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The Effect of Body Position and Head Rotation on Intracranial Pressure Readings in Premature Infants with Ladd Monitoring

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THE EFFECT OF
BODY POSITION AND HEAD ROTATION
ON INTRACRANIAL PRESSURE READINGS
IN PREMATURE INFANTS WITH LADD MONITORING

by

Martha J. Barthel

A Thesis Submitted to the Faculty of the Graduate School
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VITA

The author, Martha J. Barthel, was born September 24, 1954 in Evanston, Illinois.

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CHAPTER I

INTRODUCTION

The improved survival rates of premature infants can be attributed to advances in neonatal respiratory technology. As a result, periventricular – intraventricular hemorrhage and its neurologic sequelae have emerged as new problems and now surpass respiratory distress syndrome as the leading cause of neonatal morbidity and mortality (Reichert & Fuller, 1980; Tarby & Volpe, 1982; Volpe, 1981).

An increase in intracranial pressure (ICP) can be associated with varying degrees of intraventricular hemorrhage along with other neurological disorders and, if untreated, may progress to a life threatening situation in the neonate. In the monograph by Lundberg (1960) electronic monitoring of ventricular fluid pressure, using a polyethylene catheter connected to a pressure transducer, was first reported. Technological advances have resulted in an increased acceptance of ICP monitoring as a valuable technique for early detection and prompt therapeutic management of patients susceptible to increased ICP (Vidyasagar, Raju & Chiang, 1978; Bada, 1983; Vidyasagar & Raju, 1977). Monitoring of ICP is of critical importance in the management of infants and children with diseases that affect normal intracranial homeostatic mechanisms. The main objective of ICP monitoring is to detect variations in ICP pressure. Early identification of the causes
of these pressure variations may, in some instances, lead to amelioration of secondary brain changes due to increased ICP.

Since 1960, the major focus of research related to neurological problems has been on identifying characteristics of ICP in various disease processes and the underlying physiologic mechanisms responsible for the development of increased ICP (Langfitt, 1968; Symon & Dorsch, 1975; Shapiro, 1975). Only recently have investigators reported associations between routine patient care activities, such as: body position change (Mitchell & Mauss, 1978; Shalit & Umansky, 1977; Lundberg, 1960; Mitchell, Ozuna & Lipe, 1981; Perrin, 1981); head rotation (Mitchell et al., 1981; Shalit & Umansky, 1977; Bell, Lorig, Martin & Weiss, 1975; Goldberg, Joshi, Moscoso & Castillo, 1983); tracheal suctioning (Shapiro, 1975; Shalit & Umansky, 1977; Lundberg, 1960; Mitchell & Mauss, 1978; Perrin, 1981); painful procedures, for example venipuncture (Lundberg, 1960; Mitchell & Mauss, 1978; Perrin, 1981) and emotional upsets (Lundberg, 1960) with alterations in ICP.

The relationships between patient care activities and changes in ICP are of great interest to the medical and nursing fields in planning for patient care. To the author's knowledge, little research has been done on the neonatal population. Therefore, the focus of this study was to determine the effects of selected procedures on the ICP of premature infants as measured by the Ladd Monitor.

Need for the Study

Through technological progress ICP monitoring has become an acceptable practice for those patients with increased ICP. In the
neonate, particularly in the premature infant, increased ICP has been associated with intraventricular hemorrhage, post-hemorrhagic hydrocephalus, bacterial meningitis and hypoxic-ischemic encephalopathy (Volpe, 1981). Detection of alterations in ICP is important in the management of premature infants because of the potential for injurious effects on maturing neuronal structures secondary to sustained elevations of ICP. The literature provides few guidelines for the nursing care of premature infants with increased ICP. In neonatal intensive care units (NICU) there are usually routines which may dictate the timing of nursing care procedures or how infants are positioned. These routines will vary within and among both institutions and nurses. The use of such therapeutic appliances such as umbilical catheters, peripheral intravenous lines, intubation tubes may limit repositioning of the infant to head turning only. Even when total body repositioning is possible proper body alignment may not be maintained. It has been documented that relative to compressive forces and tension exerted on the internal jugular vein, venous blood flow may be diminished (Watson, 1974). This alteration of flow may precipitate changes in ICP.

A common practice utilized in the nursing care of critically ill patients, is to condense many of the daily nursing procedures into a shortened time frame to allow patients longer rest periods. Mitchell et al. (1981) found that the above practice resulted not only in increases in ICP, but also in successively higher baseline values of ICP. Although their findings were not significant, further investigation of this widely accepted nursing care practice is warranted.
Additional research is needed not only to identify various factors that may be associated with alterations in ICP, but also to test the validity and generalizability of the findings reported in the literature.

Statement of Problem

The problem addressed in this study was to ascertain the effects of six position changes on ICP in premature infants.

Purpose of Study

The present investigation was designed to determine the effects of head rotation to the right and left on ICP when the premature infant was in the supine position, and also to determine the effect on ICP of turning to each of four positions (i.e., supine to right lateral, right lateral to supine, supine to left lateral, left lateral to supine).

Hypothesis

The hypotheses to be tested in this study are:

1. Head rotation of the premature infant to the right or left will not produce a significant change in ICP.

2. Turning of the premature infant from the supine to right lateral position, right lateral to supine position, supine to left lateral position, and left lateral to supine position will not produce a significant change in ICP.

Conceptual Framework

The conceptual framework for this study is based on the physiology of increased ICP and adaptation. The normal range of ICP
for infants, both term and pre-term, is from 1 mmHg to 7 mmHg (Salmon, Hajjar & Bada, 1977; Welch, 1980; Bada, Menke & Khanna, 1980). The ventricular system is filled with cerebrospinal fluid (CSF) which is secreted by the choroid plexuses. The choroid plexuses are vascular networks that project into the lateral, third and fourth ventricles. The rate of cerebrospinal fluid production is estimated to be 0.3 - 0.4 ml/hr (Cutler, Page, Galicich & Watters, 1968). There are approximately 10 ml of cerebrospinal fluid present within the central nervous system at any one time, with 400 - 500 ml produced and reabsorbed daily. Cerebrospinal fluid flows from the lateral ventricle through the foramen of Munro into the third ventricle. From the third ventricle the fluid flows through the aqueduct of Sylvius into the fourth ventricle. From the fourth ventricle the fluid enters the central canal which extends through the inferior half of the medulla oblongata and through the entire length of the spinal cord. Cerebrospinal fluid leaves the fourth ventricle through the two foramina of Luschka and the foramen of Magendie and enters the subarachnoid space of the spinal cord and brain. As cerebrospinal fluid diffuses over the brain convexities it is reabsorbed into the circulatory system through the arachnoid villi which are located in the subarachnoid space and project into the dural venous sinuses. The major site for cerebrospinal fluid reabsorption is the superior sagittal sinus (Guyton, 1981; Snell, 1980; Moore, 1983). Normally, the pressure of the cerebrospinal fluid is regulated by the rate of cerebrospinal fluid production by the choroid plexus, and the resistance to absorption through the arachnoid villi (Guyton, 1981).
The adaptive mechanisms for pressure changes within the cranio­spinal compartment are explained on the basis of the Monroe-Kellie Doctrine in 1783 with Burrows Modification in 1846 (Langfitt, 1968). This modified Monroe-Kellie Doctrine can be explained as follows.

After the fontanels have closed and the cranial sutures have fused the cranium is essentially considered to be a non-distensible structure. The skull of the neonate, because of the presence of a fontanel and open sutures is considered to be a less rigid structure. In spite of the differences between the neonate and adult skull, this structure allows for only limited expansion of its contents.

Intracranial volume is composed of brain tissue, the intravascular system and the cerebrospinal fluid. Volume can be added to any of these compartments by a variety of mechanisms. For example, additional brain volume can result from tumor growth, hematoma or edema formation. Hypercapnia, profound hypoxia or acidosis can increase intravascular volume through vasodilitation, and cerebrospinal fluid volume can be altered by blockage of flow, increased production or decreased reabsorption. Any volume change in one of the major compartments, must be accompanied by a reciprocal change in one or more of the other compartments if the adaptive physiologic mechanisms are to maintain ICP within a normal range. Changes in ICP can be accommodated by the brain, under normal conditions, by a redistribution of intracranial volume. Another way in which the neonate may compensate for an increase in intracranial volume is by separation of his cranial sutures as observed by a progressive increase in head circumference.
Langfitt (1968) and Miller (1975) have identified compensatory mechanisms which allow for an increase in volume in any of the intracranial compartments before ICP begins to rise. These compensatory mechanisms consist of: 1) decreased formation of cerebrospinal fluid, 2) increased reabsorption of cerebrospinal fluid, 3) displacement of brain tissue water, and 4) reduction in cerebral intravascular volume by vasoconstriction. The degree of compensation or stretch within the system is referred to as compliance. Specifically, compliance expresses the ratio of change in volume which occurs secondary to change in pressure. Elastance, the inverse of compliance, refers to the change in pressure secondary to a change in volume (Langfitt, 1968; Miller, 1975). The ability to compensate for change in ICP is determined by the volume and rate of displacement of cerebrospinal fluid, intravascular blood, and/or brain tissue. Elastance is influenced by the rate at which volume expands within the intracranial cavity, so that a rapid increase of a given volume will produce a greater rise in ICP than if the volume change is slow. Because the cerebrospinal and intravascular volume compensating mechanisms are finite, any increase in volume that exceeds what can be displaced eventually exhausts the body's compensatory capacity producing a pronounced rise in ICP. Any additional volume at this point would produce a disproportionate and further dramatic rise in ICP.

The major risk of increased ICP is related to decreased cerebral perfusion which limits the availability of substrates needed to sustain cerebral metabolism and oxygenation resulting in ischemia and impaired cerebral function. Cerebral blood flow is primarily influenced by
arterial blood pressure (BP), partial pressure of carbon dioxide (PaCO₂), and partial pressure of oxygen (PaO₂). Cerebral blood flow is maintained through the process of autoregulation. Autoregulation refers to the change in the diameter of the resistance vessels in order to maintain continuous (relatively constant) cerebral blood flow over a broad range of perfusion pressures (Langfitt, 1968; Kuchinsky & Wahl, 1978). Autoregulation has been shown to be present in the brain of fetal and neonatal sheep (Purves & James, 1969) and in neonatal dogs (Hernandez, Brennan and Bowman, 1980). The exact mechanisms underlying autoregulation in the premature infant are currently not entirely understood. Lou, Lassen and Friis-Hansen (1979) noted that in premature infants cerebral blood flow appears to be passive to pressure such that cerebral blood flow does not remain constant between a range of arterial pressures as it does in the older infant or adult, but instead is labile in response to changes in systemic blood pressure. A possible explanation for the premature infant's lack of autoregulation was proposed by Haruda and Blanc (1981). They found that the walls of parenchymal cerebral arteries and veins in infants less than 30 weeks gestation were composed only of a layer of endothelium and lacked the smooth muscle, elastin and collagen necessary to maintain autoregulatory control of blood flow through vasoconstriction and vasodilation. Bada, Chua, Salmon and Hajjar (1979) recorded ICP during exchange transfusion in eight infants. Seven or eight infants were premature weighing less than 2500 grams. One infant was term and weighed 4050 grams. Data were reported on five of eight infants who had simultaneous mean arterial pressures (MAP) recorded. In all five
cases both ICP and MAP decreased during blood withdrawal and increased with infusion. A significant positive correlation \((P<0.001)\) was noted between ICP and MAP and the duration of blood withdrawal, such that when blood was withdrawn over a longer period of time ICP and MAP measurements were higher. Also noted was a significant negative correlation \((P<0.001)\) between ICP and MAP and duration of infusion time. This indicates with prolonged infusion time the ICP and MAP values are lower. Bada and associates state that the changes in MAP and ICP which corresponded to withdrawal and infusion of blood during exchange transfusion reflect alterations in circulating blood volume. These findings suggest a direct relationship between MAP and cerebral blood flow, and support Lou and colleagues (1979) hypothesis of impaired autoregulation in the preterm infant.

