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The Effects of a Cervical Collar in the Treatment of Apnea of Prematurity

by

Denise Florence Munro

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Nursing

Faculty of Nursing

Edmonton, Alberta

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Dr. Louise Jensen, Thesis Supervisor

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March 26, 2002

Abstract

Apnea of prematurity is a common infant problem in the Neonatal Intensive Care Unit (NICU). Due to the decreased muscle tone of preterm infants' upper airway, apnea is frequently induced by neck flexion leading to upper airway obstruction. The purpose of this study was to examine the effects of a cervical collar on the incidence of apneic episodes and negative physiologic sequelae among preterm infants receiving a means of ventilatory support. A within-subject, repeated measures design was used with 16 neonates. No statistically significant difference in the incidence of apneic episodes with the collar when compared to the control period was found. There may have been a clinical intolerance to the cervical collars as shown by the increasing number of bradycardic and oxygen desaturation episodes, necessitating discontinuation of the study. However, no conclusions can be made as some neonates also deteriorated during the control period.

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CHAPTER ONE

Introduction

Apnea of prematurity is the most common occurring respiratory event in infants less than 37 weeks gestation (Grisemer, 1990). According to Blanchard and Aranda (1986), approximately 25% of preterm infants experience apnea and more than 80% whose birthweight is less than 1,000 grams. Apnea of prematurity occurs in the first day of life in all infants less than 34 weeks gestational age (Barrington & Finer, 1991). Apneic episodes of greater than 15 seconds duration have also been reported to occur every hour in infants less than 32 weeks gestation during the first week of life (Henderson-Smart, 1981). Only when infants reach a post-conceptional age of 37 weeks do apneic episodes become a rare occurrence (Henderson-Smart, 1981).

Apnea refers to any episode of the cessation of airflow (lasting 3 to 20 seconds) with or without respiratory effort, and may be of a central, obstructive, or mixed origin (Stark & Thach, 1976; Dransfield, Spitzer, & Fox, 1983; Barrington & Finer, 1991; Ruggins, 1991; Calhoun, 1996; Jenni et al., 1996; Lemke et al., 1998). The type of apnea is classified according to the presence or absence of airway obstruction. However, there is new evidence that all three types of apnea may have an obstructive component involving the easily collapsible upper airway, suggesting that all types of apnea may be due to a mixed component (Mathew, Roberts, & Thach, 1982; Ruggins, 1991; Stark, 1991; Thach, 1992; Holditch-Davis, Edwards, & Wigger, 1994; Lemke et al., 1998). Investigators have also found that the airway obstruction may be induced and aggravated by neck flexion (Thach & Stark, 1979; Wilson, Thach, Brouillette, & Abu-Osba, 1980; Dransfield et al., 1983; Ruggins & Milner, 1991). Several authors have examined neck flexion as an

antecedent to apnea in neonates (Stark & Thach, 1976; Jenni et al., 1997); however, to date, little research on interventions for neck flexion-induced apnea has been reported (Thach & Stark, 1979). Therefore, the use of a cervical collar, as a proposed intervention, should be examined in the neonatal population.

Purpose of the Study

The primary purpose of this study was to examine the relationship between the therapeutic use of a cervical collar on the incidence of apneic episodes among preterm infants receiving continuous positive airway pressure (CPAP) or high flow nasal cannula (HFNC), whose gestational age is 32 weeks or less, and birthweight is less than or equal to 1500 grams (Ballard et al., 1991). In addition, the effects of the cervical collar on two physiologic correlates (bradycardic and hypoxic episodes) were investigated.

The hypotheses tested in this study were as follows:

Primary Hypothesis:

1. The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the overall frequency of apneic episodes as measured by heart rate, respiratory impedance, and oxygen saturation levels when compared to the frequency of apneic episodes experienced during the control period.

Secondary Hypotheses:

1. The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the number of isolated bradycardic episodes experienced without apnea and significant oxygen desaturation episodes, when compared to the control period.

2. The use of a cervical collar in preterm infants receiving CPAP or HFNC will

significantly decrease the number of isolated oxygen desaturation episodes experienced without significant bradycardia or apnea, when compared to the control period.

3. The use of a cervical collar in preterm infants receving CPAP or HFNC will significantly decrease the number of combined isolated bradycardic and oxygen desaturation episodes, when compared to the control period.

4. The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the number of apneic and bradycardic episodes when compared to the control period.

5. The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the number of apneic and oxygen desaturation episodes when compared to the control period.

6. The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the number of combined apneic, bradycardic and oxygen desaturation episodes when compared to the control period.

Significance of the Study

This research involved studying a new clinical strategy for the treatment of apnea of prematurity for infants less than 32 weeks gestational age. Apnea of prematurity is an extremely common problem in the NICU and it is a significant contributor to infant morbidity and mortality. Although there are current treatments for apnea, there is not one efficacious intervention for apneic episodes induced by upper airway obstruction and neck flexion. A significant part of nursing care in the NICU involves stimulating, repositioning, and arousing preterm infants from the physiologic deterioration resulting

from apneic episodes. With the effective use of a cervical collar, nurses' handling of the preterm infant may be reduced, stimulation and repositioning of the infants decreased, and therefore more developmentally appropriate care may be applied.

CHAPTER TWO

Review of the Literature

Apnea of Prematurity

Apnea not only causes airflow cessation, but it may also be accompanied by a number of negative physiologic correlates including bradycardia, oxygen desaturation, and fluctuation in pulse and blood pressure (Levitt, Mushin, Bellman, & Harvey, 1988; Finer, Barrington, Hayes, & Hugh, 1992; Miller & Martin, 1992; Lemke et al., 1998; Urlesberger, Kaspirek, Pichler, & Muller, 1999). Due to their immaturity, decreased lung reserve, and subsequently frequent chronic lung disease development, preterm infants are more prone to apnea and its negative physiologic consequences (Finer et al., 1992; Miller & Martin, 1992). For these reasons, apnea of prematurity may lead to prolonged hospital stays, medication therapy, dependency on ventilation assistance; and possibly a less than optimal neurodevelopmental outcome; and thus, increased healthcare resource usage (Perlman & Volpe, 1985; Levitt et al., 1988; Miller & Martin, 1992; Abbey, Cooper, & Kwentus, 1989; Calhoun, 1996; Jenni et al., 1996). Despite many interventions for the treatment of apnea, it is a medical condition that continues to persist in the preterm population. It is therefore imperative that the incidence of apnea of prematurity be reduced to a minimum.

Much of the therapy involved with preterm infants is the treatment and management of apnea. Nurses, as the primary caregivers, have many responsibilities when caring for infants with apnea of prematurity (Peters, 1992). They must be able to accurately assess the infant and be competent in detecting any changes in heart rate and respiration (Grisemer, 1990). A large proportion of their role also involves appropriate stimulation

and resuscitation of the infant from apneic episodes. Nurses can help to reduce the chance of apneas by providing the infants with proper neck positioning, thereby reducing the chance of airway obstruction. Another nursing intervention that helps to reduce apneic episodes in preterm infants includes the promotion of a stable physiologic state, achieved by limiting the amount of their handling and providing the infants with a quiet, dim environment (Grisemer, 1990; Lawhon, 1996; Peters, 1999). These interventions reduce the disturbance of the infants, resulting in a more stable physiologic state.

Apnea Detection and Duration

Although, healthcare personnel are able to detect apneic episodes, not all apneic episodes are identified and appropriately classified without the use of monitoring devices (Muttitt, Finer, Tierney, & Rossman, 1988; Poets, Stebbens, Richard, & Southall, 1995). Continuous noninvasive monitoring of heart rate, oxygen saturation, airway patency, by way of exhaled carbon dioxide (CO_2) via an end-tidal catheter or nasal thermistor, and respiratory effort, by way of respiratory indices, is required for reliable, accurate recordings and classification of all apnea types (Anderson, Martin, Lough, & Martinez, 1982; Reed, Roberts, & Thach, 1985; Muttitt et al., 1988; Curley & Thompson, 1990; Jenni et al., 1996). However, not all of these devices are available to care givers, nor do they function effectively under all therapeutic circumstances. End-tidal CO_2 has proven to be ineffective with the administration of nasal air pressure (Kobayashi, Katayama, & Miyasaka, 1992), and the nasal thermistor does not record accurately in the thermal environment of the isolette and has proven to be variable in detection of apneas (Weese-Mayer et al., 2000).

A significant difference of opinion exists amongst researchers concerning the

definition of apnea by duration. Apneic episodes have been defined as an episode of absent airflow lasting 3, 5, 10, 15, or 20 seconds (Mathew et al., 1982; Dransfield et al., 1983; Barrington & Finer, 1990; Lemke et al., 1998). It has also been interpreted as pathologic when the cessation of respirations occurs for greater than or equal to 20 seconds, or for less than 20 seconds when accompanied by a significant desaturation or bradycardia (Barrington & Finer, 1991). Other researchers have defined apnea according to its respiratory pattern (Stark & Thach, 1976; Grosswaser et al., 1995). Some have reported "periodic breathing" (a respiratory pattern of apnea, involving three or more respiratory pauses, greater than or equal to 3 seconds, with intervals of breathing between pauses that are less than 20 seconds) as part of the apnea profile (McNamara & Sullivan, 1996; Lemke et al., 1998), while others consider it a normal pattern exhibited by preterm infants that does not require extra consideration or intervention. The length of time and the duration of apnea researchers choose will have a substantial effect on research results. Lemke et al. (1998) defined apnea as a respiratory pause of $\geq$ 3 seconds, and 4977 apneic episodes were recorded in 42 subjects, each subject being studied for 2-3 hour periods; whereas Finer et al. (1992) defined apnea as any episode $\geq$ 15 seconds, and 2082 episodes were reported in 47 infants, each infant studied for a 12 hour period. The definition of apnea researchers choose will obviously affect the frequency of detected and reported apneic episodes, thus compounding difficulties in interpreting results across studies. (Mathew et al., 1982; Barrington & Finer, 1991; Barrington, Finer, & Li, 1996; Lemke et al., 1998).

Apnea Types

Apnea of prematurity can be classified as central, mixed, or obstructive

(Dransfield et al., 1983; Jenni et al, 1996; Lemke et al., 1998). Central apnea occurs when there is absence of airflow and respiratory effort (Ruggins & Milner, 1991; Holditch-Davis et al., 1994; Lemke et al., 1998). Obstructive apnea results when airflow is absent, but respiratory effort continues, although it is frequently disrupted in pattern and intensity (Calhoun, 1996; Jenni et al., 1996; Lemke et al., 1998). Mixed apnea contains elements of both the central and obstructive apneas (Barrington & Finer, 1991). It results from a central respiratory cessation that either follows or precedes an obstructive event (Calhoun, 1996, Lemke et al., 1998). In a study of 47 infants at less than 34 weeks gestation, Finer et al. (1992) reported that a gradual decrease in heart rate interrupted with accelerations was the common pattern found in all apnea types. However, they also noted that while significant oxygen desaturation episodes occurred with all types of apnea, bradycardic episodes (defined as a heart rate decrease to less than 100 beats per minute) occurred more frequently with central apneas.

Pathophysiologically, central apnea is secondary to the immature development of the preterm infant's central nervous system (Barrington & Finer, 1991; Holditch-Davis et al., 1994). Central apnea occurs less frequently (approximately 20-40%) in preterm neonates compared to obstructive and mixed apneas (Miller, Carlo, & Martin, 1985; Ruggins & Milner, 1991; Idiong et al., 1998; Lemke et al., 1998). Obstructive apnea is related to anatomical factors, such as hypotonic muscles of the neonatal upper airway (Thach & Stark, 1979; Thach, 1992; Mathew et al., 1982; Lemke et al., 1998). A purely obstructive apneic episode is rare, but has often been associated with movements or attributed to external obstructions, such as occlusion by a pillow, blanket, or bed mattress (Thach & Stark, 1979; Ruggins & Milner, 1991; Lemke et al., 1998). Lemke et al. (1998)

studied 42 preterm infants and reported that 208 of 4977 (4%) apneic episodes were obstructive. Similarly, in the study by Thach and Stark (1979) involving eight preterm infants, 4 out of 43 (9%) apneic episodes were obstructive in origin. Finally, mixed episodes are the most common occurring apnea in preterm infants (Mathew et al., 1982; Ruggins & Milner, 1991; Lemke et al., 1998). In a descriptive study of 76 preterm infants, Dransfield et al. (1983) reported that 68% of the 433 recorded apneic episodes were of mixed origin. As well, in a descriptive study of nine preterm infants, 70 of the 105 (66.6%) apneic episodes were from a mixed source (Mathew et al., 1982). In both studies, mixed apneas were detected more frequently due to the improved monitoring techniques of apnea detection and upper airway involvement.

Underlying Causes of Apnea

There are many underlying causes and mechanisms of apnea. Apnea may be a manifestation of an underlying disease process, such as sepsis, temperature instability, maternal drug ingestion, metabolic disorders, neurological complications, and/or abdominal distension (Grisemer, 1990). However, apnea of prematurity results from causes other than an underlying disease state (Grisemer, 1990; Miller & Martin, 1992). The developmental immaturity of the preterm neonates' central nervous system and respiratory drive are major contributors to apnea of prematurity, as are their suppressed ventilatory responses to chemoreceptor and mechanoreceptor information (Miller & Martin, 1992; Stark, 1991). The triggering of the Hering-Breuer inspiratory inhibitory reflex, initiated by the pulmonary stretch receptors, may also be a factor in respiratory cessation (Stark & Thach, 1976; Thach & Stark, 1979; Hannam, Ingram, & Milner, 1998; Lemke et al., 1998). In addition, preterm infants have a more compliant chest wall and

decreased muscle tone in the upper airway, as well as poorly coordinated diaphragmatic muscle movements; all factors which predispose them to respiratory disruption (Thach & Stark, 1979; Grisemer, 1990; Miller & Martin, 1992). Current research has suggested that infants are unable to appropriately manage a stressful environment, which in turn renders them susceptible to disorganized and negative responses (Ruggins, 1991; Lawhon, 1996; Peters, 1999). Sleep states and positioning are associated with apnea of prematurity (Catlett & Holditch-Davis, 1990; Gaultier, 1990; Stark, 1991; Holditch-Davis et al., 1994). The underlying causes of apnea are outline in Figure 1.

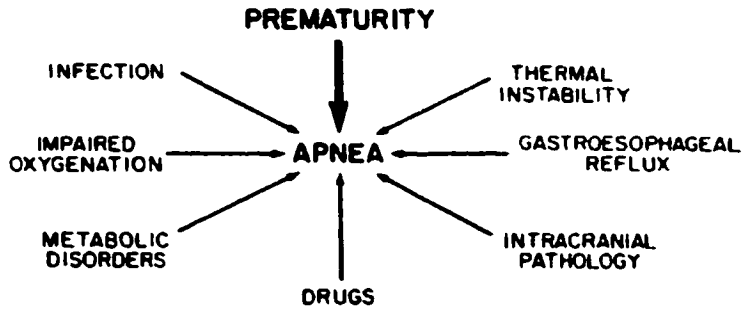

Figure 1. Precipitating Factors of Apneic Episodes in Preterm Infants.
From: "Pathogenesis of Apnea in Preterm Infants" by R. J. Martin et al., 1986, Journal of Pediatrics, 109, p.733. Adapted with permission of the author.

Environment

The environment of the NICU is often overwhelming, foreign, and stressful for the vulnerable, continually developing preterm infant (Wolke, 1987; Catlett & Holditch-Davis, 1990; Grisemer, 1990; Peters, 1999). Preterm infants are exposed to an unfamiliar environment of excessive noise and light; and excessive, and/or inappropriate

handling (Orr et al., 1985; Catlett & Holditch-Davis, 1990; Peters, 1992; VandenBerg, 1993). Due to the immaturity and limited physiologic reserves of preterm neonates, they have a diminished tolerance to these environmental stressors and their neurobehavior becomes disorganized and fatigued, demonstrating unstable physiologic responses (Catlett & Holditch-Davis, 1990; Oehler, Strickland, & Norlund, 1991; VandenBerg, 1993; McNamara & Sullivan, 1996; Peters, 1999). As a result, they experience an increased number of apneic, bradycardic and hypoxic episodes, which places them at risk for intraventricular hemorrhage, cerebral ischemia, injury to the developing brain, and a poor neurodevelopmental outcome (Perlman & Volpe, 1985; Wolke, 1987; Anthony, Edwards, & Levene, 1993; VandenBerg, 1993; Als et al., 1994; Koons et al., 1994).

Neonatal nurses play a significant role in reducing the effects of the stressful environment to which the infants are exposed (Catlett & Holditch-Davis, 1990). Strategies include reducing noise and light levels, and modifying tactile stimulation (Catlett & Holditch-Davis, 1990). By limiting environmental stimulation and excessive handling, neonatal nurses provide infants with developmentally supportive care (Lawhon, 1996). It is postulated that developmentally supportive care may help reduce the incidence of apnea of prematurity, in addition to improving the infants' neurobehavioral outcome (Lawhon, 1996; Peters, 1999). However, this has yet to be established under controlled circumstances (Tyebkhan, Peters, McPherson, & Coté, 1999).

Sleep State

Sleep state also plays a role in apnea of prematurity (Holditch-Davis et al., 1994). Changes in sleep state have a major impact on breathing in the newborn (Stark, 1991). In active, rapid-eye-movement (REM) sleep, the infant experiences irregular breathing,

whereas in quiet, non-REM sleep, the infant's respiratory pattern is regular (Stark, 1991). Preterm infants spend up to 65% or more of their time in active sleep, during which they experience more apnea and irregular breathing patterns (Martin, Miller, & Carlo, 1986; Marchal, Bairam, & Vert, 1987; Stark, 1991). As the infant's gestational age increases and neurological and physiologic development matures, the infant spends more time in quiet sleep and as a result apnea frequency and irregular respiratory patterns significantly decrease (Stark, 1991; Holditch-Davis et al., 1994; Holditch-Davis & Edwards, 1998).

Body Positioning

Different positions precipitate apnea (Reed et al., 1985; Levene & McKenzie, 1989; Ruggins, 1991; Stark, 1991). Infants tend to experience fewer apneic episodes in the prone position versus the supine position (Levene & McKenzie, 1990; Stark, 1991; Heimler, Langlois, Hodel, Nelin, & Sasidharan, 1992; Jenni et al., 1997). The prone position supports the abdomen and the compliant rib cage and as a result, improves tidal volume, and decreases asynchronized, sporadic respiratory effort (Wagaman et al., 1979; Gaultier, 1990; Kurlak, Ruggins, & Stephenson, 1994; Jenni et al., 1997). Kurlak et al. (1994) studied 35 preterm infants in both the supine and prone positions, each for a two-hour period, and found that significantly greater central ($p=0.025$) and mixed ($p= 0.012$) apneic episodes occurred when infants were placed in the supine position. Other investigators, however, have reported no difference in apnea frequency in the supine and prone positions (Orr et al., 1985; Barrington, Finer, & Wilkinson, 1987; Goto, Maeda, Mirmiran, & Ariagno, 1999).

