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Factors contributing to endotracheal suctioning induced heart rate alterations in anesthetized newborn piglets

Gunderson, Laurie Porter, Ph.D.
The Ohio State University, 1989

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FACTORS CONTRIBUTING TO ENDOTRACHEAL SUCTIONING INDUCED HEART RATE ALTERATIONS IN ANESTHETIZED NEWBORN PIGLETS

DISSERTATION

Presented in Partial Fulfillment of the Requirements for the Degree Doctor of Philosophy in the Graduate School of the Ohio State University

By

Laurie Porter Gunderson, R.N., B.S.N., M.S.N.

* * * * *

The Ohio State University
1989

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1989
To My Parents
ACKNOWLEDGEMENTS

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# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>ACKNOWLEDGEMENTS</th>
<th>iii</th>
</tr>
</thead>
<tbody>
<tr>
<td>VITA</td>
<td>iv</td>
</tr>
<tr>
<td>LIST OF TABLES</td>
<td>xii</td>
</tr>
<tr>
<td>LIST OF FIGURES</td>
<td>xiii</td>
</tr>
<tr>
<td>CHAPTER</td>
<td>PAGE</td>
</tr>
<tr>
<td>I. FACTORS CONTRIBUTING TO ENDOTRACHEAL SUCTIONING INDUCED HEART RATE ALTERATIONS IN ANESTHETIZED NEWBORN PIGLETS</td>
<td>1</td>
</tr>
<tr>
<td>Introduction to the Problem</td>
<td>1</td>
</tr>
<tr>
<td>Purpose</td>
<td>3</td>
</tr>
<tr>
<td>Hypothesis</td>
<td>3</td>
</tr>
<tr>
<td>Research Questions</td>
<td>4</td>
</tr>
<tr>
<td>Definitions of Variables</td>
<td>4</td>
</tr>
<tr>
<td>Independent Variables</td>
<td>4</td>
</tr>
<tr>
<td>Dependent Variables</td>
<td>5</td>
</tr>
<tr>
<td>II. REVIEW OF THE LITERATURE</td>
<td>6</td>
</tr>
<tr>
<td>Introduction</td>
<td>6</td>
</tr>
<tr>
<td>The Effect of Endotracheal Suction on Oxygenation</td>
<td>6</td>
</tr>
<tr>
<td>Adult Studies</td>
<td>6</td>
</tr>
<tr>
<td>Infant Studies</td>
<td>10</td>
</tr>
<tr>
<td>Methods of Preventing Suctioning Hazards</td>
<td>15</td>
</tr>
<tr>
<td>Hyperoxygenation</td>
<td>15</td>
</tr>
<tr>
<td>Hyperinflation</td>
<td>15</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>16</td>
</tr>
<tr>
<td>Modified Endotracheal Suctioning Adaptors</td>
<td>23</td>
</tr>
<tr>
<td>Heart Rate</td>
<td>31</td>
</tr>
<tr>
<td>Mechanical Stimulation</td>
<td>33</td>
</tr>
<tr>
<td>Hemodynamics</td>
<td>34</td>
</tr>
<tr>
<td>Negative Pressure</td>
<td>36</td>
</tr>
<tr>
<td>Endotracheal Solutions</td>
<td>36</td>
</tr>
</tbody>
</table>

vii
TABLE OF CONTENTS (cont.)

II. REVIEW OF THE LITERATURE (cont.) PAGE

- Conceptual Framework ........................................ 39
- Transition to Extrauterine Life .............................. 39
- Respiratory Distress Syndrome ............................... 39
- Mechanical Ventilation ........................................ 41
- Endotracheal Suctioning ....................................... 42
- Hypoxemia ..................................................... 44
- Mechanical and/or Neural Factors ............................ 44

III. METHODOLOGY ................................................. 48

- Research Design ................................................ 48
- Randomization .................................................. 50
- Setting .......................................................... 51
- Subjects ........................................................ 52
- Protection of Animal Subjects ................................ 53
- Instrumentation ................................................ 53
- Data Collection Process ...................................... 53
- Data Gathering ................................................ 53
- Data Analysis .................................................. 61
- Law of Initial Value ......................................... 63
- Repeated-Measures Analysis of Variance ................. 64

IV. DATA ANALYSIS AND INTERPRETATION ........................ 65

- Introduction .................................................... 65
- Description of the Sample .................................... 65
- Characteristics of the Piglets ............................... 65
- Characteristics of Group I and Group II Piglets .......... 68
- Respiratory and Ventilator Data for All Piglets .......... 68
- The Effect of Endotracheal Suctioning on Arterial Blood Gases .......................... 70
- Oxygen .......................................................... 70
- Arterial Oxygen Values for Group I Pre-Drug ............. 70
- Arterial Oxygen Values for Group I Post Drug ............ 73
### TABLE OF CONTENTS (cont.)

#### IV. DATA ANALYSIS AND INTERPRETATION (cont.)

<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen Saturation</td>
<td>75</td>
</tr>
<tr>
<td>Arterial oxygen saturation for group I pre-drug</td>
<td>75</td>
</tr>
<tr>
<td>Arterial oxygen saturation for group I post drug</td>
<td>78</td>
</tr>
<tr>
<td>Arterial Hemoglobin Values</td>
<td>80</td>
</tr>
<tr>
<td>Arterial Hemoglobin Values for Group I Pre-Drug</td>
<td>80</td>
</tr>
<tr>
<td>Arterial Hemoglobin Values for Group I Post Drug</td>
<td>80</td>
</tr>
<tr>
<td>Oxygen</td>
<td>81</td>
</tr>
<tr>
<td>Arterial Oxygen Values for Group II Pre-Drug</td>
<td>81</td>
</tr>
<tr>
<td>Arterial Oxygen Values for Group II Post Drug</td>
<td>85</td>
</tr>
<tr>
<td>Oxygen Saturation</td>
<td>87</td>
</tr>
<tr>
<td>Arterial Oxygen Saturation for Group II Pre-Drug</td>
<td>87</td>
</tr>
<tr>
<td>Arterial Oxygen Saturation for Group II Post Drug</td>
<td>90</td>
</tr>
<tr>
<td>Arterial Hemoglobin Values</td>
<td>92</td>
</tr>
<tr>
<td>Arterial Hemoglobin Values for Group II Pre-Drug</td>
<td>92</td>
</tr>
<tr>
<td>Arterial Hemoglobin Values for Group II Post Drug</td>
<td>93</td>
</tr>
<tr>
<td>Carbon Dioxide</td>
<td>93</td>
</tr>
<tr>
<td>PaCO₂ for Group I Pre-Drug</td>
<td>93</td>
</tr>
<tr>
<td>PaCO₂ for Group I Post Drug</td>
<td>96</td>
</tr>
<tr>
<td>PH</td>
<td>98</td>
</tr>
<tr>
<td>Arterial pH Units for Group I Pre-Drug</td>
<td>98</td>
</tr>
<tr>
<td>Arterial pH Units for Group I Post Drug</td>
<td>100</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>103</td>
</tr>
<tr>
<td>Arterial Bicarbonate Values for Group I Pre-Drug</td>
<td>103</td>
</tr>
<tr>
<td>Arterial Bicarbonate Values for Group I Post Drug</td>
<td>105</td>
</tr>
<tr>
<td>Carbon Dioxide</td>
<td>107</td>
</tr>
<tr>
<td>PaCO₂ for Group II Pre-Drug</td>
<td>107</td>
</tr>
<tr>
<td>PaCO₂ for Group II Post Drug</td>
<td>109</td>
</tr>
<tr>
<td>IV. DATA ANALYSIS AND INTERPRETATION (cont.)</td>
<td></td>
</tr>
<tr>
<td>---------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>PH  ........................................... 112</td>
<td></td>
</tr>
<tr>
<td>Arterial pH Units for Group II  ............... 112</td>
<td></td>
</tr>
<tr>
<td>Pre-Drug ...................................... 112</td>
<td></td>
</tr>
<tr>
<td>Arterial pH Units for Group II  ............... 114</td>
<td></td>
</tr>
<tr>
<td>Post Drug ..................................... 114</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate .................................... 117</td>
<td></td>
</tr>
<tr>
<td>Arterial Bicarbonate Values for Group II Pre-Drug 117</td>
<td></td>
</tr>
<tr>
<td>Arterial Bicarbonate Values for Group II Post Drug 119</td>
<td></td>
</tr>
<tr>
<td>The Effect of Endotracheal Suctioning on Heart Rate 121</td>
<td></td>
</tr>
<tr>
<td>Heart Rate Values for Group I  ............... 121</td>
<td></td>
</tr>
<tr>
<td>Pre-Drug ...................................... 123</td>
<td></td>
</tr>
<tr>
<td>Heart Rate Values for Group I  ............... 125</td>
<td></td>
</tr>
<tr>
<td>Post Drug ..................................... 125</td>
<td></td>
</tr>
<tr>
<td>Heart Rate Values for Group II  ............... 128</td>
<td></td>
</tr>
<tr>
<td>Pre-Drug ...................................... 128</td>
<td></td>
</tr>
<tr>
<td>Heart Rate Values for Group II  ............... 131</td>
<td></td>
</tr>
<tr>
<td>Post Drug ..................................... 131</td>
<td></td>
</tr>
<tr>
<td>Discussion .................................... 135</td>
<td></td>
</tr>
<tr>
<td>V. SUMMARY, LIMITATIONS, IMPLICATIONS FOR NURSING PRACTICE, RECOMMENDATIONS FOR FURTHER STUDY . 141</td>
<td></td>
</tr>
<tr>
<td>Summary ....................................... 141</td>
<td></td>
</tr>
<tr>
<td>Limitations .................................... 143</td>
<td></td>
</tr>
<tr>
<td>Implications for Nursing Practice ............. 145</td>
<td></td>
</tr>
<tr>
<td>Recommendations for Further Study .......... 146</td>
<td></td>
</tr>
<tr>
<td>APPENDICES ..................................... 148</td>
<td></td>
</tr>
<tr>
<td>A. Computer Randomization Program ............ 148-150</td>
<td></td>
</tr>
<tr>
<td>B. Endotracheal Suctioning Method A .......... 151-152</td>
<td></td>
</tr>
<tr>
<td>C. Endotracheal Suctioning Method B .......... 153-154</td>
<td></td>
</tr>
<tr>
<td>D. Endotracheal Suctioning Method C .......... 155-156</td>
<td></td>
</tr>
<tr>
<td>E. Endotracheal Suctioning Method D .......... 157-159</td>
<td></td>
</tr>
</tbody>
</table>
TABLE OF CONTENTS (cont.)

APPENDICES (Cont.)

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>Institutional Laboratory and Animal Care and Use Committee Approval</td>
<td>160-162</td>
</tr>
<tr>
<td>G</td>
<td>Endotracheal Intubation of the Newborn Piglet</td>
<td>163-164</td>
</tr>
<tr>
<td>H</td>
<td>Carotid Artery Catheterization</td>
<td>165-167</td>
</tr>
<tr>
<td>I</td>
<td>Arterial Blood Gas Sampling from an Arterial Line</td>
<td>168-169</td>
</tr>
</tbody>
</table>

LIST OF REFERENCES ............................................. 170
LIST OF TABLES

<table>
<thead>
<tr>
<th>TABLE</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. General Characteristics, Resting Heart Rates, Respiratory Rates and Rectal Temperatures of the Sample</td>
<td>67</td>
</tr>
<tr>
<td>2. Mean Weight, Mean Heart Rate, Mean Respiratory Rate and Mean Rectal Temperatures of the Sample, Group I and Group II</td>
<td>68</td>
</tr>
<tr>
<td>3. Ventilator Settings For Twenty-Two Newborn Piglets</td>
<td>69</td>
</tr>
<tr>
<td>4. Time Lines for the Suctioning Protocols</td>
<td>122</td>
</tr>
</tbody>
</table>
## LIST OF FIGURES

<table>
<thead>
<tr>
<th>FIGURE</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Relationship of Factors Contributing to Endotracheal Suctioning Induced Heart Rate Alterations Before and After Administration of Atotate</td>
<td>47</td>
</tr>
<tr>
<td>2. Research Design</td>
<td>49</td>
</tr>
<tr>
<td>3. Time Lines for No Suction and Suction Protocols and Room Temperature and Body Temperature Injectate</td>
<td>58</td>
</tr>
<tr>
<td>4. Group I Pre-Drug PaO₂</td>
<td>72</td>
</tr>
<tr>
<td>5. Group I Post Drug PaO₂</td>
<td>74</td>
</tr>
<tr>
<td>6. Group I Pre-Drug Arterial Oxygen Saturation</td>
<td>77</td>
</tr>
<tr>
<td>7. Group I Post Drug Arterial Oxygen Saturation</td>
<td>79</td>
</tr>
<tr>
<td>8. Group II Pre-Drug PaO₂</td>
<td>83</td>
</tr>
<tr>
<td>9. Group II Post Drug PaO₂</td>
<td>86</td>
</tr>
<tr>
<td>10. Group II Pre-Drug Arterial Oxygen Saturation</td>
<td>89</td>
</tr>
<tr>
<td>11. Group II Post Drug Arterial Oxygen Saturation</td>
<td>91</td>
</tr>
<tr>
<td>12. Group I Pre-Drug PaCO₂</td>
<td>95</td>
</tr>
<tr>
<td>13. Group I Post Drug PaCO₂</td>
<td>97</td>
</tr>
<tr>
<td>14. Group I Pre-Drug pH</td>
<td>99</td>
</tr>
<tr>
<td>15. Group I Post Drug pH</td>
<td>102</td>
</tr>
<tr>
<td>16. Group I Pre-Drug HCO₃</td>
<td>104</td>
</tr>
<tr>
<td>17. Group I Post Drug HCO₃</td>
<td>106</td>
</tr>
<tr>
<td>Figure</td>
<td>Description</td>
</tr>
<tr>
<td>--------</td>
<td>--------------------------------------</td>
</tr>
<tr>
<td>18.</td>
<td>Group II Pre-Drug PaCO(_2)</td>
</tr>
<tr>
<td>19.</td>
<td>Group II Post Drug PaCO(_2)</td>
</tr>
<tr>
<td>20.</td>
<td>Group II Pre-Drug pH</td>
</tr>
<tr>
<td>21.</td>
<td>Group II Post Drug pH</td>
</tr>
<tr>
<td>22.</td>
<td>Group II Pre-Drug HCO(_3)</td>
</tr>
<tr>
<td>23.</td>
<td>Group II Post Drug HCO(_3)</td>
</tr>
<tr>
<td>24.</td>
<td>Group I Pre-Drug Heart Rate</td>
</tr>
<tr>
<td>25.</td>
<td>Group I Post Drug Heart Rate</td>
</tr>
<tr>
<td>26.</td>
<td>Group II Pre-Drug Heart Rate</td>
</tr>
<tr>
<td>27.</td>
<td>Group II Post Drug Heart Rate</td>
</tr>
</tbody>
</table>
CHAPTER I
FACTORS CONTRIBUTING TO ENDO TRACHEAL SUCTIONING INDUCED
HEART RATE ALTERATIONS IN ANESTHETIZED NEWBORN PIGLETS

Introduction to the Problem
Maintaining a patent airway is a critical factor in caring for the mechanically ventilated infant. Endotracheal suctioning, a procedure to remove mucus and secretions from the tracheobronchial tree of the premature infant, is frequently performed by the neonatal critical care nurse to maintain airway patency. However, the endotracheal suctioning procedure is not without complications. The hazards of endotracheal suctioning of premature infants include hypoxia (Poole, Abraham, & Fisk, 1979; Cabal, Devaskar, Siassi, Plajstek, Waffarn, Blanco & Hodgman, 1979; Simbruner, Coradello, Fodor, Havelec, Lubec & Pollack, 1981; Cunningham, Baun, & Nelson, 1984), bradycardia and other arrhythmias (Cordero & Hon, 1971; Cabal et al., 1979; Simbruner et al., 1981; Cunningham et al., 1984), atelectasis (Brandtater & Muallem, 1969), lung perforation (Anderson & Chandra, 1976), pneumothorax (Vaughn, Menke & Giacoia, 1978; Newman, Pomerance, & Brown, 1985), bacteremia (Storm, 1980), increased
intracranial pressure (Perlman & Volpe, 1983; Marshall, 1984; Perlman & Volpe, 1984; Trevathan, 1984; Volpe & Perlman, 1984), introduction of a foreign body, (Schreiner, Smith & Gresham, 1976), and mucosal necrosis, (Rasche & Kuhns, 1972).

Techniques to reduce the side effects associated with endotracheal suctioning include hyperoxygenation, hyperventilation and hyperinflation (Cabal, Siassi, Blanco, & Hodgman, 1974; Thibeault & Nelson, 1979; Thibeault & Gregory, 1986). High oxygen concentrations have been associated with retinopathy of prematurity (Phelps, 1987). Increasing the ventilatory rate and pressure may lead to barotrauma (Fox, Morray & Martin, 1987). Consequently the interventions to reduce the negative sequelae are associated with potentially detrimental consequences.

Modified endotracheal tube adapters were developed to permit the infant to remain connected to the ventilator during the suctioning procedure. Cabal et al., 1979; Zmora & Merritt, 1980; Gunderson, McPhee & Donovan, 1986, recommend the use of an adapter to allow the infant to receive continuous positive airway pressure (Cabal et al., 1979) and oxygen during the suctioning procedure. Use of an adapter during the endotracheal suctioning results in a reduction of the hypoxia, bradycardia and the duration of these negative events. However, none of these procedures
take into account the possible mechanisms of the bradycardic response.

Cabal et al., (1979) hypothesized that hypoxia leads to bradycardia. Yet, Gunderson et al., (1986) noted that there was no relationship between hypoxia and bradycardia during endotracheal suctioning of premature infants. During seven suctioning events (5 partially-ventilated and 2 non-ventilated) the infants became bradycardic without evidence of hypoxia. That is, the bradycardic episodes were characterized by an abrupt deceleration with rapid recovery (Gunderson, et al., 1986). Therefore, the bradycardia associated with endotracheal suctioning may not be directly related to the hypoxia. The question then becomes: what other variables in the endotracheal suctioning procedure may cause the heart rate alterations? The focus of this investigation was: To identify factors that contribute to the heart rate alterations during endotracheal suctioning.

**Purpose**

The purpose of this study was to explore the mechanical and neurogenic factors that may be associated with endotracheal suction induced heart rate alterations.

**Hypothesis**

The hypothesis that was tested in this study was: Heart rate alterations are initiated by the endotracheal
suctioning procedure and are due to mechanical and/or neural stimulation which is vagally mediated.

**Research Questions**

What factors contributed to endotracheal suctioning induced heart rate alterations in the anesthetized premature piglet? The following research questions were addressed:

1. What was the effect of suction catheter insertion into the endotracheal tube versus suction catheter insertion and the application of negative pressure on arterial blood gases?

2. What was the effect of room temperature (24.42 degree centigrade) normal saline versus body temperature (37 degree centigrade) on arterial blood gases?

3. What was the effect of suction catheter insertion into the endotracheal tube versus suction catheter insertion and the application of negative pressure on heart rate?

4. What was the effect of room temperature (24.42 degree centigrade) normal saline versus body temperature (37 degree centigrade) on heart rate?

**Definition of Variables**

**Independent Variables**

In this study the independent variables were components of the endotracheal suctioning procedure. The independent variables included: mechanical stimulation
which was the catheter insertion, the application of negative pressure and the temperature of the normal saline injectate solution.

