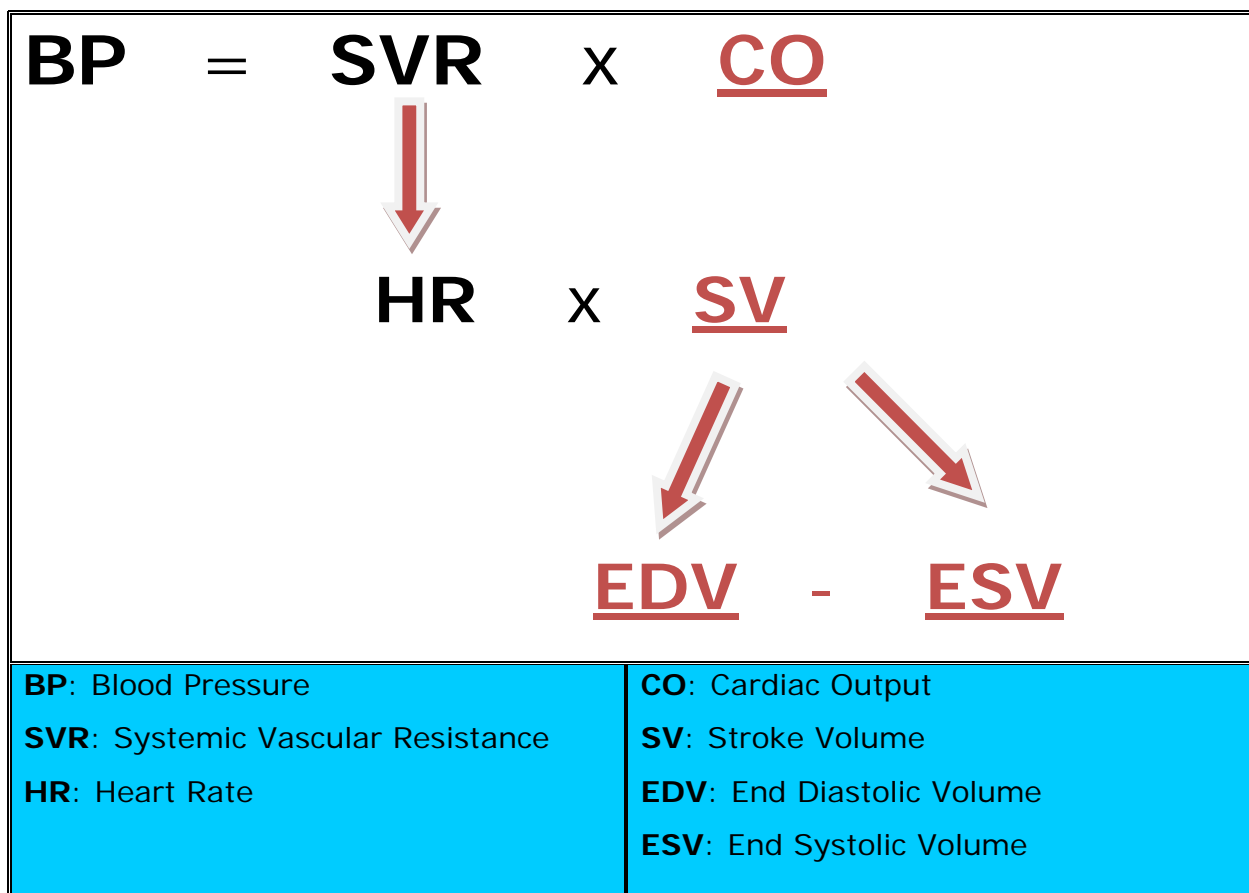


Figure 2.1: Determinants of blood pressure

As may be seen from Figure 2.1, blood pressure (BP) is determined by the formula: $BP = \text{systemic vascular resistance (SVR)} \times \text{cardiac output (CO)}$. SVR refers to the vascular tone which exerts pressure on the ejection of blood from the left ventricle and, thereby, determines diastolic blood pressure (Smeltzer *et al.* 2008: 786). Diastolic blood pressure refers to the blood pressure during the relaxation phase of

the heart (Marieb 2007: 725). Cardiac output, in turn, is defined by Marieb (2007: G-4) as the amount of blood pumped out of the ventricle of the heart in one minute. This volume is not constant and it may be increased in order to increase the blood pressure (Urden *et al.* 2006: 269). Cardiac output (CO) is determined by heart rate (HR) multiplied by stroke volume (SV). SV is the amount of blood ejected at each contraction of the heart muscle or systole (Marieb 2007: G-18), and is determined by end diastolic volume (EDV) minus end systolic volume (ESV). EDV refers to the amount of blood in the right heart chamber during rest, also referred to as preload, while ESV refers to the amount of blood which is ejected from the heart after the heart has contracted— also referred to as contractility (Marieb 2007: 700). SV is determined by the following three factors, namely:

- ⇒ **Preload:** Preload is determined by the volume of blood in the ventricle at the end of diastole. At the end of diastole the filling of the ventricle with blood volume is at a maximum. Preload is, thus, the term used to describe the degree of stretch of the heart muscle (Smeltzer *et al.* 2008: 786). Preload is also referred to as EDV and it is influenced by a decrease in the circulating blood volume or hypovolaemic shock (Stone *et al.* 2008: [1]).
- ⇒ **Afterload:** Afterload refers to the pressure exerted by the SVR on the ejection of blood from the left ventricle during contraction of the heart muscle, namely, during systole (Marieb 2007: 702). Factors influencing vascular tone include the use of vasodilatory drugs as well as any other condition which may cause a loss of vascular tone, for example, in anaphylactic shock, septic shock and neurogenic shock (Stone *et al.* 2008: [2]; Urden *et al.* 2006: 1012).
- ⇒ **Contractility:** Contractility refers to the power of contraction of the heart muscle independent of muscle stretch or preload (Marieb 2007: 702). An increase in contractility may be caused by either a neural response or by medication that increases SV. A decrease in contractility may be caused by hypoxia, acidosis or by medication that decreases SV (Smeltzer *et al.* 2008: 786). Cardiogenic shock influences contractility and also EDV (Stone *et al.* 2006: [2]).

2.2.2 Stages

The severity of shock may be described in terms of a “stage” (O’Shea 2005: 191). Shock syndrome is divided into four stages so as to facilitate an understanding of the physiological response, signs and symptoms associated with shock syndrome. These stages include the initial, compensatory, uncompensated and irreversible stages (Urden *et al.* 2006: 1009).

2.2.2.1 *Initial stage*

Urden *et al.* (2006: 1009) maintain that the initial stage of shock is characterised by a decrease in the cardiac output – the amount of blood per minute that is ejected from the left side of the heart (Smeltzer *et al.* 2008: 358) – and, thus, a decrease in the blood supply to the tissue of the body.

During the initial stage of shock cells which are deprived of oxygen are no longer able to produce their own energy. The cells then switch to anaerobic metabolism that leads, in turn, to the formation of lactic acid as a by-product. The lactic acid causes systematic acidosis which damages the cells (O’Shea 2005: 176).

2.2.2.2 *Compensatory stage*

During the compensatory stage of shock the body tries to maintain homeostasis by activating the compensatory mechanisms in order to increase perfusion on a cellular level. These compensatory mechanisms are managed as part of the body’s nervous system and include the neural, hormonal and chemical responses (Urden *et al.* 2006: 1009).

The neural response refers to the activation of those bodily functions that are controlled by the body’s nervous system and include an increase in heart rate and contractility, vasoconstriction of the vascular system, and the redistribution of blood to vital organs such as the brain and heart in order to facilitate a rise in blood

pressure. The hormonal response causes a number of hormonal reactions which, in turn, influence various systems. Included in these hormonal responses is the Renin-Angiotension system which is responsible for the retention of water and sodium, thus causing an increase in the circulating blood volume and, in turn, leading to an elevation in blood pressure. The Renin-Angiotension system also stimulates the release of the adrenocorticotrophic hormone (ACTH) which is a hormone responsible for the production of glucocorticoids. This hormone raises the blood glucose levels which are essential in helping the body to manage stressors (Marieb 2007: 731). These hormonal responses give rise to the activation of the chemical response through the release of chemicals, namely, adrenalin and noradrenalin, which, in turn, further enhance the compensatory mechanisms of the body (Urden *et al.* 2006: 1009).

2.2.2.3 *Progressive stage*

During the progressive stage of shock, the compensatory mechanisms of the body begin to fail and the patient presents with clearer signs and symptoms of a shocked state (Collins 2000: [3]; Hoyt & Selfridge-Thomas 2007: 722). Those mechanisms that were initially helpful become ineffective and, in fact, cause additional tissue damage. Additional pathologic responses to the shock state (e.g. severe lactic acidemia and inflammatory activation) may lead to either multiple system deterioration or even failure (Shoemaker & Beez 2010: 5). The signs and symptoms of shock are now discernible in the effects of tissue hypoperfusion on the lungs, heart, brain, kidneys, liver, gastro intestinal system, and circulation causing, in turn, multi-organ death syndrome (MODS) (Smeltzer *et al.* 2008: 361).

It is the opinion of Morton *et al.* (2005: 1270) that this stage is characterised by a failure of those compensatory mechanisms which are normally needed in order to maintain adequate tissue perfusion. The failure of these compensatory mechanisms may be seen in metabolic and circulatory dysfunction as well as in the activation of both the inflammatory and the immune response. During the progressive stage of shock, organ failure becomes more pronounced.

2.2.2.4 Refractory stage

The final stage of shock is the critical point at which no treatment will reverse the process (Hoyt & Selfridge-Thomas 2007: 722). The extent of the damage to the cells and tissue leads to cellular destruction and may be so severe that death is inevitable. In fact, death may be imminent even should the metabolic and circulatory problems be resolved (Morton *et al.* 2005: 1270). The refractory stage is also characterised by MODS, brain damage and cellular death (Collins 2000: [4]; Shoemaker & Beez 2010: 5).

2.2.3 Classification

The classification of shock which was introduced by Hinshaw and Cox in 1972, is still used by clinicians today in order to provide a conceptual framework for both research and communication purposes (Irwin, Rippe & Goodheart 2003: 174). The classification includes the following four main categories, namely:

- **Hypovolaemic shock:** Shock caused by a decrease in the circulating blood volume that may have been caused by haemorrhage.
- **Obstructive shock:** Shock caused by an extra cardiac obstruction of blood flow, for example as a result of cardiac tamponade.
- **Cardiogenic shock:** Shock caused by dysfunction of the cardiac muscle as a result of myocardial infarction.
- **Distributive shock:** Shock due to a deranged distribution of blood and volume as is seen in neurogenic, anaphylactic and septic shock.

In the view of both Morton *et al.* (2005: 1255) and Shoemaker and Beez (2010: 5), shock may be classified according to the organ or system that causes the decrease in tissue oxygenation and perfusion. In other words, a loss of circulating volume causes hypoperfusion and, thus, hypovolaemic shock. Major injury to the heart muscle that causes mechanical and structural dysfunction and damage to the heart may result in cardiogenic shock. The body's response to an allergen causes

anaphylactic shock while a disruption to the sympathetic nervous tone in the brain causes neurogenic shock. The inflammatory response triggered by a severe infection which results in the maldistribution of circulating blood volume and cellular hypoperfusion leads to septic shock (Morton *et al.* 2005: 1256).

Stone and Humphries (2003: 107) maintain that the main cause of hypovolaemic shock is an exogenous or endogenous loss of fluid, electrolytes, blood or plasma. Accordingly, hypovolaemic shock caused by blood loss is referred to as haemorrhagic shock. Cardiogenic shock is caused by a pump failure of the heart, arrhythmia or valvular dysfunction. Obstructive shock may be due to an obstruction of the systemic circulation or the pulmonary circulation, although it may also be caused by pericardial or cardiac disease. The abnormal distribution of fluid volume that manifests as distributive shock may be caused by neurogenic, anaphylactic and septic shock.

Jones and Klines (2006: [3]) maintain that, because shock is usually concealed in another diagnosis, it is rarely listed as a primary coding diagnosis. As a result, the number of shocked patients entering the emergency unit is often underestimated. The main distinguishing aetiology of shock is tabulated in Table 2.1 (Boon, Colledge & Walker 2006: 186 and Smeltzer *et al.* 2008: 373). The clinical manifestation of shock as indicated in Table 2.1 is derived from Urden *et al.* (2006: 1012–1025).

Table 2.1: A summary of the classification of shock

Class	Aetiology	Clinical manifestation
Hypovolaemic	Decrease in circulating volume as a result of: <ul style="list-style-type: none"> - internal or external bleeding - haemorrhage - burns - dehydration 	Compensatory phase: <ul style="list-style-type: none"> - sinus tachycardia - decreased peripheral perfusion - normal blood pressure - increased heart rate

Obstructive	<p>Disorders causing a mechanical obstruction of the blood flow through the heart:</p> <ul style="list-style-type: none"> - pulmonary embolism - cardiac tamponade - dissected aorta - tension pneumothorax 	<ul style="list-style-type: none"> - systolic blood pressure <90mmHg - decreased level of consciousness - cool, clammy skin - rapid and laboured breathing - weak peripheral pulses
Cardiogenic	<p>Cardiac failure which influences cardiac contractility:</p> <ul style="list-style-type: none"> - myocardial infarction - structural problems - disrythmia 	<ul style="list-style-type: none"> - Systolic blood pressure < 90 mmHg - decreased level of consciousness - cool, clammy skin - decreased urine output - chest pain.
Distributive	<p>Caused by distorted distribution of blood and fluid volume which manifests in :</p> <ul style="list-style-type: none"> - neurogenic shock - anaphylactic shock - septic shock 	<p>Specific to presenting condition</p>
Neurogenic	<p>Major damage to the brain or spinal cord, causing a disruption in either the brainstem or in the vasomotor control</p>	<ul style="list-style-type: none"> - decrease in blood pressure - bradycardia - dry, warm skin
Anaphylactic	<p>Vasodilatation and activation of the acute inflammatory response as a result of an allergen: e.g. a bee sting</p>	<p>Impact on various systems, most commonly:</p> <ul style="list-style-type: none"> - rash - swelling of throat - pulmonary oedema - anxiety

Septic	Activation of the systemic inflammatory response causing endothelial damage and, ultimately, organ damage.	- warm, flushed skin Septic shock is recognised by the presence of more than two indicators of the inflammatory response as well as the dysfunction of one or more organs. The most common organs which fail include the lungs, heart or vascular system
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Source: Adopted from Boon *et al.* (2006:186), Smeltzer *et al.* (2008: 373) and Urden *et al.* (2006:1012–1025)

Although shock is caused by a decrease in tissue perfusion as a result of an inadequate blood supply to the tissue, the aetiology of shock may differ (see Table 2.1). The clinical manifestation of shock also differs despite the fact that the management of shock is focused on improving the oxygen supply on a cellular level and preventing cellular death (Urden *et al.* 2006: 1010; Shoemaker & Beek 2010: 5).

The medical management of shock depends on the type of shock present and will be directed towards treating the underlying disorder. It is not possible for the body's compensatory mechanisms to compensate for all stages of shock and, thus, it is essential that fluid and medical management be introduced timeously in order to ensure both adequate perfusion as well as a sufficient supply of oxygen to the cells (Smeltzer *et al.* 2008: 356).

In view of the research topic, haemorrhagic shock only, – a subdivision of hypovolaemic shock – will be discussed in-depth in the literature review.

2.3 HAEMORRHAGIC SHOCK

Haemorrhagic shock is caused by a decrease in the circulating volume as a result of blood loss. This decrease in circulating volume leads to both inadequate tissue perfusion and the activation of the shock response (Urden *et al.* 2006: 1012). The aetiology, pathophysiology, assessment, diagnosis, classification, and physical examination of shock, as well as the principles pertaining to the management of haemorrhagic shock, will be addressed.

2.3.1 Aetiology

For the purpose of this study the focus will be on haemorrhagic shock which is one of the major causes of shock in patients entering an emergency unit (Kane *et al.* 2007: 61; Shoemaker & Beez 2010: 5). According to Kauvar, Lefering and Wade (2006: S3), shock which arises from blood loss is the second leading cause of early death among the injured with central nervous system injuries only proving more fatal.

Irwin *et al.* (2003: 174) are of the opinion that haemorrhagic shock is almost always considered in patients presenting in a shocked state with a decrease in tissue perfusion. Haemorrhagic shock is caused by absolute hypovolemia. Absolute hypovolaemia, in turn, arises from the loss of intravascular volume caused by loss of whole blood, plasma and other body fluids, as well as a loss of intravascular integrity. Whole blood may be lost as a result of trauma, surgery or gastrointestinal bleeding while plasma may be lost because of either severe burn wounds or large lesions. Vomiting and diarrhoea may be responsible for a significant loss of other bodily fluids (Urden *et al.* 2006: 1012). Intravascular volume loss may be as a result of external haemorrhage as in trauma or else internal haemorrhage caused by internal injuries (Collins 2000: [4]).

Relative hypovolaemia may also cause shock and is caused by vasodilatation and an increase in capillary membrane permeability as a result of sepsis or anaphylaxis. A loss of sympathetic stimulation due to neurological injury also causes vasodilatation with thermal injuries also affecting capillary membrane permeability. A decrease in colloid osmotic pressure may cause relative hypovolaemia as a result of low sodium levels and intestinal obstruction (Urden *et al.* 2006: 1012).

It is the opinion of Morton *et al.* (2005: 1255) that haemorrhagic shock is caused by inadequate circulating volume, mostly because of blood loss or dehydration. Shock resulting from acute volume loss occurs because the body's normal compensatory mechanisms are overwhelmed and are not able to compensate for the loss of circulating volume. This, in turn, may lead to a number of secondary complications, namely, hypotension, electrolyte and acid-base disturbance and organ dysfunction as a result of inadequate blood and oxygen supply. Peitzman, Rhodes, Yearly, Schwab and Fabian (2002: 13) maintain that haemorrhagic shock is usually caused by acute blood loss in injured patients, but that it may also be due to a loss of red blood cells, plasma, extra cellular fluid or all three. The severity of haemorrhagic shock depends on the intensity and duration of the shock. Prolonged mild shock may be just as dangerous as acute, intense shock (Shoemaker & Beez 2010: 5).

A number of injuries may accompany haemorrhagic shock, for example, injuries to the chest and abdomen, and fractures to the long bones (e.g. the femur, tibia and fibula). These injuries may cause various degrees of circulating blood volume loss and, in combination with other injuries, such as lacerations, multiple bruising and bleeding wounds, may further increase the likelihood of haemorrhagic shock. Injury to organs such as the liver, spleen and major chest vessels may also cause significant blood volume loss that may, in turn, lead to haemorrhagic shock (Jacobs & Hoyt 2000: 71). The incidences of life threatening injuries are divided into categories of severity by Hill and Davies (2002: 198). Head injuries cause mortality in 70% of patients presenting with trauma. Chest and abdominal injuries account for 35% of deaths in patients, and spinal injuries 5%. In RSA the main cause of

death, according to the National Injury Mortality Surveillance System of 2005, was homicide-related deaths and accidental deaths, with both accounting for 38,7% of the total national deaths. It was also found that 74,3% of all accidental deaths were as a result of transport-related accidents (SA Health *Info* 2005).

2.3.2 Pathophysiology

The pathophysiology of haemorrhagic shock may best be illustrated in the flow diagram depicted in Figure 2.2.

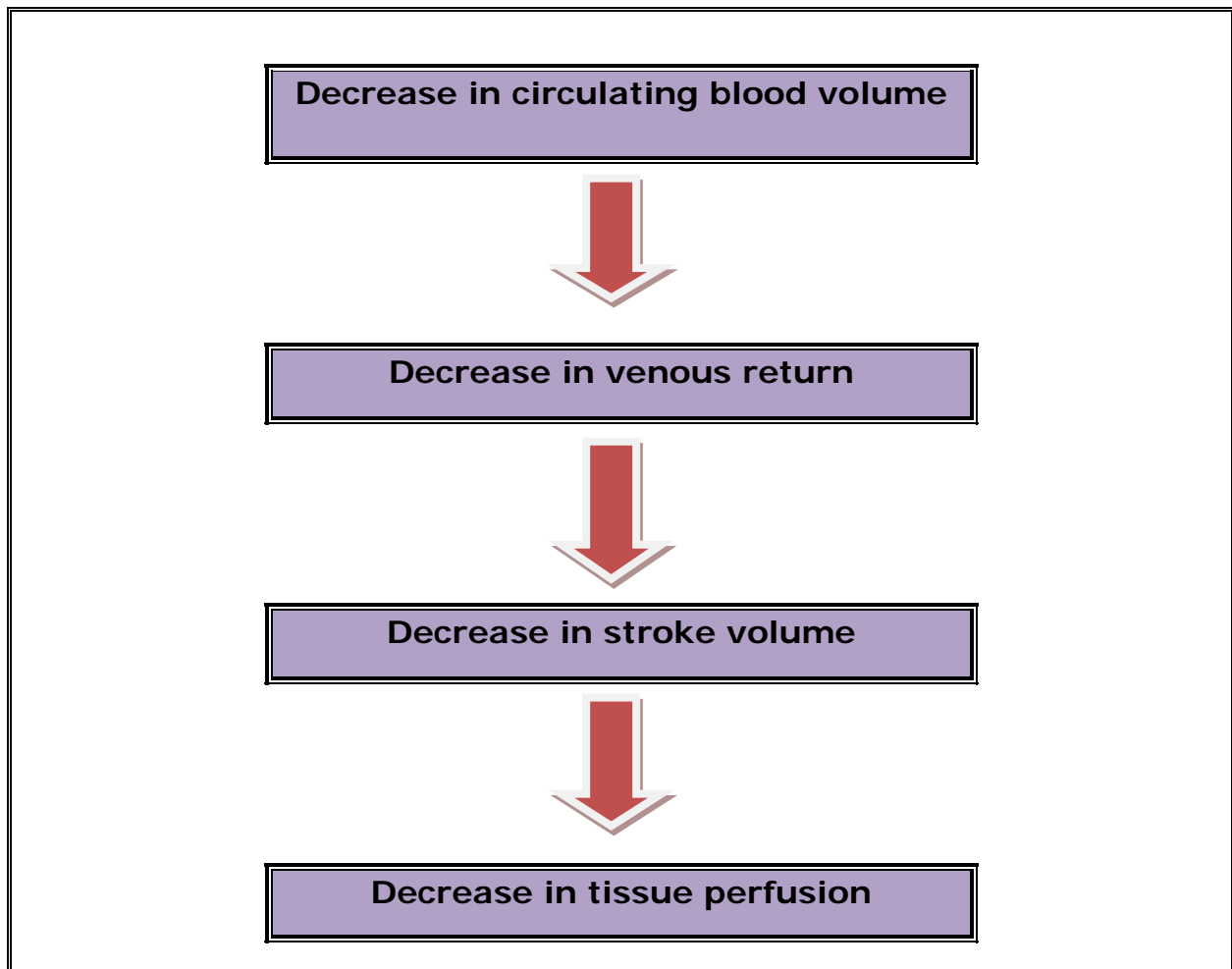


Figure 2.2: Summary of the pathophysiology of haemorrhagic shock
(adapted from Smeltzer et al. 2008: 359)

As is evident in Figure 2.2, a loss of blood as a result of haemorrhage causes a decrease in the circulating blood volume which, in turn, causes a decrease in the venous return to the heart. A decrease in the venous return then decreases the stroke volume which lowers the cardiac output. A low cardiac output causes a decrease in the cellular perfusion which implies a diminished supply of oxygen to the cells. This, in turn, causes inadequate tissue perfusion and impaired cellular metabolism (Smeltzer *et al.* 2008: 361). Patients may lose up to 15% of intravascular volume before manifesting signs of shock (Halaas 2006: [1]). Compensatory mechanisms may be activated by this loss of volume, thus causing cellular hypoperfusion, which, in turn, stimulates the pulmonary, endocrine and circulatory systems in order to ensure adequate oxygenation of the cells. Figure 2.2 depicts the compensatory mechanisms operating in haemorrhagic shock

The compensatory mechanism of the body activated by shock is depicted in Figure 2.3. This compensatory mechanism involves the *brain* which, during hypoperfusion of cells, increases the production and release of the anti-diuretic hormone (ADH) – an hormone which is responsible for vasoconstriction by causing an increase in blood pressure (Morton 2006: 1254). The brain also relays a message of thirst in order to boost the oral intake of fluids so as to remedy the depleted fluid status.

The *cardiovascular compensatory mechanisms* include an increase in heart rate, contractility, increased SVR, constriction of veins and the shunting of blood away from the kidneys, gastro-intestinal tract, skin and skeletal muscle (Morton 2006: 1253). The *adrenal gland* is responsible for the increased release of aldosterone, which is the hormone responsible for the re-uptake of sodium and water through the kidneys (Marieb 2007: 627). This, in turn, increases preload which increases blood pressure (Morton 2006: 1254) and also aids in the excretion of adrenalin and noradrenalin, which activate the “fight-and-flight” response. This response causes blood to be shunted away from the organs such as the skin, kidneys and the gastro intestinal tract to the more vital organs such as the brain and heart in order to ensure adequate perfusion (Smeltzer *et al.* 2008: 358).

The *liver* aids the compensatory mechanisms by releasing stored blood and constricting veins, while the *kidneys* present with decreased urine output and an increase in sodium and water uptake. Water from the intestinal tract is reabsorbed into the capillary bed, thus enhancing the fluid status (Morton 2006: 1253).

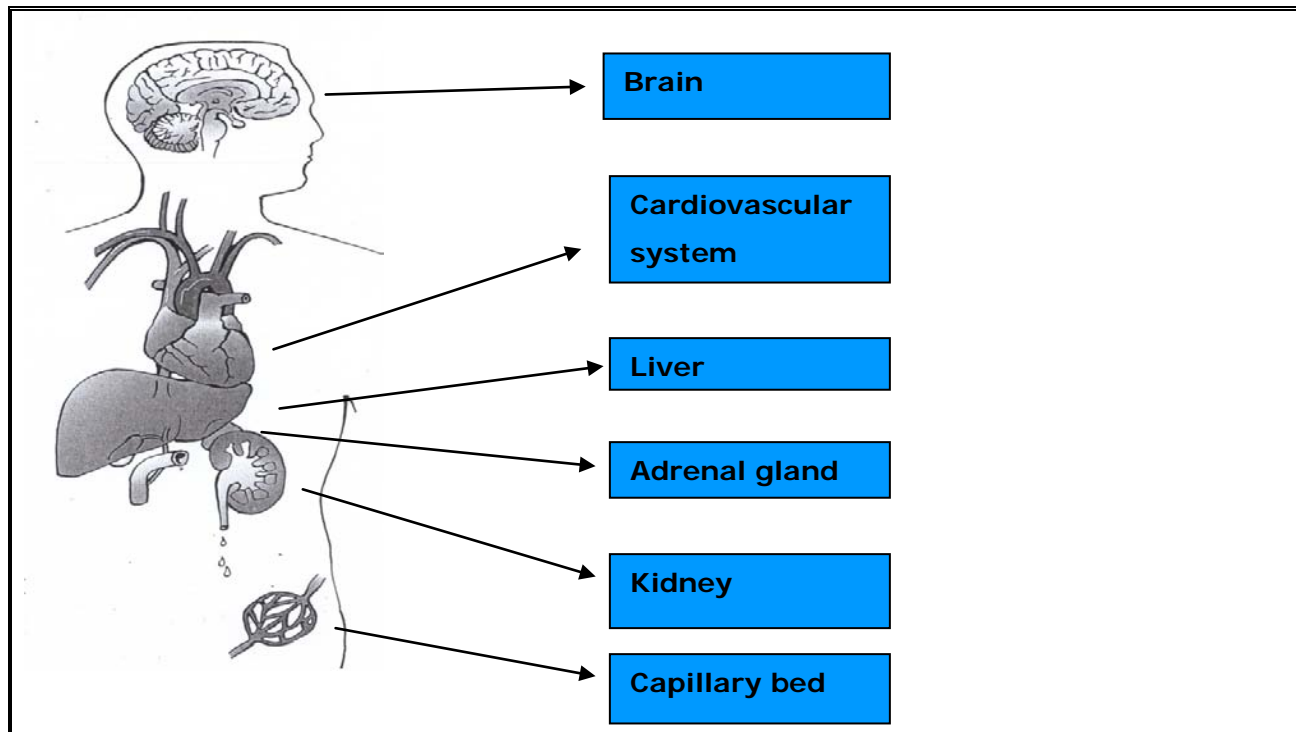


Figure 2.3: Compensatory mechanisms of haemorrhagic shock (adopted from Morton *et al.* 2005: 1253).