Bed Position

Jenni et al. (1997) studied 12 preterm infants less than 31 weeks gestation in a prone,

head elevated tilt position. The infants were randomly placed in a prone, head elevated tilt position (15 degrees) or a prone horizontal position. A significant reduction of 48.5% ($p < .01$) of bradycardic and hypoxemic episodes was found in the head-elevated-tilt position. A concern the authors identified is the possibility that head elevation, if performed rapidly, could impact cerebral perfusion and oxygen supply (Anthony et al., 1993).

Upper Airway Obstruction

Upper airway obstruction is a major component of apnea of prematurity (Pickens, Schefft, & Thach, 1988; Jenni et al., 1996). The obstruction has been attributed to the decreased muscle tone of the preterm infant's upper airway (Dransfield et al., 1983). This hypotonia leads to increased mobility of the upper airway, which results in its tendency to collapse, forming an obstruction (Thach & Stark, 1979; Ruggins & Milner, 1991). Some researchers have identified the site of the obstruction as the pharynx (Stark & Thach, 1976; Wilson et al., 1980), while others have identified it as the larynx (Ruggins & Milner, 1991). In a descriptive study of nine preterm infants, pharyngeal pressure was measured via a pharyngeal catheter, during both respiratory effort and apnea (Mathew et al., 1982). It was inferred from the pharyngeal pressure changes that the site of obstruction was in the high pharyngeal area. A problem with this study was the actual measurement method used. The authors reported occasional obstruction of the catheter, rendering it inoperative.

Ruggins and Milner (1991), using an ultra fine fiberoptic scope, continuously examined the upper airways of 12 preterm infants for a mean length of time of 43 minutes. A non-patent larynx during mixed and obstructive apneas was found. The upper

airway obstruction at the vocal cords during central apneas was also noted. However, it has been established that the introduction of the fiberoptic scope itself may have induced glottic closure (Finer & Etches, 1989; Ruggins, 1991).

Lemke et al. (1998) noted laryngeal obstruction in their study of 41 preterm infants, all less than 34 weeks gestation. The infants were studied using a new, highly sophisticated monitoring technique of cardiac airflow oscillation observed in an amplified respiratory flow tracing. Central apneas were detected when the oscillation was present, obstructive apneas occurred when no oscillation was detected, and mixed episodes were recorded when the oscillation occurred during part of the apnea. With this method, the transmission of cardiac airflow oscillation indicates airway patency. Surprisingly, a significant number of central apneas (13%, $p< 0.05$) were associated with decreased oscillation, suggesting airway instability. Nasal airflow and respiratory effort recordings confirmed this conclusion.

Neck Flexion

Upper airways muscles, in particular the genioglossus and those of the pharyngeal wall, are more hypotonic in preterm infants and therefore predispose them to obstruction with neck flexion (Stark & Thach, 1976). Thach and Stark (1979) proposed that neck flexion may be the main cause of upper airway closure in neonates. In their descriptive study, eight preterm infants less than 33 weeks gestational age, were monitored in three different positions, the supine position with unflexed neck and the sitting position with neck flexed and neck unflexed. Respiratory effort was detected by esophageal pressure changes, airflow was monitored via an end-tidal CO_2 catheter, and heart rate was recorded via an electrocardiogram (ECG). The findings revealed that the infants had

statistically more apneic episodes in the sitting-flexed position. It was concluded that when spontaneous neck flexion occurred, the hypotonic pharyngeal wall musculature collapsed and the tongue was displaced posteriorly, which resulted in complete obstruction of the upper airway. Maintaining the infant's head in a neutral or slightly extended position is the suggested treatment to prevent this occurrence (Stark & Thach, 1976; Gleason et al., 1983; Reed et al., 1985; Thach, 1992; Reiterer, Abbasi, & Bhutani, 1994; Makofsky, 1997; Lemke et al., 1998). Figure 2 illustrates the correct neck position for the neonate, as well as two incorrect positions.

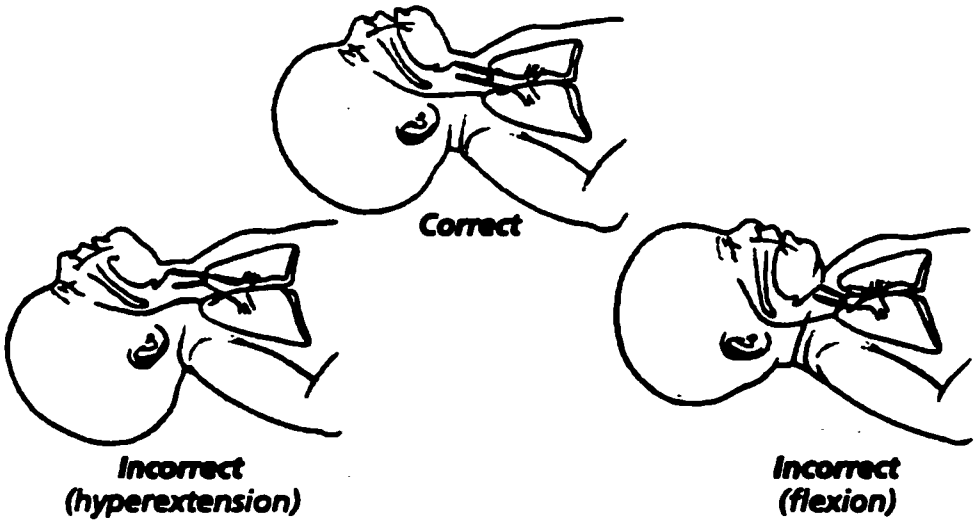

Figure 2. Neck Positions in the Supine Neonate.

From: Neonatal Resuscitation Textbook (4th ed.) (p.2-6), by the National Neonatal Resuscitation Program Steering Committee, 2000, American Heart Association, American Academy of Pediatrics. Adapted with permission from the author.

Treatment of Apnea

Pharmocologic Therapy

Many forms of treatment are used for apnea of prematurity and its negative physiologic correlates. Treatment varies according to the cause of the apnea, the type of apnea, the severity of the apneic episodes, the gestational age of the infant, and the location of the neonate (i.e., level of care provided). The more preterm the infant and the more severe the apneas, the greater the number of interventions required. Current forms of treatment for central apnea include the pharmacologic therapy of methylxanthines, caffeine, and/or doxapram (Jamali, Coutts, Malek, & Peliowski, 1991; Calhoun, 1996; Poets, Darraj, & Bohnhorst, 1999). Methylxanthines, such as theophylline, and caffeine, work as central nervous stimulants and smooth muscle relaxants, whereas doxapram is a respiratory stimulant (Finer, Peters, Duffley, & Coward, 1984; Calhoun, 1996; Lee, Charles, Steer, Flenady, & Grant, 1996; Young & Mangum, 1998; Erenberg et al., 2000). However, aminophylline, caffeine, and doxapram are not without side effects. Adverse effects from caffeine are rare, but may include restlessness and vomiting; whereas theophylline toxicity can result in central nervous system irritability, tachycardia, hyperglycemia, and jitteriness (Calhoun, 1996; Young & Magnum, 1998). The adverse effects of doxapram occur quite rarely in neonates, however if a high dosage is administered ($\geq$ 1.0 mg/kg/hr), irritability of the central nervous system, jitteriness, tachycardia, as well as seizures and hypertension have been documented (Bairam, Faulon, Monin, & Vert, 1992; Finer et al., 1992; Young & Mangum, 1998; Poets, Darraj, & Bohnhorst, 1999). Although methylxanthines are efficacious in decreasing apnea frequency, apneic episodes and concurrent episodes of negative physiologic correlates

continue to occur despite this intervention (Muttitt, Tierney, & Finer, 1987).

Oscillating Beds

Oscillating beds have also been used to treat central and obstructive apneas (Jones, 1981; Groswasser et al., 1995; Svenningsen, Wittstrom, & Hellstrom-Westas, 1995). In a study of eight preterm infants, it was suggested that obstructions were significantly reduced to a median of 1.8 episodes per hour from 2.5 ($p = .034$). This was attributed to the stimulation of the vestibular apparatus, which may have induced automatic responses in the dilating muscles of the upper airway (Groswasser et al., 1995). Other researchers have not supported these findings and have found that the oscillating beds have been proven ineffective for the prevention of apnea (Saigal, Watts, & Campbell, 1986; Osborn & Henderson-Smart, 2000).

Endotracheal Intubation

Various treatments exist for mixed and obstructive components of apneas (Calhoun, 1996). For severe mixed and obstructive apneas, in which the infant experiences excessive negative physiologic effects, (e.g., profound and/or prolonged bradycardia and/or hypoxic episodes along with cessation of respiratory effort and/or airway closure), positive pressure is the suggested form of treatment (Gitterman, Fusch, Gitterman, Regazzoni, & Moessinger, 1997). In this case, positive pressure is delivered following endotracheal intubation by mechanical ventilation (Gitterman et al., 1997). The endotracheal tube is positioned in the upper trachea of the infant thus providing a physical splint to prevent the collapse of the hypotonic upper airway musculature. However, endotracheal intubation is maintained only in level III nurseries (i.e., NICUs). It is also known to be a highly invasive procedure associated with stress and trauma to the

infant (Barrington, Finer, & Etches, 1989). The procedure may also place the infant at risk for developing disorders of the upper airway (tracheomalacia, subglottic stenosis) as well as chronic lung disease (Benjamin, Thompson, & O'Rourke, 1990; Page, Giehl, & Luke, 1998). Infants may still develop apneic episodes when ventilated if the mechanical breaths are set at a low frequency (Page et al., 1998).

Positive Pressure Ventilation

By far, the most frequent method of treatment for apnea of prematurity, related to mixed and obstructive sources, is continuous positive airway pressure (CPAP). CPAP therapy is also limited to level III nurseries because of the acuity level of the infants who require this form of treatment. CPAP, which is delivered by nasal prongs, nasal mask, nasal cannula or a nasopharyngeal tube, maintains the patency of the upper airway by delivering air and oxygen to the infant's airway under pressure. (Engelke, Roloff, & Kuhns, 1982; Abbey, et al., 1989; Higgins, Richter, & Davis, 1991; Lin, Wang, Lin, & Yeh, 1998; Sreenan, Lemke, Hudson-Mason, & Osiovich, 2001). The increased airway pressure overcomes the negative pharyngeal pressure induced by inspiration (Engelke et al., 1982; Abbey et al., 1989). It also acts as a splint for the upper airway, preventing its collapse (Higgins et al., 1991; Kurz, 1999; McNamara & Sullivan, 1999; Sreenan, Lemke, & Hudson-Mason, 2001). In a non-randomized study of 14 preterm infants less than 32 weeks gestation, Miller et al. (1985) reported that a nasal mask delivering CPAP significantly reduced obstructive and mixed apneas ($p < 0.01$). Airflow was monitored via a modified nasal mask pneumotachometer, respiratory effort was measured via mercury strain gauges, and heart rate was recorded via an ECG. Measured responses of the infants were taken for three sequential 45-minute periods with, without, and again with the

treatment of CPAP. A concern with this study is that the infants were not randomized and the size of the sample was small.

Although CPAP has resulted in effective treatment for reducing apneic episodes in preterm infants, they continue to occur, thus leaving infants at continued risk for immediate, short (hypoxic and bradycardic episodes), and long-term (ventilation dependency and possibly poor neurological outcome) negative sequelae (Kurz, 1999). Another disadvantage of CPAP therapy is that it is difficult to ensure that adequate pressure is reaching the infant's upper airway (Higgins et al., 1991). The infant's movements and subsequent displacement of the device, as well as the opening and closing of the infant's oral cavity may result in a loss of air pressure (Grisemer, 1990; Higgins et al., 1991). CPAP irritates the nares and the entire delivery system adds to overall infant discomfort (Peters, 1989). Thus, during CPAP therapy, infants often require sedation (Higgins et al., 1991). Another drawback to CPAP is that it may also cause abdominal distension, which may result in feeding difficulties or the infant not being fed enterally at all (Miller et al., 1985). Infants who cannot effectively be fed enterally must have nutrition supplied parenterally, increasing the risk for infection and trauma, and reducing intravenous access sites.

Nasal Intermittent Positive Pressure Ventilation

A similar respiratory support to CPAP is the delivery of nasal intermittent positive pressure ventilation (NIPPV) (Lemyre, Davis, & De Paoli, 2000). It is a means of support administered via nasal prongs or a nasopharyngeal tube. NIPPV delivers not only positive distending airway pressure, but also a low rate of mechanical ventilations that are synchronized with the infant's respirations (Lemyre, Davis, & De Paoli, 2000). Very few

studies have been performed on this type of treatment, however it is an effective support in the older children and adult populations (Lemyre, Davis, & De Paoli, 2000).

Oxygenation

Another form of therapy for central apnea is the avoidance of hypoxemia (Stark, 1991; Miller & Martin, 1992). When preterm infants are faced with a hypoxic challenge, they will initially respond with hyperventilation, followed by a subsequent drop in respirations and episodes of apnea and periodic breathing (Stark, 1991). The hypoxemia is thought to induce a central respiratory depression (Miller & Martin, 1992), thus by eliminating hypoxemia, the incidence of central apneas will be reduced.

Positioning

A current noninvasive intervention for upper airway obstruction includes infant positioning. Placing the infant's head in a slightly extended position has reduced the frequency of obstruction by preventing neck flexion and the collapse of the hypotonic muscles of the upper airway (Stark & Thach, 1976; Thach & Stark, 1979; Wilson et al., 1980; Grisemer, 1990). In an effort to ensure proper positioning of the head and neck relative to the torso, neck rolls have been placed under the infant at the cervical level (Dransfield et al., 1983; Miller et al., 1985; Grisemer, 1990). This positioning, however, is difficult to maintain as infants may be restless and experience an activity level change with sleep-wake cycles (Reed et al., 1985; Grisemer, 1990; Stark, 1991; Thach, 1992). Despite years of effort and research, treatment for mixed and obstructive components of apnea continues to remain problematic.

Cervical Collars

There has been minimal research on neck flexion and upper airway obstruction as

etiological antecedents to apnea. It is proposed that the use of a cervical collar may be helpful by keeping the head in a slightly extended position relative to the trunk, thus decreasing the likelihood of inadvertent neck flexion (Dodd, Simon, McKeown, & Patrick, 1995; Jacques & Karmel-Rose, 1997). In a descriptive study, Dodd et al. (1995) evaluated the use of cervical collars and their effect on the maintenance of upper airway patency in 38 anaesthetized adults. Tidal volume, the volume of gas inspired with each breath, was measured via a facial mask as a means of detecting airway obstruction. It was theorized that if tidal volume was unchanged, airway obstruction was not suspected. If, however, it was decreased, some degree of airway obstruction was thought to have occurred. During the collar phase of the study, no statistically significant difference in tidal volume, as measured by a facial mask, was noted in 33 patients. However, complete airway obstruction occurred in five of the subjects. While this study is the first of its kind, considerable design and demographic issues interfere with the credibility of the results. The researchers used broad inclusion criteria; resulting in an extremely heterogeneous sample of 23 women and 15 men, all requiring minor surgery, age ranging from 17-79 years. This heterogeneous sample does not control for extraneous variables that may have skewed the results of the study, such as age, size, and individual responses to anesthetic agents. In addition, the collars were not custom-made and came in only three sizes, fitting each patient very differently. A large fitting collar caused complete upper airway obstruction in four of the subjects by compressing the mandible posteriorly. Finally, the placement of the facial mask, used for measuring tidal volume, may itself have induced upper airway obstruction if too much pressure was applied on the mandible, resulting in inappropriate neck flexion.

Collars have also been used with infants 4 months of age and older for the treatment of torticollis secondary to the shortening of the sternocleidomastoid muscle (Jacques & Karmel-Ross, 1997). Such collars have been custom-made out of plastic or foam for each patient. They have been efficacious in this cohort in improving the infant's stability to hold his or her head midline. It is postulated that this collar intervention, using the foam prototype, could be adapted for use with the preterm infant and be used in conjunction with CPAP or HFNC to reduce apneic episodes. Figure 3 illustrates the foam collar utilized in older infants with torticollis.

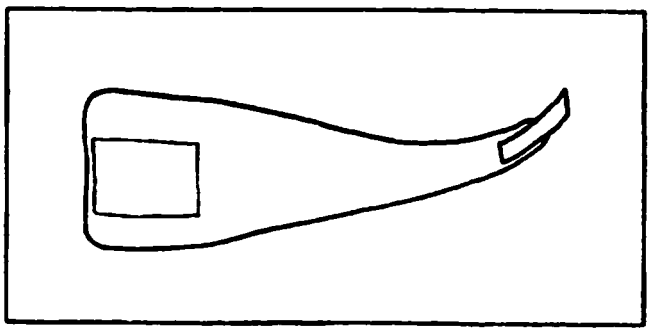

Figure 3. The Foam Collar Used in the Treatment of Older Infants with Torticollis From: "The Use of Splinting in Conservative and Post-Operative Treatment of Congenital Muscular Torticollis" by C. Jacques and K. Karmel-Ross, 1997, Physical and Occupational therapy in Pediatrics, 17(2), p.85. Adapted with permission from the author.

Summary

From the research that has been done, it is apparent that to some degree airway collapse is present in virtually all apneic episodes. The classical division of central, obstructive, and mixed apnea may be somewhat inaccurate, as it appears that upper

airway obstruction may be a common underlying pathophysiologic mechanism contributing to or occurring with all apnea (Wilson et al., 1980; Ruggins & Milner, 1991; Lemke et al., 1998). Researchers to date have focused on the frequency of apnea induced by neck flexion and upper airway obstruction, but there has been no research performed on any treatment for these types of apneas. In many of the studies, researchers have reported placing the infant's neck in a neutral or slightly extended position, while in others it was either not reported or included as a variable (Heimler et al., 1992; Kurlak et al., 1994; Jenni et al, 1997). It is therefore imperative that more research is done where focus is placed on a specific intervention for neck flexion-induced apneas, such as the use of a cervical collar.