**Dependent Variables**

The dependent variables were arterial partial pressure of oxygen (PaO₂), and heart rate. The partial pressure of oxygen in the blood was measured by arterial blood gases (ABG's). Heart rate was defined as the number of R waves per minute on the electrocardiogram (ECG).
CHAPTER II

REVIEW OF THE LITERATURE

Introduction

The organization of the review of the literature is based on the dependent and independent variables. The dependent variables are the physiologic manifestations associated with the risks of endotracheal suctioning. These variables deal with changes in oxygenation, changes in heart rate and changes in hemodynamics. The independent variables that may have an effect on the physiologic risks associated with endotracheal suctioning are: mechanical stimulation; negative pressure; and the temperature of the solutions instilled down the endotracheal tube.

The Effect of Endotracheal Suction on Oxygenation

Adult Studies

Berman and Stahl (1968) explored the use of a double lumen suction/oxygen delivery catheter. Subjects for this study were 12 males between the ages of 27 and 81 years and diagnosed with cardiopulmonary disease. The study took place during the immediate post operative period when a subject was breathing spontaneously. ABG samples were
drawn when the subjects were receiving room air and then with five liters per minute of supplemental oxygen. The room air oxygen tensions ranged from 45 to 85 millimeters of mercury (mmHg) and the oxygen tensions with supplemental oxygen ranged from 57 to 200 mmHg. Following the baseline oxygen tensions, each subject was then suctioned with 70 mmHg negative pressure using a double-lumen oxygen-suction catheter. The duration of the suctioning procedures ranged from 15 to 45 seconds. When no supplemental oxygen was delivered to the subjects, the PaO₂ fell one to 23 mmHg below the baseline room air values. When 5 liters per minute of supplemental oxygen was delivered through the double lumen suction catheter, the PaO₂ increased 7 to 90 mmHg above the PaO₂ obtained during endotracheal suctioning without any supplemental oxygen, and up to 68 mmHg above the baseline room air values. Endotracheal suctioning with supplemental oxygen was at least 30 seconds longer in duration than endotracheal suctioning without supplemental oxygen. The results support the premise that hypoxic episodes occur with endotracheal suctioning. The data also supports the fact that supplemental oxygen can reverse the hypoxic trend following the endotracheal suctioning procedure (Berman and Stahl, 1968).

Boutrous (1970) investigated the effect of endotracheal suctioning on 22 preoperative adult patients.
The results indicated that: (a) a decrease in $\text{PaO}_2$ occurred during periods of complete disconnection from the ventilator; (b) an even greater decrease in $\text{PaO}_2$ was incurred during suctioning; and (c) a period of hyperinflation following suctioning yielded a higher post-suction $\text{PaO}_2$. Boutrous (1970) inferred that prolonged suctioning and the lack of post-suctioning hyperinflation would result in significant and prolonged hypoxia.

Fell and Cheney (1971) conducted a two-fold investigation focusing on endotracheal suctioning. The first component of the study used dogs as an animal model. In the second component, the investigators applied the findings from the dog study to explore the effects of endotracheal suctioning in 26 adults with varying degrees of respiratory failure. Two groups were used to investigate several procedures to decrease hypoxia. Twenty subjects were exposed to hyperinflation with 100 percent oxygen for one minute prior to suctioning and eighteen subjects were exposed to 5 liters per minute of supplemental oxygen delivered via a side arm of the endotracheal tube during the suctioning procedure and no side arm flow during the suctioning procedure. There was a 30 minute period between the supplemental oxygen procedure and the procedure without supplemental oxygen.

With the human subjects the $\text{PaO}_2$, after hyperinflation with 100 percent oxygen, rose and then
declined after 15 seconds of suctioning. Only one of the 20 subjects had a PaO₂ less than 65 mmHg after 15 seconds of suction. The second group of 18 subjects with no hyperinflation had decreased PaO₂ values after 15 seconds of suctioning. The delivery of sidearm oxygen flow did not have any influence on the levels of PaO₂. Following 15 seconds of suction, seven had PaO₂ values less than 65 mmHg and a reported low of 38 mmHg. The investigators indicate that patients with respiratory failure should be hyperinflated with 100 percent oxygen for one minute prior to suctioning. If one minute of hyperinflation cannot be done, the investigators recommend 1 or 2 hyperinflations with 100 percent oxygen prior to suctioning. The insufflation with side arm oxygen during suctioning did not retard the fall in partial pressure in oxygen in humans, although it did retard the fall in PaO₂ in dogs. The results from the dog investigation are similar to the findings of Berman and Stahl (1968). Fell and Cheney (1971) hypothesized that significant shunts and atelectasis may have been present in the study subjects, therefore, this may have been one of the reasons for the variance in study findings.

Adlkofer and Powaser (1978) compared the effects of preoxygenation versus no preoxygenation prior to endotracheal suctioning. The sample was comprised of 64 adult patients. There was no descriptive data regarding
the diagnosis, ages and ventilator settings for the sample. In this non-randomized study, 54 subjects were not preoxygenated prior to the endotracheal suctioning procedure. The 54 subjects who were not preoxygenated were divided into 3 groups based on their decline in PaO₂. Subjects in group I had less than a 10 millimeter of mercury fall in PaO₂, subjects in group II had between a 10 to 20 millimeter of mercury fall in PaO₂ and subjects in group III had a fall of more than 20 mmHg in PaO₂. The remaining 10 subjects had their arterial oxygen elevated to 200-300 mmHg by hyperinflation with 100 percent oxygen prior to suctioning. Five of the 10 subjects demonstrated declines in PaO₂ below the control levels following endotracheal suctioning. There were considerable differences in the rate of decline in PaO₂ during endotracheal suctioning. The rate of decline in PaO₂ could not be attributed entirely to the duration of the suctioning procedure. The investigators recommended that preoxygenation should be carried out in all patients and that lung hyperinflation after endotracheal suctioning may be beneficial.

Infant Studies

Dangman, Hegyi, Hiatt, Indyk, and James (1976) used transcutaneous monitoring to explore changes in oxygenation associated with routine nursing care procedures for premature infants. During endotracheal
suctioning, the decrease in transcutaneous partial pressure of oxygen (tcPO$_2$) was as great as 40 mmHg and the time for recovery took up to seven minutes. The endotracheal suctioning events were also associated with bradycardia. The bradycardia was more transient in nature than the decreases in tcPO$_2$. The investigators did not specify the size or characteristics of the sample, nor did they define "transient" when they were referring to the bradycardic episodes.

Speidel (1978) investigated the use of an umbilical catheter with an oxygen electrode at its tip for monitoring premature infants arterial oxygen concentrations (SaO$_2$). In the three cases that were presented there was a marked, but unspecified, fall in SaO$_2$ when the infant was handled and manipulated. The fall in SaO$_2$ was compounded when manipulations occurred back to back without allowing the infant to recover. In one case one infant that was pharyngeally suctioned had an average fall in SaO$_2$ of 31 mmHg. The information presented in this work supports other studies that have found that suctioning is frequently associated with a decline in arterial oxygen. Vidyasagar and Asonye (1979) discussed their clinical experience with transcutaneous oxygen monitoring of forty infants with gestational ages ranging from 26-42 weeks. They noted that tcPO$_2$ monitoring provides useful trends in assessing the infants
physiologic reactions to routine procedures. One case of chest physiotherapy followed by endotracheal suctioning resulted in a 35 mmHg fall in tcPO$_2$. These observations support the fact that the endotracheal suctioning procedure is associated with decreases in tcPO$_2$.

Long, Philip and Lucey (1980) examined the impact that a variety of routine nursing procedures including positioning, blood pressure monitoring and endotracheal suctioning had on tcPO$_2$. Forty-five premature infants that were less than 36 weeks of gestation, had birth weights of less than 2,500 grams and no congenital anomalies were the subjects for this investigation. The infants were randomly assigned through the use of a table of random numbers to a control or one of two experimental groups. Both of the experimental groups used transcutaneous monitoring for data collection. The only difference in the two experimental groups was that the caretakers in one group (group III) were allowed to see the transcutaneous values whereas the other group (group II) were not. There was a statistically significant difference between group II and group III during endotracheal suctioning. Group II infants had a greater number of undesirable events that were longer in duration as compared to the events for group III. Group II infants also experienced a fall in tcPO$_2$ of greater than 20 torr as compared to group III. The investigators concluded
that transcutaneous monitoring is a valuable tool to use when caring for premature infants. They also noted that the number and duration of negative events associated with endotracheal suctioning can be minimized by altering the technique used for endotracheal suctioning based on the infant's tcPO$_2$ values.

Norris, Campbell and Brenkert (1982) explored the effects of 3 routine nursing procedures, including endotracheal suctioning, on tcPO$_2$. The subjects were 25 premature infants diagnosed with respiratory distress syndrome. The suctioning procedure called for disconnecting the infant from the ventilator, inserting the catheter and applying intermittent suction during catheter withdrawal. The infant was then reconnected to the ventilator and the above procedure was repeated. The tcPO$_2$ tracings indicated that the mean fall was 25.5 mmHg with a mean time for recovery of 265 seconds. The investigators postulated that the dramatic decline in tcPO$_2$ may be due to the interruption of the infants airway coupled with airway occlusion via the suction catheter.

Danford, Miske, Headley and Nelson (1983) evaluated the clinical application of transcutaneous monitoring of 40 infants with gestational ages between 24 and 38 weeks of age. The effects of ten different procedures, including chest physiotherapy followed by suctioning, were examined. Unfortunately the investigators did not specify
if the suctioning was performed on intubated or non-intubated infants. There was a significant fall in tcPO₂ immediately following the initiation of the chest physiotherapy followed by suctioning in 40-70 percent of the infants. The significant fall was reported to be a drop of 11 mmHg in greater than 90 percent of the infants with chest physiotherapy and suctioning. There was also a late rise of 13 mmHg in 90 percent of the infant's tcPO₂ following the completion of the intervention. The investigators postulate that the late rise in tcPO₂ may be related to the beneficial effects of the chest physiotherapy and suctioning procedure in and of itself on pulmonary function. However, the investigators failed to describe whether the suctioning was nasopharyngeal and/or endotracheal suctioning. Therefore, it was impossible to determine if the increase in tcPO₂ occurred following suctioning of the endotracheal tube.

Murdoch and Darlow (1984) investigated the effects of routine handling of premature infants. The investigators observed 5 infants over an extended period of time undergoing a variety of procedures. During this time frame the investigators observed 22 endotracheal suctioning events. Hypoxemia, as indicated by a fall in the tcPO₂, occurred in all of the 22 cases with endotracheal suctioning. Bradycardia and apnea also occurred in some of the cases, however the investigators
did not specify the exact number of bradycardic and/or apneic episodes. Based on their findings, Murdoch and Darlow (1984) recommend that endotracheal suctioning should not be performed on a set schedule. Instead, the timing of the suctioning event should be based on the infant's individual needs.

**Methods of Preventing Suctioning Hazards**

**Hyperoxegenation**

The complication of hypoxemia can be alleviated in the adult by using increased oxygen concentrations prior to or after endotracheal suctioning. However, the practice of increasing the fractional inspired oxygen concentration ($\text{FI}O_2$) prior to or following endotracheal suctioning in neonates remains controversial. Simbruner et al., (1981) does not recommend the use of increased $\text{FI}O_2$. On the other hand, Cunningham et al., (1984) recommended that further research needs to be done before incorporating this intervention into policies and procedures for endotracheal suctioning of the premature infant.

**Hyperinflation**

Brandstater and Muallem (1969) studied 6 newborn infants between the ages of 7 to 34 days. The gestational age for these infants was not provided. The investigators explored the consequences of endotracheal suctioning on lung stability. They found that following the suctioning
procedure, lung collapse occurred. In order to re-expand the lung, greater than normal pressures were required to reexpand the lung. In this study three deep breaths using pressures of 25 centimeters of water (cm H$_2$O) for a period of 2 seconds following the suctioning procedure helped to reexpand the collapsed lung. Brandstater and Muallem also noted that once the lung collapsed, it would remain collapsed, and re-expansion would occur only when high pressures or volumes were used. Therefore, the investigators recommended using a pressure of at least 25 cm H$_2$O following the endotracheal suctioning of infants to help reexpand the collapsed lung.

**Hyperventilation**

Raval, Yeh, Mora and Pildes (1980) conducted a non-randomized study exploring the effects of endotracheal suctioning on tcPO$_2$. The subjects were six premature infants with gestational ages of less than 37 weeks and one post term infant with a gestational age of 42 weeks. All three suctioning protocols included chest physiotherapy, suctioning of the endotracheal tube followed by oral and nasal suctioning. The first protocol involved suctioning the endotracheal tube followed by no hand ventilation. The second protocol called for suctioning the endotracheal tube, followed by hand ventilation using an FIO$_2$ 10 percent greater than the presuctioning parameters. The third protocol stipulated
that following endotracheal suctioning the infant would be ventilated by means of a manual resuscitation bag using 100 percent oxygen. The investigators did not describe how the different sequences for suctioning were determined and only 4 of the 7 infants were studied using the third protocol.

For all three protocols, the tcPO₂ was 84 ± 5.5 mmHg prior to beginning the chest physiotherapy. The tcPO₂ fell to a low of 63 ± 4.5 mmHg within 10 to 20 seconds after chest physiotherapy. Recovery to within baseline limits was 220 ± 21 seconds.

In the first suctioning protocol, subjects baseline tcPO₂ values were 75.4 ± 8 mmHg. The tcPO₂ declined significantly to 52.8 ± 4.8 mmHg following the completion of the suctioning protocol. The decline in tcPO₂ continued even after the endotracheal tube was reconnected to the ventilator. Recovery time for these infants ranged from 35 to 425 seconds. During recovery, two of the infants experienced bradycardic episodes, though the exact heart rate values were not specified.

The second suctioning protocol resulted in an immediate rise in the tcPO₂. However, the mean change in tcPO₂ was not significantly different from baseline. Immediately following the endotracheal suctioning procedure, the tcPO₂ decreased, though the exact value was not specified.
The third suctioning protocol resulted in hyperoxemia with tcPO\textsubscript{2} values greater than 150 mmHg. Overall, the tcPO\textsubscript{2} increased from 89.5 ± 6.5 mmHg to 98.5 ± 8.5 mmHg within 10 to 20 minutes following the completion of the endotracheal suctioning procedure.

When the three different protocols were compared there were no significant changes between the first and second protocols. The third protocol showed a significant improvement in oxygenation when compared to the other two protocols. Overall, for all of the protocols, endotracheal suctioning resulted in a 30 percent decrease in tcPO\textsubscript{2}. The use of hand ventilation following endotracheal suction resulted in a rapid recovery in tcPO\textsubscript{2}. The investigators recommended using 100 percent oxygen only when the infant was hypoxemic before the start of the suctioning procedure. When the baseline tcPO\textsubscript{2} was normal, the investigators recommend endotracheal suctioning and hand ventilation with a 10 percent increase in FIO\textsubscript{2} from baseline.

Simbruner, et al., (1981) explored the use of transcutaneous oxygen monitoring, heart rate monitoring, and blood pressure monitoring in 10 preterm infants undergoing endotracheal suctioning. The endotracheal suctioning procedure called for the complete disconnection of the endotracheal tube from the ventilator, suctioning the endotracheal tube followed by hand ventilation with a
manual resuscitation bag. The 10 subjects were divided into two groups. Group A had 5 infants weighing less than 1250 grams and Group B had 5 infants weighing more than 1750 grams. Infants were suctioned every two hours. The suctioning procedure required that the entire circuit was disconnected from the ventilator and 0.5 milliliters (mls) of normal saline was instilled into the endotracheal tube. The tube was then reconnected to the ventilator for several breaths. The endotracheal tube was once again disconnected from the ventilator and the suctioning procedure was performed. The duration of the suctioning procedure was not controlled. The negative pressure used for suctioning was 200 cm H₂O in all cases. Following completion of the suctioning, infants were ventilated with a manual resuscitation bag and supplemental oxygen. The investigators indicated that the tcPO₂ and heart rate parameters decreased while blood pressure increased. Five minutes after hand ventilation with a manual resuscitation bag was completed, the blood pressure and tcPO₂ values were higher than the values obtained during the presuctioning control period. The investigators concluded that endotracheal suctioning caused hypoxemia in 1 out of 2 occasions, even though the infant might have been well oxygenated prior to the suctioning procedure. In the study, hypoxemia was not reversed by hand ventilation with a manual resuscitation bag and the episodes of the
hypoxemia were up to 2 minutes in duration. The severity of the respiratory disease appeared to have been a major factor involved in the precipitous fall in tcPO\textsubscript{2}. In conclusion, Simbruner et al. (1981) recommend decreasing the overall time required to perform endotracheal suctioning and using some type of endotracheal tube adaptor that would allow the infant to remain connected to the ventilator during the suctioning procedure.

Norris, Campbell and Brenkert (1982) used a convenience sample of 25 premature infants to examine the effects of suctioning, repositioning and heel sticks on tcPO\textsubscript{2}. Suctioning, in this study, was a combination of endotracheal and/or nasopharyngeal. The endotracheal suctioning protocol stipulated disconnecting the endotracheal tube from the ventilator or continuous positive airway pressure (CPAP). The suction catheter was inserted and removed using intermittent negative pressure. The infant was then reconnected to the ventilator or CPAP and the entire procedure was repeated. The study found that the infants that had a higher pre-procedure tcPO\textsubscript{2} values sustained the largest decline following suctioning. The findings lead the investigators to hypothesize that the administration of supplemental oxygen prior to initiating the suctioning procedure may lead to a greater fall in tcPO\textsubscript{2}. This conjecture is interesting as pre-
oxygenation is often recommended prior to performing the endotracheal suctioning procedure.

Cunningham et al. (1984) investigated four different preoxygenation techniques and the subsequent effects on heart rate and oxygenation. Eight premature infants requiring mechanical ventilation served as subjects. Protocol A increased the FIO$_2$ by 10 percent for five breaths delivered over 15 seconds, protocol B increased the FIO$_2$ by 20 percent for five breaths delivered over 15 seconds, protocol C increased the FIO$_2$ by 10 percent for 10 breaths delivered over 30 seconds and protocol D increased the FIO$_2$ by 20 percent for 10 breaths delivered over 30 seconds. The suctioning procedure was performed by the primary investigator and consisted of two passes of the suction catheter and three inflation periods followed by a period of hand ventilation with a manual resuscitation bag. The negative pressure was 80-100 cm H$_2$O and was applied continuously for three seconds during withdrawal of the catheter.

The investigators divided the subjects into two groups for the analysis. Group I had three subjects with hyaline membrane disease and group II was comprised of the five remaining subjects. Group I subjects for all four protocols had a steady decline in tcPO$_2$ that was not immediately reversed with hand ventilation with a manual resuscitation bag. The downward trend in tcPO$_2$ continued
even after the subjects were reconnected to the ventilator. Overall, there was a significant (39 percent) decline in tcPO$_2$ for group I. The tcPO$_2$ fluctuated only slightly from baseline for protocols A and C. However, for protocols B and D, the tcPO$_2$ rose with the first inflation and remained elevated above baseline until the infants were reconnected to the ventilator. When the data for both groups was examined in its entirety, protocol A had more hypoxic episodes (tcPO$_2$ less than 40 mmHg) and bradycardic episodes (heart rate less than 120 beats per minute). The fewest bradycardic episodes were seen with protocol D. The investigators concluded that the type of preoxygenation technique depends on the infants underlying disease state. Further investigations are needed before recommending preoxygenating premature infants.

