Manual Head and Neck Alignment During Patient Turning in states of Decreased Intracranial Adaptive Capacity

by

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ABSTRACT

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Immobility following brain injury creates a practice problem for nurses in the intensive care unit (ICU). Turning reduces the occurrence of complications that accompany immobility but problems arise when undesirable physiologic responses accompany turning. Intracranial pressure (ICP), cerebral perfusion pressure (CPP) and intracranial pressure waveforms (ICPW) are the physiologic responses and dependent variables in this experimental study. Adequate intracranial adaptive capacity is present when responses after turning are normal. Active or experimental alignment of the head and neck promises to promote rather than challenge adaptive capacity as evidenced by responses in ICP, CPP and ICPW. Five subjects with cerebral hemorrhages and edema are the sample and the setting is the neurosurgical ICU in a 500-bed private hospital. Each subject undergoes four observation sessions spaced at two hour intervals. Randomization of the type of turn takes place before data collection. Comparison of baseline values for ICP, CPP and ICPWs before each turn to those after the turn takes place. Statistical differences between experimental and standard methods within subjects and the aggregate are absent. Sample size limits the generalizability of findings but single case analysis shows responses are unique. Two predictions for practice are present. Patients with ICP levels above 15 mm Hg respond with less adaptive capacity to turning and ICP levels above 15 mm Hg have more elevations in the P2 component of the ICPW.

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CHAPTER I

INTRODUCTION

Nurses caring for patients with brain injury face a practice problem that occurs when undesirable physiologic responses follow ordinary nursing interventions such as turning. Some patients tolerate the turning procedure while other individuals respond with sustained increases in intracranial pressure (ICP) and decreases in cerebral perfusion pressure (CPP). When turning of the patient is omitted or delayed, complications such as pneumonia, pulmonary edema and pressure necrosis may occur. The quandary then is how to approach the problem of immobility in patients with intracranial pathology. Therefore, identification of a method to turn patients who demonstrate poor tolerance for this activity is important to the recovery and health of the individual.

Turning is a change in body position from supine to sidelying or from sidelying to supine. Two nurses accomplish this task in the intensive care unit (ICU). During this maneuver the alignment of the head and neck appears unimportant. The neck flexes laterally, extends backward, or remains stationary. Furthermore, head rotation happens when endotracheal tubes connected to respirators pull the head in an opposite direction from the body.

The purpose of this study is to observe and describe physiological responses that follow head and neck alignment.

Two types of head and neck alignment include: (a) active securing the head and neck into a neutral position, and (b) passive touching of the head and neck without active alignment. Passive alignment of the head and neck most closely reflects the turn used in routine nursing practice. Physiologic responses that occur after the two types of alignment are of interest. The physiological responses include measurements of intracranial pressure (ICP), cerebral perfusion pressure (CPP), and intracranial pressure waveforms (ICPW).

Statement of Problem

What effect does turning have upon critically ill patients with intracranial pathology? This question is important to nurses practicing in the ICU because patients here are often immobilized by injuries resulting from trauma or surgical interventions. Therefore, it becomes the nurses' responsibility to design a plan that will mobilize the patient without endangering the brain. Sometimes a plan for turning is absent for the craniotomy or head injured patient (Fontaine & McQuillan, 1989). For example, shift reports include statements such as, "I did not turn my patient all shift because we tried once and then the ICP increased a lot and the blood pressure dropped." In practice, nurses view brief elevations of ICP as a serious problem. Nurses are reluctant to turn patients and risk sustained elevation of ICP (Parsons & Wilson, 1984) and changes in CPP. Critical care nurses are aware that turning does influence ICP and

CPP; however, exactly how much or what direction individual responses may take is a mystery.

Review of the Literature

Several research studies structured around the problem of turning and the concomitant effects on ICP have been useful. Physicians, were the first to report that nursing care activities caused rises in ICP (Lundberg, 1960; Shalit & Umansky, 1977). Several positioning studies followed. The classic study on this topic was the descriptive work of Mitchell and Mauss (1978) which was the first to discuss the relationship between patient-nursing care activities and ICP. Subjects included nine adults with ventricular catheters in place for the purpose of pressure-controlled venticulostomy drainage. The investigators predicted frequencies of ventricular fluid drainage during activity and non activity and compared predicted to actual occurrences. Although one subject demonstrated no increase in ventricular fluid drainage, eight others showed more than predicted drainage with activity over a 24 hour period. Consistent increases in ventricular fluid drainage (an indirect measurement of ICP) occurred with chewing, coughing, use of the bed pan, and restless movements. In addition, individuals demonstrated differing responses to turning and conversations regarding their clinical condition (See Table 1).

In a two part study, Lipe and Mitchell (1980) concentrated upon the internal jugular vein and its relationship to neck rotation and body position change. In the first experiment, 10 healthy adults were studied and internal jugular flow was monitored by ultrasound and a Duplex Echo-Doppler scanner. Together, these instruments detected velocity of blood flow. The researchers found compromised flow in six out of the 10 healthy subjects in three body positions: supine, left lateral and right lateral. The left lateral position demonstrated more events of compromised flow compared to other positions. Compromised flow consisted of an increase in internal jugular vein flow velocity combined with reduced lumen size during expiration. The second experiment took place because head rotation involuntarily occurred in subjects turned in experiment one. Five adult subjects were monitored with the Duplex Echo-Scanner to study head rotation and jugular vein compression. The conclusion in experiment two was that head position specifically with 90 degree rotation to the right, indeed affected jugular vein dynamics. Together the experiments demonstrated undesirable events while the subjects were on the left lateral side and with head rotation to the right.

Various body positions and their effect on ICP were studied by Mitchell, Ozuna, and Lipe (1981). Ventricular fluid drainage was measured following eight position changes: arm extension, hip flexion, turning from supine to right, right to supine, supine to left and left to supine, head rotation to the right and to the left. With eighteen subjects they found that drainage increased in all patients in at least one turn. A large percentage (88%) of patients displayed elevations in pressure on half of the turns. Of interest was the fact that in some cases turning from supine to the left lateral position created a unique decrease in drainage of ventricular fluid at one minute following the turn. In addition, the investigators also reported a steady increase in ventricular fluid pressure when nursing activities were spaced only 15 minutes apart. This increase did not occur when the same activities were spaced one hour apart.

Table 1

Summary of Studies on Positioning

Author	Type and Sample Size	Problem	Measurement Tool	Results
Magnaes (1976)	Quasi experimental N=120	Pressure dynamics in patients as they moved between lateral and sitting positions	Mini-transducer potentiometer	Rapid change in body position initiated CSF pressure waves. Waves were transient or sustained. Sustained waves were found only in diseased states or increased ICP
Shalit & Umansky (1977)	Quasi-experimental N=21	Effect of rotation, flexion of head to side upon ICP	Pressure transducer IVC, bolt, feeding tubes	Rotation of head from neutral to side caused rapid increase in ICP. Increases were more pronounced in subjects previously elevated. If the whole body followed the head ICP was restored.
Mitchell & Mauss (1978)	Descriptive N=9	Relationship between patient care activities and ICP variations	Pressure controlled ventriculostomy	Each subject displayed individual patterns. All consistently display increased ICP with coughing, chewing use of bed-pan, and restlessness
Lipe & Mitchell (1980)	Quasi-experimental N=10 (#1) N=5 (#2)	Internal Jugular velocity and lumen size before and after position changes	Duplex echo-doppler scanner	Experiment #1 Compromised flow occurred most often in left lateral position. Experiment #2 Compromised flow occurred most often in head rotation to the right.
Mitchell, Ozuna & Lipe (1981)	Quasi-experimental N=20	Relationship of nursing care activities to ICP	Pressure controlled Ventriculostomy	No significant increases following ROM exercises. Turning did increase ICP in all subjects at least once and 88% twice. Increases in ICP in one direction matched other directions. Spaced activities of at least 60 minutes resulted in no cumulative increase in ICP.

Table 1

Summary of Studies on Positioning (Continued)

Author	Type and Sample	Problem	Measurement Tool	Results
Ropper, O'Rourke & Kennedy (1982)	Quasi-Experimental N=19	Effect of head position on ICP and compliance	Either bolt or IVC or both	Compliance estimate was performed by recording four baseline ICP readings followed by injection of 0.1 cc of saline. Pressures were recorded at 10-second intervals. Compliance was calculated by determining the areas under the ICP curve. Ten cases had lower ICPs with HOB up 60 degrees, and two decreased when flat and seven were unchanged. Compliance improved with elevation in five, improved with lowering in four and was unchanged in 10. Optimal positioning was set on individual basis.
Parsons & Wilson (1984)	Descriptive N=18	Effects of body positions turning, head rotation, ROM head elevation upon ICP and cerebrovascular status	Subarachnoid bolt	All position changes produced transient increases in ICP except raising the HOB CPP did not fall be-low 50 mm Hg.
Boortz-Marx (1985)	Descriptive N=4	Events in ICU that affect ICP	Subarachnoid bolt/IVC	Elevations in ICP followed turning, physical assessment, suctioning and bathing. Environmental stimuli did not affect ICP
Mitchell, Amos & Astley (1986)	Quasi-Experimental N=3 (African baboons)	Effects of turning and lateral neck flexion on ICP and Cardiovascular variables	ICP, Epidural fiberoptic sensor and Cannulae in internal jugular vein and auxiliary artery	ICP increased in 96% of turns and demonstrated clinically significant increases with lateral neck flexion, ICP increased to statistical significance in 74% of all turns.
March, Mitchell, Grady & Winn (1990)	Experimental N=4	Effects on ICP, CPP, SABP and CBF velocity with changes in back rest elevation	Bolts, A-lines, transcranial doppler	Changes in ICP, CPP and TCD flow velocities resulting from changes in backrest position are individual and not consistent across all head injury patients or within a single patient.

Parsons and Wilson (1984), in a study of eighteen severely head injured subjects, found that all position changes, except one, produced transient increases in mean ICP and CPP. The one position where ICP decreased had the head of bed elevated. The device for ICP measurement in this study was a subarachnoid bolt (a direct method). Blood pressures were monitored through an arterial catheter and then mean ICP was subtracted to yield a value for CPP.

All subjects in the Parson and Wilson (1984) study had baseline mean ICPs less than 15 mm Hg and CPPs greater than 50 mm Hg. The authors noted the pressure changes were transient with trends that returned to baseline after one minute. However, the range in age of subjects was from five years to 67 years. Another study involving children was conducted by Hobdell, Adamo, Carusa, Dihoff, Neveling, and Roncoli (1990). Thirteen subjects aged 1.5 to 11 years were studied in a pediatric ICU and the authors reported no significant difference in the mean ICP after turning. Nine of the children had subarachnoid bolts in place and four a ventriculostomy type device.

In the descriptive work of Boortz-Marx (1985), elevations of ICP were present following the turns of four subjects with severe head injuries. The amounts of increase or decrease in ICP were listed during the care of subjects in the ICU. Suctioning was followed by a mean increase in ICP of 10.65 mm Hg above baseline. In some events ICP was lowered by repositioning activity. Flexion and rotation of the head and neck were followed by a mean rise of 2.75 mm Hg. Two bolts and two ventriculostomies were the measuring tools in this study.