Cerebral blood flow may also be influenced by local metabolic factors that are vasoactive. Variations in arterial \(\text{PaCO}_2\) can produce changes in cerebral blood flow because elevated \(\text{PaCO}_2\) is a potent stimulus to vasodilation. Purves and James (1969) noted that cerebral blood flow increased approximately 7% for each mmHg that \(\text{PaCO}_2\) was raised over 40 mmHg. Rahilly (1980) using jugular venous occlusion plethysmography found significant increases in cerebral blood flow in seven term infants given 2% \(\text{CO}_2\) and air to breathe. Changes in \(\text{PaO}_2\) effects cerebral blood flow but to a lesser extent than alterations in \(\text{PaCO}_2\). In studies on fetal sheep, Purves and James (1969) demonstrated that a reduction of maternal \(\text{PaO}_2\) by 7 - 14 mmHg at a constant \(\text{PaCO}_2\) caused significant increases in cerebral blood flow through vasodilation. Rahilly (1980) noted significant decreases in cerebral
blood flow in eight infants breathing a mixture of 100% $O_2$ and air. The cerebral vessels are also sensitive to alterations in pH. Bucciarelli and Eitzman (1979) have shown in perinatal goats that acidemia produced an increase in cerebral blood flow. Based on these studies regulation of cerebral blood flow appears to be similar in both term and preterm animals. In summary, cerebral blood flow varies directly with $PaCO_2$ and pH levels, inversely with $PaO_2$ values, and may be passive to changes in arterial blood pressure in the premature infant.

As ICP increases a critical level may be attained at which further elevation is associated with decreased cerebral blood flow. This may be explained by the loss of autoregulatory mechanisms and compression of cerebral veins, thereby, obstructing flow from the venous sinuses (Shapiro, Langfitt & Weinstein, 1966; Osterholm, 1970). The resultant decrease in venous sinus drainage produces a back pressure which is projected to the cerebral arterial system causing a further decrease of cerebral blood flow. As blood flow progressively slows, cerebral ischemia occurs. Ischemia exacerbates the rise in intracranial pressure eventually leading to a total cessation of cerebral perfusion.

Patients with increased ICP demonstrate clinical manifestations which reflect dysfunction of the deep brain structures. Alterations in cardio-respiratory function are due to axial distortion of the brain stem affecting neuronal cell conductivity of the pontomedullary center which is responsible for respiratory and cardiovascular control (Thompson & Malina, 1959). Alterations in consciousness appear to
result from dysfunction of the ascending reticular activating and
limbic systems (Plum & Posner, 1980). Unfortunately, these are usually
late signs reflective of a decompensatory state, and if immediate ac-
tion is not undertaken to lower and control ICP death will ensue.

The dynamic changes which occur within the individual, in order
to maintain intracranial pressure within a normal range of values, can
be viewed within a nursing framework of adaptation. Adaptation is an
active and dynamic process which encompasses a range of protective
adjustments enacted by the individual in an attempt to maintain equi-
librium between oneself and one's internal and external environments.
These protective adjustments may be physiologic, psychologic and/or
sociocultural.

Adaptation theory has been utilized by Roy (1970, 1971, 1973,
1976) in a systems model as a basis for a conceptual framework for
nursing. Roy views man holistically and in constant interaction with a
changing environment. Roy's Adaptation Model recognizes man as a
biopsychosocial being who must respond to both internal and external
environmental changes. Adaptation varies from person to person and
changes over time within the individual. The level of adaptation,
according to Roy, is determined by the individual's ability to respond
to changing environmental stimuli. In this framework the environmental
stimuli are identified as focal stimuli, which is the particular factor
or situation that has changed; contextual stimuli, which comprises all
internal and external stimuli of the current situation that the person
is able to identify and measure; and residual stimuli which consists of
the inherent characteristics of the individual that are applicable to
that particular situation. According to Roy's Model adaptation occurs within a certain limit which is designated as the adaptation zone. If the focal, contextual and residual stimuli all fall within this zone a positive response to that particular situation will occur. If the above environmental stimuli fall outside the zone a negative response or maladaptation occurs. For Roy, adaptation is a positive response to stimuli that maintains or strengthens the integrity of the individual. Roy conceptualizes adaptation as occurring in one or more of four modes; the physiological mode, the self-concept mode, the role function mode and/or the interdependence mode.

In this study, the premature infant as a biopsychosocial being has been observed to determine the effect of selected body position changes upon intracranial pressure. According to Roy an adaptive response within the physiologic mode would be reflected in the ability of the infant to adjust to changing environmental stimuli, such as, head rotation and body position change by innate compensatory physiological mechanisms, and maintain homeostasis with minimal changes in intracranial pressure. Failure of these compensatory mechanisms results in elevated intracranial pressure. Within Roy's nursing framework, the goal of nursing practice is to manipulate internal and/or external environmental stimuli so as to facilitate the patient's adaptation, thereby maintaining ICP within an accepted range of values. This study will investigate the effects of nursing care activities (i.e., head rotation and body positioning) on the ICP of neonates.
CHAPTER II

REVIEW OF THE LITERATURE

Only since 1975 have medical and nursing clinical studies focused on the effect of patient care activities on ICP. Mitchell and Mauss (1978), in a descriptive pilot study, observed continuously for up to twenty-four hours eight patients, ages 21–76, to determine what patient-nurse activities were associated with transient or sustained rises in ICP. These patients were monitored by a pressure controlled external ventricular drainage system. The activities associated with the patient which resulted in elevations in ICP were: rapid eye movement (REM), sleep, painful procedures, tracheal suctioning, coughing, chewing, use of bedpan, conversation about the patient's condition, and position changes in bed. The investigators cited three activities that were not associated with changes in the patient's ICP; manipulation of patient tubing, bathing, and passive range of motion exercises. Cooper and Hulme (1966) observed elevations in ICP associated with REM sleep in patients with head trauma. It is believed that elevations in ICP are produced as a result of increased cerebral blood flow which occurs during REM sleep (Reivich, Isaacs, & Evarts, 1967). Increased cerebral blood flow is due to vasodilatation which is controlled by neuronal and humoral mechanisms, such as variations in autonomic tone and alterations in the levels of $\text{PaO}_2$ and $\text{PaCO}_2$ (Aserinsky, 1965; Meyer, Teraura, & Sakamoto, 1971).
Further documentation on the effects of various sleep stages on ICP in children was provided by DiRocco, McLone, Shimoji and Raimondi (1975). Using an extra ventricular catheter connected to a pressure transducer, ICP was continuously monitored for 24 hours in five hydrocephalic children ranging in age from 7 weeks to 8 years. Intracranial pressure values for all five patients were within the normal range of 1 - 10 mmHg during quiet wakefulness and rose above baseline levels during sleep. In three of five patients, correlations between sleep phase and pattern of ICP were obtained using an electroencephalogram. In these patients elevations in ICP were noted during the first stage of sleep and became more pronounced during the slow wave stage. Greatest increases in pressure were recorded during REM sleep at which time ICP rose in excess of five to seven times the baseline levels recorded during wakefulness. Although this study is limited, due to the small sample size, it clinically documents and supports the previous findings of Cooper and Hulme (1966).

Several investigators have reported that body positions were associated with changes in ICP. For instance, Nornes and Magnaes (1971) recorded supratentorial epidural pressure in fourteen patients ages one to 62 years during posterior fossa surgery. They found that neck flexion, extreme hip flexion and the prone position consistently produced elevations in epidural pressure readings. In this study elevated ICP was significantly reduced by reducing the degree of hip or neck flexion, or supporting the shoulders and pelvis with padding while in a prone position. Generalization of these findings is limited due to the specialized positions used during posterior fossa
surgery and the possible effects of anesthetic agents on ICP. Raju, Vidyasagar, Torres, Grundy and Bennett (1980) monitored ICP, using a Ladd Monitor, during intubation and anesthesia in ten infants from seven days to 10 months old. A mixture of halothane, nitrous oxide and oxygen was the anesthetic agent used for all infants. Four infants were intubated while awake and five infants were intubated after administration of curare. The mean pre-anesthetic ICP was 16.5 cmH$_2$O for both groups with a range of 8 - 25 cmH$_2$O. Infants undergoing intubation while awake had a significant increase in mean ICP to 89.7 cmH$_2$O. Intracranial pressure in infants intubated after administration of curare rose to a mean of 33.6 cmH$_2$O which was higher than baseline ICP but less than infants intubated in an awake state. The mean ICP throughout anesthesia stabilized at 27.1 cmH$_2$O for all infants and decreased to a mean of 14.3 cmH$_2$O following termination of anesthesia and extubation in six of 10 infants. The authors state that the neck extension required for intubation may result in elevated ICP through venous obstruction. Another factor responsible for elevated ICP, particularly during awake intubation is the effect of valsalva maneuvers encountered in a struggling child. However, the primary reason cited for increased ICP during intubation was the cerebral vasodilation caused by the anesthetic agents used. A secondary effect was attributed to the depressant myocardial effects of the anesthetics resulting in increased venous pressure and possibly decreased internal jugular venous outflow. The findings of this study suggest that the causes of elevated ICP during intubation and anesthesia are multifactorial.
Shalit and Umansky (1977) monitored ICP in 21 comatose patients with brain edema. Intracranial pressure was measured by both intra-ventricular catheter and subdural transducer in different patients. Their findings demonstrated that routine bedside procedures, (i.e., body position change, suctioning, head rotation, flexion or extension) often resulted in significant changes in ICP. The effect of changes in body position was studied 35 times in 11 of these patients. In seven of these 11 patients every change in body position was accompanied by either a significant increase or decrease in ICP. In the remaining four patients body position changes reportedly did not markedly affect ICP. The authors failed to state which specific body positions led to either increases or decreases in ICP, or to what level ICP was altered by these maneuvers. Intracranial pressure was noted to both increase and decrease following 28 observations of tracheal suctioning in seven patients. Decreases in pressure were noted to occur in those patients who had elevated pCO₂ levels prior to suctioning. Coughing, which followed suctioning in all patients, was noted to produce elevations in ICP. Consistency of patient response along with magnitude of pressure change were not reported. These findings support the early observations of Lundberg (1960) who reported that suctioning along with body position changes, precipitated increases in ventricular fluid pressure.