Overall, hypoxemia does occur with endotracheal suctioning. However, the proposed beneficial effect of supplemental oxygen during or after suctioning is less well established. The practice of hyperoxygenation has potential dangers for premature infants. Prolonged exposure to high oxygen concentrations in the blood can be damaging to some body tissues. The organ most vulnerable to the adverse effects of excessive oxygenation is the retina. Research has shown that high concentrations of oxygen can lead to retinal damage, more commonly referred to as retinopathy of prematurity (Phelps, 1987). Due to
the potential risk of retinopathy of prematurity, increasing the FIO₂ is not always a viable alternative for treating or preventing hypoxemic complications that have been associated with endotracheal suctioning.

**Modified Endotracheal Suctioning Adaptors**

Urban and Weitzner (1969) used a Rovenstine adaptor that permitted continuation of ventilation and oxygen delivery during the suctioning procedure in adults. Subjects for this study were seven males with a variety of medical and surgical diagnoses. The age range for these individuals was from 23-81 years of age. The data indicated that hypoxia can and does occur during endotracheal suctioning when the patient is disconnected from the ventilator. The ventilator flow was increased in an attempt to compensate for the flow through the suction catheter. In this investigation, suctioning via the adaptor prevented or alleviated the hypoxic trends. However, the negative aspect was that suctioning through the adaptor increased the duration of the suctioning procedure. The implications of this study were that a system which allows the patient to remain connected to the ventilator during the suctioning procedure has the potential benefit for preventing or reducing hypoxia.

Further literature searches revealed no other information of the Rovenstine adaptor. Since Urban and Weitzner's work, other investigators have studied several
different adaptors that allow for the individual to remain connected to the ventilator during the suctioning procedure.

Belling, Kelley and Simon (1978) compared the use of a swivel respirator adaptor to disconnecting the patient from the ventilator for endotracheal suctioning. The subjects for this study were 20 post-surgery cardiac patients with ages ranging from 40 to 60 years. Two of the 20 subjects required positive end expiratory pressure (PEEP). Each subject was randomly exposed to two protocols of endotracheal suctioning. One protocol disconnected the patient from the ventilator and one protocol used an adaptor to prevent disconnection from the ventilator during endotracheal suctioning. No preoxygenation techniques were employed in this study. The data revealed that there was an average decline of 24.6 percent in PaO₂ when the patient was suctioned via the swivel adaptor. When the subject was disconnected from the ventilator for the suctioning procedure, there was an average decline of 67.4 percent in PaO₂. Based on the data, the investigators recommend the use of an adaptor for endotracheal suctioning.

Bodai (1982) used a valve system which fits all commercially available respiratory circuit manifolds. The valve system allows for continuous ventilation of the patient during suctioning. The subjects were seven men
with ages ranging from 26-60 years of age and in severe respiratory failure. All subjects required PEEP ranging from 8 to 20 cm H\textsubscript{2}O. Subjects were randomly exposed to a suctioning protocol that called for disconnection from the ventilator, and to a suctioning protocol that used an in line adaptor. ABG's were drawn one minute prior to suctioning and at one and five minutes following the completion of the suctioning procedure. Both suctioning protocols stipulated three passes of the suction catheter. For the disconnection from the ventilator protocol vigorous bagging with 15 liters per minute of oxygen was done between each suction pass.

When the subjects were disconnected from the ventilator, the PaO\textsubscript{2} declined between 24-44 mmHg. At one minute the PaO\textsubscript{2} was still 15-35 mmHg below baseline. By five minutes the PaO\textsubscript{2} had returned to baseline parameters. During the suctioning protocol that required disconnection from the ventilator, five of the seven subjects became bradycardic.

The use of the adaptor for endotracheal suctioning procedure resulted in a decline of only 5-9 mmHg from the baseline PaO\textsubscript{2}. One minute following the completion of suctioning procedure, the decline ranged varied between 0-8 mmHg from the baseline PaO\textsubscript{2}. In all cases the PaO\textsubscript{2} was back to within baseline parameters by five minutes. Unlike the subjects that were disconnected from the
ventilator, none of the subjects suctioned through the adaptor experienced bradycardia.

In the study, the subjects with larger PEEP, had greater declines in PaO₂. The investigators hypothesized that suctioning via an adaptor helped to maintain the functional residual capacity by maintaining the PEEP resulting in better oxygenation. They went on to speculate that the hypoxemia that was seen when subjects were disconnected from the ventilator was the cause of the cardiac arrhythmias. The results indicated that the valve system modified the decline in PaO₂ and bradycardia associated with endotracheal suctioning. The valve, however, is not easily modifiable for use with the infant ventilator system.

Brown, Stansbury, Merrill, Linden and Light (1983) examined the use of an endotracheal tube adaptor. The subjects were 22 adult patients in acute respiratory failure that had underlying obstructive lung disease. The investigators randomly compared disconnecting the patient from the ventilator for endotracheal suctioning to a method that used an endotracheal tube adaptor. Four different protocols for suctioning were examined. Three of the methods involved disconnecting the patient from the ventilator, and one involved the use of an endotracheal tube adaptor. The disconnection protocols differed by the no presuction breaths versus presuction breaths delivered
to the patient. The results indicated that there was a significant desaturation with all three of the disconnection methods. The method of suctioning that used the endotracheal tube adaptor had the lowest desaturation level and the shortest recovery time. The investigators concluded that performing endotracheal suctioning via an adaptor is as good as, or better than methods of endotracheal suctioning than disconnection of the patient from the ventilator for suctioning.

Baker, Baker and Koen (1980) studied 3 different methods of endotracheal suctioning in 6 adult patients that required mechanical ventilation. All protocols required prehyperoxygenation with one hundred percent oxygen for one minute and one minute post hyperoxigenation with one hundred percent oxygen. The duration for the suctioning was 15 seconds in all cases. The first method of suctioning involved hyperoxygenating the patients with 100 percent oxygen via the ventilator prior to suctioning through the adaptor. The second method of suctioning involved hyperoxygenating the patient with 100 percent oxygen via a manual resuscitation bag prior to suctioning through the endotracheal tube adaptor. The final method of suctioning involved hyperoxygenating the patient with 100 percent oxygen via a manual resuscitation bag, disconnecting the patient from the ventilator and then suctioning. The investigators found that the first method
of suctioning was significantly better than the second method in increasing the PaO₂ (253 ± 22 mmHg versus 158 ± 26 mmHg). The investigators concluded the hyperoxygenating the patient via the ventilator was better than hyperoxygenating the patient with a manual resuscitation bag. They also noted that suctioning via the adaptor was better than disconnecting the patient from the ventilator.

The Novametrix C/D adaptor is a piece of equipment that can be used with either an adult or infant population. The adaptor was designed to fit on top of an endotracheal tube and replace the standard adaptor. The equipment has a side hole to permit access for endotracheal suctioning while still allowing the patient to be connected to the ventilator. Unfortunately, the Novametrix C/D adapter is not without fault. The adaptor must be used in place of the standard endotracheal tube adaptor. Though the adaptor is small, it is slightly longer than the standard adaptor. This added length can produce stress on the endotracheal tube, particularly rotational forces that tend to kink the soft pliable endotracheal tube. Kinking of the tubing leads to airway occlusion. The added length also increases the amount of respiratory dead space; thus complicating respiratory effort and gas exchange (Ganong, 1987). The increased
dead space can be physiologically detrimental to premature infants.

Jung and Newman (1982) investigated the Novametrix C/D adaptor. The sample for this study were eighteen adult patients admitted to the medical, surgical and respiratory intensive care units. The method of endotracheal suctioning was randomly determined so that the subject was disconnected from the ventilator and then suctioned or the subject was suctioned via the Novametrix C/D adaptor. There was a 5.55 percent decline in arterial oxygen saturation when the subjects were disconnected from the ventilator versus a 2.72 percent decline in arterial oxygen saturation when subjects were suction via an endotracheal tube adaptor (p < 0.01). When the investigators analyzed the data for only those subjects receiving PEEP there was a significant decline (p < 0.05) when the subjects were disconnected from the ventilator (12.6) versus suctioning via the adaptor (6.2) in arterial oxygen saturation. For all subjects the time to return to baseline was not significantly different, though it took slightly longer when the subjects were disconnected from the ventilator. The data indicated that the adaptor method of endotracheal suction is preferable to disconnection from the ventilator. The investigators noted that suctioning via the adaptor decreased the incidence of hypoxia and bradycardia.
Cabal et al. (1979) performed a randomized study on eight preterm infants being ventilated for respiratory distress syndrome. The investigators compared a method of suctioning that called for the disconnection of the infant from the ventilator and manual resuscitation bag to a method of endotracheal suctioning using the Novametrix C/D adaptor. The data indicated use of the Novametrix C/D adaptor minimized the negative sequelae of hypoxia and bradycardia. The negative events were significantly smaller in magnitude and shorter in duration.

Zmora and Merritt (1980) studied 13 ventilated newborn infants with a variety of pulmonary pathologies including: respiratory distress syndrome; pneumonia; asphyxia; and atelectasis following gastrointestinal surgery. Transcutaneous oxygen and heart rate data were evaluated during the suctioning procedure. The investigators compared the standard method of suctioning, disconnecting the infant from the ventilator, to the method of suctioning which used the Novametrix C/D adaptor. Although not a randomized study, the results supported previous findings that partially-ventilated suctioning, using an adaptor, prevented precipitous hypoxia.

Gunderson et al., (1986) compared the use of the Isothermal 3165 adaptor to the routine procedure of disconnecting the infant from the ventilator to perform
the endotracheal suctioning procedure. The Isothermal 3165 adapter does not replace the standard endotracheal tube adaptor, instead it replaces the ventilator circuit connecting piece that fits over the end of the endotracheal tube. Therefore, there is no increase in dead space. A port hole can be opened to allow for the passage of the suction catheter without disconnecting the infant from the ventilator. Eleven premature infants diagnosed with respiratory distress syndrome served as subjects. The ordering of the suctioning procedures was randomly determined by a sealed card system. The first method of suctioning was performed followed by the opposite method two hours later. The data from this study indicated that suctioning via the adaptor significantly decreased the incidence and severity of hypoxic events as measured by tcPO₂. Although not an intent of the study, the investigators noted that there was no relationship between the hypoxic events and bradycardia. The results of this investigation support the findings of Cabal et al. (1979) and Zmora and Merritt (1980). That is, a partially-ventilated method of endotracheal suctioning of premature infants reduces the associated hypoxia.

Heart Rate

Cabal et al. (1974) examined heart rate changes during and after endotracheal suctioning in 10 premature infants being ventilated for severe respiratory distress
syndrome. They found significant bradycardia (heart rate less than 100 beats per minute) in all patients, and periods of sinus arrest in five. The deleterious effect of endotracheal suctioning on heart rate could be ameliorated by presuction hand ventilation with a manual resuscitation bag for 30 seconds with an increased FIO$_2$. The investigators did not specify the ventilation rate, pressure of magnitude of the increase in FIO$_2$.

Cordero and Hon (1971) examined the relationship between nasopharyngeal suctioning and bradycardia. They speculated that the bradycardia observed, resulted from vagal stimulation. The authors went on to state that further investigation was needed to explore this relationship.

Simbrunner et al., (1981) investigated the effect of endotracheal suctioning on heart rate, blood pressure and tcPO$_2$. They found that heart rate and tcPO$_2$ fell, whereas blood pressure increased following the suctioning procedure. In some cases the blood pressure remained elevated for five minutes after the suctioning procedure. The investigators hypothesized that the bradycardic events were related to the hypoxemic episodes. Interestingly enough, they noted that the bradycardic changes occurred more frequently than the documented hypoxemic episodes as noted by the tcPO$_2$ recordings. Simbrunner et al., tried to explain this discrepancy in bradycardic events
hypothesizing a "dampened" tcPO2 values. However, the investigators did not define what they meant by dampened tcPO2 parameters.

Perlman and Volpe (1983) conducted an investigation examining the effects of endotracheal suctioning on cerebral blood flow, heart rate, blood pressure and oxygenation. During the suctioning procedure there were no significant changes in mean heart rate. Eleven out of the 35 subjects had marked decreases in tcPO2. The changes in tcPO2 were not associated with any changes in heart rate (Perlman & Volpe, 1983).

Cabal et al. (1979) noted a relationship between hypoxia and bradycardia. However, although not an intent of the investigation, Gunderson et al. (1986) reported no relationship between the hypoxia and bradycardic events. During 7 suctioning events, 5 partially-ventilated and 2 non-ventilated, the infants became bradycardic without becoming hypoxic. In 20 events, 4 partially-ventilated and 16 non-ventilated, the infants were hypoxic without becoming bradycardia. Gunderson et al. (1986) question whether there is a direct cause and effect relationship between hypoxia and bradycardia associated with the endotracheal suctioning procedure.

**Mechanical Stimulation**

Widdicombe (1963) investigated the physiologic response to chemical and mechanical irritation of the
lower respiratory tract. From his data he concluded that regardless of the type of stimulation, irritating the lower respiratory tract resulted in coughing. The investigator reported that chemical or mechanical irritation of the respiratory tract sometimes resulted in bradycardia. Though the bradycardic response was masked at times by the physiologic changes associated with coughing.

Naigow and Powaser (1977) Woodburne and Powaser (1980) conducted an investigation using animals as subjects. The purpose of their studies was to explore the effects of endotracheal suctioning on PaO₂. The investigators found that there was a significant and sustained fall in PaO₂ values following endotracheal suctioning. These declines were up to 5 minutes in duration. They hypothesized that the significant decline in PaO₂ as compared to baseline parameters may have been due to a reflex mechanism caused by the suction catheter stimulating the airways (Naigow & Powaser, 1977; Woodburne & Powaser, 1980). The mechanical stimulation of the airway initiated bronchoconstriction and intrapulmonary shunting resulting in sustained hypoxemia.

Hemodynamics

Simbruner et al. (1981) investigated the effects of endotracheal suction on oxygenation, heart rate and aortic blood pressure. Ten newborn infants diagnosed with
respiratory distress syndrome served as subjects for this study. The endotracheal suctioning procedure called for complete disconnection from the ventilator and the instillation of 0.5 mls of normal saline down the endotracheal tube followed by reconnection of the infant to the ventilator for several breaths prior to beginning the suctioning procedure. During endotracheal suctioning, oxygenation and heart rate decreased while blood pressure increased (Simbruner et al., 1981). Blood pressure remained elevated for up to 5 minutes in 8 of the 10 infants. One infant had a cardiac arrhythmia, though the type and duration was not specified.

Perlman and Volpe (1983) investigated the effects of endotracheal suctioning on changes in cerebral circulation, oxygenation, heart rate and blood pressure. Blood pressure readings were obtained from an umbilical or radial arterial line interfaced to a blood pressure transducer. In 34 out of 35 patients, blood pressure increased during the endotracheal suctioning event. The blood pressure values returned to baseline parameters following the suctioning procedure. The authors did not address the amount of time required to return to baseline blood pressure values following the endotracheal suctioning procedure.

The relationship between asphyxia and blood pressure changes in the pig was investigated by Dukes and Schwarte
(1931). When the pig's airway was occluded for 60 seconds, the blood pressure increased from 70 to 160 mmHg. Even after the airway was no longer occluded, blood pressure continued to rise for a short time to 190 mmHg. The investigators did not define a "short time". Length of time required to achieve pre-occlusion blood pressure values was not specified.

Based on these investigations, endotracheal suctioning appears to result in an increase in blood pressure. The changes in blood pressure associated with endotracheal suctioning have the potential for increasing the premature infant's chance of intracranial hemorrhage (Fanaroff & Martin, 1987).

**Negative Pressure**

Brandstater and Muallem (1969) examined several complications of endotracheal suctioning in paralyzed ventilated infants. The results supported the premise that the negative intrathoracic pressure during the suctioning procedure produced atelectasis. The investigators also noted that post-suctioning hyperinflation was found to return lung mechanics and values to baseline.

**Endotracheal Solutions**

Killingsworth, Slocombe, Alnoor, Robinson, and Derksen (1987) examined pulmonary dysfunction in 17 neonatal calves following the instillation of small
amounts of solution down the endotracheal tube. Data were gathered in relation to arterial oxygenation and pulmonary lung pressures. Twenty mls of room temperature normal saline (21 to 24 degrees Centigrade) was injected down the endotracheal tube resulting in hypoxia and pulmonary hypertension. The authors hypothesized that the solution stimulated irritant receptors located in the trachea and lung fields, triggering the hypertensive response. The hypertensive response indicated that the vagus nerve was responsible for the changes in pulmonary dynamics. However, the hypertensive response persisted following ligation of the vagus nerve. This finding lead Killingsworth et al. (1987) to speculate that there may be other mechanisms involved in triggering the hypoxemia and pulmonary hypertension. The findings of this study indicate that the vagus nerve is not entirely responsible for mediating changes within the pulmonary system. Further research needs to be conducted in this area.

Huber, Edmunds and Finley (1971) investigated unilateral pulmonary lavage with isotonic saline and the effect on pulmonary function and dynamics in ten anesthetized mongrel dogs. In this study, 500 mls of 0.9 percent isotonic saline were rapidly instilled into and aspirated from the left lung over a five minute time interval. The amount of fluid aspirated from the left lung ranged from 440 to 480 mls of liquid. Following the
lavage procedure the left lung was then ventilated. The investigators noted that the lavaged lung had altered alveolar structure, decreased surface activity at the alveolus, and altered pulmonary function. Therefore, this investigation found that instilling solution into the left lung resulted in an altered alveolus at the cellular level (Huber et al., 1971).

When both Huber et al., (1971) instilled 500 mls of solution into the lung field and Killingsworth et al., (1987) instilled 20 mls of an unspecified solution, they both noted alveolar changes. Fanaroff and Martin (1987) noted that in premature infants, impairment of the alveoli can lead to ventilation perfusion mismatching and atelectasis, which may in turn result in shunting and further compromise of the infants oxygenation status. Therefore, the use of solutions during endotracheal suctioning may further compromise the infant with pulmonary disease.

Francoz, Konopka, Sgroi, and Moser (1978) investigated pulmonary changes in anesthetized dogs following lavage with 1,850 mls of physiologic saline solution. They found that lavage resulted in decreased regional ventilation and perfusion. The ventilation and perfusion inequities resulted in hypoxemia. The duration of the ventilation and perfusion imbalance lasted up to six hours (Francoz et al., 1978).
Lee, Stoll and Downing (1977) investigated cardiorespiratory responses following stimulation of the laryngeal chemoreceptors in 62 piglets. The piglets were intubated and either water or milk were injected down the endotracheal tube. Injecting either type of solution resulted in apnea, bradycardia and hypertension. The authors noted that this response was more prominent in the very young piglets versus the older animals (age range 1 to 79 days).

**Conceptual Framework**

**Transition to Extrauterine Life**

During delivery, pressure on the thorax forces intra-alveolar fluid to be cleared from the mouth and nose. A marked negative intrathoracic pressure develops that assists in air being drawn into the lungs replacing the intra-alveolar fluid (Lough, Williams & Rawson, 1979; Moore, 1988). The remainder of the intra-alveolar fluid is removed via the pulmonary capillaries, arteries, veins and the lymphatic system (Moore, 1988). Once the initial breath is taken, the intrapleural pressure then becomes slightly negative to atmospheric pressure. The negative intrapleural pressure assists in normal respiration.