Magnaes (1976) explored pressure dynamics in patients as they moved between lateral and sitting positions. Rapid changes in body positions initiated elevations or "waves" in pressure transducers reflective of cerebrospinal fluid (CSF) pressure in the lumbar and ventricular region. Magnaes observed both transient and stationary waves. The stationary or sustained elevations happened only in disease or states of increased ICP. The author believed the changes following repositioning were due to alterations in cerebral blood volume reflecting the systemic blood pressure.

Another recent study done on the topic of head and neck positioning was that of Mitchell, Amos and Astley (1986) with African baboons as subjects. They found clinically important increases in ICP following lateral neck flexion. The increases were not present when the heads were in the midline position.

To summarize, several experts agreed with practicing nurses that turning challenges the intracranial adjustments reflected indirectly by sustained increases in ICP (Mitchell & Mauss, 1978; Mitchell, Ozuna & Lipe, 1981; Magneas, 1976; Parsons & Wilson, 1984; and Boortz-Marx, 1985). Other investigators suggested that most patients with increased ICP benefited from head elevation (Parsons & Wilson, 1984). A neutral neck position was advocated by some, (Shalit &

Umansky, 1977) and avoidance of head and neck flexion and rotation was recommended by others. (Mitchell and Lipe, 1980).

Five limitations were present in the positioning studies. The first was that of the measurement devices. For example, pressure controlled ventriculostomy drainage systems were used in nine subjects to measure ICP by Mitchell and Mauss (1978) and Mitchell, Ozuna, and Lipe (1981). When the drainage system's output increased the authors believed ICP increased. While this may be a correct assumption, the exact magnitude and duration of the increase were not available. More importantly, the fluid filled device may have attenuated the response by reducing ICP. A second category of limitation was the use of small samples. Boortz-Marx (1985) reported on a small sample size of four as did March et al. (1990). Mitchell et al. (1986) experimented with three African baboons. A third limitation was noted in the Boortz-Marx (1985) and Shalit and Umansky (1977) studies because definitions of turning, head elevation or rotation were not present. A fourth limitation combined pediatric and adult subjects for analysis of ICP and CPP (Hobdell et al., 1990; Parsons and Wilson, 1984). Fenichel (1988) stated that unlike adults, children younger than 10 with ICP problems experience temporary relief due to anatomical separation of cranial sutures. Therefore ICP values may not be equivalent when the adult brain was compared to the more adaptive brain of a child. A final limitation noted was lack of internal and external controls. For example, various pathological problems were present the each study group (Boortz-Marx, 1985; March, Mitchell, Grady, & Winn, 1990; Mitchell & Mauss, 1978). Furthermore, environmental controls for each study lacked documentation.

A knowledge gap in the turning dilemma has remained. For instance, what clinical features did patients have in common who demonstrated large and sustained elevations of ICP following turning? Which patients were more likely to rotate the head involuntarily following the stimuli of turning? What evidence was there that immobility in the first 72 hours following injury caused certain pulmonary or skin complications?

Research Question

According to investigators (Magnaes, 1976; Mitchell, 1986b) the stimulus of head and neck rotation may have caused transient mechanical obstruction to the jugular, vertebral and even intrathoracic venous systems. Mitchell speculated that head position could cause transient increases in intracranial CSF due to obstruction of basal cisterns. In practice and in the literature reviewed some individuals adjusted to the stimuli of head and neck rotation and flexion. Adaptive responses such as only a slight increase in ICP and a rapid return to baseline measurement occurred. Other individuals responded ineffectively to this stimulus in a physiologic sense. Would responses be any different when active alignment was given during the turn? Therefore, the primary research question that drives this investigation is:

> What effect does holding the head in a mid-line position have upon ICP, CPP, and ICPW following nurse-initiated turning, in patients with decreased intracranial adaptive capacity?

Research Hypotheses

The following three hypotheses are:

1) When the head of the critically ill patient is maintained in midline throughout a turning process a sustained increase in ICP is less likely to occur.

2) When the head of the critically ill patient is maintained in midline throughout a turning process CPP is more likely to remain above 50 mm Hg.

3) When the head of the critically ill patient is maintained in midline throughout a turning process ICPW is more likely to remain normal.

Conceptual Framework

Adaptive Capacity

To explain the physiologic responses of patients to the identical stimuli of turning, the concept of adaptive capacity is useful. One of the definitions used by Webster in describing capacity is "the ability to receive." Another Webster definition is the "maximum production or output" of a system. Is it possible that a system's adaptive capacity can be evident both in the ability to receive stimuli and to demonstrate adaptive responses? In differing states of health and illness, human systems have varying capacities for adaptation. This study explores intracranial adaptive capacity during illness.

Mitchell (1986b) describes a system with adequate adaptive capacity as one having transient or only minimal responses to potent stimuli. Therefore, a system has adequate adaptive capacity to receive input when responses demonstrated by the system are normal. In contrast, Mitchell asserts that a system with inadequate adaptive capacity will demonstrate exaggerated responses following even minimal stimuli. Direct measurement of intracranial adaptive capacity is not possible, however certain responses arising from the patient may provide information regarding adaptive capacity.

Table 2

Theoretical and Empirical Frameworks

INTRACRANIAL ADAPTIVE CAPACITY

Conceptual		Empirical	Measured Indicators
Stimuli		Turning	Turning Protocols Passive Alignment Active Alignment
	Adaptive	Ineffective	
Response	ICP levels that return to baseline within 5 minutes of turn	ICP increases greater than 10 mm Hg above baseline for more than 5 minutes	ICP values displayed on both digital and waveform analogs from intracranial monitoring devices in the brain and ventricle
	CPP levels above 50 mm Hg	CPP less than 50 mm Hg	Bedside monitor computed MAP by calculating the area under the curve divided by a time interval (Nara, et al., 1989) and subtracted mean ICP to get CPP.
	Normal P2 amplitudes	Abnormal P2 amplitudes	Percentage of P2 amplitude compared to that of P1. (See Protocol for P2 amplitude in Appendix D)

Intracranial Adaptive Capacity

Intracranial adaptive capacity is the ability of the nervous or vascular system to adjust to changing stimuli that arise either from within or external to the cranial cavity. Circumstances such as trauma and surgery alter volume-pressure relationships or the intracranial adaptive capacity of the system. Intracranial adaptive capacity is present as a result of normal physiologic events that include increased absorption of the cerebral spinal fluid and autoregulation of cerebral blood flow. Nursing activities such as turning may temporally interrupt the system's ability to adapt.

Cerebrospinal Fluid

Bruce (1978) believes that CSF is the major intracranial content responsible for adaptive volumetric changes within the cranium. Normally following an intracranial stimulus such as increased volume, displacement and shunting of CSF from the intracranial compartment to the spinal dural sac occurs. From observations following myelography Martins et al. (1972) demonstrate that the spinal dural sac acts as a reservoir for CSF. Disruptions of free CSF circulation or CSF absorption alter intracranial adaptive capacity.

Cerebral Blood Flow

The vascular system within the brain may indirectly reflect adaptive capacity. Blood vessels on the surface and base of the brain are innervated by autonomic nerve fibers. The fibers respond to systemic arterial pressure and chemical changes. According to experts, (Bornstein & Brown, 1991, and Lundgren, 1990), cerebral blood flow is autoregulated or remains constant at approximately mean arterial pressures of 50 mm Hg to 150 mm Hg. Thus, for individuals in a healthy state who have mean arterial pressures as low as 50 mm Hg or as high as 150 mm Hg the cerebral blood flow remains consistent. In disease states systemic arterial pressures often fall outside the normal autoregulatory range. A reduction or increase in cerebral blood flow occurs. The result is a change in volume of blood supplied or delivered. Without autoregulatory mechanisms, cerebral blood flow and volume become passively dependent upon changes in systemic blood pressure.

<u>Brain Tissue</u>

Brain tissue by itself has some adaptive attributes. Overall the brain has viscoelastic properties such as degrees of displacement and compression. In healthy individuals frequent but brief elevations in ICP may reflect brain displacement also known as herniation. Brain tissue displacement that persists causes tissue damage. On a cellular basis, trauma and disease change the volume balance of the central nervous system. Edema is common in many patients with brain injury. Together and separately, cerebrospinal fluid, cerebral blood flow and brain tissue are important intracranial elements that influence intracranial adaptive capacity.

Adaptive and Ineffective Responses

Intracranial pressure and cerebral perfusion pressure are indirect measurements of intracranial adaptive capacity. More recently a third clinical indicator, intracranial pressure waveforms, promises to give additional information regarding the volume pressure relationships of the brain.

Intracranial Pressure

The concept of intracranial pressure (ICP) is based on the relationship among three volumes enclosed within the cranium: blood, brain tissue and cerebrospinal fluid (CSF). The pressure exerted by the cerebrospinal fluid upon the brain is known as intracranial pressure. ICP values ranging from 0-15 mm Hg are normal. Lundberg (1960) lists ICP levels of 11-20 mm Hg as slightly elevated, 21-40 mm Hg as moderately elevated and over 40 mm Hg as severely elevated.

Brief fluctuations in ICP normally occur in response to the respiratory cycle, arterial pulsations, changes in intrathoracic pressure (coughing, sneezing and Valsalva maneuvers) and position changes. These transient elevations may reach the 50 mm Hg range and return to base line readings within seconds (Bruce, 1978, Magnaes, 1976). An example of an adaptive response would be a brief elevation in ICP lasting less than 5 minutes and values that do not exceed 10 mm Hg above baseline (Mitchell, 1986b). On the other hand, sustained ICP levels of 15 mm Hg or more reflect a state of intracranial hypertension where normal adaptive capacity may be decreased or absent.

Cerebral Perfusion Pressure

Another indirect indicator of adaptive capacity is cerebral perfusion pressure (CPP). Normal values of CPP are 60-100 mm Hg. The CPP is calculated if mean arterial pressure (MAP) and ICP are known. The arithmetic differences between mean ICP and mean arterial blood pressure is equal to CPP (CPP = MAP - ICP). McGillicuddy (1985) suggests that autoregulation maintains a stable cerebral blood flow in ranges of CPP that begin with a lower limit of 50 mm Hg.

Intracranial Pressure Waveform

In addition to pressure measurements, analog waveform patterns indirectly reflect adaptive capacity. Cardoso, Rowan and Galbraith (1983) believe that the ICP waveform has three pulsatile components. The first peak, or P1, is the percussion wave. This wave originates from pulsations of the choroid plexus in the lateral ventricles where arterial input is measured from surrounding vessels. The second peak, or P2, is the tidal wave. The tidal wave is variable in shape and amplitude, ending on the dicrotic notch. Immediately following the dicrotic notch is the third wave, or the dicrotic wave is P3.

Normally the P1 component has the greatest value followed closely by P2 and then P3 (See Figure 1). However with injury to the brain, the P2 component of the patient's waveform can elevate to levels above the P1 component.

Figure 1. Normal and abnormal configurations of the intracranial pressure waveform.