In the Shalit (1977) study, rotation of the head to right or left, and head flexion or extension, produced greater elevations in ICP than manual compression of both jugular veins in the supine position in 13 patients. With head rotation to either side, it was
stated that the ICP would return to baseline level if the body was rotated in the same direction. Again the authors failed to give numerical values for comparison.

Becht (1920) was the first to show that ligation of the jugular veins resulted in an increase in ICP that was proportional to the degree of occlusion. This increase in ICP was postulated as occurring secondary to the accumulation of venous blood within the cranial cavity. He suggested that position changes of the head compressed the jugular veins in the neck impairing venous outflow, thereby causing a rise in ICP. Watson (1974) studied the effect of head rotation on the internal jugular vein using venographic studies in 60 infants and children with various cardiac anomalies undergoing cardiac catheterization. His results indicated that rotation of the head to one side did not affect flow in the contralateral internal jugular vein. However, rotation of the head 45° caused narrowing of the middle third of the ipsilateral internal jugular vein in all cases. Rotation of the head 90° resulted in complete ipsilateral internal jugular vein occlusion in 41 of 60 children. In 24 of 41 cases, 75° of head rotation completely occluded ipsilateral internal jugular flow, and in 15 of those 24 cases only 60° of head rotation was required to produce the same effect.

Bell, Lorig and Weiss (1975) in their nursing observations reported that if the patient's head was not in line with the rest of the body, elevation in ICP would occur. This was hypothesized as the result of sternomastoid muscular compression of the internal jugular vein with resultant decrease in venous outflow. Hulme and Cooper
(1976) investigated changes in ICP associated with neck flexion, head rotation to the right and left and bilateral jugular vein compression in 18 patients. Intracranial pressure was recorded using both an intraventricular fluid pressure and a subdural pressure monitor. In the 17 reported cases, maximum increases in pressure were found to be associated with neck flexion in six patients, bilateral jugular vein compression in seven and with head rotation to the right in four patients. Lipe and Mitchell (1980) investigated how body position changes and head rotation affect the internal jugular vein. With the use of Ultrasound, it was documented in 10 patients that head rotation of 90° to the right or left produced partial or total occlusion of the ipsilateral internal jugular vein. However, since manual jugular vein compression alone did not result in an immediate rise in ICP in the Shalit and Umansky (1977) study, the authors suggested a combination of venous and CSF cisternal outflow obstruction, and some yet undefined factor which may be responsible for alterations in ICP with head rotation. The effect of head position change on ICP has also been reported in the pediatric population. Vidyasagar and Raju (1977) monitored the effect of head position on ICP using the Ladd Monitor in three groups of infants. Group one consisted of 30 healthy term infants with a mean gestational age of 39.7 weeks. The second group consisted of seven healthy preterm infants with a mean gestational age of 32.5 weeks, and the third group consisted of 15 sick preterm infants whose mean gestational age was 32.1 weeks. The major clinical diagnosis for this last group was respiratory distress syndrome. The positions selected for evaluation were horizontal, head up, or head
down. The positions were achieved by raising or lowering the head of the bed $15^\circ$. In general, ICP showed rapid increases for all groups in the head down position, and decreases following elevation of the head of the bed to below baseline values obtained in the horizontal position. However, no significant differences were found among the groups in the degree of ICP change when the head position was altered. Infants in Group III were noted to have higher values of ICP in both the horizontal and head up position as compared to infants in Groups I and II. Their higher ICP could reflect hypoxic changes which is often associated with varying degrees of respiratory distress.

Goldberg, Joshi, Moscoso and Castillo (1983) also investigated the effect of head position in 26 neonates. These infants had a mean gestational age of 33 weeks, were less than or equal to 10 days of age and weighed less than 2500 grams. Intracranial pressure was measured using the Ladd Monitor in the following head positions: head turned to the right with bed horizontal, head turned to right with bed elevated 30 degrees, head midline and bed horizontal, head midline and bed elevated 30 degrees. The results of this study showed that for the entire group the head midline position with either the bed horizontal or elevated to 30 degrees resulted in lower ICP values than the position of head right with the bed horizontal or elevated 30 degrees. These differences were not statistically significant. It was also noted that elevating the head of the bed 30 degrees resulted in lower ICP readings when compared to each corresponding flat head position. These differences were not statistically significant when ICP values were less than 7 cmH$_2$O. However, in infants whose baseline ICP values
were greater or equal to 7 cmH₂O in either the head right with bed horizontal or the head midline with bed horizontal positions, elevating the head of the bed 30 degrees resulted in statistically significant decreases in ICP. Unfortunately, the authors failed to cite in how many instances this particular finding was noted. Higher ICP values in the head right position are explained as the result of obstructed cranial venous outflow due to neck vein compression and supports similar findings by Shalit and Umansky (1977), Hulme and Cooper (1976), and Lipe and Mitchell (1980). Elevating the head of the bed also promotes hydrostatic venous drainage. The authors cite this as a factor responsible for the lower ICP values found in the elevated head positions.

According to Bell et al. (1975) ICP varies dynamically in response to respiratory patterns. Furuse, Ikeyama, Mabe, Hashuo, Kuchiwaki, Nakaya, Toyama, Tersoka, Nagai and Kageryama (1979) investigated the relationship between changes in respiratory pattern and ICP variations in seven postoperative brain tumor patients, ages 27 to 64 years. Intracranial pressure was measured via the epidural route. Respiratory pattern was measured by an impedance pneumogram. Their findings showed that type A and B variations in ICP waves were closely related to changes in the respiratory pattern. Type A pressure waves consist of sudden increases in ICP from a slightly elevated baseline to pressures of 50 mmHg or greater. Intracranial pressure remains at this elevated level for 5 - 20 minutes and then suddenly decreases. Type A pressure waves signify decreased cerebral elastance. Type B pressure waves consist of steep ascent or descent
phases, and are often associated with respiratory cycles. Type B waves represent alterations in cerebral vasomotor tone. Patients with both A or B pressure waves may exhibit signs of neurologic deterioration. In type A waves hypopnea and decreased tidal volume corresponded to ICP rise; whereas, tachypnea and/or hyperpnea resulted in a decrease in ICP. In type B waves rhythmic oscillations in ICP were closely related to breathing patterns, particularly when respirations had clearly defined apneic and hyperpneic cycles. Increases in ICP corresponded to the apneic phase while reduction in ICP coincided with hyperpnea. These findings indicate that poor ventilation and/or inadequate oxygenation with subsequent respiratory acidosis stimulates cerebral vasodilation in an attempt to increase the cerebral blood flow. The increased cerebral blood flow adds to the total intracranial volume resulting in a further elevation of ICP.

Apuzzo, Weiss, Petersons, Small, Kurze and Heiden (1977) studied the effect of positive and expiratory pressure (PEEP) ventilation on ICP in 25 adult patients with severe head trauma. Ten centimeters of PEEP was administered using a Puritan-Bennett Valve and MA-1 ventilator while ICP was measured using an intraventricular catheter. Intracranial volume pressure response was evaluated in all 25 patients to assess cerebral elastance. Twelve patients had normal values recorded for both baseline ICP and cerebral elastance. Administration of PEEP to these patients caused no change in ICP. Nine patients had normal baseline ICP along with increased cerebral elastance. Administration of PEEP to these patients resulted in a significant rise in ICP. The remaining four patients had an elevated baseline ICP along
with increased cerebral elastance. With the administration of PEEP three of the four patients had ICP that was double or greater than their baseline readings. One patient showed an increase in ICP from baseline, but this was not considered significant. It was also demonstrated that six of the 12 patients who manifested a significant elevation in ICP during PEEP had an associated decrease in cerebral perfusion pressure below 60 mmHg. The authors suggest that cardiovascular changes such as increased venous pressure, decreased venous return and decreased cardiac output, which may occur as a result of increased positive intrathoracic pressure following PEEP administration, may be partly responsible for elevations in ICP.

Based on the reports previously noted, Mitchell, Ozuna and Lipe (1981), systematically evaluated the effects of eight nursing activities on 18 patients ages 21 to 72 years. Intracranial pressure was monitored using an external ventricular drain connected to a manometer. Intracranial pressure was expressed in terms of ventricular fluid pressure. The eight nursing activities studied were flexion and extension of the hip and arm, head rotation to the right and left, and turning to four positions: supine to right lateral; right lateral to supine; supine to left lateral and left lateral to supine. No significant change in ventricular fluid pressure was found after passive flexion or extension of the hip or arm. This concurs with the findings of Mitchell and Mauss (1978) that passive range of motion (PROM) was not by itself associated with increases in ventricular fluid pressure. Increases in ventricular fluid pressure were noted only when PROM was performed with multiple activities within a short period
of time, however, this was not found to be statistically significant. Changes in body position produced increases in ventricular pressure for all turns except the supine to left lateral position. Ventricular fluid pressure values for the left lateral turn were found to be related to the patients' diagnostic category. Those patients with a subarachnoid hemorrhage had a mean decrease in ventricular fluid pressure, whereas, patients with posterior fossa lesions had a mean increase in ventricular fluid pressure. In general, changes in body position were consistent with the findings of Mitchell and Mauss (1978) and Shalit and Umansky (1977). Head rotation to the right was noted to produce greater increases in ventricular fluid pressure than head rotation to the left (Mitchell et al., 1981). Shalit and Umansky (1977) make no mention as to whether head rotation in one direction versus another produced a greater effect in ICP. Mitchell and her associates' (1981) findings regarding head rotation lend support to other investigators' postulations (Lipe & Mitchell, 1980; Watson, 1974; Shalit & Umansky, 1977; Hulme & Cooper, 1976; Becht, 1920) that obstructed venous flow may be a contributing factor to the elevation in ICP.

An interesting finding of the Mitchell et al. (1981) study was that the cumulative effect of nursing activities that were spaced 15 minutes apart (arm extension, hip flexion, supine to right lateral turn and head rotation to the right and left) resulted in successively higher, although not significant, baseline values of ventricular fluid pressure. No cumulative effects were noted with those activities spaced one hour apart. In patients whose intracranial compliance is
already compromised, the standard nursing protocol of condensing many nursing activities into a shortened time frame in order to allow for longer rest periods may actually be deleterious to such patients based on these findings. More research on this issue is warranted.

Bruya (1981) studied the effect of planned rest periods on ICP in 20 adult patients ranging in age from 20 - 70 years. The patients were equally divided into two groups. In Group I the patients received the routine nursing care of vital sign determination, respiratory toilet and bed bath without a rest period between activities. Patients in Group II were allowed a 10 minute rest period between each activity during which there would be no interruptions from any hospital personnel and extraneous noise would be controlled. Her findings identified increases in ICP in both groups with suctioning, respiratory inflation and turning patients to their side to perform back hygiene. In Group II the anticipated decrease in ICP following planned rest did not occur. For this group ICP values increased during the 10 minute rest period from a mean high of 11.8 to 14.7 mmHg following determination of vital signs, and from 15 to 18.6 mmHg following the activities of respiratory inflation, suctioning and oral care. According to Bruya these findings suggest that a 10 minute rest period is an insufficient amount of time to allow ICP to return to baseline values and that rest as defined here may not actually represent rest to the patient. It was also noted in this study that ICP decreased during the 10 minute period following the bath. The proposed reason for this decrease in ICP was due to the effect of returning the patient to a supine position from the 30 - 45° lateral
position required to perform back care rather than the effect of a
planned rest period.