CHAPTER THREE

Method

Design

A within-subject, repeated measures design was used to examine the effects of the use of a cervical collar on apneic episodes in preterm infants who were admitted to the Royal Alexandra Hospital, Level III Neonatal Intensive Care in Edmonton, Alberta. This design allowed each subject to serve as his/her own control. The independent variable was the cervical collar, the dependent variables included the frequency and severity of apneic, bradycardic and hypoxemic episodes, and the process variable was the fraction of inspired oxygen (FiO_2). Prior to the initiation of the study, two subjects participated in a pilot study. No problems with the procedure, the method, or the data collection process were identified; therefore, no changes were made.

Each recording period was divided into five intervals (see Figure 4): (a) an initial 3 hour time period to obtain baseline measurements; (b) a 15 minute time period for placement of the collar and to allow the infant to settle; (c) intervention: a 6 hour period measuring the effects of the collar; (d) a 15 minute period for removing the collar and once again allowing the infant to settle; (e) and control: a 6 hour period of measurements without the collar. Baseline was defined as the period of time of 180 minutes in which the infant was monitored in his/her environmental setting for the frequency and severity of apneic episodes, bradycardia, and/or clinically significant oxygen desaturation episodes before the introduction of intervention and control periods. The baseline period provided the researcher with information of the infant's physiological status prior to the introduction of treatment or control.

The order in which the intervention was introduced was randomly assigned to either precede or follow the control period, thus creating two treatment order protocols. The treatment order group was determined by opening a sealed, prepared envelope containing the order code once parental consent was obtained. This permitted a time plan to be generated and the necessary equipment and personnel to be on site when required. The envelopes consisted of equal numbers for each treatment order protocol. For consistency, all infants were studied while on CPAP or HFNC, and while in the prone position.

Duration	3 hours	15 minutes	6 hours	15 minutes	6 hours
	Baseline	Collar Placement	Intervention	Collar Removal	Control

Duration	3 hours	15 minutes	6 hours	15 minutes	6 hours
	Baseline	No action	Control	Collar placement	Intervention

Figure 4. Treatment Order Sequences.

Definition of Terms

Apnea was defined as an isolated respiratory pause of greater than 20 seconds, or greater than 10 seconds if accompanied by a bradycardic and/or a clinically significant oxygen desaturation episode (Mathew et al., 1982; Finer et al., 1992).

Oxygen desaturation was defined as any decrease in the subject's oxygen saturation level less than 88% (Barrington & Finer, 1991; Peters, 1998). As per the neonatal research office sleep study guidelines, a desaturation must last for a period of greater

than or equal to 10 seconds to be deemed clinically significant.

Bradycardic episode was defined as any decrease in heart rate level equal to or less than 30% from the subject's mean heart rate level measured for 60 seconds prior to the event.

Respiratory impedance was defined as the method of measuring the respiratory effort, which yield a visual respiratory waveform from which apneic episodes will be calculated.

Fraction of inspired oxygen (FiO_2) was defined as the ambient oxygen concentration.

Sample

A convenience sample of preterm infants admitted to the NICU at the Royal Alexandra Hospital was studied. The subjects were selected based on the availability of the necessary equipment and staff, as well as the availability of this particular patient group. Infants who were withdrawn from the study due to their parents' wishes, equipment malfunction, or unstable medical condition were replaced.

Inclusion criteria for the sample were:

1. informed consent given by mother or both parents when possible.
2. infants less than 32 weeks gestational age (as determined by fetal ultrasound and Ballard physical examination) (Ballard et al., 1991) with an appropriate-for-gestational-age birthweight of less than 1,500 grams.
3. at the time of the study, the infants were extubated, were hemodynamically stable, were on nasal CPAP for at least 3 days, were receiving methylxanthines and/or doxapram, and if on methylxanthines, had a therapeutic methylxanthine serum level (< 3 days since last dosage change or loading dose) of approximately 70 umol/L or greater (Muttitt et al., 1987).

Infants excluded from this study were those having confirmed or suspected sepsis (documented as temperature instability, feeding intolerance, increasing number of apneic, bradycardic, and oxygen desaturation episodes), greater than a grade II cerebral hemorrhage (Papile, Burnstein, & Burnstein, 1978), hemodynamically significant patent ductus arteriosus, necrotizing enterocolitis, and major congenital or chromosomal anomalies.

A clinically significant difference was defined as a decrease in apneic episodes by at least 50%. Therefore, with an alpha of 0.05, a power of 0.80, and an estimated standard deviation of 40%, a sample size of 20 was estimated for investigation of the primary hypothesis (Jandel Scientific, 1994). Enrollment was stopped due to increasing trends in the dependent variables suggesting possible adverse effects from the cervical collar. Overall, 16 infants were studied. Eleven infants underwent a 15 hour and 30 minute recording session during which measurements of the heart rate, respiratory impedance, oxygen saturation levels, and FiO_2 requirements were continuously monitored and collected. The remaining 5 infants were studied for shorter periods of time due to the following reasons: computer malfunction, parent's wishes to stop the study, and noted physiologic instability.

Ethical Considerations

Ethical approval was obtained from the Health Research Ethics Board, Capital Health Region. A letter of support from the medical director and patient care manager of the NICU at the Royal Alexandra Hospital accompanied the ethical approval (Appendix I). The neonatal research staff member obtained consent from parents of potential subjects. The parents were provided with a completely detailed description of the study and the

potential risks and benefits to the infants were described. Parents were informed that they may withdraw their infant by informing a member of the neonatal research staff from the study at any time with no consequences to the medical or nursing care provided to their infant. They were informed that if their infant becomes agitated for greater than a 15 minute period, or if irritability or intolerance of the collar is noted, the infant would be removed from the study. They were invited to be at their baby's bedside throughout the study. The parents were also informed of the Patient Concerns Office location and number of the Capital Health Authority. To protect confidentiality and privacy, the infant's data were collected and stored with a code number in a locked facility that was accessible only to the researcher.

Data Collection Procedure

Prior to initiating the study the researcher met with medical, nursing, research, and respiratory staff to explain the purpose of the study. The participation of the neonatal research staff was clearly outlined to them prior to the start of the study. When a potential infant met the inclusion criteria, the mother or both parents were approached by a research staff member and asked if they were willing to discuss the study. If the parents agreed, the neonatal research staff provided further explanation of the study and obtained informed consent (Appendix II). The study began during the day and when the research nurses and the Occupational Therapist were available.

The recording equipment was placed on the infant by experienced neonatal research staff. As preterm infants at this proposed site are routinely placed in the prone position, they remained prone for the duration of the study. All neck rolls were also removed from the infants' bedside. As with routine infant polygraphic sleep studies and other apnea

detection studies that are carried out by neonatal research office staff, a monitoring sheet was also be placed at the bedside. The infant's nurse noted any procedures or events that the infant was exposed to during the monitoring session on this sheet. The nurse documented the activity of the infant during the procedure and the time in which it occurred (Appendix III). Areas involving any prolonged treatments during this time were noted. Any exaggerated or erratic episodes of recording, such as loss of signal or inappropriate correlation of the two heart rate readings was noted as well. The infant's birth and medical history were recorded on a data collection form developed for this study (Appendix IV).

The cervical collar was made by the full-time Occupational Therapist of the NICU immediately after the consent has been obtained. The collar was individually fitted and applied to each participant by the Occupational Therapist. The collar was made of soft foam and was covered with stockinet, and each end was connected with velcro material. It fit snugly around the infant's neck in order to maintain the head in a neutral position relative to the trunk; however, each was made to ensure two fingerbreadths could fit between the neck and collar. The collar was somewhat thicker in the middle front portion, for more stability under the chin, avoiding inadvertent neck flexion (Jacques & Karmel-Ross, 1997). This thicker portion also designated the top from the bottom. Any adjustments to the collar were made by the Occupational Therapist. The Occupational Therapist posted an instruction sheet at the bedside with directions and information for the application and removal of the collar, as well as the signs and symptoms of skin irritation. In the development and early testing for side effects, the collar had been well tolerated by preterm infants. However, all infants were monitored for any signs of

intolerance or discomfort from the study and/or collar. To control for application variety, only the Respiratory Therapists (n=14) were trained in the application and removal of the collar. None of the 16 infants experienced skin irritation from the collar.

Figure 5. Cervical Collars Made by the Occupational Therapist.

Note. An example of three different sizes of collars used in the study.

The infant was cared for in the usual manner by the assigned NICU staff, including bedside nurse and respiratory therapist. The time the study was planned was in accordance with the nurse's care plan in order to avoid over-stimulating the infant. Each infant was studied only once and served as his/her own control. Once data collection was completed, the data collection equipment was removed from the infant's bedside. A summary of the study procedure is outlined in Appendix V.

Measurement of Variables

For each infant, heart rate, oxygen saturation and FiO_2 levels, in addition to respiratory impedance were continuously recorded from Hewlett Packard cardiorespiratory monitoring equipment routinely used in the care of critically ill preterm infants in the NICU. The equipment that was used is identical to that used for polygraphic

sleep studies routinely carried out by experienced neonatal nurses working in the neonatal research office at the proposed site. This same equipment had been used at the proposed site for other clinical studies in which apnea detection is paramount and had been noted to be reliable, valid, and sensitive in detecting apnea frequency (Barrington et al., 1987; Muttitt et al., 1988; Finer at al., 1992).

Heart Rate and Respiratory Impedance

The infant's heart rate and respiratory waveform form were recorded from the cardio-respiratory module of the Hewlett Packard Monitor (Model 24, Hewlett Packard, Waltham, MA). Calibration was accomplished using a signal-generating box, developed by the biomedical staff at the RAH, for the purpose of ECG signal calibration. With this system, continuous beat-to-beat heart rate is recorded. The time period between each "R" wave is detected (of the QRS cardiac cycle) and on the basis of that time period, the rate is calculated and displayed on a digital readout. The respiratory waveform is generated through impedance pneumography. Three appropriately placed self-adhesive electrodes were placed on the infant (on the infant's chest, below the right and left clavicles, and on the infant's lower left abdomen) and attached to a 3-lead cable that was connected to the cardiac monitoring system. Heart rate and respiratory impedance were both digitally displayed and graphically recorded. The heart rate accuracy is ± 2 beats per minutes ($\pm$ 20 mv) and with an instantaneous response time (Model 24, Hewlett Packard, Waltham, MA). The resolution is 1 beat per minute on graphic display.

The oxygen saturation level was measured using a transcutaneous pulse oximeter (Model 24, Hewlett Packard, Waltham, MA). This particular monitor functions by producing two wavelengths of light (660 and 940 nm.) directed through a vascular bed.

Each pulsation of this bed causes a change in the light path length, which alters the light absorbency. At 660 nm, the absorbency of oxyhemoglobin is greater than deoxyhemoglobin; at 940 nm, the opposite is true. The absorbency ratio of the two wavelengths, therefore, varies with the ratio of oxy- to deoxyhemoglobin in arterial blood. Beat-to-beat oxygen saturation level detection is possible without interference from non-pulsating blood and other tissues. Calibration was accomplished by a signal generated within the monitoring system. Reports of clinical trials have indicated a good correlation between arterial and transcutaneous oxygen saturation levels in neonates ($r=0.90$, $p<0.001$) and response time near instantaneous (Jannis, & Peabody, 1987). The oxygen saturation level was both digitally displayed and graphically recorded for later analysis. This machine is a reliable and valid tool for measuring oxygen saturation (Barrington, Finer, & Ryan, 1998). The oxygen saturation accuracy is ± 0.5% (± 20mv) (Model 24, Hewlett Packard, Waltham, MA). While the sensor is sensitive to light and motion, these artifacts were reduced by securely and appropriately applying the probe and shielding it from the light with a Posey® or other available cover. To further reduce inaccurate readings caused by light or motion, the pulse rate from the oximeter was compared to that from the ECG. If the heart rate values differed by more than 10 beats per minute, the oxygen saturation was deemed inaccurate and was disregarded.

Ambient Oxygen Concentration

Ambient oxygen concentration (mini-ox; Ohmeda 5120) was measured in-line, distal to the infant, and the analog transformed to a digital signal that was subsequently recorded.

Recording System

The analog voltage signals derived from the above-named monitoring devices were acquired by a Data Translation analog-to-digital converter board at a rate of 30 samples per second (No. DT2801 Data Translation Inc., Malborough, Mass.) installed in an IBM personal computer Intel 300 mHz CPU (Markham, ON) with a 6.4 gigabyte hard drive. The ASYST scientific software system (MacMillan Software Co., New York, N.Y.) was used for programming. Three programs specifically written were used for data acquisition in the NICU at the Royal Alexandra Hospital to perform the following: create correctly configured files, acquire and store data in files, and perform analysis of the neonate's record. The signals were obtained at the neonates' bedsides from the monitors mentioned above, and the analog output from each device was connected to a purpose-built junction box that was hard-wired to the computer which was taken to the bedside for the study's duration.

Data Preparation

The data were downloaded to a digital conversion device, and then the computer generated a digitized screen of all the five variables: heart rate recorded from the Hewlett Packard monitor, heart rate and oxygen saturation from the Hewlett Packard pulse oximeter tracings, respiratory impedance, and FiO_2. Each computer screen showed recordings for a time period of one minute. Each variable had a separate color on the screen, and therefore was easily identified. The clinical acquisition program calculated the mean value of all variables. Any event (a bradycardia, oxygen desaturation, and/or apnea) thought to be due to artifact, such as handling or probe/electrode displacement, was not considered. Only episodes in which there was evidence of appropriate

monitoring (such as appropriate readings and correlation of the heart rate), before and after an event were measured. For each event the computer file contained information regarding the mean value calculated for one minute before the event for heart rate and oxygen saturation, plus the entire apnea event. Figure 5 demonstrates a sample of a one-minute period of recorded data taken from a different study where similar monitoring equipment was utilized.

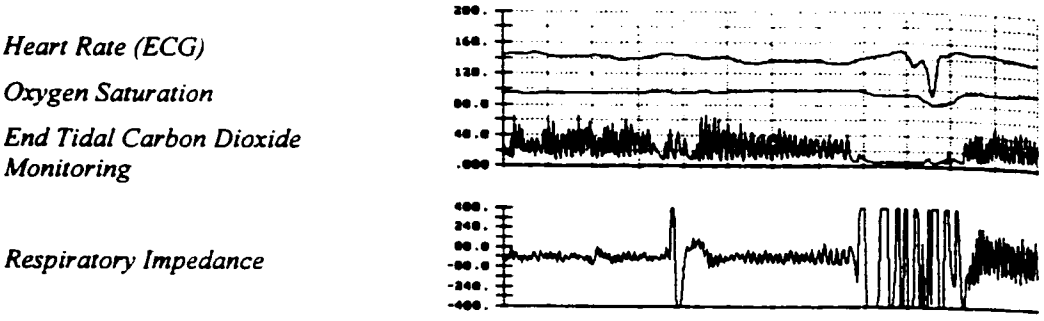

Figure 6. A Recording of Heart Rate, Oxygen Saturation, End Tidal Carbon Dioxide, and Respiratory Impedance for a One-Minute Period.
From: "Predischarge Respiratory Recordings in Very Low Birth Weight Newborn Infants" by K.J. Barrington, N. Finer, and D. Li, 1996, Journal of Pediatrics, 129(6), p. 938. Adapted with permission from the author.

Data was divided into three phases: baseline, control, and intervention. The three phases totaled 15 hours of useable data pending the activity of the infant and number of procedures taking place. To assess the possible associations between heart rate, oxygen saturation, and respiratory impedance with the changing study periods, markers were located on the digital screen by the researcher denoting the baseline period, the intervention period and the control period. Also, any epochs in which a bradycardic

and/or clinically significant oxygen desaturation and/or apnea episode occurred were marked.

As the researcher was not trained in computer tracing analysis, training was provided by the neonatal research staff, all of who are considered to be reliable in the analysis and interpretation of polygraphic sleep tracings. At least 10 traces were analyzed under the supervision of neonatal research staff. To assess for reliability, three further tracings were analyzed and results compared with those of a member of the neonatal research staff. A correlation of 0.92 resulted and was accepted as evidence of inter-rater reliability (Burns & Grove, 1997).

Data Analysis

Descriptive statistics were generated for physiologic and demographic variables. The physiologic variables included heart rate, respiratory impedance and oxygen saturation; whereas the demographic and health variables included birth weight, gestational and chronological age, apgar scores, use of antenatal steroids and/or surfactant, days intubated and on CPAP/HFNC, whether or not on enteral feedings, and types of medications. The statistics included measures of central tendency (mean, standard deviation, range and frequency) for all variables to describe the sample (Biles, 1995).

As the introduction of the intervention was randomly assigned creating two treatment protocols, a one-way analysis of variance (ANOVA) was used to assess for treatment order (period) effect on the dependent variables. Once completed, data were further analyzed to investigate the effect of the intervention. One-way ANOVA was used to test the primary hypothesis: 1) the effect of the cervical collar on isolatd apnea frequencies, as well as the secondary hypotheses: 1) the effect of the cervical collar on isolated

bradycardic events; 2) the effect of the collar on isolated significant oxygen desaturation episodes; 3) the effect of the cervical collar on combined apneic and bradycardic episodes; 4) the effect of the cervical collar on combined apneic and desaturation episodes; 5) the effect of the cervical collar on combined apneic, bradycardic, and oxygen desaturation episodes; and 6) the effect of the cervical collar on combined bradycardic and oxygen desaturation events. Confounding demographic and health variables were also tested for effect using multiple linear regression. The significance level accepted for all inferential testing was $p \leq 0.05$.

CHAPTER FOUR

Presentation of Findings

The purpose of the study was to determine if a cervical collar was an effective intervention in the treatment of apnea of prematurity. A summary of the subjects' characteristics is given, sequencing effect on the physiologic variables will be discussed, followed by the effects of the cervical collar. Finally, effects of demographic and health status variables on the outcomes will be reviewed.

Description of the Subjects

The parents of 18 infants, who met the inclusion criteria for the study, were approached and 16 parents consented, while 2 refused. The reasons for refusal included anxiety and uncertainty. Of the 16 enrolled infants, 11 were studied for the total period of 15 hours and 30 minutes, whereas 5 were studied only partially due to hemodynamic instability (n=1), equipment malfunction (n=3), or parental withdrawal of the infant due to anxiety (n=1). Figure 7 demonstrates the length of time of the infants' studies.