**Respiratory Distress Syndrome**

If infants are born before the 38th week of gestation, they frequently lack an adequate supply of surfactant in the terminal alveoli (Farrell & Perelman,
Pulmonary surfactant is secreted from the type II alveolar epithelial cells in the lungs. Surfactant is a lipoprotein comprised of phospholipids, highly saturated lecithins, lipids, phospholipids and other substances (Farrell & Perelman, 1987). The role of surfactant is to decrease the surface tension at the air-alveolar interface and to prevent the alveoli from collapsing. Without an adequate supply of surfactant, atelectasis occurs (West, 1985). The lack of pulmonary surfactant is associated with respiratory distress syndrome (RDS) or hyaline membrane disease as it is sometimes referred to (Martin & Fanaroff, 1987, p. 580).

Over 70 percent of the infants born between 28 to 30 weeks of gestation will develop respiratory distress syndrome (Martin & Fanaroff, 1987; p. 580). The diagnosis of respiratory distress syndrome is made from a combination of clinical, biochemical and roentgenologic features. The classical physiological alterations of respiratory distress syndrome include: (a) decreased lung volume; (b) increased respiratory rate; (c) increased physiologic dead space; (d) high pulmonary arterial pressure; (e) hypoperfusion of the lung; and (f) elevated partial pressure of carbon dioxide (PaCO₂) and decreased Ph (respiratory acidosis) (Martin & Fanaroff, 1987). In more severe cases of respiratory distress syndrome,
pulmonary edema leads to subsequent transudation of fluid into the lung space.

With the decreased surface area for gas exchange and increased work of breathing, infants with respiratory distress syndrome are frequently unable to adequately oxygenate. The inability to adequately exchange gases leads to a fall in arterial oxygen and an increase in arterial carbon dioxide. Medical management may initially involve supplemental oxygen delivered via a head hood. If the infant is still unable to compensate, support with intubation and positive pressure ventilation is often required (Aloan, 1987).

**Mechanical Ventilation**

When a premature infant requires mechanical ventilation, an uncuffed endotracheal tube must be inserted. The endotracheal tube provides an airway that allows for the infant to be connected to the ventilator.

Positive pressure ventilation attempts to maintain open alveolar sacs, thus promoting more surface area for gas exchange (Aloan, 1987). When positive pressure ventilation is used in conjunction with PEEP, the alveoli are not as likely to collapse. Maintaining adequate ventilation at the alveolar level enhances the infant's ability to compensate. Compensation is usually in the form of decreasing the amount of carbon dioxide and increasing the amount of oxygen available to the tissues.
Endotracheal Suctioning

Endotracheal intubation results in an open glottis, ineffective cough, and obstruction to normal mucociliary clearance. The internal lumen of the endotracheal tube frequently becomes narrowed or obstructed by the accumulation of secretions. Maintaining patency of the endotracheal tube is achieved by performing endotracheal suctioning, a nursing responsibility. Gregory (1972), Lough, Williams and Rawson (1979), Thibeault and Nelson (1979) and Thibeault (1986) recommend that endotracheal suctioning be performed every one to two hours depending on the type and amount of secretions. If secretions are not removed from the endotracheal tube the following complications may occur: increased airway resistance; atelectasis; decreased lung compliance; and decreased functional residual capacity (FRC). These complications can further compromise the premature infant's cardiopulmonary status (Cassini, 1984).

During endotracheal suctioning the catheter is inserted to just clear the tip of the endotracheal tube (Lough, Doershuk, Stern, 1985). Passing the suction catheter down the endotracheal tube reduces the internal diameter of the infant's airway. Depending on the size of the external diameter of the suction catheter, the airway may be reduced or occluded. In order to eliminate this problem Lough et al. (1985) recommend that:
The suction catheter should be sufficiently large to allow adequate removal of secretions, but not so large as to occlude the tube (and thus deflate the lung). In general, the catheter should be approximately half the diameter of the tube (p. 213).

Hodson and Truog (1983) recommend using: a 5 french suction catheter with a 2.5 millimeter (mm) internal diameter endotracheal tube; a 6 french suction catheter with a 3.0 mm internal diameter endotracheal tube; and a 8 french suction catheter with a 3.5 mm internal diameter endotracheal tube.

Negative pressure is not applied while the suction catheter is being inserted. Once the catheter has been inserted and pulled back 0.5-1 cm the negative pressure (suction) is then applied (Thibeault & Nelson, 1979; Thibeault, 1986; Aloan, 1987, p. 293). Aloan (1987) recommends using suction pressures that do not exceed 80 mmHg. Negative pressures greater than 100 mmHg may lead to tissue damage and atelectasis (Rosen & Hillard, 1960; Aloan, 1987). The negative pressure is applied continuously during catheter removal.

The suction catheter is quickly withdrawn using a rotating technique. Rotating the catheter attempts to remove secretions from all aspects of the internal wall of the endotracheal tube. The direct application of suction,
should not exceed 10 seconds (Thibeault, 1986). Thibeault (1986) recommends that if bradycardia occurs the duration of the suctioning procedure should be shorter than 10 seconds.

**Hypoxemia**

Hypoxemia is an important concept that has been addressed in both adult and neonatal investigations of endotracheal suctioning. Cassani (1984) defines hypoxemia as "a decrease in the content of oxygen in the arterial blood" (p. 9). Hypoxemia may be due to: (a) decreased oxygen; (b) alveolar hypoventilation; (c) decreased surface area at the alveolar level; (d) anatomic or intrapulmonary shunting that allows unoxygenated blood to enter the systemic circulation; and (e) perfusion inequities due to alveolar hypoventilation (Cassani, 1984).

**Mechanical and/or Neural Factors**

Nasopharyngeal suctioning often produces changes in cardiovascular and respiratory manifestations (Cordero & Hon, 1971). Investigators have hypothesized that cardiac arrhythmias may produced by vagal inhibition or stimulation resulting from reflexes triggered by stimulation of afferent vagal fibers in the pharynx, esophagus, or respiratory tract (Cordero & Hon, 1971). Cordero and Hon (1971) explained how inserting the catheter lead to alterations in heart rate as follows:
From the initial stimulation site, the impulse travels over the vagal network to the vagal medullary center and then to the heart, either by axon reflex in the vagal system without travelling centrally or by radiation of the impulse, which passes from its origin at the vagal nerve endings to a ganglion where it crosses to a branch of the sympathetic system and proceeds to the heart. The cardiac effector mechanism acts mainly as a depressor through the vagal fibers to the heart, but it also acts as a depressor through reciprocal activity of cardiac sympathetic accelerator nerves (p. 446).

Therefore, the bradycardic reflex can be triggered by an irritant, usually mechanical, such as inserting an endotracheal tube or passing a suction catheter. This response is due to the fact that vagal receptors are located throughout the airways. These receptors include irritant receptors, J-receptors and C-fibers (Paintal, 1973). These receptors may be stimulated by direct mechanical stimulation or via chemical factors (Widdicombe, 1981).

Hence the bradycardic response may be related to a variety of factors other than hypoxemia. The bradycardia may be due to the introduction of the suction catheter or stimulation of the receptors in the lung by the
instillation of solution down the endotracheal tube prior to initiating the suctioning procedure.

Figure 1 illustrates the relationship of mechanical stimulation, (the suction catheter insertion) and thermal stimulation, (the use of room temperature and body temperature normal saline), and the subsequent effect on heart rate and oxygenation prior to and following the administration of atofate. Heart rate alterations are initiated by the suctioning procedure and are due to mechanical and/or neural stimulation which is vagally mediated. The insertion of the suction catheter and the insertion of the suction catheter with the application of negative pressure will not result in dramatic heart rate alterations. The application of negative pressure will lead to alterations in PaO₂ due to the removal of gases from the lung field as compared to the no suction protocol. Following the administration of atofate, the heart rate alterations will be virtually eliminated and the changes in PaO₂ will be similar for both the no suction and suction protocols prior to and following the administration of atofate.

The endotracheal suctioning protocol that used body temperature normal saline would result in fewer heart rate alterations and less of a decline in PaO₂. Therefore, the room temperature endotracheal suctioning procedure would result in more heart rate alterations and a greater
decline in the PaO₂. Following the administration of atofate, the heart rate alterations for both the room temperature and body temperature injectate protocols would be virtually obliterated.

MECHANICAL STIMULATION    MECHANICAL/THERMAL STIMULATION
|                             |
CATHETER INSERTION          ROOM TEMPERATURE SOLUTION
APPLICATION OF SUCTION      BODY TEMPERATURE SOLUTION
VAGUS
OXYGENATION
&
HEART RATE ALTERATIONS
VAGUS
VAGAL BLOCKADE
APPLICATION OF SUCTION
CATHETER INSERTION
MECHANICAL STIMULATION

Figure 1. Relationship of factors contributing to endotracheal suctioning induced heart rate alterations before and after the administration of atofate.
CHAPTER III
METHODOLOGY

RESEARCH DESIGN

An experimental, repeated measures design was used to explore the physiologic effects of endotracheal suctioning in anesthetized neonatal swine. In this design the subjects were divided into two groups. Within the two groups, the piglets served as their own controls (Figure 2).

Data were collected on all of the piglets prior to the administration of atropine and following the administration of atropine. Atropine was selected because this drug produces vagal blockade. Therefore, the piglets heart rate alterations could be studied with and without vagal tone to determine if the heart rate alterations were due to hypoxia or vagal stimulation.
GROUP 1

\[0_1 X_A 0_2 0_3 X_A 0_4\]

ADMINISTRATION OF ATROPINE

GROUP 1

\[0_5 X_A 0_6 0_7 X_A 0_8\]

\(X_A\) REPRESENTS THE RANDOMLY PAIRED ORDERING OF THE FOLLOWING TWO SUCTIONING PROTOCOLS: (1) INSERTION OF THE SUCTION CATHETER AND REMOVAL; AND (2) THE INSERTION OF THE SUCTION CATHETER AND THE APPLICATION OF SUCTION DURING CATHETER REMOVAL.

GROUP 2

\[0_1 X_B 0_2 0_3 X_B 0_4\]

ADMINISTRATION OF ATROPINE

GROUP 2

\[0_5 X_B 0_6 0_7 X_B 0_8\]

\(X_B\) REPRESENTS THE RANDOMLY PAIRED ORDERING OF THE FOLLOWING TWO SUCTIONING PROTOCOLS: (1) THE INSTILLATION OF ROOM TEMPERATURE NORMAL SALINE; AND (2) THE INSTILLATION OF BODY TEMPERATURE (37°C) NORMAL SALINE.

Figure 2. Research Design
Randomization

The method of endotracheal suctioning was determined by a randomized computer program (Appendix A). Twenty-four piglets were randomly assigned to one of two groups. A piglet was randomly exposed to either the suctioning protocols for group I or group II (Figure 2). The suctioning protocols for group I included: (a) inserting the suction catheter into the endotracheal tube and removing the catheter (Appendix B); and (b) inserting the suction catheter into the endotracheal tube and continuously applying negative pressure during catheter removal (Appendix C). The suctioning protocols for group 2 included: (a) instilling room temperature (24.42 degrees centigrade) 0.9 percent normal saline into the endotracheal tube and then performing the suctioning procedure (Appendix D); and (b) instilling body temperature (37 degrees centigrade) 0.9 percent normal saline into the endotracheal tube and then performing the suctioning procedure (Appendix E). The suctioning protocols within each group were paired and the ordering of which suctioning protocol occurred first and which suctioning protocol occurred second was randomly determined by a computer program.

Each piglet was exposed to two different suctioning protocols followed by the administration of phenylephrine hydrochloride, 0.1 milligrams per kilogram (mg/kg) of body
weight to assure that the vagus nerve was intact. Two minutes after the administration of phenylephrine hydrochloride, the piglet was given atropine (atofate), 0.25-0.5 ml/kg of body weight to block the vagal tone.

Two minutes following the administration of atropine, a test dose of phenylephrine hydrochloride, 0.1 mg/kg of body weight was given to test for vagal blockade. Following the administration of atropine and phenylephrine hydrochloride, the two suctioning protocols were repeated in the same order.

The two randomized suctioning protocols per group comprised a study period. A study period was at least 30 minutes in duration. The 30 minute study period consisted of: (a) a 2 minute baseline data collection period; (b) the first suctioning protocol as determined by the randomization procedure; (c) a 3 minute post-suctioning data collection period; (d) a 15 minute "rest" period; (e) a 2 minute baseline data collection period; (f) the second suctioning protocol as determined by the randomization procedure; and (g) a 3 minute post-suctioning data collection period.

Setting

The setting for the study was the Biophysiological Laboratory located in the College of Nursing in a large, state affiliated university in the midwest. The barometric pressure on the twelve different days of data
collection ranged from 734 to 744 with a mean of 741 and a
standard deviation of 3.1 mmHg. The room temperature was
maintained at 24.42 degrees centigrade.

**Subjects**

The protocol was submitted to and approved by the
Ohio State Institutional and Laboratory Animal Care and
Use Committee and met with animal care and use guidelines
(Appendix F). The population for this study was newborn
swine. The subjects were twenty-four newborn Yorkshire
piglets.

The selection of an animal model was due to ethical
considerations. Manipulations such as the administration
of atropine, could not ethically be carried out in a human
neonatal population. The administration of atropine to
premature infants is known to cause tachycardia, decreased
secretions and hemodynamic alterations (Fanaroff & Martin,
1987). These changes could jeopardize the physiologic
status of premature infants, thus potentially increasing
the severity of their respiratory disease.

The piglet's pulmonary and cardiac systems are
similar to the human (Bustad, 1966; Dodds, 1982; Hill,
1986, p. 1157; Redding, Standaert, & Truog, 1986, p.
1187). Developmentally the piglet is physiologically more
advanced than the human neonate (Pond & Houpt, 1978).
These differences need to be taken into consideration when
addressing the generalizability of the findings.
Protection of Animal Subjects

The University Institutional Laboratory Animal Care and Use Committee mandates that procedures that may cause more than momentary or slight pain or distress to the animal must be performed with appropriate sedation, analgesia or anesthesia. Therefore, the piglets in this study were anesthetized with ketamine and xylazine 0.05 ml/kg subcutaneously and were sustained with additional doses as required to ablate palpebral and toe-pinch reflexes.

Instrumentation

The Gould 2600S strip chart recorder model 2007-8848-00 was used to gather data related to heart rate. The heart rate was obtained by using the Gould Electrocardiogram pre-amplifier.

Arterial blood gas (ABG) samples were analyzed by a calibrated Corning 158 semi-automated ABG analyzer. Corning has established that the accuracy and drift is less than 0.01 percent for a twenty-four hour period (Corning ABG Manual, 1982).

Data Collection Process

Data Gathering

Data collection took place between August 4, 1988 and September 1, 1988. Upon arrival at the Biophysics Laboratory the piglets were transferred to an isolette that had been warmed to an ambient air temperature of 35.5
to 36.5 degrees centigrade. The isolette air temperature was maintained in this neutral thermal zone (35.5 degrees centigrade to 36.5 degrees centigrade) by a servo controlled heater. The vital signs, including heart rate, respirations and rectal body temperature, were obtained for each piglet.

The resting heart rate and resting respiratory rate were calculated by placing the stethoscope directly on the piglet's chest wall and determining the heart rate by counting the number of beats in a sixty second time period. The normal heart rate for the newborn piglet is 180 ± 20 beats per minute (Macdonald, 1986; Pond & Houpt, 1978, p. 115). The resting respiratory rate was determined by counting the number of breaths in a sixty second time period. Neonatal piglet respirations have been reported to range between 15 and 20 breaths per minute (Watchko, Standaert, Badura & Woodrum, 1986, p. 1246).

The piglet's temperature was obtained by the use of a rectal thermometer kept in place for a minimum of 2 minutes. A rectal temperature of 38 degrees centigrade is reported as the average rectal temperature in piglets less than 24 hours of age (Pond & Houpt, 1978, p. 111). One piglet was removed from the isolette at random. The piglet was then assigned to either group I or group II according to the randomized computer program protocol
(Appendix A). Following randomization to one of the two groups, the piglet was then placed on a scale to determine its body weight in grams. Once the weight had been recorded, the piglet was positioned on its left side on warm (38°C) water packets. The warm water packs were resting on top of a surgical table. The warm packs were changed every 15-20 minutes in order to help maintain the piglet's core body temperature at 37-38 degrees centigrade (Pond & Houpt, 1978, p. 112). Immediately following placement on the table, the piglet was anesthetized with ketamine and xylazine 0.05 ml/kg and was sustained with additional doses as required to ablate palpebral and toe-pinch reflexes.

Electrocardiogram (ECG) electrodes were placed for monitoring heart rate and rhythm in lead II. Next, the piglet's length in centimeters was determined by measuring the snout to rump length. Once the piglet's length had been determined, the piglet was then placed on its back for the purpose of intubation (Appendix G). The size of the endotracheal tube was dependent upon the weight and length of the piglet. Generally, piglets that weighed 1200 grams or less were intubated with a 2.5 mm internal diameter endotracheal tube and piglets greater than 1200 grams were intubated with a 3.0 mm internal diameter endotracheal tube.
Following the placement of the endotracheal tube, ventilation was adjusted to maintain normal ABG values for neonatal piglets. The mean FiO₂ ranged between 0.30 and 1.00.

The right carotid artery was cannulated, following endotracheal intubation. A three centimeter incision was made in the piglet's neck over the carotid artery. The artery was isolated using blunt dissection technique. Once the carotid artery had been isolated a suture was placed around the proximal and distal aspects of the exposed vessel. The proximal suture was securely tied off. With tension being applied to both sutures a small nick was placed in the artery and with the aid of a vein introducer a 2.5 french umbilical line was threaded into the 5 centimeter mark. Once the catheter was in place the distal suture was securely tied. The proximal end suture was also used to secure the catheter to the vessel to prevent or minimize the risk of any inadvertent displacement of the catheter (Appendix H). The carotid artery catheter was used for ABG sampling and the administration of intravenous fluids and medications (Appendix I).

The piglet's glucose levels were maintained by 5 ml intravenous bolus infusion of 5 percent dextrose and water (D₅W). The first intravenous infusion was given immediately following the placement of the arterial line
and every thirty minutes until the piglet was euthanized.

The piglet was allowed to stabilize for 10-15 minutes following the placement of the endotracheal tube and carotid artery catheter prior to beginning the first suctioning protocol as determined by the randomization procedure.

The time line for the study for the no suction and suction protocols and the room temperature and body temperature protocols are illustrated in Figure 3. The room temperature and body temperature injectate protocols were 14 seconds longer in duration than the no suction and suction protocols due to the opening and closing of the adaptor, the injection of normal saline, and the waiting for 6 seconds.
Figure 3. Time line for suction and no suction protocols.
Prior to beginning the suctioning protocol, a two
minute graphic recording of heart rate was obtained. A
baseline arterial blood gas was drawn from the carotid
arterial line (Appendix I) 60 seconds prior to beginning
the suctioning protocol. Normal ABG parameters for a
newborn piglet breathing room air are: \( P_aO_2 > 60 \); \( P_aCO_2 < 50 \); and ph 7.35-7.45 (Redding et al., 1986, p. 1189,
Watchko et al., 1986, p. 1246).

Once the ABG had been obtained, suctioning of the
endotracheal tube was carried out 60 seconds following the
beginning of the arterial blood gas sampling procedure.
The exact protocols for the four methods of suctioning are
described in appendices B - E.