Parsons and Wilson (1984) investigated the effect of six body
position changes on the physiologic variables of heart rate (HR), mean
arterial blood pressure (MABP), mean intracranial pressure (MICP) and
cerebral perfusion pressure (CPP) in 18 patients ages five - 67 years
with a diagnosis of severe closed head injury. Intracranial pressure
was measured using a subarachnoid bolt connected to a pressure trans­
ducer. The MABP and HR were monitored using an arterial catheter and
electrocardiographic leads respectively. These measuring devices were
connected to a Hewlett-Packard monitoring system for the recording of
data. Baseline measurements for the dependent variables of HR, MABP,
MICP and CPP were recorded prior to position change for each patient.
Immediately after a position change, the highest value for each vari­
able was recorded. Recovery values for the variables were again
recorded one minute following the position change. All patients had
baseline MICP's less than 15 mmHg and MABP's that were sufficient to
support a CPP of greater than 50 mmHg prior to initiation of the
study. In six of 18 patients turning from side to back with extension
of lower extremities produced increases in all dependent variables,
although significant values (p<.05) were found only for the MABP and
the CPP. Values for all dependent variables decreased with MICP
falling below baseline values at 1 minute postintervention. However,
none of the dependent variables were significantly higher or lower
than baseline readings. In 11 of 18 patients turning from a supine to
side position with flexion of upper and lower extremities produced
significant increases (p<.05) in the HR, MABP, and MICP. One minute after intervention the HR and MABP were not significantly higher than baseline, whereas the MICP remained significantly elevated. Range of motion exercises to the upper and lower extremities in eight of 18 patients showed significant increases for HR, MABP and CPP. At one minute following intervention only the MABP remained significantly higher than baseline values. Rotation of the head to the right or left in eight of 18 patients produced significant increases in MABP, MICP and CPP. All the dependent variables had returned to baseline values at one minute postintervention. In 12 of 18 patients elevating the head of the bed to 35° resulted in decreases for all dependent variables, but significant decreases (p<.05) were noted only for the MABP and MICP. One minute postintervention the MABP, MICP and CPP were significantly lower than the baseline values. Lowering the head of the bed from 35° - 0° produced significant increases in the HR, MABP, and MICP in 14 of 18 patients and remained significantly higher than baseline values at one minute postintervention. The CPP was not significantly altered by this position change.

The authors demonstrated that although the specified position changes produced both significant increases and decreases in the dependent variables, these fluctuations were within a safe range of values as determined by the CPP consistently remaining greater than 50 mmHg. This indicated that throughout the position changes blood flow to the brain was adequate. The authors further noted that even with wide fluctuations in HR and MABP, the MICP remained stable and did not fluctuate with the MABP in the majority of cases. This finding sug-
suggests that cerebrovascular autoregulatory mechanisms were functional for this particular group of patients.

This study clearly supports previous findings on the effects of position changes on ICP. However, the authors have provided additional information on how other physiologic variables can be used to accurately assess cerebrovascular status and subsequently plan patient care. Perrin (1981) in a descriptive study observed 13 patients ages 45 - 76 years for one hour, during a period of increased patient activity, to identify other conditions or procedures within the patient's environment that were associated with changes in ICP. A Ladd Monitor inserted into the epidural space was used to monitor these patients. Significant increases in ICP were found to occur with the following conditions: functional touch defined as any physical stimuli given to the patient for the purpose of performing nursing care; painful touch which consisted of agents other than invasive procedures which may be perceived by the patient as pain producing (e.g., sternal rub, nail bed pressure); invasive procedures, environmental noise, professional verbal stimuli, passive range of motion, spontaneous movement, and oral stimulation. Manual ventilation was found to produce the greatest changes in mean ICP readings. It was hypothesized that these findings may be secondary to variations in not only the amount, but pressure and time over which the oxygen is delivered during manual ventilation. Her findings support those of Apuzzo (1977). Other activities noted by Perrin (1981) which increased ICP included turning which is consistent with Mitchell et al. (1981) and Shalit and Umansky (1977); neck hyperextension which
supported the findings of Watson (1974), Hulme and Cooper (1976), and suctioning and coughing which are consistent with studies by Shalit and Umansky (1977), and Mitchell and Mauss (1978). Those activities found to result in significant decreases in ICP included personal verbal stimuli and elevation of the head of the bed (Perrin, 1981). Specific degree of the head of the bed elevation was not identified in the Perrin (1981) study. The activity found to produce increases and/or decreases in ICP was therapeutic touch (i.e., any non-painful stimuli given to the patient by the nurse, family or friend that may be perceived by the patient as soothing). According to Perrin (1981) therapeutic touch showed the least amount of change in ICP readings as compared to the other categories. The studies cited indicate a relationship between selected activities within the environment of the patient and alterations in ICP.

Since only a limited number of studies have been done in the neonatal population, and fewer with premature infants, data on the effect of patient care activities on variations in ICP are scarce. Therefore, an investigation into the effect of body position change and head rotation and its affect on ICP in premature infants would aid in expanding the current data base. As more information is attained recommendations for future study of how nurses can facilitate the neurologic adaptation of premature infants will be identified. It is hoped that the information obtained will provide guidelines for the nursing management of these patients and stimulate further research in this area.
CHAPTER III

METHODOLOGY

Research Design

A modified replication of the Mitchell et al. (1981) study which investigated the relationship of head rotation, turning and passive range of body motion on ICP readings in the adult population was used. A quasi-experimental design was used to determine variations in ICP in premature infants during head rotation to the right and left, also turning the infant from supine to the right lateral position, right lateral to the supine position, supine to the left lateral position, and left lateral to the supine position. The six activities listed above were performed while maintaining the patient in a horizontal position. This study was conducted between 1000 and 1600 hours in order to guard against circadian variations (Tom & Lanuza, 1976) in physiological functions (e.g., heart rate, respirations, and blood pressure), which may affect ICP. In order to control for the possible effect of infant activity level on ICP, infants were categorized into one of six activity states (Appendix A) identified by Wolf (1959).

Definition of Terms

1. Premature Infant: Any infant with a gestational age of 28 to 37 weeks and birth weight of 900 - 2500 grams. The infant must be in a Neonatal Intensive Care Unit.

2. Ladd Monitor: A fiber optic pressure sensitive device applied to
the anterior fontanel of the infant for the purpose of measuring variations in ICP.

3. Turning: A change in the orientation of the patient's body to the right, left or supine horizontal position.

4. Head Rotation: A rotation of the patient's head position to the right or left while the patient was in the supine horizontal position.

5. Intracranial Pressure (ICP): Force exerted by the brain tissue, cerebrospinal fluid or blood within the skull. The normal ICP in the preterm infant is 1 - 7 mmHg. In this study a measure of ICP was obtained by means of the Ladd Monitor.

6. Infant State: Wolf's classification of a group or pattern of behaviors regularly occurring together which aid in identifying the infant's degree of arousal at any given time.

7. Positive End-Expiratory Pressure (PEEP): A method of controlled ventilation that prevents alveolar collapse by providing increased end-expiratory transpulmonary pressure.

8. Fractional Inspired Oxygen (FiO): Refers to the measurable amount of oxygen being delivered to a patient.

Assumptions

The assumption basic to this study was that the Ladd Monitor is a valid and reliable tool for measuring ICP (Von Wild & Porksen, 1980; Raju, Vidyasagar & Rapazafiratou, 1980; Meyerberg, York, Chaplin & Gregory, 1980; Vidyasagar & Raju, 1977).
Limitations

In this study the lack of randomization was a limiting factor but for ethical considerations this was unavoidable. Although a control group was not used, each infant served as his/her own control. Random sequencing of activities also would have been preferred, but a set order of activities was chosen because it was most compatible with the Neonatal Intensive Care Unit's routine. Another possible limitation of this study involved the interaction between the researchers and subject which may have altered the subject's behavior. Characteristics of the patient population were dependent on the infants admitted to the Neonatal Intensive Care Unit. Consequently, generalizability of the findings are limited.

Setting

The setting was a Neonatal Intensive Care Unit at a large metropolitan mid-western medical center. The intensive care unit was a 29 bed, level III perinatal nursery. Infants were placed in open radiant warmers or incubators which were located along the periphery of one large room and on either side of a central dividing structure. The Nursing Station, Supply Area and Respiratory Therapy Station were adjacent to the Neonatal Intensive Care Unit. The Ladd Monitor was placed on a portable cart and positioned at the head of the bed.

Sample Criteria

A non-probability convenience sample was used for this study. Infants who met the following criteria were eligible for participation in the study.
1. A gestational age of 28 - 37 weeks as determined by standard obstetric history and Dubowitz physical assessment as confirmed by a physician.

2. A birth weight of 900 - 2500 grams.

3. Infants whose head circumference, total body length and weight were appropriate for gestational age as determined by chart records.

4. Birth age equal to or greater than 72 hours.

5. No congenital anomalies of the central nervous system or other major organ systems.

6. No chromosomal abnormalities.

7. No infants requiring shunting of their cerebral ventricles or external ventricular drainage.

8. No known skin diseases or lesions from adhesives which might have interfered with the application of the Ladd Monitor.

9. No known active infections.

10. Stable respiratory function as defined by:
    a) $FiO_2$ not greater than 30%.
    b) Breathing spontaneously or requiring no more than 60 mechanical breaths per minute at a peak pressure of no greater than 14 cm $H_2O$ and a PEEP of no more than 4 cm $H_2O$.
    c) No more than a 20% change in $FiO_2$, respiratory rate, peak pressure or PEEP within the 24 hours preceding the study.
    d) Absence of chest tubes.
e) Arterial capillary or transtracheal blood gases within accepted normal ranges.

f) No more than two apenic episodes associated with bradycardia (heart rate less than 100 beats per minute) for the eight hours period preceding the study.

11. Stable cardiovascular function as defined by:

a) Systolic blood pressure not less than 50 mmHg.

b) Apical pulse rate between 120 and 170 beats per minute.

c) No known congenital heart diseases, congestive heart failure, or persistent fetal pulmonary hypertension requiring treatment with surgery or medications such as: diuretics, Digoxin and Indomethacin.

12. No known metabolic problems as defined by:

a) Normal temperature for the 12 hours preceding the study.

b) Weight changes not exceeding an increase or decrease of 1 – 2% per day over the preceding 48 hours.

c) Normal urine output of 1.5 – 4 cc / Kg / hr over the preceding 24 hours.

d) If measured, normal serum values for sodium, potassium, glucose, calcium, magnesium, creatinine, blood urea nitrogen.
13. No infants of diabetic mothers.