Characteristics of Subjects

The sample consisted of six females (38%) and ten males (62%). The gestational age of the infants ranged from 23 to 30 weeks, with a mean age of 26 ± 1.39 weeks; the biophysical assessment mean was 26.75 ± 1.77 weeks (Table 1). The birthweight of the infants was appropriate for their gestational age, with a mean weight of 905 grams (SD=213.66 grams) and a weight percentile mean of 60.88 grams (SD=25.13 grams). The age of the neonates at the time of study ranged from 28 to 46 weeks, with a mean age of 31.31 weeks (SD=4.6 weeks). All infants were receiving aminophylline and/or other medications, for a mean of 3.7 medications (SD=1.49 medications). Each infant had a therapeutic level of aminophylline documented within 3 days of the study. Figure 8

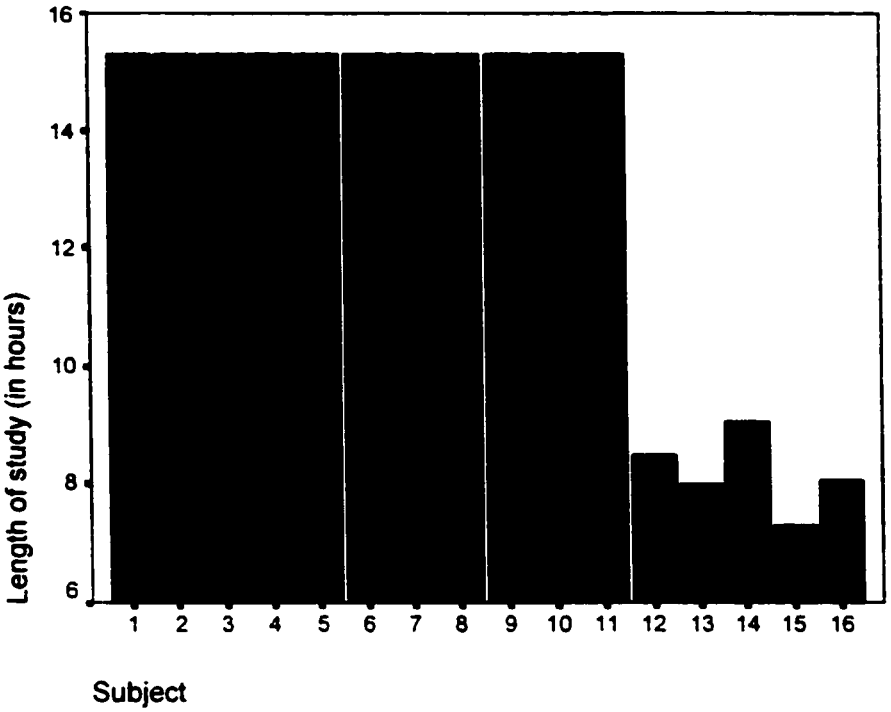

Figure 7. Length of Subjects' Studies.

Note. Subjects 1-11 were each studied for the total time of 15 hours and 30 minutes.

Subject 12 was studied for a period of 8 hours and 50 minutes. The study was stopped due to equipment errors.

Subject 13 was studied for a period of 8 hours due to physiologic instability.

Subject 14 was studied for a period of 9 hours and 3 minutes. The study was stopped due to an equipment malfunction.

Subject 15 was studied for a period of 7 hours and 30 minutes due to an equipment error.

Subject 16 was studied for a period of 8 hours and 5 minutes. The study was stopped due to a parental request.

Table 1

Sample Characteristics

	M	SD	Range
Gestational Age (in Weeks) n=16	26.06	1.39	23.00 - 30.00
Age at Time of Study (in Weeks) n=16	31.31	4.60	28.00 - 46.00
Biophysical Assessment (in Weeks) n=16	26.75	1.77	25.00 - 32.00
Gestational Birthweight (in Grams) n=16	905.00	213.67	590.00 - 1360.00
Weight Percentile n=16	60.88	25.13	25.00 - 97.00
Days Ventilated n=16	32.75	19.64	14.00 - 91.00
Days Since Last Extubation n=16	18.21	6.97	7.00 - 29.00

depicts the medications of each subject. As well, all 16 infants in the study were receiving enteral feedings.

The apgar scores of the infants are listed in Figure 9. The mean apgar score at one minute was 4 out of 10, and the mean score at 5 minutes was 6.8 out of ten, and 10 minutes, the mean score was 7.7 out of 10. Seven infants were delivered by caesarean section, due to maternal complications, and 9 infants were delivered vaginally due to spontaneous premature onset of labor. Antenatal steroids were given to 13 mothers. Antenatal steroids have beneficial effects on fetal lung development and maturity, if given for more than 24 hours, and less than 7 days prior to delivery (Schaap, et al., 2001). Three mothers did not receive antenatal steroid treatment due to the onset of a precipitous delivery.

All infants were premature and all had hyaline membrane disease. Apnea of

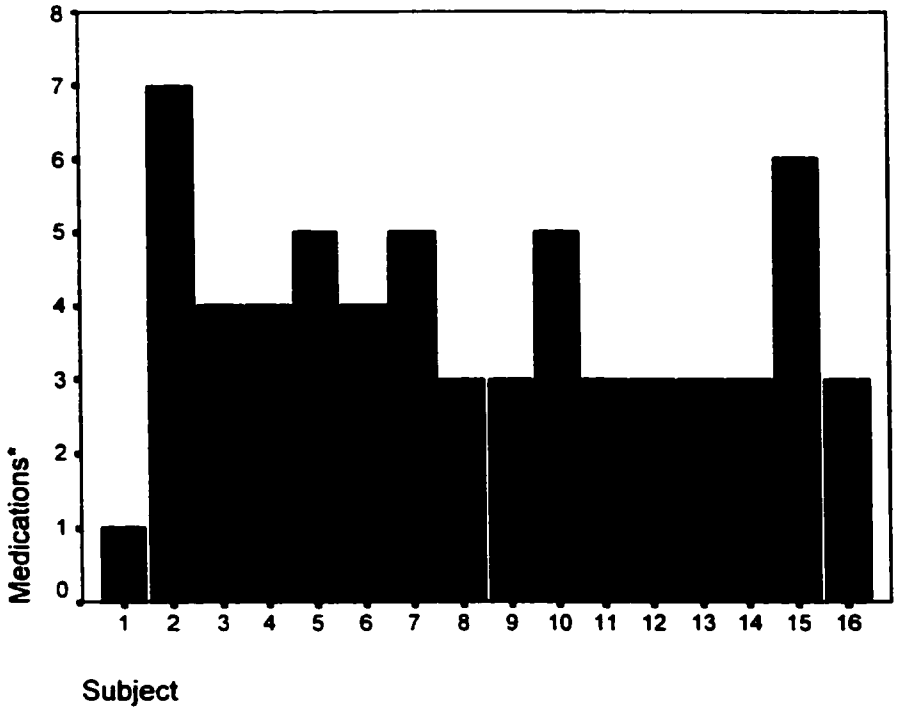

Figure 8. Patients' Medications at the Time of Study.

Note. 1= aminophylline only
2= aminophylline and antibiotics
3= aminophylline and sedation
4= aminophylline, sedation and antiemetics
5= aminophylline and antiemetics
6= aminophylline, antibiotics, sedation, and antiemetics
7=doxapram, aminophylline, and antiemetics

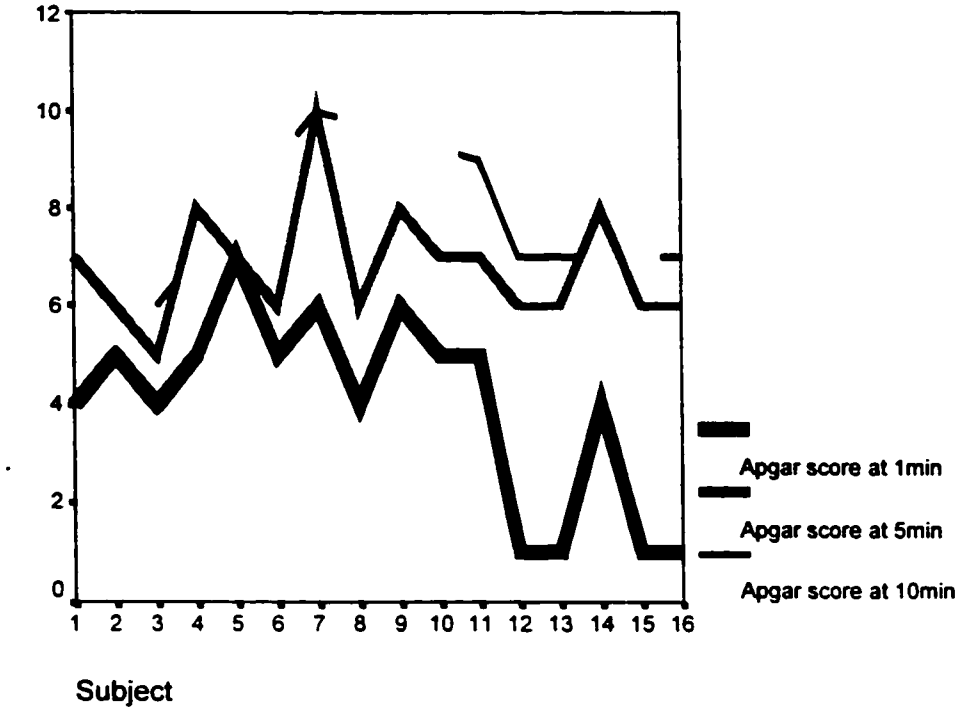

Figure 9. Subjects' Apgar Scores at One, Five and Ten Minutes.

Note. Only subjects 3, 7, 11, 12, 13, and 16, received a ten minute apgar score.

prematurity was documented for the entire sample. All but 2 infants required intubation at birth, due to premature lung development, and all but one of the intubated infants required at least one dose of surfactant. Thirteen infants received one or more doses of surfactant. At the time of the study, the days since the last extubation varied for each infant who had required endotracheal intubation. The length of time varied from 7 to 29 days (M=18.21; SD=6.97). Each infant required ventilation assistance at the time of the study; 14 were receiving CPAP, and 2 were receiving HFNC.

Description of Physiologic Variables of the Infants

Three isolated physiologic parameters were monitored and measured throughout the study: apnea, bradycardia, and oxygen desaturations. The number of episodes and the duration (in seconds) of each occurrence were measured for apnea; whereas the number of episodes and the severity (value) were recorded for bradycardias (heart rate in one minute) and oxygen desturations (oxygen saturation reading). Episodes of various combinations of the three variables were also recorded (i.e. bradycardia and apneas, bradycardias and oxygen desaturations, etc). Frequencies and measures of central tendency for episodes, duration, and values for the three isolated variables were recorded during the three study periods: the three-hour baseline period, the six-hour collar period, and the six-hour control period.

Apneic Episodes and Apnea Duration

The detection of central apnea was the only type of apnea that could be recorded and identified with the use of the respiratory impedance data. An isolated apnea consisted of a respiratory pause of greater than 20 seconds. Other apnea types could not be detected, as end-tidal CO_2 could not be monitored due to the ineffectiveness of the device when used

in conjunction with CPAP and HFNC. Isolated apneic episodes were not detected in every subject. Only 4 of the 16 subjects (25%) experienced apnea in the baseline period, 3 out of 14 (21%) in the collar period, and 3 of the 13 (23%) subjects in the control period. Nine apneic episodes occurred in the baseline period, 3 in the collar period, and 6 in the control period. The duration of the apneic episodes varied for each subject, ranging from 20 to 45 seconds (Table 2; Figures 10 and 11). During the baseline period, the mean apnea duration was 23.22 seconds (SD=3.93 seconds); in the collar period, the mean duration was 26.67 seconds (SD=3.79 seconds); and finally, during the control period, the mean duration was 26.50 seconds (SD=9.75 seconds).

Table 2

Apneic Episodes and Apnea Duration

	Apneic Episodes				Apnea Duration				
	Number of Episodes	Frequency	Percent		Seconds	Frequency	Percent	M	SD
Baseline	0.00	12	75.0		21.00	5	23.8	23.22	3.93
n=16	1.00	1	6.3		23.00	2	9.5		
	2.00	2	12.5		25.00	1	4.8		
	4.00	1	6.3		33.00	1	4.8		
					Missing	12	57.0		
Collar	0.00	11	68.8		21.00	1	6.3	23.67	3.79
n=14	1.00	3	18.8		22.00	1	6.3		
	Missing	2	12.5		28.00	1	6.3		
					Missing	13	81.3		
Control	0.00	10	62.5		21.00	4	21.0	26.5	9.75
n=13	2.00	3	18.8		30.00	1	5.3		
	Missing	3	18.8		46.00	1	5.3		
					Missing	13	68.4		

Bradycardic Episodes and Bradycardic Rates

An isolated bradycardic episode consisted of any heart rate decrease, greater than or equal to 30% from the subject's mean heart rate level, measured one minute prior to the event. The Hewlett Packard tracings, as well as the heart rate tracings from the pulse

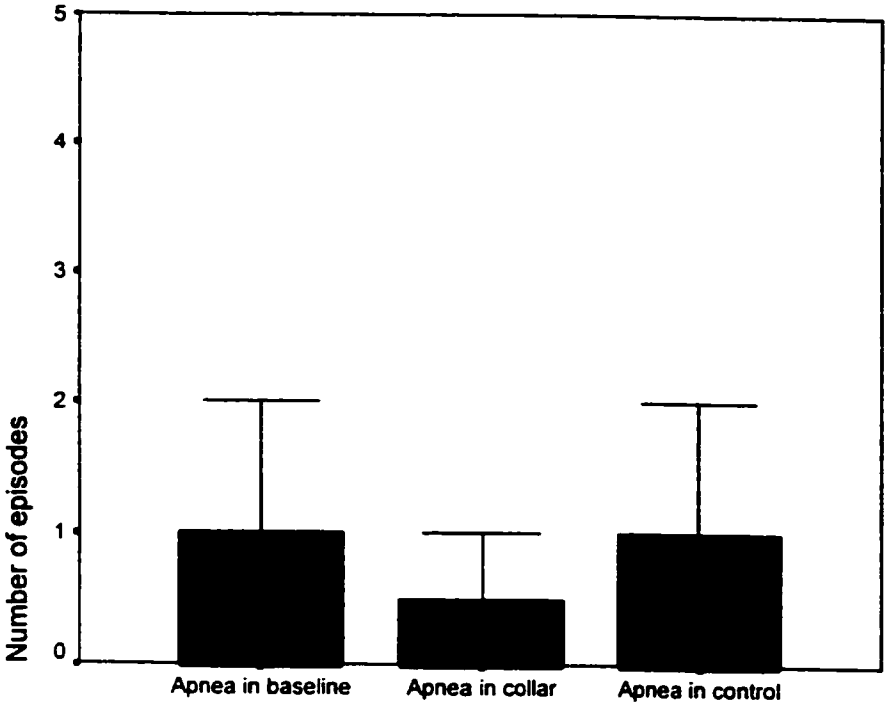

Figure 10. Apnea Episodes in Three Time Periods: Baseline, Collar, and Control.

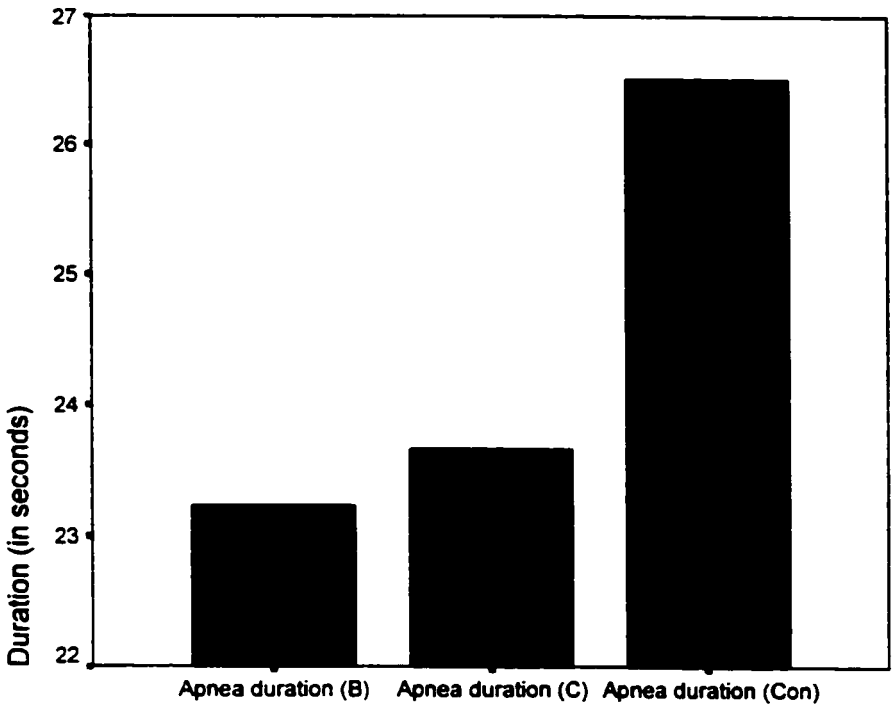

Figure 11. Mean Apnea Duration in Three Time Periods: Baseline, Collar, and Control.

Note. Mean Apnea Duration in Baseline (B), Apnea Duration During Collar Period (C), and Apnea Duration During Control Period (Con).

oximeter, provided the necessary data for the detection of bradycardias. A true bradycardia was recorded if the two heart rate tracings were of close correlation (being within 10 beats per minute of each other). During the baseline period, 5 of the 16 subjects (31%) experienced isolated bradycardic episodes; in the collar period, 12 of 14 subjects (86%) had bradycardic episodes; and in the control period, 9 of 13 subjects (69%) had documented bradycardic episodes. The bradycardic rates for the 21 episodes recorded in the baseline period had a mean of 95.71 beats per minute (SD= 8.81 beats per minute); for the 85 during the collar period, a mean of 79.95 beats per minute was recorded (SD=23.93 beats per minute); and for the 44 recorded throughout the control period, a mean of 85.23 beats per minute (SD= 20.01 beats per minute) resulted. The degree of bradycardia ranged from 26 to 119 beats per minute (Tables 3 and 4). Figures 12 depicts the mean for bradycardic episodes, and Figure 13 depicts the mean for bradycardic rates.