The negative pressure (suction) was set for 80 mmHg
(Fox et al., 1987). When negative pressure was indicated
in the protocol, the pressure was applied continuously
during the withdrawal of the suction catheter. In order
to determine the negative pressure, a mercury manometer
was used. In all cases, the the wall suction unit was
adjusted to measure 80 mmHg. Next, the tubing from the
wall unit suction apparatus was then connected to the top
of the Fischer-Porter rotometer. A 5/6 french suction was
attached to the bottom of the Fischer-Porter rotometer.
When the thumb port of the 5/6 french suction catheter was
occluded, the small ball in the Fisher-Porter rotometer to
the level of 4.8. The 4.8 value from the Fisher-Porter
rotometer was then plotted on the Fischer-Porter rotometer graph. The reading of 4.8 from the Fischer-Porter rotometer converted to a value of 4.8 liters of flow through the 5/6 french suction catheter per minute.

Twenty seconds following the completion of the suctioning procedure an ABG was drawn. ABG samples were also obtained at 60, 120 and 180 seconds following the completion of the suctioning procedure. A continuous graphic recording of the ECG was collected for three minutes following the completion of the suctioning procedure. A rest period of ten to fifteen minutes was provided to allow the piglet to return to within 10 percent of baseline heart rate parameters prior to initiating the second suctioning protocol as indicated by the randomization procedure.

Once the two different suctioning protocols had been completed, the piglet was given a bolus of phenylephrine hydrochloride 0.1 mg/kg of body weight to ensure that the vagus nerve was intact. Following the administration of the drug, the piglet's heart rate declined. Two minutes following the administration of the phenylephrine hydrochloride the piglet was given an intravenous bolus of atropine 0.05 ml/kg of body weight through the carotid arterial line to block the vagal tone. Two minutes following the administration of the atropine, the piglet was given a repeat dose of the phenylephrine hydrochloride
to ensure that the vagus nerve was blocked. Blockade of the vagus nerve was indicated by a lack of decline in heart rate as seen with the first dose of phenylephrine hydrochloride.

The piglet was allowed to stabilize for 10 minutes following the administration of the last dose of phenylephrine hydrochloride. After the stabilization period, the two different suctioning protocols were repeated in the same manner as described for the first study period. The piglet was also allowed to stabilize for 10 minutes between the two different suctioning protocols that comprised the study period.

Following the completion of the piglet's second study period, it was euthanized by injection through the carotid arterial line with 200 mg/kg of sodium pentobarbital as needed to obliterate heart rate. Death was determined when the piglet's heart was no longer beating. The lack of heart rate was determined by monitoring the ECG in lead II. If the piglet did not demonstrate a decline in heart rate within thirty seconds a repeat dose of sodium pentobarbital was administered.

Data Analysis

Data were gathered on the dependent variables in relation to the endotracheal suctioning events. Data included: (a) heart rate; and (b) ABG's.
The heart rate data was obtained from the graphic recordings. The ABG data was obtained from the five ABG samples for each suctioning procedure.

The graphic recordings of the heart rate for the two minutes prior to the suctioning procedure were converted to numerical values. The conversion process entailed counting the number of R waves occurring in a 20 second interval and then multiplying by three. The procedure was carried out for the two minutes just prior to the suctioning procedure, thus yielding six heart rate values prior to the suctioning event. Heart rate values were calculated by counting the number of R waves that coincided with the various aspects of the suctioning procedure. Following the suctioning procedure heart rate values were calculated, in the same manner as described for the pre-suctioning heart rate baseline, for a twenty second period. Therefore there were a total of nine post-suctioning heart rate values. These numerical values for heart rate were entered into the computer for data analysis.

The exact values for each of the five ABG samples were entered into the computer for analysis. Demographic data for the twenty-four piglets was entered into the computer for statistical analysis.
Law of Initial Value

When dealing with physiologic data the law of initial value (LIV) must be taken into consideration. Wilder (1957) defined LIV as follows:

Not only the intensity but also the direction of a response of a body function to any agent depend to a large degree on the initial level of that function at the start of the experiment. The higher this "initial level", the smaller is the response to function-raising, the greater is the response to function-depressing agents. At more extreme initial levels there is a progressive tendency to "no response" and to "paradoxic reactions", i.e., a reversal of the usual direction of the response. This rule holds true for 75-85 per cent of all experiments (p. 73).

Therefore a piglet's response to a treatment of procedure could have been affected by the piglet's unique pattern of physiologic function or alterations due to numerous factors (Benjamin, 1963). These factors must be taken into consideration when attempting to interpret the data from physiologic research.

One method for dealing with the problem of LIV is to statistically control for the problems inherent in the law of initial value. The covariance model is the best way for controlling for the problems associated with LIV.
(Benjamin 1963 & 1967). The analysis of covariance procedure (ANCOVA), according to Benjamin (1963), allows for the "...analysis of variance of a variable of primary interest (difference scores) after that primary variable has been made independent of an undesirable variable (initial level)" (p. 562). However, in this study the initial differences and unique patterns of physiologic response were not widely divergent from one another. Therefore, the aspect of using an analysis of covariance was not carried out.

Repeated-Measures Analysis of Variance

Repeated-measures analysis of variance (ANOVA) was used to analyze the data. Glantz (1987) recommends this approach when each experimental subject receives more than one treatment. When the overall F statistic was significant for the repeated-measures ANOVA a Duncan's post hoc test was employed to determine what values were statistically different from one another.
CHAPTER IV
DATA ANALYSIS AND INTERPRETATION

Introduction

The chapter includes a presentation and analysis of the data according to the research questions: (a) description of the sample; (b) data and analysis of the effect of suction catheter insertion into the endotracheal tube versus suction catheter insertion and the application of negative pressure on arterial blood gases; (b) data and analysis of the effect of room temperature (24.42 degree centigrade) normal saline versus body temperature (37 degrees centigrade) on arterial blood gases; (c) data and analysis of the effect of suction catheter insertion into the endotracheal tube versus suction catheter insertion and the application of negative pressure on heart rate; (d) data and analysis of the effect of room temperature (24.42 degree centigrade) normal saline versus body temperature (37 degrees centigrade) on heart rate.

Description of the Sample

Characteristics of the Piglets

The sample consisted of twenty-four newborn yorkshire piglets. General characteristics of the piglets studied are shown in Table 1. Two of the piglets were eliminated
from analysis due to congenital anomalies. The study population was comprised of 7 males and 15 females. All of the piglets were less than 24 hours of age. The investigator was unable to determine the precise age in hours due to the vague or nonexistent information regarding the exact time of delivery for each of the piglets.
**TABLE 1**

General Characteristics, Resting Heart Rates, Respiratory Rates and Rectal Temperatures of the Sample

<table>
<thead>
<tr>
<th>LITTER/IN</th>
<th>WEIGHT</th>
<th>LENGTH</th>
<th>HEART RATE</th>
<th>RESPIRATORY RATE</th>
<th>RECTAL TEMPERATURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1ST/2 F</td>
<td>1588</td>
<td>42 CM</td>
<td>144</td>
<td>40</td>
<td>38.4</td>
</tr>
<tr>
<td>1ST/1 M</td>
<td>1134</td>
<td>35 CM</td>
<td>120</td>
<td>36</td>
<td>38.4</td>
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<td>2ND/1 F</td>
<td>907</td>
<td>32 CM</td>
<td>162</td>
<td>56</td>
<td>37.0</td>
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<tr>
<td>2ND/2 F</td>
<td>907</td>
<td>32 CM</td>
<td>136</td>
<td>44</td>
<td>37.0</td>
</tr>
<tr>
<td>3RD/2 F</td>
<td>1021</td>
<td>34 CM</td>
<td>108</td>
<td>22</td>
<td>37.8</td>
</tr>
<tr>
<td>3RD/1 F</td>
<td>1474</td>
<td>39 CM</td>
<td>126</td>
<td>32</td>
<td>37.0</td>
</tr>
<tr>
<td>4TH/ ELIMINATED DUE TO CONGENITAL ANOMALIES</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4TH/1 M</td>
<td>794</td>
<td>30 CM</td>
<td>120</td>
<td>28</td>
<td>37.0</td>
</tr>
<tr>
<td>5TH/2 F</td>
<td>1588</td>
<td>33 CM</td>
<td>120</td>
<td>36</td>
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<tr>
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<td>1701</td>
<td>38 CM</td>
<td>126</td>
<td>38</td>
<td>37.4</td>
</tr>
<tr>
<td>6TH/1 F</td>
<td>1474</td>
<td>39 CM</td>
<td>146</td>
<td>40</td>
<td>37.8</td>
</tr>
<tr>
<td>6TH/2 M</td>
<td>1134</td>
<td>35 CM</td>
<td>136</td>
<td>36</td>
<td>37.8</td>
</tr>
<tr>
<td>7TH/2 F</td>
<td>964</td>
<td>34 CM</td>
<td>122</td>
<td>26</td>
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<tr>
<td>7TH/2 M</td>
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<td>36.6</td>
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<td>1474</td>
<td>42 CM</td>
<td>136</td>
<td>32</td>
<td>37.0</td>
</tr>
<tr>
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<td>40 CM</td>
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<td>32</td>
<td>37.0</td>
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<td>1134</td>
<td>37 CM</td>
<td>136</td>
<td>24</td>
<td>37.0</td>
</tr>
<tr>
<td>10TH/2 F</td>
<td>1134</td>
<td>38 CM</td>
<td>116</td>
<td>26</td>
<td>37.0</td>
</tr>
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<td>22</td>
<td>36.0</td>
</tr>
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<td>36 CM</td>
<td>118</td>
<td>26</td>
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<tr>
<td>11TH/ ELIMINATED DUE TO CONGENITAL ANOMALIES</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>12TH/2 M</td>
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<td>34 CM</td>
<td>116</td>
<td>28</td>
<td>37.0</td>
</tr>
<tr>
<td>12TH/1 F</td>
<td>1474</td>
<td>37 CM</td>
<td>130</td>
<td>32</td>
<td>37.0</td>
</tr>
</tbody>
</table>

**GROUP:**
1 = THE NO SUCTION/SUCTION PROTOCOLS  
2 = THE ROOM TEMPERATURE/BODY TEMPERATURE PROTOCOLS

**SEX:**
M = MALE  
F = FEMALE

**LENGTH:**
CM = CENTIMETERS
Characteristics of group I and group II piglets.

The mean values and standard deviations for the weight, length, heart rate, and rectal temperature per group are presented in Table 2. When the demographic variables for the 11 piglets in group I and the 11 piglets in group II were compared, the mean values were similar.

**TABLE 2**

Mean Weight, Mean Heart Rate, Mean Respiratory Rate, and Mean Rectal Temperature of the Sample, Group I and Group II

<table>
<thead>
<tr>
<th></th>
<th>WEIGHT</th>
<th>LENGTH</th>
<th>HEART RATE</th>
<th>RESPIRATORY RATE</th>
<th>RECTAL TEMPERATURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAMPLE MEAN:</td>
<td>1205/</td>
<td>36.4/</td>
<td>129/</td>
<td>32 ± /</td>
<td>37.1/</td>
</tr>
<tr>
<td>SD:</td>
<td>± 249</td>
<td>± 3.2</td>
<td>11.9</td>
<td>± 3.2</td>
<td>± 0.7</td>
</tr>
<tr>
<td>GROUP I MEAN:</td>
<td>1247/</td>
<td>36.0/</td>
<td>130/</td>
<td>32 ± /</td>
<td>37.2/</td>
</tr>
<tr>
<td>SD:</td>
<td>± 266</td>
<td>± 3.11</td>
<td>± 13</td>
<td>± 3.1</td>
<td>± 0.6</td>
</tr>
<tr>
<td>GROUP II MEAN:</td>
<td>1162/</td>
<td>36.3/</td>
<td>127/</td>
<td>31 ± /</td>
<td>36.3/</td>
</tr>
<tr>
<td>SD:</td>
<td>± 255</td>
<td>± 3.3</td>
<td>± 12</td>
<td>± 3.3</td>
<td>± 0.6</td>
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</tbody>
</table>

Respiratory and Ventilator Data for all Piglets

The size of endotracheal tubes (ETT) used and the ventilator settings with the FIO₂ are listed in Table 3. Ventilator settings and/or oxygen concentrations fluctuated in five of the piglets that were studied. Ventilator settings were adjusted to maintain arterial blood gases (ABG's) within normal limits during the baseline time periods.
<table>
<thead>
<tr>
<th>PIGLET</th>
<th>METHOD OF SUCTION</th>
<th>SIZE OF ETT</th>
<th>FIO₂</th>
<th>RATE</th>
<th>VENTILATOR SETTINGS</th>
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<td>1</td>
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<td>6</td>
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<tr>
<td>2</td>
<td>B, A, B, A</td>
<td>2.5</td>
<td>0.35</td>
<td>6</td>
<td>4/0</td>
</tr>
<tr>
<td>3</td>
<td>B</td>
<td>2.5</td>
<td>0.47</td>
<td>9</td>
<td>4/0</td>
</tr>
<tr>
<td></td>
<td>A</td>
<td></td>
<td>0.47</td>
<td>8</td>
<td>4/0</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td></td>
<td>0.47</td>
<td>6</td>
<td>4/0</td>
</tr>
<tr>
<td></td>
<td>A</td>
<td></td>
<td>0.60</td>
<td>6</td>
<td>4/0</td>
</tr>
<tr>
<td>4</td>
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<td>0.32</td>
<td>6</td>
<td>4/0</td>
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<tr>
<td>5</td>
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<td>0.34</td>
<td>8</td>
<td>4/0</td>
</tr>
<tr>
<td>6</td>
<td>B, A, B, A</td>
<td>2.5</td>
<td>0.34</td>
<td>4</td>
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<tr>
<td>7</td>
<td>OMITTED DUE TO CONGENITAL ANOMALIES</td>
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<td></td>
</tr>
<tr>
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<td>20</td>
<td>4/0</td>
</tr>
<tr>
<td>9</td>
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<td>0.32</td>
<td>10</td>
<td>4/0</td>
</tr>
<tr>
<td>10</td>
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<td>0.32</td>
<td>10</td>
<td>4/0</td>
</tr>
<tr>
<td>11</td>
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<td>0.30</td>
<td>8</td>
<td>4/0</td>
</tr>
<tr>
<td>12</td>
<td>C</td>
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<td>0.35</td>
<td>8</td>
<td>4/0</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td></td>
<td>0.60</td>
<td>8</td>
<td>4/0</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td></td>
<td>0.70</td>
<td>20</td>
<td>4/0</td>
</tr>
<tr>
<td></td>
<td>D</td>
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<td>1.00</td>
<td>20</td>
<td>4/0</td>
</tr>
<tr>
<td>13</td>
<td>D, C</td>
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<td>0.60</td>
<td>8</td>
<td>4/0</td>
</tr>
<tr>
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<td>4/0</td>
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<td>10</td>
<td>4/0</td>
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<td>0.40</td>
<td>10</td>
<td>4/0</td>
</tr>
<tr>
<td></td>
<td>A, B</td>
<td></td>
<td>0.35</td>
<td>10</td>
<td>4/0</td>
</tr>
<tr>
<td>16</td>
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<td>1.00</td>
<td>10</td>
<td>4/0</td>
</tr>
<tr>
<td>17</td>
<td>D, C</td>
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<td>0.58</td>
<td>10</td>
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</tr>
<tr>
<td></td>
<td>D, C</td>
<td></td>
<td>0.47</td>
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<td>4/0</td>
</tr>
<tr>
<td>18</td>
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<td>0.38</td>
<td>10</td>
<td>4/0</td>
</tr>
<tr>
<td>19</td>
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<td>0.33</td>
<td>12</td>
<td>4/0</td>
</tr>
<tr>
<td>20</td>
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<td>0.60</td>
<td>10</td>
<td>4/0</td>
</tr>
<tr>
<td>21</td>
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<td>0.60</td>
<td>12</td>
<td>4/0</td>
</tr>
<tr>
<td>22</td>
<td>OMITTED DUE TO CONGENITAL ANOMALIES</td>
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<td></td>
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<tr>
<td>23</td>
<td>D, C, D, C</td>
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<td>0.40</td>
<td>10</td>
<td>4/0</td>
</tr>
<tr>
<td>24</td>
<td>B, A, B, A</td>
<td>3.0</td>
<td>0.72</td>
<td>8</td>
<td>4/0</td>
</tr>
</tbody>
</table>

A = NO SUCTION PROTOCOL
B = SUCTION PROTOCOL
C = ROOM TEMPERATURE INJECTATE PROTOCOL
D = BODY TEMPERATURE INJECTATE PROTOCOL
The Effect of Endotracheal Suctioning on Arterial Blood Gases

Five arterial blood gases were withdrawn. One at baseline, one minute prior to commencing the suctioning procedure, and at 20, 60, 120 and 180 seconds following the suctioning procedure.

The components of the ABG that were analyzed included: partial pressure of oxygen (PaO₂), arterial oxygen saturation (O₂ sat), hemoglobin (Hb), partial pressure of carbon dioxide (PaCO₂), pH, and bicarbonate (HCO₃⁻).

Oxygen

Arterial oxygen values for group I pre-drug

The two different protocols examined in group I were suction and no suction. The mean PaO₂ for the no suction protocols was 150 millimeters of mercury (mmHg), and 159 mmHg for the no suction protocol at baseline, which was not statistically different.

When the pattern of mean PaO₂ values for the no suction and suction protocols were compared over time there was no significant difference between the two protocols (p = 0.1293), as illustrated in Figure 4. There was a significant decline in PaO₂ from baseline at 20 seconds post suctioning in both the no suction and suction protocols. The degree of decline was greater for the suction protocol (41 mmHg) as compared to the no suction
protocol (13 mmHg). The mean PaO₂ values for both protocols returned to within baseline parameters at one minute.
Group I Pre-Drug

No Suction & Suction

Arterial Oxygen (PaO2) mmHg

* = p < 0.05

Figure 4. Group I pre-drug PaO2.
Arterial oxygen values for group I post drug

The mean baseline PaO2 values in the suction and no suction protocols were not statistically different following the administration of atropine. The mean PaO2 value for the no suction protocols was 130 mmHg and the mean PaO2 value for the suction protocol was 139 mmHg.

When the mean PaO2 values for both the suction and no suction protocols were compared over time following the administration of atropine there was no significant difference, (p = 0.2765), as illustrated in Figure 5. Even though there was no significant difference in the two protocols over time, clinically the 20 second post suctioning mean PaO2 values were significantly lower for the suction protocol (37 mmHg) as compared to the no suction protocol (2 mmHg). The 2 mmHg decline in mean PaO2 was not significantly different from the remaining four values for the no suction protocol. However, the 37 mmHg decline for the suction protocol was significantly different from baseline and the remaining three PaO2 values at 60, 120 and 180 seconds.
Group I Post Drug

No Suction & Suction

Arterial Oxygen (PaO₂) mmHg

* = p < 0.05

Figure 5. Group I post drug PaO₂.
Examining the change from baseline to the 20 second post suctioning PaO₂ value, there was a clinically significant difference in the two suction protocols prior to the administration of atropine. The difference between the two protocols was due to the application of pressure and the removal of gases from the airway. The decline in mean PaO₂ for the no suction protocol was most likely due to partial obstruction of the airway with the suction catheter.

Following the administration of atropine, the same pattern of change for the mean PaO₂. The administration of atropine did not alter the response in the decline in PaO₂ at the 20 seconds. The 2 mmHg decline in mean PaO₂ for the no suction protocol was most likely due to partial occlusion of the airway with the suction catheter for the suction protocol. The partial occlusion of the airway in conjunction with the application of negative pressure (suction), the removal of gases from the lungs, and the decrease in compliance leads to a greater decline in mean PaO₂ both prior to and following the administration of atropine.

Oxygen Saturation

Arterial oxygen saturation for group I pre-drug.

