THE PREVENTION OF TRANSCIENT HYPOXAEIA
DURING THE SUCTION PROCEDURE
OF MECHANICALLY VENTILATED NEONATES

by

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To my Lord and saviour
who guides me
and gives me
strength

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SUMMARY

Suctioning of endotracheal tubes, in premature infants with respiratory distress syndrome, who are undergoing mechanical ventilation, is viewed as a necessary nursing practise to prevent tube obstruction.

The purpose of the study was to determine whether preoxygenation by hyperoxygenation prevents transient hypoxaemia, during endotracheal tube suctioning, of ventilated neonates with respiratory distress syndrome.

An extensive literature survey confirmed that endotracheal tube suctioning, results in transient hypoxaemia and demonstrates that this is reflected in the brain, by vasodilatation and deoxygenation :- predisposing to the genesis of intra-ventricular haemorrhages and neurodevelopmental problems. The survey further indicated that these effects are preventable by preoxygenation before endotracheal tube suctioning.

Twelve premature babies were then used in the study to determine whether preoxygenation by hyperoxygenation prevents transient hypoxaemia, during endotracheal tube suctioning.

The following conclusions during suctioning with preoxygenation were reached, by statistically processing the results:

* Bradycardia still occurred but at a significantly lesser degree.
* Significantly less desaturation.
* Smaller increase in mean blood pressure.
* Significantly faster recovery to baseline heart rate, transcutaneous oximeter saturation and respiration and a markedly faster recovery to baseline mean blood pressure.
* The mean arterial blood gas and partial pressure of oxygen showed a slight increase after suctioning.

Nursing guidelines were established for beneficial suctioning techniques for premature babies and recommendations were made for further research.
Suiging van endotracheale buise, by premature babas met respiratoriese nood sindroom, wat meganise ventilasie ondergaan, word beskou as 'n noodsaaklike verpleeg aksie om obstruksie van buise te voorkom.

Die doel van hierdie studie was om te bepaal of preoksigenasie deur middel van hiperoksigenasie tydelike hipoksemie sou voorkom, gedurende suiging van endotracheale buise, by geventileerde neonate met respiratoriese nood sindroom.

'n Uitgebreide literatuurstudie het getoon dat suiging van endotracheale buise, tydelike hipoksemie tot gevolg het en het ook gedemonstreer dat dit die brein beïnvloed, deur vasodilatasie en deoksigenering : - dit stel die babas bloot aan intra-ventrikulere bloeding en probleme tydens neurologiese ontwikkeling. Die literatuurstudie het ook uitgewys dat hierdie effekte voorkombaar is deur preoksigenasie voor die suiging van endotracheale buise.

Twaalf premature babas is in hierdie studie gebruik om te bepaal of preoksigenasie deur middel van hiperoksigenasie tydelike hipoksemie sou voorkom, gedurende suiging van endotracheale buise.

Daar is tot die volgende gevolgtrekkings gedurende suiging met preoksigenasie gekom, deur die resultate statisties te verwerk :

* Bradikardi het nog steeds plaasgevind maar tot 'n minder beduidende mate.
* Desaturasie het tot 'n minder beduidende mate gebeur.
* Daar was 'n kleiner toename in gemiddelde bloeddruk.
* Daar was 'n beduidende vinniger herstel van basislyn van die harttempo, transkutane oksiemeter saturasie en asemhalingstempo en 'n merkwaardige vinniger herstel van basislyn van gemiddelde bloeddruk.
* Die gemiddelde arteriele bloedgas en die parsiele suurstofdruk het 'n geringe verbetering getoon na suiging.

Riglyne is gestel vir verpleegkundiges rakende voordelige suigtegnieke by premature babas en aanbevelings vir verdere narvorsing is gemaak.
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CHAPTER 1

INTRODUCTION AND MOTIVATION FOR RESEARCH STUDY

1.1 INTRODUCTION

A basic tenet of neonatal intensive care is that handling, and disturbing a sick neonate in any way may cause his condition to deteriorate, usually by making him hypoxic. (Roberton, 1993: 47, 48; Avery, Fletcher and Mac Donald, 1994: 1121 - 1134; Kelner and Harvey, 1987: 113).

According to Roberton (1993: 47, 48), Avery, et al (1994: 1121 - 1134), and Kelner and Harvey (1987: 113) anything that makes an infant cry, by making his respiration irregular e.g. disconnecting the oxygen supply when suctioning the endotracheal tube, will compromise his ventilation (even on intermittent positive pressure ventilation) by increasing the pulmonary artery pressure, increasing the right to left shunt, and decreasing the Pa02 (partial pressure of oxygen) resulting in hypoxia.

Suctioning is not an innocuous procedure - it may cause bronchospasm, pneumothoraces, infection, airway damage, bradycardia due to vagal nerve stimulation, hypoxia and increases intracranial pressure, predisposing to intra-ventricular haemorrhage. Hypoxic - ischaemic cerebral injury, results from hypoxaemia, often associated with impaired cerebrovascular autoregulation. The extent of injury, is determined principally by maturity of brain at time of insult and severity and duration of insult. Because of impaired cerebrovascular autoregulation in sick premature infants, alteration in systematic blood pressure reflects directly in changes in cerebral blood flow. Care must be taken to prevent hypertension associated with rapid infusion of fluids, excessive handling and suctioning. (Avery, et al, 1994: 1121 - 1134; Wong, 1993: 244 / 696 - 697).

Shah, Kurth, Gwiazdowski, Chance, Delivoria - Papadopoulos (1992: 769 - 774) investigated, at the hospital of University of Pensylvania, the effect of suctioning the endotracheal tube, on cerebral circulation. This study confirmed that endotracheal tube suctioning results in transient hypoxaemia, reflected in the brain by vasodilatation and deoxygenation. These effects were prevented by preoxygenation, by increasing FiO2 (concentration of inspired oxygen) by 10% at the beginning of protocol to achieve a saturation of 100% before suctioning was performed.

that suction pressure should be set at 100 mmHg to prevent damage to brachial mucosa. They also suggest that nurses should not suck out the endotracheal tube too often, since secretions are rarely a problem in early respiratory distress syndrome. Suctioning 4 - 6 hourly should suffice. Suctioning should be swift and efficient, 0.5ml NaCl should be instilled into the endotracheal tube, prior to suction, to loosen secretions. If baby becomes cyanosed, saturation drops or heart rate falls below 90 beats per minute, stop suctioning and reconnect ventilator.

Many studies have explored ways to lessen hypoxia associated with endotracheal tube suctioning. Abels (1986 : 221 - 334), Hazinski (1984 : 263, 280), and Thelan, Davie and Urden (1990 : 455 - 467) believe that introduction of catheter decreases airflow. To minimise possible suction - induced hypoxaemia, presuction and postsuction oxygenation should be used.

The question arises whether preoxygenation should be provided to all ventilated patients. Thelan, et al (1990 : 455 - 467) describes how Poames and Kirchhoff concluded that the method of preoxygenation used, should be tailored to the patients needs, whether by :- hyperinflation, hyperventilation, hyperoxygenation, or insufflation.

Suctioning has been found to increase cerebral blood flow and intracranial pressure, predisposing to intra-ventricular haemorrhage. (Shah, et al, 1992 : 769 - 774). Perlman and Volpe (1983 : 329 - 334) postulated that the mechanism was related to increased arterial and central venous blood pressure and transient hypoxaemia, caused by endotracheal tube suctioning. However, Shah, et al (1992 : 769 - 774) and Levin and Morriss (1990 : 927) found that preoxygenation, prevents hypoxaemia, by maintaining SpO2 (arterial haemoglobin saturation), between 90 -99%, and prevents MAP ( mean arterial blood pressure ) from increasing significantly.

1.2 RESEARCH PROBLEM

From the above, it can be seen that most sources agree that suctioning does result in transient hypoxaemia i.e. deficient oxygenation of the blood. Whether preoxygenation should be used at all, as part of the nursing protocol, in suctioning techniques and if used - the method, time frame when it should occur and for how long delivered, during the suctioning technique, is uncertain.

The following question can then be asked :- Will nursing staff, utilising preoxygenation by means of hyperoxygenation, during suctioning of ventilated neonates with respiratory distress syndrome, prevent transient hypoxaemia?
1.3 RESEARCH PURPOSE

The aim of the study, is to determine whether utilising preoxygenation by means of hyperoxygenation, will prevent transient hypoxaemia, during endotracheal tube suctioning of ventilated neonates admitted to a neonatal unit with respiratory distress syndrome.

1.3.1 Specific objectives

* Determine, through literature survey, the most effective suctioning technique that will prevent transient hypoxaemia, during suctioning of ventilated neonates with respiratory distress syndrome.

* Evaluate the effect of using preoxygenation by means of hyperoxygenation, prior, during and after endotracheal tube suctioning in terms of preventing transient hypoxaemia, during suctioning of ventilated neonates with respiratory distress syndrome.

* Establish guidelines, of beneficial suctioning techniques for nursing staff.

1.4 HYPOTHESES

1.4.1 Null hypothesis

Utilising preoxygenation by means of hyperoxygenation, does prevent transient hypoxaemia during endotracheal tube suctioning of ventilated neonates with respiratory distress syndrome.

1.4.2 Alternative hypothesis

Utilising preoxygenation by means of hyperoxygenation, does not prevent transient hypoxaemia during endotracheal tube suctioning of ventilated neonates with respiratory distress syndrome.
1.5 ASSUMPTIONS OF THE STUDY

1.5.1 Paradigm

For the purpose of this study the Whole Person Theory from the Oral Roberts University, Anna Vaughn School of Nursing is used, in conjunction with the philosophy of the Rand Afrikaans University and guidelines of the South African Nursing Council. Based on Judeo -Christian Philosophy - it is utilised as a framework for nursing education, nursing practice and nursing research and focuses on the continuous quest for wholeness of the individual within the family and the community (RAU, 1992 : 2 , 3).

1.5.2 Meta - theoretical assumptions

Person
"A person is a spiritual being who functions in an intergrated biopsychosocial manner to achieve his quest for wholeness. A person interacts with his internal and external environment holistically " (RAU, 1992 : 5). In this study, the ventilated neonate with respiratory distress syndrome is considered as the person.

Health
"Health is a state of spiritual, mental and physical wholeness. The person's pattern of interaction with his internal and external environment determines his health status. Health can be qualitatively described on a continuum from maximum health to minimum health. Illness potential exists in those who are healthy "(RAU, 1992 : 5, 6). In this study illness refers to the condition of the ventilated neonate with respiratory distress syndrome. The treatment thereof, can however lead to a better pathway to health.

Illness
"Illness is a dynamic state that reflects the nature of the person's interactive patterns with stresses in his internal and external environment. Illness can be qualitatively described on a continuum from severe illness to minimum illness. Health potential exists in those who are ill " (RAU, 1992 : 6).
Nursing

Is a goal-directed service to assist the individual by promoting, maintaining and restoring health - by utilising the concept of Nursing for the Whole Person Theory. Promotion, maintenance and restoration of health are defined as:-

Promotion of health refers to nursing activities that contribute a greater degree of wholeness for the individual and family. This will be achieved by researching the best suctioning technique available and explaining the suction procedure and the condition of the child to the parents.

Maintenance of health refers to nursing activities directed to continuing and preserving the health status of the individual. In this study maintenance of health can be achieved by using a safe and therefore acceptable suction procedure that prevents transient hypoxaemia in the ventilated neonate with respiratory distress syndrome.

Restoration of health refers to nursing activities that facilitate the return to the previously experienced level of health of the individual. Restoration of health can be achieved by preoxygenation during endotracheal tube suctioning and preventing transient hypoxaemia and possible side-effects thereof.

1.5.3 Theoretical assumptions

This study is based on the Nursing for the Whole Person Theory. The following statement drawn from this theory is central to the theme of the study.
"The nurse, through the health delivery system, facilitates promotion, maintenance and restoration of the individual's health" (RAU, 1992: 8).

This theory relates to the study in the following way. The nurse promotes the health of the ventilated neonate with respiratory distress syndrome, by researching the best suctioning techniques available. The nurse then maintains the neonate's health by utilising this technique during her nursing care, thus preventing possible side-effects and thereby promotes the neonate to optimal health.

1.5.4 Methodological Assumptions

Nursing Science utilises the functional thought process, which implies that the main goal of the practise of science, is to understand, comprehend and explain nursing practice through reality testing (research), for better control and improvements of the practice. Thereby promoting theory development and testing and consequently validating the old body of knowledge and generating new knowledge. The criteria for validity and
accountability are fulfilled when the research findings are significant in improving nursing practice (Botes, 1993: 1-3).

One of the objectives of this study is to establish guidelines of beneficial suctioning techniques, for nursing practice, by researching to see whether preoxygenation will prevent transient hypoxaemia during suctioning and if so proven promote theory development and testing and therefore fulfil the criteria for validity and accountability.

1.6 DEFINITION OF TERMS

The following terms are used in the study, which were operationalized.

1.6.1 Respiratory distress syndrome (RDS)

A disorder primarily of prematurity, but also found in neonates of diabetic mothers, among neonates born by caesarean section and occasionally among neonates in whom no predisposing factor is apparent. The neonate is delivered prior to maturation of enzymatic pathways in the lung, that manufacture pulmonary surfactant. Manifested clinically by respiratory distress and pathologically by pulmonary hyaline membranes and diffuse alveolar actelectasis, oedema and cell injury.

1.6.2 Premature neonate

Any live-born infant whose birth occurs before the end of the last day of the 37th week gestational age and after 26 weeks gestational age for viability, following onset of last menstrual period. These infants weigh between 900 - 2600 grams. Appropriate for gestational age (AGA).

1.6.3 Endotracheal tube (ET Tube) suctioning

Suctioning method of cleaning secretions from the airways. This is performed through an artificial airway (endotracheal tube) either nasotracheally or orotracheally. It involves the use of a sterile technique. A suctioning catheter is placed into the endotracheal tube and inserted +/- 1/2 cm beyond the tip of the tube. The catheter is then, without suction, withdrawn slightly. Then using continuous suction and gentle twisting motion of the catheter, removed from the endotracheal tube.
1.6.4 Transient hypoxaemia

As a patient is suctioned, oxygen is removed along with the secretions, resulting in transient suction-induced hypoxaemia i.e. deficient oxygenation of the blood. The arterial partial pressure of oxygen (PaO2) is less than normal, due to the suctioning procedure and can be identified by a decrease in transcutaneous oximeter saturation (TO2) < 90% or a decrease in PaO2 < 6.6kPa / < 60mmHg. If hypoxia is prolonged it results in impaired cellular processes. Reoxygenation commences with the onset of reventilation.

1.6.5 Mechanical ventilation

This is the process of exchange of air between the lungs and the ambient air, accomplished by extrinsic means. It maintains positive pressure in the airways throughout the respiratory cycle, lessens the work of breathing and provides ventilation for patients who cannot, themselves, effectively ventilate. Achieved by using ventilators which intermittently, continuously assist or control pulmonary ventilation. For the purposes of this study, intermittent positive pressure ventilation is used.

1.6.6 Preoxygenation

This is presuction and postsuction oxygenation. There are four different methods that can be used, in conjunction or alone and tailored to the patients needs. Three methods are excluded from this study, namely:

- Insufflation: not commonly utilised locally.
- Hyperinflation: predisposes to pneumothoraces in neonates.
- Hyperventilation: of little use as high rates already in use.

For the purposes of this study hyperoxygenation is used.

1.6.6.1 Hyperoxygenation

Increasing the concentration of inspired oxygen (FiO2) before, during and after suctioning. Achieved by turning ventilator oxygen setting up or by using resuscitation
bag. Sufficient time is allowed to elapse, to achieve the increased oxygen concentration in the system.

During this study hyperoxygenation consists of:

Increasing the FiO2 by 10% at the beginning of the procedure i.e. 5 minutes immediately before suctioning and decreased again, slowly, by 2% at one minute intervals at the end of the procedure i.e. within 5 minutes of completing suctioning.

1.7 METHOD OF INVESTIGATION

This study is undertaken within the contest of a specific area - a private clinic with a neonatal unit.

1.7.1 Research Design

Pre - post-test control / experimental group design.

Pretest Control 1 ---+ Treatment ---+ Control 2
ET Tube suctioning

MANIPULATION OF INDEPENDENT VARIABLE: PREOXYGENATION

Post-test Experiment 1 ---+ Treatment ---+ Experiment 2
ET Tube suctioning

A quantitative, explanatory design with experimental method, to be undertaken within the context of a neonatal unit in a private clinic. The same infant is used in the pretest and then 6 hours later in the post-test. To be discussed fully in chapter 3 pg 37.