Oxygen Desaturation Episodes and Oxygen DesaturationValues

Oxygen desaturations were detected via the tracings of the pulse oximeter. An oxygen desaturation consisted of any decrease in the subject's saturation level of less than 88%, $\geq$ 10 seconds. To ensure the recording was accurate and not due to artifact, the heart rate tracings of the oximeter had to be within 10 beats per minute with those of the Hewlett Packard monitor. During the baseline period, 15 of the 16 subjects (94%) experienced oxygen desaturations; 13 of the 14 subjects (93%) had documented episodes in the collar period; and all 13 subjects (100%) had desaturations throughout the control period (Tables 5 and 6). The degree of oxygen desaturation varied for each subject, ranging from 31% to 87%. In the baseline period, 174 desaturation episodes were recorded, with a

mean value of 78.11 ± 6.17%. Throughout the collar period, 320 desaturation episodes were recorded, with a mean value of 75.24 ± 9.52%; and during the control period, 248 oxygen desaturation episodes were documented, having a mean value of 75.65 ± 7.82% (Figures 14 and 15).

Table 3

Bradycardic Episodes

Baseline(n=16)			Collar (n=14)			Control (n=13)		
Number of Episodes	Frequency	Percent	Number of Episodes	Frequency	Percent	Number of Episodes	Frequency	Percent
0.00	11	68.8	0.00	2	12.5	0.00	4	25.0
1.00	2	12.5	1.00	2	12.5	1.00	2	12.5
4.00	1	6.3	2.00	1	6.3	2.00	2	12.5
7.00	1	6.3	3.00	3	18.8	3.00	1	6.3
8.00	1	6.3	4.00	3	18.8	4.00	1	6.3
			5.00	1	6.3	5.00	2	12.5
			10.00	1	6.3	21.00	1	6.3
			45.00	1	6.3	Missing	3	18.8
			Missing	2	12.5			

Combination Episodes

Episodes of all possible combinations of the three physiologic variables were also recorded. A combination episode was documented if two or more of the three variables occurred simultaneously during recording. Episodes were documented for the following combinations of variables: 1) apnea and bradycardia, 2) apnea and desaturation, 3) apnea, bradycardia, and desaturation, 4) bradycardia and desaturation.

The number of episodes varied for each subject and each different combination (Table 7). An apnea and bradycardic episode was recorded when the two following physiologic correlates occurred simultaneously: a ≥ 30% decrease in the subject's heart rate (when compared to the previous one-minute heart rate mean), and a respiratory pause of ≥ 10 seconds. During the baseline period, this combination had a mean occurrence of

Table 4

Bradycardic Rates in Baseline

(n=16, M=95.71, SD=8.81)		
Rates	Frequency	Percent
76.00	1	3.1
80.00	1	3.1
86.00	3	9.4
89.00	1	3.1
93.00	1	3.1
94.00	1	3.1
97.00	1	3.1
98.00	3	9.4
100.00	2	6.3
101.00	1	3.1
103.00	1	3.1
104.00	3	9.4
105.00	1	3.1
108.00	1	3.1
Missing	11	34.4

Bradycardic Rates in Control Period

(n=13, M=85.23, SD=20.01)		
Rates	Frequency	Percent
28.00	1	2.0
42.00	1	2.0
43.00	1	2.0
44.00	1	2.0
56.00	1	2.0
58.00	1	2.0
70.00	2	3.9
77.00	1	2.0
78.00	4	7.8
81.00	1	2.0
82.00	4	7.8
84.00	2	3.9
85.00	2	3.9
89.00	1	2.0
91.00	2	3.9
93.00	3	5.9
95.00	2	3.9
97.00	1	2.0
98.00	1	2.0
99.00	1	2.0
100.00	1	2.0
101.00	2	3.9
103.00	1	2.0
104.00	1	2.0
105.00	1	2.0
106.00	1	2.0
107.00	2	3.9
116.00	1	2.0
119.00	1	2.0
Missing	7	13.7

Bradycardic Rates in Collar Period

(n=14, M=79.95, SD=23.92)		
Rates	Frequency	Percent
26.00	1	1.1
27.00	1	1.1
28.00	2	2.2
29.00	2	2.2
32.00	1	1.1
38.00	1	1.1
40.00	2	2.2
41.00	1	1.1
43.00	1	1.1
44.00	2	2.2
51.00	1	1.1
52.00	1	1.1
58.00	1	1.1
60.00	1	1.1
73.00	3	3.4
74.00	2	2.2
75.00	1	1.1
76.00	1	1.1
77.00	2	2.2
78.00	2	2.2
79.00	1	1.1
80.00	2	2.2
81.00	4	4.5
82.00	3	3.4
83.00	3	3.4
84.00	2	2.2
85.00	1	1.1
86.00	1	1.1
87.00	1	1.1
88.00	2	2.2
89.00	3	3.4
92.00	3	3.4
93.00	2	2.2
94.00	5	5.6
99.00	4	4.5
102.00	3	3.4
103.00	3	3.4
104.00	3	3.4
105.00	3	3.4
106.00	2	2.2
108.00	2	2.2
110.00	2	2.2
113.00	1	1.1
Missing	4	4.5

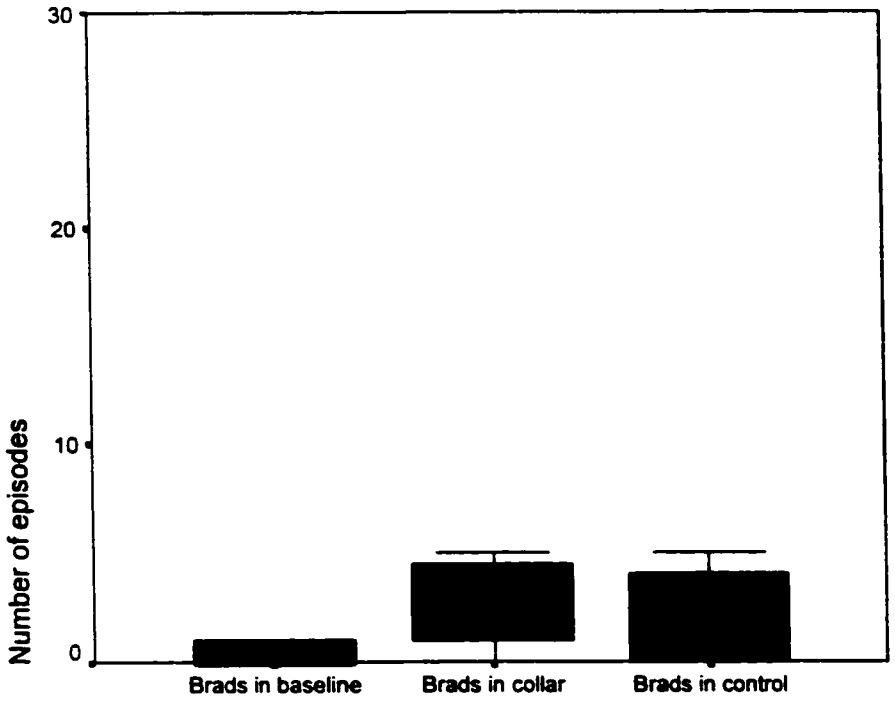

Figure 12. Bradycardia Episodes in Three Time Periods: Baseline, Collar, and Control.

Note. Brads=Bradycardia Episodes.

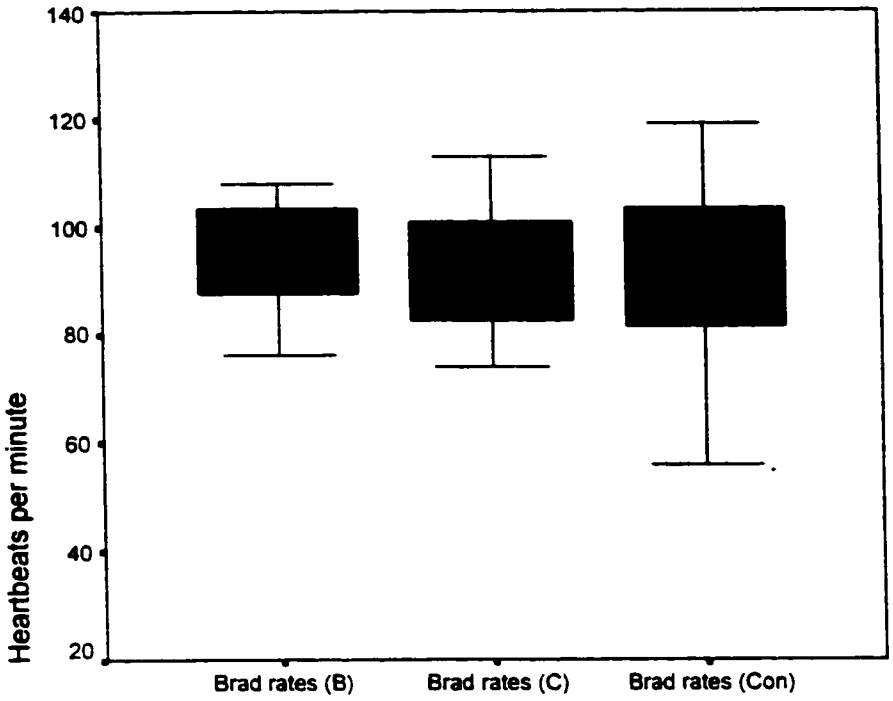

Figure 13. Bradycardia Rates in Three Time Periods: Baseline, Collar and Control.

Note. Bradycardia Rates in Baseline (Brad rates B), Bradycardia Rates During Collar Period (Brad rates C), and Bradycardia Rates During Control Period (Brad rates Con).

Table 5

Oxygen Desaturation Episodes

Baseline (n=16)			Collar (n=14)			Control (n=13)		
Number of Episodes	Frequency	Percent	Number of Episodes	Frequency	Percent	Number of Episodes	Frequency	Percent
0.00	1	6.3	0.00	1	6.3	1.00	1	6.3
1.00	1	6.3	4.00	1	6.3	4.00	1	6.3
4.00	1	6.3	5.00	1	6.3	6.00	1	6.3
5.00	1	6.3	10.00	1	6.3	7.00	1	6.3
6.00	1	6.3	13.00	1	6.3	8.00	1	6.3
7.00	1	6.3	16.00	1	6.3	10.00	1	6.3
8.00	1	6.3	17.00	1	6.3	15.00	1	6.3
9.00	1	6.3	27.00	1	6.3	16.00	1	6.3
11.00	1	6.3	29.00	1	6.3	23.00	1	6.3
12.00	1	6.3	36.00	1	6.3	32.00	1	6.3
13.00	1	6.3	40.00	3	18.8	37.00	1	6.3
15.00	1	6.3	43.00	1	6.3	38.00	1	6.3
16.00	1	6.3	Missing	2	12.5	51.00	1	6.3
22.00	2	12.5				Missing	3	18.8
23.00	1	6.3						

0.63 ± 0.96 episodes; a mean occurrence of 1.00 ± 1.52 episodes during the collar period; and a mean occurrence of 1.54 ± 2.44 episodes during the control period.

An apnea and desaturation episode was recorded when the subject's oxygen saturation level was less than 88% and there was a respiratory pause of ≥ 10 seconds. During the baseline period, this type of combination had a mean occurrence of 1.94 ± 2.84 episodes; throughout the collar period, the mean occurrence was 4.79 ± 6.93 episodes; and throughout the control period, the mean occurrence was 2.62 ± 3.38 episodes.

A combination involving the three physiologic correlates was recorded when all of the following simultaneously occurred: an oxygen saturation of less than 88%, a respiratory pause of ≥ 10 seconds, and a ≥ 30% decrease in heart rate when compared to the previous one-minute heart rate mean. During baseline, the mean occurrence for this combination was 2.25 ± 3.61 episodes; during the collar period, the mean occurrence was 5.93 ± 5.36 episodes; and during the control period, the mean occurrence was 2.92 ± 3.35 episodes.

Table 6

Oxygen Desaturation Values

Desaturation Values in Baseline

(n=16, M= 78.11, SD= 6.17)		
O_2 Percent	Frequency	Percent
55.00	1	0.6
63.00	1	0.6
64.00	3	1.7
65.00	2	1.1
66.00	6	3.4
67.00	1	0.6
68.00	2	1.1
69.00	2	1.1
70.00	5	2.9
71.00	5	2.9
72.00	5	2.9
73.00	5	2.9
74.00	6	3.4
75.00	7	4.0
76.00	4	2.3
77.00	10	5.7
78.00	11	6.3
79.00	14	8.0
80.00	13	7.4
81.00	11	6.3
82.00	10	5.7
83.00	8	4.6
84.00	18	10.3
85.00	16	9.1
86.00	6	3.4
87.00	2	1.1
Missing	1	0.6

Desaturation Values in Collar

(n=14, M=75.24, SD=9.52)		
O_2 Percent	Frequency	Percent
31.00	1	0.3
40.00	1	0.3
43.00	1	0.3
45.00	1	0.3
46.00	1	0.3
47.00	2	0.6
48.00	1	0.3
51.00	2	0.6
52.00	1	0.3
54.00	1	0.3
55.00	4	1.2
56.00	2	0.6
57.00	2	0.6
58.00	2	0.6
59.00	2	0.6
60.00	4	1.2
61.00	3	0.9
62.00	4	1.2
63.00	6	1.9
64.00	5	1.5
65.00	3	0.9
66.00	4	1.2
67.00	6	1.9
68.00	6	1.9
69.00	5	1.5
70.00	3	0.9
71.00	9	2.8
72.00	8	2.5
73.00	9	2.85
74.00	12	3.7
75.00	13	4.0
76.00	10	3.1
77.00	17	5.3
78.00	18	5.6
79.00	16	5.0
80.00	15	4.6
81.00	25	7.7
82.00	16	5.0
83.00	26	8.0
84.00	28	8.7
85.00	20	6.2
86.00	5	1.5
Missing	3	0.9

Desaturation Values in Control

(n=13, M= 75.65, SD= 7.82)		
O_2 Percent	Frequency	Percent
44.00	1	0.4
50.00	2	0.8
53.00	2	0.8
55.00	2	0.8
56.00	2	0.8
58.00	1	0.4
59.00	1	0.4
60.00	1	0.4
61.00	1	0.4
62.00	2	0.8
63.00	4	1.6
64.00	5	2.0
65.00	4	1.6
66.00	5	2.0
67.00	4	1.6
68.00	5	2.0
69.00	5	2.0
70.00	7	2.8
71.00	6	2.4
72.00	9	3.6
73.00	8	3.2
74.00	12	4.8
75.00	12	4.8
76.00	9	3.6
77.00	19	7.6
78.00	11	4.4
79.00	8	3.2
80.00	24	9.6
81.00	11	4.4
82.00	16	6.4
83.00	18	7.2
84.00	19	7.6
85.00	7	2.8
86.00	3	1.2
87.00	2	0.8
Missing	3	1.2

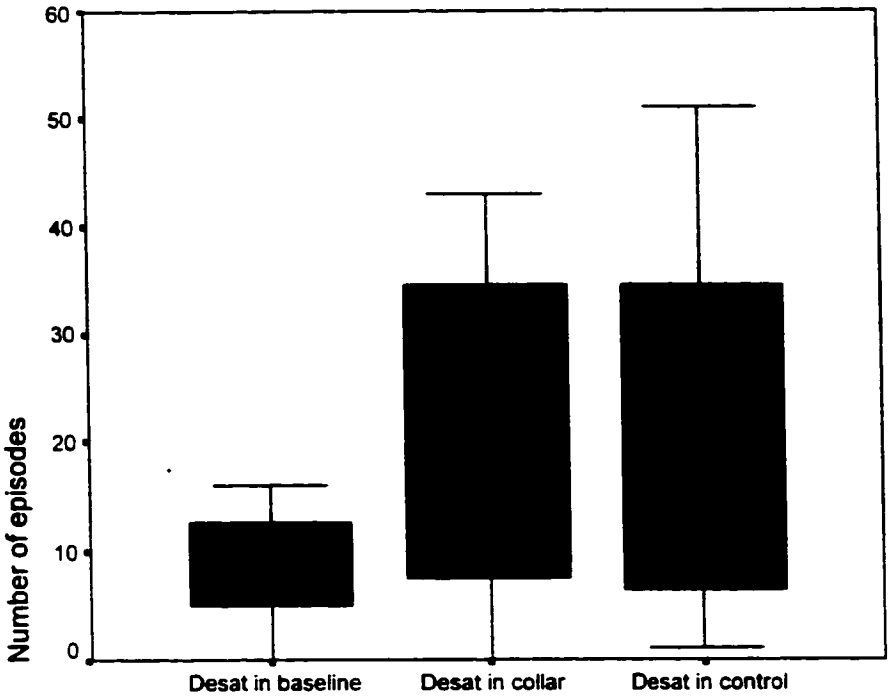

Figure 14. Desaturation Episodes in Three Time Periods: Baseline, Collar and Control.

Note. Desat = Desaturations.

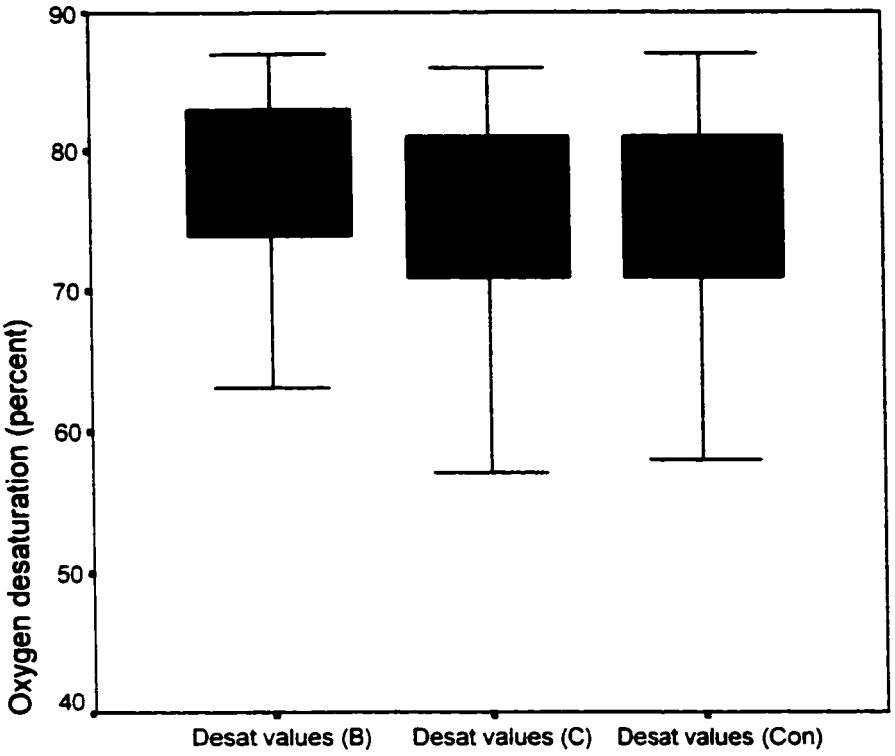

Figure 15. Desaturation Values for Three Time Periods: Baseline, Collar and Control.