Cardoso, Rowan and Galbraith (1983) claimed that the P2 component of the waveform closely reflected cerebral compliance or intracranial volume - pressure balances. They described fifteen awake patients who demonstrated decreased amplitude of the P2 with hyperventilation (a common treatment for reduction in acid content of the blood). The authors believe this reduction in amplitude reflects an improvement in volume-pressure relationships within the cranium. In another study by Portnoy, Chopp, Branch and Shannon (1982) rounding or elevation of the P2 in the ICPWs of 13 dogs occured along with increases in ICP values. Both studies suggested that the ICPW has a connection to the same factors that increase ICP. Takizawa, Gabra-Sanders and Miller (1987) explored the relationship of ICPW to CPP. They conducted a study with cats that demonstrated a negative correlation with ICPW and CPP. When CPP dropped the waveform showed an increase of P2 amplitude.

While ICP, CPP and ICPWs are accessible clinically, they are only indirect reflectors of intracranial adaptive capacity. Other measurements known as compliance and elastance are more direct indicators.

Compliance

Compliance is an expression of the relationship between the change in ICP as a result of the change in intracranial volume. According to Bruce (1978) compliance describes intracranial situations where pressure changes are induced. The following equation defines compliance:

Compliance = change in volume/change in pressure.

Normally, the volume of intracranial contents totals between 1400 and 1900 ccs and the ICP is 10 mm Hg. Following injury, a subdural hematoma may hypothetically add 30 cc to the volume bringing the total to 1930 cc. If the resultant ICP increases only to 12 mm Hg a state of adequate compliance is present. The subdural hematoma continues to enlarge adding five more cubic centimeters of volume and bringing the total 1935 ccs. A state of decreased compliance or decreased ICAC is present as ICP has sustained an increase from 12 mm Hg to 30 mm Hg (See Figure 2; compare pressures at Point B and Point C).

<u>Elastance</u>

According to Jennett and Teasdale (1981), elastance is the change in ICP resulting from any unit change in CSF volume. Elastance is the reciprocal of compliance:

Elastance = change in pressure/change in volume.

Therefore, clinical situations of decreased compliance are also states of high elastance. Decreased compliance and high elastance are present in states of decreased intracranial adaptive capacity.

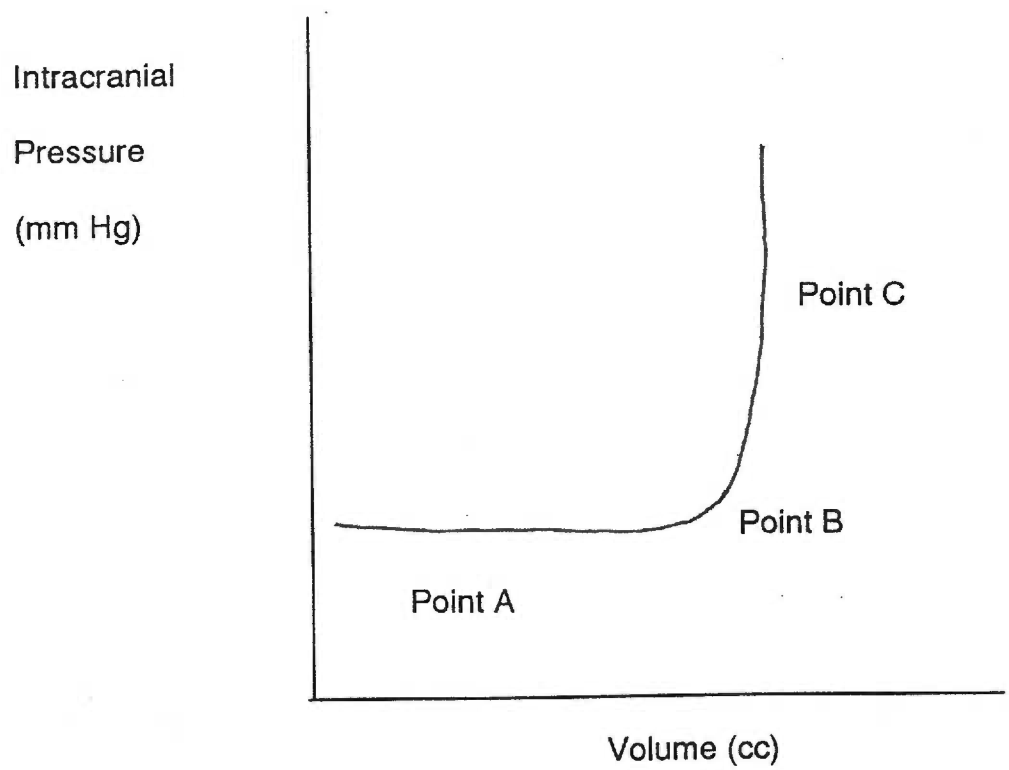


Figure 2. Volume - Pressure curve. The initial, gradually sloping section indicates adaptive response. The steep portion of the curve signifies ineffective response.

Overview of the Study

This study investigates the physiologic responses of brain injured or diseased patient and compares the standard method of turning to an experimental method where the patients' head and neck alignment enhances venous drainage, potentially preventing increases in ICP. Responses, measured as dependent variables, are studied before and after therapeutic and control turns. As undesirable physiologic responses follow turning the nurse usually chooses to avoid any movement. As a result the risk for increases in skin and pulmonary problems is present.

CHAPTER 2

METHODS

Design

This study uses a single subject experimental design as well as group analysis for five subjects. Alignment of the subject's head during the therapeutic turn is the manipulative component of the experiment. The control turn involves passive touching of the head of the subject, so that the arousal response to tactile stimuli about the head and face is constant in both conditions. The investigator performs both the passive and active alignments for all subjects. Each subject serves as his or her own control. Initially the direction and type of the turn are randomized. Three turning positions are possible. They are right-side lying, supine and left-side lying. Initial observation of subjects includes a baseline measurement of the dependent variables. Three separate data points establish the subject's baseline before the treatment as suggested by Barlow and Hersen (1973). The experimental or control method of turning takes place after three baseline measurements and before four final data points. Observations take place for 30 minutes in two hour intervals starting four hours after the subjects' normal waking hour. When the patient is found to be in the lateral position before the first observation period Table 3 determines the order of the turning sequence. On the other hand when the patient is found to be in the

Table 3

Random Sequences of Head Alignment in first Turns to Supine.

Patient Position	Supine	Lateral	Supine	Lateral
Type of Turn				
Sequence #1	T	C	C	T
Sequence #2	C	T	T	C
Sequence #3	T	T	C	C
Sequence #4	C	C	T	T

Note. T = Therapeutic Turn, C = Control Turn

Table 4

Random Sequences of Head Alignment in First Turns in the Lateral Direction

Patient Position	Lateral	Supine	Lateral	Supine
Type of Turn				
Sequence #1	T	C	C	T
Sequence #2	C	T	T	C
Sequence #3	T	T	C	C
Sequence #4	C	C	T	T

Note. T= Therapeutic Turn, C= Control Turn supine position before the first observation period application of Table 4 takes place.

Pennies with the numbers one, two, three and four taped to them are selected from a container to determine the sequence of therapeutic or control protocols. For instance, when using Table 4, the first turn is in the lateral direction. Turn sequence #2 means a penny with the number two came up and alignment of the head is first in the control manner, the second and third, therapeutic and the fourth, control.

Another characteristic of this study is the focus upon single cases. The major benefit of single case research is that each case is analyzed for individual responses to turning. According to Holm (1983), individual variations can disappear in the analysis as numbers of subjects increases. Even though this study has only five cases the responses following the stiumli of turning are unique.

Identification of Variables

Independent Variable

The independent variable in this study is the active alignment of the head in the midline position during turning (See Appendix A).

Dependent Variables

Three indirect indicators of ICAC are the dependent variables; ICP levels, ICP waveforms and CPP levels with three measurements before the intervention, and four following the intervention taking place.

Sample

Criteria for Subjects

Patients admitted to this study are critically ill due to trauma or other emergencies. To be eligible for this study the subject has intracranial pressure monitoring established as well as arterial pressure monitoring. Male or female subjects over the age of eighteen and under 85 are in included the sample. Patients with traction apparatus and spinal cord injuries that prevent turning are not included the sample.

Measurement Tools

Intracranial Pressure Monitoring Devices

The investigator hoped to use measurements from a ventricular fiberoptic device (FOD) designed by Camino Laboratories. After placement, the FOD catheter connects to a monitoring box (also known as the 420 model) which provides a digital display of the mean ICP. Continuous waveform recording is available by connection of the 420 model to a bedside pressure monitoring system.

Reliability for ICP Measurement

The Camino FOD, shown by Crutchfield et al. (1990) is more stable than the fluid-filled monitoring systems in the ventricles and subarachnoid space. In 22 cases the fiberoptic device compares favorably to fluid-filled ventriculostomies and subarachnoid bolts. The FOD is more stable than the ventriculostomy or subarachnoid bolt, with a maximum drift of 2 mm Hg per 8 hour shift (mean 0.75). The maximum recorded drift for the ventriculostomy is 11 mm Hg per shift (mean 8 mm Hg) and the corresponding figure for the bolt is 17 mm Hg (mean 11 mm Hg). To avoid "drift" in pressure lines calibration takes place before observations and then four hours later.

Narayan (1990) reports that ICP measured by ventriculostomy and by direct placement into brain tissue (intraparenchymal) are similar, (Mean difference 0.57 mm Hg) with the exception of transient increase of ICP, the peak is often higher when measured by the intraparenchymal FOD. In another study, Ostrup (1987) reports the ventricular monitoring device has a strong correlation (r=.98) to those placed in the brain tissue. Narayan (1990) found that the waveform obtained with FOD is similar in shape, but contains more detail and is less artifact-prone that the fluid-filled systems.

Space Labs (1990) claim in the operation manual the pressure module is accurate within nearly one percent of the range with zero drift or <0.1 mm Hg after a five minute warm-up and exclusive of pressure transducer drift.

A potentiometer screw on the hub of the cable facilitates zeroing to atmospheric pressure (Hollingsworth-Fridlund, Vos and Daily, 1988). Zeroing is complete before the catheter tip is in vivo. Even though the transducer pressure response is calibrated by the manufacturer, another check takes place. The next step to reach reliability in the instrument chain is calibration of the 420 model that must be matched to the patient monitoring system. Calibration happens as suggested in the operations manual (Space Labs, 1990; See Appendix B). According to Rubin (1987) the experimenter can limit the effects of error by performing a calibration.

Validity for ICP Measurement

Accuracy, suggests Rubin (1987), is the instrumentation science version of truth, which is the ability of the instrument to measure the true or correct level of the signal and in addition represent the underlying concept of the system under study (validity). Validity in this study is important as observed signals of the subjects represent the physiologic concepts of ICP, CPP and ICPWs. ICP is nearly equivalent to cerebrospinal fluid pressure. This had been supported by studies with subjects who have simultaneous monitors in the ventricle and brain tissue or subarachnoid space (Narayan, 1987, Ostrup et al., 1987, and Ropper et al., 1982). Ostrup et al. (1987) demonstrate only a 2-to-5 mm Hg difference between catheters in the CSF (ventricle) and concurrent readings from intraparenchymal catheters.