2.3.2.1 Inflammatory response

The activation of the inflammatory response forms part of pathophysiology haemorrhagic shock. Trauma causes metabolic and immunological changes which, in turn, result in both the activation of the systemic inflammatory response syndrome (SIRS) and the release of cytokines. These responses may cause damage and death to the organs (DeLong & Born 2004: 57). SIRS is defined by Smeltzer *et al.* (2008: 366) as an “overwhelming inflammatory response in the absence of infection causing relative hypovolaemia and decreased tissue perfusion”.

However, the activation of SIRS caused by trauma may be beneficial to the patient as it may serve as a trigger for the activation of the body's immune response. Nevertheless, according to (Giannoudis, Hildebrand & Pape 2004: [2]) it may also have dire consequences if left untreated. Boon *et al.* (2006: 185) describe the systemic inflammatory response as the systemic release of cytokines and other mediators that trigger interaction between the coagulation pathways, platelets, endothelial cells and white blood cells, especially the polymorphonuclear cells (PMNs). Cytokines, their mediators and their receptors were identified in the late 1980s as both indicators and predictors of mortality and morbidity in trauma patients. PMN cells express selectins, thus causing the selectins ultimately to move through the damaged endothelium of the cell into the extra vascular and interstitial space. PMN cells, together with other proteins and fluid, cause tissue oedema and inflammation.

The immune system, which is also activated, causes the release of macrophages, leukocytes and natural killer cells. Additional inflammatory mediators may be released as a result of ongoing endothelial injury, intravascular coagulation, microvascular occlusion and tissue damage (DeLong & Born 2004: 58). The final result of this process is severe organ damage which presents as Acute Respiratory Distress Syndrome (ARDS) of the lungs, Acute Tubular Necrosis of the kidneys (ATN) and a disruption of the coagulation system, resulting in Disseminating Intravascular Clotting (DIC) disorder (DeLong & Born 2004: 61).

The activation of SIRS leads to relative hypovolaemia as a result of the mediators causing vasodilatation and myocardial depression. The major cause of severe tissue damage in SIRS is the inability of the tissue to take up oxygen, even if the oxygen is supplemented. The result of this inability is both a raised lactate level interpretable on an ABG analysis and high mixed venous oxygen saturation (SvO₂). If SIRS is left untreated it will lead to progressively deteriorating organ function which will, in turn, result in MODS (Barbee *et al.* 2010: 114).

2.3.3 Assessment and diagnosis

The clinical appearance of haemorrhagic shock differs according to the amount of blood or fluid loss. The severity of shock increases according to the percentage of total body fluid lost and the ability of the body to compensate for this loss (Urden *et al.* 2006: 1012). A complete history of events leading to the shocked state is important for the diagnosis of shock, as subtle blood loss due to haemorrhage over a long period of time may give rise to shock (Collins 2000: [4]).

In the early stages of shock, the signs of the shock may be subtle as a result of the early activation of compensatory mechanisms which render the recognition of the shocked state difficult (Stone *et al.* 2008: [3]). The effect of volume loss due to haemorrhage depends on both the amount of fluid lost and the body's ability to compensate for that loss. Signs and symptoms of shock may be absent even in cases in which up to 30% of blood has been lost and it is only when the compensatory mechanisms fail that the shocked state becomes apparent (Barbee *et al.* 2010: 113; Collins 2000: [4]).

2.3.4 Classification

A classification of the severity of haemorrhagic shock has been developed by the American College of Surgeons. In terms of this classification, haemorrhagic shock is broadly categorised according to the estimated blood loss (Soreide & Grande 2001: 294). This classification was implemented as the Advanced Trauma Life Support (ATLS) programme and was aimed at optimising the care of critically injured patients. It has since become the gold standard for the management of such patients in the United Kingdom (Greaves & Johnson 2002: 23). However, despite the fact that this categorisation provides a standardisation in respect of the effect of blood loss on the body, it is based on normal adult physiology only. In the clinical field it is often difficult to estimate blood loss resulting from haemorrhage accurately, and it is essential that factors such as age and health status be taken

into consideration when assessing the effect of haemorrhage on the body (Soreide & Grande 2001: 294; Barbee *et al.* 2010: 113). The classification of haemorrhage according to ATLS is as follows:

- ⇒ Class I Haemorrhage
- ⇒ Class II Haemorrhage
- ⇒ Class III Haemorrhage
- ⇒ Class IV Haemorrhage

Each of these classes will be discussed in Sections 2.3.4.1 to 2.3.4.4.

2.3.4.1 Class I Haemorrhage

Class I haemorrhagic shock is characterised by an estimated blood loss of less than 750 ml (up to 15% of a patient's total circulatory volume). Its features include minimal clinical symptoms with a sinus tachycardia as the symptom that one may expect to observe in a clinical setting. No measurable changes will be noted in blood pressure, pulse pressure or respiratory rate. For the majority of healthy patients an amount of blood volume loss of less than 750 ml does not require replacement, as compensatory mechanisms are able to restore such a blood loss within 24 hours (American College of Surgeons 2004: 75).

In the view of Morton *et al.* (2005: 1255), clinical findings as a result of haemorrhagic shock are directly related to both the amount and the acuity of the volume loss. It is, therefore, essential that these findings be considered carefully in special circumstances such as dealing with the elderly or with patients with chronic diseases. Compensatory mechanisms in these patients may be subtle and are often missed. Urden *et al.* (2006: 1012) describe the patient in this stage of shock as generally symptom free, although with slight anxiety. Compensatory mechanisms are required to maintain the cardiac output fully in this stage.

2.3.4.2 Class II Haemorrhage

The American College of Surgeons (2004: 75) describes the estimated blood loss in Class II haemorrhagic shock as being between 750 and 1500 ml (15–30% of total circulating volume). Changes in the hemodynamic parameters that may be expected include an increased pulse rate of above 100 beats per minute (b/min) (sinus tachycardia), an increase in respiratory rate (tachypnea), and a decrease in pulse pressure, although these parameters are influenced by a decrease in cardiac output and the activation of more intensive compensatory mechanisms (Urden *et al.* 2006: 1013).

In a study conducted by Brasel, Guse, Gentilello and Nirula (2007: 814), the researchers scrutinised the value of pulse rate as a vital sign to indicate the severity of haemorrhagic shock. In their retrospective data review they found little conclusive evidence to the effect that, except in cases involving elderly patients and patients with penetrating trauma, the presence of tachycardia may be linked to severity of haemorrhage (Brasel *et al.* 2007: 814). They, therefore, recommended that pulse rate should not be used as an isolated vital sign to estimate the severity of haemorrhage, and that an absence of tachycardia with haemorrhage should raise concern about the patient's condition (Brasel *et al.* 2007: 814; Victorino, Battistella & Wisner 2003: 679).

A decrease in pulse pressure is one of the body's physiological compensatory mechanisms which may be activated to protect the cells from hypoperfusion. This decrease in pulse pressure is indicated by the difference between systolic and diastolic blood pressure with this difference being due to a rise in diastolic blood pressure. Physiologically, the inotropes produced by catecholamine cause an increase of peripheral vascular tone and resistance, thus causing the diastolic blood pressure to increase. In view of the fact that systolic blood pressure changes minimally during the early stages of shock, the monitoring of pulse pressure is a reliable indicator of the presence of shock and should, thus, be monitored. An additional indicator of Class II shock is the presence of subtle changes in the level of consciousness, including anxiety and hostility. Such patients may initially be

stabilised by using crystalloid fluid although they may also need a blood transfusion (American College of Surgeons 2004: 75).

2.3.4.3 Class III Haemorrhage

The estimated blood loss in this class of haemorrhagic shock is approximately 2000 ml (30–40% of circulatory volume) in adult patients. Clinical signs and symptoms include a sinus tachycardia, tachypnea, significant changes in the level of consciousness, and a measurable fall in systolic blood pressure. Such patients almost always require a blood transfusion (American College of Surgeons 2004: 75).

A retrospective study on the importance of blood pressure as a vital sign in estimating the severity of haemorrhagic shock was conducted. In this study the systolic blood pressure that is used as a threshold in terms of which to define hypotension was redefined. Hypotension is mostly identified by a systolic blood pressure of less than, or equal to, 90 mmHg. However, this retrospective study found that the mortality rate increased significantly for every 10 mmHg in respect of a drop in the systolic blood pressure below 110 mmHg (Eastridge, Salinas, McManus, Blackburn, Bugler, Cooke, Concertina, Wade & Holcomb 2007: 292). The study concluded that a systolic blood pressure of 110 mmHg was a better threshold of hypotension than the 90 mmHg as previously defined. The researchers suggested that this newly defined threshold be used as a diagnostic tool to facilitate the identification of hypotension and to prevent treatable complications (Eastridge *et al.* 2007: 296).

The amount of blood lost indicate that the severity of shock in Class III shock also worsens. This is known as the progressive stage of shock and, in this stage, compensatory mechanisms fail to maintain adequate tissue perfusion. Marked tachycardia above 120 b/min and the development of cardiac dysrhythmia provide evidence of this deranged tissue perfusion. The blood supply to the kidneys and

skin is impaired and the patient presents with minimal urine output as well as a cold, clammy, ashen skin (Urden *et al.* 2006: 1013).

Kauvar *et al.* (2006: S5) maintain that the degree of haemorrhage causing haemorrhagic shock is directly linked to the outcome of patients suffering from this type of shock, with the presence of haemorrhagic shock being associated with poor prognoses. In other words, prolonged hypotension is related to late mortality and the development of multi-organ failure.

2.3.4.4 Class IV Haemorrhage

According to the American College of Surgeons (2004: 74–75) this stage of hypovolaemic shock is life threatening. It is associated with the loss of more than 40% of a patient's circulating volume and, if left untreated, death may occur within minutes. A loss of 50% or more of the patient's blood volume is associated with loss of consciousness.

Urden *et al.* (2006: 1013) refer to this stage of haemorrhage as the refractory stage of shock. During this stage compensatory mechanisms cease and organ failure occurs. This stage is distinguished by severe tachycardia which presents with a pulse rate greater than 140 b/min (American College of Surgeons 2004: 74), as well as a rapid decrease in blood pressure as characterised by the absence of peripheral pulses. The skin becomes mottled and cyanotic with no capillary refill as a result of peripheral vasoconstriction. No urine output is noted and the patient will manifest lethargy and a loss of consciousness. Failure of the bodily systems and organs are noted through different clinical manifestations (Urden *et al.* 2006: 1013). Such patients almost always need a rapid blood transfusion as well as immediate surgical intervention in order to stop the haemorrhage.

The classification of haemorrhage has been modified by Gutierrez *et al.* (2004: 373) and is summarised in Table 2.2:

Table 2.2: Classification of haemorrhage

Parameter	Class I	Class II	Class III	Class IV
Blood loss ml	< 750	750–1500	1500–2000	> 2000
Blood loss %	< 15%	15–30%	30–40%	> 40%
Pulse rate per min	< 100	> 100	> 120	> 140
Blood pressure	Normal	Decreased slightly	Decreases	Decreased
Respiratory rate per min	14–20	20–30	30–40	> 35
Urine output ml/hr	> 30	20–30	5–15	Negligible
Central nervous system symptoms	Normal	Anxious	Confused	Lethargic

Source: Gutierrez et al. (2004: 374)

Table 2.2 illustrates the differences between each stage of haemorrhagic shock. Class I shock is the least severe with minimal physiological changes, while Class IV shock affects other bodily functions.

2.3.5 Principles of management

The need for consistency in the management of trauma patients was first recognised in 1976, when a tragedy changed the management of trauma within the first hour of injury. Before 1980, the management of trauma patients by American doctors had lacked consistency and the outcomes were often unfavourable (American College of Surgeons 2004: 4). In order to decrease the mortality of critically injured patients, a course was devised to provide a universally accepted guideline for the management of trauma patients. The first Advanced Trauma Life Support (ATLS) programme was implemented by the Southeast Nebraska

Emergency Medical Services. One year later, after recognising trauma as a surgical disease, the American College of Surgeons incorporated ATLS into their educational programme (American College of Surgeons 2004: 4).

The course was first accepted by the Canadians in 1981, and it soon spread to the rest of the world. The South African Resuscitation Council accepted and implemented the ATLS course in 1992 (American College of Surgeons 2004: 7). The course is now taught and implemented in 43 countries worldwide and ATLS is now the international standard for the initial evaluation and management of trauma patients (Collicot 2005: [3]).

In accordance with the principles of ATLS, the medical management of haemorrhagic shock includes the restoration of circulating blood volume and the determination of the source of volume loss. The choice of fluid used for volume replacement will depend on the severity of shock and the type of fluid lost (Morton *et al.* 2005: 1256).

The most important strategy in the nursing management of patients with haemorrhagic shock is to prevent the shocked state from actually occurring. This may be done by the early recognition of underlying shock and the rapid management of shock. It is not always possible to prevent shock and it is, thus, the responsibility of the nurses to assist in the management of shock and to restore intravascular volume through adequate fluid administration (Smeltzer *et al.* 2008: 368).

The concept behind the ATLS course was initially difficult to accept, although it was based on treating the most serious threat to life first. The second concept which represented a shift away from traditional management was that patients could be treated without a definitive diagnosis first having been made. The development of the mnemonic "**ABCDE**" had its origin in the paradigm shift that a detailed history was not needed in the initial management of a severely injured trauma patient, as had been believed in the past (American College of Surgeons 2004: 4). The

mnemonic represents the notion that certain conditions kill faster than others, and should therefore receive priority (Proehl 2009: 2).

Tintinalli, Kelen and Stapczynski (2004: 221) are in agreement with this edict of the American College of Surgeons and they also emphasise the importance of the management of patients in the first hour of injury, namely, the “golden hour”. Accordingly, the “**ABCDE**” mnemonic defines the order in which evaluation and intervention should take place (American College of Surgeons 2004:5). As contained in the ATLS manual (American College of Surgeons 2004:76) the mnemonic stands for following:

- ⇒ **A**irway with cervical spine protection
- ⇒ **B**reathing
- ⇒ **C**irculation and control of external bleeding
- ⇒ **D**isability or neurological status
- ⇒ **E**xposure (undress) and **E**nvironment (temperature control)

Tintinalli *et al.* (2004: 221) promote the use of this mnemonic devised by the American College of Surgeons and, in addition, include the attainment of the end points of resuscitation. According to the ATLS manual and also the concepts adopted by Halaas (2006: [1–9]) and Tintinalli *et al.* (2004: 221) based on the principles of ATLS, the management of a patient suffering from haemorrhagic shock should be conducted as discussed in the following sections.

2.3.6 Physical examination

The physical examination is directed at an immediate diagnosis of life-threatening injuries and it includes the assessment of the following: **A**- Airway, **B** – Breathing, and **C** – Circulation. **D** – Disability and **E** – Exposure also form part of the physical examination but are assessed only once the patient’s airway, breathing and circulation has been stabilised. Other interventions that form part of the physical examination will also be discussed.

2.3.6.1 Airway and breathing

The securing of an adequate airway is the first and most important priority, as an occluded airway may kill a patient within three minutes. The “jaw thrust” or “head tilt chin lift” airway manoeuvre may be used to secure an open airway. Supplementary oxygen may be administered to keep the oxygen saturation greater than 95%. If the patient is not able to breathe effectively on his own, then endotracheal intubation may be considered (Greaves & Johnson 2002: 25).

Halaas (2006: [2]) adds the need for the mechanical ventilation of patients with a decreased respiratory drive to supplement the assessment of the airway and breathing of a patient. Irwin *et al.* (2003: 183) concur that the control of the airway is the most important priority, and that the stabilisation of the cardiovascular system would mean nothing if adequate oxygenation to relieve hypoxia has not been established. Patients who are able to communicate effectively and who are fully conscious rarely need airway assistance. Endotracheal intubation should be considered if the patient is unable to protect his own airway, and is not fully conscious. As well as the assessment of breathing, the management of any life-threatening chest injuries that may cause hypovolaemia or hypoxia is also essential. These injuries should be recognised by means of a clinical examination and treated immediately (Greaves & Johnson 2002: 29).

2.3.6.2 Circulation

Greaves and Johnson (2002: 29) maintain that shock in a trauma patient should be considered to be caused by haemorrhage until proven otherwise. According to both the ATLS manual (American College of Surgeons 2004: 76) and Gutierrez (2004: 376), the circulation priorities include the control of obvious haemorrhage, the assessment of tissue perfusion, and adequate intravenous access. Bleeding from wounds may be stopped by applying direct pressure to the wounds with other interventions to stop massive internal bleeding being considered if safe. The emphasis on the management of haemorrhagic shock should be on the control of the haemorrhage (Greaves & Johnson 2002: 29; Spahn, Cerny, Coats, Duranteau,

Fernandez-Mondejar, Gordini, Stahel, Hunt, Komadina, Neugebauer, Ozier, Riddez, Shultz, Vincent & Rossaint 2007: [2]).

In addition to the control of haemorrhage it is essential that volume replacement also take place. Halaas (2006: [2]) describes the placement of two large, bore IV lines for fluid resuscitation and beginning with one litre of a crystalloid solution such as Ringers lactate for adult patients. In an article written by Shafi and Kauder (2004: 38), they maintain that two intravenous cannulas, 16 gauge or larger, are generally adequate for resuscitation. However, if the peripheral veins are not accessible then an 8,5 French catheter may be placed in either the femoral vein or the subclavian vein for rapid infusion. The fluid should be warmed to body temperature before infusing so as to enable environmental control and to prevent a lowering of the body temperature.

2.3.6.3 *Disability*

Utilising the Glasgow Coma Scale (GCS)– a widely accepted system for evaluating neurological function (Halaas 2006: [3])– a speedy neurological evaluation should be carried out. This evaluation includes the pupil response, best motor response, as well as a determination of the level of consciousness. Decreased brain perfusion as a result of haemorrhagic shock may mask the same signs and symptoms as intracranial injury in the neurological assessment. It is essential that cerebral perfusion and oxygenation first be restored before any decision is made that that neurological fallout is as a result of intracranial injury (American College of Surgeons 2004: 76). However, the neurological assessment does also contribute as a reference point in conducting future assessments in order determine whether the patient's condition is improving or deteriorating (Greaves & Johnson 2002: 31).

2.3.6.4 *Exposure and environmental control*

Exposure of the patient will ensure that no injuries have been missed. Accordingly, the patient's clothes should be removed or cut off completely. A complete head to toe inspection and assessment, which is known as the secondary survey, should be

carried out (Proehl 2009: 5). The patient should be covered with warm blankets to keep his temperature normal (American College of Surgeons 2004: 76), while fluids, especially blood products, should be warmed before administration in order to prevent hypothermia (Greaves *et al.* 2002: 31).

2.3.6.5 Additional actions

Additional actions that are considered during the “ABCDE” phase of management during haemorrhagic shock include:

- **Gastric decompression:** Gastric distension renders the management of shock difficult and it may lead to aspiration of the stomach contents, with fatal complications. Gastric decompression is performed by inserting a tube into the oesophagus via the nose or mouth and then applying suction to the tube in order to drain the stomach content (American College of Surgeons 2004: 76).
- **Urinary catheter insertion:** The placement of a urinary catheter allows for the continuous monitoring of urine output, which is essential in order to estimate adequate renal perfusion (American College of Surgeons 2004: 77).
- **Vascular access lines:** As earlier discussed, it is essential that vascular access be established as soon as possible by means of two peripheral intravenous subclavian or femoral lines. Blood samples should also be taken in order to ascertain type and cross match of blood, as well as for laboratory analyses. Arterial blood gas analyses should also be taken and essential radiological investigations carried out (American College of Surgeons 2004: 77).

2.4 FLUID RESUSCITATION

Shafi and Kauder (2004: 38) discuss the importance of fluid resuscitation during haemorrhagic shock. They, together with Spinella and Holcomb (2009: 238), Greaves *et al.* (2002: 29) and Spahn *et al.* (2007: [2]), recognise the importance of early aggressive volume resuscitation, be it blood or other intravenous fluid, in the decrease of mortality and morbidity after trauma. The need for the type and

amount of volume resuscitation is determined by the severity of the shock and may be estimated according to the ATLS categorisation of shock.

According to the ATLS Manual (American College of Surgeons 2004: 78), Gonzales (2008: 151) and Santry & Halam (2010: 233) the volume expander of choice for class II haemorrhage is crystalloid fluids. Ringers lactate solution and normal saline 0,9% are the two most frequently used isotonic fluids. Isotonic fluid is fluid with the same tonicity of that of body cells and, thus, no inflow or outflow of fluid across the cell membrane takes place (*Dorland's pocket medical dictionary* 2001). A review article sited in Kwan, Bunn and Roberts (2003: 6) was not able to find evidence either against or for the recommended guidelines for the use of Ringers lactate and normal saline 0,9% made by the American College of Surgeons (2004: 78). The writers did, however, conclude that colloid fluid remains superior to crystalloid fluid as a result of the fact that colloid fluid is retained in the vascular space for longer than crystalloid fluid, thus increasing the vascular volume for longer (Kwan *et al.* 2003: 3). However, Gonzales (2008: 151) recommends that more clinical studies should be conducted before globally deciding on a preferred resuscitation fluid. In addition, Gutierrez *et al.* (2004: 378) maintain that a preferred resuscitation fluid has not yet been established. There remain limitations and side effects in respect of the use of both colloid fluid and crystalloid fluid (see Table 2.3).

Table 2.3: Fluid replacements in haemorrhagic shock

Crystalloids		
Fluids	Advantages	Disadvantages
0,9% Sodium chloride (NaCl) solution	Widely available and not expensive	Requires large volume of infusion which may cause pulmonary oedema
Lactated ringers	Lactate ions help buffer metabolic acidosis	Requires large volume for infusion and may cause pulmonary oedema

Crystalloids		
Fluids	Fluids	Fluids
Hypertonic saline (3%, 5%, 7,5%)	Small volume needed to restore intravascular volume	Danger of hypernatremia
Colloids		
Fluids	Advantages	Disadvantages
Albumin (5%, 25%)	Rapidly expands plasma volume	Expensive, requires human donors, limited supply, may cause congestive cardiac failure
Dextran (40, 70)	Synthetic plasma expander	Interferes with platelet aggregation, not recommended for haemorrhagic shock
Hetastarch	Synthetic, less expensive than albumin, effect lasts 36 hours	Prolonged bleeding and clotting time.

Source: Adopted from Smeltzer *et al.* (2008: 357)

Table 2.3 indicates the different crystalloid and colloid fluids used regularly in the emergency unit for the resuscitation of patients. Although 0,9% sodium chloride (NaCl) solution is widely available and inexpensive, it has been associated with the worsening of lactic acidosis through hyperchloremic acidosis which is caused by massive NaCl 0,9% infusion. Of the two most frequently used crystalloid solutions for the resuscitation of haemorrhagic shock patients, Ringers lactate has proved to be superior to NaCl 0,9%. This is as a result of the fact that the lactate in Ringers solution is metabolised by the kidneys and liver to form bicarbonate which acts as a buffer against lactic acid (Shafi & Kauder 2004: 40). Hypertonic saline is not frequently used because of the danger of causing hypernatremia. Colloid fluids may be defined as fluid which contains large molecules which expand the intravascular volume by pulling fluid into the intravascular space (Smeltzer *et al.* 2008: 367).

Albumin, dextran and hetastarch are all examples of colloid fluids. Albumin is a non-synthetic colloidal solution but it requires human donors. Dextran and hetastarch, on the other hand, are both synthetic colloidal fluid and are not ideal for the resuscitation of haemorrhagic shock.

The types of blood and blood product available consist of uncross-matched Type-O blood for initial resuscitation or until cross-matched blood is available. It is recommended that fresh frozen plasma and platelets be transfused when coagulopathy or hypothermia is present, or when blood transfusion exceeds more than six units. Staff treating patients with haemorrhagic shock should be aware of the dangers of massive blood transfusion. These dangers include hypothermia, coagulopathy, acid/base disturbances, infection risk related to massive blood transfusion and the activation of the systemic inflammatory response syndrome (Shiler & Napolitano 2010: 217).

The use of cell salvage either in theatre or post operatively is becoming increasingly popular. The amount of blood products to be transfused is determined by both the patient's physiological status and reaction to therapy (Shafi & Kauder 2004: 41).

2.4.1 Initial fluid therapy

The dosage of initial fluid therapy recommended by the ATLS Manual (American College of Surgeons 2004: 77) consists of 1–2 litres of crystalloid fluid as a rapid bolus for adults. However, it is difficult to predict the fluid required for a trauma patient with the best guideline being the "3 for 1" rule. This rule implies that each millilitre of blood loss should be replaced with three millilitres of crystalloid fluid.

Gutierrez *et al.* (2004: 374) suggest a simple way in which to calculate the estimated acute blood loss by using the formula presented in Figure 2.4:

$$\mathbf{EBL = EBV \times \ln(Hi/Hf)}$$

- **EBL:** Estimated blood loss
- **EBV:** Estimated blood volume
- **In:** Intravascular space
- **Hi:** Initial haematocrit
- **Hf:** Final haematocrit

Figure 2.4: Estimated acute blood loss

Figure 2.4 illustrates that, in order to calculate the estimated blood loss (EBL), the estimated blood volume (EBV) needs to be considered. An adult's blood volume comprises either 7% of body weight or 7 ml/kg of body weight. Thus, the EBV of an adult male weighing, for example, 70 kg is estimated to be 4,9 litres.

A fluid ratio of three millilitre (ml) of fluid for each ml of blood loss is recommended. However, in cases of uncontrolled haemorrhage, a ratio of 10 ml fluid to every 1 ml blood loss is recommended. Nevertheless, these guidelines are not rigid as the amount of fluid used for resuscitation is, ultimately, determined by the desired end points of resuscitation. The complications of massive fluid resuscitation include increased haemorrhage as a result of hypothermia, dilution of thrombocytopenia, dilution of procoagulant factor, decreased blood viscosity, and the blow out of haemostatic plugs when blood pressure is elevated (Shafi & Kauder 2004:40; Shiler & Napolitano 2010: 217). It is the opinion of both Kwan *et al.* (2003: 1) and Santry and Alam (2010: 229) that, while these resuscitation strategies include the management of blood pressure and tissue perfusion through volume administration, these strategies may actually exacerbate the bleeding. There was inconclusive evidence found both for and against the use of large volume resuscitation as it has proved to be life saving in some patients but detrimental to

others (Kwan *et al.* 2003: 6; Gonzales 2008: 151). According to both Stone and Humphries (2003: [5]) and Garner *et al.* (2010: 1136), the concept of permissive hypotension remains unproven although it must be borne in mind that it requires quick surgical intervention. The goal of resuscitation should be to stop the bleeding and maintain an adequate blood pressure in order to ensure perfusion (Greaves *et al.* 2002: 29; Spahn *et al.* 2007: [2]; Spinella & Holcomb 2009: 238).