1.7.2 Variables

Independent variable
Preoxygenation, by means of hyperoxygenation, during suctioning procedure.
Dependant variable
Transcient hypoxaemia.

Other Variables
These variables will be predetermined and held constant.

i) Number of aspirations during suctioning procedure.
ii) Duration of each aspiration.
iii) Size of catheter used to suction with.
iv) Instillation of NaCl, into endotracheal tube, prior to suctioning.
v) Use of chest physiotherapy.
vi) Suction pressure
vii) Catheter length passed.

1.7.3 Sampling

Population
All ventilated neonates admitted to a private neonatal unit, in Johannesburg.

Sample plan
Systematic sampling of the first 10 - 20 ventilated neonates admitted into the unit.

The criteria will be covered in chapter 3 pg 42.

1.7.4 Measuring strategy

Direct physiologic dimensions are utilised during this study. The infants represented a random sample. There is an experimental and control phase, both carried out on the same infant. The treatment is under control of the researcher. The dependent variable is measured twice, before and after manipulation of the independent variable. The instrument includes measurements of arterial blood gases, heart rate, blood pressure, transcutaneous oximeter saturation and respiration, before and after manipulation of independent variable.

1.7.4.1 Reliability and validity of measuring instrument

Use of the Van Drimmelin arterial blood gas machine will be made, withdrawing arterial blood gas specimens 5 minutes before starting suctioning procedure and 30 minutes
after completing suctioning procedure during the control and experimental phases. (This section will be discussed in depth in Chapter 3 - the method of investigation undertaken).

1.8 SUMMARY

Suctioning of ventilated neonates with respiratory distress syndrome causes transient hypoxaemia. Suction techniques differ. To prevent hypoxaemia, which predisposes to intra-ventricular haemorrhages, preoxygenation can be utilised. This research study examines the manipulation of preoxygenation, during suctioning with the purpose of preventing hypoxaemia.

1.9 DIVISION OF CHAPTERS

Chapter 1
Consists of introduction, description of the research problem, purpose, hypotheses and definition of terms. The paradigm on which the study is based, as well as the associated assumptions are discussed.

Chapter 2
The literature survey discusses different suction techniques. The means of delivering preoxygenation during these suction techniques and the effect it has on transient hypoxaemia, is also included.

Chapter 3
The method of investigation which is undertaken, with referral to the sampling plan, measuring instruments and collection of data.

Chapter 4
Analysis of data collected.

Chapter 5
Findings of the research study are discussed and recommendations are made, for implementation of results, improvements in nursing practice and areas where further research in this field can be done.
2.1 INTRODUCTION

This chapter includes a literature survey conducted on suctioning techniques of premature infants with respiratory distress syndrome; transient hypoxaemia and sequelae thereof. The role of preoxygenation in preventing transient hypoxaemia and the different methods of preoxygenating the incubated premature infant with respiratory distress syndrome, is also discussed.

Premature births occur before the end of the 37th gestational week. The majority of babies who weigh less than 2600g and almost all infants below 1500g are born prematurely. Most of them are appropriately grown, some are small for gestational age (SGA). (Korones, 1986: 144; Klaus and Fanaroff, 1986: 69; Avery, et al, 1994: 70).

Respiratory distress syndrome is a acute disorder that is symptomatic at birth or soon thereafter. Primarily characterised by respiratory distress, it occurs almost exclusively in premature infants. In essence, respiratory distress syndrome is a partial persistence of the fetal cardiopulmonary state i.e. increased pulmonary vascular resistance and a deficiency in surfactant activity. At birth, and for a short time thereafter, some infants breathe with little or moderate difficulty. Soon respiration becomes more laboured as surfactant is dissipated. Consequently, normal functional residual capacity (FRC) is not possible. Alveoli that are inflated during inspiration are again collapsed at end expiration. Atelectasis becomes more widespread and vasospasm causes pulmonary hypoperfusion. This results in hypoxia and hypercapnia. The failure of lung expansion and persistence of high vascular resistance causes the fetal circulation to persist in each patient to a variable extent. Therefore lung compliance diminishes, requiring far more pressure than normal lungs for equal amounts of expansion. Treatment of respiratory distress syndrome may require therapy with positive end expiratory pressure (PEEP) and intermittent positive pressure ventilation (IPPV). (Avery, et al, 1994: 436 - 438; Korones, 1986: 221 - 224; Klaus and Fanaroff, 1986: 181 - 185).

Ventilation requires endotracheal tube insertion into the trachea, which interferes with the physiologic mechanisms for clearance of respiratory secretions and may itself stimulate secretions. Meticulous care is needed to prevent accumulation of secretions which can obstruct the tube. Secretions consist of sputum which is a complex fluid...

Suctioning of endotracheal tube, in premature infants with respiratory distress syndrome, who are undergoing mechanical ventilation, is viewed as a necessary nursing practice to prevent tube obstruction. Suctioning can be performed up to 8 times in 24 hours. The ideal suctioning technique is the one that removes the greatest amount of secretions with the least amount of risk to the patient. (Levin and Morriss, 1990 : 927 - 929).

2.2 SUCTION PROTOCOLS

Most suction protocols, used at different institutions, follow more or less the same basic guidelines, with occasional variations. i.e. use of NaCl (Normal Saline 0,9%); use of chest physiotherapy; variations in suction pressures; catheter size; length of catheter pass; duration of each aspiration; number of aspirations during entire suction procedure and whether to preoxygenate and method used.


2.2.1 Basic procedure

* Aseptic technique to be used.
* Resuscitation bag is connected to correct concentration of oxygen and potency checked, should the infant become apnoeic / brady during procedure.
* Observe vital signs - heart rate, respiration, colour, saturation, blood pressure, arterial blood gas.
* Auscultate chest. Physiotherapy with need - positioning, percuss and vibrate.
* Appropriate size catheter to be used in relation to size of endotracheal tube.
* Check suction pressure.
* NaCl to be used when necessary (0,25ml to 0,5ml).
* Preoxygenate infant if necessary.

*** Disconnect ventilator. Insert catheter +/- 1/2cm beyond the tip of the endotracheal tube tube, withdraw slightly before suctioning to prevent mucosal haemorrhage and erosion of tracheobronchial tree.
* Withdraw in one continuous movement. Suction for no longer than 20 seconds.
* Reventilate. Observe vital signs and re-establish transcutaneous oximeter
saturation > 90% ***

* Used warmed NaCl when necessary - disconnect ventilator and instill into endotracheal tube. Reconnect. Allow a few breaths. Disconnect and repeat from *** to ***. Repeat procedure x 3 maximum times.

* Suction nasopharynx and oropharynx thereafter.

* Observe patient's condition till stable. Auscultate chest to evaluate effectiveness of suctioning and ensure adequate bilateral ventilation. Note tolerance of procedure.

* Note amount and type of secretions obtained.

2.2.2 Variations in suction protocol

2.2.2.1 The use Of NaCl

Hodge (1991:12), and Hazinski (1984:280) feel that instillation of NaCl before suctioning is controversial and has not been thoroughly investigated. The rationale of NaCl use, +/- 0,5ml into endotracheal tube, prior to suctioning is that it facilitates the removal of fixed secretions and prevents formation of mucous crust within the artificial and proximal airway. However, NaCl droplets are large in diameter and seldom move beyond the area of the carina. There is also the potential risk of contamination of contents during opening of NaCl vial. Most other sources, Avery, et al (1994:61,64), Merenstein (1985:312), and Levin and Morriss (1990:927-929) among others feel that NaCl instillation should be used only when necessary.

2.2.2.2 Chest physiotherapy

Chest percussion causes hypoxia and contravenes minimal handling. Fox, et al (1978:977-981) concluded in their study, that chest physiotherapy may cause hypoxia and was not warranted as a routine. Merenstein (1985:312), and Levin and Morriss (1990:927-929) feel that physiotherapy - including postural drainage, percussion and vibration should be done on patient's needs only. Roberton (1986:303) and Hazinski (1984:263) add that there is a place for selective chest physiotherapy in babies with broncho pulmonary dysplasia (BPD), pneumonia, old respiratory distress syndrome > 4 / 5 days with many secretions and atelectasis post extubation. Hodge (1991:12) agrees that infants in acute phase of respiratory distress syndrome produce little or no mucous unless an infection is present.
2.2.2.3 Suction pressure

The negative pressure of suctioning can contribute to atelectasis, decrease lung compliance cause mucosal damage and also worsen hypoxia. Therefore, suction pressure should not exceed 80 - 120mmHg / < 20 kPa, depending on the weight of the infant. (Wong, 1994: 696 - 697; Kelner and Harvey, 1987: 118 - 119; Hodge, 1991 : 9; Perlman and Volpe, 1983 : 330).

2.2.2.4 Catheter size

Catheter size can contribute to suction - induced atelectasis, hypoxia, decreased lung compliance and intrapulmonary shunting, because negative pressure created during suctioning pulls air from distal airways. A appropriate sized catheter, which does not totally occlude the endotracheal tube and allows atmospheric air simultaneously within the tube, is necessary to decrease amount of negative pressure generated in the airways. The appropriate catheter size interval - to - external diameter ratio is 0,50 - 0,66.

Example : use size 5 French catheter - 2,5mm endotracheal tube
use size 6 French catheter - 3mm endotracheal tube

Levin and Morriss (1990 : 927 - 929) suggest the following:-

<table>
<thead>
<tr>
<th>Catheter Size</th>
<th>Endotracheal tube</th>
<th>Max Neg Pressure (cmHzO)</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>2,0</td>
<td>60 - 90</td>
<td>Infant</td>
</tr>
<tr>
<td>6</td>
<td>2,5</td>
<td>90 - 110</td>
<td>Child</td>
</tr>
<tr>
<td>6 1/2</td>
<td>3,0</td>
<td>110 - 150</td>
<td>Older child</td>
</tr>
<tr>
<td>6 1/2 - 8</td>
<td>3,5</td>
<td>Negative pressure that occurs during suctioning of the endotracheal tube, can be controlled, by setting prior to the suction procedure.</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>4,0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Catheter sizes related to endotracheal tube size.

2.2.2.5 Catheter length passed

Catheter length passed is also important, not to advance beyond length of endotracheal tube, thereby decreasing risk of pneumothoraxes and narcotising tracheobronchitis. Centimetre markings on endotracheal tube are used to determine the appropriate length.

Avery, et al (1994: 64) describe the following safe length of catheter advancement.

<table>
<thead>
<tr>
<th>WEIGHT:</th>
<th>CM:</th>
</tr>
</thead>
<tbody>
<tr>
<td>500g</td>
<td>7 cm</td>
</tr>
<tr>
<td>600g - 1000g</td>
<td>8 cm</td>
</tr>
<tr>
<td>1100g - 1500g</td>
<td>9 cm</td>
</tr>
<tr>
<td>1600g - 2000g</td>
<td>10 cm</td>
</tr>
<tr>
<td>2100g - 2500g</td>
<td>11 cm</td>
</tr>
<tr>
<td>2600g - 3000g</td>
<td>12 cm</td>
</tr>
<tr>
<td>3100g - 3500g</td>
<td>13 cm</td>
</tr>
<tr>
<td>3600g - 4000g</td>
<td>14 cm</td>
</tr>
</tbody>
</table>

Avery, et al 1994: 64

### 2.2.2.6 Duration of each aspiration

Duration of each aspiration during suctioning is also important. Longer durations of each aspiration increases the risk of complications and even 20 second suction durations can cause hypoxaemia and elevate cerebral blood flow velocity, increase systemic blood pressure, increase intracranial pressure and reduce oxygenation. Reducing duration of suctioning will reduce risk of trauma, pulmonary haemorrhage and prolonged hypoxia. Sources agree that a maximum of 20 second duration should be used, preferably +/- 10 seconds long. (Dietch, 1993: 13; Perlman and Volpe, 1983: 330; Shah, et al, 1992: 770; Levin and Morriess, 1990: 927 - 929).

### 2.2.2.7 Number of aspirations during the suction procedure

The greater the number of aspirations during the entire suction procedure can also increase risk of hypoxaemia. Wong (1994: 696 - 697), suggests limiting to +/- 3 aspirations in one period and to rest the infant 30 to 60 seconds after each aspiration, to allow oxygen tension to return to normal. Hodge (1991: 12) feels that repetitive catheter passers are not to be used unless amount of secretions warrants it. Ventilated infants with respiratory distress syndrome, in the first few days, have few secretions and rarely warrant repeated passes, unless they become a problem i.e. pulmonary haemorrhage or pneumonia. (Hazinski, 1984: 280; Wong, 1994: 696 - 697).
2.3 EFFECTS OF SUCTIONING: TRANSCIENT HYPOXAEAMIA

Infants with respiratory distress syndrome have problems with oxygenation. Anything which increases their oxygen consumption without improving arterial oxygenation, leads to hypoxia and potential deterioration in their condition. Babies with respiratory distress syndrome deteriorate with handling because this leads to excess movement, crying and agitation, all which increases oxygen consumption and right to left shunting. Almost all nursing and medical procedures lead to hypoxic episodes, including suctioning (Roberton, 1986: 304; Merenstein, 1985: 312; Korones, 1986: 236 - 238; Klaus and Fanaroff, 1986: 107, 184; Levin and Morriss, 1990: 909; Johansen, Dungca, Hoffmeister and Wells, 1985: 594 - 595).

As early as 1978 Fox, Schwartz and Shaffer (1978: 977 - 978) reported that bradycardia following suctioning had been observed in neonates. By 1983 Perlman and Volpe (1983: 329) noted that suctioning had been associated with hypoxaemia, bradycardia and hypertension in infants.

In the 1990's Levin and Morriss (1990: 927 - 929), and Hodge (1991: 7 - 8) feel that suctioning is complicated by morbidity, from short-term hypoxia (transcient), to dysrrhythmias, pulmonary artery hypertension, atelectasis, structural mucosal damage, bacterial growth and death. Suction induced atelectasis occurs when alveolar lung volume diminishes. Suctioning creates negative airway pressure, causing collapse of tiny air sacs, resulting in arterial oxygen desaturation and transient hypoxaemia - lasting until air sacs are re-expanded. Transient hypoxaemia results in vasoconstriction of renal, splanchnic and pulmonary vessels, which could cause PDA (Patent ductus arteriosus), NEC (Necrotising entero colitis) and intrapulmonary shunting. Dysrhythmias occurs as a result of transient hypoxaemia or vagal stimulation from suction catheter.

2.4 SEQUELAE OF TRANSCIENT HYPOXAEAMIA

2.4.1 Intra-ventricular haemorrhage and neurodevelopment problems

Hodge (1991: 8) further describes how transient hypoxaemia and bradycardia with increased blood pressure contribute to pathogenesis of intra-ventricular haemorrhage and hypoxic brain injury. The risk of intra-ventricular haemorrhage (IVH) maybe as high
as 61 - 70%, in the VLBW (very low birth weight) infant, < 1000gms (Dietch, 1993 : 7), and as high as 40 - 60% in prems < 34 weeks or < 1500gms. (Merenstein, 1985 : 412; Korones, 1986 : 398; Roberton, 1986 : 543; Van De Bor, Van Bel, Lineman and Ruys, 1986 : 1125). Hypoxic - ischaemic brain injury is the major cause of neurologic impairment and mortality in premature infants. Incidence of 5 - 15% of premature infants < 32 weeks or < 1500gms which survive, exhibit cerebral palsy, and 20 - 25% exhibit less prominent disabilities.

Peri-ventricular leukomalacia (PVL) or hypoxic - ischaemic brain injury is an ischaemic lesion of the white matter adjacent to external angles of lateral ventricles of the brain. (Roberton, 1986 : 543 - 545; Dietch, 1993 : 8 - 9; Doran, 1992 : 7 - 8). Peri-ventricular leukomalacia at autopsy correlates with spastic diplegia in premature infants who live beyond the 1st month of life. Spastic diplegia - is a type of spastic quadriplegia / cerebral palsy where lower extremities are affected more than upper extremities (Doran, 1992 : 7 - 8).

With intra-ventricular haemorrhage, mortality rates and incidence of progressive post-haemorrhage hydrocephalus will vary with severity of haemorrhage. In recent figures by Dietch (1993 : 13 - 14), grade IV intra-ventricular haemorrhage, when intracerebral involvement occurs with severe haemorrhage, mortality rate approaches 60% and progressive ventricular dilation will occur in nearly 80% of these survivors. Dietch also describes long-term prognosis, as follows:-

Grade I IVH - neurological sequelae +/- 15% for survivors e.g. spastic diplegia, spastic quadriplegia often with intellectual retardation.