Arterial Pressure Monitoring Device

Achievement of direct blood pressure monitoring is via cannulation of the radial artery in four subjects. The femoral artery was cannulated in one subject. The cannula attaches to fluid-filled tubing and a disposable transducer. A cable from the transducer connects to the patient monitoring system via a pressure module (Model 90402) that serves as an interface between the transducer and the PC Bedside Monitor (Model 90303). The pressure module uses its own microprocessor to perform real-time processing for one or two pressure signals to derive systolic, diastolic, and mean pressure value.

The investigator secures the disposable transducers manufactured by American Edwards (Catalog Number 43-600F) directly to head dressings. Durward et al.(1983) suggest that the site of maximum clinical interest for transducer position is the level of the foramen of Monro. Standard placement is level with the left cardiac ventricle. According to Bedford (1985) mean arterial pressure of 70 mm Hg at the left ventricle in the seated position usually means there is only marginal mean arterial pressure 50 mm Hg in the brain.

Validity and Reliability for Arterial Pressure Monitoring

Calibrations for A-lines take place before the investigation and four hours later (See Appendix B). Although the American Association of Critical Care Nurses (1985) indicates this calibration procedure should take place at least once in eight hours, Rubin (1987) recommends four hour intervals for increased reliability and avoidance of drift. Rubin (1987) suggests that validity is the trait of the tool to measure the true or correct level for blood pressure. American Edwards (1986) claim an accuracy of ± 1.5 mm Hg or ± 2.5% of reading for arterial pressure transducers in this study.

Patient Monitoring System

Space Labs systems Model 90303 is the bedside monitor for digital recording of mean ICP levels, mean arterial pressure and CPP. Correspondence between digital readouts of the pressures to the "hard copy" analog is consistent with continuous recordings; however, in some situations the graphic analogs may be four to 10 seconds ahead as the investigator activates the computed recording and then completes hand-written documentation of all three variables.

Internal Validity

History. Anticipated threats to the internal validity include the threat of history, maturation and selection. The threat of history (Polit & Hungler, 1987) refers to the occurrence of external events surrounding the treatment. These events can affect the dependent variables. Examples of historical events are suctioning, pain, coughing, and "bucking the ventilator." Musculoskeletal resistance can alter the dependent variables well. Resistance and restlessness observed in the fifteen minute post-intervention phase is documented.

Maturation. Maturation refers to processes occurring within the subjects during the course of the study. Polit and Hungler (1987) comment that maturation is a result of time rather than a result of the treatment. Intracranial adaptive mechanisms have the capacity to deteriorate rapidly. Again events surrounding either clinical improvement or deterioration are present in observation records. These threats challenge the internal validity but are realistic in experiments conducted on human subjects in the field.

Selection. Selection is a term used to classify threats to internal validity. An example of a selection threat in this study is the grouping of fluid-filled and fiberoptic ICP monitoring devices. Another selection threat is present in the number of assistants needed to perform the therapeutic turn in four observation sessions. On occasion a shift change occurs during clinical observation. Training of assistants to perform the intervention in a consistent manner is important. However, the number of assistants threatens consistency of treatment performance.

External Validity

Because of the anticipated small size of the sample the extent to which the results extend to subjects whose characteristics may differ is limited. Medical diagnosis or the preferred treatment options of the institution limit application across settings.

<u>Contextual Variables</u>

Because this study is performed in the clinical setting rather than the laboratory, several variables have characteristics that potentially may confound the findings. They include the following: medical diagnoses, type of lesion, concurrent injuries, medications in use, endotracheal suctioning, head of bed elevation, neurological nursing assessments, conversations in the room and touching of the patient by health care workers or visitors.

<u>Medical Diagnosis</u>. Subjects' diagnoses vary in this study. Additional points of comparison between subjects are the type of lesion sustained following the primary insult. Gennarelli et al. (1982) found that persons with acute subdural hematomas have more ineffective responses compared to persons with diffuse injury, who have more adaptive responses. Brain shift or pathologic displacement is available by computerized tomagraphy (CAT), which is routinely performed in the clinical setting.

<u>Pharmacological Agents</u> Osmotic diuretics, neuromuscular blocking agents, barbiturates and vasopressors have an impact on this study as well. Osmotic diuretics change the volume dynamics of the intracranial cavity and subsequently change ICP. Neuromuscular blocking agents paralyze the muscles; therefore the response following stimulus of turning upon a flaccid system differs from that of the systems without neuromuscular blocking adjuncts. Barbiturates are effective in controlling ICP in some

situations (Marshall, Smith and Shapiro, 1979; Rockoff, Marshall et al., 1979; Rea and Rockswold, 1983). One major side effect of higher dose therapy is arterial hypotension, which may endanger cerebral blood flow and consequently reduce CPP levels. When arterial hypotension occurs as a side effect of barbiturates or secondary complications, vasopressors are effective. Vasopressors act upon the cerebral blood flow regulators affecting cerebral perfusion pressures. The peripheral vasoconstrictive action increases cardiac output and enhances oxygenation to the cerebral cortex.

Endotracheal Suctioning Endotracheal suctioning (Boortz-Marx, 1985; Snyder, 1983) is another variable that needs investigator control. Unit nurses caring for the subjects plan to suction the subjects ten minutes before the intervention rather than after if the clinical condition supports this action. Nurses avoid suctioning if feasible during the observation and until the 15 minute post observation stage is complete.

Head of Bed Elevation When possible, head of bed elevations are uniform from subject to subject. Shalit and Umansky (1977), and Parsons and Wilson (1984) note that certain levels of elevation of the head of the bed cause decreased ICP. Head of bed elevations remain at 30 degrees from flat. When a subject's blood pressure drops at 30 degree's elevation, a lower position of 15 or 20 degrees is an option. The head of bed elevation standard is 30 degrees.

March, Mitchell, Grady and Winn (1990) suggest changing the standard according to individual responses.

Neurological Assessments Avoidance of neurological assessments that involve pupil checks and motor-sensory testing takes place when possible during observation periods. Snyder (1983) found that the stimuli such as light into the eyes, nailbed pressure and reflex testing increase ICP levels in certain individuals.

Environmental Issues Environmental issues such as where the location of a subject's room in reference to the nursing station are significant. The ideal is to observe each subject in the same room and bed so those noise levels might be consistent. Much of the health care planning for the subjects happens at the nurse station while physicians and nurses discuss various patients. Constant use of the telephone and computers is an additional source of stimuli to the subject.

To assess other types of stimuli, additional facts regarding family structure and personal histories are useful. The investigator records all conversations of either health care workers or family members in the room regarding the patient. Johnson, Omery, and Nikas (1989) found the effect of conversation on ICP in comatose patients is individualized and is related to the level of consciousness.

Human Subjects Review

Consideration and acceptance of the study by the Human Subjects Committee at Oregon Health Sciences University and

Salem Hospital Administration is the first step. Then the investigator asks the primary physician for permission to admit the subject to the study. After physician approval, the procedure for acquisition of consent continues with the primary nurse asking a family member if he/she is willing to discuss the study with the investigator, after which the investigator requests consent. The investigator informs the family of the possible risks, benefits and promises confidentiality.

Risks to Subjects

A potential risk to participation in this study is that the intermittent presence of a researcher in the room might be uncomfortable to the subject or to family members who visit. Participation in this study does not increase the risk of injury. However, every effort to prevent any injury that could result during this study is taken.

Benefits

A potential benefit of participation in this study is that the researcher made the information about the subject's condition available to the nursing staff. It is very possible that this study provided information like ICP, CPP and ICPWs to critical care nurses and physicians.

Confidentiality

The subjects' name nor items that may be traced to the subject are absent. The subjects' records are identified by a code number rather than by name.

Data Collection

Data collection begins after consent is given. Several technical procedures are undertaken. Clocks in the patient room and on the recorder match the hour and minutes and if possible the seconds. The investigator performs calibration of ICP and arterial pressure lines for electronic accuracy. The standard of 30 degrees head of bed elevation remains. Selection of pennies from a container determines the random direction and sequence of the first turn. Baseline measurements of the dependent variables, ICP, CPP and ICPWs, take place at 5, 3 and 1 minutes before the turn. Four measurements of the variables follow the turn (see Table 5). Conversations by staff and visitors along with administered medication are present on the event record. A total of four turns occurs for each subject and then data collection is complete. The investigator may discontinue the study if for any reason a patient wished it terminated, family members found the study cumbersome or interruptive to their socialization needs, or the physiological status deteriorated so that inspection and observation interrupt resuscitation attempts. None of the events listed as reasons to terminate the studies were present in any observation session.

Data Analysis

Two steps occur in the data analysis for this study. Single case analysis is the first step. Values obtained are visually inspected for direction, duration and magnitude of change (See Appendix C). Computation of single case descriptive statistics occurs. Then the second step in data analysis is to study the aggregate. Data available before and after turning along with both therapeutic or control methods is analyzed.

Repeated measures ANOVA is used to rate the difference between the therapeutic versus control method before and after the turn. Simultaneously recording of the three dependent variables takes place seven times in each Case. The major advantage of repeated measures is that the same subjects serve as their own control while exposed to different levels of independent variables (Huck, Cormier, & Bounds, 1974).

The use of repeated measures ANOVA is appropriate when subjects serve as their own control. Variance within the group is of interest and defined in this study as sets or combinations of time (before or after) and treatment. A within subjects or repeated measures design has merit because cases are observed under all treatment conditions and the measure of error variability is smaller than with a between subjects design. In this study sets of data collected before and after the interventions are one group. The second group is comparison of therapeutic data to control data. A two-factor repeated measures ANOVA is useful to the investigator seeking answers to the relationship of time to treatment. Time and treatment are the two main effects (Moore and McCabe, 1989). The interaction of treatment and time is available when using a two-way repeated measures ANOVA. Interaction described by Snedecor (1956) serves to rule out the compounding of the main effects. In other words, interactions are those variations attributable to joint effects rather than two influences acting singly (Guilford, 1967). The interaction demonstrates for this study that the difference between the before and after measures is not the result of the type of the turn. The alpha or level of significance for F is .05 that means that there is a probability of five in 100 that the findings are the result of chance errors.

Table 5

Observational Flow Chart with Data Points

Observation / preturn					Observation / postturn		
5 min	3 min	1 min	TURN	1 min	5 min	10 min	15 min
*	*	*		*	*	*	*

Note. Asterisks indicate data points referred to throughout this paper.