The use of blood and blood products is indicated in Class III or Class IV haemorrhage as it will restore circulatory volume, enhance oxygenation, and improve coagulation. Spinella and Holcomb (2009: 235) suggest a transfusion ratio of 1:1:1 of red blood cells, plasma and platelets for ongoing haemorrhage and large quantities of blood loss. The National Institute of Health Consensus Conference recommends that a haematocrit of less than, or equal to, 21%, and a haemoglobin level of 7 g/dl be used as a transfusion trigger point (Shafi & Kauder 2004: 41). However, it is essential that this trigger point be carefully considered in the event of acute blood loss as a result of trauma as initial laboratory results may not adequately reflect a true haemoglobin level.

2.5 EVALUATION OF RESUSCITATION

The vital signs used to diagnose the degree of haemorrhagic shock should also be used to assess the resuscitation efforts of healthcare professionals (American College of Surgeons 2004: 78).

According to articles by Englehart and Schreiber (2006: 573), Husain *et al.* (2003: 489), Porter and Ivatury (1998); Schulman (2002: 32cc8); Shirley (2007: 192) and Shoemaker and Beez (2010: 5), research has proven that, once uncompensated shock has been treated, a significant number of patients still suffer from inadequate tissue perfusion despite the fact that the clinical signs are normal. These patients may suffer from persistent compensated shock, inadequate tissue perfusion and even death unless the clinical end points are taken into consideration.

2.5.1 Vital signs to assess resuscitation efforts

Bilkovski, Rivers and Horst (2004: 530) maintain that the normalisation of vital signs, including heart rate, blood pressure and urine output, should be considered to be the macro or clinical end points of resuscitation. End points of resuscitation refer to those parameters that are used to estimate whether or not resuscitation has been successful and whether shock has been corrected (Shafi & Kauder 2004: 39).

A review article written by Porter and Ivatury (1998) indicates that shock was previously defined by using its clinical markers. These clinical markers include the vital signs, namely, blood pressure, pulse rate and urine output, and they were used to determine when the end point of resuscitation had been reached, namely, successful resuscitation. The above-mentioned clinical end points are still useful when assessing a patient in uncompensated or Class IV haemorrhagic shock (Shoemaker & Beez 2010: 5). However, these traditional end points are not adequate in identifying ongoing oxygen debt as a result of hypoperfusion, and more physiological end points are needed to identify the reversal of shock by monitoring the restoring of both oxygen debt and aerobic metabolism and the alleviating of tissue acidosis (Bakker 1999: [2]; Barbee *et al.* 2010: 113; Shoemaker & Beez 2010: 5; Tintinalli *et al.* 2004: 224).

Uncompensated shock presents with markedly high pulse rate, blood pressure below, or equal to, 90 mmHg systolic, and no urine output (American College of Surgeons 2004: 74-75; Urden *et al.* 2006: 1013). When the clinical end points are normalised it becomes plain to see that the patient is no longer in uncompensated shock although the patient may still have an oxygen debt which may lead to anaerobic metabolism, the formation of lactic acid and increased mortality (Rivers, Ander & Powell 2001: 204; Shoemaker & Beez 2010: 5).

2.5.2 Metabolic markers

Both Engelhart and Schreiber (2006: 569) and Tisherman *et al.* (2004: 898), identify metabolic parameters, such as serum pH, lactate, base deficit and bicarbonate, as markers which may be used to identify metabolic acidosis in shock. In other words, these metabolic markers help to identify compensated shock before it progresses to uncompensated shock. A change in the acid-base balance in arterial blood is often picked up before any change in cardiac output as a result of haemorrhage is noted.

Bilkovski *et al.* (2004: 530) and Tisherman *et al.* (2004: 899) validate the use of the metabolic end points of resuscitation as being metabolic indicators which indicate whether critical oxygen delivery to tissue is taking place. According to them, resuscitation end points should be uniform in their goals so as to avoid under- or over-resuscitation and, in addition, they need to serve as a basis for the comparison of the outcome of resuscitation. Metabolic end points are, thus, part of a road map to restore normal oxygen delivery to tissue. (Bilkovski *et al.* 2004: 354). Other writers, such as Bakker (1999: [6]); Porter & Ivatury (1998); Shafi and Kauder (2004: 39) and Schulman (2002: 32cc8) all agree with Bilkovski *et al.* (2004: 354). The studies that have already been discussed all emphasise that these end points of resuscitation are more valuable as trended values than as isolated figures. The most commonly used end points of resuscitation include:

- ⇒ Base deficit
- ⇒ Bicarbonate level
- ⇒ Central venous oxygen saturation
- ⇒ Lactic acidosis

2.5.2.1 **Base deficit**

Base deficit is an indicator of tissue hypoxia with a worsening in the arterial and venous blood's base deficit, indicating a worsening of tissue hypoxia. Base deficit may be used to identify when simple haemorrhage is deteriorating and may cause

haemorrhagic shock. The haemorrhagic shock is characterised by a worsening base deficit due to increasing tissue hypoxia (Tisherman *et al.* 2004: 905).

Schulman (2002: 32cc4) is of the opinion that base deficit represents the amount of base solution needed to neutralise one litre of acidic blood. Accordingly, the greater the patient's acidosis, the larger the base deficit. Base deficit is easily obtained from an arterial blood gas (ABG) analysis. Tisherman *et al.* (2004: 905) maintain that a base deficit – or excess (BE) – value of –2 to –5 reflects mild hypo perfusion, a value of –6 to –15 moderate hypo perfusion, and above –15 severe hypo perfusion. Engelhart and Schreiber (2006: 570) go on to state that the base deficit may be regarded as a marker of the severity of injury and that it correlates with mortality. Base deficit is effective in road mapping the efficacy of resuscitation although it does also need to be seen as part of the metabolic parameters. If the base deficit remains elevated during volume administration, then it is a sign of ongoing tissue hypoxia (Barbee *et al.* 2010: 115; Tisherman 2004:906).

2.5.2.2 Bicarbonate level

In a later study by Engelhart and Schreiber (2006: 571) they identified additional metabolic markers for resuscitation with bicarbonate levels being said to act as an indicator of worsening metabolic acidosis. Bicarbonate serves as a buffer for the hydrogen ions that are released during anaerobic metabolism. Accordingly, bicarbonate levels decrease as acidosis becomes worse. Bicarbonate values of 17.7 and 17.5 were associated with mortality while levels of 19.8-20mEq/l were found in surviving patients (Engelhart & Schreiber 2006: 571).

In articles by Engelhart and Schreiber (2006: 571) Porter and Ivatury (1998) on the optimal end points of resuscitation, it was recommended that these metabolic end points be reached at least 24 hours after injury. They also suggest that emergency practitioners use at least one of these metabolic end points with which to guide resuscitation. Engelhart and Schreiber (2006: 575) also reviewed the literature on regional end points of resuscitation, namely, capnometry and near

infrared spectroscopy. However, these methods of evaluating resuscitation are not used in all emergency units.

2.5.2.3 Central venous oxygen saturation

Central venous oxygen saturation measurement refers to the measurement of venous blood in terms of which the balance between oxygen consumption and oxygen delivery is reflected. This value may be used as part of the goal-directed therapy strategies. Goal-directed therapy refers to resuscitation strategies which are directed at specific physiological end points which indicate that organ perfusion has been restored (Bilkovski *et al.* 2004: 534).

The use of central venous oxygen saturation (SvO₂), as well as central venous pressure monitoring in haemorrhagic shock patients, is still under investigation with observational studies only having been carried out. Nevertheless, these observational studies do appear to be promising (Kane *et al.* 2007: 68; Tisherman *et al.* 2004: 904).

In a review study conducted by Rivers, Ander and Powell (2001: 210), it was found that SvO₂ of less than 60% was associated with heart failure and shock. The results of a study conducted by Rady, Rivers and Nowak (1996: 218) showed that the evaluation of lactate and base deficit levels alone is not a sufficient means of evaluating the end points of resuscitation. They found that shocked patients who had been resuscitated to normal vital signs continued to manifest a high lactate level and low SvO₂ which both indicate anaerobic metabolism and an oxygen debt. These findings gave rise to the evaluation of SvO₂ in trauma patients. It is suggested that a patient suffering from haemorrhage with a SvO₂ of less than 65% requires additional resuscitation. In the literature review conducted by Rivers *et al.* (2001: 208) it was concluded that SvO₂ is a better indicator of tissue hypoxia than vital data, and that it may be beneficial in the evaluation of therapeutic interventions in the resuscitation of shocked patients.

2.5.2.4 Lactic acidosis

Hypoxia as a result of inadequate tissue perfusion causes anaerobic metabolism that, in turn, gives rise to lactic acid. The interpretation of lactic acid serves as an indirect indicator of the imbalance of oxygen delivery and oxygen consumption by tissue (Schulman 2002: 32cc4; Shirley 2007: 195). The use of lactate levels as an indicator of tissue hypoxia has been used frequently in different settings and the prognostic value thereof has been well documented (Englehart & Shreiber 2006: 570; Jansen, van Bommel & Bakker 2009: 2827; Schulman 2002: 2cc4; Shirley 2007: 195).

Lactic acidosis is the result of increased lactate production that exceeds lactate use or metabolism (Nichol, Egi, Pettila, Bellomo, French, Hart, Davies, Stachowski, Reade, Bailey & Cooper 2010: [1]). Lactic acidosis may be divided into two types, namely A and B. Type A refers to lactate production in terms of which tissue hypoxia is present, while type B is characterised by both normal tissue oxygenation and an impaired lactate usage (Tintinalli *et al.* 2004: 29).

It has been found that lactate trends over time are highly predictive of mortality (Englehart & Schreiber 2006: 570; Husain *et al.* 2003: 485; McNelis *et al.* 2001: 481; Nichol *et al.* 2010: [2]; Schulman 2002: 32cc4). Lactate is used extensively to guide the resuscitation of patients in shock with the normal value for lactic acid being below 2 mmol/L (Englehart & Schreiber 2006: 570; Nichol *et al.* 2010: [1]). A review of trauma patients also studied by these researchers showed that patients whose lactate was normalised within 24 hours manifested 0–10% mortality, while those whose lactate was normalised only after 48 hours manifested a mortality rate of 80–86%. Tisherman *et al.* (2004: 906) documented a 100% survival rate for patients whose lactate levels were normalised within 24 hours, a 25% mortality rate for patients whose lactate levels were normalised only between 24 to 48 hours and an 86% mortality rate for those patients whose lactate was normalised only after 48 hours. Despite the fact that the cut-off value for lactate has not been clearly identified, Schulman (2002: 32cc4) and Nichol *et al.* (2010: [7]) note that, when lactate levels continue to increase, the source of shock has not yet been

eliminated. On the other hand, if the lactate levels decrease then shock is reversing.

The use of lactate as an end point of resuscitation is described by Englehart and Schreiber (2006: 570), Jansen *et al.* (2009: 2827) and Schulman (2002: 2cc4). Lactate may be used as an alternative or additional metabolic end point in respect of other physiological and metabolic end points in order to evaluate resuscitation. However, its use in the emergency unit is limited as a result of both a lack of data from emergency units on the use of lactate as a metabolic end point of resuscitation and a lack of availability of tests verifying its usefulness (Nichol *et al.* 2010: [6]; Tisherman *et al.* 2004: 907; Jansen *et al.* 2009: 2835). The use of serial lactate levels to assess resuscitation has been proposed by Englehart and Schreiber (2006: 570), Jansen *et al.* (2009: 2827), Nichol (2010: [7]) and Shirley (2007: 195). These writers all propose that lactate values which remain elevated despite resuscitation strategies indicate that additional steps should be taken in order to improve tissue perfusion.

Shirley (2007: 195), together with Jansen *et al.* (2009: 2835), Nichol *et al.* (2010: [6]) and Tisherman *et al.* (2004: 907), comment on the prognostic value of lactic acid in predicting mortality. These writers are of the opinion that, irrespective of the cause of increased lactic acid, lactic acid is always associated with a higher mortality rate. The lactate value on admission correlates strongly with outcome and serial blood lactate levels are useful in calculating the prognosis of a patient. If lactate is decreased within the first 24 hours then outcome may be beneficial while prolonged lactate clearance time is associated with a poor outcome. The recognition of high lactate values early in resuscitation is essential to improve the outcome of patients (Nichol *et al.* 2010 [8]; Shirley 2007: 195; Tisherman *et al.* 2004: 906). With reference to the importance of admission serum lactate levels in an emergency unit, these levels may be regarded as an instrument with which to identify patients with serious injury or illness immediately, to predict their survival and to guide resuscitation efforts (Shirley 2007: 194).

2.5.3 Specific considerations

However, there are also various other factors which influence the outcome of patients presenting with haemorrhagic shock after resuscitation. Specific factors which should be taken into account during the evaluation of resuscitation efforts include age, pregnancy and pain response (see Sections 2.5.2.1 to 2.5.2.3).

2.5.3.1 Age

Trauma is one of the leading causes of death in paediatric patients with blunt trauma accounting for 80 to 95% of injuries and the remainder of injuries being caused by penetrating trauma. In view of the anatomical and physiological differences between paediatric patients and adults, the order and frequency of their injuries also differ. Paediatric patients most often sustain injuries to the head, then to the musculoskeletal system, the thorax and, lastly, the abdomen (Jacobs & Hoyt 2000: 251). These differences in the paediatric anatomy and physiology compared with that of adults mean that the management process also differs and adult guidelines do not apply to paediatric patients.

Athey, Dean, Ball, Wiebe and Melese-d'Hospital (2001: 170) state that the needs of children involved in trauma are different to those of adult patients, and that children suffer from other types of disease and injury compared to adults. In light of these factors paediatric patients require unique resuscitation strategies and proper emergency care which are the result of proper paediatric emergency training. The paediatric patient's physiological and emotional response to trauma also differs from that of the adult. This implies that the equipment and facility requirements also change in the case of paediatric patients (Athey *et al.* 2002: 173).

It is evident from the epidemiology that trauma is among the top four leading causes of death in all these countries while, in most of these countries, trauma is the leading cause of death for people between the ages of one and 44 years (Kane

et al. 2007: 61; Kauvar *et al.* 2006: S3; Garner *et al.* 2010: 1131). Articles cited in Dick and Baskett (1999: 88) conclude that patients above the age of 44 years sustain more serious injuries caused by trauma while fatalities double for patients above the age of 75 years. Post-traumatic complications are also more severe, characterised by longer hospital stays, CCU admissions and an increased risk of complications in the elderly.

2.5.3.2 *Pregnancy*

Pregnant females are as exposed to trauma as any other person who participates in all normal daily activities, with trauma being the leading cause of unnatural death during pregnancy (Colburn 1999: 22–23).

In view of a number of hemodynamic and physiological changes in respect of almost all the organs during the three trimesters of pregnancy, it is suggested by Colburn (1999: 23) that, in the case of the pregnant mother, routine trauma practices be changed. Together with a perinatal team, these routine trauma practices should, where possible, strive to attain the common goal of the stabilisation of the mother, without compromising the fetus (Ruffolo 2009: 704).

2.5.3.3 *Pain response*

The pain response refers to a physiological response to actual and potential tissue damage. Pain management in the emergency unit is often not carried out adequately. This failure may be as a result of a lack of knowledge of pain management on the part of the healthcare providers or a fear in respect of the side effects to pain management such as hypotension and respiratory failure (Jacobs & Hoyt 2000:449). The source of pain may be injuries such as fractures and lacerations or else the pain may be as a result of procedures such as IV line insertion and wound care. Pain may also be caused by painful stimulation of various areas of the body. In addition, the source of pain may be the skin or subcutaneous tissue, muscles, joints, tendons, blood vessels or organs (Jacobs & Baker 1995:495).

The body responds physiologically to pain and this response may be seen in an increase in the heart rate and/or an increase in the force of the contraction of cardiac muscle (Victorino *et al.* 2003: 679). Such a response will elevate both the patient's pulse rate and the strength of the pulse. Physiological pain responses may also cause peripheral vasoconstriction and the release of adrenal gland hormones that increase the blood pressure (Jacobs & Hoyt 2000:449). These physiological responses to pain may mimic physiological responses to both blood volume loss and to hypovolaemic shock.

2.6 CONCLUSION

Trauma is the leading cause of death in patients between the ages of one and 44 years in the RSA. In addition, compensated and uncompensated haemorrhagic shock is frequently under-diagnosed in trauma patients and this has a definitive effect on the mortality and morbidity of these patients. These facts demonstrate the necessity of improving resuscitation strategies in order to improve the outcome of patients presenting with haemorrhagic shock.

The use of end points of resuscitation, especially lactate values, in the emergency unit may prove beneficial in the effective resuscitation of trauma patients. Special attention should be paid to the age and physiological status of patients during resuscitation. The consequences of delayed resuscitation in haemorrhagic shock patients are associated with an increase in mortality and morbidity although this increase is preventable.

Chapter 3 provides an in-depth discussion of the research method which was used in the research.

3 RESEARCH METHOD

3.1 INTRODUCTION

Chapter 2 provided an in-depth literature review of relevant topics as they applied to this study. The literature review focused on haemorrhagic shock and the evaluation of resuscitation efforts.

Chapter 3 contains a detailed discussion of the research method used in this study. The research method refers to the research design and methodology used in a study. As suggested by Polit and Beck (2004: 48), the research design been used to plan the research method (see Chapter 1, Table 1.1). The research design (see Section 3.3.1) and research methodology (see Section 3.3.2) are also discussed in depth.

In other words the chapter focuses on both the research design used and the research methodology implemented in order to address the research question, as well as the aim and objectives of the study. In addition, the ethical considerations as they relate to the study are delineated.

3.2 AIM AND OBJECTIVES

The overall aim of this study is to determine the correlation between the 24-hour serum lactate levels and the 48-hour mortality rate of patients presenting with haemorrhagic shock in the emergency unit of the hospital.

In order to achieve this aim, the objectives of the research are to

- o audit the serum lactate levels as indicted in patients' files on admission, as well as during a 4 to 8 hour, 10 to 14 hour and 22 to 26 hour timeframe following haemorrhage caused by trauma

-
- o document the 48-hour mortality rate in patients presenting with haemorrhage caused by trauma
 - o determine the relationship between the serum lactate levels and the 48-hour mortality rate in patients presenting with haemorrhage caused by trauma
 - o make recommendations for future resuscitation strategies for patients presenting with haemorrhagic shock in order to improve the outcome of such patients

3.3 RESEARCH DESIGN

A research design refers to the blueprint for conducting a study. The aim of this blueprint is to maximise control over factors that may interfere with the validity of the findings (Burns & Grove 2005: 211). In other words, the research design guides the researcher in planning and implementing the study so as to achieve the set goals. A research design is referred to by Polit and Beck (2006: 509) as a “general plan for addressing research questions, including specifications for enhancing the studies’ integrity”. Green and Thorogood (2004: 34) refer to the research design as “the what, how and why of data production” in order to answer the research question. Accordingly, the research design refers to the way in which the researcher will address the research question (Burns & Grove 2005: 211; Polit & Beck 2004: 731).

A quantitative, retrospective, non-experimental, descriptive, correlational and cross-sectional research design was chosen for this study. Each of these will be discussed in-depth in Chapter 3, Sections 3.3.1–3.3.6 The design chosen is consistent with the paradigm and assumptions on which this study is based (see Sections 1.7.3 and 1.7.4).

3.3.1 Quantitative design

Quantitative research is a formal, objective and systematic process for generating information about the world (Burns & Grove 2005: 26). In addition,

this method of research is used to describe variables, examine relationships among variables and determine cause–effect interactions between variables. Quantitative research is currently the method of choice for scientific investigations in healthcare practice. It must be remembered that it requires rigorous control in order both to identify and to limit the effects of extraneous variables not under study (Burns & Grove 2005: 26).

According to Polit and Beck (2004: 15), quantitative research refers to a set of orderly and disciplined procedures aimed at acquiring knowledge. This research study is based on a traditional, positivistic and scientific method which was used in order to conduct the research. Quantitative research is systematic and, thus, the researcher follows a logical series of steps according to a set plan of action. The researcher is able to implement a degree of control over the study and this control ensures minimum bias and a high degree of validity. Quantitative studies rely on empirical evidence – evidence from an objective viewpoint that is gathered by the senses, for example taste, smell, sight, touch and hearing. Empirical evidence is grounded in reality and, as such, does not form part of the researcher’s beliefs, unlike qualitative studies.

In quantitative research, specific questions generate knowledge which directly enhances the understanding of the phenomenon under study. In this research the phenomenon being studied was the serum lactate levels as indicated in patients’ files on admission and, if available, during a 4 to 8 hour, 10 to 14 hour and 22 to 26 hour timeframe following trauma. During a quantitative study, evidence is gathered according to a specified plan with formal instruments being used to collect the required information (Somekh & Lewin 2005: 215).

The data were documented in terms of the clinical audit checklist and analysed by a professional statistician using the Statistical Package for the Social Sciences (SPSS), version 14.0, micro-computer program. Frequencies were determined for the responses to all the data collected. In addition, one of the strengths of quantitative design, as described by Yauch and Steudel (2003: 473), that it facilitates comparison, was taken into consideration with the objective of the study being to compare 24-hour serum lactate levels with the

48-hour mortality of patients admitted with haemorrhagic shock to the emergency unit.

3.3.2 Retrospective design

A retrospective design is defined by Polit and Beck (2004: 731) as “a study design that begins with the manifestation of the dependant variable in the present, and then searches for the presumed cause occurring in the past”. The dependant variable refers to that attribute being studied that is subject to change as a result of another attribute (independent variable) which influences the dependant variable (Polit & Beck 2004: 716).

Retrospective design is regarded by Burns and Grove (2005: 234–235) as part of the time-dimensional designs in terms of which the cause and effect that is proposed has already taken place. Retrospective study designs are important in nursing studies because a pattern of reaction to interventions is identified that influences nursing actions (Burns & Grove 2005: 235).

Retrospective research requires the analysis of data which were not necessarily collected for research purposes (Gearing, Mian, Barber and Ickowicz 2006: 126). Retrospective data include clinical administration data such as laboratory results, nursing notes, emergency room files and documentation on special investigations. The use of a retrospective chart review is believed to comprise 25% of all scientific articles written in emergency medical journals (Gearing *et al.* 2006: 126).

For the purpose of this study the files of patients were retrospectively investigated and information relevant to the study captured on a data sheet and then analysed by a professional statistician.

3.3.3 Non-experimental design

Retrospective design stems from non-experimental, quantitative research (Polit & Beck 2008: 272). Non-experimental design is most commonly used in nursing research as a result of the fact that most human attributes are not subject to change and it is, thus, not possible to experiment on them. Even if it were possible to manipulate these human attributes this would not always be ethical, thus making non-experimental research design the design of choice (Polit & Beck 2004: 188).

A non-experimental study design was used in this research. This type of study design implies that none of the human attributes presented on the patients' files was used as part of the data collected for the purposes of the research. This was done in order to protect the anonymity and ethical rights of the patients concerned.

3.3.4 Descriptive design

As suggested by Burns and Grove (2005: 248), descriptive designs help to identify challenges in current practice with a view to improving the practice outcomes for patients. When a study is not structured formally as an analytical or as an experimental study, thus implying that the study does not aim specifically to test a hypothesis, such a study is termed a descriptive study and it belongs to the observational category of studies (WHO 2001b: 16).

In descriptive studies the researcher's intention is to "portray an accurate picture of reality" (Stommel & Wills 2004: 437). Descriptive research may also be described as being "concerned with the current status of something and 'asks' what is and reports things as they are" (McMillan & Schumacher 2001: 283).

The purpose of descriptive research is "to observe, explore, describe and document aspects of a situation as it naturally occurs" (Polit & Beck 2006: 192). Descriptive studies are characterised by little or no researcher control.

Subjects are examined as they exist in their natural settings, for example, in their homes, in hospital or at work (Burns & Grove 2005: 28). Descriptive research is "... the exploration and description of a phenomenon in real-life situations. It provides an accurate account of characteristics about particular individuals, situations or groups" (Burns & Grove 2005: 24). The outcomes of descriptive research include the description of concepts, identification of relationships and development of hypotheses that may provide a basis for future quantitative research (Burns & Grove 2005: 25).

This study aimed to investigate and to describe the serum lactate levels as indicated in patients' files on admission as well as during a 4 to 8 hour, 10 to 14 hour and 22 to 26 hour timeframe following trauma. The study also aimed to describe the 48-hour mortality rate of patients admitted to the emergency unit with haemorrhagic shock.

3.3.5 Correlational design

Non-experimental research consists of two broad classes, namely, correlation or "ex post facto" research and descriptive research. For the purposes of this research a correlational research design was used. A correlational design involves the relationship among variables, namely, the dependant and independent variables without any manipulation of the variables. In Latin, correlational design is referred to as *ex post facto* which means 'after the fact'. In other words, the data is collected after the phenomena have taken place, and after the dependant variable has caused the independent variable (Polit & Beck 2004: 188).

Correlation research establishes whether there is a relationship between two phenomena or variables. Novice researchers often make the mistake of trying to establish causality between variables – as is the case in experimental and quasi-experimental research – rather than simply recognising a relationship between two variables (Burns & Grove 2005: 239).

In this research the relationship between the two variables, namely, 24-hour serum lactate levels and the 48-hour mortality of patients admitted with haemorrhagic shock to the emergency unit were investigated. A correlational design was used as the researcher did not intend to manipulate the independent variable. Instead, the variable in this research was studied after the incident had taken place, and thus, as confirmed by Polit and Beck (2004: 189), manipulation or control of the variable was exceedingly difficult. Other measures which may be applied in order to minimise control over variables in a correlational study include the use of a large, representative sample for the purposes of the research. In this study it was not possible to obtain a large sample, although representativeness was assured by including inclusion and exclusion criteria for the sample selected for the research (see Chapter 3, Sections 3.4.1). In terms of correlational design it is regarded as important to consider the entire spectrum of outcomes from the variables represented in the sample, thus ensuring a representative sample, as the variance in respect of the variables is not controlled as in experimental study designs (Burns & Grove 2005: 239).

3.3.6 Cross-sectional design

Cross-sectional studies entail the collection of data from a cross-section of the population. This cross-section of the population may comprise the whole population or a proportion (sample) of it (WHO 2001b: 17). In addition, Somekh and Lewin (2005: 216) point out that a cross-sectional study involves the collection of quantitative data in respect of at least two variables at a single point in time and from a number of cases.

The fact that the data for this research were collected from patients' files correlated with the inclusion and exclusion criteria for the research. The study adhered to the variable that the patients selected were all adult patients entering the emergency unit of the hospital concerned and presenting with haemorrhagic shock (see Chapter 3, Section 3.4). This meets the criterion for a cross-sectional study that specifies that the data collected must fit at least two variables. The cross-sectional design also specifies that the data must be

collected at a point in time and from a number of cases. The data in this research were collected retrospectively from a sample representing the study population, which consisted of 46 patients' files for the study period of January 2008 to June 2008 (see Chapter 3, Section 3.4.1.1).