14. Infants were excluded if they or their mothers (if infant is breast fed or less than 7 days old) were receiving any medications known to affect ICP, blood pressure, intrathoracic pressure, central venous pressure or body fluid compartments, such as anti-hypertensive agents, pressor drugs, sedative-hynotics or neuromuscular blockers. If the infants received IV fluids, total parenteral nutrition, vitamin supplements, electrolytes, antibiotics and theophylline they were included in the study.

15. Infants who were cared for in an isolette, open radiant warmer or crib were included in the study.

16. Consent for inclusion in study by a parent or legal guardian.

17. The primary physician and/or neonatologist, head nurse or clinical nurse specialist and the infant's primary nurse were consulted prior to including the infants in the study.

Discussion of Sample Criteria

During the initial phase after birth the premature infant undergoes changes in several physiologic parameters that may produce alterations in ICP which may not be directly related to simultaneous nursing care interventions. These physiologic parameters include changes in blood pressure, venous pressure, intrathoracic pressure, partial pressure of carbon dioxide (PaCO₂), partial pressure of oxygen (PaO₂), acid-base balance, temperature, electrolyte balance, fluid compartment size, fetal-neonatal circulatory adaptations, blood volume and hemo-
globin content. The aim of the selection criteria was to identify a
patient population in which the above mentioned physiologic parameters
were considered stable.

Requirements for defined ranges for gestational age, birth
weight, along with body measurements which were appropriate for
gestational age were chosen to specifically define a segment of the
premature infant population under investigation who are at greatest
risk for developing intraventricular hemorrhage and subsequent ele­
vation of ICP. A birth age of 72 hours or greater was chosen to insure
that infants had successfully completed their transitional period, had
adjusted to their environmental surroundings and were in a stable con­
dition. Infants with chromosomal or congenital abnormalities were
excluded as these infants were not representative of the normal pre­
mature infant. Infants with neurologic disorders requiring shunting of
their ventricles or external ventricular drainage were excluded as
these infants had alterations in the normal flow and reabsorption of
cerebrospinal fluid. Infants with active infections were excluded due
to their possible unstable condition and to avoid cross contamination.
Infants with unstable respiratory and metabolic states were excluded
due to their labile state and the effects of altered PaO₂, PaCO₂, and
acid-base balance on ICP. Infants with heart disease were excluded
because of abnormal structural and functional alterations in their
circulatory system which may affect ICP in those infants with deficient
cerebral autoregulatory control. Infants of diabetic mothers were
excluded because of the difficulty in assessing gestational age and
their potentially unstable metabolic state. Infants receiving any
medications known to affect intracranial pressure, blood pressure, intrathoracic pressure, central venous pressure or body fluid compartment size were excluded due to the direct or indirect action these agents may have on ICP.

Protection of Human Subjects

This proposal was approved by the Institutional Review Board (IRB) of the hospital in which this study was performed. The medical diagnostic workup, treatment, daily care, monitoring and outcome of the infant was not altered, impeded or necessarily improved by participation in this study. No known pain, discomfort or deleterious effects, both physically and psychologically, were rendered. Patient privacy and confidentiality were ensured by coding the data. All patients and their families participating in this study remained anonymous. As required by the IRB, the Parent or Legal Guardian was asked to complete the Consent Form (Appendix B).

Although patients participating in this study did not benefit directly from their participation, information obtained from this study may be used to meet future patients' needs. The findings of this study may also serve as an additional data base to be utilized for the development of guidelines in Nursing Management of premature neonatal patients.

Techniques for Data Collection

Instrument Selection

The Ladd Fiber Optic Pressure Monitor is an automatic system designed for safe and accurate monitoring of ICP. This monitor has
been in wide use since receiving Food and Drug Administration approval in 1976. The clinical value of this fiber optic method of measuring ICP has been documented for both premature and term infants by Von Wild and Pörksen (1980), Raju et al. (1980), Hill and Volpe (1981), Meyerberg et al. (1980), and Bada (1983).

The Ladd Monitor consists of a sensor, a monitor and a recorder. The sensor consists of a mirror mounted on a pressure sensitive diaphragm, three fiber optic cables and a pneumatic tube. Light transmitted through the light source fiber is reflected by the mirror to the two receptor fibers. When the mirror is in the neutral position each receptor fiber receives the same amount of light. A change in pressure acting against the diaphragm causes the mirror to move from the neutral position. The monitor contains a photoelectric detector that compares the amount of light returned by the two receptor fibers. Differences in light intensity causes the detector to activate a bellows which increases or decreases the air pressure inside the sensor to match the external pressure, thus returning the diaphragm and mirror to the neutral position. A pressure transducer in the bellows measures the air pressure applied to the sensor and this value is displayed on the digital readout of the monitor. The technical specifications for the Ladd Monitor are listed in Appendix C.

The Ladd Monitor used for this study was obtained from Ladd Research Industries, Inc. located in Burlington, Vermont. Prior to the initiation of the study the Ladd Monitor passed inspection from the Biomedical Electrical Engineering Department of the Medical Center in which the study was conducted. Insurance coverage for loss, damage,
and/or liability to the monitor was assumed by Ladd Research Industries, Inc.

**Application of the Monitor**

The method used to apply the Ladd Monitor required securing a small fiber optic sensor to the skin of the anterior fontanel with a soft and compliant self-adhesive foam material such as Reston. In infants with abundant hair it was necessary to shave a small portion of hair over the anterior fontanel prior to application of the monitor.

To insure uniform application of the Ladd Monitor the principal investigator applied the monitor using the two step technique described by Hill and Volpe (1981). In step one, half of the self-adhesive foam material was applied to the infant's scalp; then, under direct vision the fiber optic sensor was gently applied to the skin and manually held in place while the ICP reading on the monitor was recorded. In step two, the remaining half of the self-adhesive foam material was applied to the infant's scalp and another ICP reading was recorded. Correct application of the monitor was determined by obtaining identical ICP readings with the fiber optic sensor secured by the self-adhesive foam material, as in step two, and with the sensor manually applied to the anterior fontanel as in step one.

**Procedure**

The same order of activity occurred in all patients: head rotation to right and left, turning from the supine to right lateral position, right lateral to supine position, supine to left lateral position, left lateral to supine position. These procedures were
performed with the infant in a horizontal position. One research assistant performed all of the activities to decrease the amount of variability in the performance of these activities. The data which were collected by the principal investigator of this study, were recorded from the digital readout of ICP every 15 seconds for 5 minutes before and after each activity was performed. The length of time required to perform each activity was 15 seconds as determined by a stopwatch. Blood pressure, pulse rate, and respiration rate were obtained at the beginning and end of the data collection period for each subject. The following time sequence for data collection was utilized.

<table>
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<th>Rest</th>
<th>Data Collection</th>
<th>Activity</th>
<th>Data Collection</th>
<th>Rest</th>
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<td>5 Minutes</td>
<td>15 Seconds</td>
<td>5 Minutes</td>
<td>5 Minutes</td>
</tr>
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</table>

The duration of the study was approximately two hours and conducted between 1000 and 1600 hours. An example of the data collection form utilized for each activity is in Appendix D. An example of the patient profile used for recording demographic data is in Appendix E.

Reliability of Observations

The principal investigator was the only person who applied the monitor and collected the data. The principal investigator was instructed by a representative of the Ladd Research Industries, Inc. on how to read and calibrate the Ladd Monitor. Reliability coefficients of her accuracy in recording were .9.

Statistical Analysis

Descriptive statistics were utilized to describe the population. Frequency tables were used to analyze distribution with regard to age,
sex, and medical diagnosis. Inferential statistics were used to analyze the data. The mean, standard deviation (SD), variance and standard error of the mean (SEM) were computed for every 15 seconds for 5 minutes prior to the selected position change (preintervention measurements) and again for 5 minutes following that position change (postintervention measurements). An analysis of variance for repeated measures (ANOVA) was used to analyze the data with P_0.05 indicating statistical significance.
CHAPTER IV

RESULTS

Demographic and Physical Characteristics

As shown in Table 1, thirteen neonates with mean ± SEM gestational age of 31.4 ± .5 weeks (range 28 - 35 weeks) and a mean birth weight of 1521 ± 97 grams (range 900 - 2100 grams) were included in this study. There were eight white male, four white female and one black female infants. The mean Apgar scores at 1 and 5 minutes were 4.9 ± .6 (range 1 - 8) and 7.6 ± .3 (range 6 - 9) respectively. All infants were average for gestational age. All of the neonates were premature (Table 2). Other additional clinical diagnosis listed in their charts included respiratory distress syndrome (92%) / hyaline membrane disease (30%).

At the time of data collection (Table 2) the corrected mean age and weight of the infants were 34.77 ± .4 weeks (range 33 - 37 weeks) and 1755.38 ± 66.0 grams (range 1420 - 2330 grams). The apical pulse rate (range 148 - 170), respiration rate (range 48 - 60), blood pressure (range 88 - 78), and temperature (range 37.0 - 37.5°C. rectal) of each infant were within normal limits. All of the infants were on room air and breathing without ventilatory assistance. All infants were on 2 to 3 hour feedings with Dextrostix readings of 45 mg/DL to 90 mg/DL and received daily supplements of multivitamins. Four infants received Theophylline as medical management for their respiratory
<table>
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<th>Weight (grams)</th>
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<th>Apgar Scores 5 min.</th>
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Mean ± SEM

31.4 ± .5  1521 ± 97  4.9 ± .6  7.6 ± .3
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disease. Infants were categorized into three of six activity states identified by Wolf (1959) on the basis of observations made during the study (Appendix A). Eight infants were in stage two or active sleep, three infants were in stage one or quiet sleep, and two infants were in stage three or a drowsy state.

**Data Presentation**

**Position 1**

Position 1 involved rotation of the infant's head to the right while the infant was in the supine, horizontal position. During the first 5 minutes of data collection (preintervention), the mean ± SEM ICP for the entire group was noted to vary between a high of 4.0 ± .6 mmHg to a low of 3.4 ± .6 mmHg (Figure 1). An ANOVA for repeated measures showed no significant (F (19, 240) = .09, p>.05) variation in ICP over time within the preintervention period. After the infant's head was turned to the right, the mean ICP increased significantly (p<.05) from 3.4 ± .6 mmHg to 5.9 ± .8 mmHg. This increase in pressure represents a change of 75.7%. During the postintervention period the mean ICP varied between a high of 7.0 ± .7 mmHg to a low of 5.9 ± .8 mmHg. During the postintervention period there appeared to be a slight increase in ICP over time, but an ANOVA for repeated measures showed it was not a significant (F (19, 240) = .13, p>.05) variation in ICP over time within the postintervention period. However, for the entire 10 minute data collection period a significant (F (39, 480) = 4.35, p<.05) change in ICP occurred between the pre- and postintervention periods. A significant (F (1, 24) = 11.95, p<.05) change in ICP was also noted
Figure 1. Mean ICP ± SEM (vertical line) of 13 premature infants during the pre- (0-5 minutes) and postintervention (5.15-10 minutes) periods. Head rotation to the right occurred immediately after the preintervention period (5 minutes). The preintervention period significantly (p<.05) differed from the postintervention period.
between the fifth minute preintervention and the first and fifth minute postintervention period.