CHAPTER 3

ANALYSIS OF DATA

Sample

Description

Eight cases in the neurosurgical ICU (NICU) at Salem Hospital over a 10 month period in 1991 make up the sample. Six subjects have an intracerebral hemorrhage (ICH), one an epidural hematoma and another cerebral edema. The intracerebral hemorrhage category includes a ruptured cerebral aneurysm, hypertensive hemorrhage and bleeding into brain tissue from an arterial-venous malformation. Four subjects have traumatic injuries in addition to their brain injury: one a leg fracture, one a neck fracture, one facial fracture, and the other a jaw and clavicle fracture. The variables are present for Cases A, B and C in the Appendix D but are not a part of the statistical analysis due to technical inconsistencies like arterial line transducer placement and improper control turn methods. For example, observation of Subject A with a neck fracture is useful as a trial experimenting with the mechanics of strip chart recording and timing of data point collection. However, the intended design is useless because the presence of a cervical collar. Eight cases arrive over a period of 10 months but five subjects fit the criteria for statistical evaluation. The first three cases are technically dissimilar due to the

Table 6

Demographic Data and Variables for Study Subjects

Case	Sex	Age	Diagnosis	Confounding Variables	Random Table
A	M	16	ICH	C-collar, respirator, 5cm peep, Versed, CT=hemorrhagic contusions anterior, right temporal lobe and bilateral frontal lobes, Rt-to-Lf shift 2 millimeters	*********
B	M	61	ICH	Respirator, Versed, MS, Labetalol, CT=Lf. intracerebral hematoma, Craniotomy	Table 4 #3
C	M	19	ICP	Respirator, Pavuon, Rigid posturing, CT=Left punctate, peducular bleed, brain stem shearing	
1	M	59	ICH	Respirator, Versed drip, Nimodipine, Mannitol drip, CT=Rt Subarachnoid Bleed, Anterior Cerebral Artery, Craniotomy for Aneursym Clipping	Table 4 #3
2	F	65	ICH	"T"piece blow by, Nipride, Fractures of Tibia, Fibula, CT= Bleed into Rt. Temporal, Occipital Lobe, Rt Uncal Herniation, Craniotomy	Table 4 #4
3	F	35	Edema	Respirator, Versed, MS, Fractured skull, clavicle and mandible, CT=Rt temporal skull fracture, Rt to Lf shift of 6 millimeters	Table 4 #3
4	F	24	Epidural	"T "piece blow by, Fractured skull, Injuries result of a crime. CT=Epidural Left Temporal along with intracerebral bleed and depressed skull fracture	Table 4 #4
5	F	82	ICH	Mannitol drip, Lasix, CT=Rt uncal herniation, Bleed into Parietal, 12 millimeter shift Rt-to-Left, entrapment of left Ventrical	Table 4 #1

wrong reference point for the arterial line transducer and improper control alignment.

Confounding Variables within Sample

When the five formal cases are grouped several differences are evident. Two subjects are on respirators, two have endotracheal tubes with a T piece attached to an oxygen source and one subject did not need an artificial airway. Four cases had injury in the right cerebral hemisphere. Two CAT scans report uncal herniation (temporal lobe). In addition, some subjects received very aggressive administration of osmotic diurectics while others did not. Skull fractures were present in two cases that may have changed pressure outputs by providing temporary relief. Post operative events such as increased swelling within the brain may have altered some pressure values while cases treated medically escaped this physiologic challenge. Finally, the mean (+/- SD) age for subjects is 53 (+/- 26.72) years

Single Case Analysis

In the next section of this paper each case is presented individually. A brief history and a report of dependent variable trends with graphics for each variable follow.

Case 1

Case 1 is a 59-year-old man who arrives in the NICU after suffering a seizure while showering. A right subarachnoid hemorrhage is the diagnosis. Four days later after medical stabilization Case 1 has a craniotomy for clipping of an anterior cerebral artery aneurysm and evacuation of clot. Intracranial pressure monitoring is present post-operatively with a fluid filled system and a right ventricular catheter. Arterial pressure is present by cannulation of the right femural artery and attachment to pressure set ups and finally bedside display. Observation sessions begin close to 24 hours following surgery. Due to diagnostic procedures the first collection points occur nine hours after normal waking time (5:00 a.m.).

Intracranial Pressure

In Case 1 baseline mean (±SE) ICP is 15 (±0.33) mm Hg and those following the first therapeutic turn are 13 (±1.58) mm Hg. A reductive trend for ICP is present with a total drop of 8 mm Hg from the preturn baseline to the last data point. Two hours later after the second therapeutic turn a rise of 2 mm Hg occurs within one minute and a rise of 4 mm Hg after five minutes, however; this elevation is not sustained. Comparing baseline and ending values, the trend for ICP is downward. A control type turn also demonstrates lower trends following the intervention (3 mm Hg). The final observation session, a control type, shows a slight increase following the turn (2 mm Hg). To summarize, the control and therapeutic type turns have similar ICP values. For instance, in all turns at one minute following the turn there is a slight elevation of ICP with increases ranging from 1 mm Hg to 4 mm Hg compared to those before the turn. Sustained elevations do not continue and the end point trend is toward lower values than original.

Cerebral Perfusion Pressure

The first turn with therapeutic alignment shows CPP dropping from a mean (±SE) of 64 (±0.00) mm Hg in the preturn to 58 (±2.48) mm Hg in the postturn. The last collection point is 12 mm Hg lower than the first measurement before the turn approximately 20 minutes earlier. Two hours later in another therapeutic turn mean CPPs fall 10 mm Hg from the first collection point and continue dropping 15 minutes after the turn. During the same session Case 1 receives an intravenous push of Morphine Sulfate. In addition, the subject has concurrent medications including intravenous drips of Mannitol and Versed along with a recent 60 mg dose of sublingual Nimodipine. Control sessions three and four demonstrate a downward trend in CPP before the turn rather than after. Head of bed elevation in sessions three and four is lower than sessions one and two by about 10 degrees.

Intracranial Pressure Waveforms

In the preturn baseline P2 amplitudes average (±SE) 134 (±0.03)% greater than P1 and following the turn 140 (±0.04)% of P1. In session two the ICPWs continue with P2/P1 ratio abnormalities. For example, P2 amplitude averages are 137 (±0.03)% of P1s in the preturn and 119 (±0.14)% in the postturn. Again in session three the amplitude of P2 remains higher than P1 but approach normal once with an 89% after the turn. In the fourth session the P2 component of the ICPW continues to be elevated.

Summary

Case One demonstrates slight increases in ICP immediately following all turns. However, ICP values return to less than or equal to baseline levels in all sessions. During the second observation (therapeutic) a 10 mm Hg drop in CPP concerned the nursing staff enough so that the head of the bed elevation is reduced from 30 degrees to approximately 15 degrees. The nurses are under medical orders to medicate this subject with Morphine Sulfate and Versed to achieve sedation. Administration of both drugs takes place within minutes of the CPP drop and Nimodipine 60 mg within one-half hour. All P2 amplitudes elevate in every data point for this subject. To summarize, Case 1 demonstrates ineffective responses in two categories with baseline ICP values over 15 mm Hg, and abnormal ICPWs. The standard error (±SE) calculations demonstrate variability in ICP and CPP after a large majority of the turns as Table 7 shows. In some situations the standard error more than doubles. On the other hand, ICPWs show little variability after the turns

Finally, the analysis of Case 1 does not support the research hypotheses. The first hypothesis states that when the head is held in midline a sustained increased in ICP is less likely to occur. The findings do not support this hypothesis. Minor elevations in ICP did not persist in either control or therapeutic turns. The second hypothesis states that when the head is held in midline CPPs are more likely to remain above 50 mm Hg. In both therapeutic turns CPP drops following the turn but never reaches 50 mm Hg. Control turns show one drop and one elevation in CPP following the turn. The third hypothesis predicts that when the head is held in midline ICPWs are more likely to remain normal. The findings show more elevations in P2 waves following the therapeutic turn than control turns. Therefore all three hypotheses find no support.

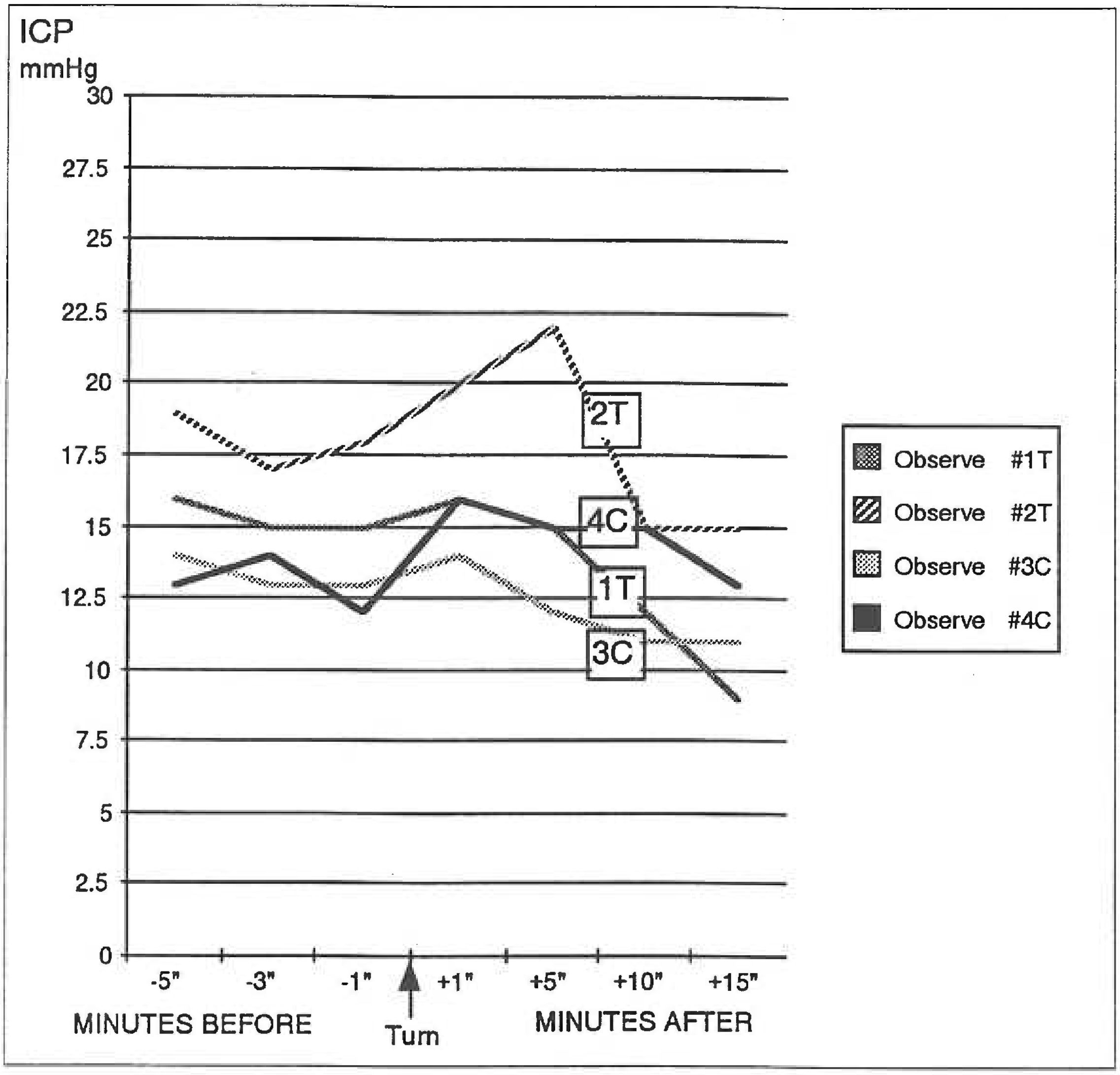


Figure 3.Intracranial pressure trends from Case 1 before and after therapeutic (T) or control (C) turns in four observation sessions.