3.4 RESEARCH METHODOLOGY

Research methodology refers to the application of all the steps, strategies and procedures for gathering and analysing data in a research investigation in a logical and systematic way (Burns & Grove 2005: 26). The selection of the research methodology or strategy forms the core of a research design and is probably the single most important decision the investigator makes in terms of the research.

Offredy and Vickers (2010: 65) refer to the methodology in research as a systematic way of gathering data from a given population so as to understand a phenomenon and to generalise facts obtained from a large population. Methodology embraces the research design, population, instruments used to collect data, ethical considerations, as well as the data analysis and its interpretation. Accordingly, methodology helps both the researcher and the reader to understand the process of the research, thus according the research scientific merit.

The relevant steps in the research method that form part of this chapter and as applied to this research will be discussed in terms of the sample plan, data collection instrument, data collection and data analysis.

3.4.1 Sample plan

A sample plan is defined by Polit and Beck (2004: 731) as a plan that describes the sample method, the size of the sample and the procedure followed to select the sample. A sample frame is developed to ensure that the

sample that is selected is representative of the population. Figure 3.1 summarises the sample plan that was used in this study.

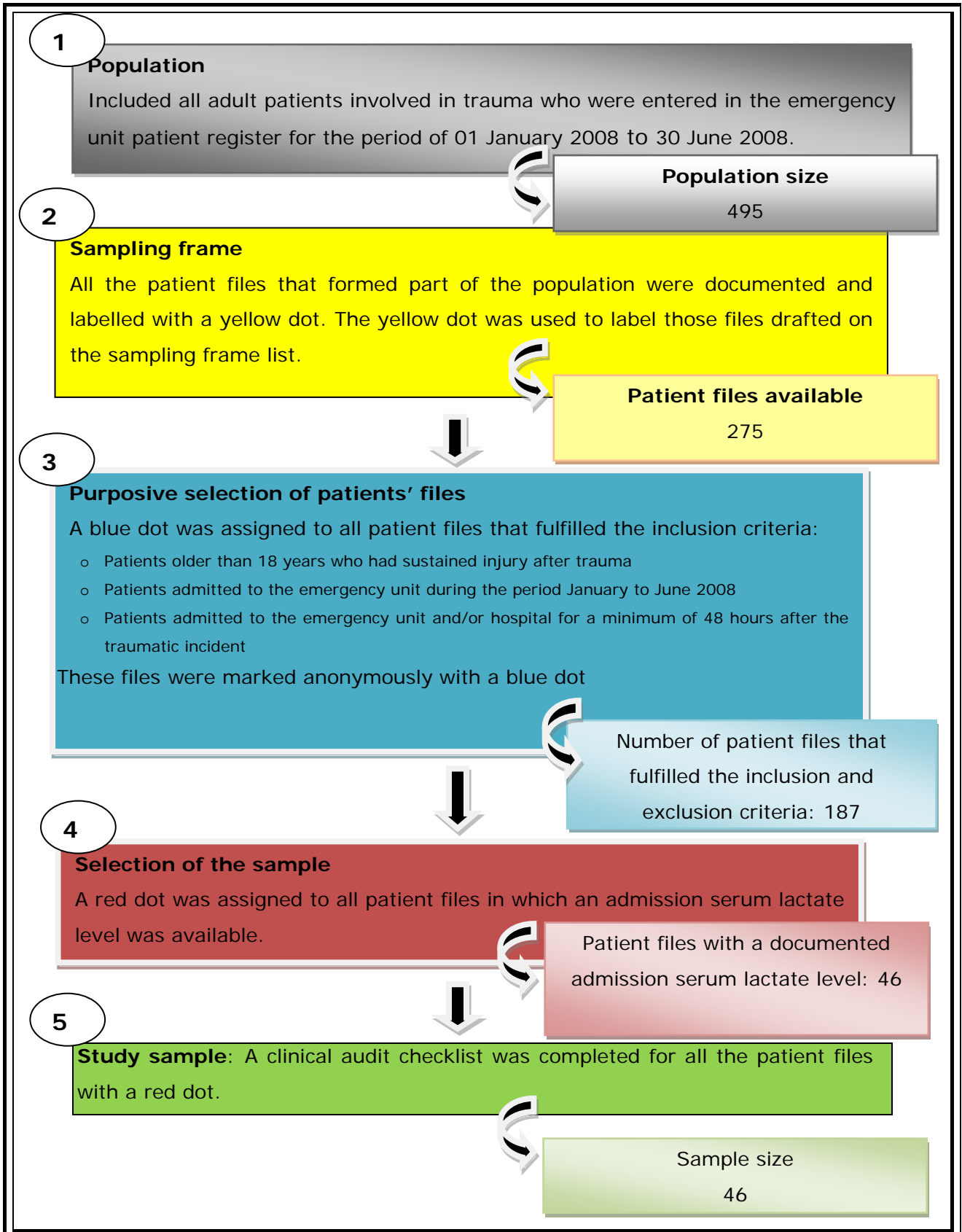


Figure 3.1: Summary of the sample plan

Figure 3.1 illustrates the process used to select those patients' files which were chosen as part of the sample. The sample plan consisted of a few basic steps, namely, the 1) population, 2) sampling frame, 3) purposive selection of patients' files, 4) selection of the study sample and 5) completion of clinical audit check lists for those files that formed part of the study sample.

3.4.1.1 Population

The population refers the entire set of individuals with some common characteristic(s). The accessible population comprises those individuals who conform to the eligibility criteria and who are available for a particular study (Burns & Grove 2005: 342; De Vos, Strydom, Fouche & Delport 2002: 198). In addition, Polit and Beck (2006: 259) are of the opinion that a population refers to the entire aggregate of cases that meet specified criteria. Bryman (2004: 85), on the other hand, describes a population as the totality of persons, events, organisation units, case records or other sampling units from which the sample is selected and with which the research problem is concerned. The population may also be regarded as the group to which results of a study are generalised (Trochim 2006). Somekh and Lewin (2005: 217) refer to a population as the "complete set of units being studied when time, costs and accessibility often prohibit the collection of data from every member or about every item".

The population for this study included the files of male and female trauma patients who presented with haemorrhagic shock and who were admitted to the emergency unit of the hospital during the study period. The population selection process commenced with the decision on the population of the research. The population was chosen by reviewing the admission book of the emergency unit and then selecting the records of patients entering the emergency unit from January 2008 to June 2008. A total of 495 files were identified.

3.4.1.2 Sampling frame

Once the study population has been identified, the accessible population is listed and the sample is drawn from this. This is referred to as a *sampling frame* (Trochim 2006). The sampling frame for this study was established by

noting down all the files identified, and assigning a random number to each file in order to maintain confidentiality. In order to insure the anonymity of the subjects, no personal information obtained from the files was used as part of the sampling frame. For the purposes of this study a sampling frame was drawn which included a list of the files from which the sample was chosen. From the 495 files identified, 275 only were accessible from the record department. The files which could not be obtained had either been lost or misplaced, thus making them impossible to access.

3.4.1.3 Purposive selection of files

The purposive selection of a sample is described by Polit and Beck (2004: 294) as the selection of a sample based on the researcher's knowledge of the population. The sample is selected according to criteria which will ensure that the sample is representative of the entire population. The purposive selection of the sample is a subjective sampling method and it has proved to be beneficial should the researcher be aiming to select a sample which is of specific interest to the research topic (Polit & Beck 2004: 295).

In this study, the sample was selected on the basis of inclusion and exclusion criteria, thus making it representative of the population. The sample frame was drafted, and files were then purposively selected according to the inclusive and exclusive criteria. A total of 187 patient files from the population were purposively selected as part of the sample selection process.

Based on the discussion in "Background to the problem" (see Chapter 1, Section 1.2), in which the arguments of reputable researchers such as Bakker (1999: [2]), Barbee *et al.* (2010: 113), Englehart and Schreiber (2006: 573), Husain *et al.* (2003: 489), Porter and Ivatury (1998), Schulman (2002: 32cc8) and Shoemaker and Beez (2010: 5) were described, the inclusion criteria for the patients' files that were selected included the following:

- Patients who had been admitted to the emergency unit of the hospital
- Files that were available for the period January 2008 to June 2008
- Patients who were 19 years or older

-
- Patients presenting with haemorrhagic shock as a result of traumatic blood loss
 - Patients admitted to the emergency unit and/or hospital for a minimum period of 48 hours following the traumatic incident.

The exclusion criteria for selecting the files were formulated and incorporated patients with the following:

- **Head injuries:** Patients with a confirmed head injury and in respect of whom the management had been taken over by a neurosurgeon were excluded. These patients were not included in the population for the study in view of the severe physiological changes that may take place in patients with head injuries. McQuillan *et al.* (2009: 394) are of the opinion that patients who survive the initial brain injury are, nevertheless, at risk of a number of complications affecting various systems which, in turn, enhance the mortality and morbidity of such patients.
- **Pregnant females:** Pregnant females were not included in the study population because of the physiological changes which take place during pregnancy (Colburn 1999: 23). The treatment of pregnant patients presenting with trauma would not be the same as that of other trauma victims, and their outcomes are usually influenced by the pregnancy (Ruffolo 2009: 704).
- **Younger than 19 years:** According to Athey *et al.* (2001: 170), the needs of patients younger than 19 years of age (infant, child and adolescent) and involved in trauma differ from those of adult patients. These patients require unique resuscitation strategies as their physiological and emotional responses to trauma differs from those of adults. Accordingly, the equipment and facility requirements also change when dealing with this age group. *Dorland's Pocket Medical Dictionary* (2001: 18) defines adolescence as the period between puberty (after childhood) and the completion of physical growth. This study was conducted using adult patients' files and, thus, the paediatric patient (0–10 years) and the adolescent (11–18 years) were not included in the research.

3.4.1.4 Selection of the sample

Sample selection is described by Polit and Beck (2004: 731) as the process in terms of which a portion of the population is chosen that is representative of the

entire population. In this study, the files selected in the sampling process were patient files that indicated an admission lactate level and that were available. These files were assigned a red dot and the clinical audit checklists were completed for all these files. Serum lactate levels were also obtained for a 4 to 8 hour, 10 to 14 hour and 22 to 26 hour timeframe following trauma, if available. A total of 46 files with an available admission serum lactate level was used.

3.4.1.5 Sample

A sample comprises a subset of the population selected to participate in a study (Polit & Beck 2006: 260). According to Somekh and Lewin (2005: 218), a sample is studied in order to understand the population from which the sample was drawn. It is both more cost-effective and practical to conduct a study on a sample than on an entire population. Terre Blanche and Durrheim (2004: 44) state that sampling involves decisions about “which people, events, behaviour or social processes are selected and/or observed”. The aim of sampling is to select subjects that will be representative of the population in respect of which the researcher aims to draw conclusions regarding what is being studied (Terre Blanche & Durrheim 2004: 44).

Complete coverage of the population is seldom possible and, even if it were possible, both time and cost considerations would usually make this a prohibitive undertaking. Sampling is, thus, the science and practice of selecting information from populations in a manner that allows defensible inferences to be drawn from the data (Salks & Allsop 2007:157). The use of samples may, therefore, result in more accurate information because, with a sample, time, money and effort may be concentrated in order to produce a higher quality of research information (De Vos *et al.* 2002:199).

For the purpose of quantitative studies – this study – the individuals in the sample are termed subjects (Polit & Beck 2004: 27). However, for this specific research, the subjects themselves were not used for the sample but rather the subjects’ files. A sample is selected through a sampling process. It is important to select a sample that is representative of the population and, hence, which has similar characteristics to the population (Polit & Beck 2004: 291).

The method of sample selection may be based on either a probability or a non-probability approach. For the purposes of the research, non-probability sampling, more specifically, purposive sampling, was used. This method of sampling is often perceived as problematic in that the sample selected may not be representative of the population. Nevertheless, this form of sampling is used extensively in research, especially in quantitative research in terms of which specific traits of a population have to be studied (Polit & Beck 2004: 294).

The researcher opted to use purposive sampling because, in order to address the research question, it was essential that the admission serum lactate level be available in the files of patients who had been admitted to the emergency unit of the hospital. The representativeness of the population in respect of the sample was assured by the undertaking that the sample would adhere to the inclusion and exclusion criteria for the research. This was achieved by the purposive selection of patients' files for the study sample.

⇒ **Sample size**

According to Polit and Beck (2006: 267), sample size is a major issue in conducting and evaluating quantitative research. There is no simple equation to determine the size of the sample needed, although quantitative researchers are generally advised to use the largest sample possible. According to Somekh and Lewin (2005: 218), it is the sample size rather than the relative size or proportion of the population sampled that is the crucial factor. Salks and Allsop (2007: 158) maintain that, the bigger the sample size, the smaller the error in respect of approximating the characteristics of the entire population, despite the fact that it would cost more to administer the survey and analyse the data. The sample size is dependent on the accuracy required and the likely variation of the population characteristics being investigated.

Generally, as the sample size increases proportional to the population size, sampling errors are likely to decrease. If the sampling error is small, the sample is likely to be representative of the population. Representativeness may be enhanced by selecting suitable sample size, taking into account the population

size. If a sample is representative of the population, it may be possible to generalise the research findings to the population as a whole (Burns & Grove 2005: 343–345; Polit & Beck 2006: 299).

This study sample included patients' files that had been selected for the study in order to document the serum lactate levels on admission, as indicated in the files, and, where available, during a 4 to 8 hour, 10 to 14 hour and 22 to 26 hour timeframe following trauma. The sample obtained consisted of 46 patients' files which, in turn, represents 9.29% of the study population.

3.4.2 Data collection

Research data, particularly in quantitative studies, are often collected according to a structured plan which indicates what information is to be gathered and the way in which to gather this information. Structured methods yield data that are relatively easy both to quantify and to analyse. Structured methods also enhance the objectivity of the data collected by eliminating bias as a result of the researcher's personal feelings or beliefs (Burns & Grove 2001: 50). The data collection instrument in a quantitative study relies on the measurement of variables – the assignment of numbers to the number of times the occurrence which is being studied takes place (Polit & Beck 2004: 413).

Measurement refers to the process, based on specific rules, of allocating numbers to objects, events or situations (Burns & Grove 2007: 40). A component of measurement is "instrumentation" which is the application of specific rules to the development of a measurement device or instrument (Burns & Grove 2007: 40).

In this study biophysical measures were used. Biophysical measures refer to the evaluation of the physiological status of subjects which, in turn, stems from the increased patient-centred research which is becoming more apparent in nursing research (Polit & Beck 2004: 321). Biophysical measures are categorised in one of six categories. For the purpose of this study one such category was used and

discussed, namely, the description of the physiological outcomes of nursing and healthcare. Polit and Beck (2008: 440) are of the opinion that these studies do not focus on specific interventions but rather that they are designed to indicate the extent to which standard procedures may influence an individual.

In order to collect the necessary data or information for a study it is essential that researchers develop a measuring instrument in order to ensure uniformity and consistency. According to McMillan and Schumacher (2001: 185) there might be a need to develop a new measuring instrument for research evaluation for a specific setting. In this study a clinical audit checklist was used to collect the data (see Annexure B).

3.4.2.1 Clinical audit checklist

The data collection instrument, namely, a clinical audit checklist, was developed before the data collection had taken place and, as suggested by Burns and Grove (2005: 424), it was developed to suit the type of data that needed to be collected. The data for this study were obtained through a retrospective audit of patients' files.

Clinical audit checklists are one of the recordkeeping tools used to strengthen the research design (Burns & Grove 2005: 395). According to Burns and Grove (2005: 395) checklists comprise a technique which may be used to indicate whether a specific behaviour or an incident has occurred. Each time that a behaviour or incident occurs that is relevant to the study, it is marked on the checklist while all other incidents or behaviours are ignored. Checklists are instruments which may be used by the observer to observe phenomena. The data that are collected in this way are exhaustive and are entered on the checklist at different time intervals (Polit & Beck 2004: 388).

Audits performed on medical records are not a new phenomenon in the medical field and they may, in fact, be extremely beneficial in either legal reviews or case reviews while, at the same time, safeguarding the personal information of patients (McCartney 2009: 64). Clinical audits have not only proved beneficial to patients by contributing to an improvement in patient care, but healthcare

providers have also reported the fact that clinical audits have led to improved team work (Johnston, Crombie, Davies, Alder & Millard 2000: 25). The clinical audit checklists used in this research clearly encompass the same potential benefits for the emergency unit of the hospital.

Johnston *et al.* (2000: 26) identify barriers to clinical audits and classify these barriers into five main categories which include a lack of resources and a lack of expertise. In this study the researcher decided on the information to be included in the clinical audit checklist after consulting the relevant literature and also on the recommendation of a professional statistician. This included parameters proposed by the Utstein technique of recording.

The Utstein recording technique was developed because the treatment of trauma patients both in and outside of the hospital setting required a multidisciplinary team approach. This multidisciplinary team approach made it difficult to establish both a uniform pattern and a uniform set of criteria in respect of the recording of events pertaining to these trauma patients. The European Resuscitation Council, the American Heart Association, the Australian Resuscitation Council and the Resuscitation Council of Southern Africa, among others, have drawn up guidelines for the recording of cardiac arrest in patients, namely, the 'Utstein style' of recording (Dick & Baskett 1999: 83).

In 1994, a group from the International Trauma Anaesthesia and Critical Care Society (ITACCS) met at the 7th Annual Symposium in Paris and decided to develop a documentation system for the recording of trauma patients based on the Utstein style – specifically for the out-of-hospital trauma patient but also including early in-hospital treatment (Dick & Baskett 1999: 83). In 1995, Spaitte proposed that the principals of data collection be based on the stratification of information into both essential and supplementary data (Dick & Baskett 1999: 81). The aim of this proposed stratification of data was to improve the quality of care of, and research into, trauma patients specifically in the pre-hospital environment. In 1998, during the 11th ITACCS Annual Symposium in Vienna, the final document on the Utstein recording style was presented after an urgent need had been identified in 1996 for a uniform terminology and reporting system in respect of trauma patients. Since then the Utstein report has enjoyed

various inputs from different disciplines and organisations worldwide (Dick & Baskett 1999: 83).

Erasmus and Zemlin (2009: 595) emphasise the importance of relevant and precise data in patient files. As a result of the fact that clinical audits are often retrospective in nature (see Chapter 3, Section 3.3.2), it is important to make certain that an adequate and representative sample is used in a study in order to ensure valid conclusions and generalisability of research findings. Clinical audits are used both to improve patient outcomes and the level of general care in the clinical environment (Erasmus & Zemlin 2009: 594).

3.4.2.2 Structure of the clinical audit checklist

In respect of the clinical audit checklist that was developed for this research, a portion of the Utstein style of reporting as well as the recommendations of the professional statistician were used as part of the checklist (see Annexure B). It was decided by the researcher and professional statistician that the clinical audit checklist should comprise three sections, namely:

- Section A: Demographic information
- Section B: 24-hour lactate levels
- Section C: 48-hour mortality

The contents of each section will now be discussed.

⇒ Section A: Demographic information

There was no reference made to name and surname, hospital number, date of admission or race in order to ensure the anonymity of all the patients whose files were used as part of the research.

The demographic information included in the clinical audit checklist comprised the age and gender of the patients whose files were used:

- **Age:** As a result of the fact that age influences the outcome of trauma, due to the physiological response to trauma (Dick & Baskett 1999: 88), the **age** or estimated age – where the actual age was unknown – was included as part of the demographic information. In most countries trauma is seen as

the biggest killer of patients up to the age of 44 years (Kane *et al.* 2007: 61; Kauvar 2006: S3; Garner *et al.* 2010: 1131). For the purpose of this research only patients of 19 years or older were used in accordance with the inclusion and exclusion criteria of both the population and the sample.

- **Gender:** Gender influences the mortality rate and physiological response of patients involved in trauma. Brasel *et al.* (2007: 813) and Dick and Baskett (1999: 89) are of the opinion that the mortality rate resulting from trauma is twice as high in males as in females and, thus, gender was included as part of the demographic information in order to investigate the correlation of the findings of the study with this statement.

⇒ **Section B: 24-hour lactate levels**

As discussed in Chapter 2, Section 2.5.2.4, greater accuracy in the measurement of serum lactate levels aids in the recognition of tissue hypoxia as a result of hypovolaemia. Serum lactate levels normalise more slowly than both the pulse rate and blood pressure in patients suffering from shock caused by haemorrhage. Although a serum lactate level on admission is an important predictor of the severity of injury, raised serum lactate levels after 12 hours are associated with mortality, as specified by Cerovic *et al.* (2003: 1304). The aim of this research is to evaluate the correlation between the 24-hour serum lactate levels and the 48-hour mortality rate of patients presenting with haemorrhagic shock in the emergency unit of the hospital concerned.

Based on the aim and objectives of the research (see Chapter 3, Section 3.2) it was decided to document the serum lactate levels as indicated in patient files on admission as well as during a 4 to 8-hour, 10 to 14-hour and 22 to 26-hour timeframe following trauma. These timeframes concur with both the findings in the literature (Cerovic *et al.* 2003: 1302- 3; Englehart and Schreiber 2006:570 and Schulman 2002: 32cc4) and the recommendations of the professional statistician.

Section B of the clinical audit checklist contained the following time frames, as suggested by Cerovic *et al.* (2003: 1302- 3), Englehart and Schreiber (2006:

570) and Schulman (2002: 32cc4), and in accordance with which the serum lactate levels were measured within a span of 24 hours:

- 0 to 4 hours after admission:
- 4 to 8 hours after admission
- 10 to 14 hours after admission
- 22 to 26 hour after admission

⇒ **Section C: 48-hour mortality**

It has been concluded in various studies that lactate trends over time are predictive of mortality (Husain *et al.* 2003: 486; McNelis *et al.* 2001: 48). A review of trauma patients in the study by Engelhart and Schreiber (2006: 571) showed that patients whose lactate was normalised within 24 hours manifested a mortality rate of 0 to 10%, while those whose lactate was normalised only after 48 hours manifested a mortality rate of 80 to 86%. The cut-off value of lactate has not been clearly identified, although Schulman (2002: 32cc4) did find that, when lactate levels are still increasing, the source of shock has not yet been eliminated. On the other hand, if the levels drop, then the shock and its side effects may be reversed.

Accordingly, it was regarded as important to include the 48-hour mortality as an item on the clinical audit checklist. In addition, this further enabled the researcher to determine the relationship between the serum lactate levels and the 48-hour mortality rate in patients.

The literature suggests that it is important to evaluate the incidence of resuscitation against 24-hour mortality, as this is the time frame in which the largest number of deaths as a result of hypovolaemia occur (Kauvar *et al.* 2006: S4). In the emergency unit of the hospital, the 24-hour mortality rate is not taken into account in the evaluation of the resuscitation of adult patients suffering from shock caused by haemorrhage. Despite the fact that the most significant cause of mortality and morbidity worldwide is haemorrhage, it is, nevertheless, possible to resolve many of the complications caused by haemorrhage through the implementation of both earlier and better

resuscitation strategies, as well as earlier control of haemorrhage (Kauver *et al.* 2006: S7).

3.4.2.3 Pre-test

A pre-test is defined by Polit and Beck (2008: 762) as the collection of data prior to commencement of the research. A pre-test may be regarded as a trial administration of a newly developed instrument in order to identify flaws.

The researcher conducted a pre-test using a study population of 69 patient files. The population used for the pre-test was files of male and female trauma patients who presented with haemorrhagic shock and who were admitted to the emergency unit of the hospital. A total number of 51 of these files met the inclusion criteria for the study although only 10 files contained admission lactate values. Based on these files 10 clinical audit checklists were completed.

Accordingly, the sample for the pre-test consisted of 10 patient files that had been selected from a population of 69 patient files which were not included in the study itself. The data collected during the pre-test was analysed carefully in order to determine whether the clinical audit checklist was capable of capturing the desired data. The clinical audit checklist used for the pre-test is contained in Annexure B.1. However, it was subsequently altered on the basis of the feedback from the pre-test and administered in the main study as suggested by De Vos *et al.* (2002: 177).

There were two alterations made to the clinical audit checklist which had been used for the pre-test. These changes included the inclusion of decimals in the serum lactate values, thus contributing to the recording of more accurate blood lactate levels. The second change involved the inclusion of the type of incident that had caused the haemorrhage. Both these changes were suggested and approved by the professional statistician who assisted the researcher in the interpretation of the data (see Annexure B.1 and B.2).

3.4.3 Data collection

The data were collected by the researcher herself. She was supervised by personnel in the record department of the hospital to ensure both the confidentiality of the information. The data, which were collected over a period of four months from July 2009 to November 2009, were collected by means of a clinical audit checklist.

3.4.4 Data analysis

In most social research, data analysis involves three major steps, namely, cleaning and organising the data for analysis, describing the data, and testing hypotheses and models (Trochim 2006). Data preparation involves checking or logging the data in; checking the data for accuracy; entering the data into the computer; transforming the data; and developing and documenting a database that integrates the various data (Trochim 2006). A professional statistician then analysed the data using the Statistical Package for the Social Science (SPSS) version 14.0 microcomputer program.

The data were interpreted by means of frequency tables, box plot histograms and X graph charts. The analysis of the data corresponded with the sections contained in the clinical audit checklist and the data were interpreted accordingly (See Chapter 4).

3.5 VALIDITY AND RELIABILITY OF THE RESEARCH

According to Somekh and Lewin (2005: 216), validity refers to whether or not the instrument used succeeds in collecting the data which is necessary in order to answer the research questions. A measure may be reliable (always generate the same results) but not valid (not measure the intended concept).

3.5.1 Validity

Validity also refers to the ability of an instrument to measure exactly what it is supposed to measure (De Vos *et al.* 2002: 167). In this study validity was assured through consultation with multi-disciplinary members involved in the same area of study, literature reviews and constructive inputs from supervisors and the statistician.

The validity of an instrument is supported by either a greater or a lesser degree of evidence – no easy task. There are different approaches to assessing the validity of an instrument and both face validity and content validity were used in this study. The application of these approaches is summarised below:

- Face validity is a subjective judgement of whether an instrument appears to measure what it is supposed to measure (Flokstra-de Blok & Dubois 2009: 216). Face validity was found to be present in this study because all the sections of the clinical audit checklist focused on lactate values (0 to 48 hours) and the mortality of patients. These variables were based on previous, related studies and were consistent with variables found in the literature. Accordingly, it would appear that the clinical audit checklist did seem to be measuring what it was supposed to measure.
- Content validity is concerned with the sampling adequacy of the content area being measured. Areas covered by the instrument should represent a wide area of the topic under study. This is considered to be a subjective exercise because the researcher who is designing the instrument determines the content to be included. The use of content validity used in the research is consistent with the views of Polit and Beck (2006: 329). In this study the researcher asked two physicians, a surgeon, two emergency care nurses, two supervisors and a statistician to evaluate every item of the clinical audit checklist for content validity.

3.5.2 Reliability

The reliability of an instrument may be equated with the clarity, stability, consistency and accuracy of the measuring tool (Trochim 2006). Reliability is

thus the consistency of an instrument or the ability of the instrument to be applied repeatedly to measure that which the researcher wishes to measure. If used on a similar group of respondents in a similar context the instrument should yield similar results (Gerrish & Lacey 2010: 370). According to Somekh and Lewin (2005: 216), reliability refers to the stability or consistency of measurements – whether or not the same results would be achieved if the test or measure were applied repeatedly.