**Position 2**

Position 2 involved rotation of the infant's head to the left while the patient was in a supine, horizontal position. During the preintervention data collection period, the ICP for all infants varied from a high of $3.9 \pm 0.6$ mmHg to a low of $3.4 \pm 0.5$ mmHg (Figure 2). An ANOVA for repeated measures indicated there was no significant ($F (19, 240) = 0.07, p>0.05$) variation in ICP over time within the preintervention period. An immediate increase in ICP from $3.7 \pm 0.5$ mmHg to $7.6 \pm 1.1$ mmHg occurred upon turning the infant's head to the left. This significant ($p<0.05$) increase in pressure represents a change of 81.6%. During the postintervention phase there appeared to be a general decrease in ICP over time with a maximum pressure of $7.6 \pm 1.1$ mmHg decreasing to a minimum pressure of $5.6 \pm 0.6$ mmHg. However, the mean ICP remained markedly increased as compared to preintervention values.

Again, during the postintervention period an ANOVA for repeated measures showed no significant ($F (19, 240) = 0.45, p>0.05$) variation in ICP over time. However, for the entire 10 minute data collection period a significant ($F (39, 480) = 4.11, p<0.05$) change in ICP occurred between the pre- and postintervention periods. A significant ($F (1, 24) = 4.89, p>0.05$) change in ICP was also noted between the fifth minute preintervention and the first and fifth minute postintervention period.
Figure 2. Mean ICP ± SEM (vertical line) of 13 premature infants during the pre- (0-5 minutes) and postintervention (5.15-10 minutes) periods. Head rotation to the left occurred immediately after the preintervention period (5 minutes). The preintervention period significantly (p<.05) differed from the postintervention period.
Position 3

Position 3 involved changing the infant's position from the supine to the right lateral position. The ICP for the entire group varied from a high of $4.2 \pm 0.7$ mmHg to a low of $3.4 \pm 0.6$ mmHg during the preintervention period (Figure 3).

The ICP remained relatively constant with almost no variation occurring during the first 2 minutes of the preintervention period. An ANOVA for repeated measures showed no significant ($F(19, 240) = 0.08, p > 0.05$) variation in ICP over time within the preintervention period. Immediately upon turning the infant from the supine to right lateral position the mean ICP increased from $3.8 \pm 0.6$ mmHg to $5.5 \pm 0.8$ mmHg. This increase in pressure was not statistically significant although it represents a 35.2% change in mean ICP. During the postintervention phase, the ICP varied between a high of $5.8 \pm 1$ mmHg to a low of $4.8 \pm 0.7$ mmHg. An ANOVA for repeated measures also showed no significant ($F(19, 240) = 0.09, p > 0.05$) variation in ICP over time within the postintervention period and no trends were noted. No significant ($F(39, 480) = 0.76, p > 0.05$) variation in ICP was noted between the pre- and postintervention phases during the 10 minute data collection period, nor was there a significant ($F(1, 24) = 1.88, p > 0.05$) variation in ICP between the fifth minute preintervention and the first and fifth minute postintervention period.

Position 4

In position 4 the infant was turned from the right lateral to the supine position. During the preintervention period the mean ICP varied
Figure 3. Mean ICP ± SEM (vertical line) of 13 premature infants during the pre- (0-5 minutes) and postintervention (5.15-10 minutes) periods. Turning from the supine to the right lateral position occurred immediately after the preintervention period (5 minutes). No statistically significant differences were found between the pre- and postintervention periods.
Position 4:
Turning: Right Lateral to Supine

Figure 4. Mean ICP ± SEM (vertical line) of 13 premature infants during the pre- (0-5 minutes) and postintervention (5.15-10 minutes) periods. Turning from the right lateral to the supine position occurred immediately after the preintervention period (5 minutes). No statistically significant differences were found between the pre- and postintervention periods.
from a high of 5.5 ± 1 mmHg to a low of 4.8 ± .9 mmHg (Figure 4). An ANOVA for repeated measures indicated there was no significant (F (19, 240) = .03, p>.05) change in the ICP over time during the preintervention period. After turning the infant to the supine position the mean ICP decreased from 5.4 mmHg to 3.9 ± .7 mmHg. This decrease in pressure represents a ~29.6% change in mean ICP. During the postintervention phase of measurement, the mean ICP varied between a high of 4.1 ± .7 mmHg to a low of 3.1 ± .6 mmHg. Again, an ANOVA for repeated measures showed no significant (F (19, 240) = .13, p>.05) variation in ICP during the postintervention period. Also, the ANOVA for repeated measures indicated there was no significant (F (39, 480) = .90, p>.05) variation between the pre- and posttreatment periods during the 10 minutes of data collection nor was there a significant (F (1, 24) = .96, p>.05) change in ICP between the fifth minute preintervention and the first and fifth minute postintervention period.

Position 5

Position 5 involved turning the infant from the supine to the left lateral position. During the preintervention period, the ICP presented a relatively stable pattern with a minimum and maximum mean ICP of 4.1 ± .6 mmHg and 3.4 ± .6 mmHg respectively (Figure 5). An ANOVA for repeated measures confirmed that there was no significant (F (19, 240) = .10, p>.05) variation in ICP over time during the preintervention period.

Turning the infant from the supine to the left lateral position resulted in a nonsignificant increase in ICP from 4.1 ± .7 mmHg to 5.6
Figure 5. Mean ICP ± SEM (vertical line) of 13 premature infants during the pre- (0-5 minutes) and postintervention (5.15-10 minutes) periods. Turning from the supine to the left lateral position occurred immediately after the preintervention period (5 minutes). No statistically significant differences were found between the pre- and postintervention periods.
\[ - \pm 0.6 \text{ mmHg representing a } 31.3\% \text{ change in pressure}. \text{ During the postintervention phase of data collection, there appeared to be a slight decreasing trend in mean ICP from a maximum pressure of } 5.7 \pm 0.6 \text{ mmHg to a minimum pressure of } 4.5 \pm 0.6 \text{ mmHg.}

\text{During the postintervention period an ANOVA for repeated measures again showed no significant } (F (19, 240) = 0.03, p>0.05) \text{ variation in ICP over time. An ANOVA for repeated measures also confirms that there was no significant } (F (39, 480) = 1.13, p>0.05) \text{ variation between the pre- and postintervention phases during the 10 minute data collection period nor was there a significant } (F (1, 24) = 1.0, p>0.05) \text{ change in ICP between the fifth minute preintervention and the first and fifth minute postintervention period.}

\text{Position 6}

\text{In position 6 the infant was turned from the left lateral to the supine position. The mean ICP for the position presented a stable pattern from the preintervention through the postintervention phase with only a } 0.46\% \text{ change in ICP between pre- and postintervention measurements (Figure 6). An ANOVA for repeated measures showed that no significant changes occurred in ICP over time during both the preintervention } (F (19, 240) = 0.15, p>0.05) \text{ and postintervention } (F (19, 240) = 0.09, p>0.05) \text{ time periods. Also, no significant } (F (39, 480) = 0.11, p>0.05) \text{ changes in ICP were noted between the pre- and postintervention phases of the 10 minute data collection period. Similar to the previous finding no significant } (F (1, 24) = 0.04, p>0.05) \text{ variation in}
Figure 6. Mean ICP ± SEM (vertical line) of 13 premature infants during the pre- (0-5 minutes) and postintervention (5.15-10 minutes) periods. Turning from the left lateral to the supine position occurred immediately after the preintervention period (5 minutes). No statistically significant differences were found between the pre- and postintervention periods.
ICP occurred between the fifth minute preintervention, and the first and fifth minute postintervention period.

The preintervention mean ICP varied from a high of $4.9 \pm .6$ mmHg to a low of $3.9 \pm .5$ mmHg. Measurements of the mean ICP during the postintervention phase produced similar findings. The maximum and minimum mean ICP values during the postintervention phase were $4.7 \pm .8$ mmHg and $3.9 \pm .7$ mmHg respectively.
CHAPTER V

DISCUSSION

Head Rotation

The results of this study indicate that head rotation to either the right or left were the only position changes which produced marked and significant ($p<.05$) elevations in ICP. Therefore, the null hypothesis that head rotation to the right or left would not produce a significant change in ICP was rejected. Although preintervention values for ICP with head rotation to the right and left were similar, head rotation to the left resulted in initially higher ICP values than head rotation to the right. A possible explanation of why head rotation to the left may result in greater elevations of ICP may be derived from the anatomical position of the internal jugular vein.

The internal jugular veins are normally the largest neck veins and are considered the primary route for cerebral venous blood outflow. The internal jugular veins descend vertically through the neck and terminate posterior to the medial part of the clavicle. At this point the left internal jugular vein joins the left subclavian vein at a $90^\circ$ angle to form the brachiocephalic vein. However, the angle at which the right internal jugular vein joins the right subclavian vein to form the brachiocephalic vein is obtuse. In addition, the diameter of the right internal jugular vein is usually larger than the left as it receives a greater blood flow from the cranial cavity due to the anatomical position of the superior sagittal sinus (Romanes, 1964; 1966).
As a result of its smaller size and the angle at which it joins the subclavian vein the left internal jugular vein may be more sensitive to compression with position change. In turn, this sensitivity may be reflected in initially higher ICP values with head rotation to the left.

During the entire postintervention phase the ICP remained significantly (p<.05) elevated. However, a general decreasing trend in ICP was noted with head rotation to the left, whereas there appeared to be a slight increase in ICP over time with head rotation to the right. The 5 minute postintervention data collection period proved to be too short a time frame to allow the ICP to return to the preintervention levels and adequately assess whether or not the trends noted during this postintervention phase were an asymptotic or periodic phenomena.

It has been previously documented in adults (Lipe & Mitchell, 1980) through the use of ultrasound, and in children, (Watson, 1974) through the use of venographic studies, that head rotation to the right or left side either partially or totally occludes the ipsilateral internal jugular vein. It is possible that the combined effect of the anatomic position of the internal jugular vein as it traverses next to a relatively bony neck and clavicular area, coupled with compression from the sternomastoid muscle, results in varying degrees of occlusion of the internal jugular vein during head rotation. It has been postulated that the obstructed venous outflow may be a contributing factor to the elevated ICP values associated with head rotation (Becht, 1920; Watson, 1974; Hulme & Cooper, 1976; Shalit & Umansky, 1977; Lipe & Mitchell, 1980). Becht (1920) conclusively demonstrated that ligating
the internal jugular vein produced elevations in ICP secondary to accumulation of venous blood in the cranial cavity. Another contributing factor related to the elevated ICP values associated with head rotation could be related to cerebral circulation. Compared to the total body volume cerebral circulation is approximately four times greater in the infant than the adult (Volpe, 1981). As a result, this increased cerebral circulation might exacerbate rises in ICP secondary to internal jugular occlusion when the head is rotated to the right or left. Because the internal jugular veins are considered the primary route for cerebral venous drainage, it is possible that any degree of internal jugular occlusion produced through head rotation could result in decreased cerebral venous outflow. This decreased cerebral venous outflow results in cerebral venous stasis producing an increase in intracranial volume which is reflected in elevated ICP values. Although data was not recorded, the ICP was observed to return to pre-intervention values immediately upon repositioning the infant's head to the supine, horizontal position for all 13 subjects in positions 1 and 2. These findings are similar to those of Shalit & Umansky (1977) who reported immediate decreases in ICP with turning the patient's body toward the same direction in which the head was turned. Returning the infant's head to the supine horizontal position as in the present study or repositioning the body as Shalit & Umansky (1977) did, resulted in an improved body alignment with regard to the torso and head, and may have helped to facilitate cerebral venous drainage by decreasing the degree of internal jugular occlusion produced by head rotation. It is important to note that although head rotation to the right and left
produced statistically significant findings, these elevated ICP values remained within what is considered the upper limits of normal for the premature infant (Salmon, Hajjar & Bada, 1977; Welch, 1980; Bada, Menke & Khanna, 1980).