Note Desaturation Values in Baseline (Desat values B), Desaturation Values During Collar Period (Desat values C), and Desaturation Values During Control Period (Desat values Con).

Table 7

Frequency of Combination Episodes

	M	SD
	Baseline (n=16)	
Apneas and Bradycardias	0.63	0.96
Apneas and Desaturations	1.94	2.84
Apneas, Bradycardias and Desaturations	2.25	3.61
Bradycardias and Desaturations	2.19	3.21
	Collar (n=14)	
Apneas and Bradycardias	1.00	1.52
Apneas and Desaturations	4.8	6.93
Apneas, Bradycardias, and Desaturations	5.93	5.36
Bradycardias and Desaturations	5.93	5.85
	Control (n=13)	
Apneas and Bradycardias	1.54	2.44
Apneas and Desaturations	2.62	3.38
Apneas, Bradycardias, and Desaturations	2.92	3.35
Bradycardias and Desaturations	5.85	9.91

The final combination recorded involved bradycardias and desaturations. Episodes for this combination were recorded when an oxygen saturation of less than 88% occurred together with a heart rate decrease of $\geq$ 30% from the previous one-minute heart rate mean. During the baseline period, the mean occurrence for this combination was 2.19 $\pm$ 3.21 episodes; throughout the collar period, the mean occurrence was 5.93 $\pm$ 7.15 episodes; and finally, throughout the control period, the mean was 5.85 $\pm$ 9.91 episodes (Figures 16, 17, 18, and 19).

Effect of Sequencing on Physiologic Parameters

One-way analysis of variance (ANOVA) was done to assess for an order (sequence) effect of the cervical collar. Sequencing did not account for significant differences in the dependent variables. A summary of the one-way ANOVA of the treatment order effect on the isolated physiologic parameters is found in Table 8. The results of the cervical

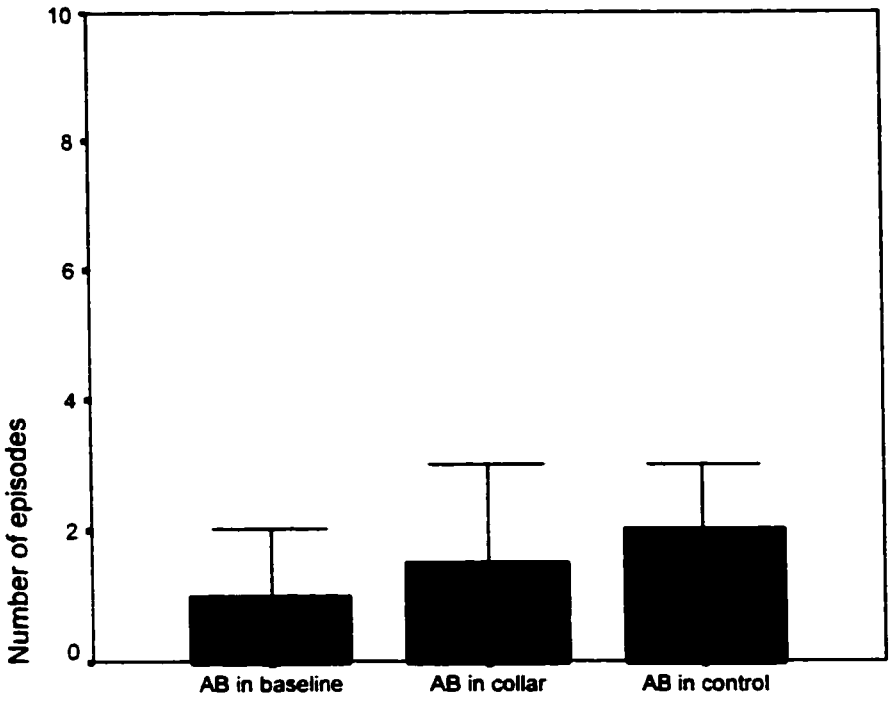

Figure 16. Combined Apnea and Bradycardia Episodes in Three Time Periods: Baseline, Collar, and Control.

Note. Apnea and Bradycardia Episodes in Baseline (AB in Baseline), Apnea and Bradycardia Episodes During Collar Period (AB during collar), and Apnea and Bradycardia Episodes During Control Period (AB during control).

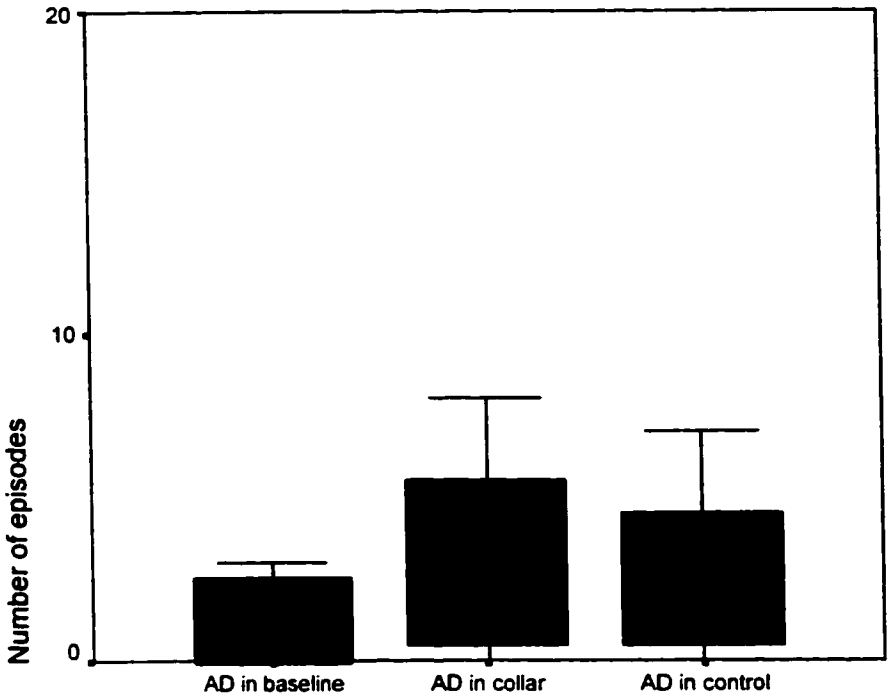

Figure 17. Combined Apnea and Desaturation Episodes in Three Time Periods: Baseline, Collar, and Control.

Note. Apnea and Desaturation Episodes in Baseline (AD in baseline), Apnea and Desaturation Episodes During Collar Period (AD during collar), and Apnea and Desaturation Episodes During Control Period (AD during control).

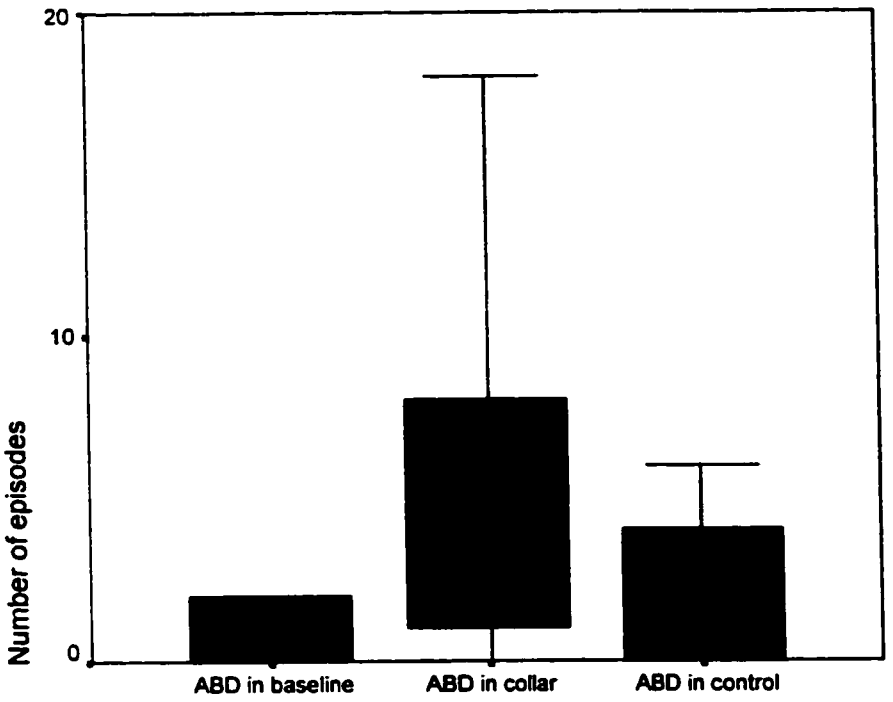

Figure 18. Combined Apnea, Bradycardia, and Desaturation Episodes in Three Time Periods: Baseline, Collar, and Control.

Note. Apnea, Bradycardia, and Desaturation Episodes in Baseline (ABD in baseline), Apnea, Bradycardia and Desaturation Episodes During Collar Period (ABD during collar), and Apnea, Bradycardia, and Desaturation Episodes During Control Period (ABD during control).

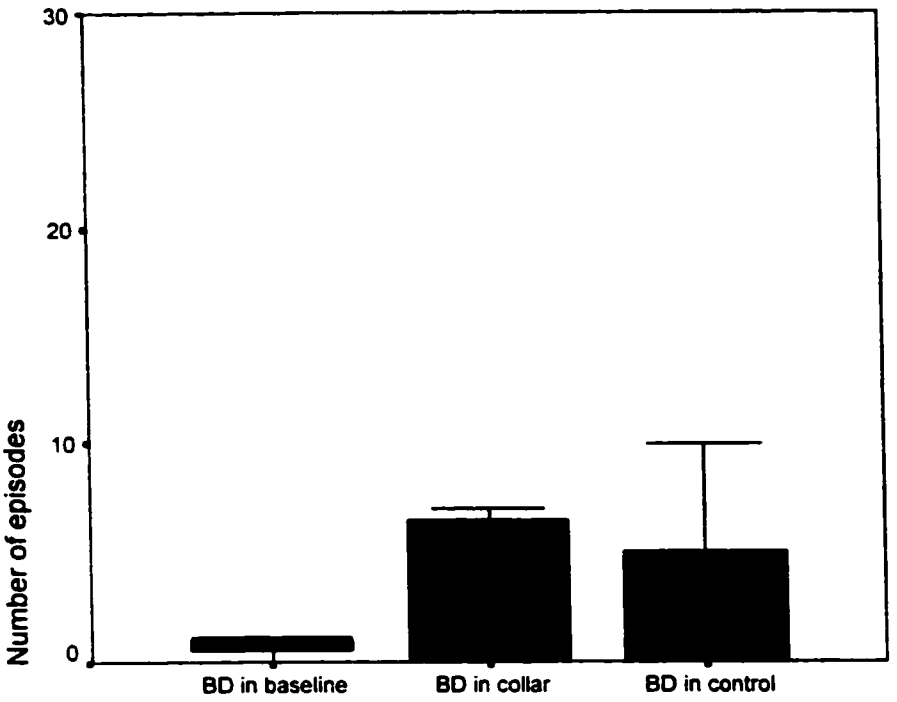

Figure 19. Combined Bradycardia and Desaturation Episodes in Three Time Periods: Baseline, Collar, and Control.

Note. Bradycardia and Desaturation Epiosdes in Baseline (BD in baseline), Bradycardia and Desaturation Episodes During Collar Period (BD during collar), and Bradycardia and Desaturation Episodes During Control Period (BD during control).

collar sequence on the combined episodes of the physiologic parameters are shown in Table 9.

Table 8

Treatment Order Effect of Isolated Physiologic Variables

Episodes		SS	df	MS	F	Sig.
Apneas in Baseline	Between Groups	2.38	1	2.38	1.90	0.19
	Within Groups	17.56	14	1.25		
	Total	19.94	15			
Apneas During Collar Period	Between Groups	7.14E-02	1	7.14E-02	0.38	0.55
	Within Groups	2.29	12	0.19		
	Total	2.36	13			
Apneas During Control Period	Between Groups	1.68	1	1.68	2.44	0.15
	Within Groups	7.56	11	0.69		
	Total	9.23	12			
Bradycardias in Baseline	Between Groups	6.83	1	6.83	0.99	0.34
	Within Groups	96.60	14	6.90		
	Total	103.44	15			
Bradycardias During Collar Period	Between Groups	97.79	1	97.79	0.73	0.41
	Within Groups	1617.14	12	134.76		
	Total	1714.93	13			
Bradycardias During Control Period	Between Groups	0.86	1	0.86	0.03	0.88
	Within Groups	376.22	11	34.20		
	Total	377.08	12			
Desaturations in Baseline	Between Groups	24.77	1	24.77	0.45	0.51
	Within Groups	766.98	14	54.79		
	Total	791.75	15			
Desaturations During Collar Period	Between Groups	0.00	1	0.00	0.00	1.00
	Within Groups	3055.71	12	254.64		
	Total	3055.71	13			
Desaturations During Control Period	Between Groups	21.37	1	21.37	0.08	0.78
	Within Groups	2961.56	11	269.23		
	Total	2982.92	12			

Table 9

Treatment Order Effect: Combined Physiologic Variables

		SS	df	MS	F	Sig
Apneas and Badycardias						
Baseline	Between Groups	0.67	1	0.67	0.71	0.41
	Within Groups	13.08	14	0.93		
	Total	13.75	15			
Collar	Between Groups	2.57	1	2.57	1.12	0.31
	Within Groups	27.43	12	2.29		
	Total	30.00	13			
Control	Between Groups	1.23	1	1.23	0.19	0.67
	Within Groups	70.00	11	6.37		
	Total	71.23	12			
Apneas and Desaturations						
Baseline	Between Groups	14.05	1	14.05	1.84	0.19
	Within Groups	106.89	14	7.64		
	Total	120.94	15			
Collar	Between Groups	5.79	1	5.79	0.11	0.74
	Within Groups	618.57	12	51.55		
	Total	624.36	13			
Control	Between Groups	7.44	1	7.44	0.63	0.44
	Within Groups	129.64	11	11.79		
	Total	137.08	12			
Apneas, Bradycardias, and Desaturations						
Baseline	Between Groups	13.35	1	13.35	1.02	0.33
	Within Groups	181.65	14	12.98		
	Total	195.00	15			
Collar	Between Groups	3.50	1	3.50	0.11	0.74
	Within Groups	369.43	12	30.79		
	Total	372.93	13			
Control	Between Groups	2.62	1	2.62	0.21	0.65
	Within Groups	132.31	11	12.03		
	Total	134.92	12			
Bradycardias and Desaturations						
Baseline	Between Groups	7.17	1	7.17	0.68	0.42
	Within Groups	147.27	14	10.52		
	Total	154.44	15			
Collar	Between Groups	28.57	1	28.57	0.53	0.48
	Within Groups	647.14	12	53.93		
	Total	675.71	13			
Control	Between Groups	109.14	1	109.14	1.12	0.31
	Within Groups	1068.56	11	97.14		
	Total	1177.70	12			

Effect of the Cervical Collar on Physiologic Parameters

One-way ANOVA was used to determine the treatment (collar) effect, over time, on isolated and combined physiologic parameters.

Primary Hypothesis I: The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the overall frequency of apneic episodes. There was no evidence to support this hypothesis. There were no statistically significant differences found in either apneic episodes during baseline (n= 9), collar period (n= 3), and control period (n=6) (F= .59, p=. 556), or in apnea duration (F=.48, p=.625) (Table 10).

Table 10

One-Way Analysis of Variance of the Cervical Collar Effect on Isolated Physiologic Variables

	SS	df	MS	F	Sig.
Apneic Episodes					
Between Groups	0.94	2	0.47	0.59	0.56
Within Groups	31.52	40	0.79		
Total	32.46	42			
Apnea Duration					
Between Groups	40.55	2	20.28	0.48	0.63
Within Groups	627.72	15	41.84		
Total	668.27	17			
Bradycardic Episodes					
Between Groups	169.30	2	84.65	1.54	0.23
Within Groups	2195.44	40	54.89		
Total	2364.74	42			
Bradycardia Rates					
Between Groups	4327.26	2	2163.63	4.75	0.01
Within Groups	66845.82	147	454.73		
Total	71173.09	149			
Desaturation Episodes					
Between Groups	1133.79	2	566.90	3.32	0.05
Within Groups	6830.38	40	170.76		
Total	7964.18	42			
Desaturation Values					
Between Groups	986.479	2	493.24	7.20	0.01
Within Groups	50611.58	739	68.49		
Total	51598.05	741			

Table 11

Post Hoc-Comparisons-Scheffe

Dependent Variable: Desaturation Episodes

			Mean Difference (I-J)	SE	Sig.	95% CI	
	(I) Time Factor	(J) Time Factor				Lower Bound	Upper Bound
Scheffe	Baseline	Collar	-11.9821	4.7822	0.054	-24.1401	0.1758
		Control	-8.2019	4.8793	0.255	-20.6068	4.2030
	Collar	Baseline	11.9821	4.7822	0.054	-0.1758	24.1401
		Control	3.7802	5.0331	0.756	-9.0157	16.5761
	Control	Baseline	8.2019	4.8793	0.255	-4.2030	20.6068
		Collar	-3.7802	5.0331	0.756	-16.5761	9.0157

* The mean difference is significant at the .05 level.

Dependent Variable: Bradycardic Rates

			Mean Difference (I-J)	SE	Sig.	95% CI	
	(I) Time factor	(J) Time factor				Lower Bound	Upper Bound
Scheffe	Baseline	Collar	15.7613	5.1965	0.012	2.9109	28.6118
		Control	10.4870	5.6559	0.183	-3.4994	24.4734
	Collar	Baseline	-15.7613	5.1965	0.012	-28.6118	-2.9109
		Control	-5.2743	3.9604	0.414	-15.0680	4.5193
	Control	Baseline	-10.4870	5.6559	0.183	-24.4734	3.4994
		Collar	5.2743	3.9604	0.414	-4.5193	15.0680

* The mean difference is significant at the .05 level.

Dependent Variable: Desaturation Values

			Mean Difference (I-J)	SE	Sig.	95% CI	
	(I) Time factor	(J) Time factor				Lower Bound	Upper Bound
Scheffe	Baseline	Collar	2.8686	0.7795	0.001	0.9567	4.7805
		Control	2.4560	0.8184	0.011	0.4487	4.4632
	Collar	Baseline	-2.8686	0.7795	0.001	-4.7805	-0.9567
		Control	-0.4126	0.7001	0.841	-2.1298	1.3046
	Control	Baseline	-2.4560	0.8184	0.011	-4.4632	-0.4487
		Collar	0.4126	0.7001	0.841	-1.3046	2.1298

* The mean difference is significant at the .05 level.