In general, reliability refers to the extent to which the independent administration of the same instrument consistently yields the same (or similar) results under comparable conditions (De Vos *et al.* 2002: 168). Two aspects of reliability were applied in this study, namely, internal consistency and equivalence. An instrument may be said to be reliable if it always generates the same results (Somekh & Lewin 2005: 216). The clinical audit checklist was developed in such a way that specific data were obtained that were relevant to the subject of investigation. This was done in order to enhance the reliability of the instrument. The same data were collected each time using the clinical audit checklist. The data were collected by the researcher herself while the clinical audit checklist was tested for internal consistency by the conducting of a pre-test.

The clinical audit checklist was compiled by the researcher under the supervision of a professional statistician and researcher's supervisors who are specialists in the field of trauma and emergency care. This clinical audit checklist was accredited by three specialists in the field of emergency and intensive care.

3.6 ETHICAL CONSIDERATIONS

One of the first internationally recognised efforts to establish ethical standards is known as the Nuremberg Code (Polit & Beck 2008: 168). These standards were developed following the Nazi atrocities which were made public during the Nuremberg trials. Several other international standards have subsequently been developed, of which the Declaration of Helsinki is the most notable. The

principles of the Declaration of Helsinki are followed by the Faculty of Health Science, Research Ethics Committee, University of Pretoria.

Research ethics involve the protection of the rights of both the respondents who participate in research as well as the institutions in which the research is carried out while at the same time maintaining scientific integrity (Babbie & Mouton 2001: 531; Burns & Grove 2005: 181). A researcher is responsible for conducting research in an ethical manner. Failure to do so undermines the scientific process and may, indeed, have negative consequences (Brink *et al.* 2006: 30).

The Nuremberg Code which guides ethical research is described by Burns and Grove (2005: 177). The Code reflects the three concepts most appropriate for this study which the researcher aimed to uphold. These concepts include the principles of justice, informed consent and ethical approval. The carrying out of research in an ethical way commences with identification of the topic and continues through to the end at which point the findings are published. It is, thus, essential that the conduct of researchers be characterised by not only expertise and diligence, but also by honesty and integrity (De Vos *et al.* 2002: 24). These ethical principles are described below.

3.6.1 Justice

The principal of justice forms part of the Belmont Report which was brought out in the United States in 1978 by the National Commission for the protection of human subjects in biomedical and behavioural research (Polit & Beck 2004: 143). In this study the principle of justice was upheld by the researcher's honouring of the patients' right to privacy. This involved protecting the patients' identities in such a way that it was not possible to trace any information back to the subjects. It is, thus, essential that the patients should remain anonymous throughout the research, even to the researcher (Polit & Beck 2004: 149).

The process of the purposive selection of subjects ensured the anonymity of the patients. Purposive sampling may also be referred to as judgemental sampling

and it involves the selection of participants by the researcher based on the researcher's personal judgement of which patients' files is most representative of the population (Polit & Beck 2004: 729). This purposive selection of files was accomplished by adhering to the inclusion and exclusion criteria of the research as stated in the sample plan (see Chapter 3, Section 3.4.1). No personal information that could be traced back to a specific patient was included in the clinical audit checklist and, accordingly, the patients remained anonymous.

3.6.2 Informed consent

According to Polit and Beck (2004: 151) informed consent means that the participants in a study are furnished with adequate information about the research. In view of the fact that the study conducted was retrospective in nature and because the patients' files only were utilised as information, consent to access the patient files was obtained. This was accomplished in accordance with the policy of the hospital on the utilisation of patients' files. Consent was obtained from both the hospital and the emergency unit in which the research was conducted.

Both a consent form and a letter requesting permission to conduct the study are included in the appendices (see Appendices A.1 and A.2). The letter explains both the purpose and the nature of the research whilst also explaining that there were no risks involved in conducting the research and that the anonymity of the patients was ensured. The researcher also adhered to ethical considerations in respect of the data collection.

The head of the emergency unit gave written consent for the research to be conducted in the unit as did the superintendant of the hospital. Permission to access files was also obtained from the superintendant of the hospital.

3.6.3 Ethical approval

Before commencing the research, the research proposal was submitted to the Ethical Committee of the Faculty of Health Science at the University of Pretoria, (See Annexure A, sections A1). In order to ensure that there would be no bias during the research the ethical dimensions were evaluated by an external review board. This external review board was also responsible for determining the risk benefit ratio in respect of the patient who's files were included as part of the research. The risk benefit ratio is based on the principal of beneficence – to do good and to protect from harm (Polit & Beck 2004: 156). The Ethical Committee decided unanimously that the risk benefit ratio of the research was low and that it would be possible for the research to be conducted without any harm being done to patients.

The researcher obtained permission to conduct the research from the emergency unit of the hospital concerned (see Annexure A, section A.2). The superintendant of the hospital as well as the head of the department of the emergency unit both gave permission for the study to be conducted. Babbie and Mouton (2001: 530) and Burns and Grove (2005: 181) are all the opinion that ethical considerations both protect the institutions in which the research is conducted as well as helping to maintain scientific integrity. In addition, all the information obtained was used solely for the purposes of this study and any publication relating to the study in journals would be subject to prior permission from the hospital.

A summary of the ethical principles as specified in the Nuremburg Code and to which the researcher adhered during this study are presented in Table 3.1.

Table 3.1: Summary of the application of the principles of the Nuremberg Code

Principles of the Nuremberg Code	Application to this research
Voluntary consent is essential	A retrospective study was conducted and permission to access patient files was obtained from the hospital. This was done in accordance with both the policy of the Research Ethics Committee of the Faculty of Health Sciences, University of Pretoria and the procedure specified by the hospital.
Study should yield fruitful results for the good of society	The results of the study will be used to make recommendations in respect of future resuscitation strategies for patients presenting with haemorrhagic shock in order to improve the outcomes of such patients.
Previous results should justify the study	The researcher conducted an in-depth literature review on the research topic (see Chapter 2)
Study should avoid unnecessary physical and mental discomfort, suffering and injury	There was no perceived risk of harm to the patients whose files were used for the research as their files were used to obtain retrospective information.
No study should be conducted if it is believed that it will cause death or disabling injury.	In view of the fact that this study was a retrospective study there was no human intervention and, thus, no harm to patients occurred.
The degree of risk should never exceed the benefits of the study	No risk was posed to the patients whose files were used for the study sample as this was a retrospective study
The study should be performed by qualified persons only	The researcher is a qualified registered nurse and has successfully completed a research methodology module. In addition, the supervisors overseeing the researcher were both recognised researchers.

Principles of the Nuremberg Code	Application to this research
Subjects should be free to withdraw at any time	Consent to obtain information from patients' files was obtained from the hospital in accordance with both hospital policy and with the requirements of the ethics committee.
The researcher should be prepared to stop the study if a continuation of the research is likely to cause harm	Approval for the study was granted by the Research and Ethics Committee of the University of Pretoria which foresaw no harm and approved the research unconditionally.

Source: Adapted from Burns and Grove (2005: 177)

3.7 CONCLUSION

This chapter covered the research design and methodology. It emphasised the rationale behind the utilisation of the research design and described in detail the population, sampling and sample, research instrument, data collection, ethical considerations, study limitations and data analysis.

Chapter 4 will discuss the data analysis, the results and the interpretation of the results by means of a literature control.

4 RESULTS, DISCUSSION AND ANALYSIS

4.1 INTRODUCTION

Chapter 3 provided an overview of the research method. In this chapter the results of the analysis of the data obtained in the study and a discussion on the findings is presented. This discussion is divided into three sections (Sections 4.2.1 to 4.2.3). Section 4.2.1 presents the results of the demographic information included in the clinical audit checklist, Section 4.2.2 contains an analysis of lactate levels documented over a 24-hour period while Section 4.2.3 provides an analysis of the 48-hour mortality rate of patients presenting with haemorrhage caused by trauma.

4.2 RESULTS AND DISCUSSION

The data were collected by means of a clinical audit checklist which had been designed to capture the specific data that were relevant to this research (see Annexure B, Section B.2). The clinical audit checklist was devised by the researcher under the direct guidance of a statistician and consisted of the following sections:

- **Section A:** Demographic information
- **Section B:** 24-hour lactate levels
- **Section C:** 48-hour mortality

The findings will be discussed in terms of each section and item as they appear in the clinical audit checklist (see Annexure B).

4.2.1 Section A: Demographic information [V2-V4; V16]

The demographic information provided the basic data that were required for the research to be conducted. This demographic data included the patients' date of admission, age and gender. The type of incident which had caused the trauma (see Annexure B.2, item V16) did not initially form part of the clinical audit checklist, but was included on the recommendation of the statistician as being of additional interest (see discussion Section 4.2.1.4).

4.2.1.1 *Date of admission [V2]*

The files of adult patients who had been admitted to the emergency unit in the period from January 2008 to June 2008 were used. Figure 4.1 illustrates the distribution of patients entering the emergency unit at that time. It may be deduced from Figure 4.1 that, of the 46 files that were used for the research (N = 46), most of the files pertained to February 2008 (12 files), March 2008 (10 files) and June 2008 (10 files). No files were used of patients admitted during May 2008.

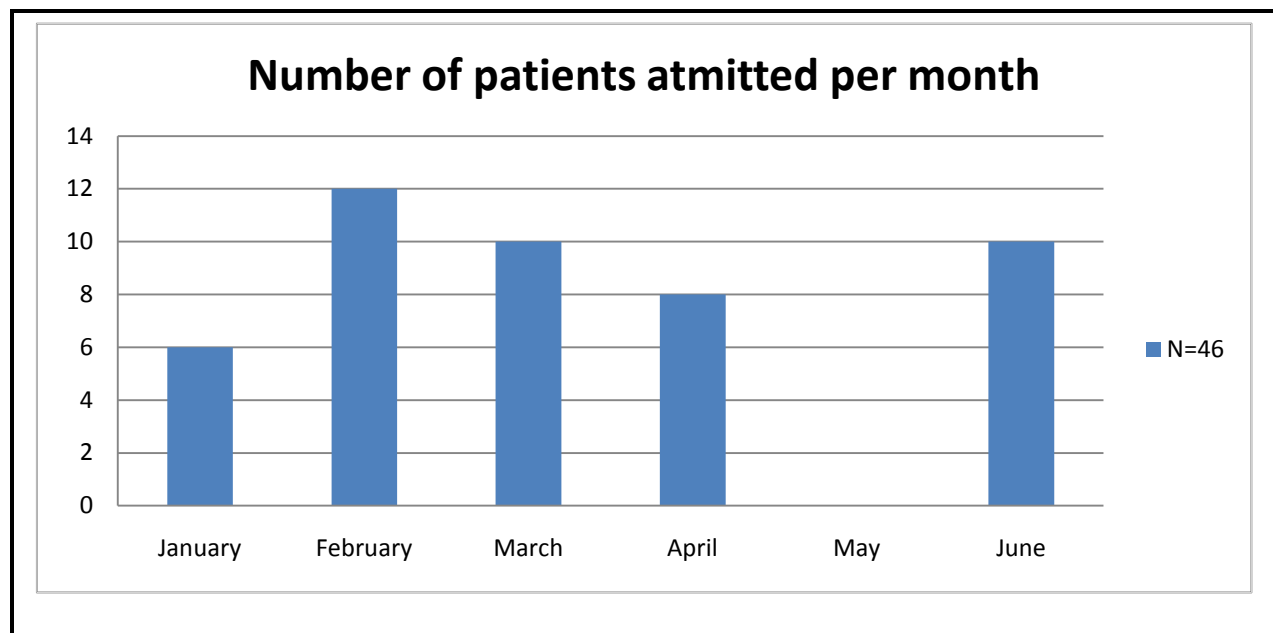


Figure 4.1: Distributions of patients entering the emergency unit from January 2008 to June 2008

Discussion: According to statistics from South African Health/Info (2005), the most common months in which death caused by trauma-related accidents occur are March and May with the fewest deaths being registered in January and February.

Analysis: The distribution of patients entering the emergency unit as illustrated in Figure 4.1 shows that the occurrence of trauma is not distributed evenly throughout the year and appears to be erratic and haphazard. This implies that personnel working in emergency units should at all times be prepared to treat patients entering the emergency unit with haemorrhage caused by trauma.

4.2.1.2 Gender [V3]

Adult female and male patient files were included in the population of the research. Table 4.1 indicates the gender distribution of the patients whose files were used in research (N = 46). Table 4.1 illustrates the frequency of both male and female patients whose files were used for the research, namely, 80,4% (37 files) of the patients who were admitted to the emergency unit were male, while only 19,6% (9 files) were that of female patients.

Table 4.1: Gender distribution of files used for the research

Gender	Frequency	Percentage (%)
Male	37	80.4
Female	9	19.6
Total/N	46	100.0

Discussion: Studies such as those conducted by Brasel *et al.* (2007: 813), Acosta, Yang, Winchell, Simons, Fortlage, Hollingsworth-Fridlund, and Hoyt (1998: 529) and Dick and Baskett (1999: 89) illustrate that injury due to trauma is more prevalent in males than in females.

Analysis: The frequency distribution of male and female patients as seen in Table 4.1 is derived from the statistics of the hospital for the study period. These statistics clearly indicate that haemorrhage due to trauma is more prevalent in male patients than in female patients. This is in accordance with the literature on trends observed worldwide. Accordingly, the findings of this research imply that personnel working in the emergency unit of hospitals should be prepared to work with patients who are often male patients, bigger and stronger than their female counterparts. This may make management of these patients more difficult. Nevertheless, female patients presenting with haemorrhage should not be underestimated in terms of size, strength and severity of injuries and standardised care should be rendered, irrespective of gender.

Figure 4.2 depicts the gender distribution for trauma patients as presented in Table 4.1.

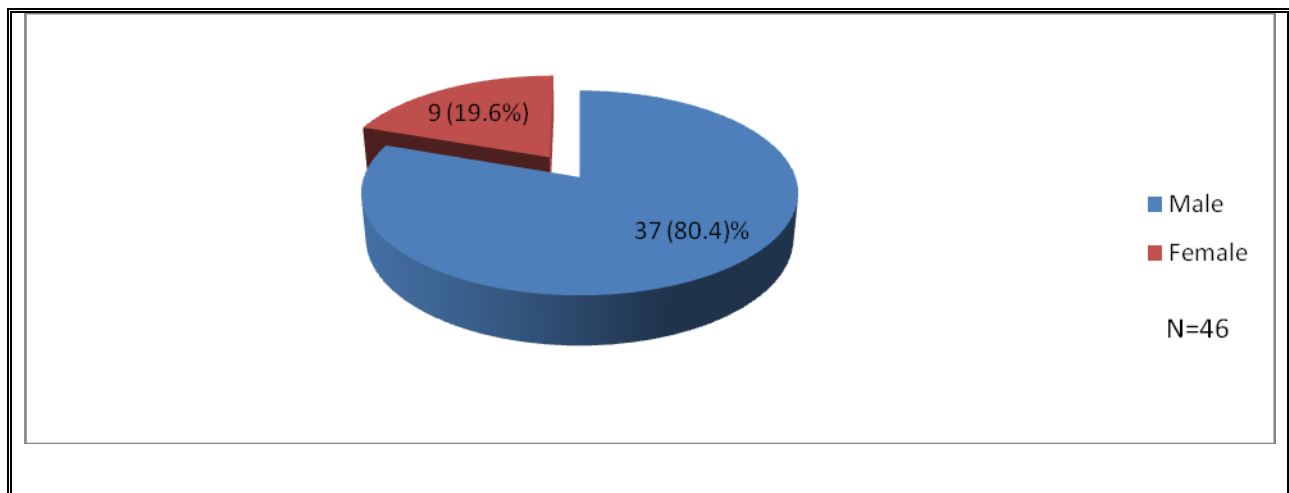


Figure 4.2: Gender distribution for patients' files used in the sample of the research

4.2.1.3 Date of Birth [V4]

The inclusion criteria for the research stated that adult patients only were to be included in the research. The clinical audit checklists were completed for the files of adult patients of 19 years or older who had entered the emergency unit in the period from January 2008 to June 2008.

In the research conducted the average age of the patient was 34 years (N = 46) with five patients (10,8%) being between the ages of 10 and 20 years. The age group with the largest representation of 22 files (47,8%) was between the ages of 20 and 30 years with 11 patients (23,9%) being between the ages of 30 and 40 years. The age groups representing 40 to 50 years and 50 to 60 years comprised seven files (15,2%) each with three patients only (4,6%) in the sample in the age category of 60 to 70 years.

Figure 4.3 illustrates the age distribution of patient files used for the sample utilised in the research.

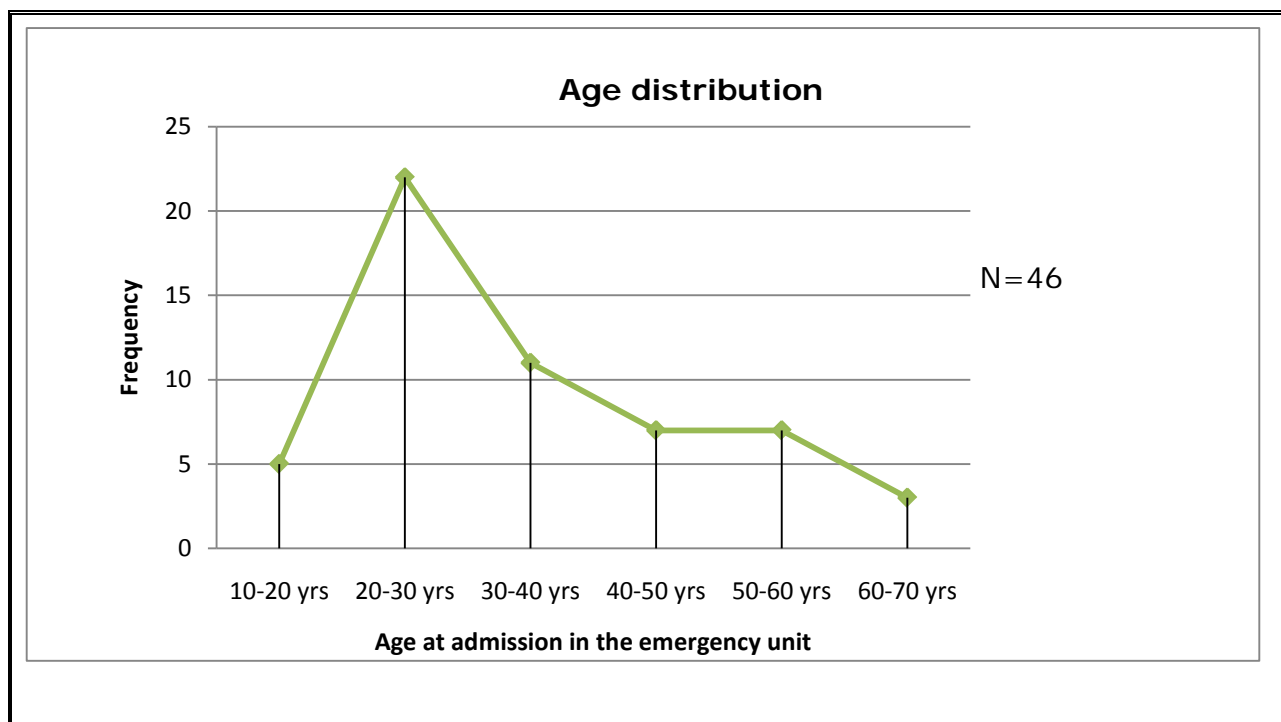


Figure 4.3: Age distribution of admissions to the emergency unit

Discussion: The findings, as illustrated in Figure 4.3, are in agreement with the findings contained in the literature on research conducted on trauma patients. For example, according to Cerovic *et al.* (2003: 1301), the mean age of patients who sustained injuries caused by trauma was 37,5 years. Stahel, Heyde and Ertel

(2005: 200); Kane *et al.* (2007: 61) and Garner *et al.* (2010: 1131) all propose that trauma is still the greatest killer of patients younger than 45 years of age.

Analysis: As seen in Figure 4.3, the majority of the patients entering the emergency unit of the hospital with haemorrhage due to trauma were below the age of 45 years. This is in accordance with the findings of researches as quoted above and it implies that the RSA is also faced with the challenges faced by countries elsewhere in the world. This clearly means that there is an urgent need for the effective initial management of patients entering an emergency unit and presenting with haemorrhage caused by trauma. The outcome of these patients is of cardinal value, particularly in view of the fact that they are often otherwise healthy young individuals.

4.2.1.4 Type of incident [V16]

Figure 4.4 presents a graphic representation of the type of incident recorded on the clinical audit checklist as the cause of the trauma. In other words, the findings depicted in Figure 4.4 reveal the causes of injury to the patients whose files were utilised in the study (N = 46). Pedestrian vehicle accidents contributed to 12 (30%), of the total incidents recorded, with motor vehicle accidents being the second largest cause of injury and comprising 11 (28%) of the total number of injuries. Gunshot wounds and stab wounds contributed to seven (18%) and five (13%) of the total incidents respectively. A minority of injuries were caused by assaults—three (8%) of the total number of injuries. Motorbike accidents were the cause of two incidences (5%) of the injuries sustained.

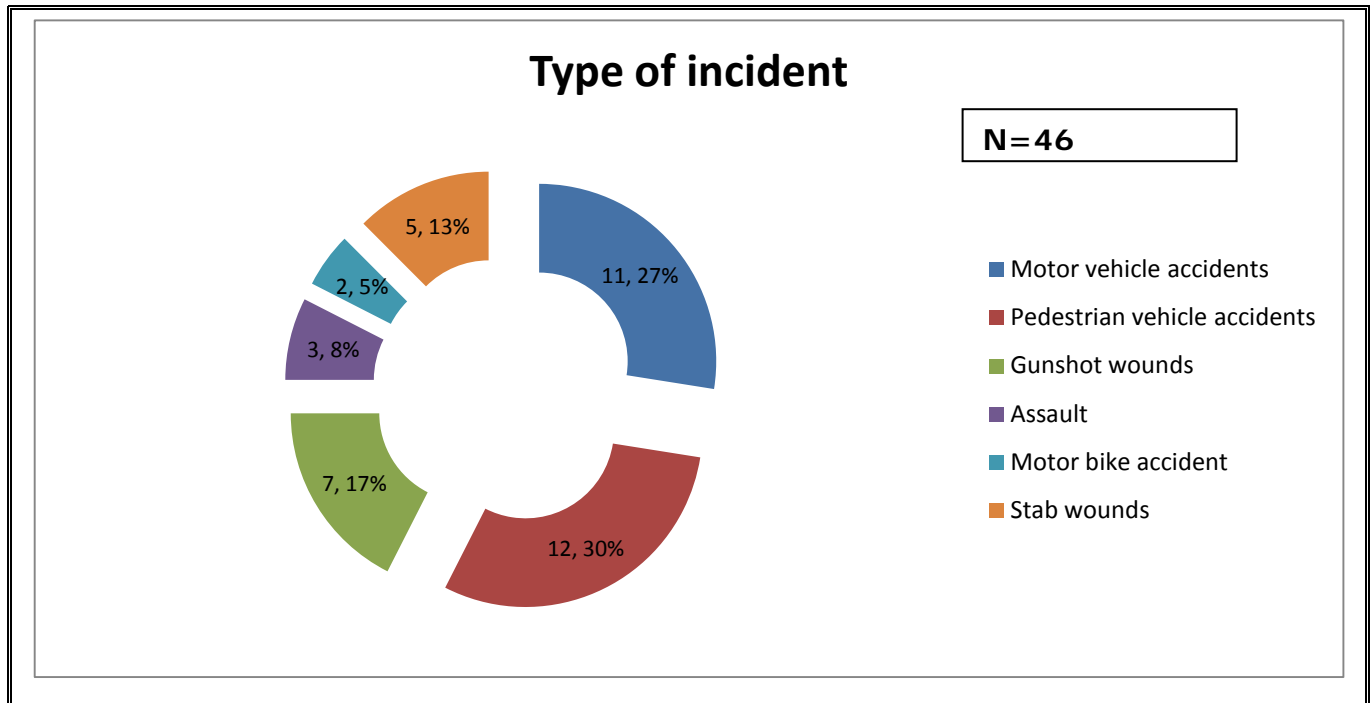


Figure 4.4: Types of injury sustained

Discussion: These findings from the research may be compared to the international trends as discussed in the study by Acosta *et al.* (1998: 532). In this retrospective study of Acosta *et al.* (1998: 532) on deaths resulting from trauma in a Level I emergency unit over a period of 11 years it was found that the major cause of death among these patients was as a result of penetrating injuries which had led, in turn, to severe haemorrhage. Motor vehicle accidents accounted for 80% (434 of 537) of all blunt trauma deaths while homicide was the cause of 75% (272 of 363) of all penetrating trauma.

According to statistics in South Africa, violence is the leading cause of trauma related deaths (38,77%) while accidents contribute to 38,78% of the total deaths. In addition, these statistics indicate that the majority of accidental deaths are as a result of transport-related incidents (SA HealthInfo: 2005).

According to Kauvar *et al.* (2006: S4) violence, whether self-inflicted, interpersonal related or as a result of war, accounted for half of the trauma deaths (1,6 million)

registered globally by the World Health Organization (WHO) in 2000 with road accidents accounting for 1,2 million of the deaths related to trauma.

Analysis: The information depicted in Figure 4.4 is significant as it provides evidence of the fact that the emergency unit of the hospital is confronted with global trends and the same difficulties which are faced worldwide. An awareness of the type of incidents that will have to be dealt with makes it easier to offer recommendations for future resuscitation strategies for patients presenting with haemorrhagic shock in order to improve the outcomes of these patients.

4.2.2 Section B: 24-hour lactate levels [V5-V13]

In this section of the clinical audit checklist serum blood lactate levels were obtained over a period of 24 hours. The inclusion criteria for the research specified that an admission serum blood lactate level had to be available if the inclusion criteria of the research were to be met.

Research, such as studies conducted by Cerovic *et al.* (2003: 1302-3); Shirley (2007: 195) and Schulman (2002: 32cc4), all involved the testing of serum blood lactate levels at different time intervals – 8 hours, 12 hours and 24 hours – after admission. It was found that the serum blood lactate levels peaked at 8, 12 and 24 hours and were, thus, reliable predictors of patient survival rates. These guidelines were followed when determining the timeframes (in consultation with a professional statistician) at which the serum blood lactate levels of patients were documented during the clinical audit (see Annexure B). Section B of the clinical audit pertaining the 24-hour lactate levels was, therefore, divided into the following subsections:

4.2.2.1 Time of admission to emergency unit [V5]

The admission time of patients to the emergency unit was included as part of Section B in order to estimate the average time until the first lactate value was

obtained. Figure 4.5 indicates both the admission times of patients to the emergency unit as well the specific time at which the first lactate value was obtained. It would appear that peak admission times for patients admitted to the emergency unit were between 8:00 and 10:00 in the morning and between 20:00 and 22:00 in the evenings.