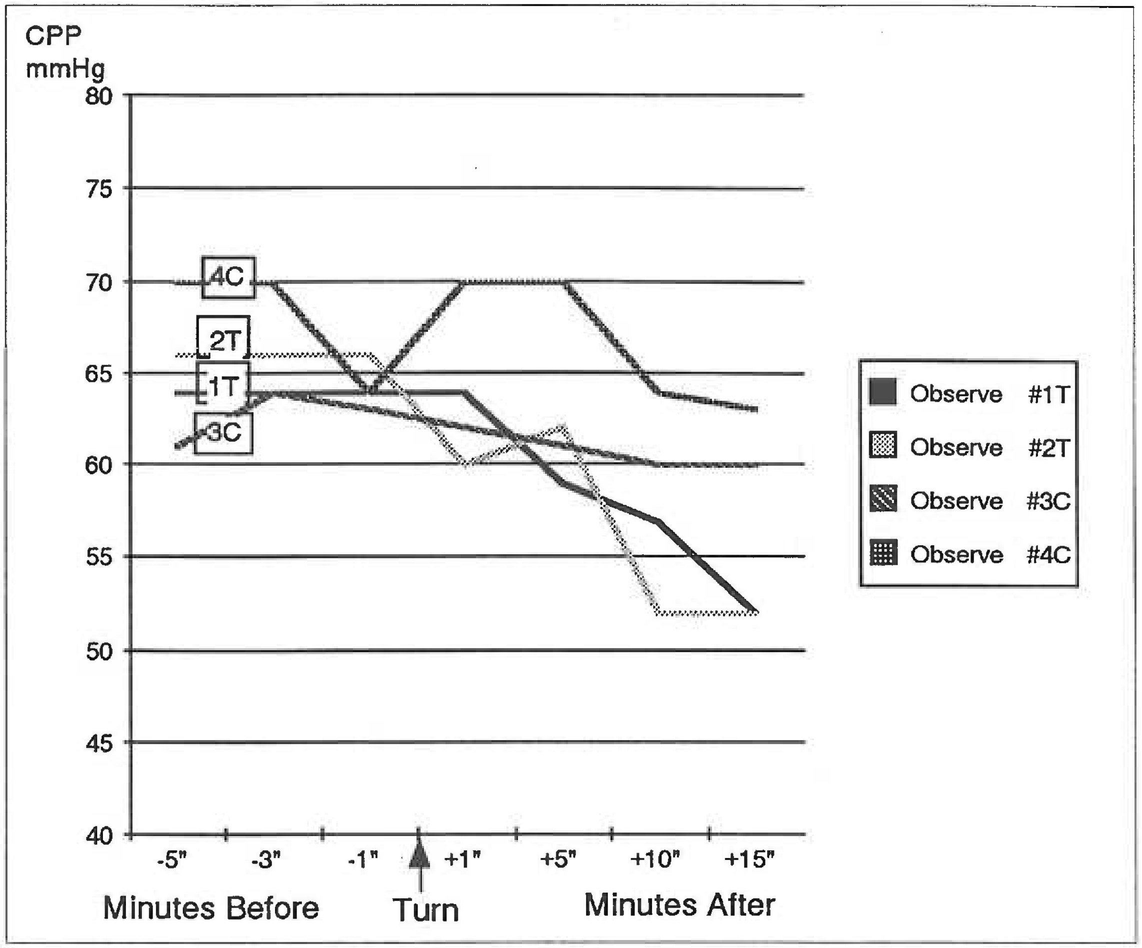


Figure 4. Cerebral perfusion pressure trends from Case 1 before and after therapeutic (T) or control (C) turns in four observation sessions.

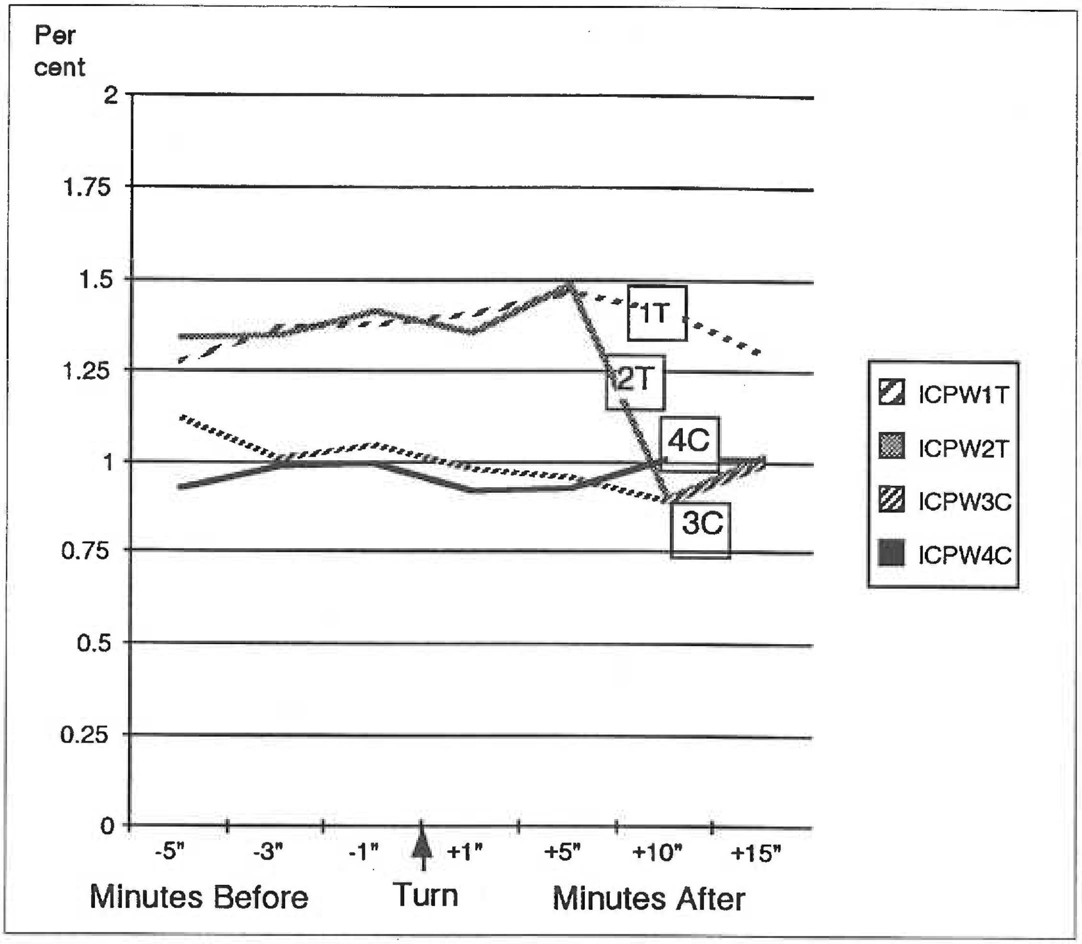


Figure 5. Intracranial pressure waveform trends from Case 1 before and after therapeutic (T) or control (C) turns in four observation sessions.

Case 2

Case 2 is a 65-year-old retired homemaker who has left leg pain and headache before admission. The family reports Case 2 fell at home without striking her head. Shortly before admission to a community hospital she became less communicative. The relationship between the left leg pain and the fall is unclear. Transfer to a larger hospital takes place and on arrival in the emergency room the Glasgow Coma Score is four to five. The X-ray reports indicate a left fracture of the tibia and fibula. In addition the CAT Scan results show a right temporal and occipital bleed with uncal herniation. Shortly after admission the subject has a craniotomy for removal of the intracerebral hemorrhage. After surgery a fiberoptic intraparenchymal monitoring device and arterial pressure monitor are present. Observation sessions begin within 12 hours of admission and 10 hours of surgical intervention. Normal waking time for this subject is 0600 hours and the first collection points occur eight hours after normal waking. Pathology reports show tissue from the operative site is that of an arterial-venous malformation.

Intracranial Pressure

Baseline mean (±SE) ICPs are 3 (±0.00) mm Hg and those following the control turn are 3.5 (±1.26) mm Hg; however, ICP more than doubled from 3 mm Hg to 7 mm Hg one minute after the turn. The elevation does not continue and decreases to baseline by the next collection point five minutes later. On the other hand, two hours later another control type turn demonstrates a slight drop of 2 mm Hg immediately following the turn. In both therapeutic turns ICP dropped slightly after the intervention but then returned to baseline. To summarize, in three of the four turns ICP decreased by 2 mm Hg when one was the control and the another therapeutic. The third demonstrated a reduction of 5 mm Hg immediately following the therapeutic turn.

Cerebral Perfusion Pressure

The CPP mean (±SE) before the first control turn is 63 (±0.33) mm Hg whereas following the turn pressures rise slightly to a mean of 65 (±3.93) mm Hg. Before the second turn the CPP average (±SE) is 56.6 (±0.67) mm Hg rising to 66 (±1.38) mm Hg after the control alignment turn. In session three a brief elevation of CPP immediately following the therapeutic turn is present. The preturn mean (±SE) for CPP is 64 (±0.33) mm Hg and 74 (±3.72) mm Hg after the turn. In the fourth and final session mean (±SE) CPP value is 71 (±0.58) mm Hg before the turn increasing slightly to 75 (±2.10) mm Hg after the therapeutic turn.

Intracranial Pressure Waveform

The ICPW has normal ratios for P2s when compared to P1 in control session one as all P2 waves are 73% smaller than P1 waves. Preturn averages (±SE) for the P2 are 66 (±0.03)% of P1 and after the turn 68 (±0.03)%. This normal waveform configuration is consistent with normal values of ICP and CPP. In control session two ICPWs elevate slightly elevated with P2 values of 82 (±0.01)% before the turn and 81.5 (±0.01)% after. A slight rise in overall ICP to upward levels of normal in this session is also consistent with concurrent increased amplitude in the P2 component of the waveform. In session three the ICPW has P2 waves that are 122% larger than P1 waves in preturn measurements but decrease to 109% after the therapeutic turn. The ICPW has P2 amplitudes 91 (±0.05)% higher than P1 before the turn that increase to 103 (±0.09)% after the turn in session four. Control sessions include several normal amplitudes for P2 waves unlike therapeutic sessions where thirteen out of fourteen measurements are abnormally high. Therefore, both therapeutic turns had abnormally elevated P2s before the turn. On the other hand the therapeutic turns show reductions in the P2 elevations by 23% in the third turn and 24% in the fourth. Measurements taken following control turns show an 9% increase and 3% decrease in P2 amplitude.

Summary

Responses for Case 2 fall into the effective category. The ICP remains under 12 mm Hg throughout all sessions. There is an increase in CPP in all turns. Fluctuations after the turn are notable as standard error doubles or increases 10 fold. Control turns show normal ICPWs while a majority in therapeutic turns demonstrate abnormal elevations.