As reported by other investigators one of the most striking and consistent associations between position change and ICP elevations occurred with head rotation. The study supports the findings of Bell et al. (1975), Shalit and Umansky (1977) and Parsons and Wilson (1984) that head rotation to either the right or left results in significant increase in ICP. However, the authors cited above failed to mention whether head rotation in one direction versus another produced a greater effect on ICP. In the present study head rotation to the left produced initially greater elevations in ICP than head rotation to the right. This finding is in contrast to data reported by Hulme and Cooper (1976) and Mitchell et al. (1981) who stated that rotation of the head to the right resulted in greater increases in ICP. Mitchell et al. (1981) and Hulme and Cooper (1976) do not postulate why head rotation to the right resulted in higher ICP values. In this study it is suggested that the left internal jugular vein, due to its anatomical position and relationship to other structures (Romanes, 1964; 1966), may be more susceptible to the varying degrees of occlusion that has been documented to occur with head rotation (Watson, 1974; Lipe & Mitchell, 1980). This, in turn, could aid in explaining the higher ICP values secondary to obstructed cerebral venous outflow that were observed in the present study with head rotation to the left. The findings in the present study were similar to those of Goldberg et al.
(1983) in terms of the type of population under investigation. However, the major focus of their study was to examine only the effect of head position to the right and in the midline with the head of bed horizontal and elevated 30 degrees. Although Goldberg et al. (1983) reported greater elevations in ICP with head rotation to the right with the head of bed horizontal and elevated to 30 degrees, their findings were not significant which is in contrast to the statistically significant elevations in ICP encountered in the present investigations with head rotation to the right.

**Turning**

In this study turning resulted in both increases and decreases in mean ICP. However, none of these findings were statistically significant. Therefore, the null hypothesis that turning from the supine to right lateral, right lateral to supine, supine to left lateral and left lateral to supine positions would not produce a significant change in ICP was not rejected.

Nonsignificant increases in ICP occurred with turning the infant from the supine to both the right and left lateral position. Preintervention values for ICP were similar for the right and left lateral turns. Turning the infant to the left lateral position resulted in slightly higher ICP values than turning the infant to the right. During the postintervention phase, values for the ICP were again similar for the right and left lateral positions. The left lateral turn, however, showed much less variation in ICP than the right lateral turn. Also, a slight decreasing trend in ICP was noted with the left lateral turn. No discernible trend in ICP was noted with turning the infant to
the right lateral position; although the postintervention values for the right and left lateral positions were not significant, the ICP remained elevated for both positions throughout the data collection period. This suggests that the allotted time frame for data collection may have been too limited to adequately assess trends in ICP.

It was unexpectedly noted that the ICP values for two subjects decreased with turning to the right or left lateral position from a supine position and increased with turning to the supine position from the right or left lateral position. Upon inspection, these infants were noted to have a more elongated head shape along the anterior-posterior axis with a prominent occiput which resulted in forward flexion of the neck when the infants were in a supine position. Nornes and Magnaes (1971) reported associations between neck flexion and elevations in epidural pressure readings during routine positioning of patients for posterior fossa surgery. Hulme and Cooper (1976) suggest that impairment of collateral cerebral venous and CSF outflow is the mechanism most likely to result in elevated ICP readings associated with neck flexion. The majority of cerebrospinal fluid initially flows inferior to the basal cisterns and then upward within the subarachnoid space over the cerebral cortex to be reabsorbed by the arachnoid villi. It is postulated that neck flexion may partially or completely obstruct the basal cisterns resulting in increased cerebrospinal fluid volume within the cranial cavity. This, in turn, may lead to increased ICP values. It is also suggested that neck flexion may reduce collateral venous outflow through compression of the anterior and posterior cerebral veins and, thereby, increase the intracranial blood volume with a
resultant increase in ICP even in the absence of internal jugular vein compression. It is postulated that this neck flexion with possible subsequent cerebrospinal fluid and collateral cerebral venous obstruction may have been responsible for the higher ICP values noted in the supine position of these two subjects, and that the lateral position provided for better body alignment resulting in lower ICP values.

There is very little literature that aids in explaining the dynamics of the ICP change that occurs with turning the body of the adult or infant. With changing the body position from the supine to the lateral position there may be some degree of both forward and/or lateral neck flexion along with flexion of the knees and hips. The most common explanation cited for increases in ICP with turning from the supine to the lateral position involves compression of the internal jugular vein resulting in pooling of cerebral venous blood and subsequent increases in ICP (Shalit & Umansky, 1977; Mitchell & Mauss, 1978; Mitchell et al., 1981; Parsons & Wilson, 1984). The mechanism described by Hulme and Cooper (1976) of forward neck flexion resulting in impaired collateral cerebral venous and cerebrospinal outflow also cannot be discounted as a possible factor. Hip flexion which was reportedly associated with elevations in ICP in the lateral position in the Mitchell and Mauss (1978) study and in the sitting position by Nornes and Magnaes (1971) may be another contributing factor. However, the mechanism relating hip flexion to elevations in ICP is not explained. Parsons and Wilson (1984) in attempting to explain the elevations noted for heart rate and blood pressure in their study remind the reader that turning involves movements of large muscle
groups which produces a massage effect upon the vasculature. This causes an increase in systemic venous return which results in an increase in the stroke volume of the heart leading to an increased cardiac output. In premature infants who may have immature autoregulatory mechanisms this increase in cardiac output may contribute to a rise in ICP. However, due to the lack of muscle mass in the preterm infant it is uncertain whether the movements of muscle groups elicited through turning produce any effect on the systemic vasculature. Respiratory patterns and the use of mechanical ventilation have also been implicated as factors which may be responsible for altered ICP values. It has been reported by Furuse and associates (1975) that changes in respiratory pattern produced alterations in ICP. They found that hyperpnea was associated with decreases in ICP secondary to vasoconstriction due to blowing off CO₂, and hypopnea and/or apnea was associated with increases in ICP secondary to vasodilation resulting from elevated CO₂ levels. Apuzzo and associates (1977) postulated that the administration of PEEP which results in an increased intrathoracic pressure produces cardiovascular changes such as increased venous pressure, decreased venous return and decreased cardiac output which may result in an increase in ICP by inhibiting cerebral venous drainage. Although respiratory patterns and the use of mechanical ventilation have been shown to alter ICP, in this study none of the infants required ventilatory support nor did they present any abnormal breathing patterns.

The association of turning the patient in bed with increases or decreases in ICP has not been as consistent as findings regarding head
rotation. In general, the findings of this study demonstrated that turning produced both increases and decreases, although not statistically significant, in ICP. This is consistent with findings by Shalit and Umansky (1977). However, Shalit and Umansky (1977) failed to state which specific body positions were associated with either increases or decreases in ICP and whether or not these findings were statistically significant. Therefore, it is difficult to draw further comparisons with the Shalit and Umansky (1977) study.

In the present study increases, although statistically nonsignificant, were noted with the supine to lateral position turning combinations. This is consistent with the findings of Bruya (1981) and Parsons and Wilson (1984). However, the authors do not make a distinction between the right and left lateral turn in their studies. The increases in ICP noted during turning in this study also support findings of Perrin (1981). Although Perrin (1981) makes a global statement that increases in ICP were associated with turning, she does not specifically delineate to what positions subjects in her study were turned. The present investigation was similar to the Mitchell et al. (1981) study in terms of design, data collection and data analysis. However, the findings of Mitchell and associates (1981) varied with regard to the supine to lateral turning combinations. In the Mitchell et al. (1981) study the supine to right lateral turn consistently produced increases in ICP which is supported by the present investigation. The supine to left lateral turn in the Mitchell et al. (1981) study, however, resulted in increases or decreases in ICP depending on the subject's diagnostic category; whereas, in the present study only increas-
es in ICP were noted. The nonsignificant increases found only with turning from the supine to the lateral positions in this study is in contrast to Mitchell and Mauss (1978) who reported that turning in any direction was associated with elevations in ICP. Again, specific directions of position change were not identified in the Mitchell and Mauss (1978) study.

In the present study nonsignificant decreases occurred with turning the infant from the right lateral to the supine position. This was similar to findings of Bruya (1981) and Parsons and Wilson (1984), who also noted decreases in ICP with turning the patient from a lateral to a supine position. Parsons and Wilson (1984) and Bruya (1981) again do not differentiate the right from left lateral to supine turning combination in their studies. However, the findings of the present study along with those of Bruya (1981) and Parsons and Wilson (1984) are in contrast with Mitchell and associates (1981) who reported that turning from both the right and left lateral position to the supine position consistently resulted in increases in ICP. It is postulated in this study that the associated decrease in mean ICP, although not statistically significant, may have been due to an overall improvement in body alignment in the infant which may have led to an improved cerebral venous and cerebrospinal fluid outflow from the cranial cavity.

No change in ICP from the preintervention through the postintervention periods occurred in the present study with the left lateral to supine turn. Based on findings from previous turning combinations in this study and on results from other investigators, Mitchell and associates, 1981; Parsons and Wilson, 1984; Bruya, 1981, this absence of change in
ICP was an unexpected finding and is unsubstantiated in the literature. Again this suggests that the five minute data collection period in the postintervention phase may have been inadequate to assess trends in ICP. Finally, it must be stressed that although in this study turning was associated with both increases and decreases in ICP these findings were not statistically significant and the range of ICP values remained well within the normal limits for all position changes during both the preintervention and postintervention time periods.

Limitations

The major limitations of this study have previously been addressed. However, during data analysis it was noted that ICP values did not return to baseline values during the postintervention period with head rotation to the right and left, and with turning from the supine to the right and left lateral positions. This indicates that the allotted time frame for data collection in this study was too limited to adequately assess temporal trends in ICP in response to a given stimulus. In order to provide data that can be generalized a much larger and heterogenous population needs to be studied. The results of this study add information to the growing data base of neuroscience nursing, but cannot be used to establish norms of nursing care for these patients.