The mean O₂ sat for the no suction and suction protocols were not significantly different at baseline.
The mean $O_2$ sat for the no suction protocol was 97.4% and the mean $O_2$ sat for the suction protocol was 97.5%.

The comparison of the mean $O_2$ sat values for the no suction and suction protocols revealed no significant difference, (\(p = 0.6583\)), as illustrated in Figure 6. Clinically the mean $O_2$ sat values were not different for the no suction protocol. At the 20 second post suctioning the mean $O_2$ sat value fell 1.7% below baseline. By the one minute sampling the mean $O_2$ sat values returned to within baseline parameters. In comparison to the profound changes in $PaO_2$ described above with no drug, $O_2$ sat is not a sensitive indicator of changes in $PaO_2$ due to the properties of oxyhemoglobin dissociation curve.
Group I Pre-Drug

No Suction & Suction

Percent Arterial Oxygen Saturation

Figure 6. Group I pre-drug arterial oxygen saturation.
Arterial oxygen saturation for group I post drug.

The mean $O_2$ sat for the no suction and suction protocols were not significantly different at baseline following the administration of atropine. The mean $O_2$ sat value for the no suction protocol was 96.8% and the mean $O_2$ sat value for the suction protocol was 97.1%.

The comparison of the mean $O_2$ sat values for the no suction and suction protocols following the administration of atropine over time revealed no significant difference ($p = 0.7145$), as illustrated in Figure 7. There was no significant difference in any of the five mean $O_2$ sat values for the no suction protocol. However, for the suction protocol the mean $O_2$ sat fell at 20 seconds post suctioning to 94.4%. At one minute, the mean $O_2$ sat returned to within baseline parameters.

The change in the mean $O_2$ sat was not significant for the no suction and suction protocols prior to the administration of atropine. Following the administration of atropine there was a significant difference in the 20 second post suctioning $O_2$ sat. The decrease was most likely related to the application of negative pressure and the removal of gases from the lung fields.
Figure 7. Group I post drug arterial oxygen saturation.
Arterial Hemoglobin Values

Arterial hemoglobin values for group I pre-drug.

The mean Hb values for the no suction and suction protocols were not significantly different at baseline. The mean Hb value for the no suction protocol was 8.9 grams per decaliter (gm/dl) and the mean Hb value for the suction protocol was 8.8 gm/dl. The comparison of the mean Hb values for the no suction and suction protocols over time revealed no significant different, (p = 0.6968).

Arterial hemoglobin values for group I post drug.

The mean Hb values for the no suction and suction protocols were not significantly different at baseline following the administration of atropine. The mean Hb value for the no suction protocol was 9.5 gm/dl and the mean Hb value for the suction protocol was 9.6 gm/dl. The comparison of the mean Hb values for the no suction and suction protocols over time following the administration of atropine were not significantly different, (p = 0.4690). There was no change in the mean Hb values for the no suction protocols at the 20 second post suctioning ABG sampling following the administration of atropine. By three minutes the mean Hb value rose for the no suction protocol and declined for the suction protocol. The decline could be attributed to the amount of blood being drawn. Likewise, the increase in mean Hb values could be attributed to the quantity of blood being
drawn and the possibility of dehydration. The decreased blood volume, seen with dehydration could lead to the hemoglobin becoming more concentrated in the samples, thus interfering with the calculated values. If this assumption is plausible, the piglets could have been dehydrated even though replacement fluids of five cubic centimeters of 5% dextrose in water was given every one half hour to maintain adequate hydration.

A second reason for the changes in hemoglobin values could have been due to instrumentation. The Co-oximeter used to determine the arterial hemoglobin values was designed and calibrated for human blood. The Corning company and the Corning Manual (1982) stipulated that if other than human blood is to be analyzed, the values may or may not be accurate and should be used for detecting trends only. Therefore, the variations may be due to instrumentation rather than actual changes.

Oxygen

Arterial oxygen values for group II pre-drug

In group II the effect of room temperature versus body temperature injectate for the suctioning protocols was examined. At baseline the mean PaO\textsubscript{2} value for the room temperature protocol was 157 mmHg versus 124 mmHg for the body temperature protocol.

The mean PaO\textsubscript{2} values for the room temperature and body temperature injectate protocol over time were
significantly different, \( p = 0.0004 \), as illustrated in Figure 8. The Duncan's post hoc analysis revealed that the mean \( \text{PaO}_2 \) at 20 seconds (95 mmHg) was significantly different from mean baseline \( \text{PaO}_2 \) (157 mmHg) for room temperature injectate. The three remaining mean \( \text{PaO}_2 \) values for the room temperature protocol were also significantly lower than baseline.
Group II Pre-Drug

Room Temperature & Body Temperature

Arterial Oxygen (Pao2) mmHg

* = p < 0.05

Figure 8. Group II pre-drug Pao2.
The Duncan's post hoc analysis for the body temperature protocol revealed that the post 20, 120 and 180 second mean PaO\textsubscript{2} values were significantly different from the mean baseline value of 124 mmHg, as illustrated in Figure 6. At 20 second after the completion of the endotracheal suctioning protocol, the mean PaO\textsubscript{2} fell to 101 mmHg. However, the one minute post suctioning mean PaO\textsubscript{2} value was not significantly different from baseline. At two minutes, the mean PaO\textsubscript{2} values rose significantly above baseline to 149 mmHg and then fell to 142 mmHg by the three minute sampling which was within baseline parameters.

When the room temperature and body temperature protocols were compared using a Duncan's post hoc analysis, the mean baseline values were statistically different (157 mmHg versus 124 mmHg). At the 20 second post suctioning ABG the mean PaO\textsubscript{2} for the room temperature injectate protocol was 95 mmHg. Whereas the mean PaO\textsubscript{2} for the body temperature injectate protocol was 101 mmHg. Both protocols were statistically lower than their respective baseline values. The comparisons for the one, two, and three minute mean PaO\textsubscript{2} values revealed that they were not significantly different from one another. However, the room temperature protocol remained significantly lower than baseline and the body temperature
protocol was higher than baseline as illustrated in Figure 8.

**Arterial oxygen values for group II post drug**

The room temperature and body temperature protocols were not significantly different from each other when the mean PaO₂ values were compared at baseline following the administration of atropine. The mean PaO₂ value for the room temperature protocol was 142 mmHg and the mean PaO₂ value for the body temperature protocol was 128 mmHg.

The comparison of the mean PaO₂ values over time for the room temperature and body temperature protocols following the administration of atropine revealed no significant difference (p = 0.4937) as illustrated in Figure 9.
Group II Post Drug

Room Temperature & Body Temperature

Arterial Oxygen (PaO2) mmHg

* = p < 0.05

Figure 9. Group II post drug PaO2.
In summary, even though the baseline values for the two protocols were significantly different from each other prior to the administration of atropine, the degree of change is still very important. The post 20 second PaO$_2$ value fell 39 percent from baseline to a value of 95 mmHg. There was only a 19 percent change in PaO$_2$ for the body temperature injectate protocol. One may argue that the greater degree of change for the room temperature protocol was due to the higher baseline value.

The changes in PaO$_2$ following the administration of atropine were not as dramatic as the changes seen prior to the administration of the drug. The PaO$_2$ declined 54 mmHg for the room temperature protocol and 38 mmHg for the body temperature protocol. One possibility for the smaller change could have been due to the baseline values being less discrepant (142 and 128 mmHg). However, there was still a greater decline for the room temperature protocol as compared to the body temperature protocol.

Oxygen Saturation

*Arterial oxygen saturation for group II pre-drug.*

The mean O$_2$ sat for the room temperature and body temperature injectate protocols were not significantly different at baseline. The mean O$_2$ sat value for the room temperature injectate was 97.0% and the mean O$_2$ sat for the body temperature injectate was 96.5%.
The comparison of the mean $O_2$ sat values for the room temperature and body temperature injectate protocols were not significantly different ($p = 0.4270$), over time as illustrated in Figure 10.

However, clinically there was a marked decline in the mean $O_2$ sat for the room temperature and body temperature injectate protocols. The 20 second post suctioning mean $O_2$ sat declined 6.0% from 97.0% to 91.0% for the room temperature protocol with the values not returning to baseline by three minutes. There was only a 4.0% decline in mean $O_2$ sat from 96.5% to 92.5% for the body temperature injectate protocol with return to baseline by one minute.
Group II Pre-Drug

Room Temperature & Body Temperature

Percent Arterial Oxygen Saturation

* = p < 0.05

Figure 10. Group II pre-drug arterial oxygen saturation.
Arterial oxygen saturation for group II post drug.

The mean O$_2$ sat for the room temperature and body temperature injectate protocols were not significantly different at baseline following the administration of atropine. The mean O$_2$ sat for the room temperature injectate was 96.8% and the mean O$_2$ sat for the body temperature injectate was 96.8%.

The comparison of the mean O$_2$ sat for the room temperature and body temperature injectate protocols over time following the administration of atropine revealed no significant difference, (p = 0.8880), as illustrated in Figure 11.
Figure 11. Group II post drug arterial oxygen saturation.
The mean O$_2$ sat made a significant decline from baseline to the 20 second post suctioning value following the administration of atropine. The decline was 3.9% for the room temperature and 5.7% for the body temperature injectate protocol. Both protocols returned to baseline parameters by the one minute sampling.

The decline in mean O$_2$ sat may have been due to the instillation of solution and the application of negative pressure. The solution may have stimulated receptors in the lungs and the negative pressure may have removed gases from the lung fields, thus resulting in a decline in O$_2$ sat.

**Arterial Hemoglobin Values**

**Arterial hemoglobin values for group II pre-drug.**

The mean Hb values for the room temperature and body temperature injectate protocols were not significantly different at baseline. The mean Hb value for the room temperature injectate was 9.5 gm/dl and the mean Hb value for the body temperature injectate was 9.6 gm/dl. The comparison of the mean Hb values for the room temperature and body temperature injectate protocols over time revealed no significant difference (p = 0.7543).

The mean Hb values for the room temperature and body temperature protocols rose slightly (0.01 gm/dl) for the 20 second post suctioning ABG. The increase, when examined in relation to the other values, was probably due
to the normal fluctuations of sampling. Overall, the mean Hb values did not change for the room temperature and body temperature injectate protocols.

**Arterial hemoglobin values for group II post drug.**

The mean Hb values for the room temperature and body temperature injectate were identical at baseline following the administration of atropine. The mean Hb value for the room temperature injectate and body temperature injectate protocols was 10.4 gm/dl. The comparison of the mean Hb values for the room temperature and body temperature injectate protocols over time following the administration of atropine was not significant, (p = 0.7429). The mean Hb values fluctuated slightly, (0.1 gm/dl), over time. The fluctuations were characteristic of the normal changes that may be seen from one ABG sampling to the next.

**Carbon Dioxide**

**PaCO₂ for group I pre-drug.**

The mean PaCO₂ for the no suction and suction protocols were not significantly different from each other at baseline. The mean PaCO₂ for the no suction protocol was 38.4 mmHg and the mean PaCO₂ for the suction protocol was 38.5 mmHg.

When the pattern of the mean PaCO₂ for the no suction and suction protocols were compared over time there was no significant differences, (p = 0.9055), as illustrated in Figure 12. Both of the protocols showed a slight rise in
mean PaCO₂, (39.5 mmHg for no suction and 42.0 mmHg for suction), at the 20 second ABG. By the one minute sampling the mean PaCO₂ had returned to baseline parameters. The increase in the PaCO₂ might have been due to partial airway obstruction with the catheter for both protocols. For the suction protocol, the removal of gases from the airway may have contributed to the greater rise in mean PaCO₂.
**Group I Pre-Drug**

No Suction & Suction

Arterial Carbon Dioxide (PaCO₂) -- mmHg

* = p < 0.05

*Figure 12. Group I pre-drug PaCO₂.*
**PaCO₂ for group I post drug.**

The mean PaCO₂ for the no suction and suction protocols were not significantly different from each other at baseline following the administration of atropine. The mean baseline PaCO₂ for the no suction protocol was 43.5 mmHg and the mean baseline PaCO₂ for the suction protocol was 40.7 mmHg.

When the pattern of mean PaCO₂ for the no suction and suction protocols following the administration of atropine were compared over time, there was no significant difference in, \( p = 0.3524 \), as illustrated in Figure 13.

The mean PaCO₂ following the administration of atropine reflected the same pattern of rise at 20 seconds as it did prior to the administration of atropine. The increase in mean PaCO₂ was greater for the suction protocol than it was for the no suction protocol (2.6 mmHg versus 1.3 mmHg). The one minute post suctioning mean PaCO₂ tensions declined to within baseline parameters for both protocols by one minute.
Group I Post Drug

No Suction & Suction

Arterial Carbon Dioxide (PaCO₂) -- mmHg

30 32 34 36 38 40 42 44 46 48 50 52

-60 0 20 60 120 180

Time in Seconds

No Suction

Suction

* = p < 0.05

Figure 13. Group I post drug PaCO₂
Arterial pH Units for group I pre-drug.

The mean arterial pH units for the no suction and suction protocols were identical at baseline. The mean arterial pH units for both the no suction protocol and suction protocol were 7.44.

When the two protocols were examined over time, the pattern of change was not significantly different, \( (p = 0.5510) \), as illustrated in Figure 14. The change in mean arterial pH from baseline to the 20 second post suctioning sample was not significant for the no suction and suction protocols. The lack of a significant change would be appropriate considering that the overall change for the \( \text{PaCO}_2 \) was not significant. The mean baseline pH of 7.44 fell only slightly to 7.43 at the 20 second post suctioning sampling for the suction protocol. The mean \( \text{PaCO}_2 \) rose from a baseline value of 38.5 mmHg to 42.0 mmHg for the suction protocol. Thus the decline in pH is linked to the rise in mean \( \text{PaCO}_2 \) for the suction protocol. There was no change in pH for the no suction protocol.
Group I Pre-Drug

No Suction & Suction

Arterial pH Units

7.48

7.39

7.3

-60  +20  +60  +120  +180

Time in Seconds

■ No Suction  ■ Suction

* = p < 0.05

Figure 14. Group I pre-drug pH.
Arterial pH units for group I post drug.

The mean arterial pH units for the no suction and suction protocols were significantly different at baseline following the administration of atropine. The mean arterial pH units for the no suction protocol was 7.37 and the mean arterial pH units for the suction protocol was 7.40.

The comparison of the arterial pH units for the no suction and suction protocols over time following the administration of atropine revealed no significant difference, (p = 0.1564), as illustrated in Figure 15.

There was no change in the mean arterial pH units for the no suction protocol from baseline to the 20 second post suctioning sampling following the administration of atropine. There was a 0.02 decline in mean arterial pH units from baseline to 20 seconds post suctioning. The decline correlates with the 2.6 mmHg rise in the PaCO₂ for the suction protocol following the administration of atropine. The reason for the change, for the suction protocol as compared to the no suction protocol, may have been due to the application of negative pressure, the removal of gases from the airway and the partial obstruction of the airway with the suction catheter.

For both the no suction and suction protocol following the administration of atropine, the mean baseline PaCO₂ values were higher than the mean baseline
PaCO₂ values prior to the administration of atropine. The reason for the rise, or retention of carbon dioxide following the administration of atropine was due to increased dead space due to bronchodilation resulting from the sympathetic predominance following vagal blockade.
Group I Post Drug

No Suction & Suction

Arterial pH Units

7.48

7.39

7.3

-60  +20  +60  +120  +180

Time in Seconds

■ No Suction  ■ Suction

Figure 15. Group I post drug pH.
Bicarbonate

Arterial bicarbonate values for group I pre-drug.

The mean arterial HCO$_3$ values for the no suction and suction protocols were not significantly different at baseline. The mean HCO$_3$ value for the no suction protocol was 26.5 millimoles per liter (mm/l) and the mean HCO$_3$ value was 26.2 mm/l.

The comparison of the mean HCO$_3$ values for the no suction and suction protocols over time revealed no significant differences, (p = 0.9433), as illustrated in Figure 16. The lack of significant differences from one ABG sampling to the next corresponds to the fact that the bicarbonate system is slower to respond as a buffer for the carbon dioxide. Therefore, one would not expect to see major alterations HCO$_3$ values over 50 seconds.
Figure 16. Group I pre-drug $\text{HCO}_3$. 
Arterial bicarbonate values for group I post drug.

The mean HCO$_3$ values for the no suction and suction protocols were not significantly different at baseline after the administration of atropine. The mean HCO$_3$ value for the no suction protocol was 25.0 mm/l and the mean HCO$_3$ value for the suction protocol was 25.1 mm/l.

The comparison of the mean HCO$_3$ values for the no suction and suction protocols over time following the administration of atropine revealed no significant difference, (p = 0.7743), as illustrated in Figure 17. There was a slight increase in the mean HCO$_3$ from baseline to the 20 second post suctioning sampling. The increase was 0.4 mm/l for the no suction protocol and 0.1 mm/l for the suction protocol. It was difficult to discern if the slight increase was related to the actual suctioning procedure or to the normal fluctuations that occur from one sampling to the next.
Figure 17. Group I post drug HCO₃. 
Arterial Carbon Dioxide

\( \text{PaCO}_2 \) for group II pre-drug.

The mean \( \text{PaCO}_2 \) for the room temperature and body temperature injectate protocols were significantly different at baseline. The mean \( \text{PaCO}_2 \) for the room temperature injectate protocol was 38.3 mmHg and the mean \( \text{PaCO}_2 \) was 41.0 mmHg.

When the pattern of mean \( \text{PaCO}_2 \) for the room temperature and body temperature injectate protocols were compared over time there was no significant difference, \((p = 0.4870)\), as illustrated in Figure 18. The mean \( \text{PaCO}_2 \) values at the post 20 second ABG sampling rose significantly to 3.6 mmHg from the baseline value for the room temperature injectate protocol and 2.1 mmHg for the body temperature injectate protocol. By the one minute ABG sampling, the mean \( \text{PaCO}_2 \) values declined to within baseline parameters.
Group II Pre-Drug

Room Temperature & Body Temperature

Arterial Carbon Dioxide (PaCO2) -- mmHg

* = p < 0.05

* = p < 0.05

Figure 18. Group II pre-drug PaCO₂.
**PaCO₂ for group II post drug.**

The mean PaCO₂ for the room temperature and body temperature injectate were not significantly different from each other at baseline following the administration of atropine. The mean baseline PaCO₂ for the room temperature injectate protocol was 45.0 mmHg and the mean baseline PaCO₂ for the body temperature injectate protocol was 44.3 mmHg.

The pattern of mean PaCO₂ values for the room temperature and body temperature injectate protocols following the administration of atropine were analyzed over time. The results revealed that there was no significant difference, (P = 0.3767), in the pattern of response as illustrated in Figure 19.
Group II Post Drug

Room Temperature & Body Temperature

Arterial Carbon Dioxide (PaCO2) — mmHg

* = p < 0.05

Figure 19. Group II post drug PaCO2.
The mean PaCO₂ for the body temperature component following the administration of atropine was significantly higher (50.0 mmHg) at 20 second post suctioning when compared to the baseline value. But, by the one minute sampling the mean PaCO₂ had returned to within baseline parameters.
Arterial pH units for group II pre-drug.

The mean arterial pH units for the room temperature and body temperature injectate protocols were significantly different at baseline. The mean arterial pH units for the room temperature injectate was 7.46 and the mean arterial pH units for the body temperature injectate was 7.42.