Grade II - III IVH - neurological deficit increases by 30 - 40%.

Grade IV IVH - neurological deficit increases by 90% (Dietch, 1993 : 13 - 14).

Nursing plays an important role in reducing risk factors contributing to intra-ventricular haemorrhage, particularly during the first four days of life, when a germinal matrix haemorrhage and its extension is most likely to occur. (Dietch, 1993 : 8; Roberton, 1986 : 543 - 545; Lapido, 1989 : 9; Merenstein, 1985 : 412 - 414). The germinal matrix does not dissipate till term, prominent between 24 to 32 weeks gestational age.

2.4.1.1 Structure of the germinal matrix

It is a gelatinous region composed of proliferating cells that are precursors of neuronal and glial cells. It provides weak support for an immature microvascular network that will
remodel into a mature capillary bed, when the matrix disappears. Vessel walls are thin and fragile endothelium, without smooth muscle, collagen or elastin. These endothelial cells are highly dependent on oxidative metabolism and maybe easily injured by hypoxic insult. (Korones, 1986: 398 - 400; Klaus and Fanaroff, 1986: 365; Dietch, 1993: 8 - 9; Van De Bor, et al, 1986: 1125 - 1126; Lapido 1989: 9 - 10).

2.4.1.2 Pathogenesis of intra-ventricular haemorrhage and peri-ventricular leukomalacia

Pathogenesis is multifactorial. Mechanism underlying association of intra-ventricular haemorrhage with peri-ventricular leukomalacia is unknown. Intra-ventricular haemorrhage is thought to be caused by increased cerebral perfusion pressure. Peri-ventricular leukomalacia may result from altered circulation pattern occurring with intra-ventricular haemorrhage. (Doran, 1992: 8; Dietch, 1993: 9 - 11). Arterial concentration of PaCO2 (partial pressure of carbon dioxide), and PaO2 (partial pressure of oxygen) influence cerebral blood flow. Extreme alterations in arterial PaCO2, PaO2 and systematic blood pressure overrides autoregulation of cerebral blood flow. Cerebral autoregulation - is a homeostatic mechanism that maintains relatively constant cerebral perfusion over a wide range of systematic blood pressure by means of cerebral arterial constriction or dilation. (Wong, 1993: 696 - 697; Korones, 1986: 394 - 395; Dietch, 1993: 9 - 11).

Hypercarbia and hypoxia, alone or in combination causes cerebral vasodilatation and increased cerebral blood flow. This mechanism is complicated by effects of abrupt increases in systematic blood pressure, which may directly increase cerebral blood flow and cerebral blood flow velocity, causing rupture of delicate germinal matrix vasculature. On the other hand, hypocarbia and hyperoxaemia cause cerebral vasoconstriction and decreased cerebral blood flow. Prolonged insult, as in asphyxia at birth, cause initial decrease in cerebral blood flow accompanied by decreased systematic blood pressure. Decreased perfusion results in injury to matrix vessels. (Dietch, 1993: 9 - 11; Doran, 1992: 7 - 8; Wong, 1993: 696 - 697; Roberton, 1986: 543 - 545).

Brain glucose utilisation increases during hypoxic - ischaemic episodes. During hypoxia, neither glucose or ketones can be oxidised completely. Energy supplied by anaerobic glycolysis results in build-up of lactic acid, which produces severe extracellular oedema, through cell damage, causing endothelial leak and breakdown of blood - brain barrier. Cerebral oedema results in regional necrosis. When ischaemia is added to hypoxaemia, brain acidosis increases because tissue lactate cannot be removed and tissue carbon dioxide is not adequately buffered by bicarbonate. (Doran, 1992: 8 - 9; Wong, 1993: 696 - 697; Korones, 1986: 394 - 395; Klaus and Fanaroff, 1986: 365 - 366). This process eventually results in the poor long-term prognosis of intra-ventricular haemorrhage and peri-ventricular leukomalacia.
LBW (low birth weight) infants, ventilated with respiratory distress syndrome, are at greater risk for initial germinal matrix haemorrhage during the first 72 hrs of life and for extension of initial haemorrhage during the first week of life. Nursing care may well influence onset or extension of first lesion. Fluctuations in systematic blood pressure and cerebral arterial / venous pressure may be caused by hypoxia and hypocarbia, resulting from nursing procedures such as, handling and suctioning of endotracheal tube. (Dietch, 1993 : 9 - 11; Van De Bor, et al, 1986 : 1125; Doran, 1992 : 12; Perlman and Volpe, 1983 : 329).

2.5 STUDIES SHOWING SEQUELAE OF TRANSIENT HYPOXAEemia

Several studies show that suctioning is associated with hypoxaemia, bradycardia and increased systematic blood pressure, in infants with respiratory distress syndrome. Korones (1986 : 12 - 13) feels that bradycardia, is the first response to abrupt onset of hypoxaemia. The rationale that being reflex bradycardia is caused by increased vagal activity resulting from hypoxaemic stimulation, of chemoreceptors in the wall of aortic arch. Dietch (1993 : 13) feels that even 20 second suction episodes can elevate cerebral blood flow, increase systematic blood pressure, increase intracranial pressure and reduce oxygenation.

Van De Bor, et al (1986 : 1125 - 1130) studied 49 consecutively admitted infants, < 34 weeks gestation and analysed the neonatal factors, among others, associated with occurrence of intra-ventricular haemorrhage. The results showed that oral and endotracheal tube suctioning increased cerebral blood flow and might cause or extend intra-ventricular haemorrhages.

Perlman and Volpe (1983 : 329 - 334) studied the relationship of suctioning to changes in cerebral circulation in 35 premature newborn infants, admitted to the neonatal intensive care, of St. Louis Children's Hospital. All infants were ventilated, with respiratory distress syndrome, on the 1st postnatal day. The birth weight ranged from 720g to 2000g. Gestational age was 26 to 35 weeks. Prominent increase in cerebral blood flow velocity was found in nearly all patients and also marked increase in systematic blood pressure and intracranial pressure. No significant changes were found in mean heart rate with suctioning. Marked decrease in transcutaneous partial oxygen saturation accompanied suctioning, which gradually returned to baseline values a over 2 minutes period afterwards. Perlman and Volpe postulated that whatever the mechanism of systemic pressure response, in the presence of dysfunctional cerebral autoregulation, increased systematic blood pressure would be expected to cause an increased cerebral blood flow, resulting in increased intracranial pressure, which predisposes to pathogenesis of intra-ventricular haemorrhage. This is substantiated by Hodge (1991 : 8).
Brazy (1988 : 457 - 461) investigated the effects of crying on cerebral blood volume. 36 episodes of spontaneous crying were observed in 11 infants, whose gestational age was 26 to 40 weeks, aged 17 hours to 24 postnatal days. The infants were receiving headbox oxygen, CPAP (continuous positive airway pressure) or intermittent ventilation at a rate of 4 to 22 breathes per minute. 6 infants were receiving no respiratory support, while 5 premature infants had respiratory disease i.e. respiratory distress syndrome or broncho pulmonary dysplasia. In 86% of episodes, baseline cerebral blood volume rose and remained elevated during the cry. This study also noted a decrease in PaO2 (partial pressure of oxygen) during crying, in infants recovering from respiratory distress syndrome. Crying occurs with suctioning, causing similar surges of cerebral blood volume, with increased risk of intra-ventricular haemorrhage.

Shah, Kurth, Gwiazclowski, Chance and Delivoria - Papadopolous (1992 : 769 - 774) investigated fluctuations in cerebral oxygenation and blood volume during endotracheal tube suctioning in premature infants. Infants admitted to the Hospital of University of Pennsylvania were all ventilated for respiratory distress syndrome, but were clinically stable. Infants excluded - were those on muscle relaxants, narcotics, barbiturates, sedative - hypnotic drugs, seizure disorders, congenital heart diseases, and any haemodynamically unstable infants.

The study included 12 premature infants, birth weight ranged from 850 to 2160g, gestational age 24 to 33 weeks and postnatal ages 1 to 14 days. 7 infants had apgars > 7 at 1 and 5 minutes and the rest were < 6 at 1 and 5 minutes and 1 infant had a grade IV intra-ventricular haemorrhage.

On suctioning there was an increase in cerebral blood volume, a decrease in arterial haemoglobin saturation, an increase in mean arterial blood pressure and a decrease in heart rate. By 1 minute reventilation brain intravascular haemoglobin saturation, cerebral blood volume, arterial haemoglobin saturation, mean arterial blood pressure and heart rate returned to presuction levels. Similar responses were noted in infants with Apgars > 7 or < 7, the infant with the intra-ventricular haemorrhage and the infants aged < 3 or >3 postnatal days.

Therefore, as gathered from the literature survey, suctioning results in transcient hypoxaemia - arterial haemoglobin desaturation and decreased partial pressure of oxygen (PaO2), causing cerebral deoxygenation and increased systematic blood pressure and increased cerebral blood volume which predisposes to pathogenesis of intra-ventricular haemorrhage and neurodevelopmental sequelae. In this research study the research hypothesis postulates, that utilising preoxygenation during suctioning techniques of ventilated neonates with respiratory distress syndrome, does prevent transcient hypoxaemia and therefore sequelae of transcient hypoxaemia.
2.6 PREVENTION OF TRANSCIENT HYPOXAEMLA - BY PEOXYGENATION

2.6.1 Preoxygenation

Preoxygenation as described in Chapter 1 - is the provision of presuction and postsuction oxygenation. There are 4 methods that can be used in conjunction or alone and tailored to the patient's needs: - insufflation, hyperoxygenation, hyperinflation, hyperventilation. (see chapter 1 : PG 16 - 17).

2.6.1.1 Insufflation


2.6.1.2 Hyperoxygenation

Increasing the concentration of inspired oxygen (FiO2) before, during and after suctioning. This is achieved by turning ventilator oxygen setting up or by using a resuscitation bag. Allow sufficient time to elapse to achieve that oxygen concentration in the system. (Thelan, 1990 : 457 - 459).

2.6.1.3 Hyperinflation

Increasing the patients ideal volume, usually by 150%. Achieved by using the sigh component of the ventilator or by using some resuscitation bags. Does not refer to changes in FiO2, although increasing the FiO2 frequently accompanies hyperinflation. (Thelan, 1990 : 457 - 459).

2.6.1.4 Hyperventilation

Increase in ventilatory rate without necessarily changing the tidal volume or oxygen level delivered. (Thelan, 1990 : 457 - 459).
2.6.2 Studies indicating preoxygenation prevents transient hypoxaemia

The use of preoxygenation is supported by several sources. Merenstein (1985: 312-318), suggests hyperinflating with 6 to 8 breaths at the same oxygen concentration, matching pressure to ventilator settings, and increasing FiO2 (fraction of inspired oxygen) if clinically indicated. Merenstein feels that hyperinflating the lungs minimises hypoxia without danger of exposure to dangerously elevated partial pressure of oxygen (PaO2), and restores functional residual capacity and prevents atelectasis. Merenstein feels that hyperoxygenation, increasing FiO2 to 100%, raises PaO2, associated with hyperoxia and side-effects of retinopathy. (Avery, Fletcher and MacDonald, 1994: 61, 64).

Merenstein (1985: 309) adds that stable concentration of oxygen is necessary to maintain PaO2 within normal limits. A sudden increase or decrease in oxygen concentration may result in disproportionate increase or decrease in PaO2 caused by vasodilatation / constriction in response to oxygen. Lowering the concentration of inspired oxygen (FiO2) must be done slowly to avoid the "flip-flop" phenomenon. Korones (1986: 238-239) also feels that, except for circumstances in which abruptly increased oxygen concentration are maintained for less than 10 minutes, an abrupt change to decrease oxygen concentration is contraindicated. (Abels, 1986: 226-334).

Since introduction of catheter decreases airflow, Levin and Morriss (1990: 927-929), Hazinski (1984: 263 / 280), Abels (1986: 226-334), and Wong (1994: 696-697) among others, suggest providing supplemental oxygen to maintain arterial haemoglobin saturation between 90-94%. Either by, hyperoxygenation increasing FiO2 by 10%, higher than patient's maintenance level and / or hyperventilating with 8 to 10 ventilated / manual breaths. Hyperinflate using a bag. Peak inspiratory pressure during manual breaths should be approximately equal to pressure provided by ventilator, as excessive pressure may cause a pneumothorax.

Levin and Morriss (1990: 927-929), Hazinski (1984: 263 / 280), and Wong (1994: 696-697) also suggest that hyperoxygenation and / or hyperventilation, should be performed immediately before introduction of suction catheter into endotracheal tube and continue after completing suctioning, to allow oxygen tension to return to normal. Monitor arterial haemoglobin saturation using a pulse oximeter while suctioning. (Avery, Fletcher and MacDonald, 1994: 61, 64).

Research studies support the use of preoxygenation, in one and / or combination method. Hodge (1991: 10-12) refers to the study by Barnes, Asonye, Vidyasagar (1981). Their study found that children < 3 years with long - term respiratory problems, treated with hyperinflation alone or in combination with preoxygenation and / or hyperventilation prevented oxygen desaturation after tracheal suctioning. Hodge (1991: 10-12) however, also identified risks of certain methods of preoxygenation:
Hyperinflation - has the risk of causing pneumothoraces. Changes in heart rate and blood pressure may occur.

Insufflation - can increase dead space and increase risk of contamination of suction catheter. It lengthens suction time due to difficulty in advancing catheter through adapter and creates site for trapping secretions.

Hyperventilation - new-borns are on high rates and hyperventilation beyond bagging capabilities could cause desaturation and decrease heart rate +/- 10% below baseline.

Hodge feels that use of hyperoxygenation / hyperinflation warrants further investigation to demonstrate its effectiveness in the new-born population, who are at risk of retinopathy of prematurity, intra-ventricular haemorrhage and further lung injury.

As early as 1978 Fox, et al (1978 : 977 - 981) looked at pulmonary physiotherapy in neonates and hyperventilation after suctioning. The study included 13 randomly selected, new-born infants, admitted to a intensive care unit of the Children's Hospital of Philadelphia. Neonates were intubated and breathing spontaneously and recovering from respiratory disease. Birth weight ranged from 1,250 to 3,200 kg and gestational age from 28 to 40 weeks. Postnatal age was 2 to 17 days. All were stable, on PEEP (positive end expiratory pressure) and breathing spontaneously on CPAP (continuous positive airway pressure).

Fox, et al, concluded that suctioning accompanied by physiotherapy caused transient hypoxaemia. Even though reversed by hyperventilation, this in itself caused agitation, crying, increased blood pressure with further risk of pneumothorax and intra-ventricular haemorrhage. They suggested that hyperventilation was useful in weak / paralysed patient's and suggested further studies of hyperoxygenation before suctioning or use of double - lumen catheter might prevent hypoxaemia.

In the study, mentioned earlier, by Shah, et al (1992 : 769 - 774) suctioning caused increased cerebral blood volume, decreased arterial haemoglobin saturation, increased mean arterial blood pressure and decreased heart rate. They repeated the study in 8 of the 12 original infants x3 times, and a similar response was noted. They then chose 6 of the 12 infants and preoxygenated them - increasing FiO2 (fraction of inspired oxygen) by 10% at beginning of protocol i.e. 5 minutes prior to suctioning, to achieve arterial haemoglobin saturation of 100% before suctioning. Brain intravascular haemoglobin saturation and arterial haemoglobin saturation did not change significantly during suctioning, after preoxygenation. Arterial haemoglobin saturation remained > 90% during suctioning in all preoxygenation studies. There was only a slight increase in mean arterial blood pressure, compared to control group, and heart rate did not drop significantly.

Shah, et al, concluded that suctioning, resulted in arterial desaturation, cerebral deoxygenation, increased cerebral blood volume which could result in pathogenesis of
intra-ventricular haemorrhage and neurodevelopmental problems. These effects were prevented by increasing FiO2 before suctioning.

Shah, et al, also postulated that cerebral deoxygenation during suctioning, could be related to physiologic response - time, of cerebral circulation to transient hypoxaemia. 30 - 60 seconds is required for the brain to recognise changes in oxygen supply for cerebrovascular autoregulation to respond, during which time cerebral oxygenation decreased. They further suggested re-evaluation of suctioning techniques.