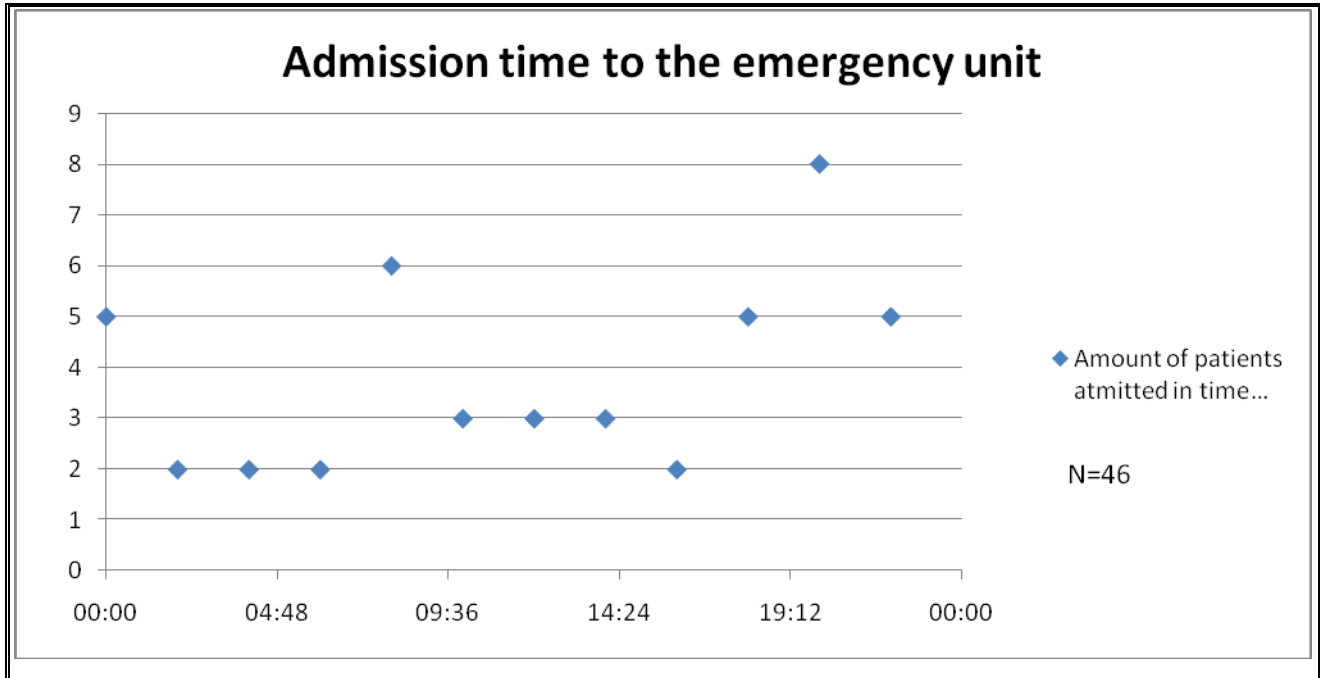


Figure 4.5: Admission time to the emergency unit

Discussion: As is illustrated in Figure 4.5, the admission times of patients to the emergency unit are unpredictable. These unpredictable admission times may easily lead to overcrowding in the emergency department. Creswell (2003: 174) defines overcrowding in an emergency unit as the inability of both the emergency department itself and the personnel working in the department to meet the needs of patients entering the emergency department at a specific time. This implies that personnel working in an emergency unit should always be primed to treat emergencies when they arrive. Statistics from SA Health *Info* (2005) indicate the time of day at which most fatal incidences in Tshwane took place in 2005. It was found that most violence-related incidents took place between 19:00 and midnight.

Most motor vehicle accidents peaked between 18:00 and midnight with pedestrian vehicle accidents being most prevalent between 7:00 and 09:00.

Analysis: As may be seen in Figure 4.5, the statistics obtained from the hospital in the study are in accordance with research conducted in other emergency unit settings. Most patients were admitted between 18:00 and midnight and between 8:00 and 10:00 in the morning. Figure 4.4 depicts the injuries with which these patients presented, with the most prevalent of the incidences relating to these injuries being pedestrian vehicle accidents and motor vehicle accidents. This finding correlates with the statistics from Tshwane as discussed in SA Health *Info* (2005).

4.2.2.2 Timeframe: 0 to 4 hours after admission [V6]

This section refers to the first lactate levels that were obtained from the patients after admission. Files that did not contain a documented admission lactate were not included in the research. Table 4.2 indicates the frequency of the lactate levels obtained in the period 0 to 4 hours after admission and the corresponding percentages.

As illustrated in Table 4.2, the average admission lactate level recorded on the clinical audit checklist was 4,092 mmol/L. As discussed in Chapter 2, Section 2.5.2.4, this indicates the presence of ongoing tissue hypoxia and hypoperfusion. The median value (the precise middle value in a score allotment) was 3,6 mmol/L, which also represents a significantly raised lactate value. The standard deviation (the degree of variability in a set of scores statistically measured) was 2,3739 mmol/L with the variance, calculated as the square root of the standard deviation, at 5,635 mmol/L. The lowest recorded lactate value was 1,0 mmol/L. This level may be seen as representing a normal value since it is below 2 mmol/L (Englehart & Schreiber 2006: 570).

Table 4.2: Frequency of lactate levels 0 to 4 hours after admission

Frequency of lactate values 0 to 4-hours after admission	Statistical data
Mean	4,092 mmol/L
Median	3,600 mmol/L
Variance	5,635
Standard deviation	2,3739
Minimum	1,0 mmol/L
Maximum	13,1 mmol/L
Range	12,1 mmol/L
N	46

Figure 4.6 presents the lactate values recorded for patients who survived compared to those patients who died 0 to 4 hours after admission.

Discussion: Three patient mortalities were recorded in the period 0 to 4 hours after admission. The average recorded lactate level for the deceased patients was 6,217 mmol/L – a value which was higher than the average recorded lactate level of the patients who survived. The lactate values recorded for the deceased patients were all above 2,8 mmol/L and, although only slightly increased, still higher than the normal lactate value as defined by Englehart and Schreiber (2006: 570). The highest lactate value recorded was 13,1 mmol/L – a level which pertained to a patient who had died. This findings show a marked correlation with the findings of both Cerovic *et al.* (2003: 1303) and Schulman (2003: 32cc4) who maintained that the higher the lactate value, the more severe the injury, and the higher the mortality rate.

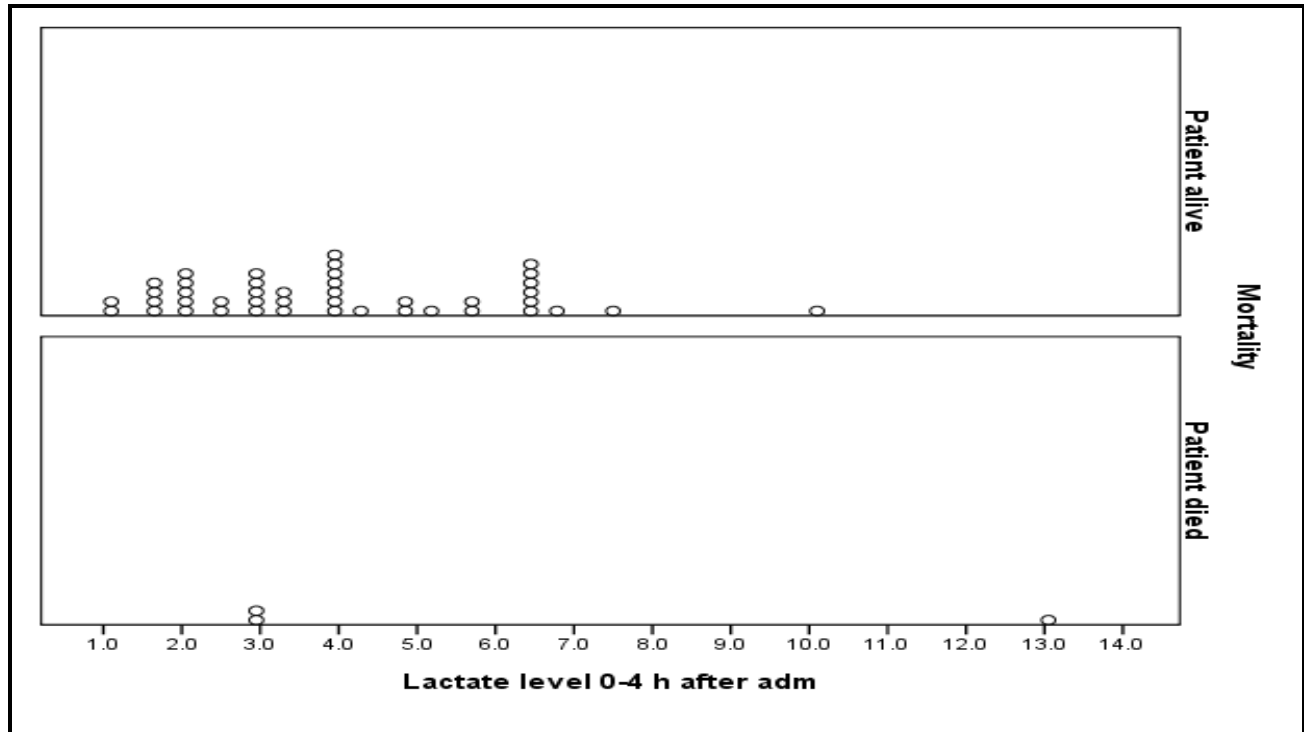


Figure 4.6: Lactate levels of patients who survived compared with those of patients who died 0 to 4 hours after admission

Analysis: The findings depicted in both Table 4.2 and Figure 4.6 imply that, overall, patients admitted to an emergency unit present with an elevated serum blood lactate level which is the result of haemorrhage caused by trauma. This elevated serum blood lactate level may serve as a good indicator to initiate immediate resuscitation which is generally associated with improved outcome (Shirley 2007: 198). An initial peak lactate value also serves as an indication of the probability of MODS developing (Englehart & Shreiber 2006: 570).

4.2.2.3 Timeframe: 4 to 8 hours after admission [V7-V8]

Figure 4.7 depicts both the serum blood lactate levels obtained from patient files for the period 4 to 8 hours after admission as well as the exact times at which these levels were obtained. Fifteen of the 20 lactate values recorded were above 2,0 mmol/L. The average lactate value recorded was 3,43 mmol/L which indicates a raised lactate value.

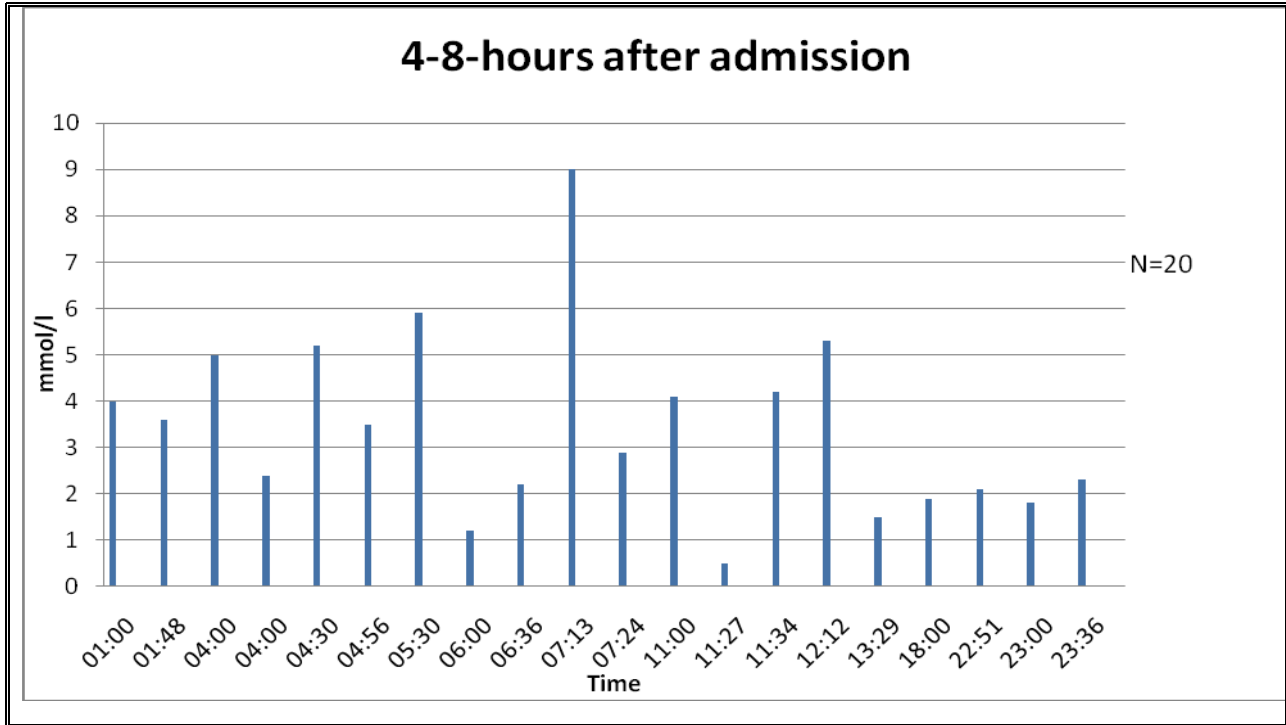


Figure 4.7: Serum blood lactate levels 4 to 8 hours after admission

Table 4.3 indicates the frequency of lactate values and the corresponding percentages of the total lactate values obtained for the period 4 to 8 hours after admission.

Table 4.3: Lactate levels obtained for the period 4 to 8 hours after admission

Lactate level (mmol/L)	Frequency	Percentage (%)
0 - 1	1	2,2
1,1 - 2	4	8,8
2,1 - 3	5	11
3,1 - 4	2	2,2
4,1 - 5	3	6,6
5,1 - 6	4	8,8
6,1 - 7	0	0
7,1 - 8	0	0
8,1 - 9	1	2

Discussion: The literature suggests that the most significant raise in serum blood lactate was noted 8 hours after admission (Cerovic *et al.* 2003: 1303). These findings were used as a guideline to determine the recording of serum blood lactate levels on the clinical audit checklist 4 to 8 hours after admission. However, 20 files (N = 20) only contained a serum blood lactate level recorded for the period of 4 to 8 hours after admission. In the arguments presented in Chapter 2, Section 2.5.2.4 under the heading of *Lactic acidosis*, raised serum blood lactate levels 4 to 8 hours after admission serve as an indication of the under resuscitation of haemorrhagic shock patients. A prolonged increased serum blood lactate level in patients presenting with haemorrhage reflects underlying tissue hypoperfusion and tissue hypoxia (Schulman 2002: 32cc4; Shirley 2007: 195).

Analysis: The lack of documented serum blood lactate levels 4 to 8 hours after admission implies that lactate levels are not used as an endpoint of resuscitation in the emergency unit of the hospital and, subsequently, patients present with prolonged tissue hypoxia and hypoperfusion. This finding may serve as a basis on which to make recommendations for future resuscitation strategies for patients presenting with haemorrhagic shock in order to improve the outcomes of such patients.

4.2.2.4 Timeframe: 10 to 14 hours after admission [V9-V10]

Figure 4.8 presents the lactate values obtained from patient files 10 to 14 hours after admission. However, only 19 serum lactate values were recorded on the clinical audit checklist for this period. One patient had died 13 hours and 41 minutes after admission. The average lactate value for the patients who survived during this period was 3,528 mmol/L. The highest lactate value obtained was 9,3 mmol/L and the lowest lactate level 0,6 mmol/L. The mean lactate value of 3,528 mmol/L indicates a raised lactate value.

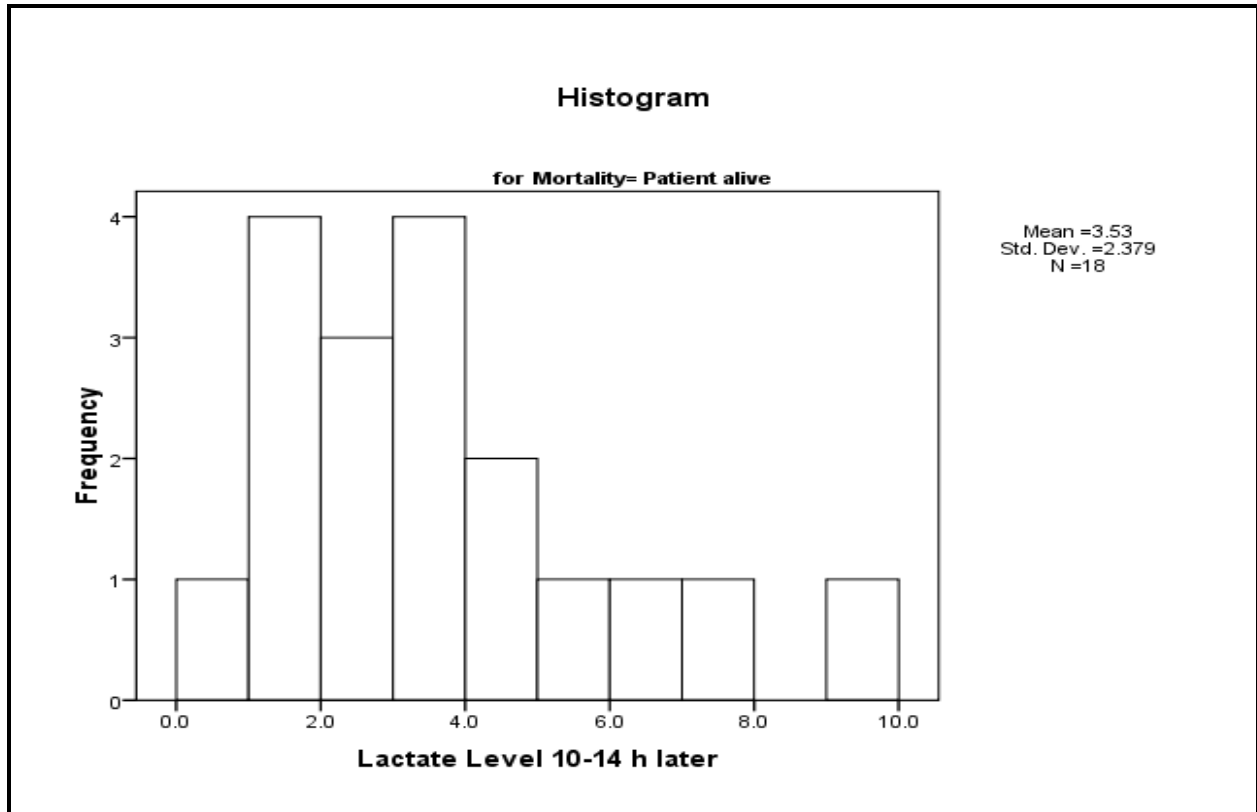


Figure 4.8: Lactate values 10 to 14 hours following admission

Figure 4.9 illustrates the serum lactate value recorded on the clinical audit checklist for the deceased patient compared to those recorded for the patients who survived.

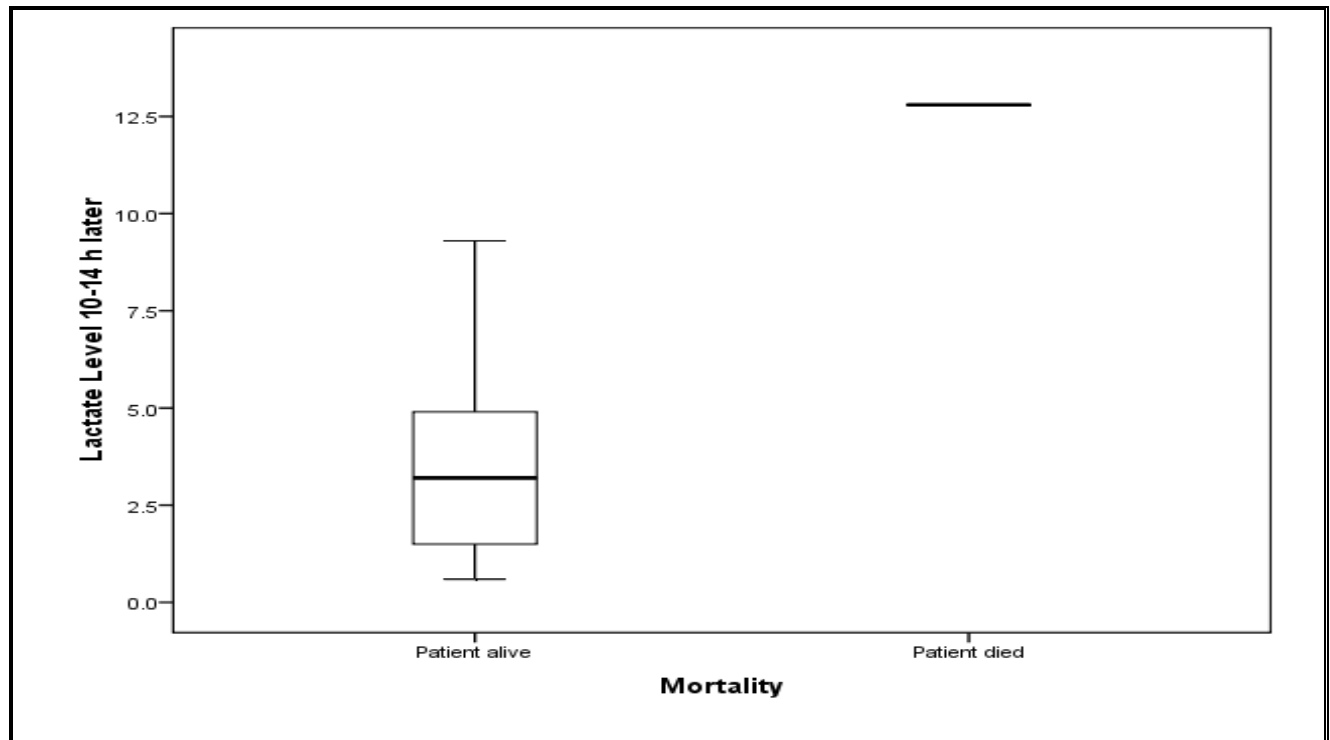


Figure 4.9: Mortality 10 to 14 hours after admission

Discussion: Cerovic *et al.* (2003: 1303) indicate the prognostic value of raised lactate levels 12 hours after admission to an emergency unit. A raised lactate level 12 hours after admission is a strong indicator of deteriorating survival rates. In other words, the higher the lactate values after this period, the higher the mortality rate (Cerovic *et al.* 2003: 1304). The authors Schoemaker and Beez (2010: 8) also indicate higher survival rates and better outcomes for patients who manifested minimum tissue hypoxia and hypoperfusion 12 hours after admission. The patient who had died 10 to 14 hours after admission had presented with a significantly raised lactate value of 12,8 mmol/L. This supports the views of Cerovic *et al.* (2003: 1304) and Schulman (2002: 32cc4) that the higher the lactate values 12 hours after admission, the slimmer the chances of survival.

Analysis: Although the literature does suggest that raised lactate values 12 hours after admission are of significant prognostic value, only 19 of the patient files used in this research contained a documented serum lactate value (N = 19). The average recorded lactate value was also elevated which suggests that the patients had

remained hypoperfused. These findings indicate the need for revised resuscitation strategies for patients presenting with haemorrhage and the introduction of end points of resuscitation.

4.2.2.5 Timeframe: 22 to 26 hour after admission [V11-V13]

Figure 4.10 presents the lactate values obtained from patients 22 to 26 hours after admission. However, 10 lactate values only were available and recorded on the clinical audit checklist for this period (N = 10). The average lactate value was 2,190 mmol/L which represents a slightly elevated value with the highest recorded value being 5,2 mmol/L, indicating a progressive state of shock (Schulman 2002: 32cc4). From the data obtained it would appear that the majority of the lactate values had almost normalised within the 24-hour period.

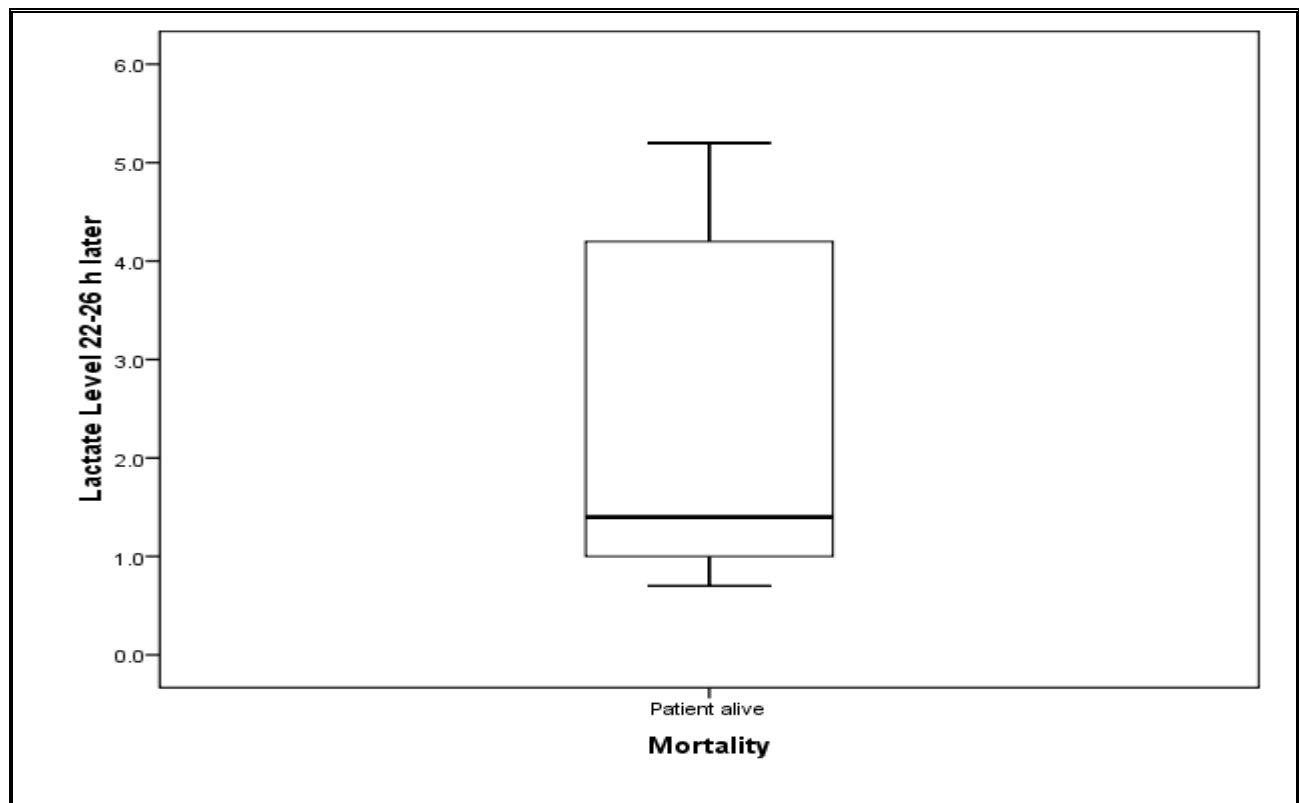


Figure 4.10: Lactate values 22 to 26 hours after admission

Discussion: Schulman (2002: 32cc4) and Shirley (2007:195) both indicate a correlation between the time of normalisation of lactate values compared to the mortality rate of patients. According to them, lactate values which normalised within 24 hours after admission indicate a close to 100% survival rate. This view is supported by both Englehart and Schreiber (2006: 570) and Porter and Ivatury (1998), thus indicating a 0 to 10% mortality rate for patients whose lactate levels are normalised within 24 hours after admission. The literature suggests that it is important to evaluate the resuscitation against 24-hour mortality as this is the period in which the most significant number of deaths as a result of hypovolaemia occur (Acosta *et al.* 1998: 528; Kauvar *et al.* 2006: S4).