Finally, the findings in Case 2 do not support the research hypotheses. The first hypothesis regarding ICP states that if the head is held in midline a sustained increase is less likely to occur. The ICP never demonstrated a sustained increase following either the experimental or control turns. CPPs were fairly consistent before and after both types of turns. The trend is toward a slight rise in CPP following every turn. Thus the second hypothesis is rejected because CPPs rise rather than fall below 50 mm Hg following all turns. Normal ICPW configurations are present in 30% of those collected from this subject, occurring to a large extent in the control turns. The third hypothesis predicting that normal ICPWs will follow therapeutic turns is unsupported. Elevated P2 amplitudes occur more frequently following therapeutic turns than control.

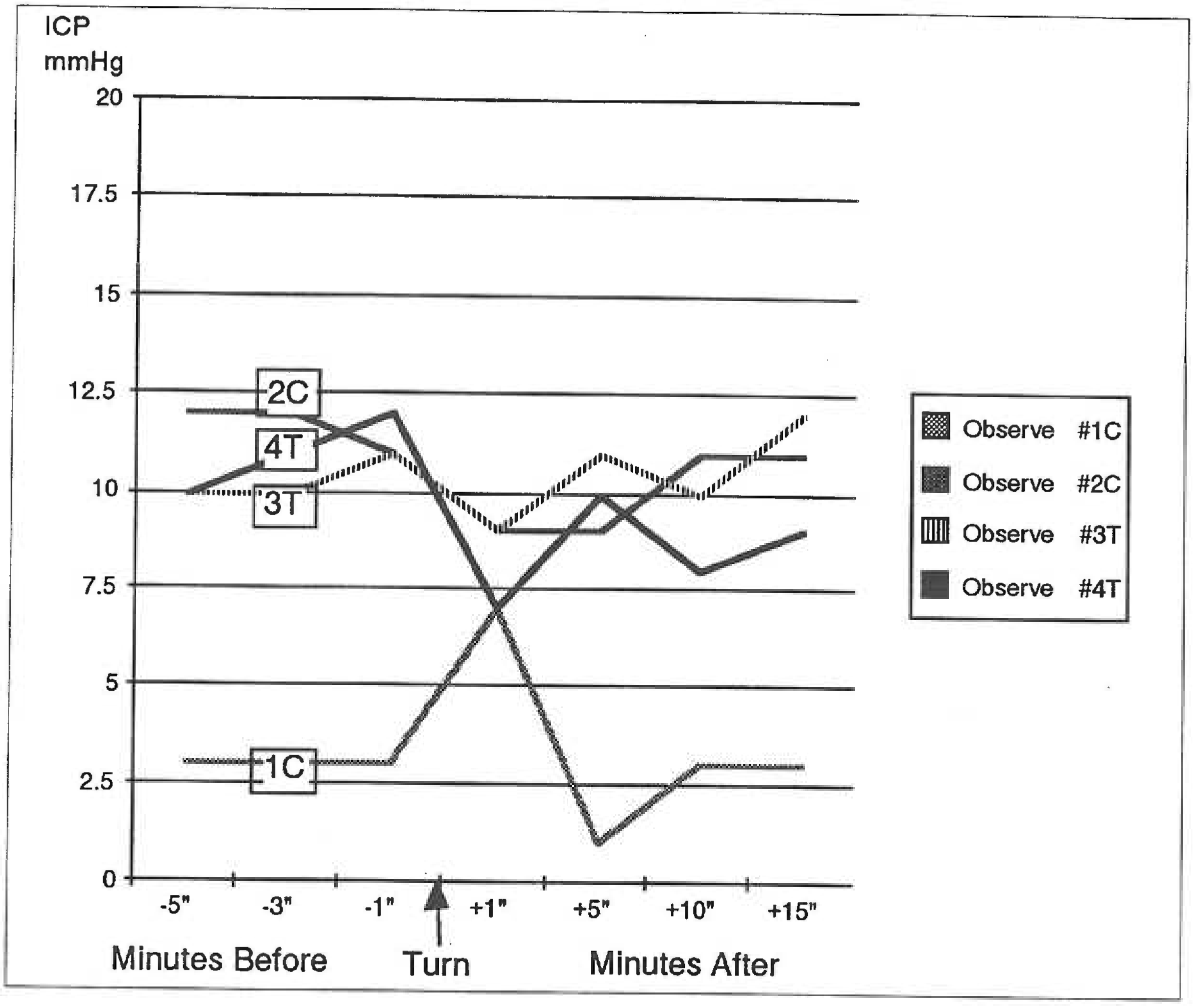


Figure 6. Intracranial pressure trends from Case 2 before and after therapeutic (T) or control (C) turns in four observation sessions.

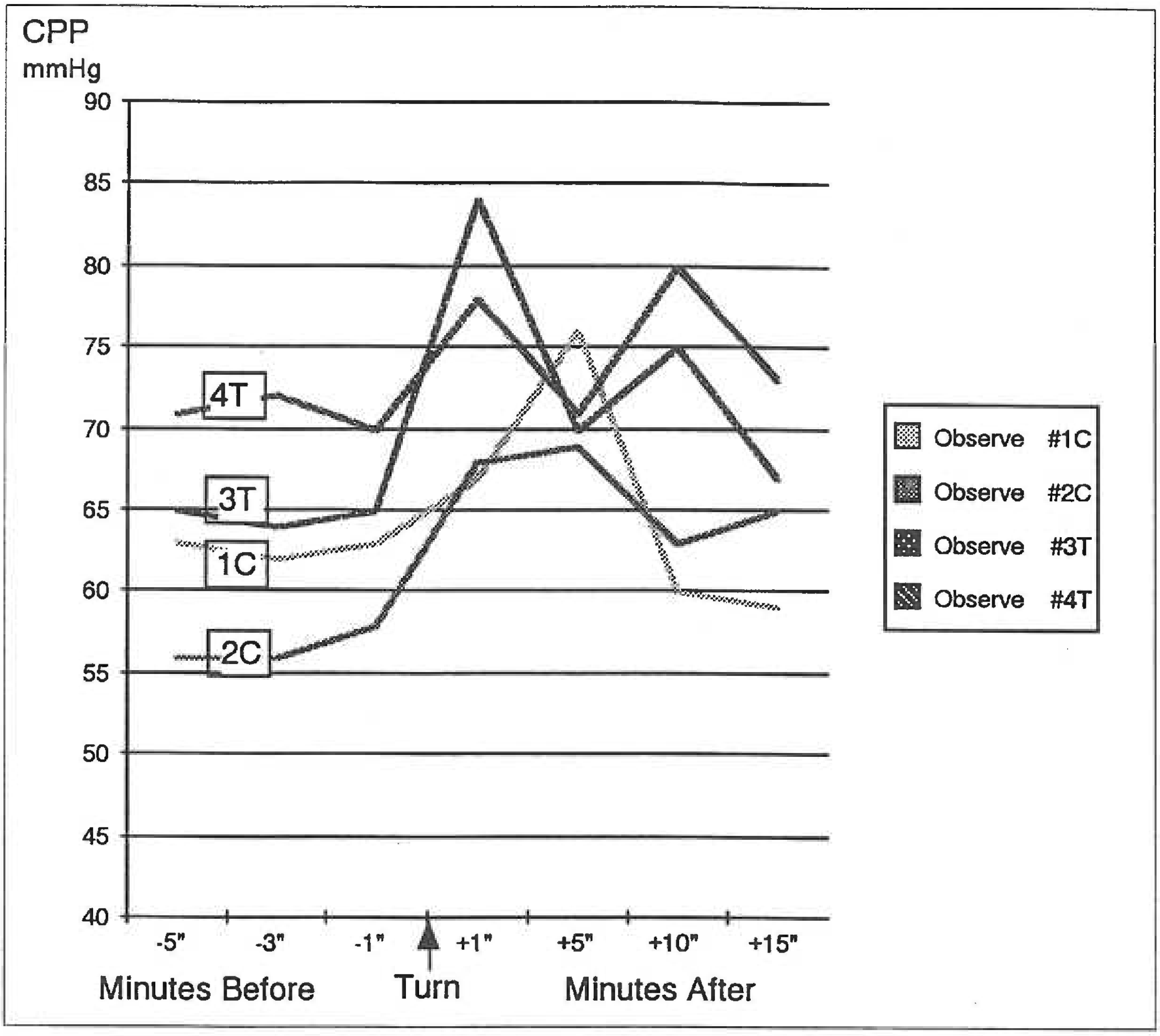


Figure 7. Cerebral perfusion pressure trends from Case 2 before and after therapeutic (T) or control (C) turns in four observation sessions.

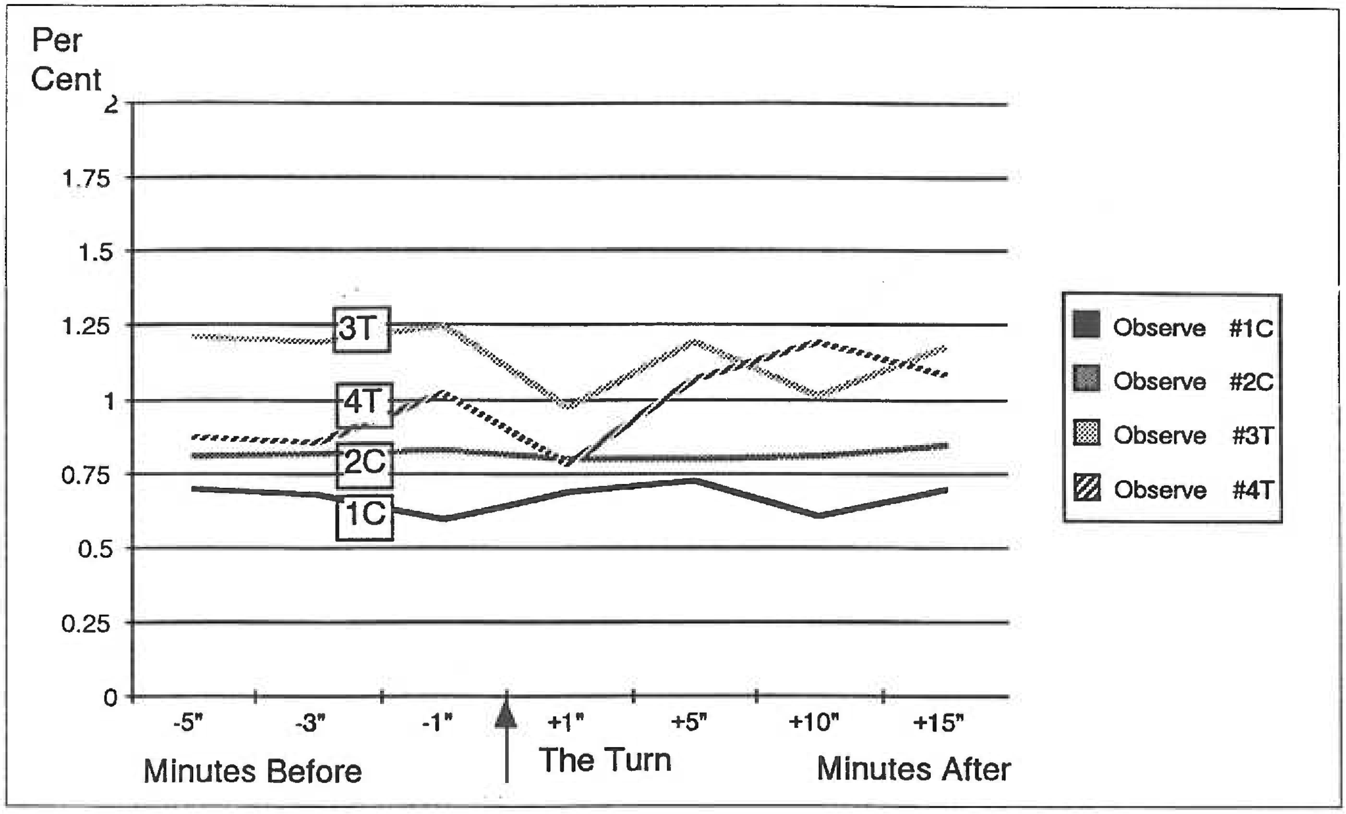


Figure 8. Intracranial pressure waveform trends in Case 2 before and after therapeutic (T) or control (C) turns in four observation sessions.