2.7 POSSIBLE SIDE-EFFECTS OF PREOXYGENATION

Even though hypoxaemia, caused during suctioning, has been shown to be reversed by using preoxygenation in previous studies, concerns about the potential deleterious effects of hyperoxia on the immature lung and retina has led to the practice of not ventilating with an increased concentration of inspired oxygen (FiO2) before suctioning.

2.7.1 Broncho pulmonary dysplasia

Broncho pulmonary dysplasia is a complex symptom process, resulting in constant and recurring lung injury with ongoing repair and healing of injury. As healing occurs, increased inspired oxygen tensions, barotrauma and infection continue to injure cells that are taking part in the healing process. Thickening and eventual necrosis of alveolar walls, basement membranes and bronchiolar epithelial lining layers occurs. Atelectasis and fibrosis is also present and impairs diffusion of oxygen from alveolar lumens to capillaries. It is a progressive chronic lung disease that follows protracted periods of mechanical ventilation, with high concentrations of oxygen via the endotracheal tube i.e. oxygen concentration of 80% or greater for at least +/- 6 days. (Merenstein, 1985 : 327; Korones, 1986 : 262 - 265).

Klaus and Fanaroff (1986 : 174 - 175) feels that the precise concentration of oxygen toxic to the lung probably depends on many variables i.e. maturation, nutritional and endocrine status, duration of exposure to oxygen and other oxidants, such as concentration of oxygen > 60% over a period of many days.

Immaturity of lung and severity of respiratory distress syndrome set the stage for injuries from barotrauma (ventilator pressures) and oxygen, and these injuries are inherent to mechanical ventilation. Barotrauma is now thought to be a more important cause than oxygen toxicity. Rarely, if ever, does broncho pulmonary dysplasia develop unless mechanical ventilatory support has been used. Infants in only headbox oxygen do not develop broncho pulmonary dysplasia. A number of other factors have been cited as

2.7.2 Retinopathy of prematurity

Association between oxygen administration, prematurity and retinal changes lead to blindness. Degree of retinal vascularization at birth determines the susceptibility to the insult of hyperoxia. The majority of retinal vascularization is complete by 32 weeks. Highest incidence of disease and blindness occurs at < 28 weeks and < 1500g. When hyperoxia is involved it is the partial pressure of oxygen in arterial blood (PaO2) rather than concentration administered (FiO2) that is important i.e. PaO2 > 100% torr. (Merenstein, 1985: 327; Korones, 1986: 265 - 268; Klaus and Fanaroff, 1986: 174 - 175).

Abnormal events occur in the retina during which a number of stages unfold. These events begin with vascular changes, progressing to retinal oedema and detachment, culminating in fibrosis. Progression through all these stages to scar formation occurs in few infants. In most, the process arrests in the early stages of vascular change. Retinopathy begins with arterial constriction, which may or may not be associated with oxygen administration or with a number of other factors. (Korones, 1986: 265 - 268; Klaus and Fanaroff, 1986: 174 - 175; Johnson, et al, 1985: 646).

Current resurgence in incidence of retinopathy is attributable to increased survival rate among infants < 28 weeks and birth weights < 1000grams. Investigators believe that other factors cannot be excluded e.g. factors inherent in disorders and therapy for survival of these smallest infants. (Korones, 1986: 265 - 268; Klaus and Fanaroff, 1986: 174 - 175; Roberton, 1986: 722 - 724).

While oxygen may play a role, studies have shown:
* Many infants who developed retinopathy despite low oxygen concentrations.
* A few infants who did not develop retinopathy when exposed to high oxygen concentrations.
* That full-term infants are known to have developed retinopathy, even despite having not received oxygen.
* Also many low birth weight infants developed retinopathy yet never had received oxygen.

Substantial data suggests retinal hypoxia is a more likely cause of neovascularization that characterises the disease. The disease has been reported in cyanotic congenital
heart disease, anencephalic infants, who were stillborn or did not survive more than a few days, and also in stillborn infants. It has been suggested that retinopathy is associated with complications of pregnancy, that cause retinal hypoxia, where retinal changes have been found to be over twice as frequent as among control infants. Extreme prematurity < 1000g, is susceptible to other factors i.e. intra-ventricular haemorrhage, recurrent apnoea, septicaemia, hypoxia and hypercarbia. Treatment with prostaglandin synthetase inhibitors eg Indomethacin for patent ductus arteriosus and vitamin E deficiency are associated with increased incidence of retinopathy. (Korones, 1986 : 265 - 268; Roberton, 1986 : 722 - 724).

Consensus exists that the greater hazard is if PaO2 > 100% torr. (Korones, 1986 : 265 - 268; Merenstein,1985 : 327; Fanaroff, 1986 : 174 - 175). Gunderson and Cusson (1993 : 71 - 72) describe how the study done by Edmond and colleagues, reported that only PaO2 at 41mmHg was required to achieve 90% saturation in LBW(low birth weight) infants i.e. low PaO2 will result in high oxygen saturation. They also noted that small changes in oxygen saturation (1 - 2%) result in large increases in PaO2. In order to prevent hyperoxia ( PaO2 > 100mmHg ) and negative sequelae i.e. oxygen toxicity, broncho pulmonary dysplasia and retinopathy; and hypoxia (PaO2 < 50mmHg), they suggest keeping pulse oximetry values for oxygen saturation between 90 - 95%.

Gunderson and Cusson (1993 : 71 - 72) also note that the American Academy of Paediatrics and American college of obstetricians and gynaecologists suggest monitoring PaO2 between 50 - 80 mmHg. This is supported by Korones (1986 : 265 - 268) and Klaus and Fanaroff (1986 : 174 - 175).

Klaus and Fanaroff (1986 : 174 - 175) add that PaO2 for premature infants be kept at 40 - 60 mmHg. This is because the structure of haemoglobin is altered in the fetus to allow increased oxygen haemoglobin affinity - resulting in higher oxygen saturation at relatively low PaO2 +/- 35 - 40 mmHg. (Gunderson and Cusson, 1993 : 71 - 72; Klaus and Fanaroff, 1986 : 174 - 175, Roberton, 1986 : 304 - 306).

2.8 SUMMARY

Suctioning of endotracheal tubes in premature infants with respiratory distress syndrome, who are undergoing mechanical ventilation is viewed as a necessary clinical practice to prevent tube obstruction. The above literature confirms that endotracheal tubes suctioning results in transient hypoxaemia and demonstrates that this is reflected in the brain by vasodilatation and deoxygenation - predisposing to the genesis of intra-ventricular haemorrhage and neurodevelopmental problems.

These effects are preventable by preoxygenation before suctioning. There however, exists the concern about the possible effects of hyperoxia causing broncho pulmonary...
dysplasia and retinopathy of prematurity. Literature, however suggests that broncho pulmonary dysplasia can be regarded as a iatrogenic symptom complex associated with prolonged oxygen and respiratory therapy initiated for respiratory distress syndrome i.e. barotrauma from ventilator pressures, endotracheal tubes injury, infections and immaturity causing the overall pathway of broncho pulmonary dysplasia.

Literature also suggests that the effect of oxygen on the retina is dependent on, the stage of development of the retinal vessels; length of exposure to oxygen and high PaO2 (partial pressure of oxygen) levels in arterial blood. Literature also emphasises that retinopathy is not a preventable disease in LBW (low birth weight) infants because the causes in these infants are still unknown.

Therefore, the need of re-evaluating our method of suctioning, to prevent transient hypoxaemia by utilising preoxygenation by hyperoxgenation, is of the greatest significance to the future nursing care of, ventilated premature infants with respiratory distress syndrome.

In chapter 3 the methodology of the study will be discussed with pertinence to suctioning technique and utilisation of preoxygenation by hyperoxgenation.
 CHAPTER 3

METHOD OF RESEARCH

3.1 INTRODUCTION

Suctioning of endotracheal tubes, in premature infants with respiratory distress syndrome, who are undergoing mechanical ventilation, is viewed as a necessary nursing practice to prevent tube obstruction. Suctioning can be performed up to 8 times in 24hrs. During this study, the concentration of inspired oxygen is increased by 10% for 5 minutes and maintained throughout the suction procedure. This is performed only once on each infant and then decreased again after completion of suctioning.

This chapter gives an overview of how the research is to be conducted and what method will be utilised.

3.1.1 Specific objective

Evaluate the effect of using preoxygenation, by means of hyperoxygenation, prior, during and after endotracheal tube suctioning in terms of preventing transient hypoxaemia, during suctioning of ventilated neonates with respiratory distress syndrome.

This objective will be reached by using preoxygenation during the experimental phase of the research design, while the infant is monitored.

3.2 RESEARCH DESIGN

A quantitative, explanatory design with experimental method, is to be undertaken within the context of a neonatal unit, in a private clinic within the Johannesburg area.
3.2.1 Pre - post-test control / experimental group design

The same infant is used in the pretest and then 6 hours later in the post-test. During the pretest control phase the infant is suctioned without preoxygenation. During the post-test experimental phase the infant is suctioned with preoxygenation.

Pretest Control 1 → Treatment → Control 2
ET Tube Suctioning

MANIPULATION OF INDEPENDENT VARIABLE : PREOXYGENATION

Post-test Experiment 1 → Treatment → Experiment 2
ET Tube Suctioning

This study is designed to prevent other elements from intruding into observation of the specific cause and effect being examined:

1) Systematic sampling
2) Controlled manipulation of the independent variable
3) Control of the experimental situation, including a control.

In this study, the treatment is under control of the researcher. The dependent variable is measured twice, before and after the manipulation of the independent variable. Characteristics of infants included in the study are narrowly defined, the independent variable is provided in a precisely defined way, the dependent variable is carefully operationalized and the situation in which the study is conducted is controlled to prevent interference of unstudied factors from modifying the dynamics of the process being studied.

3.2.2 Variables

Independent variable

Preoxygenation, by means of hyperoxygenation, during suctioning procedure.

Hyperoxygenation by increasing the FiO2 (concentration of inspired oxygen), during the experimental phase, by 10% at beginning of the procedure i.e. 5 minutes immediately
before suctioning and maintaining throughout. Wean FiO2 10% slowly, by 2% at one minute intervals at the end of the procedure i.e. within 5 minutes of completing suctioning.

**Dependent variable**

Transcient hypoxaemia

As a patient is suctioned, oxygen is removed along with the secretions, resulting in transcient suction - induced hypoxaemia i.e. deficient oxygenation of the blood. The arterial partial pressure of oxygen (PaO2) is less than normal, due to suctioning procedure and can be identified by a decrease in transcutaneous oximeter saturation (TO2) < 90% or a decrease in PaO2 < 6,6 kPa / < 50 mmHg.

**Other variables**

i) Number of aspirations = 3
ii) Duration of each aspiration = maximum 20 seconds
iii) No physiotherapy to be used
iv) Instillation of named NaCl 0,2 - 0,5ml during second aspiration
v) Maximum negative pressure
vi) Catheter size
vii) Length of catheter passed

Points v, vii, viii are illustrated in the table below and depend on birth weight and thus endotracheal tube size.

<table>
<thead>
<tr>
<th>Max Neg Pressure</th>
<th>Tube size</th>
<th>Birth weight</th>
<th>Catheter size</th>
<th>Catheter length passed</th>
</tr>
</thead>
<tbody>
<tr>
<td>80 mmHg</td>
<td>2,5</td>
<td>1000 - 1500g</td>
<td>6</td>
<td>+/- 9cm</td>
</tr>
<tr>
<td>100 mmHg</td>
<td>3,0</td>
<td>1600 - 2000g</td>
<td>6</td>
<td>+/- 10cm</td>
</tr>
<tr>
<td>120 mmHg</td>
<td>3,5</td>
<td>2100 - 2600g</td>
<td>8</td>
<td>+/- 11cm</td>
</tr>
</tbody>
</table>

These are recommended values as seen in chapter 2 PG 23 - 24. and catheter length passed also depends on where the endotracheal tube is strapped and how much is sticking outside the infant.
3.3 RESEARCH METHODOLOGY

3.3.1 Gathering of data

The same infant is used during the pretest and the post-test 6 hours later. During the pretest control phase the infant is suctioned without preoxygenation. During the post-test experimental phase the infant is suctioned with preoxygenation - FiO2 (concentration of inspired oxygen) is increased 10%, 5 minutes before suctioning and maintained throughout the suction procedure. On completion of suction procedure FiO2 is then weaned by 10% by decreasing 2% at 1 minute intervals. Weaning is complete 5 minutes after suctioning is complete. Observations are monitored throughout the whole process.

A specific protocol is followed during endotracheal tube suctioning of ventilated infants with respiratory distress syndrome, including monitoring of vital signs and manipulation of variables. This specific protocol has been assembled from the literature survey and suctioning techniques currently being utilised at several institutions in the Witwatersrand area.

3.3.1.1 Basic Procedure

Goal

* Ensure patent airway for unobstructed flow of gas and for proper lung expansion
* Maintain proper oxygenation during procedure.
* Ensure an atraumatic, safe procedure.

Equipment

* Sterile suction catheter, appropriate size.
* Sterile disposable glove.
* Sterile 0,9% NaCl (less than 6hrs old) in syringe warmed to body temperature.

Preparation

* Check Laerdal resuscitator and tubing, place next to patient and check oxygen
concentration is correct.
* Set audible beep volume on cardiac monitor.
* Aspirate nasogastric tube.
* Ensure pulse oximeter and ventilator are accessible. Ensure tracing is visible.
* Check suction pressure is set correctly.
* Check the endotracheal tube is securely strapped.
* Estimate correct catheter length to be used.

Procedure

* Take arterial blood gas 5 minutes before procedure.
* Record vital signs at beginning of procedure - heart rate, respiration, blood pressure and transcutaneous oximeter saturation.
* Observe transcutaneous oximeter saturation on monitor throughout and listen to audible beep of heart rate.
*** Disconnect ventilator and insert catheter (measured length) +/- 1/2 cm beyond the tip of the endotracheal tube. Withdraw by +/- 1/2 cm and only then apply suction. Withdraw catheter, while rolling in a smooth manner through fingers. Procedure to take no longer than 20 seconds.
* Observe consistency and volume of secretions on withdrawal of catheter.
* Reattach circuit to patient and wait 30 seconds while ventilating the patient to re-establish transcutaneous oximeter saturation > 90%.
* Disconnect ventilator and instil 0.9% warmed NaCl, a few drops to 0.5ml pm. Reattach ventilator and allow a few breaths. ***
* Repeat from *** to *** maximum x 3 times aspirations.
* Suction patient's naso and oropharynx thereafter.
* Discard glove and catheter.
* Observe the patient's condition.
* Record vital signs - heart rate, respiration, blood pressure and transcutaneous oximeter saturation.
* Record volume and consistency of secretions.
* Do arterial blood gas 30 minutes after suction procedure complete.

Exclude physiotherapy from procedure. All subjects are day 1 to 4 days postnatal age with respiratory distress syndrome and won't have excessive secretions.

Control suction procedure to be done without preoxygenation and experimental suction procedure to be done 6hrs later, on the same infant, utilising preoxygenation by hyperoxygenation, as described on PG 38.

Preoxygenation by hyperoxygenation is used only, instead of other methods.

Insufflation, can increase dead space and risk of contamination of suction catheter, lengthen suction procedure due to difficulty in advancing catheter through adapter and create site for tapping secretions (Hodge, 1991: 10-12). Insufflation cannot at present be utilised in South Africa as the specific catheter required is not supplied at present.

Newborns are on high rates and hyperventilating beyond bagging capabilities could cause desaturation, and decrease heart rate +/- 10% below baseline (Hodge, 1991: 10-12).

3.3.2 Sampling

Population

All ventilated neonates admitted to a private neonatal unit, Johannesburg.

Sample plan

Systematic sampling, of the first 20 ventilated neonates with respiratory distress syndrome admitted to a private neonatal unit was carried out. If they fitted the criteria as listed below, they were included in the study.

Criteria for inclusion

<table>
<thead>
<tr>
<th>CRITERIA</th>
<th>RATIONALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational age 26 - 37 weeks</td>
<td>Distressed newborns have impaired autoregulation of cerebral blood flow</td>
</tr>
<tr>
<td>appropriate for gestational age</td>
<td>predisposing to intra-ventricular haemorrhage. Incidence of intra-</td>
</tr>
<tr>
<td></td>
<td>ventricular haemorrhage increases when birth weight &lt; 1000grams or</td>
</tr>
<tr>
<td>Mean birth weight 900g - 2600g</td>
<td>&lt; 28 weeks, reported at 61 - 70% (Dietch, 1993: 7)</td>
</tr>
<tr>
<td>Ventilated babies on IPPV</td>
<td></td>
</tr>
<tr>
<td>(intermittent positive pressure ventilation) with respiratory distress syndrome.</td>
<td></td>
</tr>
</tbody>
</table>
* Postnatal age 1 to 4 days
Progressive chronic lung disease, broncho pulmonary dysplasia follows protracted periods of mechanical ventilation (Korones, 1986: 262-265)

EXCLUDE BABIES
If babies have a seizure disorder or congenital heart disease, or if haemodynamically unstable.
See above rationale.
These conditions may alter arterial blood gas results thus providing a false picture.