When the mean arterial pH units for the room temperature and body temperature injectate protocols were compared over time there was no significant difference, \( p = 0.0880 \), as illustrated in Figure 20. There was a significant difference in the mean baseline arterial pH units. The remainder of the mean arterial pH values for the room temperature injectate protocol and the body temperature injectate protocol did not differ significantly.
Group II Pre-Drug

Room Temperature & Body Temperature

Arterial pH Units

7.48
7.39
7.3

-60  +20  +60  +120  +180

Time in Seconds

Room Temperature  Body Temperature

* = p < 0.05

Figure 20. Group II pre-drug pH.
The mean arterial pH units for the room temperature and body temperature injectate protocols reflected a decrease that is linked to the rise in mean PaCO$_2$. Both the room temperature and body temperature injectate protocols declined at the 20 second post suctioning sampling. The body temperature protocol returned to baseline by the three minute post suctioning sampling. Whereas, the room temperature injectate protocol mean arterial pH units were still below the baseline value at the three minute post suctioning sampling. When the mean arterial pH units for the room temperature protocol were examined in conjunction with the mean PaCO$_2$ values, the two patterns correspond. There was an increase above baseline for the mean PaCO$_2$ that corresponded to the decreases in mean arterial pH units.

**Arterial pH units for group II post drug.**

The mean arterial pH units for the room temperature and body temperature injectate protocols were not significantly different at baseline after the administration of atropine. The mean arterial pH units for the room temperature injectate was 7.36 and the mean arterial pH units for the body temperature injectate was 7.37.

The comparison of the room temperature and body temperature injectate protocols over time following the
administration of atropine were not statistically significant, \( p = 0.8122 \), as illustrated in Figure 21. There was an identical decline of 0.03 for the mean arterial pH units for the room temperature and body temperature protocols following the administration of atropine at the 20 seconds post suctioning. The pattern of decline for the mean arterial pH units at the 20 second post suctioning sampling corresponds with the rise in the mean \( \text{PaCO}_2 \).
Group II Post Drug

Room Temperature & Body Temperature

Arterial pH Units

* = p < 0.05

Figure 21. Group II post drug pH.
Bicarbonate

Arterial bicarbonate values for group II pre-drug.

The mean HCO$_3$ values for the room temperature and body temperature injectate protocols were not significantly different at baseline. The mean HCO$_3$ value for the room temperature injectate protocol was 26.7 mm/l and the mean HCO$_3$ value for the body temperature injectate was 26.5 mm/l.

When the mean HCO$_3$ values for the room temperature and body temperature injectate protocols were compared over time there was no significant difference, (p = 0.9591), as illustrated in Figure 22. The mean HCO$_3$ level declined 0.1 mm/l for the room temperature protocol and 0.3 mm/l for the body temperature protocol. The variation may have been due to the normal fluctuation in the HCO$_3$ that occurs normally from one sampling to the next.
Figure 22. Group II pre-drug HCO₃.
Arterial bicarbonate values for group II post drug.

The mean HCO$_3$ values for the room temperature and body temperature injectate protocols were not significantly different at baseline following the administration of atropine. The mean HCO$_3$ value for the room temperature injectate protocol was 25.5 mm/l and the mean HCO$_3$ value for the body temperature injectate protocol was 25.0 mm/l.

The comparison of the mean HCO$_3$ values for the room temperature and body temperature injectate protocols over time after the administration of atropine were not significantly different, (p = 0.3965), as illustrated in Figure 23. There was a slight, but insignificant increase in the mean HCO$_3$ values for the room temperature and body temperature protocols following the administration of atropine. The mean HCO$_3$ values rose 0.4 mm/l for the room temperature protocol and 1.2 mm/l for the body temperature protocol. The increase may have been related to the rise in mean PaCO$_2$ values at the post 20 second sampling.
Group II Post Drug

Room Temperature & Body Temperature

Arterial Bicarbonate Values

Time in Seconds

- Room Temperature  ■ Body Temperature

Figure 23. Group II post drug HCO$_3$. 
The Effect of Endotracheal Suctioning on Heart Rate

Heart rate for each of the suctioning events was calculated by counting the number of R waves that occurred in a 20 second interval and then multiplying by three. Thus, there were six values for heart rate prior to the suctioning procedure. These six values were then averaged to yield one value for baseline heart rate.

The heart rate during the endotracheal suctioning procedure was calculated for each component of the suctioning protocol (Table 4). Therefore, there were nine values during the suctioning procedure for the no suction and suction protocols. During the room temperature and body temperature suctioning protocols there were thirteen values for the actual suctioning procedure. The reason for the discrepancy in the number of heart rate values in the two groups was due to the administration of solution in the room temperature and body temperature protocols. In both instances, the instillation of solution added four extra steps to the overall suctioning procedure.
Table 4
Time line for the suctioning protocol for catheter insertion without solution

CATHETER INSERTION WITHOUT SOLUTION

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Time line for the suctioning protocol for catheter insertion with solution

CATHETER INSERTION WITH SOLUTION

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<tr>
<td>OBTAIN 180 SEC ABG</td>
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<td>180</td>
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The post-suctioning heart rate was calculated in the same manner as the pre-suctioning heart rate. Thus the number of R waves in a twenty second interval were counted and multiplied by three to yield a value for heart rate in beats per minute. The total number of post-suctioning heart rate values was nine. The post-suctioning heart rate values were not averaged due to significant fluctuations from one 20 second interval to the next.

Heart Rate Values Group I Pre-Drug

The mean heart rate values for the no suction and suction protocols were not significantly different from each other at baseline. The mean baseline heart rate for the no suction protocol was 115 beats per minute and the mean baseline heart rate for the suction protocol was 110 beats per minute.

When the pattern of heart rate changes for the no suction and suction protocols were compared over time, there was no significant difference in the pattern of response, \( p = 0.9157 \), as illustrated in Figure 24.
Group I Pre-Drug

No Suction & Suction

Heart Rate (Beats Per Minute)

Components of the Suction Procedure

* = p < 0.05

Figure 24. Group I pre-drug heart rate.
The mean heart rates for the no suction and suction protocols were not significantly different. However, when one examines the change for each method several interesting findings emerge. There was a 9 beat per minute decline for the no suction protocol from baseline following the first withdrawal of the suction catheter as compared to a 14 beat per minute decline for the suction protocol. Following the second withdrawal of the suction catheter the heart rate declined 7 more beats per minute for the no suction protocol and rose 6 beats per minute for the suction protocol. The heart rate made its largest decline following the first withdrawal of the suction catheter. Following the second withdrawal of the suction catheter the difference in heart rate was obliterated.

Heart Rate Values Group I Post Drug

The mean heart rate values for the no suction and suction protocols were significantly different from each other at baseline following atropine administration. The mean baseline heart rate for the no suction protocol was 155 beats per minute and the mean baseline heart rate for the suction protocol was 161 beats per minute.

When the pattern of heart rate changes for the no suction and suction protocols were compared over time, there was no significant difference, \( p = 0.9563 \), as illustrated in Figure 25.
There was no change in heart rate following the first withdrawal of the suction catheter for the no suction and suction protocols following the administration of atropine. Following the second withdrawal of the suction catheter resulted in an increase of 3 beats per minute for the no suction protocol and no change for the suction protocol. Overall, the mean heart rates for both methods of suctioning were essentially unchanged.
Group I Post Drug

No Suction & Suction

Heart Rate (Beats Per Minute)

![Graph showing heart rate over time with labels for No Suction and Suction.]

Figure 25. Group I post drug heart rate.
Heart Rate Values Group II Pre-Drug

The mean heart rate values for the room temperature and body temperature injectate protocols were not significantly different from each other at baseline. The mean baseline heart rate for the room temperature injectate protocol was 97 beats per minute and the mean baseline heart rate for the body temperature injectate protocol was 99 beats per minute.

When the pattern of heart rate changes for the room temperature and body temperature injectate protocols was compared over time, there was a significant difference, (p = 0.0180), as illustrated in Figure 26.
Group II Pre-Drug

Room Temperature & Body Temperature

Heart Rate (Beats Per Minute)

Components of the Suction Procedure

* = p < 0.05

Figure 26. Group II pre-drug heart rate.
A Duncan's post hoc analysis was conducted to determine which values differed. The post hoc analysis revealed that for the room temperature protocol the first time the Novametrix C/D adaptor was opened the mean heart rate declined significantly from the baseline reading of 97 beats per minute to 89 beats per minute. During the insertion of the suction catheter the mean heart rate increased to 91 beats per minute and then declined to 90 beats per minute during the first withdrawal of the catheter. The mean heart rate of 90 beats per minute was significantly different from the mean baseline heart rate. When the port hole of the Novametrix C/D adaptor was closed for the second time, the mean heart rate made a significant decline from baseline to 81 beats per minute. The mean heart rate returned to within baseline parameters only to make one more significant decline to 85 beats per minute during the final closing of the Novametrix C/D adaptor following the completion of the entire suctioning procedure.

The Duncan's post hoc analysis revealed that the opening of the Novametrix C/D port hole for the first time during the body temperature injectate protocol caused the mean heart rate to decline significantly from 99 beats per minute at baseline to 90 beats per minute. The mean heart rate immediately returned to within baseline parameters only to fall again when the port hole was closed to 91
beats per minute. Following the closure of the port hole
the mean heart rate returned to within baseline
parameters. The last, and greatest, decline in mean heart
rate occurred when the Novametrix C/D adaptor was opened
for the second time. During the second opening of the
port hole the mean heart rate fell to 83 beats per minute.
For the remainder of the body temperature injectate
protocol the mean heart rate remained within baseline
parameters.

The mean decline from baseline heart rate was 16
beats per minute for the room temperature protocol and 5
beats per minute with the body temperature protocol
following the first withdrawal of the suction catheter.
Following the second withdrawal of the suction catheter
the mean heart rate declined 11 beats per minute for the
room temperature protocol and 6 beats per minute for the
body temperature protocol. By 20 seconds post suctioning
the heart rate values had returned to within baseline
parameters.

Heart Rate Values Group II Post Drug

The mean heart rate values for the room temperature
and body temperature injectate protocols were
significantly different at baseline following the
administration of atropine. The mean baseline heart rate
for the room temperature injectate protocol was 155 beats
per minute and the mean baseline heart rate for the body
temperature injectate protocol was 150 beats per minute.

When the pattern of heart rate changes for the room
temperature and body temperature protocols was compared
over time, there was no significant difference, (p =
0.7994), as illustrated in Figure 27.

The mean heart rate for the room temperature and body
temperature injectate protocols during the actual
suctioning procedure were not significantly different from
their respective baseline values. The mean heart rate for
the room temperature injectate protocol did not fluctuate
from baseline following the first and second withdrawal of
the suction catheter. The mean heart rate for the body
temperature injectate protocol fluctuated 3 beats per
minute following the first withdrawal of the suction
catheter and 1 beat per minute following the second
withdrawal of the suction catheter. By 20 seconds post
suctioning the mean heart rate for both suctioning
protocols had decline 4 beats per minute. By three
minutes post suctioning, the mean heart rate for the room
temperature protocol decline 6 beats per minute and the
mean heart rate for the body temperature injectate
protocols declined 6 beats per minute. The reason for the
gradual downward drift for both the room temperature and
body temperature injectate protocols appears to have been
associated with the completion of the actual suctioning
procedure. The piglets were no longer being manipulated and therefore the mean heart rates were allowed to recover.
Group II Post Drug
Room Temperature & Body Temperature

Heart Rate (Beats Per Minute)

Components of the Suction Procedure
- Room Temperature
- Body Temperature

Figure 27. Group II post drug heart rate.
Discussion
When the application of suction was compared to the no suctioning protocol there was a decrease in PaO₂, a decline in arterial oxygen saturation and a rise in PaCO₂. The changes were similar to the changes seen with catheter insertion without suction as compared to catheter insertion with suction that has been documented by other investigators (Boba, Cincotti, Piazza & Landmesser, 1959; Rosen & Hillard, 1960; Woodburne & Powaser, 1980; Baker et al., 1983).

The changes in ABG values are congruent with the findings of an unpublished in vitro study by the investigator. The instillation of the 5/6 french suction catheter down a 3.0 endotracheal tube without the application of negative pressure resulted in a 14 mmHg decline in the pressure being measured at the distal end of the endotracheal tube via a pneumotachograph interfaced to a Gould 2400S recorder. When the same suction catheter was inserted and negative pressure applied, the pressure fell 21 mmHg. Thus, simply instilling the suction catheter would result in occluding the airway and subsequently the PaCO₂ would rise. With the application of suction, not only would the PaCO₂ rise but the PaO₂ would decline due to the removal of gases form the alveoli, leading to a decline in arterial oxygen saturation. Hence, the modest decline in the piglet's
mean $\text{PaO}_2$ and rise in mean $\text{PaCO}_2$ with the insertion of the suction catheter without the application of negative pressure is substantiated by the in vitro work. In addition, the insertion of the suction catheter and the application of negative pressure resulted in an exaggerated decline in the piglets mean $\text{PaO}_2$ and similar rise in mean $\text{PaCO}_2$ when compared to the no suction protocol.

The heart rate alterations associated with the endotracheal suctioning of newborn swine were present prior to the administration of atropine for the no suction and suction protocols. Numerous investigations have documented that bradycardia is frequently associated with the suctioning procedure (Cabal et al., 1979; Zmora & Merritt, 1980; Cunningham et al., 1984; and Gunderson et al., 1986). Following the administration of atropine, to produce vagal blockade, the heart rate alterations were obliterated. The obliteration of heart rate variability indicates that the alterations are due to mechanical and neural stimulation and not hypoxemia as speculated by Cabal et al., (1979). Therefore, it appears that the heart rate alterations that are associated with endotracheal suctioning are not due to a lower $\text{PaO}_2$ but instead are related to mechanical stimulation from the insertion of the suction catheter. This is substantiated by the fact that, vagal blockade did not have an effect on
the pattern of decline in PaO₂, arterial oxygen saturation and rise in PaCO₂.

The lack of heart rate variability following the administration of atropine indicates that the heart rate alterations are vagally mediated is congruent with the findings of Winston, Gravelyn, & Sitrin, (1987), and McCauley, & Boller, (1988). Winston (1987) examined the administration of nebulized normal saline (control), parenteral atropine and nebulized atropine on heart rate alterations associated with endotracheal suctioning of adult patients. The findings indicate that the nebulized atropine, (0.05 mg/kg ideal body weight), prevented the bradycardic episodes associated with endotracheal suctioning.

With the room temperature injectate and body temperature injectate protocols the PaO₂ fell, the arterial oxygen saturation declined and the PaCO₂ rose. The changes following the endotracheal suctioning protocol followed the pattern of change associated with the application of suction. Again, the pattern of change for the PaO₂, arterial oxygen saturation, and PaCO₂ conforms to the changes that have been reported in the literature (Boba et al., 1959; Rosen & Hillard, 1960; Woodburn & Powaser, 1980; Baker et al., 1983).

The decline in PaO₂ with the instillation of aerosolized nonissomolar fluids has been reported to cause
bronchoconstriction in humans, whereas aerosolized isotonic saline solution may not (Hogg, 1981; Sheppard, Rizk, Boushey & Bethel, 1983; Eschenbacher, Boushey & Sheppard, 1984). Killingsworth et al. (1987) found that bronchoconstriction occurred with nonaerosolized isotonic saline solutions. It has been noted that the alterations in PaO₂ may have been due to the mechanical stimulation of irritant receptors by the flow of fluid over the epithelium (Killingsworth et al., 1987; Widdcombe, Kent, & Nadal, 1962). The temperature of the solution may also be a factor in the degree of change in ABG values. Eschenbacher et al., (1984), and Killingsworth et al. (1987), found that the cool, room temperature solutions, triggered bronchoconstriction in cats and newborn calves. In this investigation the room temperature injectate protocol resulted in greater declines in PaO₂ than did the body temperature injectate. The persistence of the decline in PaO₂, following ligation of the vagus nerve in Killingsworth et al., (1987) investigation is congruent with the findings following vagal blockade in this study.

When room temperature injectate was used for the suctioning protocol, the time to return to baseline parameters was greater than the time required by the body temperature injectate protocol. Prior to the administration of atropine, the PaO₂ never returned to baseline for the room temperature protocol and the PaO₂
was back to within baseline parameters by the one minute post suctioning ABG for the body temperature injectate protocol. Following vagal blockade, the PaO$_2$ returned to baseline parameters at the three minute post suctioning ABG for the room temperature injectate protocol and again by the one minute post suctioning ABG for the body temperature injectate protocol. Therefore, the body temperature normal saline injectate may have less of an impact on the epithelium and irritant receptors.

The heart rate alterations may also be linked to the temperature of the normal saline. The heart rate for the room temperature injectate protocol declined 8 beats per minute when the solution was instilled. Whereas, when the body temperature normal saline was instilled there was essentially no change in heart rate. The magnitude of heart rate alterations was greater and longer in duration for the room temperature injectate protocol than were the changes for the body temperature injectate protocol. Therefore, the stimulation of the irritant receptors may be shorter in duration for the body temperature injectate protocol.

Overall, the lack of heart rate alterations for the no suction, suction, room temperature and body temperature injectate protocols, following the administration of atropine to produce vagal blockade, indicates that the heart rate alterations associated with endotracheal
suctioning are vagally mediated. Also, the body
temperature injectate protocol may have less of an impact
on ABG parameters and heart rate than the room temperature
injectate.
CHAPTER V
SUMMARY, LIMITATIONS, IMPLICATIONS FOR NURSING PRACTICE,
RECOMMENDATIONS FOR FURTHER STUDY

Summary

The purpose of this experimental study was to identify the mechanical and/or neural factors contributing to the alterations in heart rate associated with endotracheal suctioning. The investigation examined: the effects of catheter insertion alone; catheter insertion with negative pressure application; and the effects of room temperature (24.42 degree centigrade) normal saline versus body temperature (37 degree centigrade) on oxygenation and heart rate prior to and following vagal blockade with atropine. The subjects were 22 newborn swine less than 24 hours of age. The piglets were randomly assigned to one of two groups. In group I, the 11 piglets were exposed to both the suction and no suction protocols. The 11 piglets in group II were exposed to room temperature (24.42 degree centigrade) and body temperature (37 degree centigrade) injectate suctioning protocols. Preoperatively the subjects were anesthetized with ketamine and xylazine 0.05 cc per kilogram. The
electrocardiogram in lead II was recorded on a Gould 2600S recorder. Arterial blood gases were drawn one minute before, and at 20 seconds, one, two and three minutes following the suctioning protocol. The arterial blood gases were analyzed on a calibrated Corning semi-automated arterial blood gas machine. Data were analyzed using a repeated measures analysis of variance. The application of suction in group I resulted in a mean decline of 40 mmHg in $\text{PaO}_2$ versus a mean decline of 13 mmHg in $\text{PaO}_2$ for the no suction protocol at the 20 second post suctioning ABG. Following vagal blockade with atropine, the decline in the mean $\text{PaO}_2$ was 37 mmHg for the suction protocol as compared to a 2 mmHg for the no suction protocol. Following the withdrawal of the suction catheter, mean heart rate declined 15 beats per minute with suction and 9 beats per minute with no suction. The difference was obliterated following the second withdrawal of the suction catheter. There was no difference in the heart rate between the suction protocol (1 beat per minute) and the no suction protocol (2 beats per minute) following vagal blockade, indicating that the decline in heart rate with endotracheal suctioning is vagally mediated.

In group II room temperature injectate resulted in a 61 mmHg decline in the mean $\text{PaO}_2$ at 20 seconds following the completion of the endotracheal suctioning protocol. Body temperature injectate resulted in a 24 mmHg decline
in the mean PaO$_2$ at 20 seconds following the completion of the suctioning procedure. The decline in PaO$_2$ was 54 mmHg for the room temperature protocol and 38 mmHg for the body temperature protocol following vagal blockade with atropine.