Recommendations for Further Study

The primary recommendation of this investigator is to have this type of study replicated with a larger population. In a larger study variables such as gestational age, presence of IVH or other disease
states, requirement for assisted ventilation, activity level, feeding time, or other factors could be isolated and analyzed in relation to ICP with position change. This would greatly enhance generalizability. Another recommendation would be to lengthen the time of data collection in an attempt to analyze trends in ICP over time with emphasis placed on determining the length of time required for ICP to return to baseline levels following any given activity or stimulus. Because of the limited information available on factors affecting intracranial pressure in the neonatal population, another consideration is to conduct a descriptive study. This would help to identify other factors impacting on intracranial pressure which could then be systematically evaluated. Further studies, based on evidence from Parsons and Wilson (1984), could also investigate the physiologic variables of heart rate, blood pressure, ICP, cerebral blood flow, cerebral perfusion pressure, $PCO_2$ and $PO_2$ related to specific position changes in premature infants. This type of study would enable nursing to more accurately determine, by combining a variety of information, which factors have either a deleterious, beneficial or neutral effect on intracranial pressure. By investigating correlations between variables more information will be obtained that will make formulation of nursing care for premature infants with altered intracranial pressure status more accurate and generalizable.
SUMMARY

Intracranial pressure was recorded using the Ladd Monitor on 13 infants between 33 and 37 weeks of age. The primary clinical diagnosis for all infants was prematurity. Other clinical diagnosis included respiratory distress syndrome in 92% / hyalin membrane disease in 30%. Intracranial pressure was recorded on all patients for each of the six standardized position changes every 15 seconds for 5 minutes prior to the position change and again every 15 seconds for 5 minutes following the selected position change. Increases in ICP were noted with the following position changes, head rotation to the right and left, and turning from the supine to the right and left lateral positions. However, statistically significant elevations in ICP occurred only with head rotation to the right and left. Decreases, although not statistically significant, in ICP were noted in the right lateral to supine turn. However, with the left lateral to supine turn no demonstrable changes in ICP from baseline through postintervention were noted. With all position changes ICP remained within the range of normal values for the premature infant. Because both the identification and systematic analysis of factors which impact on ICP in the premature infant has had only limited investigation to date further study in this area is needed and encouraged.
REFERENCES


Rahilly, P. Effects of 2% carbon dioxide, 0.5% carbon dioxide, and 100% oxygen on cranial blood flow of the human neonate. Pediatrics, 1980, 66 (5), 685-689.

Raju, T., Vidyasagar, D., & Rapazafiratou, C. Intracranial pressure monitoring in the neonatal ICU. Critical Care Medicine, 1980, 8 (10), 575-581.


Thompson, R., & Malina, S. Dynamic axial brain stem distortion as a mechanism explaining the cardiorespiratory changes in increased intracranial pressure. *Journal of Neurosurgery*, 1959, 16, 665-675.


APPENDIX A
# Infant Activity States

<table>
<thead>
<tr>
<th>State</th>
<th>Characteristic Behaviors</th>
</tr>
</thead>
<tbody>
<tr>
<td>State 1:</td>
<td>Lies still with only occasional startle, twitch or sucking movement. Breathing regular.</td>
</tr>
<tr>
<td>Quiet sleep</td>
<td></td>
</tr>
<tr>
<td>State 2:</td>
<td>Rapid eye movement (REM), irregular breathing, brief smiles, or crying sounds.</td>
</tr>
<tr>
<td>Active sleep</td>
<td></td>
</tr>
<tr>
<td>Drowsy</td>
<td></td>
</tr>
<tr>
<td>State 4:</td>
<td>Wide-eyed and focused on environment. Regular breathing and minimal activity.</td>
</tr>
<tr>
<td>Quiet, alert</td>
<td></td>
</tr>
<tr>
<td>State 5:</td>
<td>Eyes open, much body and facial movement. Low tolerance for hunger or fatigue. Breathing irregular.</td>
</tr>
<tr>
<td>Active, awake</td>
<td></td>
</tr>
<tr>
<td>State 6:</td>
<td>Much motor activity, clinched eyes, irregular breathing.</td>
</tr>
<tr>
<td>Crying</td>
<td></td>
</tr>
</tbody>
</table>

APPENDIX B
Patient's Name__________________________________________Date:__________________

Project Title: The Effect of Body Position and Head Rotation on Intracranial Pressure Readings in Premature Infants with Ladd Monitoring.

Patient Information:

Previous studies on adults have shown that various activities such as turning from side to side may result in changes in intracranial pressure (i.e., pressure within the head). Since such procedures are daily functions of patient care the purpose of this study is to determine what effect, if any, procedures such as body and head position changes have on the pressure within the head of premature infants.

To study the effect of body and head position changes on the pressure within the head requires placing a small monitor on the anterior fontanel (soft spot) of your baby's head. This monitor is used to detect pressure changes that occur inside the head. The monitor is placed on the skin over the anterior fontanel and held in place by a soft self-adhesive foam material. In infants with abundant hair it is necessary to shave a small portion of hair over the anterior fontanel prior to application of the monitor. In many cases the hair has already been shaved to allow for the administration of IV fluids.

Once the monitor is in place I will observe the digital readout on the monitor and record every 15 seconds for 5 minutes before and after your baby's body position is changed by my assistant from back to right side, right side to back, back to left side, left side to back; and head is positioned to the right side and then left side while your baby is lying on his/her back.

The time table for the study is as follows: First, we collect our information from the monitor for 5 minutes. Then, one of the above activities is done by my assistant. We again collect information from the monitor for 5 minutes. After that we wait 10 minutes before starting the procedure over again with the next activity. Using this time table the duration of the study is approximately 2 hours.
Potential Benefits and Risks

Taking the reading simply involves recording the digital readout on the monitor. There are no known or anticipated risks involved in the reading or application of the monitor. No pain or discomfort is associated with the reading or application of the monitor. In some cases only a small amount of hair over the soft spot will be shaved.

It is not expected that your baby will directly benefit from participation in the study; however, we hope that with the results of this study, nursing can gain more knowledge about what functions affect patient conditions and use that knowledge to improve care for infants in the future with similar problems.

There will be no cost involved for participating in this study.

Consent:

I have fully explained to the nature and purpose of the above-described procedure and the risks that are involved in its performance. I have answered and will answer all questions to the best of my ability.

(Signature: Principal Investigator)

I have been fully informed of the above-described procedure with its possible benefits and risks. I give permission for my/ my child's participation in this study. I know that Martha Barthel, R. N. or her associates will be available to answer any question I may have. If, at any time, I feel my questions have not been adequately answered, I may request to speak with a member of the Medical Center Institutional Review Board. I understand that I am free to withdraw this consent and discontinue participation in this project at any time without prejudice to my/my child's medical care. I have received a copy of this informed consent document.

I understand that biomedical or behavioral research such as that in which you have agreed to participate, by its nature, involves risk of injury. In the event of physical injury resulting from these research procedures, emergency medical treatment will be provided at no cost, in accordance with the policy of Loyola University Medical Center. No additional free medical treatment or compensation will be provided except as required by Illinois law.
In the event you believe that you have suffered any physical injury as a result of participation in the research program, please contact Dr. R. Henkin, Chairman, Institutional Review Board for Protection of Human Subjects at the Medical Center, telephone (312) 531-3777.

I agree to allow my name and medical records to be available to other physicians and researchers for the purpose of evaluating the results of this study. I consent to the publication of any data which may result from these investigations for the purpose of advancing medical knowledge, providing my name or my child's name or any other identifying information (initials, social security number, etc.), is not used in conjunction with such publication. All precautions to maintain the confidentiality of medical records will be taken.

(Signature: Patient/Parent/Legal Representative)

Signature: Witness to Signature
APPENDIX C
Technical Specifications of the Ladd Monitor

Measurement Range: -37 to +185 mmHg (absolute pressure)

Rate of Response: 2.5 mmHg per second

System Sensitivity: less than 0.074 mmHg regardless of pressure level.

System Accuracy with Digital Readout: 1% of reading

Zero Point Stability: ± 0.37 mmHg less than 0.74 mmHg shift due to patient temperature rise from 37 - 41°C unaffected by changes in ambient pressure.

Repeatability: ± 0.37 mmHg

APPENDIX D
DATA COLLECTION FORM

<table>
<thead>
<tr>
<th>5 Minutes</th>
<th>5 Minutes</th>
<th>Nursing Activity</th>
<th>5 Minutes</th>
<th>5 Minutes</th>
</tr>
</thead>
</table>

Code Number: ______________________________________

Procedure: ______________________________________

Infant Activity State ________________________________

Intracranial Pressure Readings:

<table>
<thead>
<tr>
<th>5 Minutes Prior to Procedure</th>
<th>5 Minutes Post Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
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</tbody>
</table>

APPENDIX E
**Patient Profile**

<table>
<thead>
<tr>
<th>Code #</th>
<th></th>
<th>Date</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td></td>
<td>Race</td>
<td></td>
</tr>
<tr>
<td>Date of Birth:</td>
<td></td>
<td>Gestational Age:</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Weight (grams)</th>
<th>Length (cm)</th>
<th>Head Circumference (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apgars (1 min.)</td>
<td></td>
<td>Apgars (5 min.)</td>
</tr>
</tbody>
</table>

**Primary Diagnosis**

**Current Diagnosis**

**Age at time of data collection**: ________ weeks.

<table>
<thead>
<tr>
<th>Current Weight (grams)</th>
<th>Current Length (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current Head Circumference (cm)</td>
<td></td>
</tr>
</tbody>
</table>

- size of anterior fontanel (cm)
  - description of fontanel:
    - sunken: soft/firm
    - flat: soft/firm
    - full: soft/firm
tense/bulging

**Vital signs**: T _____ P _____ R _____ BP _____

**Respiratory Status**:

- Spontaneous Ventilation: _______ Room air
  - Head Hood: _____ % O₂
  - apnea mattrace: yes/no

**Ventilator**: yes/no parameters

**C-PAP**: yes/no parameters

**Arterial Blood Gas**:

<table>
<thead>
<tr>
<th>pH</th>
<th>pCO₂</th>
<th>pO₂</th>
<th>Bicarb</th>
<th>Base excess</th>
</tr>
</thead>
</table>

**Nutritional state**: NPO

<table>
<thead>
<tr>
<th>Hyperalmentation</th>
<th>IV</th>
<th>PO</th>
<th>OG/NG</th>
<th>Type</th>
<th>Amount</th>
<th>Frequency</th>
</tr>
</thead>
</table>

The thesis submitted by Martha J. Barthel, R. N., has been read and approved by the following committee:

Dorothy Lanuza, R. N., Ph.D., Director
Associate Professor, Medical Surgical Nursing

Elizabeth Brophy, R. N., Ph.D.
Associate Professor, Psychiatric Public Health Nursing

Marcia Maurer, R. N., M. S.
Assistant Professor, Maternal Child Health Nursing

The final copies have been examined by the director of the thesis and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the thesis is now given final approval by the Committee with reference to content and form.

The thesis is therefore accepted in partial fulfillment of the requirements for the degree of Master of Science in Nursing.

Date: July 17, 1984
Director's Signature