Secondary Hypotheses: I. The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the number of isolated bradycardic episodes experienced without apnea and significant oxygen desaturations, when compared to the control period. There was no statistically significant difference found among bradycardic episodes during baseline (n=21), collar period (n=85), and control (n=44) (F=1.54, p= .226) (Table 10). However, a significant difference resulted when the bradycardic

rates were tested (F= 4.75, p=.01). The difference occurred between the baseline and collar period, increasing from 21 to 85 events (p=. 01) (Table 11).

II. The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the number of isolated oxygen desaturations experienced without significant bradycardia or apnea, when compared to the control period. Significant differences resulted (F= 3.32, p= .05) (Table 10); however the post-hoc analysis revealed that the significant findings did not occur between the collar (n=320) and control period (n=258) (p=.75) (Table 11). The differences resulted between the baseline and collar period, increasing by 168 episodes during the collar period (p=.05). Differences were also found in the desaturation values (F=7.2, p=.001), between the baseline and collar periods (p=. 001) as well as the baseline and control, increasing by 96 episodes (p=. 01) period.

Table 12

One-Way Analysis of Variance of the Cervical Collar Effect on Combined Physiologic Variables

	SS	df	MS	F	Sig.
Apneas and Bradycardias					
Between Groups	5.99	2	2.99	1.04	0.36
Within Groups	114.98	40	2.88		
Total	120.97	42			
Apneas and Desaturations					
Between Groups	64.41	2	32.21	1.46	0.24
Within Groups	882.37	40	22.06		
Total	946.79	42			
Apneas, Bradycardias, and Desaturations					
Between Groups	110.91	2	55.46	3.15	0.05
WithinGroups	702.85	40	17.57		
Total	813.76	42			
Bradycardias and Desaturations					
Between Groups	137.68	2	68.84	1.37	0.26
Within groups	1997.05	40	49.93		
Total	2134.74	42			

III. The use of a cervical collar in preterm infants receving CPAP or HFNC will significantly decrease the number of combined bradycardic and oxygen desaturation episodes, when compared to the control period. There was no evidence to support this hypothesis (F=1.37, p= .264). No statistically significant difference was found amongst the three time periods: baseline (n=35), collar period (n=83), and control period (n=76) (Table 12).

IV. The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the number of apneic and bradycardic episodes when compared to the control period. There was no statistically significant evidence to support this hypothesis (F=1.04, p=.362). No statistically significant difference was found amongst the three time periods: baseline (n=10), collar (n=14), and control (n=20) (Table 12).

V. The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the number of apneic and oxygen desaturation episodes when compared to the control period. No statistically significant difference occurred in the baseline (n=31), collar (n=67) or control (n=34) periods to support this hypothesis (F=1.46, p=. 244) (Table 12).

VI. The use of a cervical collar in preterm infants receiving CPAP or HFNC will significantly decrease the number of combined apneic, bradycardic and oxygen desaturation episodes when compared to the control period. No statistically significant differences resulted in the baseline (n=36), collar (n=83), and control (n=38) periods to support this hypothesis (F= 3.15, p= .053) (Table 12).

Individual Changes in Physiologic Variables

Although statistical significance was not found, clinical significance was noted for several of the neonates. The majority of the infants experienced increases in isolated desaturation episodes, as well as combined apneic episodes during the collar period, and some infants experienced an increase in combination episodes during the control period. The increases concerned the Registered Nurses caring for the neonates. Clinical deterioration was evident, resulting in termination of the study.

Comparing the collar and control periods, 11 of 14 infants (79%) had an increasing number of events during the collar period; 6 of these infants (55%) experienced clinically relevant findings. Subjects 3, 4, 7, and 10 had an increase of 8 to 20 desaturation episodes in the collar period. Subjects 3 and 4 also had an increase of 9 to21 combined bradycardia and desaturation episodes with the collar. Subject 1 experienced an increase of 8 apnea and desaturation episodes, and an increase of 14 apnea, bradycardia and desaturation episodes during the collar period. Subject 5 had an increase of 35 bradycardic events with collar. These subjects were all randomized to the collar period in the last six hour period (Table 13).

There were also clinically significant findings noted during the control period with 4 of 13 subjects (31%) having increased events. Subjects 1 and 2 had an increase of 20 to 21 desaturation episodes; subject 4 experienced an increase of 8 combined apnea and bradycardia events; and subject 6 had an increase of 9 bradycardia events during the control period. Subjects 1 and 4 were randomized to the control period in the first six hour period, and subjects 2 and 6 were randomized to the control period in the last six hour period.

Table 13

Number of Episodes for all Variables for all Subjects

Subject	Collar Period	Baseline Period							Collar Period							Control Period						
		A	AB	AD	ABD	B	D	BD	A	AB	AD	ABD	B	D	BD	A	AB	AD	ABD	B	D	BD
1	2	6	0	1	6	0	15	2	2	0	14	19	0	27	1	3	0	6	5	0	47	7
2	1	2	1	8	15	0	4	1	0	0	4	10	0	16	6	6	5	2	5	4	37	5
3	2	0	0	0	0	0	12	1	0	0	0	1	4	43	23	0	0	1	4	0	23	2
4	2	0	3	0	2	0	1	2	0	0	1	6	5	17	17	0	8	0	4	3	7	8
5	2	0	0	0	0	7	5	1	0	1	0	1	45	4	0	0	1	0	0	10	7	0
6	1	0	0	0	0	0	12	0	0	0	0	0	1	41	4	0	0	7	0	10	37	0
7	2	0	0	1	1	0	11	0	0	1	8	5	1	25	4	0	1	11	3	0	15	0
8	1	1	2	0	0	4	0	3	3	5	1	2	10	5	7	0	0	1	0	6	11	0
9	1	7	0	3	3	0	16	1	1	1	6	6	3	10	0	1	6	7	6	2	8	0
10	2	0	1	6	1	1	12	1	0	0	2	13	4	40	4	0	0	3	12	2	32	6
11	2	0	0	0	1	0	7	0	0	0	2	3	2	0	0	0	0	0	0	1	4	3
12	2	0	2	8	0	0	23	0	0	2	24	13	4	37	1	No data collected						
13	1	0	0	2	2	0	22	0	0	0	0	4	2	40	3	No data collected						
14	1	0	0	0	0	7	22	12	No data collected							0	1	0	2	4	10	5
15	2	0	0	0	2	0	5	5	No data collected							0	1	0	1	1	14	37
16	1	1	0	2	5	0	9	6	0	0	0	6	3	13	9	No data collected						

Note. *Collar Period: 1-Collar applied in the first six hour period
2-Collar applied in the last six hour period

A-apnea
AB-apnea and bradycardia
AD-apnea and oxygen desaturations
ABD-apnea, bradycardia, and oxygen desaturations
B-bradycardia
D-oxygen desaturations
BD-bradycardia and oxygen desaturations

Predictors of Physiologic Variables

As the collar did not result in less physiologic events, other factors were explored to account for the noted trends in events. Specific demographic and health status variables were tested for effect on the dependent variables using multiple linear regression. The predictor variables examined included: ventilation at time of study (CPAP or HFNC), medications at time of study, age at time of study, gender, and days since last extubation. These variables were tested for effect on all the isolated and combined physiologic parameters. No significant predictors were found for all isolated and combined episodes (Appendix VI).

Chapter V

Discussion of Findings

In this study, a within-subject, repeated measures design was conducted with premature infants to test the effectiveness of a cervical collar in the treatment of apnea of prematurity. An overview of the findings related to the hypotheses will be discussed, followed by the limitations of the study and implications of the findings for future nursing practice and research.

Collar Effects on Apnea of Prematurity

There are three different types of apnea: central, obstructive, and mixed (Stark & Thach, 1976; Dransfield, Spitzer, & Fox, 1983; Barrington & Finer, 1991; Ruggins, 1991; Calhoun, 1996; Jenni et al., 1996; Lemke et al., 1998). Researchers suggest that all three types of apnea may all have one common obstructive condition involving the easily collapsible upper airway (Mathew, Roberts, & Thach, 1982; Ruggins, 1991; Stark, 1991; Thach, 1992; Holditch-Davis, Edwards, & Wigger, 1994; Lemke et al., 1998). The upper airway obstruction may be induced and aggravated with neck flexion (Thach & Stark, 1979; Wilson, Thach, Brouillette, & Abu-Osba, 1980; Dransfield et al., 1983; Ruggins & Milner, 1991). Historically, neck rolls have been used in the neonatal population to prevent neck flexion, however the movements and activity of the neonates often decrease the effectiveness of this intervention, and apneas persist (Grisemer, 1990). Interventions for the prevention of neck flexion in the neonatal population have not been investigated. The focus of this study was the use of cervical collars, as a non-invasive treatment, in the prevention of neck flexion-induced apnea.

The demographic characteristics of this sample were similar to those of previous

studies on the treatment of apnea of prematurity (Mathew, Roberts, & Thach, 1982; Dransfield, Spitzer, & Fox, 1983; Finer et al., 1992; Barrington, Finer, & Li, 1996; Lemke et al., 1998). The 16 preterm infants had a mean gestational age of 26 weeks and a mean study age of 31 weeks. All infants experienced apnea of prematurity before and during the study, with subject 11 experiencing 23 episodes in 15 hours and 30 minutes, and subject 1 experiencing up to 161 episodes throughout the same time period. The range in episodes was anticipated as all preterm infants experience apnea, in many different forms, to many different degrees (Finer et al., 1992; Lemke et al., 1998). Apnea of prematurity and its negative physiologic correlates become rare only when infants reach a post-conceptional age of at least 37 weeks (Henderson-Smart, 1981). The enrollment for this study was limited to infants with a gestational age of less than or equal to 32 weeks in order to capture apneic episodes amongst the entire sample. All infants were studied in the prone position in order to control for positioning effects (Levene & Mckenzie, 1989; Stark, 1991; Jenni et al., 1997). Parent handling was also limited during data collection to a period no greater than 30 minutes in length. However, handling was not recorded by the nurses, therefore the degree to which this was adhered to may have varied. Limiting the handling was done in attempt to control positioning effects and effects of environmental stimulation (Peters, 1999). All other aspects of care were routine, as disruption or prevention of the needs of the infant was to be completely avoided.

The major purpose of the study was to examine the effects of a cervical collar on apneic episodes in preterm infants. There was no significant sequence effect found for the order in which the infants received the collar treatment, whether it was in the first six

hour period or the last. The primary hypothesis was that the use of a cervical collar would significantly decrease the overall frequency of apneic episodes as measured by heart rate, respiratory impedance, and oxygen desaturations when compared to the control period. No statistically significant decrease was found amongst any of the physiologic variables during the intervention period. Apnea duration was also tested, and again no statistically significant findings resulted. Bradycardic episodes also did not decrease with the collar. However, heart rate was found to decrease significantly more in the collar period when compared to the baseline period. Furthermore, although oxygen desaturations did not decrease during the collar period when compared to the control period, a significant increase in the number of desaturation episodes did occur from the baseline to collar period. No statistically significant results were found in the severity (value) of oxygen desaturations between the collar and control period. The degree of oxygen desaturations worsened between the baseline and collar period, as well as between the baseline and control period. No significant changes in the number of combined physiologic variables occurred during the collar period.

The increase between the collar and baseline periods for the bradycardic rates and the oxygen desaturation episodes may have been caused by the cervical collar, but the findings are difficult to interpret as no statistical significance was detected when compared to the control period. Both the control and collar periods were six hours in length; whereas the baseline period was only a three hour study period, suggesting that perhaps more episodes were recorded due to the increased length of the study periods.

Eleven of 14 infants (79%) had an increasing number of events during the collar period, when compared to the control period. The variables that increased during the

collar period versus the control group included the number of isolated bradycardic and isolated oxygen desaturation episodes, as well as the number of combined apnea and desaturation episodes, combined apneic, bradycardic, and desaturation events, and combined bradycardia and desaturation episodes. The significant increases occurred when the collar was placed on during the last six hour period, suggesting that the physiologic deterioration may be due to time versus intolerance of the intervention. There were, however, 4 out of 13 infants (31%) who experienced negative physiologic effects during the control period versus the collar period. Two of the subjects were randomized to the control period in the first six hour period, and two were randomized to control period in the last six hours.

During the study, a number of the Registered Nurses, who were the primary caregivers of the studied neonates, also voiced concern with the suspected deterioration of the neonates in the study. Therefore, the data was distributed to two Neonatologists for feedback and guidance as to whether to continue with the study. Both Neonatologists concurred that the study should be discontinued due to the increasing trends of deterioration during the study period, therefore the study was stopped before the entire sample was recruited.

The fraction of inspired oxygen was also recorded during the study to determine the effect of oxygen content on the dependent variables. The device used to measure the fraction of inspired oxygen was available for 14 of 16 (88%) subjects. The oxygen content the sample received ranged from 21% (oxygen content at room air) to 42%. The oxygen content was increased to varying degrees, for all subjects, after several apneic episodes were documented. The degree of increase (and decrease) varied as no direction

or guidelines were provided for the oxygen concentration. Each Registered Nurse increased it as they felt was required for that particular infant. Once the oxygen content was increased, the number of apneic events would decrease to a minimum for varying lengths of time. However, when the oxygen content was lowered to its previous level, the episodes would begin to again increase. It was difficult to form a conclusion on the effect of this process variable as it varied to great degrees for all subjects. The data indicate, however, that an increase of oxygen does appear to decrease the number of apneic episodes for each infant (Miller & Martin, 1992).

Several demographic and health status factors were tested for their effect on the dependent variables. The effect of these predictors was analyzed using multiple linear regression. The predictors tested included: age at time of study (in weeks), ventilation means at time of study, gender, medications at time of study, and days since last extubation. The predictors were tested for effect on the number of episodes for all the isolated variables as well as all the combined variables. The age at the time of study was tested due to the effect it may have on the frequency of apnea of prematurity (Grisemer, 1990; Henderson-Smart, 1981), but this variable did not affect results. The type of ventilation was also considered, as two different ventilation means were accepted: high-flow nasal cannula and continuous positive airway pressure via nasal prongs (Sreenan et al., 2000). No differences were found. The number of days since last extubation was also considered as this may have an effect on apneic events, yet no effect was noted. Premature infants have the tendency to experience many apneic episodes shortly after extubation and/or if extubated too early in their respiratory course (Gitterman, et al., 1997). The gender was also tested as a predictor due to its potential effect on apnea, yet

again nothing was found of significance. However, authors of one study reported an effect of gender on apnea of prematurity, suggesting that female infants experience more respiratory pauses than do males (Holditch-Davis, et al., 1994). The types of medications were also examined due to their potential effects on apneic episodes, yet no differences were found (Hrabovsky & Mullett, 1986; Vandenplas, Deneyer, Verlinden, Aerts, & Sacre, 1989). These variables also did not affect the results.

Limitations of the Study

The convenience sample of premature neonates limits the generalization of the findings to this specific patient population only. The findings are exclusive to premature infants with similar characteristics. Also, the completed studies of only 16 infants weakened the validity of any conclusions.

The inability to monitor all types of apnea may have affected the results. Due to the lack of functionality of the end-tidal carbon dioxide device and nasal thermistor in this type of environment, only central apnea could be recorded. Many obstructive and mixed apneas may have been missed due to this lack in detection. The different modes of ventilation that were accepted may have also skewed the findings, as only one study has been done suggesting that high-flow nasal cannula is as effective as continuous positive airway pressure (Sreenan et al., 2000). The two different modes are routinely used in the NICU at the Royal Alexandra Hospital so both were accepted.

The various combinations of medications may have also altered the results. Each infant was on aminophylline and/or antiemetics and sedation. Research suggests that antiemetics may have a role in decreasing the frequency of apneic episodes, as does sedation (Hrabovsky & Mullett, 1986; Vandenplas, et al., 1989). Aminophylline given

intravenously (IV) or orally (theophylline) was accepted which may have also altered the findings, as research has suggested that IV aminophlline may have a more consistent and complete absorbency and may therefore be more effective (Jones & Baillie, 1979). However, other research suggests that the theophylline serum concentration may be maintained at a similar therapeutic level when either aminophylline or theophylline is administered (al-Omran & al-Alaiyan, 1997).

All the neonates were being fed completely enterally during the study via a nasogastric or orogastric tube. However, tube position, and whether the tube was indwelling or intermittent, was not assessed and these factors may have affected the number of apneic episodes experienced by the neonates (Symington, Ballantyne, Pinell, & Stevens, 1995).

A cervical collar has never been used and/or tested in the neonatal population; therefore no previous research exists for comparison. The model for the study's collar was created from the adult prototype used for neck stabilization after trauma and during anesthesia, as well as from the pediatric model used in the treatment of torticollis secondary to congenital shortening of the sternocleidomastoid muscle. Although, there were no statistically significant findings found, several modifications with the collar may be done. It was measured to fit snugly and to maintain the head in a neutral position relative to the trunk. The degree of neutrality was determined by the subjective opinion of the Occupational Therapist, therefore this may have fluctuated for each subject. The degree of head position relative to the trunk should be measured to ensure the same position is attained for each subject. As well, the collar may need to be made wider in order to ensure the head is maintained in a slightly extended position relative to the trunk.

The collar may need to be created with stiffer foam to decrease possible pliability, and ensure maintenance of the head position. Also, the collar may need to be made with lighter material as it may have been too heavy causing possible compression of the upper airway.

Different variables that were not controlled for may have also influenced the findings, such as the environmental setting (the lighting and noise) and anemia of prematurity (Grisemer, 1990; Peters, 1999). Infants are prone to experience more apneic episodes in a noisy, bright environment (Peters, 1999). Infants also tend to become more apneic when anemic due to their decreased oxygen carrying capacity (Grisemer, 1990).

Implications for Nursing

Apnea of prematurity is a significant health problem in the NICU that continues despite many different forms of interventions. Although several modes of treatment exist for apnea of prematurity, including ventilation and pharmacotherapy, they are not without their adverse effects, and neonates continue to experience apneic episodes and negative physiologic correlates despite these current treatments.

Many of the infants appeared to have a clinically significant deterioration in the collar period versus the control, suggesting that there may have been some intolerance to the intervention. However, no conclusions can be made on the effect of the cervical collars as some neonates also deteriorated during the control period. Several of the infants experienced less apneic episodes in the three hour baseline period versus the longer six hour study periods, suggesting that the length of time may also have influenced the number of episodes recorded.