Analysis: The lactate levels obtained 22 to 26 hours after admission showed a decrease in value. This decrease is associated with a favourable prognostic outcome. However, very few lactate values were recorded for this time period which makes it difficult to indicate the outcomes of most of the cases. This inadequate recording of lactate levels may be seen as a limitation of this research. Despite the fact that it would appear that the hypoperfusion of cells had been alleviated, this finding is inconclusive in view of the small number of lactate levels recorded for this period.

4.2.3 Section C: 48 hour mortality [V14-V15]

For the purposes of this research, the 48-hour mortality rate of patients presenting with haemorrhagic shock in the emergency unit was also investigated. Various studies indicate that, when lactate levels are not normalised within 24 hours after admission, the mortality rate of such patients increases (Englehart & Schreiber 2006: 570; Schulman 2002: 32cc4; Shirley 2007: 195). In other words, the mortality rate increases significantly in patients whose lactate level are not normalised within 48 hours after admission. Schulman indicates that the mortality rate for these patients is between 80 and 86% (Schulman 2002: 32cc4). However, the study conducted by Cerovic *et al.* (2003: 1302) indicates much higher lactate levels in non-surviving patients than in surviving patients after 48 hours.

Shoemaker and Beez (2010: 8) detected that all patients who die within 48 hours after a shocked state caused by haemorrhage suffered from hypoperfusion on a cellular level. In view of the fact that patient death 48 hours after admission to the emergency unit is influenced by other factors, the mortality of such patients was not considered as part of this research.

4.2.3.1 *Date and time of death [V14-V15]*

Figure 4.11 indicates the lactate levels for patients who died 0 to 4 hours after admission compared to those patients who survived.

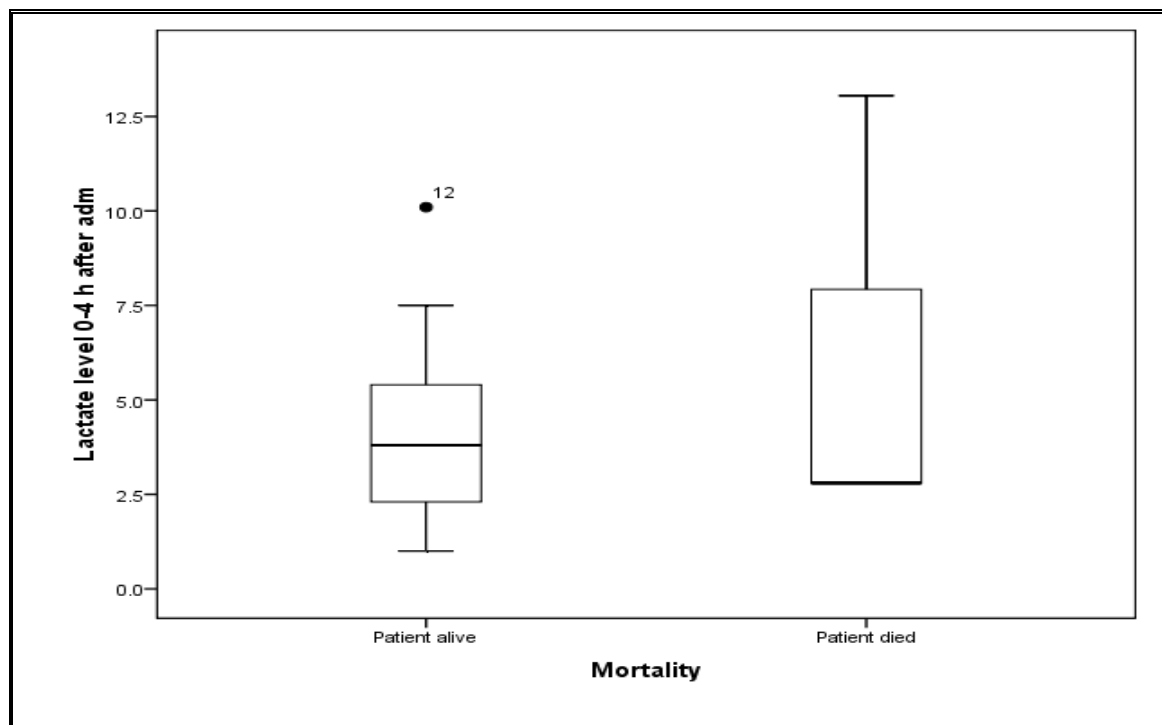


Figure 4.11: Lactate levels of patients who died 0 to 4 hours after compared to patients who survived

Figure 4.12 indicates the lactate level for the patient who died 10 to 14 hours after admission compared to the lactate levels of patients who survived.

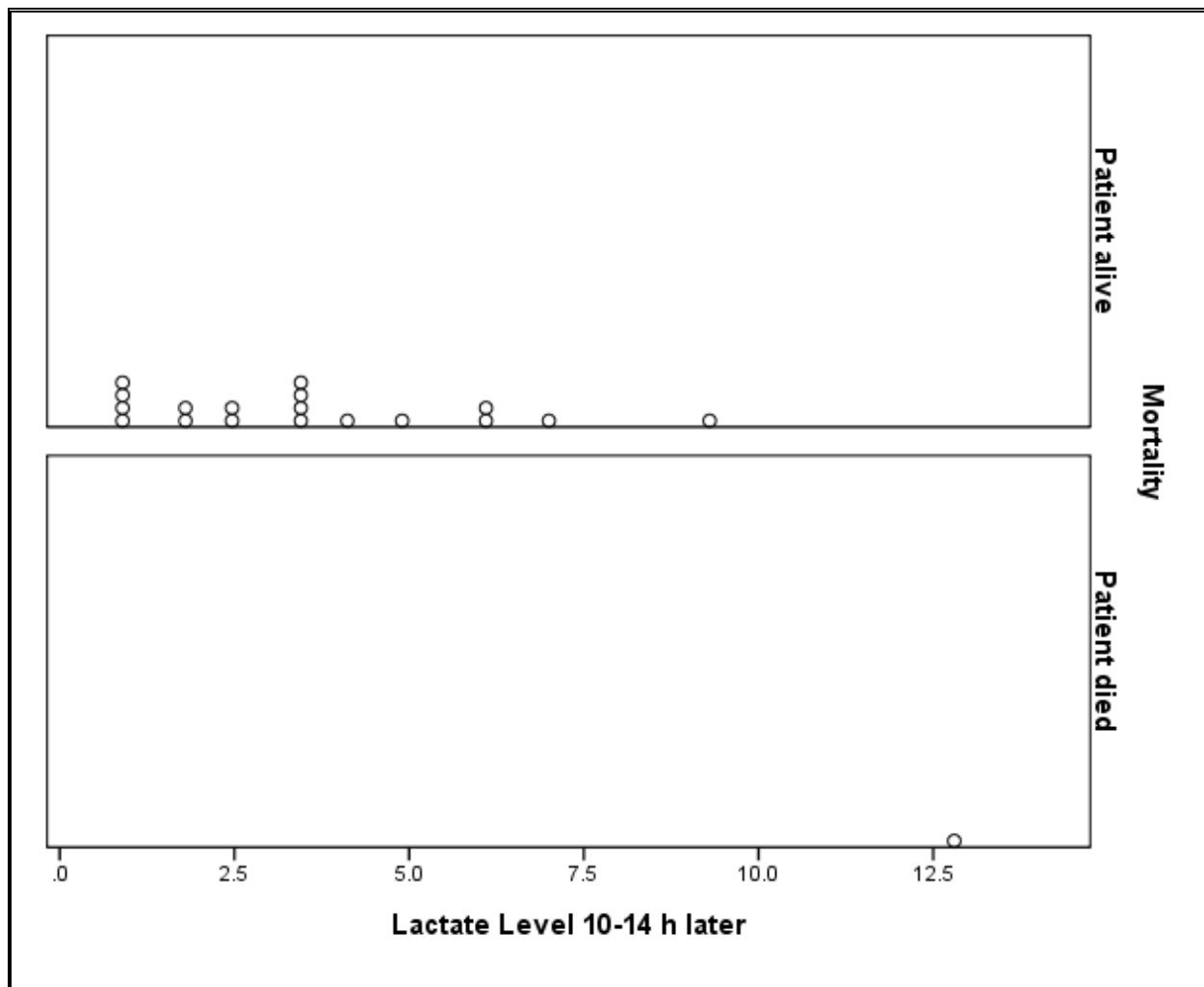


Figure 4.12: Lactate values of deceased patient compared to those of patients who survived 10 to 14 hours after admission

Discussion: Three patients died 0 to 4 hours after admission. These three patients all presented with an elevated lactate level which, according to various researchers, influences the mortality rate of patients presenting with haemorrhage caused by trauma (Englehart & Schreiber 2006: 570; Schulman 2002: 32cc4; Shirley 2010: 195). Figure 4.12 illustrates that the patient who died 10 to 14 hours after admission also presented with an elevated lactate level. These findings are significant in this research in light of the fact that all the patients who died manifested a raised lactate level, irrespective of time of death. This supports the view that an elevated lactate level is an indicator of mortality (Englehart &

Schreiber 2006: 570; Cerovic *et al.* 2003: 1302; Schulman 2002: 32cc4; Shirley 2007: 195; Shoemaker & Beez 2010: 8).

Analysis: The lactate levels recorded for those patients presenting with haemorrhage caused by trauma in the emergency unit, and of those who died indicate that lactate may, in fact, be used as a predictor of mortality for such patients. All four patients who died, irrespective of the time frame, had manifested an elevated serum lactate level. This supports the need for the revision of resuscitation strategies in order to improve the outcomes of patients presenting with haemorrhage as a result of trauma.

4.3 CONCLUSION

Chapter 5 concludes the study. It presents the findings of the research that were then statistically presented and interpreted using graphs and tables. The chapter also discussed the findings as they relate to the literature and other research findings. An analysis is then conducted which compares the findings obtained in this study with those of other research. The chapter also discusses the limitations of the study, as well as outlining recommendations, thereby laying the foundation for future research.

5 CONCLUSIONS AND RECOMMENDATIONS

5.1 INTRODUCTION

Chapter 4 contained a discussion and an analysis of the results of the study while. Chapter 5 presents the conclusions and recommendations based on these research results. The research objectives were used as a basis for the conclusions drawn.

5.2 OVERVIEW OF THE STUDY

Trauma is seen as one of the most significant causes of mortality in South Africa and, as such, causes injury and death to otherwise healthy individuals. Patients presenting with shock as a result of haemorrhage are admitted to the emergency unit of the hospital on a daily basis. These patients require immediate treatment in order to pre-empt the life-threatening complications of haemorrhagic shock.

The subtle changes in the condition of these patients as a result of compensatory mechanisms mean that haemorrhagic shock is often under diagnosed, especially in the initial stages. The measurement of physiological end points alone in patients presenting with haemorrhagic shock in the emergency unit of the hospital have been judged to be insufficient. The failure to utilise additional end points in order to monitor resuscitation is also seen as problematic in patients presenting with haemorrhage. Despite the fact that additional end points of resuscitation are not used in the emergency unit of the hospital as part of the protocols and procedures of the unit, it is however used globally. These end points include base deficit, bicarbonate, central venous oxygen saturation and lactate levels.

In view of the fact that an ongoing state of shock may have dire consequences for patients, the early recognition of the severity of shock is extremely important in order to reduce the effects of tissue hypoperfusion and hypoxia on the cells of the body. Accordingly, both resuscitation and the evaluation of the resuscitation are vital in the first 12 hours after admission. Indeed, if resuscitation is performed within the first 12 hours after injury the survival rates of these patients is almost 100%. This indicates the need for the consideration of serum lactate values in the resuscitation of patients presenting with haemorrhagic shock in an emergency unit. These serum lactate values may be used to prevent ongoing tissue hypoxia and hypoperfusion. In addition, they may also be used as a strategy with which to improve the 48-hour mortality rate of patients presenting with haemorrhagic shock in the emergency unit of the hospital.

The results obtained in respect of serum lactate values and mortality will now be discussed on the basis of the research objectives which were established.

5.3 CONCLUSIONS IN RELATION TO THE OBJECTIVES GUIDING THE STUDY

Upon completion of the data analysis the researcher drew certain conclusions based on the following research objectives:

- **Objective 1:** audit the serum lactate levels as indicated in patient files on admission as well as during a 4 to 8 hour, 10 to 14 hour and 22 to 26 hour timeframe following haemorrhage caused by trauma
- **Objective 2:** document the 48-hour mortality rate in patients presenting with haemorrhage caused by trauma
- **Objective 3:** determine the relationship between the serum lactate levels and the 48-hour mortality rate in patients presenting with haemorrhage caused by trauma

- **Objective 4:** make recommendations for future resuscitation strategies for patients presenting with haemorrhagic shock in order to improve the outcomes of such patients

5.3.1 Objective 1: Audit serum lactate levels

Objective 1 involved auditing the serum lactate levels as indicated in patient files on admission as well as during a 4 to 8 hour, 10 to 14 hour and 22 to 26 hour timeframe following haemorrhage caused by trauma. The conclusions pertaining to this objective will now be discussed.

During the first four hours following admission to the emergency unit, the average serum lactate level recorded on the clinical audit checklist was 4,092 mmol/L. The highest serum lactate level recorded was 13,1 mmol/L and this was the serum lactate level of a patient who died. The findings of the study support the contention that, overall, patients with haemorrhagic shock admitted to an emergency unit do present with an elevated serum lactate level. Serum lactate levels may, thus, serve as one of the measures which may be utilised to initiate the immediate resuscitation which is generally associated with an improved outcome.

Fifteen of the 20 serum lactate values recorded in the 4 to 8 hours following admission were above 2,0 mmol/L with the average lactate value recorded being 3,43 mmol/L. This indicates a prolonged raised serum lactate value despite fluid resuscitation. It must be borne in mind that a prolonged increased serum lactate level in patients presenting with haemorrhage always reflects underlying tissue hypoperfusion and tissue hypoxia.

Nineteen only serum lactate results were documented in the 10 to 14 hours after admission to the emergency unit. One patient with a serum lactate of 12,8 mmol/L since admission died during this time frame. The average serum lactate value

obtained from the files of the surviving patients was 3,528 mmol/L with a mean value of 3,528 mmol/L. It is evident that the serum lactate level remained raised throughout this timeframe which, in turn, indicates that hypoperfusion and hypoxia may have been present during this stage.

A raised serum lactate level 12 hours after admission to the emergency unit is regarded as a strong indicator of deteriorating survival rates. The patient who died 10 to 14 hours after admission to the emergency unit had presented with a significantly raised serum lactate value of 12,8 mmol/L.

The serum lactate values obtained from patients' files in the 22 to 26 hours after admission indicated that the average value had returned to just above normal, namely, 2,190 mmol/L. As a result of the fact that their serum lactate values had normalised within the first 24 hours after admission to the emergency unit these patients manifested an almost 100% survival rate. It, thus, remains important to evaluate the resuscitation efforts against 24-hour mortality, as this is the time frame in which the largest number of deaths as a result of hypovolaemia occur.

5.3.2 Objective 2: Document 48-hour mortality rate

The second objective of the study was to document the 48-hour mortality rate in patients presenting with haemorrhage caused by trauma. The conclusions pertaining to this objective will now be discussed.

The audit revealed that three patients died 0 to 4 hours following admission to the emergency unit. The average recorded lactate levels for these deceased patients was 6,217 mmol/L – levels which were higher than those of the patients who survived. The fourth patient died 10 to 14 hours after admission.

5.3.3 Objective 3: Determine the relationship between the serum lactate levels and the 48-hour mortality rate

The third objective of the study was to determine the relationship between the serum lactate levels and the 48-hour mortality rate of patients presenting with haemorrhage caused by trauma. The conclusions pertaining to this objective will now be discussed.

Mortality increases in patients whose lactate levels are not normalised within 48 hours. This was not consistent with the research findings, as four only of the 46 patients (9%) had died during the first 48 hours after admission and all four of these patients had presented with increased serum lactate levels.

The fact that all four of the patients who had died, irrespective of time frame, had had elevated serum lactate levels gives credence to a revision of the resuscitation strategies in order to improve the outcomes of patients presenting with haemorrhage due to trauma. Accordingly, serum lactate levels may be used to predict the mortality for patients admitted to an emergency unit with haemorrhage caused by trauma.

5.3.4 Objective 4: Recommendations

Based on the research results, recommendations will be made regarding intervention strategies in terms of which to improve the outcomes of patients admitted with haemorrhage due to trauma in an emergency unit. The researcher is of the opinion that serum lactate may be used to evaluate resuscitation efforts. In addition, it is clear from the results that further research relating to the research question should be conducted.

Recommendations will be made with regard to management, education, and nursing practice (see Sections 5.2.4.1 to 5.2.4.4).

5.3.4.1 Management

It is proposed that the resuscitation strategies in respect of patients presenting with haemorrhagic shock be changed and that this transformation start at managerial level. These changes should include the implementation of standard operating procedures and protocols for the resuscitation of patients with haemorrhagic shock. These standard operating procedures and protocols should include exact guidelines for the use and interpretation of metabolic end points such as lactate values.

Patient files should be audited on a regular basis to ensure that best practice guidelines are being followed in the resuscitation of patients presenting with haemorrhagic shock. Clinical audits have proven to be extremely valuable in guiding research and should be used to ensure evidence-based practice.

The improvement of resuscitation strategies should also be facilitated by conducting regular Mortality and Morbidity (M&M) meetings in the emergency unit of hospitals in respect of patients presenting with haemorrhagic shock. This would help improve the resuscitation of patients presenting with haemorrhage by providing personnel with constructive feedback so as to better their own practice.

5.3.4.2 Education

Education relating to haemorrhagic shock and the evaluation of resuscitation efforts should be a focus area in all emergency units throughout South Africa. This education may include programmes such as the Emergency Nursing programme or continuous professional programmes.

It is essential that nurse practitioners and healthcare professionals working in the emergency unit be knowledgeable, skilled and vigilant in respect of recognising the severity of haemorrhagic shock. Education should, thus, focus on the monitoring of physiological and metabolic end points in the evaluation of resuscitation efforts. Healthcare professionals should be made aware of the fact that monitoring and evaluating physiological end point alone is not sufficient and that additional end points such as base deficit, bicarbonate levels, central venous oxygen saturation and lactate levels are, at present, used internationally.

Nurse practitioners should also be made aware of the importance of good record keeping as this forms the basis of many studies and provides the structure for valuable information. It is, thus, essential that nurse practitioners be educated in respect of good record-keeping skills as this, in turn, will facilitate future retrospective studies such as this one.

5.3.4.3 *Nursing practice*

Haemorrhagic shock is often under diagnosed, especially during the initial stages. This under diagnosis is due mainly to the subtle changes which may come about in a patient's condition as a result of the compensatory mechanisms. Nurse practitioners should be aware of the importance of early recognition and the use of end points in monitoring these patients.

It is important to assess and to reassess patients admitted as a result of trauma on a regular basis as this will allow the nurse practitioner to pick up subtle changes in the patient's condition. During assessment the nurse practitioner should not only take the physiological end points into consideration, but also the metabolic end points as this will ensure that ongoing tissue perfusion and hypoxia are monitored and evaluated.

It is essential that nurse practitioners become involved in clinical audits to monitor patient management and outcome. Participation in clinical governance teams would facilitate the documentation of patient management the monitoring of outcomes and the planning of actions in order to improve clinical practice.

5.3.4.4 *Multidisciplinary approach*

During the study the researcher realised that multidisciplinary team often form clinical governance teams, which then take responsibility for auditing patient management and outcomes. This could be beneficial in the emergency unit in which this study was conducted.

It is recommended that more members of the multi-disciplinary team be involved in the auditing process, in evaluating the results of the process and in suggesting and implementing change in emergency units in order to bring about change and improve patient outcomes.

5.4 FUTURE RESEARCH

It is recommended that future research be conducted in the emergency unit used for this study based on the research results. The topics for future research could include:

- resuscitation management for patients with haemorrhagic shock
- point-of-care lactate monitoring of patients presenting with haemorrhagic shock in the emergency unit
- importance of evaluating more than one end point of resuscitation in patients presenting with haemorrhagic shock

5.5 PERSONAL REFLECTION

In my role as an emergency nurse practitioner working in a busy emergency unit, the resuscitation of patients presenting with haemorrhagic shock is something with which I deal on a regular basis. I firmly believe that, in view of the fact that haemorrhagic shock is common in South Africa and because it presents in often otherwise healthy individuals, it is of the utmost importance that these patients receive optimal care.

Although I was very familiar with the research topic, the study did not progress without challenges. The biggest challenge I faced in this study was retrieving the data for my study. In view of the fact that this was a retrospective study, I was forced to rely solely on patient charts that had been completed years before the study commenced. One of the difficulties which I faced was the fact that the patient charts that contained incomplete or faulty information could not be used as part of the research, thus negatively influencing the size of the study sample.

Another challenge involved both the record-keeping system itself, as well as the personnel who assisted me with the retrieving of records. This was a very haphazard system and prone to human error. Despite the fact that some of the staff members working in some sections of the record department were very helpful others were not interested in assisting me with the retrieval of patient records and this directly influenced the time spent on data collection and, in fact, delayed the process for months. I carried out the data collection on my own, and I found it extremely tiring, frustrating and difficult to manage by myself. In retrospect, it would have been advisable to include other emergency nurse practitioners in this process.

The use of a clinical audit to revise patient cases and documentation was a new concept introduced into the emergency unit of the hospital. It would, however, have been advisable that personnel working in the emergency unit of the hospital be

made familiar with clinical audits in order to help improve record-keeping skills and patient care by reviewing mistakes made and successes experienced.

The research methodology has taught me that, although there may be fewer ethical implications in conducting a retrospective study, there are several variables which may influence the outcome of the research. The variables which played a role in influencing this research included the following:

- o extraneous variable – the blood gas machine which was used to obtain the lactate levels which were used in the research. This machine had not been regularly serviced and maintained, which meant that lactate results were not always available as part of the blood gas analysis. In addition, printing paper for the blood gas machine was not always available which, in turn, meant that lactate levels were not always written down. Another extraneous variable was the fact that the patient report form used for patients presenting in the emergency unit of the hospital did not always require that a lactate level be documented. This meant that the lactate levels for some of the patients presenting with haemorrhagic shock were not always documented.

5.6 SUMMARY

In Chapter 5 the findings of the research were synthesised in order to make it easier to generalise the research findings. Although this study did not produce statistically significant results, the study may, nevertheless, be seen as a stepping stone in the improved care of patients presenting with haemorrhagic shock.

The research was aimed at explaining the relationship between lactate values and mortality in patients with haemorrhagic shock admitted to the emergency unit. It also served as a basis on which to make recommendations for future resuscitation strategies for patients presenting with haemorrhagic shock in order to improve the outcomes of such patients.

The findings of the research indicate the need to take into account serum lactate values in the resuscitation of patients with haemorrhagic shock in an emergency unit. These serum lactate values may be used to prevent ongoing tissue hypoxia, hypoperfusion and subsequent death. It is proposed that, if the resuscitation strategies in respect of patients with haemorrhagic shock were changed to include additional metabolic end points such as serum lactate levels, the outcomes of these patients would improve. This implies that standard operating procedures and protocols for the resuscitation of patients with haemorrhagic shock should be implemented and that these standard operating procedures and protocols should include exact guidelines for the use and interpretation of metabolic end points such as serum lactate values.

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BIBLIOGRAPHY

A

Acosta, J.A., Yang, J.C., Winchell, R.J., Simons, R.K., Fortlage, D.A., Hollingsworth-Fridlund, P. & Hoyt, D.B. 1998. Lethal injuries and time to death in a Level I trauma center. *Journal of the American College of Surgeons*, 186(5): 528–533.

American College of Surgeons. 2008. *Advanced trauma life support (ATLS)*. [Online]. Available: <http://www.facs.org/trauma/atls/index.html> [Accessed: 12 December 2008].

American College of Surgeons Committee on Trauma. 2005. *Advanced trauma life support for doctors (ATLS): Student course manual*. 7th edition. Chicago: American College of Surgeons.

Anderson, L.E. 1995. *Mosby's pocket dictionary of nursing, medicine & professions allied to medicine*. London: Mosby.

Athey, J., Dean, M., Ball, J., Wiebe, R. & Melese-d'Hospital, I. 2001. Ability of hospitals to care for pediatric emergency patients. *Pediatric Emergency Care*, 17(3): 170–174.

B

Babbie, E. 2005. *The basics of social research*. 3rd edition. Belmont: Thomson Wadsworth.

Babbie, E. & Mouton, J. 2001. *The practice of social research*. Cape Town: Oxford University Press.

Bakker, J. 1999. Blood lactate levels. *Current Opinion in Critical Care*, 5(3): 234–248.

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

Barbee, R.W., Reynolds, P.S. & Ward, K.R. 2010. Assessing shock resuscitation strategies by oxygen debt repayment. *Shock*, 33(2): 113–122.

Bilkovski, R.N, Rivers, E., & Horst, H.M. 2004. Targeted resuscitation strategies after injury. *Current Opinion in Critical Care*, 10(6): 529–538.

Boon, N.A., Colledge, N.R. & Walker, B.R. 2006. *Davidson's principals and practice of medicine*. 20th edition. New York: Elsevier.

Brasel, K.J., Guse, C., Gentilello, L.M. & Nirula, R. 2007. Heart rate: is it a true vital sign? *The Journal of TRAUMA: Injury, Infection, and Critical Care*, 62(4): 812–817.

Brink, H., Van der Walt, C. & Van Rensburg, G. 2006. *Fundamentals of research methodology for health care professionals*. 2nd edition. Cape Town: Juta.

Bryman, A. 2004. *Social research methods*. 2nd edition. New York: Oxford University Press.

Burns, N. & Grove, S.K. 2005. *The practice of nursing research: Conduct, critique and utilization*. 5th edition. Missouri: Saunders Elsevier.

Burns, N. & Grove, S.K. 2007. *Understanding nursing research: Building an evidence-based practice*. 4th edition. Missouri: Elsevier.

Burns, N. & Grove, S.K. 2009. *The practice of nursing research: Appraising, synthesis, and generation of evidence*. 6th edition. Missouri: Saunders Elsevier.

C

Cerovic, O., Golubovic, V., Spec-Marn, A., Kremzar, B., Vidmar, G. 2003. Relationship between injury severity and lactate levels in severely injured patients.

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

Intensive Care Medicine, 29: 1300–1305.

Colburn, V. 1999. Trauma in pregnancy. *Journal of Perinatal & Neonatal Nursing*, 13(3): 21–32.

Collicott, P.E. 2005. ATLS celebrating 25th anniversary. *Bulletin of the American College of Surgeons*, 90(4): 17–21. [Online]. Available: http://www.facs.org/fellows_info/bulletin/2005/collicott0505.pdf [Accessed 26 August 2007].

Collins English Dictionary. 2006. Concise edition. Glasgow: HarperCollins.

Collins, T. 2000. Understanding shock. *Nursing Standard*, 14(49): 35–41.

Creswell, J.W. 2003. *Research design: Qualitative, quantitative and mixed methods approaches*. 2nd edition. University of Nebraska, Lincoln: Sage Publications.

D

DeLong, W.G. & Born, C.T. 2004. Cytokines in patients with polytrauma. *Clinical Orthopaedics and Related Research*, 422: 57–65.