* Arterial Blood Gas: -
  
  PaO₂ 6.6 - 12 kPa / 50 - 90 mmHg (Arterial partial pressure of oxygen in the blood).

  Within normal parameters.
  Hyperoxia and possible side-effects occur > 100 mmHg / > 13.3 kPa.
  Therefore sources suggest keeping PaO₂ < 90 mmHg / < 12 kPa.

* Haemoglobin > 10mg% (Hb)

  Within normal parameter. A fall in haemoglobin, accentuated by blood loss associated with multiple blood sampling, results in a shift to the right in the oxygen dissociation curve and thus lowers oxygen carrying capacity of the blood. (Roberton, 1986: 265 - 266; Korones, 1986: 233-234).

Ventilator settings: -

FiO₂ (concentration of inspired oxygen)

If > 90% unable to preoxygenate by increasing FiO₂ by 10%

PIP (peak inspiratory pressure) < 25cmH₂O

If > 25cm H₂O increased risk of pneumothoraces synonymous with physiological PEEP.
3.3.3 Measuring strategy

Direct physiologic dimensions are used. There is a control pre-test and experimental post-test, 6hrs apart, on the same infant. Infants systematically selected, on admission if they fit the criteria. The treatment is controlled by the researcher. The dependent variable is measured twice, before and after the manipulation of the independent variable.

The instruments include measurement of arterial blood gases, heart rate, blood pressure, transcutaneous oximeter saturation and respiration, occurs before and after manipulation of independent variable.

3.3.3.1 Instruments

3.3.3.1.1 ABL 520 blood gas system

The ABL 520 is designed and intended primarily for use on human blood. The ABL 520 measures pH, PaCO2 (partial pressure of carbon dioxide), PaO2 (partial pressure of oxygen), Hb (haemoglobin) and SpO2 (saturation).

History

Founded in 1935, Radiometer was originally involved in developing measuring instruments for the radio and chemical industries. Production of the Acid-Base Laboratory (ABL) series, started in 1973 with the ABL, the first computer-controlled totally automatic pH / blood gas instrument. Successive refinements have been incorporated into each new member in the series, improving accuracy and reliability. The ABL 520 is the latest in this series and is the first instrument in the world to combine measurement of blood gas and all the oximetry parameters. (Radiometer, 1991a: 1.2 - 1.5).

Accuracy and reliability

Is achieved through a combination of factors from high quality components and precise manufacturing standards, through calibration intervals and automatic self-diagnostic functions. Allows calibration intervals to be adjusted to match individual needs - from 30 minutes up to 8 hours, or after every measurement.
Blood sampling is convenient with designed arterial blood samplers and heparinized capillary tubes. The machine has a sample preheater to adjust even ice-cooled samples to precisely 37°C. There are sensors to detect samples compromising air bubbles and to monitor the sample size, and automatic verification of each electrode response before results are released.

Radiometer has specified the instrument according to a set of accepted reference methods to ensure its reliability. This informs the operator about accuracy and precision. Quality control is a method for verifying these specifications by making measurements in the same way as sample measurements, but on a prepared material with predetermined value ranges. (Radiometer, 1991a: 1.2 - 1.5).

Validity

Refers to content and construct validity.

Statistic factor - is defined as the factor by which the control range must be expanded to define the statistics range - which is the range within which all results from all valid measurements on control solution are included in the quality control statistics. (Radiometer, 1991b: 5.2 - 5.4).

Valid measurements on control solution are those which results have no question marks for any of the parameters, and which are not "unknown". All results outside the statistics range or with at least one parameter marked with "?" are excluded from the statistics. (Radiometer, 1991b: 5.2 - 5.4).

The machine performs a number of calibrations to ensure that the signals are converted to accurate and valid values during measurements. i.e. status and sensitivity of pH and PaCO2 electrodes and zero point and sensitivity of PaO2 electrode. (Radiometer, 1991b: 5.2 - 5.4).

3.3.3.1.2 Other instruments

Standardised international equipment being used:

- Heart rate - using cardiac monitor
- Respiratory rate - using cardiac monitor
- Blood pressure - using dynamap
Transcutaneous oximeter saturation

Transcutaneous oximeter saturation (TO2) remains an adjunct and not a substitute for intermittent blood sampling but because arterial blood gas sampling requires removal of blood, repeated sampling has potential for numerous risks and complications. TO2 is a non invasive continuous monitoring of oxygen status by means of pulse oximetry, between gases. (Klaus and Fanaroff, 1986: 176 - 177; Korones, 1986: 236 - 238; Levin and Morriss, 1990: 927 - 929; Gunderson and Cusson, 1993: 71 - 72).

However, TO2 reflects the oxygen saturation only, of the arterial blood i.e. percentage of arterial haemoglobin that is saturated with oxygen. No information about haemoglobin concentration, cardiac output, efficiency of oxygen delivery to tissues, or consumption or sufficiency of oxygen is provided.

Limitations of TO2 :-

* Adhesive and probe can damage skin.
* Sensitive to spontaneous movement of extremities.
* Patient's pulse must be present and unimpaired i.e. cannot be placed distal to blood pressure cuff.
* Requires sufficient haemoglobin in blood i.e. undependable in severe anaemia.
* Can be affected by bright ambient light eg phototherapy. (Place reflective material over it to insulate against light filtering to probe sensor).
* May pick up venous pulsations eg high PEEP (positive end expiratory pressure) and severe right sided heart failure.
* Dyes and high levels of bilirubin (720mg / dL) in the bloodstream may effect accuracy. To compensate for variable arterial perfusion, light intensities on transmitters can be adjusted so that level of light received at detector provides an acceptable pulse wave.
* Time lag between physiologic changes within the body and actual detection by monitor occurs, therefore continue data collection for 2 - 3 minutes following completion of treatment / intervention.

Triangulation of values with other biophysical measures i.e. heart rate, blood pressure and using multiple measures will provide a more accurate clinical picture, of physiologic patterns and possible subsequent alterations, in those patterns. (Gunderson and Cusson, 1993: 71 - 72; Klaus and Fanaroff, 1986: 176 - 177; Korones, 1986: 236 - 238).
3.3.4 Ethical considerations

During collection of data, the following ethical considerations will be taken into account.

3.3.4.1 Consent

* Written permission obtained from management of private clinic.
* Written informed consent obtained from parents of infants treated in the neonatal unit.
* Co-operation from nursing staff in the neonatal unit.

3.3.4.2 Risks to infants

As outlined from literature survey, the risks of oxygen toxicity i.e. retinopathy and broncho pulmonary dysplasia are minimal as they occur only if infant is on high concentrations of oxygen - FiO2 > 60% for many days; if PaO2 (partial pressure of oxygen) > 100mmHg; and from high ventilator pressures among other factors already mentioned.

The risks of increasing FiO2 (concentration of inspired oxygen) by 10% for 5 minutes and maintaining throughout suction procedure are outweighed by the benefits of reducing the risk of transient hypoxaemia. Besides the fact that no suction protocol has been standardised as yet, and preoxygenation is administered ad-lib at present or not at all in various neonatal units in South Africa.

The procedure was done once only. The blood gases were fitted into nursing care routine as far as possible. Two extra blood samples of 0.2mL each would not dramatically / significantly reduce Hb of infant and costs of any extra blood samples will be covered by researcher.

3.3.5 General reliability and validity of study

3.3.5.1 Reliability

Is the extend to which measures are consistent in the study. This is achieved by :-
Statistical methods

Test - retest method with equivalent forms - ensure stability and equivalence by obtaining the score on one form, allowing a time lapse and correcting with that obtained on the other score, using a correlation coefficient.

Data Analysis

Statistical analysis is used for data that are in the form of categories (nominal data), and is used for comparing samples.

3.3.5.2 Validity

Reflects the authenticity of the study. This is achieved by :-

Content validity

which is operationalized by :-

i) Experts review the objectives and test items and decide on the appropriateness of the test items.

ii) Obtained from instrument validity and reliability already discussed previously in this chapter. See pg 44 - 46.

Construct validity

i) Theoretical foundation of Nursing of the Whole Person theory interplaying with the measurements of the constructs that make up the theory.

ii) Use of literature survey - from primary and secondary sources and in terms of geographical aspect i.e. national and international background.
Criterion-related validity

Predicts effective clinical performance - able to estimate the extent to which the criterion will be manifested in the future and is defensible on logical and theoretical grounds.

Internal validity

As the statistical methods and data analysis is used uniformly for all the infants, results can be internally compared and as these methods are reliable they make the study internally valid, within a theoretical foundation.

External validity

This reliable statistical method and data analysis can possibly be applied in other neonatal settings outside the one used in the study. Even though the sample is small the findings might be generalised and therefore the study has external validity.

3.4 DATA ANALYSIS

The data obtained was tabulated for each infant then analysed. Data was compared between control phase where no preoxygenation was used and the experimental phase when preoxygenation was used during suctioning. Statistical analysis of results were obtained using the package 'Statistica' for windows 95, which was then represented in graphic format. The post-hoc comparison, the analysis of covariance procedures and the statistical t-test for dependant samples is used, where "p" is the probability of rejecting the null hypothesis when it is in fact true. If the p value is larger than 0.05 then the result is not significant. If p < 0.05 then the result is significant at the 5% level. If p < 0.01 then the result is highly significant at the 1% level.

3.5 SUMMARY

Chapter 3 dealt with the research design and methodology. It describes how the research was conducted and the method used. Reliability and validity is also discussed. Chapter 4 will cover the analysis of the results.
CHAPTER 4

RESULTS OF THE STUDY

4.1 INTRODUCTION

The specific objective of the study is to evaluate the effect of using preoxygenation by means of hyperoxygenation, prior, during and after endotracheal tube suctioning in terms of preventing transient hypoxaemia, during suctioning of ventilated neonates with respiratory distress syndrome.

4.2 DESCRIPTION OF RESULTS

The data of each baby is set out in a table format. All the babies' details are included i.e. age, gestation, diagnosis and drug therapy. Other details of suctioning are also included i.e. maximum negative pressure used during suctioning, endotracheal tube size, birth weight, size of suction catheter used and length of suction catheter passed down endotracheal tube.

The babies' vital signs are then tabulated as follows: heart rate, respiration, blood pressure and transcutaneous oximeter saturation is measured for each baby during the control pre - test 10 minutes before suctioning, 5 minutes before suctioning, during second aspiration during suctioning, on completion of suctioning, 5 minutes after suctioning and 30 minutes after suctioning. These same vital signs are then measured 6 hours later for each baby during the experimental post - test at the same intervals.

The arterial blood gas is also measured on each baby during the control and experimental phases, 10 minutes prior to suctioning and 30 minutes after completing suctioning. The time taken to regain baseline heart rate and saturation is also measured.

The results are also graphically depicted in figures 1 - 13 discussed later in this chapter.

4.3 RESULTS OF EACH CASE

The results of each baby are tabulated as discussed above.
4.3.1 RESULTS OF BABY 1

<table>
<thead>
<tr>
<th>VENTILATOR SETTINGS</th>
<th>VENTILATOR SETTINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONTROL</td>
<td>EXPERIMENTAL</td>
</tr>
<tr>
<td>FIO2</td>
<td>FIO2</td>
</tr>
<tr>
<td>RATE</td>
<td>RATE</td>
</tr>
<tr>
<td>PRESSURE</td>
<td>PRESSURE</td>
</tr>
<tr>
<td>34%</td>
<td>34%</td>
</tr>
<tr>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>18/5</td>
<td>18/5</td>
</tr>
</tbody>
</table>

BABY: 1
DIGNOSIS: HMD Gr III, IVH Gr III
AGE: DAY 4
GESTATION: 26 WEEKS

BIRTH WEIGHT: 940 g
TUBE SIZE: 2.5
CATH SIZE: 6
LENGTH PASSED: 8.5 cm

MAX NEG PRESSURE: 80 mmHg
DRUGS: Pen G, Amikin, Phenobarb

<table>
<thead>
<tr>
<th>10 MINUTES BEFORE</th>
<th>5 MINUTES BEFORE</th>
<th>DURING 2nd ASPIRATION</th>
<th>ON COMPLETION</th>
<th>5 MINUTES AFTER</th>
<th>30 MINUTES AFTER</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR 142</td>
<td>144</td>
<td>142</td>
<td>114</td>
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<td>120</td>
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<tr>
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<td>21</td>
<td>16</td>
<td>21</td>
<td>24</td>
<td>21</td>
</tr>
<tr>
<td>BP 48/22</td>
<td>39/18</td>
<td>52/23</td>
<td>60/28</td>
<td>45/25</td>
<td>62/25</td>
</tr>
<tr>
<td>SATS 95</td>
<td>98</td>
<td>95</td>
<td>84</td>
<td>89</td>
<td>87</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>90</td>
<td></td>
</tr>
<tr>
<td>10 MINUTES BEFORE</td>
<td>5 MINUTES BEFORE</td>
<td>DURING 2nd ASPIRATION</td>
<td>ON COMPLETION</td>
<td>5 MINUTES AFTER</td>
<td>30 MINUTES AFTER</td>
</tr>
<tr>
<td>HR 142</td>
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<td>BP 48/22</td>
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<td>87</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>90</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>AB GAS</th>
<th>INCREASED</th>
<th>DECREASED</th>
<th>Fi O2</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>7.279</td>
<td>7.247</td>
<td>7.307</td>
</tr>
<tr>
<td>PCO2</td>
<td>39.2</td>
<td>43.4</td>
<td>36.8</td>
</tr>
<tr>
<td>PO2</td>
<td>64.4</td>
<td>76.3</td>
<td>71.8</td>
</tr>
<tr>
<td>SBC</td>
<td>17.9</td>
<td>17.6</td>
<td>18.4</td>
</tr>
<tr>
<td>BE</td>
<td>-7.8</td>
<td>-7.9</td>
<td>-7.3</td>
</tr>
<tr>
<td>SATS</td>
<td>88.9</td>
<td>92.1</td>
<td>92.3</td>
</tr>
</tbody>
</table>

TIME TAKEN TO REGAIN BASELINE HEART RATE AND TRANSCUTANEOUS OXIMETER SATURATION.