It was apparent that the prevention of hypoxemia greatly affected the number of

apneic episodes, suggesting that perhaps the oxygen concentration be of primary concern when assessing the causes of apnea. As it is the discretion the Registered Nurse to adjust the oxygen content, the amount of oxygen delivered greatly varies for each patient. The oxygen concentration should be stringently monitored and increased by all nurses when caring for premature infants when apneic episodes are occurring, too often prolonged or multiple apneic events (isolated apneas or apneas accompanied by bradycardias and/or oxygen desaturations) are deemed individually acceptable (Grisemer, 1990).

Although all types of apnea may have an upper airway obstruction component, not all apnea can be treated and prevented with a device for the prevention of neck flexion. Many different causes of apnea of prematurity exist, and it is imperative that all possible factors be assessed prior to initiating treatment.

Conclusion

The purpose of this study was to investigate the effects of a cervical collar in preterm infants, less than or equal to 32 weeks gestation, on the incidence of apnea of prematurity. A within-subject, repeated measures design was conducted with a sample of 16 preterm neonates. The infants were commenced in one of two sequence order protocols, and the study was 15 hours and 30 minutes in length. The data revealed no statistically significant results in sequencing effects of the cervical collar. It was hypothesized that the cervical collar may be effective in the prevention of apneic episodes, relating to neck flexion, when compared to the control period, however this was not supported by the data. Statistically significant findings did result for several of the variables when the baseline and collar period was compared, however it is difficult to form any conclusions, as the two study periods differed in length of time. Clinically,

several of the neonates showed significant deterioration over time, and some during the collar period, therefore the study was stopped. To date, no research has been published on this type of intervention; therefore, the results of the study could not be compared to any previous findings. The study of cervical collars in the neonatal population for neck flexion-induced apnea has never before been done, therefore more research is needed to evaluate whether this intervention is effective. Many modifications could be made to the collar and several more variables could be controlled for in future studies. The most apparent finding was how effective an increase (from 2% to 10%) in oxygen concentration is in decreasing the occurrence of apneic events. The oxygen concentration of preterm neonates must be closely monitored and increased accordingly in order to help prevent apneic episodes in this type of population.

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Appendix 1

NEONATAL RESEARCH GROUP
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Canada T5H 3V9
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14 January 2000

To Whom It May Concern:

RE: **Project - "Use of Cervical Collars for Apnea of Prematurity"**
Principal Investigator: Denise F. Munro

We are familiar with and support the above project. We do not anticipate any difficulties in accessing the infants required for this protocol.

We wish Ms. Munro every success in this endeavor and eagerly await the results of this important study.

Sincerely,

Jeanne Van der Zalm, PhD, RN
Co-Chair, Neonatal Research Group

Robert P. Lemke, BMSc, MD, FAAP, FRCPC
Co-Chair, Neonatal Research Group

Philip C. Etches, MA, MB, FRCPC, FRCP(Lond), FAAP, FRCPCH, DCH
Director, Neonatal Intensive Care Unit, Royal Alexandra Hospital Site
Head, Section of Newborn Services, Capital Health Authority
Clinical Professor, Department of Pediatrics, Faculty of Medicine, University of Alberta

PCE/sdw

Royal Alexandra

Appendix II

INFORMATION LETTER

Title of Project:	Effects of Cervical Collars in the Treatment of Apnea of Prematurity
Principle Investigator:	Denise F. Munro, RN, MN Student Phone number: 477-4647 or 445-1155 (pager)
Co-Investigators:	Kathy Peters, PhD, RN Phone number: 492-4503 or 477-4760 Robert P. Lemke, MD Phone number: 477-4644 or 445-5547 (pager)

Your baby is premature and is being cared for in the Neonatal Intensive Care Nursery. One of the serious problems that often affects babies such as yours, is apnea. During apnea, babies do not breathe. This often happens because the baby's brain forgets to remind him/her to take a breath. Your baby is already receiving standard treatment but s/he is still having episodes of apnea. We are doing this study hoping to discover another way we can help the babies and decrease their apnea episodes. We think that a soft, foam neck collar will help because some apnea may be caused by the babies flexing their neck. This neck collar will help to keep babies from flexing their neck. It will be custom-made for each baby, and we think that it will help keep the airway open and stable so that apneas do not occur as often.

PROCEDURE: If your baby is in this study, he/she will be observed for apneas. We will be watching your baby for apneas by recording your baby's heart rate, breathing, and oxygen needs. All these recordings will be continuously done on a computer that will be beside your baby's bedside. This computer will be connected to the equipment that is already on your baby. This study will last for approximately sixteen hours. In these sixteen hours your baby will only be wearing the soft neck collar for six hours, and for the rest of the time, s/he will be monitored without the collar. At the end of the study, we will compare the amount of apneas your baby had with and without the collar. Nothing else will change in your baby's care except that your baby will wear a neck collar for a short period of time (six hours).

We hope that the neck collar will help to decrease apneas in premature babies. There is a chance though, that your baby might not tolerate the collar. Your baby might become upset and uncomfortable. We will closely watch your baby for any signs of discomfort and will stop the study. Also, your baby's neck might get irritated and reddened from the collar and its position on your baby's neck. In this situation, we will make some changes to the collar and readjust it. If the redness stays or worsens, we will stop the study. If, during the study, information is found that might change your decision to have your baby continue taking part in the study, you will be told as soon as possible.

RISK and BENEFIT: There will likely be no risk or harm, or any direct benefit, to your baby if you agree to have him/her take part in this study. This study is being done to help improve the care of premature babies.

VOLUNTARY PARTICIPATION: You do not have to consent to have your baby in this research study if you do not want him/her to be. If you agree to have your baby take part, you do have the choice to take your baby out of the study at any time by simply informing a member of the

research staff (Neonatal Research Office phone number: 477-4666).

CONFIDENTIALITY: Your baby's name will not be used in this research study. The researcher will erase your baby's name, and other notes that identify your baby, from all the information and results of the study. Your baby will only be known by a code number. All the results will be kept in a locked cabinet. After the research study is done, the results will be separated from the consent forms and the list of code numbers for seven years. The consent forms will be kept for five years. Once the study is complete, if we want to look at the data again later, we will go back and ask permission of the ethics committee.

We will submit information and results of this study for publication in health care journals. It might also be discussed at conferences. Only the results, and not your baby's name or any material that might identify your baby, will be used. If you have any questions or concerns about this study at any time, you can call the research staff at the number above or the Capital Health Authority Patient Concerns Office at 407-9790. The Patient Concerns Office exists to hear concerns that patients or parents have about care provided at Capital Health Authority Sites and has no direct connection with this research study or the investigators.

CONSENT FORM

Title of Project: Effects of Cervical Collars in the Treatment of Apnea of Prematurity

Principal Investigator: Denise F. Munro, RN, MN Student
Phone number: 477-4647 or 445-1155 (pager)

Co-Investigators: Kathy Peters, PhD, RN. Phone number: 492-4503 or 477-4760
Robert P. Lemke, MD, FRCPC. Phone number: 477-4644 or 445-5547 (pager)

Do you understand that you have been asked if your baby can take part in a research study?
YES NO

Have you read and received a copy of the information sheet?
YES NO

Do you understand the risks and benefits to your baby by having him/her take part in this study?
YES NO

Have you had the chance to ask questions and talk about the study?
YES NO

Do you understand that you can take your baby out of the study at any time, without having to give a reason and without his/her care being affected?
YES NO

Has confidentiality been explained to you?
YES NO

Do you understand who will be able to see your baby's medical records?
YES NO

Do you want the researcher to let your family doctor know that your baby is taking part in this research study?
YES NO

If so, please indicate the name of the doctor we should contact

_____

Who explained this study to you? _____

I agree to have my baby take part in this study:

YES NO

_____	_____	_____
(Signature of parent/ guardian)	(Date)	(Signature of witness)
_____		_____
(Printed name)		(Printed name)

I believe that the person signing this form understands the study and voluntarily agrees to have their baby take part.

_____ _____

(Signature of investigator or designee) (Date)

Appendix III

Monitoring Sheet

Please record the OFFSET NUMBER (the number of computer hours and minutes noted in the top right of the screen) at the START & FINISH of ALL activity and handling

OFFSET #	ACTIVITY AND HANDLING

Appendix IV

CERVICAL COLLARS DATA COLLECTION FORM

SUBJECT # _____ **Treatment Order Group _____**

History:

Name:_____ Randomization #:_____ Hospital ID #:_____

DOB:_____/_____/_____ Birthweight (grams):_____ Gender: M F
(year) (month) (day)

Gestational Age (weeks): Dates:_____ U/S:_____ Ballard Physical Assessment:_____

Weight Percentile:_____ Apgars:_____(1 min.) _____(5 min.) _____(10 min.)

Chronological Age at time of study (days, weeks):_____

Diagnosis:_____

C/S: Y N, Antenatal Steroids: Y N, if yes, how many doses? _____

Surfactant: Y N, if yes, how many doses?_____

Computer Acquisition

File Name:_____

Start Date:_____/_____/_____ Time:_____ Baseline Time:_____
(year) (month) (day)

Time without collar:_____ Time with collar:_____

Stop Date:_____/_____/_____ Time:_____ Problems with Collar: N Y
(year) (month) (day)

SUBJECT # _____ **Treatment Order Group _____**

Total number of isolated apneic episodes (> 20 seconds):

During baseline: _____, Without collar_____, With collar_____

Number of apnea with bradycardic episodes (>30% decrease from previous 60 seconds):

During baseline:_____, Without collar_____, With collar_____

Number of apnea with oxygen desaturation episodes (< 88%):

During baseline:_____, Without collar_____, With collar_____

Number of apneic episodes with both bradycardia and desaturation:

During baseline:_____, Without collar_____, With collar_____

Number of isolated bradycardic episodes (>30% decrease from previous 60 seconds):

During baseline:_____, Without collar_____, With collar_____

Number of isolated oxygen desaturation episodes (<88%):

During baseline:_____, Without collar_____, With collar_____

Number of isolated bradycardic and oxygen desaturation episodes:

During baseline:_____, Without collar:_____, With collar:_____

Ventilation

Days Ventilated:_____ Number of intubations:_____

(comments):_____

Date and Time of last extubation prior to this study: Date:_____/_____/_____
(year) (month) (day)

Time:_____

Days Supplemental oxygen:_____

Days NPCPAP:_____ Days HFNC:_____

SUBJECT # _____ **Treatment Order Group _____**

Feedings: Y N

Date started on feeds:_____/_____/_____ age:_____(days, weeks)
(year) (month) (day)

Date on full feeds:_____/_____/_____ age:_____(days, weeks)
(year) (month) (day)

Medications at time of study:

Antibiotics: Y N Type:_____ #Doses:_____ Duration:_____

Type:_____ #Doses:_____Duration:_____

Type:_____ #Doses:_____Duration:_____

Aminophylline/Theophylline: Y N, #Doses_____ Start date:_____

Last therapeutic serum level of theophylline:_____ Date:_____/_____/_____
(year) (month) (day)

Doxapram: Y N, Route: IV PO, Dose:_____ Duration:_____

Other: (include only sedation and analgesic and cisapride or maxeran)

	Type	Dose	Duration
1.	_____	_____	_____
2.	_____	_____	_____
3.	_____	_____	_____

COLLAR TOLERANCE/ COMMENTS:

_____

_____

_____

Appendix V

Study Protocol

1. Identification of an eligible infant

2. NRO staff to obtain consent

3. Reveal treatment order group by opening sealed randomization envelope

4. Negotiate with nursing staff and occupational therapist for date and time to start study

5. Have occupational therapist make the infant's collar

6. Calibrate and ensure proper functioning of monitoring and data gathering equipment

7. Ensure infant is in the prone position

8. Ensure all neck rolls have been removed

9. Start baseline period and continue until 180 minutes of useable data is collected

10. Move to next phase of the study (15 minute period—either application of collar or nothing)

11. Start 6 hour control or intervention period

12. Move to next phase of the study (15 minute period—either collar removal or application)

13. Start 6 hour control or intervention period

14. Conclude study and remove equipment from infant's bedside

The total recording session will be 15 hours and 30 minutes in length pending the activity and procedures experienced by the infant during the baseline period. If the infant undergoes many events during this time, the session will be lengthened.

Appendix VI

Multiple Regression Results for Prediction of Physiologic Variables Outcomes

Apneic Episodes

	R	R^2	Adjusted R^2	SEE	Change Statistics			
Model					**R^2 Change**	**F Change**	**df**	**Sig. F Change**
1	**0.430**	**-0.324**	**1.3991**	**0.185**	**0.185**	**0.363**	**5, 8**	**0.860**
		Unstandardized Coefficients		*Standardized Coefficients*	*t*	*Sig*		
Model		B	SE	Beta				
1	(Constant)	6.02	4.11		1.47	0.18		
	Age at Time of Study(wks)	-3.39E-02	0.09	-0.15	-0.42	0.68		
	Gender	-0.77	1.00	-0.30	0.77	0.46		
	Days Since Extubation	-2.547E-02	0.07	-0.15	-0.36	0.73		
	Medications	-0.18	0.25	-0.28	-0.72	0.50		
	Ventilation	-1.14	1.60	-0.31	-0.88	0.41		

Bradycardic Episodes

	R	R^2	Adjusted R^2	SEE	Change Statistics			
Model					**R^2 Change**	**F Change**	**df**	**Sig. F Change**
1	**0.452**	**-204**	**-0.293**	**2.3373**	**0.204**	**0.411**	**5, 8**	**0.829**
		Unstandardized Coefficients		*Standardized Coefficients*	*t*	*Sig*		
Model		B	SE	Beta				
1	(Constant)	10.16	6.87		1.48	0.18		
	Age at Time of Study(wks)	-4.51E-02	0.15	-0.11	-0.30	0.77		
	Gender	-1.91	1.67	-0.43	-1.14	0.29		
	Days Since Extubation	-5.68E-02	0.12	-0.19	-0.48	0.64		
	Medications	-0.26	0.42	-0.24	-0.62	0.55		
	Ventilation	-2.40	2.68	-0.31	-0.90	0.40		

Oxygen Desaturation Episodes

	R	R^2	Adjusted R^2	SEE	Change Statistics			
Model					**R^2 Change**	**F Change**	**df**	**Sig. F Change**
1	**0.592**	**0.351**	**-0.055**	**7.7898**	**0.351**	**0.864**	**5, 8**	**0.544**
		Unstandardized Coefficients		*Standardized Coefficients*	*t*	*Sig*		
Model		B	SE	Beta				
1	(Constant)	11.03	22.90		0.48	0.64		
	Age at Time of Study(wks)	0.16	0.51	0.10	0.31	0.77		
	Gender	-1.39	5.57	-0.09	-0.25	0.81		
	Days Since Extubation	-0.15	0.39	-0.14	-0.38	0.72		
	Medications	-1.61	1.40	-0.40	-1.15	0.28		
	Ventilation	10.16	8.93	0.36	1.14	0.29		

Combined Apnea and Bradycardia Episodes

	R	R^2	Adjusted R^2	SEE	Change Statistics			
Model					**R^2 Change**	**F Change**	**df**	**Sig. F Change**
1	**0.492**	**0.242**	**-0.232**	**1.1040**	**0.242**	**0.510**	**5, 8**	**0.763**
		Unstandardized Coefficients		*Standardized Coefficients*	*t*	*Sig.*		
Model		B	SE	Beta				
1	(Constant)	-1.15	3.25		-0.35	0.73		
	Age at Time of Study (wks)	-5.79E-02	0.07	-0.28	-0.81	0.44		
	Gender	0.54	0.79	0.25	0.68	0.51		
	Days Since Extubation	1.98E-02	0.06	0.14	0.36	0.73		
	Medications	0.14	0.20	0.26	0.70	0.51		
	Ventilation	1.59	1.27	0.43	1.26	0.24		

Combined Apnea and Desaturation Episodes

	R	R^2	Adjusted R^2	SEE	Change Statistics			
Model					**R^2 Change**	**F Change**	**df**	**Sig. F Change**
1	**0.695**	**0.483**	**0.160**	**2.6940**	**0.483**	**1.496**	**5, 8**	**0.291**
		Unstandardized Coefficients		*Standardized Coefficients*	*t*	*Sig.*		
Model		B	SE	Beta				
1	(Constant)	-2.89	7.92		-0.36	0.73		
	Age at Time of Study (wks)	-0.21	0.18	-0.34	-1.17	0.28		
	Gender	9.99E-02	1.93	0.02	0.05	0.96		
	Days Since Extubation	7.95E-02	0.14	0.19	0.59	0.57		
	Medications	0.45	0.48	0.29	0.93	0.38		
	Ventilation	6.45	3.09	0.59	2.09	0.07		

Combined Apnea, Bradycardia, and Desaturation Episodes

	R	R^2	Adjusted R^2	SEE	Change Statistics			
Model					**R^2 Change**	**F Change**	**df**	**Sig. F Change**
1	**0.451**	**0.203**	**-0.295**	**4.3217**	**0.203**	**0.408**	**5, 8**	**0.831**
		Unstandardized Coefficients		*Standardized Coefficients*	*t*	*Sig.*		
Model		B	SE	Beta				
1	(Constant)	8.47	12.70		0.67	0.52		
	Age at Time of Study (wks)	-8.04E-02	0.28	-0.10	-0.29	0.78		
	Gender	-0.17	3.09	-0.02	-0.05	0.96		
	Days Since Extubation	-0.20	0.22	-0.38	-0.94	0.38		
	Medications	0.72	0.78	0.35	0.93	0.38		
	Ventilation	-4.07	4.95	-0.29	-0.82	0.44		

Combined Bradycardia and Desaturation Episodes

	R	R^2	Adjusted R^2	SEE	Change Statistics			
Model					R^2 Change	F Change	df	Sig. F Change
1	**0.737**	**0.543**	**0.257**	**2.9027**	**0.543**	**1.899**	**5, 8**	**0.200**
		Unstandardized Coefficients		*Standardized Coefficients*	*t*	*Sig.*		
Model		B	SE	Beta				
1	(Constant)	11.03	22.90		0.48	0.64		
	Age at Time of Study (wks)	0.16	0.51	0.01	0.31	0.77		
	Gender	-1.39	5.57	-0.09	-0.25	0.81		
	Days Since Extubation	-0.15	0.39	-0.14	-0.38	0.72		
	Medications	-1.61	1.40	-0.40	-1.15	0.28		
	Ventilation	10.16	8.93	0.36	1.14	0.29		