Department of Health. 2006. *Health facility definitions*. [Online]. Available: <http://www.doh.gov.za/docs/misc/hfd-f.html> [Accessed: 2 May 2007].

De Vos, A.S., Strydom, H., Fouche, C.B. & Delpont, C.S.L. 2002. *Research at grass roots for the social sciences and human service professions*. 2nd edition. Pretoria: Van Schaik.

Dick, W.F. & Baskett, P.J.F. 1999. Recommendations for uniform reporting of data following major trauma — the Utstein style: a Report of a Working Party of the

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

International Trauma Anaesthesia and Critical Care. *Resuscitation*, 42(2): 81–100.

Dorland, W.A.N. 2001. *Dorland's pocket medical dictionary*. Philadelphia, PA: Saunders/Harcourt Health Science.

E

Eastridge, B.J., Salinas, J., McManus, J.G., Blackburn, L., Bugler, E., Cooke, W.H., Concertino, V.A., Wade, C.E. & Holcomb, J.B. 2007. Hypotension begins at 110mmHg: Redefining "hypotension" with data. *The Journal of TRAUMA: Injury, Infection, and Critical Care*. 63(2): 291–299.

Encarta. 2003. *Dictionary tools*. Microsoft, United States of America.

Englehart, M.S. & Schreiber, M.A. 2006. Measurement of acid-base resuscitation endpoints: Lactate base deficit, bicarbonate or what? *Current Opinion in Critical Care*, 12(6): 569–574.

Erasmus, R.T. & Zemlin, A.E. 2009. Clinical audits in the laboratory. *Journal of Clinical Pathology*, 62: 593–597. [Online]. Available: <http://www.jcp.bmjournals.com/cgi/content/full/62/7/593> (Accessed: April 2009)

F

Flokstra-de Bok, B.M.J. & Dubois, A.E.J. 2009. Quality of life in food allergy: Valid scales for children and adults. *Current Opinion in Allergy and Clinical Immunology*, 9: 214–221.

G

Garner, J., Watts, S., Parry, C., Bird, J., Cooper, G. & Kirkman, E. 2010. Prolonged permissive hypotensive resuscitation is associated with poor outcome in primary

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

blast injury with controlled hemorrhage, *Annals of Surgery*, 251(6): 1131–1139.

Gauteng Provincial Health: The Hospital. (2004). Patient statistics. Unpublished: The Hospital.

Gearing, R.E., Mian, I.A., Barber, J.B. & Ickowicz, A. 2006. A methodology for conducting retrospective chart review. *Research in Child and Adolescent Psychiatry*, 15(3): 126–134.

Gerrish, K. & Lacey, A. 2010. The research process in nursing. 6th edition. Oxford: Wiley-Blackwell.

Giannoudis, P.V., Hildebrand, F. & Pape, H.C. 2004. Inflammatory serum in patients with multiple trauma: Can they predict outcome? *British Editorial Society of Bone and Joint Surgery*, 86-B(3): 313–323.

Gonzales, E.A. 2008. Fluid resuscitation in the trauma patient. *Journal of Trauma Nursing*, 15(3): 149–157.

Gottschalk, S.B., Wood, D., DeVries, S., Wallis, L.A. & Bruijns, S. 2006. The Cape Triage Score: A new triage system South Africa. Proposal from the Cape Triage Group. *Emergency Medicine Journal*, 23(2): 149–153.

Greaves, I. & Johnson, G. 2002. *Practical emergency medicine*. New York: Arnold.

Green, J. & Thorogood, N. 2004. *Qualitative methods for health research*. London: Sage.

Gutierrez, G., Reines, D.H. & Wulf-Gutierrez, M.E. 2004. Clinical review: hemorrhagic shock. *Critical Care*, 8(5): 373–381.

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

H

Halaas, Y. 2006. General approach to the trauma patient. *eMedicine from web MD*. [Online]. Available: <http://www.emedicine.com/ent/TOPIC492.HTM> [Accessed 24 August 2007].

Hill, G. & Davies, K. 2002. *Blunt chest trauma: a challenge to accident and emergency nurses*. *Accident and Emergency Nursing*, 10: 197-204.

Hofstee, E. 2006. *Constructing a good dissertation: A practical guide to finishing masters, MBA or PhD on schedule*. Sandton: EPE.

Hoyt, K.S. & Selfridge-Thomas, J. (Eds). 2007. *Emergency Nurses Association: Emergency nursing core curriculum*. 6th edition. Missouri: Saunders Elsevier.

Husain, F., Martin, M.J., Mullenix, P.S., Steele, S.R. & Elliott, D.C. 2003. *Serum lactate and base deficit as predictors of mortality and morbidity*. Scientific paper: Department of Surgery, Madigan Army Medical Centre, Fort Lewis, WA 98431, USA.

I

Irwin, R.S., Rippe, J.M. & Goodheart, H.P. 2003. *Irwin and Rippe's intensive care medicine*. 5th edition. Philadelphia, PA: Lippincott Williams & Wilkins.

J

Jacobs, B.B. & Hoyt, K.S. 2000. *Trauma Nursing Core Curriculum (TNCC) Provider Manual*. 5th edition. Emergency Nurses Association. Missouri: Saunders Elsevier.

Jansen, T.C., van Bommel, J. & Bakker, J. 2009. Blood lactate monitoring in critically ill patients: A systematic health technology assessment. *Critical Care Medicine*, 37(10): 2827-2839.

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

Johnston, G., Crombie, I.K., Davies, H.T.O., Alder, E.M. & Millard, A. 2000. Reviewing audit: Barriers and facilitating factors for effective clinical audit. *Quality in Health Care*, 9(1): 23–36.

Jones, A.E. & Kline, J.A. 2006. Shock. In: Marx, J.A., Hockberger, R.S. & Walls, R.M. 2006. *Rosen's emergency medicine: concepts and clinical practice*. 6th edition. Philadelphia: Mosby Elsevier.

K

Kane, S.K., MacCallum, M.J. & Friedrich, A.D. 2007. Resuscitation of the trauma patient. *International Anesthesiology Clinics*, 45(3): 61–81.

Kaplow, R. & Hardin, S.R. 2007. *Critical care nursing: Synergy for optimal outcomes*. London: Jones and Barlett.

Kauvar, D.S., Lefering, R. & Wade, C. 2006. Impact of hemorrhage on trauma outcome: An overview of epidemiology, clinical presentation, and therapeutic considerations. *The Journal of Trauma: Injury, Infection and Critical Care*, 60(6): S3-S11.

Kwan, I., Bunn, F. & Roberts, I. 2003. Timing and volume of fluid administration for patients with bleeding. *Cochrane Database of Systematic Reviews*, 3(CD002245.DOI: 10.1002/14651858.CD002245).

L

LoBiondo-Wood, G. & Haber, J. 2006. *Nursing research: Methods and critical appraisal for evidence-based practice*. Missouri: Mosby Elsevier.

Luft, F.C. 2001. Lactic acidosis update for critical care clinicians. *Journal of the American Society of Nephrology*, 12: S15–S19.

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

M

Marieb, E.N. 2007. *Human anatomy & physiology: International edition*. 7th edition. San Francisco: Pearson Benjamin Cummings.

McCartney, P.R. 2009. Audit trails and electronic record discovery. *The New Networking*, 34(1): 64.

McMillan, J.H. & Schumacher, S. 2001. *Research in education: A conceptual introduction*. 5th edition. New York: Addison, Wesley Longman.

McNelis, J, Marini, C.P., Jurkiewicz, A., Szomstein, S., Ritter, G., Nathan, I.M. 2001. Prolonged lactate clearance is associated with increased mortality in the surgical intensive care unit. *The American Journal of Surgery*, 182(5): 481–485.

McQuillan, K.A., Makic, M.B.F. & Whalen, E. 2009. *Trauma Nursing: from resuscitation through rehabilitation*. Missouri: Saunders Elsevier.

Meij, H.S. & van Papendorp, D.H. 1997. *Concise physiology: An up to date and complete review of human physiology*. Pretoria: Kagiso Tertiary.

Merriam-Webster Online Dictionary. 2007. [Online]. Available: <http://merriam-webster.com/dictionary> [Accessed: 14 January 2010].

MercSource-Health Information. 2009. *Pure and Simple. Online Dictionary*. [Online]. Available: http://www.mercksource.com/pp/us/cns/cns_home.jsp [Accessed: 19 March, 2010].

Morton, P.G., Fontaine, D.K., Hudak C.M. & Gallo, B.M. 2005. *Critical care nursing: A holistic approach*. 8th edition. Philadelphia: Lippincott Williams & Wilkins.

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

N

National Department of Health. 2006. Health facility definitions. [Online]. Available: <http://www.doh.gov.za/docs/misc/hfd-f.html> [Accessed: 2 May 2007].

Nicol, A. & Steyn, E. 2004. *Handbook of trauma for Southern Africa*. Oxford: Oxford University Press.

Nichol, A.D, Egi, M., Pettila, V., Bellomo, R., French, C., Hart, G., Davies, A., Stachowski, E., Reade, M.C., Bailey, M. & Cooper, D.J. 2010. Relative hyperlactatemia and hospital mortality in critically ill patients: a retrospective multi-centre study. *Critical Care*, 14: R25. [Online]. Available from: <http://ccforum.com/content/14/1/R25>. [Accessed 9 July 2010].

O

Offredy, M. & Vickers, P. 2010. *Developing a healthcare research proposal: an interactive student guide*. Oxford: Wiley-Blackwell.

O'Shea, R.A. 2005. *Principles and practice of trauma nursing*. London: Elsevier Churchill Livingstone.

Oxford English Dictionary. 2005. 6th edition. Oxford: University Press.

P

Peitzman, A.B., Rhodes, M., Yearly, D.M., Schwab, C.W. & Fabian, T. 2002. *The trauma manual*. 2nd edition. Lippincott Williams & Wilkins.

Polit, D.F. & Beck, C.T. 2004. *Nursing research: Principals and methods*. 7th edition. Philadelphia: Lippincott Williams & Wilkins.

Polit, D.F. & Beck, C.T. 2006. *Essentials of nursing research: Methods, appraisal,*

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

and utilization. Philadelphia: Lippincott Williams & Wilkins.

Polit, D.F. & Beck, C.T. 2008. *Nursing research: Principals and methods.* 8th ed. Philadelphia: Lippincott Williams & Wilkins.

Polit, D.F. & Beck, C.T. 2010. *Essentials of nursing research: Appraising evidence for nursing practise.* 7th ed. Philadelphia: Lippincott Williams & Wilkens.

Porter, J.M. & Ivatury, R.R. 1998. In search of optimal end points of resuscitation in trauma patients: a review. *The Journal of TRAUMA: Injury, Infection and Critical Care*, 44(5): 908–914.

Proehl, J.A. 2009. *Emergency nursing procedures.* 4th edition. Saunders: Elsvier.

R

Rady, M.Y., Rivers, E.P. & Nowak, R.M. 1996. Resuscitation of the critically ill in the ED: Responses of blood pressure, heart rate, shock index, central venous oxygen saturation, and lactate. *American Journal of Emergency Medicine*, 14(2): 218–225.

Rivers, E.P., Ander, D.S. & Powell, D. 2001. Central venous oxygen saturation monitoring in the critically ill patient. *Current Opinion in Critical Care*, 7(3): 204–211.

Ruffolo, D.C. 2009. Trauma care and managing the injured pregnant patient. *Journal of Obstetric, Gynecologic, and Neonatal Nursing*, 38(6): 704–714.

S

Sale, J.A., Lohfeld, L.H. & Brazil, K. 2002. Revisiting the qualitative-quantitative debate: implications for mixed-methods research. *Quality & Quantity*, 36: 43–53.

Salks, M. & Allsop, J. 2007. *Researching health: qualitative, quantitative, and mixed*

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

methods. London: Sage.

Santry, H.P. & Halam, H.B. 2010. Fluid resuscitation: past, present, and the future. *Shock*, 33(3): 229–241.

Schulman, C. 2002. Is your patient fully resuscitated? Here's how to find out. *Nursing*, 32(5): 32cc1–32cc8.

Shafi, S. & Kauder, D.R. 2004. Fluid resuscitation and blood replacement in patients with polytrauma. *Clinical Orthopaedics & Related Research*, 422: 37–42.

Shiler, K.C. & Napolitano, L.M. 2010. Complications of massive transfusion. *Chest*, 137: 209–220.

Shirley, T.L. 2007. POC lactate: a marker for diagnosis, prognosis, and guiding therapy in the critically ill. *Point of Care*, 6(3): 192–200.

Shoemaker, W.C. & Beez, M. 2010. Pathophysiology, monitoring, and therapy of shock with organ failure. *Applied Cardiopulmonary Pathophysiology*, 14: 5–15.

Smeltzer, S.C., Bare, B.G., Hinkle, J.L. & Cheever, K.H. 2008. *Brunner & Suddarth's textbook of medical-surgical nursing*. 11th edition. Lippincott Williams & Wilkins.

Somekh, B. & Lewin, C. 2005. *Research methods in the social sciences*. London: Sage.

Soreide, E. & Grande, C.M. 2001. *Prehospital trauma care*. Buffalo: New York .

South Africa. 2003. *National Health Act (Act 61 of 2003)*. Pretoria: Government Printers.

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

South African HealthInfo-Health knowledge network. 2005. 7th Annual Report of the National Injury Mortality Surveillance System 2005 section 5. Tshwane / Pretoria metropolitan area [Online]. Available: <http://www.sahealthinfo.org/violance/2005injury.htm> [Accessed 27 March 2010].

Spahn, D.R., Cerny, V., Coats, T.J., Duranteau, J., Fernandez-Mondejar, E., Gordini, G., Stahel, P.F., Hunt, B.J., Komadina, R., Neugebauer, E., Ozier, Y., Riddez, L., Shultz, A., Vincent, J & Rossaint, R. 2007. *Management of bleeding after major trauma: a european guideline*. *Critical Care*, 11(1). [Online]. Available <http://ccforum.com/content/11/1/R17> [Accessed: 20 September 2010]

Spinella, P.C. & Holcomb, J.B. 2009. Resuscitation and transfusion principles for traumatic hemorrhagic shock. *Blood Reviews*, 23: 231–240.

Stahel, P., Heide, C.E. & Ertel, W. 2005. Current concepts of polytrauma management. *European Journal of Trauma*, 31: 200–211.

Stedman's Medical Dictionary for the Health Professions and nursing: Illustrated. 2005. 5th edition. Baltimore: Lippencott Williams & Wilkins.

Stommel, M. & Wills, C.E. 2004. *Clinical research: Concepts and principals for advanced practice nurses*. Philadelphia: Lippincott.

Stone, C.K. & Humphries, R.L. 2008. *Current emergency diagnosis & treatment*. 5th edition. London: McGraw-Hill.

Struwig, F.W. & Stead, G.B. 2001. *Planning, designing and reporting research*. Cape Town: Maskew Miller Longman/Pearson.

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

T

Terre Blanche, M. & Durrheim, K. 2004. *Research in practice: Applied methods for the social sciences*. Cape Town: University of Cape Town.

The Hospital [Online]. [Accessed 29 June 2010].

Tintinalli, J.E., Kelen, G.D. & Stapczynski, J.S. 2004. *Emergency medicine: A comprehensive study guide*. 6th edition. London: McGraw-Hill.

Tisherman, S.A., Barie, P., Bokhari, F., Bonadies, J., Daley, B., Diebel, L., Eachempati, S.R., Kurek, S., Luchette, F., Puyana, J.C., Schreiber, M. & Simon, R. 2004. Clinical practice guidelines: Endpoints of resuscitation. *The Journal of TRAUMA: Injury, Infection and Critical Care*, 57: 898-912.

Trochim, W.M. 2006. *The research methods knowledge base*. [Online]. Available: <http://www.socialresearchmethods.net.kb> [Accessed 20 September 2009].

U

Urden, L.D, Stacy, K.M. & Lough, M.E. 2006. *Thelan's critical care nursing: Diagnosis and management*. 5th edition. Philadelphia: Mosby Elsevier.

V

Van Eeden, I.E. 2010. Personal communication. 11 June, Clinical facilitator, The Hospital, Pretoria.

Van Niekerk, L.R. 2007. Personal communication. 2 November, Middle manager, The Hospital, Pretoria.

Victorino, G.P., Battistella, F.D. & Wisner, D.H. 2003. Does tachycardia correlate

Relationship between lactate values and mortality in patients with haemorrhagic shock in an emergency unit

with hypotension after trauma? *American College of Surgeons*, 196(5): 679-684.

W

World Health Organization (WHO). 2001b. *Health research methodology: a guide for training in research methods*. 2nd ed. Manila: World Health Organization.

Y

Yauch, C.A. & Steudel, H.D. 2003. Complementary use of qualitative and quantitative cultural assessment methods. *Organizational Research Methods*, 6(4): 465-481.

ANNEXURE A
(Ethical approval to conduct the research)

ANNEXURE A.1

(Ethical committee University of Pretoria)

Faculty of Health Sciences Research Ethics Committee

28/01/2009

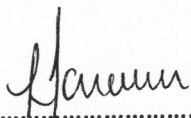
Number	:	S2/2009
Title	:	A correlation study between lactate values and mortality in patients presenting with haemorrhagic shock in an accident and emergency unit
Investigator	:	E N Zarrabi, Department of Nursing Science, University of Pretoria (SUPERVISORS: DR T HEYNS / PROF SP HATTINGH)
Sponsor	:	None
Study Degree:		M. Cur (Trauma and Emergency Nursing Science)

This Student Protocol has been considered by the Faculty of Health Sciences Research Ethics Committee, University of Pretoria on 27/01/2009 and found to be acceptable.

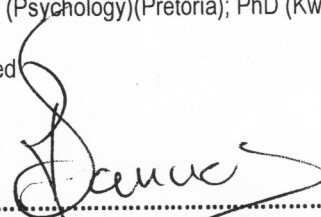
Dr AG Nienaber	(female) BA (Hons) (Wits); LLB (Pretoria); LLM (Pretoria); LLD (Pretoria); Diploma in Datometrics (UNISA)
Prof V.O.L. Karusseit	MBChB; MFGP (SA); M.Med (Chir); FCS (SA)
Prof J A Ker	Deputy Dean: MBChB (Pretoria); MMed (Int) (Pretoria); MD (Pretoria)
Prof M Kruger	CHAIRPERSON (female) MBChB.(Pretoria) M. Med.Paed.(Pretoria) M. Phil. (Applied Ethics) (Stell) PhD.(Leuven)
Dr N K Likibi	MBChB.; Med.Adviser (Gauteng Dept. of Health)
Dr T S Marcus	(female) BSc (LSE), PhD (University of Lodz, Poland)
Mrs M C Nzeku	(female) BSc (NUL); MSc Biochem (UCL,UK)
Snr Sr J. Phatoli	(female) BCur (Et.AI); BTech Oncology
Mr Y M Sikweyiya	MPH (Umea University Umea, Sweden); Master Level Fellowship (Research Ethics) (Pretoria and UKZN); Post Grad. Diploma in Health Promotion (Unitra); BSc in Health Promotion (Unitra)
Dr L Schoeman	(female) BPharm (North West); BAHons (Psychology)(Pretoria); PhD (KwaZulu-Natal); International Diploma in Research Ethics (UCT)
Dr R Sommers	(female) MBChB; M.Med (Int); MPhar.Med
Prof C W van Staden	MBChB (Pretoria); MMed(Psych) (Pretoria); MD (Warwick,UK); FCPsych (SA); FTCL (London); UPLM (UNISA)
Prof TJP Swart	BChD, MSc (Odont), MChD (Oral Path)
Dr AP van der Walt	BChD, DGA (Pretoria)

Student Ethics Sub-Committee

Prof R S K Apatu	MBChB (Legon,UG); PhD (Cantab); PGDip International Research Ethics (UCT)
Dr A M Bergh	(female) BA (RAU); BA (Hons) (Linguistics) (Stell); BA (Hons) (German) (UNISA); BEd (Pretoria); PhD (Pretoria); SED (Stell)
Mrs N Briers	(female) BSc (Stell); BSc Hons (Pretoria); MSc (Pretoria); DHETP (Pretoria)
Dr S I Cronje	BA (Pretoria); BD (Pretoria); DD (Pretoria)
Dr M M Geyser	(female) MBChB (Pretoria); BSc (Computer Science)(Pretoria); BSc Hons (Pharm) (Potchefstroom); MpraxMed (Pretoria); MSc (Clinical Epidemiology) (Pretoria); FCEM (SA); Dip PEC (SA)
Prof Daleen Millard	(female) B.lur (Pretoria); LLB (Pretoria); LLM (Pretoria); AIPSA Diploma in Insolvency Law (Pretoria); LLD (UJ)
Dr S A S Olorunju	BSc (Hons). Stats (Ahmadu Bello University –Nigeria); MSc (Applied Statistics (UKC United Kingdom); PhD (Ahmadu Bello University – Nigeria)
Dr L Schoeman	CHAIRPERSON (female) BPharm (North West); BAHons (Psychology)(Pretoria); PhD (KwaZulu-Natal); International Diploma in Research Ethics (UCT)
Dr R Sommers	SECRETARIAT (female) MBChB; M.Med (Int); MPhar.Med



.....
DR L SCHOEMAN; BPharm, BA Hons (Psy), PhD;
 Dip. International Research Ethics
CHAIRPERSON of the Faculty of Health Sciences
 Student Research Ethics Committee, University of Pretoria



.....
DR R SOMMERS; MBChB; M.Med (Int); MPhar.Med
SECRETARIAT of the Faculty of Health Sciences
 Research Ethics Committee, University of Pretoria

ANNEXURE A.2 (The Hospital)

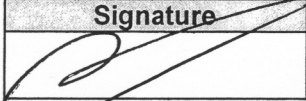
INITIAL CONSENT BY DEPARTMENTAL HEAD

I A. Engelbrecht head of EMERGENCY UNIT department of STEVE BIKO ACADEMIC hospital in consultation with the Chief Executive Officer / Superintendent of this Hospital grant permission to submit an application to conduct a clinical trial/evaluation to the Chairperson (s) of the relevant Ethics, Research and Therapeutic Committees of this Hospital.

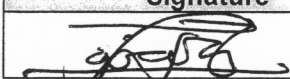
The officer conducting the trial/evaluation will be E.N. ZARRABI

Designation / Rank PROFESSIONAL NURSE

THE HEAD OF THE DEPARTMENT MUST SIGN HERE!

HEAD OF DEPARTMENT			DATE		
Signature	Initial(s)	Surname	Day	Month	Year
	A.	Engelbrecht	19	01	09

THE APPLICANT MUST SIGN HERE

TRIALIST-INVESTIGATOR			DATE		
Signature	Initial(s)	Surname	Day	Month	Year
	E.N.	ZARRABI	19	01	09

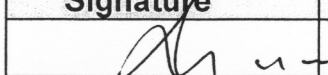
THE APPLICANT THAT APPLY FOR THIS STUDY MUST SEE TO IT THAT THE SUPERINTENDENT / C.E.O. OF THE HOSPITAL WHERE THE STUDY WILL BE DONE - SIGN HERE BEFORE THE ETHICAL COMMITTEE RECEIVE THIS APPLICATION FORM.

APPROVAL BY HOSPITAL CHIEF EXECUTIVE OFFICER:

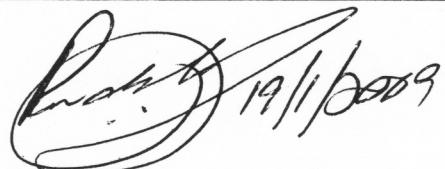
I Dr N. T. Sebastian Chief Executive Officer / superintendent of STEVE BIKO ACADEMIC Hospital, hereby agree that this trial / evaluation be conducted in the EMERGENCY Department of this hospital.

The officer conducting the trial will be : E . N . ZARRABI

The officer controlling supplies will be: DR A. ENGELBRECHT

HOSPITAL C.E.O / Superintendent			DATE		
Signature	Initial(s)	Surname	Day	Month	Year
	N.T.	SEBASTIAN	19	01	2009

19/1/2009

 19/1/2009

ANNEXURE B

(Clinical audit checklist)

ANNEXURE B.1

Clinical audit checklist used for Pre-test

Clinical audit checklist

		For Office Use Only
Audit number		V1 <input type="text"/>
Section A: Demographic information		
1.	Date of admission in A&E unit	YYMMDD
2.	Gender	V2 <input type="text"/>
	Male	1
	Female	2
3.	Date of birth	YYMMDD
Section B: 24-hour serum lactate levels		
1.	Admission time to A&E unit	HHMM
0-4hours after admission		
2.	Admission lactate level	mmol/L
3.	Document lactate levels at:	
3.1	4 to 8 hours after admission	
	Time	HHMM
	Level	mmol/L
<i>Document available lactate level closest to 6 hours</i>		
3.2	10 to 14 hours after admission	
	Time	HHMM
	Level	mmol/L
3.3	22 to 26 hours after admission (closest to 24 hours)	
	Date	YYMMDD
	Time	HHMM
	Level	mmol/L
<i>Document available lactate level closest to 24 hours and keep within the 24 hour limit</i>		
Section C: 48-hour mortality		
1.	Date of death	YYMMDD
	If patient did not die within first 48-hours following admission, leave space open please	
2.	Time of death	HHMM

ANNEXURE B.2

Clinical audit checklist used for research

Clinical audit checklist

For Office Use Only

Audit number

V1

Section A: Demographic information

1.	Date of admission in A&E unit	YYMMDD
2.	Gender	
	Male	1
	Female	2
3.	Date of birth	YYMMDD

V2 y y m m d d

V3

V4 y y m m d d

Section B: 24-hour serum lactate levels

1.	Admission time to A&E unit	HHMM
0-4hours after admission		
2.	Admission lactate level	mmol/L
3.	Document lactate levels at:	
3.1	4 to 8 hours after admission	
	Time	HHMM
	Level	mmol/L
<i>Document available lactate level closest to 6 hours</i>		
3.2	10 to 14 hours after admission	
	Time	HHMM
	Level	mmol/L
3.3	22 to 26 hours after admission (closest to 24 hours)	
	Date	YYMMDD
	Time	HHMM
	Level	mmol/L
<i>Document available lactate level closest to 24 hours and keep within the 24 hour limit</i>		

V5 h h m m

V6

V7 h h m m

V8

V9 h h m m

V10

V11 y y m m d d

V12 h h m m

V13

Section C: 48-hour mortality

1.	Date of death	YYMMDD
If patient did not die within first 48-hours following admission, leave space open please		
2.	Time of death	HHMM
3.	Type of incident	

V14 y y m m d d

V15 h h m m

V16

ANNEXURE C

(Letter from the statistician)

LETTER OF STATISTICAL SUPPORT

Date: 2008/10/24

This letter is to confirm that the student, **Ms N Zarrabi (20047348)**, studying at the University of Pretoria discussed the Project with the title:


“Correlation between lactate and mortality in haemorrhagic shock” with me.

I hereby confirm that I am aware of the project and also undertake to assist with the statistical analysis of the data generated from the project.

The DATA ANALYSIS will include descriptive statistics to summarize the findings (frequency tables, medians, modes), as well as point biserial correlation coefficients to evaluate the correlation between serum lactate levels and mortality.

The SAMPLE SIZE CALCULATION was made as follows: A simple random sampling will be conducted.

Name Dr Lizelle Fletcher

Signature 

Date 24 October 2008