<table>
<thead>
<tr>
<th></th>
<th>CONTROL</th>
<th>EXPERIMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>75 Sec</td>
<td>25 Sec</td>
</tr>
<tr>
<td>SATS</td>
<td>110 Sec</td>
<td>90 Sec</td>
</tr>
</tbody>
</table>

KEY: HR - heart rate, RESP - respiration, BP - blood pressure, SATS - transcutaneous oximeter saturation, AB GAS - arterial blood gas
FiO2 - fraction of inspired oxygen, RATE - ventilator breathe rate, PRESSURE - peak inspiratory pressure
PFC - persistant fetal circulation, IVH - intra-ventricular haemorrhage, HMD - hyaline membrane disease grade I - IV
### 4.3.2 RESULTS OF BABY 2

#### VENTILATOR SETTINGS

<table>
<thead>
<tr>
<th>Control</th>
<th>Experimental</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO2</td>
<td>RATE</td>
</tr>
<tr>
<td>37%</td>
<td>19</td>
</tr>
<tr>
<td>32%</td>
<td>12</td>
</tr>
</tbody>
</table>

#### BABY: 2

- **DIAGNOSIS:** HMD Gr III
- **AGE:** DAY 3
- **GESTATION:** 32 WEEKS

#### BIRTH WEIGHT: 1625 g

- **TUBE SIZE:** 3.0
- **CATH SIZE:** 6
- **LENGTH PASSED:** 9 cm

#### MAX NEG PRESSURE: 100 mmHg

- **DRUGS:** Survanta 60 hrs previously

#### TIME TAKEN TO REGAIN BASELINE HEART RATE AND TRANSCUTANEOUS OXIMETER SATURATION.

<table>
<thead>
<tr>
<th>HR</th>
<th>RESP</th>
<th>BP</th>
<th>SATS</th>
<th>PH</th>
<th>PCO2</th>
<th>PO2</th>
<th>SBC</th>
<th>BE</th>
<th>SATS</th>
</tr>
</thead>
<tbody>
<tr>
<td>131</td>
<td>136</td>
<td>137</td>
<td>136</td>
<td>105</td>
<td>148</td>
<td>150</td>
<td>160</td>
<td>143</td>
<td>143</td>
</tr>
<tr>
<td>29</td>
<td>35</td>
<td>33</td>
<td>37</td>
<td>58</td>
<td>68</td>
<td>32</td>
<td>38</td>
<td>19</td>
<td>29</td>
</tr>
<tr>
<td>64/39</td>
<td>60/34</td>
<td>61/35</td>
<td>60/37</td>
<td>78/46</td>
<td>73/46</td>
<td>74/46</td>
<td>74/46</td>
<td>58/37</td>
<td>68/45</td>
</tr>
</tbody>
</table>

- **AB GAS**
  - **INCREASED FIO2 10% preoxygenation**
  - **DECREASED FIO2 2% at 1 min. intervals**

- **FiO2 weaning complete**
  - 7.418
  - 7.341
  - 28.2
  - 35.7
  - 90.1
  - 97
  - 20.3
  - 19.7
  - -5.7
  - -5.9
  - 96.9
  - 98.8

<table>
<thead>
<tr>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 Sec</td>
<td></td>
<td>80 Sec</td>
<td></td>
<td>Did not drop</td>
<td></td>
<td>20 Sec</td>
<td></td>
</tr>
</tbody>
</table>

**KEY:**
- HR - heart rate, RESP - respiration, BP - blood pressure, SATS - transcutaneous oximeter saturation, AB GAS - arterial blood gas
- FiO2 - fraction of inspired oxygen, RATE - ventilator breathe rate, PRESSURE - peak inspiratory pressure
- PFC - persistent fetal circulation, IVH - intra-ventricular haemorrhage, HMD - hyaline membrane disease grade I - IV
4.3.3 RESULTS OF BABY 3

<table>
<thead>
<tr>
<th>VENTILATOR SETTINGS</th>
<th>VENTILATOR SETTINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONTROL</td>
<td>EXPERIMENTAL</td>
</tr>
<tr>
<td>FIO2</td>
<td>FIO2</td>
</tr>
<tr>
<td>RATE</td>
<td>RATE</td>
</tr>
<tr>
<td>PRESSURE</td>
<td>PRESSURE</td>
</tr>
<tr>
<td>28%</td>
<td>26%</td>
</tr>
<tr>
<td>16</td>
<td>12</td>
</tr>
<tr>
<td>18/5</td>
<td>18/5</td>
</tr>
</tbody>
</table>

BABY: 3
DIAGNOSIS: HMD Gr III
AGE: DAY 4
GESTATION: 31 WEEKS

<table>
<thead>
<tr>
<th>BIRTH WEIGHT:</th>
<th>MAX NEG PRESSURE:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1040 g</td>
<td>80 mmHg</td>
</tr>
<tr>
<td>TUBE SIZE:</td>
<td>DRUGS: Pen G, Amikin, Claforan</td>
</tr>
<tr>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>CATH SIZE:</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td></td>
</tr>
<tr>
<td>LENGTH PASSED:</td>
<td></td>
</tr>
<tr>
<td>9 cm</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>10 MINUTES BEFORE</th>
<th>5 MINUTES BEFORE</th>
<th>DURING 2nd ASPIRATION</th>
<th>ON COMPLETION</th>
<th>5 MINUTES AFTER</th>
<th>30 MINUTES AFTER</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>RESP</td>
<td>BP</td>
<td>SATS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>154</td>
<td>61</td>
<td>52/34</td>
<td>94</td>
<td>150</td>
<td>66</td>
</tr>
<tr>
<td>158</td>
<td>58/33</td>
<td>54/33</td>
<td>93</td>
<td>152</td>
<td>65</td>
</tr>
<tr>
<td>162</td>
<td>60/34</td>
<td>60/36</td>
<td>94</td>
<td>152</td>
<td>65</td>
</tr>
<tr>
<td>140</td>
<td>22</td>
<td>60/36</td>
<td>83</td>
<td>185</td>
<td>77</td>
</tr>
<tr>
<td>152</td>
<td>29</td>
<td>63/37</td>
<td>88</td>
<td>195</td>
<td>75</td>
</tr>
<tr>
<td>195</td>
<td>75</td>
<td></td>
<td>92</td>
<td>185</td>
<td>66</td>
</tr>
<tr>
<td>173</td>
<td>66</td>
<td></td>
<td></td>
<td>173</td>
<td>55</td>
</tr>
<tr>
<td>160</td>
<td>55</td>
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<tr>
<td>160</td>
<td>53</td>
<td></td>
<td></td>
<td>160</td>
<td>59</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>AB GAS</th>
<th>INCREASED FIO2 10%</th>
<th>DECREASED FIO2 2%</th>
<th>FiO2 weaning</th>
<th>CONTROL</th>
<th>EXP</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>CONTROL</td>
<td>EXP</td>
</tr>
<tr>
<td>PCO2</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>33.4</td>
<td></td>
</tr>
<tr>
<td>35.2</td>
<td>35.6</td>
<td>35.2</td>
<td>35.6</td>
<td>36.7</td>
<td></td>
</tr>
<tr>
<td>PO2</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>61.5</td>
<td></td>
</tr>
<tr>
<td>83.1</td>
<td>57.4</td>
<td>83.1</td>
<td>57.4</td>
<td>67.1</td>
<td></td>
</tr>
<tr>
<td>SBC</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>19.8</td>
<td></td>
</tr>
<tr>
<td>20.2</td>
<td>20.3</td>
<td>20.2</td>
<td>20.3</td>
<td>20.7</td>
<td></td>
</tr>
<tr>
<td>BE</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>-4</td>
<td></td>
</tr>
<tr>
<td>-4.1</td>
<td>-3.9</td>
<td>-4.1</td>
<td>-3.9</td>
<td>-3.8</td>
<td></td>
</tr>
<tr>
<td>SATS</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>CONTROL EXP</td>
<td>90.8</td>
<td></td>
</tr>
<tr>
<td>95.7</td>
<td>98.5</td>
<td>95.7</td>
<td>98.5</td>
<td>91.2</td>
<td></td>
</tr>
</tbody>
</table>

TIME TAKEN TO REGAIN BASELINE HEART RATE AND TRANSCUTANEOUS OXIMETER SATURATION.

| HR: CONTROL | 15 Sec | HR: EXPERIMENT | 5 Sec |
| SATS: CONTROL | 95 Sec | SATS: EXPERIMENT | 25 Sec |

KEY: HR - heart rate, RESP - respiration, BP - blood pressure, SATS - transcutaneous oximeter saturation, AB GAS - arterial blood gas, FIO2 - fraction of inspired oxygen, RATE - ventilator breathe rate, PRESSURE - peak inspiratory pressure, PFC - persistent fetal circulation, IVH - intra-ventricular haemorrhage, HMD - hyaline membrane disease grade I - IV
### 4.3.4 RESULTS OF BABY 4

<table>
<thead>
<tr>
<th>VENTILATOR SETTINGS</th>
<th>VENTILATOR SETTINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CONTROL</strong></td>
<td><strong>EXPERIMENTAL</strong></td>
</tr>
<tr>
<td>FIO2</td>
<td>RATE</td>
</tr>
<tr>
<td>55%</td>
<td>40</td>
</tr>
<tr>
<td>41%</td>
<td>32</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BIRTH WEIGHT: 1375 g</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAX NEG PRESSURE: 100 mmHg</td>
</tr>
<tr>
<td>DRUGS: Pen G. Amikin</td>
</tr>
</tbody>
</table>

**BABY:** 4  
**DIAGNOSIS:** HMD Gr II  
**AGE:** DAY 2  
**GESTATION:** 31 WEEKS  
**LENGTH PASSED:** 8.5 cm

| TIME TAKEN TO REGAIN BASELINE HEART RATE AND TRANSCUTANEOUS OXIMETER SATURATION. |
|-----------------------------|-----------------------------|
| HR: CONTROL | EXPERIMENT | SATS: CONTROL | EXPERIMENT |
| 20 Sec | 10 Sec | 35 Sec | 20 Sec |

**KEY:**  
HR - heart rate, RESP - respiration, BP - blood pressure, SATS - transcutaneous oximeter saturation, AB GAS - arterial blood gas  
FiO2 - fraction of inspired oxygen, RATE - ventilator breathe rate, PRESSURE - peak inspiratory pressure  
PFC - persistent fetal circulation, IVH - intra-ventricular haemorrhage, HMD - hyaline membrane disease grade I - IV
### 4.3.5 RESULTS OF BABY 5

#### VENTILATOR SETTINGS

<table>
<thead>
<tr>
<th>CONTROL</th>
<th>EXPERIMENTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO2</td>
<td>RATE</td>
</tr>
<tr>
<td>68%</td>
<td>60</td>
</tr>
<tr>
<td>54%</td>
<td>48</td>
</tr>
</tbody>
</table>

#### BABY: 5
- DIAGNOSIS: HMD Gr II, PFC, Pneumothorax
- AGE: DAY 3
- GESTATION: 34 WEEKS
- BIRTH WEIGHT: 2325 g
- TUBE SIZE: 3.0
- CATH SIZE: 6
- LENGTH PASSED: 11 cm
- MAX NEG PRESSURE: 100 mmHg
- DRUGS: Amikin, Cefotaxime, Pavulon, Morphine, Isuprel

#### Time Taken to Regain Baseline Heart Rate and Transcutaneous Oximeter Saturation

<table>
<thead>
<tr>
<th></th>
<th>10 MINUTES BEFORE</th>
<th>5 MINUTES BEFORE</th>
<th>DURING 2nd ASPIRATION</th>
<th>ON COMPLETION</th>
<th>5 MINUTES AFTER</th>
<th>30 MINUTES AFTER</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>150</td>
<td>148</td>
<td>146</td>
<td>147</td>
<td>125</td>
<td>140</td>
</tr>
<tr>
<td>RESP</td>
<td>60</td>
<td>48</td>
<td>60</td>
<td>48</td>
<td>60</td>
<td>48</td>
</tr>
<tr>
<td>BP</td>
<td>59/33</td>
<td>58/32</td>
<td>62/34</td>
<td>58/33</td>
<td>70/44</td>
<td>65/41</td>
</tr>
<tr>
<td>SATS</td>
<td>99</td>
<td>97</td>
<td>99</td>
<td>97</td>
<td>92</td>
<td>94</td>
</tr>
</tbody>
</table>

#### AB GAS

<table>
<thead>
<tr>
<th></th>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>7.471</td>
<td></td>
<td>7.495</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCO2</td>
<td>24.5</td>
<td></td>
<td>23.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PO2</td>
<td>80.7</td>
<td></td>
<td>69.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBC</td>
<td>17.8</td>
<td></td>
<td>18.1</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>BE</td>
<td>-4</td>
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<td>-3.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>SATS</td>
<td>96.5</td>
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<td>95.1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

- **CONTROL** - 8 Sec
- **EXP** - 48 Sec
- **CONTROL** - 5 Sec
- **EXP** - 22 Sec

**KEY:**
- HR - heart rate
- RESP - respiration
- BP - blood pressure
- SATS - transcutaneous oximeter saturation
- AB GAS - arterial blood gas
- FiO2 - fraction of inspired oxygen
- RATE - ventilator breathe rate
- PRESSURE - peak inspiratory pressure
- PFC - persistent fetal circulation
- IVH - intra-ventricular haemorrhage
- HMD - hyaline membrane disease grade I - IV
### 4.3.6 RESULTS OF BABY 6

#### VENTILATOR SETTINGS

<table>
<thead>
<tr>
<th></th>
<th>CONTROL</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO2</td>
<td>65%</td>
<td>28</td>
<td>22/5</td>
</tr>
<tr>
<td>RATE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PRESSURE</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>EXPERIMENT</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO2</td>
<td>63%</td>
<td>28</td>
<td>22/5</td>
</tr>
<tr>
<td>RATE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PRESSURE</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### BABY:
- 6

#### DIAGNOSIS:
- HMD Gr III

#### AGE:
- DAY 3

#### GESTATION:
- 32 WEEKS

#### BIRTH WEIGHT:
- 1535 g

#### TUBE SIZE:
- 3.0

#### CATH SIZE:
- 6

#### LENGTH PASSED:
- 10 cm

#### MAX NEG PRESSURE:
- 100 mmHg

#### DRUGS:
- Pen G, Amikin Survanta day 0

#### 10 MINUTES BEFORE | 5 MINUTES BEFORE | DURING 2nd ASPIRATION | ON COMPLETION | 5 MINUTES AFTER | 30 MINUTES AFTER
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
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<td>167</td>
<td>155</td>
<td>166</td>
<td>156</td>
<td>115</td>
</tr>
<tr>
<td>RESP</td>
<td>53</td>
<td>51</td>
<td>55</td>
<td>52</td>
<td>30</td>
</tr>
<tr>
<td>BP</td>
<td>52/39</td>
<td>52/35</td>
<td>50/38</td>
<td>52/34</td>
<td>65/45</td>
</tr>
<tr>
<td>SATS</td>
<td>92</td>
<td>92</td>
<td>91</td>
<td>92</td>
<td>87</td>
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</tbody>
</table>

#### AB GAS

<table>
<thead>
<tr>
<th></th>
<th>CONTROL</th>
<th>EXP</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>7.333</td>
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</tr>
<tr>
<td>PCO2</td>
<td>41.2</td>
<td>35.8</td>
</tr>
<tr>
<td>PO2</td>
<td>81</td>
<td>69.3</td>
</tr>
<tr>
<td>SBC</td>
<td>21</td>
<td>21.8</td>
</tr>
<tr>
<td>BE</td>
<td>-4.1</td>
<td>-3.1</td>
</tr>
<tr>
<td>SATS</td>
<td>94.8</td>
<td>93.2</td>
</tr>
</tbody>
</table>

#### INCREASED
- FiO2 10% preoxygenation

#### DECREASED
- FiO2 2% at 1 min. intervals
- FiO2 weaning complete

<table>
<thead>
<tr>
<th>FiO2</th>
<th>CONTROL</th>
<th>EXP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>81.9</td>
<td>92.6</td>
</tr>
<tr>
<td></td>
<td>22.1</td>
<td>21.9</td>
</tr>
<tr>
<td></td>
<td>-2.8</td>
<td>-3</td>
</tr>
<tr>
<td></td>
<td>95.3</td>
<td>98.5</td>
</tr>
</tbody>
</table>

#### TIME TAKEN TO REGAIN BASELINE HEART RATE AND TRANSCUTANEOUS OXIMETER SATURATION.

<table>
<thead>
<tr>
<th></th>
<th>CONTROL</th>
<th>EXP</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>10 Sec</td>
<td></td>
</tr>
<tr>
<td>SATS</td>
<td>95 Sec</td>
<td></td>
</tr>
</tbody>
</table>

#### KEY:
- HR - heart rate
- RESP - respiration
- BP - blood pressure
- SATS - transcutaneous oximeter saturation
- AB GAS - arterial blood gas
- FiO2 - fraction of inspired oxygen
- RATE - ventilator breathe rate
- PRESSURE - peak inspiratory pressure
- PFC - persistent fetal circulation
- IVH - intra-ventricular haemorrhage
- HMD - hyaline membrane disease grade I - IV

56
### 4.3.7 RESULTS OF BABY 7

#### VENTILATOR SETTINGS

<table>
<thead>
<tr>
<th></th>
<th>CONTROL</th>
<th>EXPERIMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO2</td>
<td>RATE</td>
<td>PRESSURE</td>
</tr>
<tr>
<td>48%</td>
<td>41</td>
<td>22/5</td>
</tr>
<tr>
<td>42%</td>
<td>34</td>
<td>22/5</td>
</tr>
</tbody>
</table>

#### BABY:
- **Number:** 7
- **Diagnosis:** HMD Gr II - III, Pneumothorax
- **Age:** DAY 2
- **Gestation:** 35 WEEKS
- **Birth Weight:** 2360 g
- **Tube Size:** 3.0
- **Cath Size:** 6
- **Length Passed:** 10.5 cm
- **Max Neg Pressure:** 100 mmHg
- **Drugs:** Pen G, Amikin, Survanta day 1