In group II, following the first withdrawal of the suction catheter, the mean heart rate declined 16 beats per minute for the room temperature protocol and 8 beats per minute for the body temperature protocol. Following the withdrawal of the suction catheter for the second time, the heart rate alterations were virtually obliterated. The mean decline in heart rate for the room temperature protocol was 5 beats per minute and 2 beats per minute for the body temperature protocol following atropine administration. There was no difference in the mean heart rate between the room temperature injectate protocol and the body temperature injectate protocol following vagal blockade, indicating the decline in heart rate is vagally mediated. The findings suggest that the decline in PaO$_2$ is associated with the application of suction and the room temperature injectate protocol caused a greater decline in PaO$_2$ and heart rate.

**Limitations**

The threats to the internal validity of the study include history and selection. History, in this case, refers to the effects of the passage of time and the
deterioration of the animal preparation over time. In order to maintain the animal preparation in an optimal state, bolus intravenous injections of five percent dextrose and water were given every half hour to maintain the serum glucose and vascular volume. However, with each arterial blood gas obtained, 0.6 cubic centimeters of blood were removed. Even though the smallest volume of blood was used, there was an eventual decline in arterial hemoglobin values. Each suctioning protocol required 3.0 cubic centimeters of blood for analysis and all four protocols required a total of 12.0 cubic centimeters. One is left to question that the differences in baseline and corresponding post suctioning values for the arterial blood gas values were a result of the length of time required to study each animal or as a result of the administration of the atropine. One way to determine if the difference was due to deterioration over time or as a result of the administration of atropine would be to procure more piglets and perform the same study excluding the administration of atropine. The pattern of arterial blood gas values could then be examined to determine if they were similar to the pattern obtained for the piglets that were exposed to atropine.

The second threat to internal validity is selection. Each pair of piglets received by the investigator was selected by a breeder who was not affiliated or aware of
the purpose and intent of the investigation. The possibility does exist that the piglets were not selected from the litter by random, but rather due to the size, temperament, physical condition or due to some other characteristic.

The external validity, or generalizability, of the research findings to other samples is limited. The results from this study can only be generalized to the population of newborn yorkshire piglets less than 24 hours of age. However, the potential for future inquiry based on the findings of the investigation is imperative. The pattern of heart rate alterations may not be similar for the more neurologically advanced newborn piglet as compared to the premature human infant. The potential for fewer heart rate alterations of a lesser magnitude may exist for the newborn piglets. The newborn piglets in the study had healthy lungs. Whereas a population of premature infants with respiratory distress syndrome would have decreased surfactant and the potential for physiologic shunting. Therefore, premature infants may respond differently to endotracheal suctioning than did the subjects in this study.

Implications for Nursing Practice

Based on the data from the study, it would appear that endotracheal suctioning leads to alterations in ABG and heart rate parameters. Therefore, the frequency and
duration of the suctioning procedure should be examined. Endotracheal suctioning should be performed only when necessary rather than on a set schedule such as every two hours.

A second implication, would be to examine the duration of the suctioning procedure. The amount of time required to pass and remove the catheter should be as short in duration as possible.

Until the use of room temperature and body temperature injectate for endotracheal suctioning protocols has been investigated using a population comprised of premature infants with respiratory distress syndrome, it would appear that the body temperature injectate may be potentially less detrimental. The clinical implications of the data generated by the study suggest that the use of body temperature normal saline for instillation down the endotracheal tube should be incorporated into endotracheal suctioning protocols. The bottle of normal saline could be easily placed in the isolette and warmed rather than storing the solutions at the bedside.

**Recommendations for Further Study**

Further explorations could be carried out using premature piglets that are approximately 110 days in gestation that have been delivered by caesarean section. The effects of the four protocols could be examined to
determine if the patterns of response to the endotracheal suctioning protocols were similar to the more neurologically advanced newborn piglet.

The implications for further research generated by the study is a need to focus on the effects of endotracheal suctioning using a population comprised of premature infants. The next step is to examine the effects of room temperature and body temperature injectate on transcutaneous partial pressure of oxygen, arterial oxygen saturation and heart rate. The subjects for the study would be premature infants diagnosed with respiratory distress syndrome that are less than 96 hours of age.
APPENDIX A

COMPUTER RANDOMIZATION PROGRAM
RANDOMIZATION

GROUP 1:
1 = CATHETER INSERTION WITHOUT SUCTION
2 = CATHETER INSERTION WITH SUCTION

GROUP 2:
1 = ROOM TEMPERATURE SOLUTION
2 = BODY TEMPERATURE SOLUTION

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APPENDIX B

ENDOTRACHEAL SUCTIONING METHOD A
Appendix B
Endotracheal Suction
Method A
Catheter Insertion and Removal

Equipment Needed:

1. Suction catheter - use size 5/6 french with a 2.5 and 3.0 mm internal diameter endotracheal tube.
2. Suction apparatus - pressure set to 80 millimeters of mercury for a flow of 4.8 liters per minute through the catheter.

Procedure:

1. Connect the suction catheter to the tubing of the negative pressure source.
2. Open the port hole in the Novametrix C/D adapter and insert the suction catheter without negative pressure until resistance is met. Pull the catheter back 0.5 centimeters. The insertion of the suction catheter should not exceed 8 seconds.
3. Withdraw the suction catheter using a rotating motion.
4. The withdrawal of the suction catheter should not exceed 10 seconds.
5. Immediately re-establish ventilation by occluding the porthole in the Novametrix C/D adapter.
6. Wait six seconds before repeating the procedure.
7. Open the port hole in the Novametrix C/D adapter and insert the suction catheter without negative pressure until resistance is met. Pull the catheter back 0.5 centimeters. The insertion of the suction catheter should not exceed 8 seconds.
8. Withdraw the suction catheter using a rotating motion.
9. The withdrawal of the suction catheter should not exceed 10 seconds.
10. Immediately re-establish ventilation by closing the porthole in the Novametrix C/D adapter.
APPENDIX C

ENDOTRACHEAL SUCTIONING METHOD B
Appendix C
Endotracheal Suction
Method B
Catheter Insertion with Negative Pressure During Catheter Removal

Equipment Needed:

1. Suction catheter - use size 5/6 french with a 2.5 and 3.0 mm internal diameter endotracheal tube.
2. Suction apparatus - pressure set to 80 millimeters of mercury for a flow of 4.8 liters per minute through the suction catheter.

Procedure:

1. Connect the suction catheter to the tubing of the negative pressure source.
2. Open the port hole in the Novametrix C/D adapter and insert the suction catheter without negative pressure until resistance is met. Pull the catheter back 0.5 centimeters. Insertion of the suction catheter should not exceed 8 seconds.
3. Apply the negative pressure to the suction catheter continuously during its removal.
4. Withdraw the suction catheter using a rotating motion.
5. The withdrawal of the suction catheter should not exceed 10 seconds.
6. Immediately re-establish ventilation by occluding the porthole in the Novametrix C/D adapter.
7. Wait 6 seconds before repeating the procedure.
8. Open the port hole in the Novametrix C/D adapter and insert the suction catheter without negative pressure until resistance is met. Pull the catheter back 0.5 centimeters. Insertion of the suction catheter should not exceed 8 seconds.
9. Apply the negative pressure to the suction catheter continuously during its removal.
10. The withdrawal of the suction catheter should not exceed 10 seconds.
11. Immediately re-establish ventilation by closing the porthole in the Novametrix C/D adaptor.
APPENDIX D

ENDOTRACHEAL SUCTIONING METHOD C
Appendix D  
Endotracheal Suction  
Method C  
Room Temperature Injectate  

Equipment Needed:

1. Bottle of room temperature 0.9% normal saline for tracheal instillation.  
2. Suction catheter - use size 5/6 french with a 2.5 and 3.0 mm internal diameter endotracheal tube.  
3. Suction apparatus - pressure set to 80 mmHg for a flow of 4.8 liters per minute through the suction catheter.  

Procedure:  

1. Connect the suction catheter to the tubing of the negative pressure source.  
2. Open the port hole in the Novametrix C/D adapter.  
3. Inject 0.5 ml of room temperature normal saline down the endotracheal tube.  
4. Close the port hole in the Novametrix C/D adapter and wait six seconds before continuing the procedure.  
6. Open the port hole in the Novametrix C/D adapter.  
7. Insert the suction catheter without negative pressure until resistance is met. Pull the catheter back 0.5 centimeters. Catheter insertion should not exceed 8 seconds.  
8. Apply the negative pressure to the suction catheter continuously during its removal.  
9. The suction catheter should be withdrawn using a rotating motion.  
10. The withdrawal of the suction catheter should not exceed 10 seconds.  
11. Immediately re-establish ventilation by occluding the port hole in the Novametrix C/D adapter.  
12. Wait six seconds before continuing the procedure.  
14. Open the port hole in the Novametrix C/D adapter and insert the suction catheter without negative pressure until resistance is met. Pull the catheter back 0.5 centimeters. Catheter insertion should not exceed 8 seconds.  
15. Apply the negative pressure to the suction catheter continuously during its removal.  
16. The suction catheter should be withdrawn using a rotating motion.  
17. The withdrawal of the suction catheter should not exceed 10 seconds.  
18. Immediately re-establish ventilation by closing the port hole in the Novametrix C/D adapter.
APPENDIX E

ENDOTRACHEAL SUCTIONING METHOD D
Appendix E
Endotracheal Suction
Method D
Body Temperature Injectate

Equipment Needed:

1. Bottle of 0.9% normal saline for tracheal instillation and suction catheter clearance.
2. Suction catheter - use size 5/6 french with a 2.5 and 3.0 mm internal diameter endotracheal tube.
3. Suction apparatus - pressure set to 80 millimeters of mercury for a flow of 4.8 liters per minute through the suction catheter.
4. Servo controlled heat exchanger apparatus to warm the normal saline to correspond to the piglet's body temperature.

Procedure:

1. Connect the suction catheter to the tubing of the negative pressure source.
2. Draw up 0.5 ml of normal saline heated to 37°C via the servo controlled heat exchanger.
3. Open the port hole in the Novametrix C/D adapter.
4. Inject 0.5 ml of the heated normal saline down the endotracheal tube.
5. Close the port hole in the Novametrix C/D adapter.
6. Wait six seconds before continuing the procedure.
7. Open the port hole in the Novametrix C/D adapter.
8. Insert the suction catheter without negative pressure until resistance is met. Pull the catheter back 0.5 centimeters. Catheter insertion should not exceed six seconds.
9. Apply the negative pressure to the suction catheter continuously during its removal.
10. The suction catheter should be withdrawn using a rotating motion.
11. The withdrawal of the suction catheter should not exceed 10 seconds.
12. Immediately re-establish ventilation by occluding the port hole in the Novametrix C/D adapter.
13. Wait six seconds before continuing the procedure.
14. Open the port hole in the Novametrix C/D adapter and insert the suction catheter without negative pressure until resistance is met. Pull the catheter back 0.5 centimeters. Catheter insertion should not exceed six seconds.
15. Apply the negative pressure to the suction catheter continuously during its removal.
Appendix E Continued
Endotracheal Suction
Method D
Body Temperature Injectate

16. The suction catheter should be withdrawn using a rotating motion.
17. The withdrawal of the suction catheter should not exceed 10 seconds.
18. Immediately re-establish ventilation by closing the port hole in the Novametrix C/D adapter.
APPENDIX F

APPROVAL FORM FROM THE OHIO STATE UNIVERSITY

INSTITUTIONAL LABORATORY ANIMAL CARE AND USE COMMITTEE
April 13, 1988

Dr. Robert L. Hamlin
Veterinary Medicine
Sisson Hall G-50
1900 Coffey Road
Campus

In re: Animal Use Protocol 87A0112

Dear Dr. Hamlin:

In reply to your recent communication wherein you request the period of approval be extended, be advised this has been approved. We have changed our records to reflect the ending date to be April 1, 1989.

We thank you for advising us of this change.

Sincerely,

[Signature]

Fredrick Cornhill, D. Phil.
Chairman, ILACUC

/cc: Dr. Harold Stills
    Jane Weaver, ULAR
Dr. Robert Hamlin
Vet. Physio/Pharm
Sisson Hall
1900 Coffey Rd.

Your animal use protocol:

87AO112  THE EFFECTS OF OXYGENATION ON HEART RATE, PAO 2, AND TCPO 2
IN PREMATURE PIGLETS

was approved for the period 04/87 to 04/88.

1. Do you wish to have the period of approval for this protocol extended? No
   Yes ☐

   If NO, the protocol will be inactivated as of the last date shown above. Please sign
   below and return form as instructed.

2. If YES to question #1, have any changes occurred, or do you wish to propose any
   changes, (including use of more animals than originally proposed) to the approved
   protocol as they relate to the care and use of laboratory animals? No ☐ Yes ☐

3. If NO to question #2, enter here dates of proposed extension, 4/1/89, sign
   below, and return as instructed.

4. If YES to question number 2:

   a. If changes are extensive (e.g. change in species of animal used; significant
      change in number of animals to be used; changes in surgical procedures or other
      procedures that may cause pain or distress, etc.), please complete, in its
      entirety, the enclosed animal use protocol. Review of the protocol will be
      scheduled at the earliest feasible time.

   b. If changes are not extensive, provide a description of the changes proposed
      (referring to appropriate sections of the original protocol) presented in
      sufficient detail to permit the ILACUC to evaluate your request for extension and:

      1) Complete page 1 of the enclosed animal use protocol through the period of
         protocol, entering the dates of the extension proposed.

      2) Complete the top portion of page 2 of the enclosed animal use protocol that
         asks for the listing of all animals to be used.

Return this letter and appropriate supporting information identified above to: Animal
Review Desk, Room 205 Research Center, 1316 Alumnae Rd. Thank you for your cooperation.

Chairperson, ILACUC

Signed: [Signature]
Principal Investigator
APPENDIX G

ENDOTRACHEAL INTUBATION OF THE NEWBORN PIGLET
Appendix G
Endotracheal Intubation of the Newborn Piglet

Equipment Needed:

1. Laryngoscope
2. Miller blades size 0 or 1 (curved or straight)
3. Endotracheal tube size:
   - 2.5 mm: up to 1200 gm
   - 3.0 mm: 1200 to 2500 gm
4. Trach tape
5. Stylet
6. Inflation bag (500cc)
7. Oxygen tubing
8. Suction tubing size: 5/6 F (2.5 mm to 3.0 mm tubes)
9. Stethoscope

Procedure:

1. The piglet is placed on its back.
2. Place the laryngoscope gently into the piglets mouth by introducing the blade into the side of the mouth and over the tongue.
3. Advance the blade forward into the pharyngeal area.
4. Gently lift the entire blade; as the blade is lifted the epiglottis should swing anteriorly.
5. Visualize the V shaped vocal cords.
7. Orally introduce the endotracheal tube bearing a rigid stylus.
8. The endotracheal tube is used to lift the soft palate dorsally from the glosso-epiglottic fold over the epiglottis.
9. The tip of the endotracheal tube is placed on the arytenoidal surface of the epiglottis and advanced ventrally to the floor of the larynx.
10. Before being advanced toward the trachea through the glottis, the tip must immediately be moved dorsally.
11. Once the tip of the tube is moved to the ceiling of the larynx and clears the vocal folds, it may be advanced without difficulty into the trachea and the stylus removed.
12. Auscultate and observe the chest while ventilating the piglet with the manual resuscitation bag. Breath sounds should be bilaterally equal. If not move the tube into position where breath sounds are bilaterally equal.
13. Secure the tube in place by wrapping trach tape around the tube and over the top of the snout. Tie the trach tape securely in place.
14. Quickly suction the endotracheal tube to remove any additional mucus or secretions.
APPENDIX H

CAROTID ARTERY CATHETERIZATION
Appendix H
Carotid Artery Catheterization

Equipment Needed:

1. Bottle of 0.9% Saline
2. Heparin
3. Scalpel
4. Blunt probe
5. Forceps
6. Umbilical tape
7. Umbilical vessel catheter
8. Suture material
9. Stopcock

Procedure:

Method of Insertion:

1. Prepare the catheter and stopcock setup. Fill the catheter lumen with 0.9% saline containing 5 U heparin/ml fluid.
2. Place the piglet on its back.
3. Palpate the trachea and move one centimeter to the right.
4. Palpate the right carotid artery for pulsations.
5. Clip away a 1/4 centimeter by 2 centimeter piece of skin using scissors.
6. With hemostats bluntly dissect through the skin and muscle.
7. Blot any bleeders with a 4 x 4.
8. Locate the carotid artery and remove any excess tissue.
9. Place a suture at the distal end of the carotid artery (closest to the head and tie off).
10. Place a second suture at the base of the artery and tie a loose knot. Do not tie off.
11. Have an assistant place traction on both sutures by pulling up on each suture.
12. Pick up the vessel with forceps and make a small nick in the vessel wall with eye scissors.
13. With the vessel introducer, slip inside the vessel and carefully introduce the catheter that has been previously flushed with heparinized saline into the vessel.
14. Take the loosely tied suture at the base of the vessel and allow the catheter to slip beyond the tie. Quickly tie off.
15. Blood should immediately flow back into the catheter advance the catheter to the six centimeter mark. Secure the catheter in place.
16. Flush the catheter with heparinized normal saline to ensure patency of the vessel every 10 minutes throughout the data collection procedure.
APPENDIX I

ARTERIAL BLOOD GAS SAMPLING FROM AN ARTERIAL LINE
Appendix I
Arterial Blood Gas Sampling from an Arterial Line

Equipment Needed:

1. 3 cc syringe
2. Heparinized TB syringe
3. 3 cc syringe filled with heparinized flush solution
4. Cup of ice
5. Syringe cap or stopper

Procedure:

1. Approach the arterial line at the three way stopcock.
2. Turn the stopcock lever clockwise halfway between the flush syringe and the female end of the stopcock connected to the arterial tubing.
3. Place the empty 3 cc syringe into the port (sampling port), which is at right angles to the catheter and the arterial tubing.
4. Turn the stopcock lever off to the fluid (arterial tubing) line. Lever will be "pointing" to the fluid line.
5. Draw back 2 to 3 cc of blood into the attached empty 3 cc syringe and leave connected to the stopcock. Return the stopcock lever to the midway position, then remove the blood filled syringe and cap it off with a sterile needle.
6. Place the heparinized syringe onto the sampling port. Return the stopcock lever to the off to fluid line position. Draw back 0.3 cc of blood from the catheter and leave connected to the stopcock.
7. Return the stopcock lever to the midway position, remove the sample, cap the sample, place the sample in ice, and replace the 3 cc syringe filled with blood onto the stopcock.
8. Return the stopcock lever to the off to fluid line position, draw back again into the blood filled syringe to remove air bubble, and then push the blood slowly back into the catheter.
9. Return the stopcock lever to midway, remove the empty syringe, and replace the syringe with a new flush filled syringe.
10. Return the stopcock lever to the off to fluid line position, draw back on the flush syringe just enough to clear any air bubbles. Flush the catheter slowly until clear.
11. Rotate the stopcock lever until it once again points toward the flush syringe.
PLEASE NOTE:

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12. Grasp the blood sample, hold vertically, and gently roll it between your hands until all the air bubbles have reached the tip of the syringe. You may need to gently tap the syringe. Push the air out of the syringe, cap the syringe, replace it into the ice and transfer it to the Coring 158 ABG analyzer.
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