#### Time Taken to Regain Baseline Heart Rate and Transcutaneous Oximeter Saturation:

<table>
<thead>
<tr>
<th></th>
<th>CONTROL</th>
<th>EXPERIMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR:</td>
<td>25 Sec</td>
<td>10 Sec</td>
</tr>
<tr>
<td>SATS:</td>
<td>100 Sec</td>
<td>45 Sec</td>
</tr>
</tbody>
</table>

#### Key:
- HR - heart rate
- RESP - respiration
- BP - blood pressure
- SATS - transcutaneous oximeter saturation
- AB GAS - arterial blood gas
- FiO2 - fraction of inspired oxygen
- RATE - ventilator breathe rate
- PRESSURE - peak inspiratory pressure
- PFC - persistent fetal circulation
- IVH - intra-ventricular haemorrhage
- HMD - hyaline membrane disease grade I - IV
**4.3.8 RESULTS OF BABY 8**

<table>
<thead>
<tr>
<th>VENTILATOR SETTINGS</th>
<th>VENTILATOR SETTINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONTROL</td>
<td>EXPERIMENT</td>
</tr>
<tr>
<td>FIO2</td>
<td>RATE</td>
</tr>
<tr>
<td>55%</td>
<td>30</td>
</tr>
</tbody>
</table>

**BABY:** 8  
**DIGNOSIS:** HMD Gr II  
**AGE:** DAY 1  
**GESTATION:** 35 weeks

**BIRTH WEIGHT:** 2400 g  
**MAX NEG PRESSURE:** 100 mmHg

<table>
<thead>
<tr>
<th><strong>EXP</strong></th>
<th><strong>CONTROL</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PH</strong></td>
<td>7.364</td>
</tr>
<tr>
<td><strong>PCO2</strong></td>
<td>27.7</td>
</tr>
<tr>
<td><strong>PO2</strong></td>
<td>62.7</td>
</tr>
<tr>
<td><strong>SBC</strong></td>
<td>17.9</td>
</tr>
<tr>
<td><strong>BE</strong></td>
<td>-8.9</td>
</tr>
<tr>
<td><strong>SATS</strong></td>
<td>90.6</td>
</tr>
</tbody>
</table>

**INCREASED**  
FIO2 10%  
**preoxygenation**

**DECREASED**  
FIO2 2%  
at 1 min.  
**weaning**

**TIME TAKEN TO REGAIN BASELINE HEART RATE AND TRANSCUTANEOUS OXIMETER SATURATION.**

<table>
<thead>
<tr>
<th><strong>HR:</strong></th>
<th><strong>SATS:</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CONTROL</strong></td>
<td>20 Sec</td>
</tr>
<tr>
<td><strong>CONTROL</strong></td>
<td>50 Sec</td>
</tr>
</tbody>
</table>

**KEY:**  
HR - heart rate, RESP - respiration, BP - blood pressure, SATS - transcutaneous oximeter saturation, AB GAS -arterial blood gas  
FIO2 - fraction of inspired oxygen, RATE - ventilator breathe rate, PRESSURE - peak inspiratory pressure  
PFC - persistant fetal circulation, IVH - intra-ventricular haemorrhage, HMD - hyaline membrane disease grade I - IV
### 4.3.9 RESULTS OF BABY 9

<table>
<thead>
<tr>
<th>VENTILATOR SETTINGS</th>
<th>CONTROL</th>
<th>EXCLUSIVE</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO2</td>
<td>RATE</td>
<td>PRESSURE</td>
</tr>
<tr>
<td>40%</td>
<td>33</td>
<td>22/5</td>
</tr>
<tr>
<td>36%</td>
<td>28</td>
<td>22/5</td>
</tr>
</tbody>
</table>

| BABY: | 9 |
| DIAGNOSIS: | HMD Gr II |
| AGE: | DAY 2 |
| GESTATION: | 34 WEEKS |
| BIRTH WEIGHT: | 2590 g |
| TUBE SIZE: | 3.0 |
| CATH SIZE: | 6 |
| LENGTH PASSED: | 10.5 cm |

| MAX NEG PRESSURE: | 100 mmHg |
| DRUGS: | Pen G, Amikin |

| TIME TAKEN TO REGAIN BASELINE HEART RATE AND TRANSCUTANEOUS OXIMETER SATURATION. |
|---|---|---|---|---|---|---|---|---|---|---|
| HR | CONTROL | 15 Sec | HR | EXPERIMENT | Did not drop |
| SATS | CONTROL | 140 Sec | SATS | EXPERIMENT | 30 Sec |

**KEY:**
- HR - heart rate
- RESP - respiration
- BP - blood pressure
- SATS - transcutaneous oximeter saturation
- AB GAS - arterial blood gas
- FIO2 - fraction of inspired oxygen
- RATE - ventilator breathe rate
- PRESSURE - peak inspiratory pressure
- PFC - persitant fetal circulation
- IVH - intra-ventricular haemorrhage
- HMD - hyaline membrane disease grade I - IV
# 4.3.10 RESULTS OF BABY 10

<table>
<thead>
<tr>
<th>VENTILATOR SETTINGS</th>
<th>VENTILATOR SETTINGS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CONTROL</strong></td>
<td><strong>EXPERIMENTAL</strong></td>
</tr>
<tr>
<td>FIO2</td>
<td>RATE</td>
</tr>
<tr>
<td>38%</td>
<td>16</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BABY:</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>DIAGNOSIS:</td>
<td>HMD Gr III</td>
</tr>
<tr>
<td>DAY</td>
<td>DAY 1</td>
</tr>
<tr>
<td>GESTATION:</td>
<td>33 WEEKS</td>
</tr>
</tbody>
</table>

| BIRTH WEIGHT: | 1480 g |
| TUBE SIZE: | 3.0 |
| CATH SIZE: | 6 |
| LENGTH PASSED: | 8.5 cm |

| MAX NEG PRESSURE: | 100 mmHg |
| DRUGS: | Pen G, Amikin |

<table>
<thead>
<tr>
<th>10 MINUTES BEFORE</th>
<th>5 MINUTES BEFORE</th>
<th>DURING 2nd ASPIRATION</th>
<th>ON COMPLETION</th>
<th>5 MINUTES AFTER</th>
<th>30 MINUTES AFTER</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>138</td>
<td>138</td>
<td>138</td>
<td>139</td>
<td>121</td>
</tr>
<tr>
<td>RESP</td>
<td>38</td>
<td>40</td>
<td>37</td>
<td>41</td>
<td>25</td>
</tr>
<tr>
<td>BP</td>
<td>45/32</td>
<td>48/32</td>
<td>46/32</td>
<td>49/32</td>
<td>55/38</td>
</tr>
<tr>
<td>SATS</td>
<td>99</td>
<td>98</td>
<td>99</td>
<td>98</td>
<td>93</td>
</tr>
</tbody>
</table>

| PH | 7.362 | 7.411 |
| PCO2 | 26.2 | 24.8 |
| PO2 | 85 | 67.9 |
| SBC | 17.4 | 18.8 |
| BE | -8.9 | -8.2 |
| SATS | 95.8 | 93.4 |

<table>
<thead>
<tr>
<th>AB GAS</th>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
<th>CONTROL</th>
<th>EXP</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>7.362</td>
<td>7.411</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCO2</td>
<td>26.2</td>
<td>24.8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PO2</td>
<td>85</td>
<td>67.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBC</td>
<td>17.4</td>
<td>18.8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BE</td>
<td>-8.9</td>
<td>-8.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SATS</td>
<td>95.8</td>
<td>93.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| INCREASED FIO2 10% preoxygenation |
| DECREASED FIO2 2% at 1 min. intervals |
| Fi O2 weaning complete |

**TIME TAKEN TO REGAIN BASELINE HEART RATE AND TRANSCUTANEOUS OXIMETER SATURATION.**

| HR: | CONTROL | 50 Sec | HR: | EXPERIMENT | 20 Sec |
| SATS: | CONTROL | 80 Sec | SATS: | EXPERIMENT | 35 Sec |

**KEY:** HR - heart rate, RESP - respiration, BP - blood pressure, SATS - transcutaneous oximeter saturation, AB GAS - arterial blood gas, FiO2 - fraction of inspired oxygen, RATE - ventilator breathe rate, PRESSURE - peak inspiratory pressure, PFC - persistent fetal circulation, IVH - intra-ventricular haemorrhage, HMD - hyaline membrane disease grade I - IV
# 4.3.11 RESULTS OF BABY 11

## VENTILATOR SETTINGS

<table>
<thead>
<tr>
<th></th>
<th>CONTROL</th>
<th>EXPERIMENTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO2</td>
<td>24%</td>
<td>24%</td>
</tr>
<tr>
<td>RATE</td>
<td>10</td>
<td>8</td>
</tr>
<tr>
<td>PRESSURE</td>
<td>18/5</td>
<td>18/5</td>
</tr>
</tbody>
</table>

### BABY: 11
- Diagnosis: HMD Gr I
- Age: DAY 1
- Gestation: 28 WEEKS

### BIRTH WEIGHT: 1130 g
- Tube Size: 2.5
- Cath Size: 6
- Length Passed: 8 cm
- Max Neg Pressure: 80 mmHg
- Drugs: Pen G, Amikin

### 10 MINUTES BEFORE | 5 MINUTES BEFORE | DURING 2nd ASPIRATION | ON COMPLETION | 5 MINUTES AFTER | 30 MINUTES AFTER
| HR | 154 | 149 | 151 | 146 | 93 | 122 | 173 | 171 | 154 | 155 | 155 | 148 |
| RESP | 57 | 54 | 59 | 55 | 69 | 66 | 66 | 63 | 58 | 55 | 56 | 55 |
| BP | 41/28 | 42/27 | 42/29 | 42/28 | 67/34 | 55/34 | 58/32 | 54/32 | 49/28 | 48/24 | 44/28 | 42/21 |
| SATS | 97 | 97 | 97 | 97 | 87 | 96 | 91 | 96 | 97 | 97 | 97 | 98 |

### AB GAS

| PH | 7.412 | 7.423 |
| PCO2 | 25.9 | 24.7 |
| PO2 | 78.5 | 77.9 |
| SBC | 19.5 | 19.6 |
| BE | -6.1 | -6 |
| SATS | 95.5 | 95.6 |

### TIME TAKEN TO REGAIN BASELINE HEART RATE AND TRANSCUTANEOUS OXIMETER SATURATION.

- HR: CONTROL 30 Sec  |  EXPERIMENT 15 Sec
- SATS: CONTROL 120 Sec  |  EXPERIMENT 75 Sec

**KEY:**
- HR - heart rate, RESP - respiration, BP - blood pressure, SATS - transcutaneous oximeter saturation, AB GAS - arterial blood gas
- FIO2 - fraction of inspired oxygen, RATE - ventilator breath rate, PRESSURE - peak inspiratory pressure
- PFC - persistent fetal circulation, IVH - intra-ventricular haemorrhage, HMD - hyaline membrane disease grade I - IV
### 4.3.12 RESULTS OF BABY 12

#### VENTILATOR SETTINGS

<table>
<thead>
<tr>
<th>Control</th>
<th>Experimental</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO2</td>
<td>40%</td>
</tr>
<tr>
<td>Rate</td>
<td>30</td>
</tr>
<tr>
<td>Pressure</td>
<td>22/5</td>
</tr>
</tbody>
</table>

#### BABY: 12

- **Diagnosis:** HMD Gr III
- **Age:** Day 1
- **Gestation:** 35 weeks

#### Birth Weight: 2320 g

#### Max Neg Pressure: 100 mmHg

#### Drugs: Pen G, Amikin

#### 10 MINUTES BEFORE

<table>
<thead>
<tr>
<th>HR</th>
<th>RESP</th>
<th>BP</th>
<th>SATS</th>
</tr>
</thead>
<tbody>
<tr>
<td>142</td>
<td>133</td>
<td>55/41</td>
<td>91</td>
</tr>
</tbody>
</table>

#### 5 MINUTES BEFORE

<table>
<thead>
<tr>
<th>HR</th>
<th>RESP</th>
<th>BP</th>
<th>SATS</th>
</tr>
</thead>
<tbody>
<tr>
<td>143</td>
<td>134</td>
<td>55/41</td>
<td>94</td>
</tr>
</tbody>
</table>

#### During 2nd Aspiration

<table>
<thead>
<tr>
<th>HR</th>
<th>RESP</th>
<th>BP</th>
<th>SATS</th>
</tr>
</thead>
<tbody>
<tr>
<td>95</td>
<td>118</td>
<td>54/43</td>
<td>94</td>
</tr>
</tbody>
</table>

#### On Completion

<table>
<thead>
<tr>
<th>HR</th>
<th>RESP</th>
<th>BP</th>
<th>SATS</th>
</tr>
</thead>
<tbody>
<tr>
<td>165</td>
<td>154</td>
<td>54/43</td>
<td>88</td>
</tr>
</tbody>
</table>

#### 5 Minutes After

<table>
<thead>
<tr>
<th>HR</th>
<th>RESP</th>
<th>BP</th>
<th>SATS</th>
</tr>
</thead>
<tbody>
<tr>
<td>145</td>
<td>143</td>
<td>54/43</td>
<td>85</td>
</tr>
</tbody>
</table>

#### 30 Minutes After

<table>
<thead>
<tr>
<th>HR</th>
<th>RESP</th>
<th>BP</th>
<th>SATS</th>
</tr>
</thead>
<tbody>
<tr>
<td>140</td>
<td>132</td>
<td>53</td>
<td>97</td>
</tr>
</tbody>
</table>

#### AB GAS

<table>
<thead>
<tr>
<th></th>
<th>PH</th>
<th>PCO2</th>
<th>PO2</th>
<th>SBC</th>
<th>BE</th>
<th>SATS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>7.391</td>
<td>26.9</td>
<td>50.9</td>
<td>18.9</td>
<td>-6.6</td>
<td>85.3</td>
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<td>EXP</td>
<td>7.384</td>
<td>27.3</td>
<td>50.9</td>
<td>18.7</td>
<td>-6.9</td>
<td>85</td>
</tr>
</tbody>
</table>

**Increased FIO2 10% preoxygenation**

**Decreased FIO2 2% at 1 min. intervals**

<table>
<thead>
<tr>
<th>FIO2</th>
<th>7.377</th>
<th>26.8</th>
<th>42.6</th>
<th>17.9</th>
<th>-7.7</th>
<th>76.9</th>
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</thead>
<tbody>
<tr>
<td>EXP</td>
<td>7.385</td>
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<td>48.8</td>
<td>18.9</td>
<td>-6.5</td>
<td>83.5</td>
</tr>
</tbody>
</table>

#### Fi O2 Weaning complete

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>EXP</th>
<th>Control</th>
<th>EXP</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>20 Sec</td>
<td></td>
<td>8 Sec</td>
<td></td>
</tr>
<tr>
<td>SATS</td>
<td>115 Sec</td>
<td></td>
<td>65 Sec</td>
<td></td>
</tr>
</tbody>
</table>

#### TIME TAKEN TO REGAIN BASELINE HEART RATE AND TRANSCUTANEOUS OXIMETER SATURATION.

- **HR:** CONTROL 20 Sec, EXPERIMENT 8 Sec
- **SATS:** CONTROL 115 Sec, EXPERIMENT 65 Sec

#### Key:
- HR - heart rate
- RESP - respiration
- BP - blood pressure
- SATS - transcutaneous oximeter saturation
- AB GAS - arterial blood gas
- FIO2 - fraction of inspired oxygen
- RATE - ventilator breathe rate
- PRESSURE - peak inspiratory pressure
- PFC - persistent fetal circulation
- IVH - intraventricular haemorrhage
- HMD - hyaline membrane disease grade I - IV
4.4 ANALYSIS OF RESULTS

4.4.1 Findings of biological data

* 12 babies were used in the study, 8 were male and 4 female.

* Range of gestation was between 26 and 35 weeks.

* All the babies had respiratory distress syndrome i.e. hyaline membrane disease Grade I - III, one had an intra-ventricular haemorrhage Grade III (no seizures), two had a pneumothorax, one had hyaline membrane disease Grade II compounded by persistent fetal circulation.

* Range of birth weight was between 940g and 2590gms.

* The age of the babies was between day 1 and day 4.

* Two babies were sedated and three received surfactant at least 24 hours previously.

4.4.2 Findings of physiological data

The following sequence occurs during suctioning of neonates:

The baby is disconnected from the ventilator and the suction catheter is introduced into the endotracheal tube, thereby partially or fully occluding endotracheal tube lumen. Then suctioning ensures.

The baby is temporarily not fully or adequately ventilated until suctioning routine is complete. As a result of the break in adequate ventilation, the baby starts to breathe irregularly or holds its breath and together with the lack of adequate oxygenation the heart rate drops.

There is a subsequent drop in transcutaneous oximeter saturation and arterial blood gas saturation and transient ischaemia occurs.

This sequence naturally increases the stress levels of the baby and there is a resultant increase in mean blood pressure and therefore an increased risk of intra-ventricular haemorrhage. (Avery, et al, 1994: 1121 - 1134; Wong 1993: 244 / 696 - 697).
4.4.2.1 The effect of preoxygenation on heart rate, transcutaneous oximeter saturation and mean blood pressure

The sequence described in 4.4.2 is depicted in figure 1 - where control and experimental heart rate, transcutaneous oximeter saturation and mean blood pressure is depicted during the various stages of suctioning. In the experimental phase there is a smaller drop in heart rate and transcutaneous oximeter saturation and a corresponding smaller increase in mean blood pressure as compared with control phase.

There proves to be a statistically significant interaction between control and experimental phases for heart rate (p < 0.01). The post-hoc comparison between control and experimental heart rate "during suctioning" is also significant (p < 0.01). See figure 1(a).
4.4.2.2 Box plot of heart rate

The box plot of heart rate shown in figure 2 during the various stages of suctioning shows more detail. The baseline heart rates observations for control and experimental phases are the same.

The drop in heart rate "during suctioning" is more pronounced in the control phase and the recovery after "completion of suctioning" is much higher than experimental. The higher heart rates are because there is compensation for the pronounced ischaemia that occurs during suctioning.

The heart rate of the experimental phase also returns to baseline observations faster than the control which maybe due to the fact that control heart rate results need more time to recover transcutaneous oximeter saturation lost during suctioning without preoxygenation.

The graph also shows variation during the whole procedure. The control heart rates show greater variance than the experimental heart rates. Furthermore, the babies, during the control phase, had a mean bradycardia down to 113 beats per minute as compared to the experimental phase of 132 beats per minute. This indicates babies are less stressed and more stable during the experimental phase.
4.4.2.3 Respiration during the various stages of suctioning

Figure 3 shows the control and experimental respiratory results. During the experimental phase there was no statistically significant difference between baseline and "5 minutes after" (p = 0.9) indicating that respiration had returned to baseline observations.

On the other hand, during the control phase there was a statistically significant difference between baseline and "5 minutes after" (p < 0.01) indicating that the respiration had not returned to baseline observations.

This shows that during the experimental phase the respiration of the babies treated with preoxygenation during suctioning recovered much faster than during the control phase.

It was found that the experimental respiration results were higher than the control respiration but this is due to the fact that the experimental suctioning occur 6 hrs after the control suctioning. The ventilator respiratory rates were found to be less during the experimental phase and as a consequence the babies were breathing more on their own during experimental phase.

\[
\begin{align*}
\text{mean control ventilator rates} &= 28 \\
\text{mean experimental ventilator rates} &= 23
\end{align*}
\]
4.4.2.4 Time saturation and time heart rate

Time saturation and time heart rate is the time taken to return to baseline heart rate and saturation (transcutaneous oximeter saturation) after suctioning is complete. In figure 4 time saturation is plotted as a function of time heart rate, both measured in seconds.

Figure 4 shows that for a specific time heart rate, in either the control or experimental phase, there is a specific corresponding time saturation i.e. the longer the time heart rate the longer time saturation.

Using an analysis of covariance procedure with heart rate as covariate we found a statistically significant difference between control and experimental time saturation, \( p < 0.01 \). In other words, the time saturation with time heart rate as covariate is statistically longer in the control phase than the experimental phase, where the time taken to return to baseline heart rate and transcutaneous oximeter saturation (TO2) is far less.

This is shown in figure 4 where the experimental results occupy the left lower area (less time taken to recover) whereas the control results occupy the upper and slightly more to the right area (more time taken to recover).
4.4.2.5 Time heart rate of each infant

Figure 5 shows the time heart rate (time taken to return to baseline heart rate after suctioning is complete) for each individual baby. Time heart rate for control phase ranged from 8 to 75 seconds. The mean time heart rate for control phase was 22 seconds. All the babies had shorter time heart rate during the experimental phase. Time heart rate for experimental phase ranged from 0 to 25 seconds. The mean time heart rate for experimental phase was 9 seconds.

4.4.2.6 Box - and whisker plot of time heart rate

In Figure 6 we see the variance and median between control and experimental phase. Time heart rate median for control phase = 20 seconds. Time heart rate median for experimental phase = 6 seconds. Using a two sample t-test for dependant samples, we find a highly significant difference between the mean time heart rate for the control and experimental phases (p < 0.01).
4.4.2.7 Time saturation of each infant

Time Saturation is the time taken to return to baseline transcutaneous oximeter saturation after suctioning is complete for each individual baby. This is shown in figure 7. Here there is also a difference depicted between control and experimental phases. Time saturation for control phase ranged from 35 to 140 seconds. The mean time saturation for control phase was 89 seconds. All the babies had shorter time saturation during experimental phase. Time saturation for experimental phase ranged from 20 to 90 seconds. The mean time saturation for experimental phase was 40 seconds.

![Figure 7: Time Saturation of each case](image)

4.4.2.8 Box-whisker plot of time saturation

In figure 8, a box and whisker plot for time saturation, shows the variance and median between control and experimental phases. Time saturation median for control phase = 94 seconds. Time saturation median for experimental phase = 32 seconds. The two sample t-test was also used for dependent samples. A significant difference (p<0.01) between the mean time saturation for the control and experimental phases was found.
4.4.2.9 Transcutaneous oximeter saturation

Control and experimental transcutaneous oximeter saturation drop during suctioning and recover again after completion of suctioning. Figure 9 shows that at "5 minutes after completion" the control results had yet to return to baseline meanwhile the experimental results had returned to baseline and were even marginally above baseline observations.

**Figure 9:** Control and Experimental Transcutaneous Oximeter Saturation during the various Stages of Suctioning

![Graph showing transcutaneous oximeter saturation during suctioning stages.](image)

**Figure 10:** Control and Experimental Arterial Blood gas Saturation

![Graph showing arterial blood gas saturation during suctioning stages.](image)

**Figure 11:** Control and Experimental Partial pressure of Oxygen

![Graph showing partial pressure of oxygen during suctioning stages.](image)
A post-hoc comparison between control and experimental shows that there is a significant difference between results at baseline and "on completion" for the control phase (p < 0.01) that is, the saturation had not returned to baseline. There is also a difference between results at baseline and "on completion" for the experimental phase but the difference is not significant (p = 0.80) that is, the saturation had almost returned to baseline observations.

This shows that saturation improves and recovers to baseline faster after suctioning with preoxygenation. The desaturation during suctioning is also more pronounced in the control phase. Mean desaturation for control phase was down to 85%, and the mean desaturation for the experimental phase was down to 91%.

4.4.2.10 Arterial blood gas

The arterial blood gas was taken "10 minutes prior to suctioning" and "30 minutes after suctioning". This is shown in figure 10.

The mean arterial blood gas saturation for the control phase dropped but not significantly from 92.6% to 92.4%, whereas the mean arterial blood gas saturation for the experimental phase increased but not significantly from 91.3% to 92.2%.

The arterial blood gas saturation is higher in the control phase than the experimental phase. This can be explained by the fact that the experimental results were obtained 6 hours later and by that time the FiO2 (concentration of inspired oxygen) had been weaned on 8 of the babies, 2 babies had the same FiO2 and 2 babies had a slightly higher FiO2 than during the control phase.

4.4.2.11 Partial pressure of oxygen

Figure 11 shows the control and experimental partial pressure of oxygen (PaO2) "10 minutes before suctioning" and "30 minutes after suctioning".

The mean PaO2 for the control phase stayed the same whereas the mean PaO2 for the experimental phase increased but not significantly from 67.4% to 72%.

The PaO2 for the control phase is higher than PaO2 for the experimental phase. This is explained as above, for Figure 10. The experimental results were obtained 6 hours later and by that time the FiO2 (concentration of inspired oxygen) had been weaned on 8 of the babies, 2 babies had the same FiO2 and 2 babies had a slightly higher FiO2 than during the control phase.
4.4.2.12. Mean blood pressure

There is a clear but not statistically significant increase in mean blood pressure during the control phase as compared with experimental phase. The mean blood pressure during the experimental phase recovers faster than the mean blood pressure during the control phase. This is shown in figure 12.

To compare the mean blood pressure between "10 minutes before suctioning" and "5 minutes after suctioning" a post-hoc comparison was used. The mean blood pressure for the control phase had not yet returned to baseline ($p > 0.05$), at a 5% level of significance, whereas the mean blood pressure for the experimental phase had almost returned to baseline values ($p = 0.9$). In fact at "30 minutes after suctioning" the mean blood pressure for the control phase had yet to return to baseline and the mean blood pressure for the experimental phase had dropped below baseline values.

Figure 12: Control and Experimental Mean Blood Pressure during the various Stages of Suctioning
This indicates that during the experimental phase the babies undergo less stress "during suctioning" with preoxygenation, return to baseline blood pressure faster and even may be more relaxed than prior to suctioning.

4.4.2.13 Heart rate, systolic and diastolic blood pressure during suctioning

Figure 13 is a three dimensional graph of control and experimental heart rate as a function of systolic and diastolic blood pressure during suctioning. This graph and table 4.4.2.13.1 indicates what happens to the heart rate as the systolic and diastolic changes, during suctioning.

Figure 13: 3D graph of Control and Experimental Heart Rate, Systolic and Diastolic Blood Pressure During Suctioning
4.4.2.13.1 Table of heart rate, systolic and diastolic blood pressure during suctioning

<table>
<thead>
<tr>
<th>RANGE</th>
<th>SYSTOLIC mm Hg</th>
<th>DIASTOLIC mm Hg</th>
<th>HEART RATE beats per minute</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONTROL</td>
<td>(55 - 78)</td>
<td>(38 - 46)</td>
<td>93 to 140</td>
</tr>
<tr>
<td>EXPERIMENTAL</td>
<td>(45 - 74)</td>
<td>(25 - 46)</td>
<td>118 to 152</td>
</tr>
<tr>
<td>MEAN</td>
<td>CONTROL</td>
<td>65</td>
<td>40</td>
</tr>
<tr>
<td>EXPERIMENTAL</td>
<td>58</td>
<td>38</td>
<td>132</td>
</tr>
</tbody>
</table>

The graph and the table indicate that the control systolic and diastolic blood pressure increases and that there is a corresponding bradycardia during suctioning.

The experimental systolic and diastolic blood pressure also increases during suctioning. However as can be seen the increase is not as high as during the control phase. There is also a corresponding bradycardia but to a lesser degree, during suctioning. This again shows that the babies tolerate the suctioning procedure far better when they are preoxygenated during suctioning.

4.5 SUMMARY

This chapter dealt with the description and analysis of results. Chapter 5 discusses the findings of these results and subsequent recommendations are made.
CHAPTER 5

DISCUSSION OF FINDINGS AND RECOMMENDATIONS

5.1 INTRODUCTION

The null hypothesis states that utilising preoxygenation by means of hyperoxygenation, does prevent transient hypoxaemia during endotracheal tube suctioning of ventilated neonates with respiratory distress syndrome. (see chapter 1.4.1 pg 12).

In this study the null hypothesis cannot be rejected or accepted completely. It has been shown that preoxygenation does reduce transient hypoxaemia to a significant degree but does not eliminate it altogether.

Therefore in this chapter the results are discussed and recommendations are made for nursing education, improvements in nursing practice and areas where further research in this field can be done.

5.2 DISCUSSION OF FINDINGS

From the analysis of the results it has been verified that preoxygenation by means of hyperoxygenation does lessen transient hypoxaemia to a significant degree.

There was significantly less severe bradycardia and markedly less transcutaneous oximeter desaturation and a corresponding smaller increase in mean blood pressure during suctioning.

Although there was no significant difference in the respiration the babies did recover their baseline respiration significantly faster with preoxygenation.

It was also statistically shown that there was significantly less time taken to recover baseline heart rate and transcutaneous oximeter saturation observations.

The mean arterial blood gas increased with the partial pressure of oxygen after suctioning but not significantly.

There was markedly faster recovery to baseline mean blood pressure observations. It was also found that as the blood pressure increased during suctioning there was a
corresponding bradycardia. This occurred to a much lesser degree during preoxygenation while suctioning.

There results show that babies are less stressed, more stable, and return to baseline observations faster when preoxygenated while suctioning, and therefore decrease transient hypoxaemia during suctioning and reduce the risk of intra-ventricular haemorrhages, and neurodevelopmental problems.

5.3 SHORTCOMINGS OF THE STUDY

* Only 12 cases were studied. The sampling was limited to the availability of babies at the time.

* The suction procedure was done once without preoxygenation and once 6 hours later with preoxygenation on the same baby.

5.4 RECOMMENDATIONS

5.4.1 Recommendations for nursing education

Education of nursing sisters is essential in ensuring safer suctioning procedures that lessens harmful effects such as transient hypoxaemia and sequelae thereof.

It is also important to keep up with educational international standards and the findings of latest research that suggest the following guidelines: (Which is discussed extensively in Chapter 2: literature survey.)

* Limit number of aspirations to a maximum of 3 - 5 passes.
* Limit duration of each aspiration to between 10 - 20 seconds.
* Correct catheter size to be used for corresponding endotracheal tube.
* Instillation of NaCl only when necessary.
* Gentle physiotherapy only when physiologically warranted.
* Ensure correct catheter length passed.
* Ensure correct suction pressure. Suction pressures > 20 KPa have been shown in the literature survey to be harmful to neonates.
* Allow for recovery of saturation after each aspiration.
5.4.2 Recommendations for nursing practice

Standardise all suction procedures throughout the country. Utilise preoxygenation in all ventilated babies increasing FiO2 (concentration of inspired oxygen) 5 - 10% as babies condition requires. Use the above criteria to standardise procedures.

5.4.3 Recommendations for further research

* This study only included 12 premature babies. It is recommended that a larger sample is used.

* The context of the sample was in a private clinic in Johannesburg. The context of the sample can be extended to other hospitals and cities.

* The suction procedure was done once without preoxygenation and once 6 hours later with preoxygenation on the same baby. The procedure could be repeated several times with a shorter interval time - span, between procedures.

* The control and experimental suction procedures could be done on different babies.

* Only premature babies with respiratory distress were used. A study could also include term babies who are also ventilated.

* There is a closed suction system being utilised in the United States of America. This system could be researched in a South African setting. Here oxygen is supplied throughout suctioning as the baby is not disconnected from the ventilator. It also has the added features of having markings on the catheter to ensure correct catheter length is passed during aspirations, and apparently decreases nosocomial infections.

* A study could be done to determine whether preoxygenation can occur safely and beneficially by increasing FiO2 (concentration of inspired oxygen) by 10% for longer than 5 minutes.

* A further study can determine whether preoxygenation by increasing FiO2 (concentration of inspired oxygen) more than 10% could be safe and beneficial for chronic or critically ill babies.
5.5 CONCLUSION

Suctioning of endotracheal tubes in premature infants with respiratory distress syndrome, who are undergoing mechanical ventilation is viewed as a necessary clinical practise to prevent tube obstruction.

Literature and research confirms that endotracheal tube suctioning results in transient hypoxaemia and demonstrates that this is reflected in the brain by vasodilatation and deoxygenation - predisposing to the genesis of intra-ventricular haemorrhages and neurodevelopmental problems.

Research and literature further suggests that these effects are preventable by preoxygenation before suctioning.

The purpose of this study was to determine whether utilising preoxygenation by means of hyperoxygenation, will prevent transient hypoxaemia, during endotracheal tube suctioning of ventilated neonates with respiratory distress syndrome.

It was found that preoxygenation by means of hyperoxygenation does lessen transient hypoxaemia to a significant degree.

There was significantly less severe bradycardia, markedly less transcutaneous oximeter desaturation and a corresponding smaller increase in mean blood pressure during suctioning. There was also significantly faster recovery to baseline heart rate, transcutaneous oximeter saturation, respiration, and a markedly faster recovery to baseline mean blood pressure observations. The mean arterial blood gas and partial pressure of oxygen also increased after suctioning, however not significantly.

These results show that babies are less stressed, more stable, and return to baseline observations faster, when preoxygenated while suctioning. This reduces transient hypoxaemia during suctioning and therefore the risks of intra-ventricular haemorrhages and neurodevelopmental problems.
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