Case 3

Case 3 is a 35-year-old female jockey who is thrown from a horse while racing. The Glasgow Coma Score on admission is seven. Diagnostic testing results include a CAT scan report of right temporal skull fracture with a right-to-left hemispheric shift of 6 millimeters. Concurrent injuries include a fracture of the left clavicle and right mandible. A fiberoptic monitoring device is in the brain tissue of the nondominant hemisphere. Arterial pressure monitoring via the right radial artery is present. Observation sessions begin approximately 5 hours after normal waking time of 0500 hours and 24 hours after admission to the hospital.

Intracranial Pressures

Throughout all observations ICP levels remain under ten millimeters of mercury. In two therapeutic and one control turns the ICP trend is to drop after the turn. Control session four shows the greatest drop with means (±SE) before the turn 10 (±0.88) mm Hg and after 2 (±0.48) mm Hg. In observation session number three, a control turn, the ICP has slight rise (1 mm Hg) after the turn

Cerebral Perfusion Pressure

The range of CPPs for Case 3 throughout all turns is 68 mm Hg to 130 mm Hg. In the first therapeutic turn CPP increased after the turn by 4.2 mm Hg and in the second therapeutic turn by 27.1 mm Hg. After the turn in session two a marked increase of CPP takes place with neurologic testing. Stimulation includes dolls eye, sternal rubs, extremity and reflex testing. In the control turn of observation three a slight drop of 3 mm Hg is present after the turn. The fourth observation shows a slight decrease of 4 mm Hg.

Intracranial Pressure Waveform

The amplitude of P2 compared to P1 wave is within normal limits once in all Case 3 observations. Mean (±SE) amplitudes for P2 decrease from 104 (±0.02)% to 85 (±0.06)% in observation session one that is a therapeutic turn. However, in the second session, another therapeutic turn, mean (±SE) values increase from 109 (±0.03)% to 139 (±0.11)% after the turn. Event records of the second session report restlessness and agitation while a physician did neurologic assessments that include a sternal rub and ocular reflex testing. In control session three a small increase of 3% occurs after the turn. In control session four the P2 amplitudes show a decreasing trend.

Summary

The ICPs for Case 3 remain under 10 mm Hg increasing slighty after one control turn. Increases in CPP occur in three turns and a decrease in one control. Standard error in CPPs after the turns is greater when the direction of change as shown in Table 7. Only one waveform in all has a normal P2 amplitude.

The findings in Case 3 do not support the research hypotheses. The first hypothesis states ICP will be less likely to have sustained elevations following therapeutic turns. Sustained elevations did not occur in either control or therapeutic turns. The second hypothesis predicts that CPPs will be less likely to drop to 50 mm Hg following therapeutic turns. CPPs never falls below 67 mm Hg. The third hypothesis regarding ICPWs states that elevated P2 waves are more likely to occur following control type turns. Ninety-two percent of ICPWs before and after turns elevate in this case. Weak support for hypothesis number 3 is present. Following the first therapeutic turn two normal ICPWs are present. On the other hand, all ICPWs elevate following control turns.

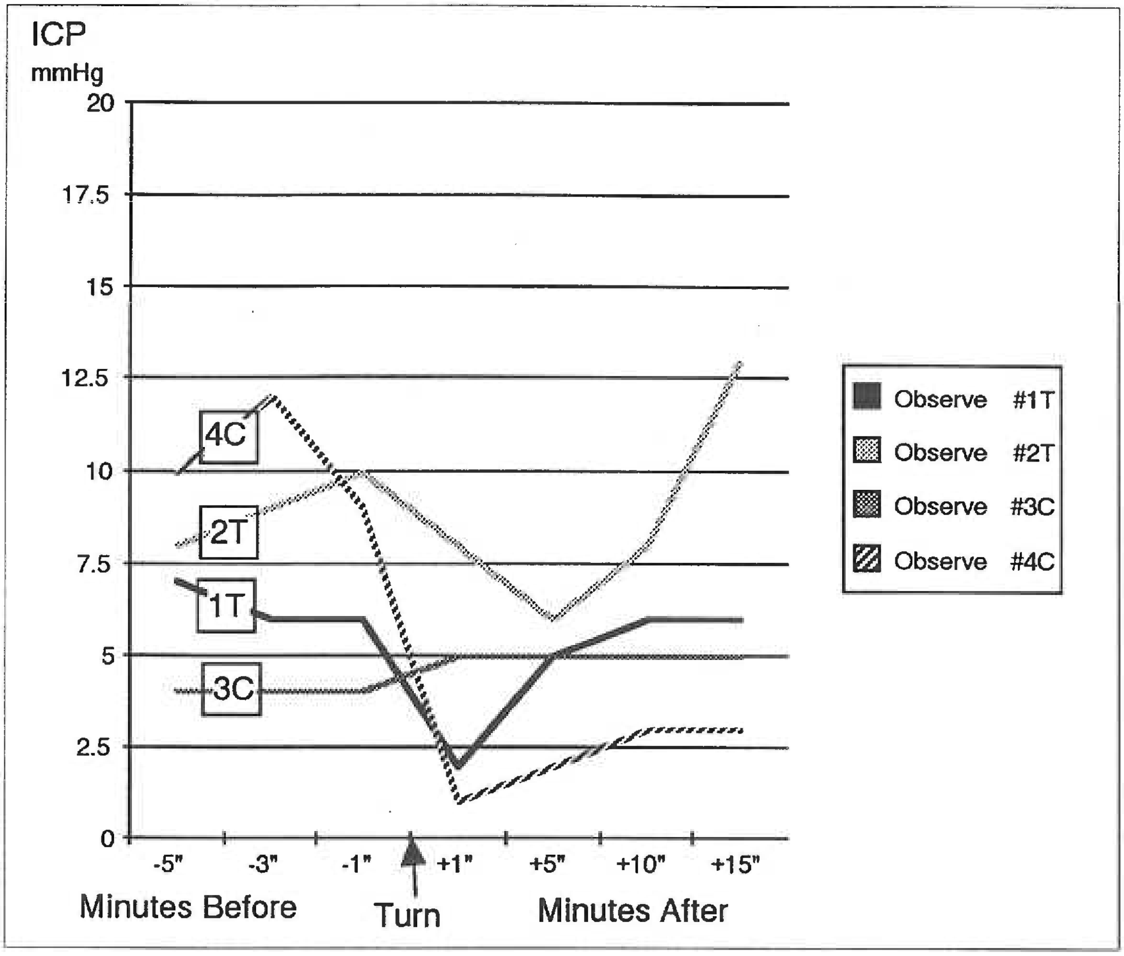


Figure 9. Intracranial pressure trends for Case 3 before and after therapeutic (T) or control (C) turns in four observation sessions.

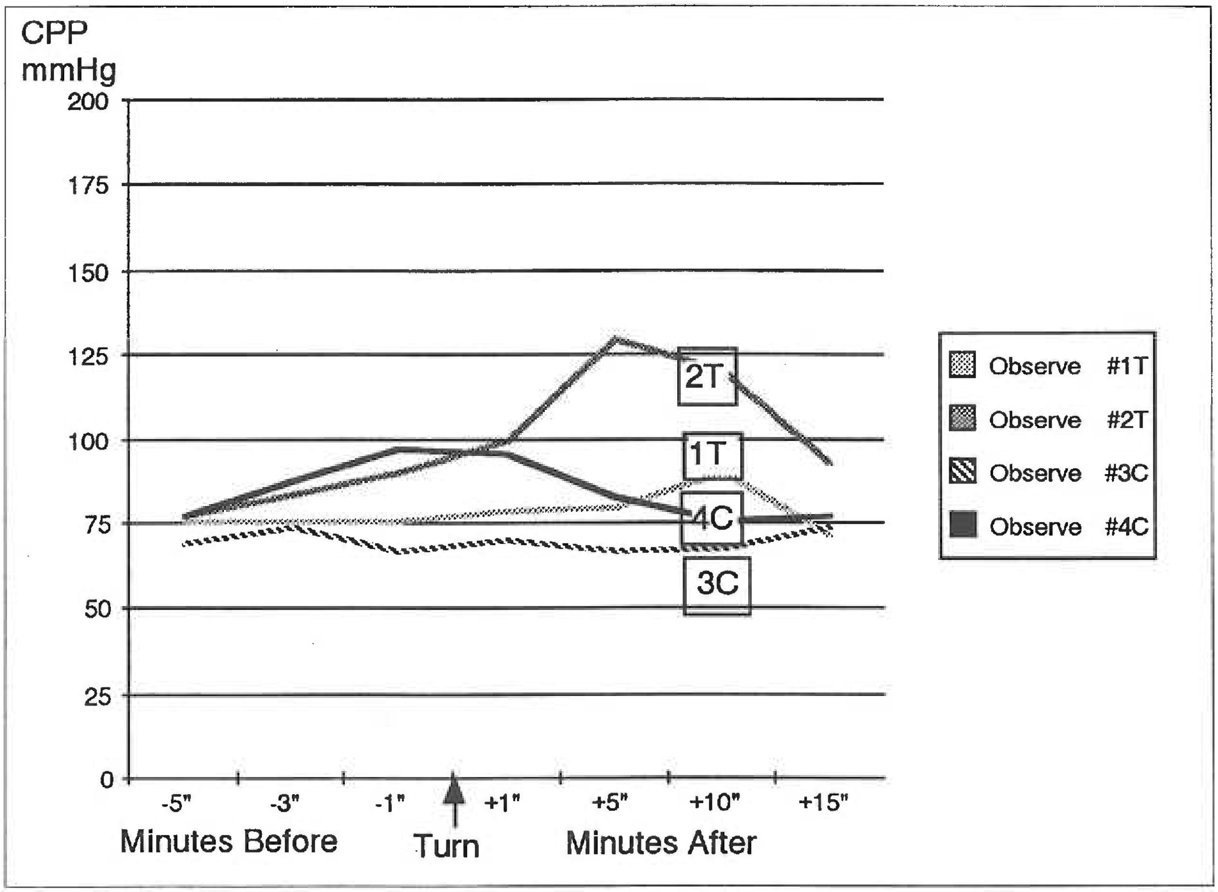
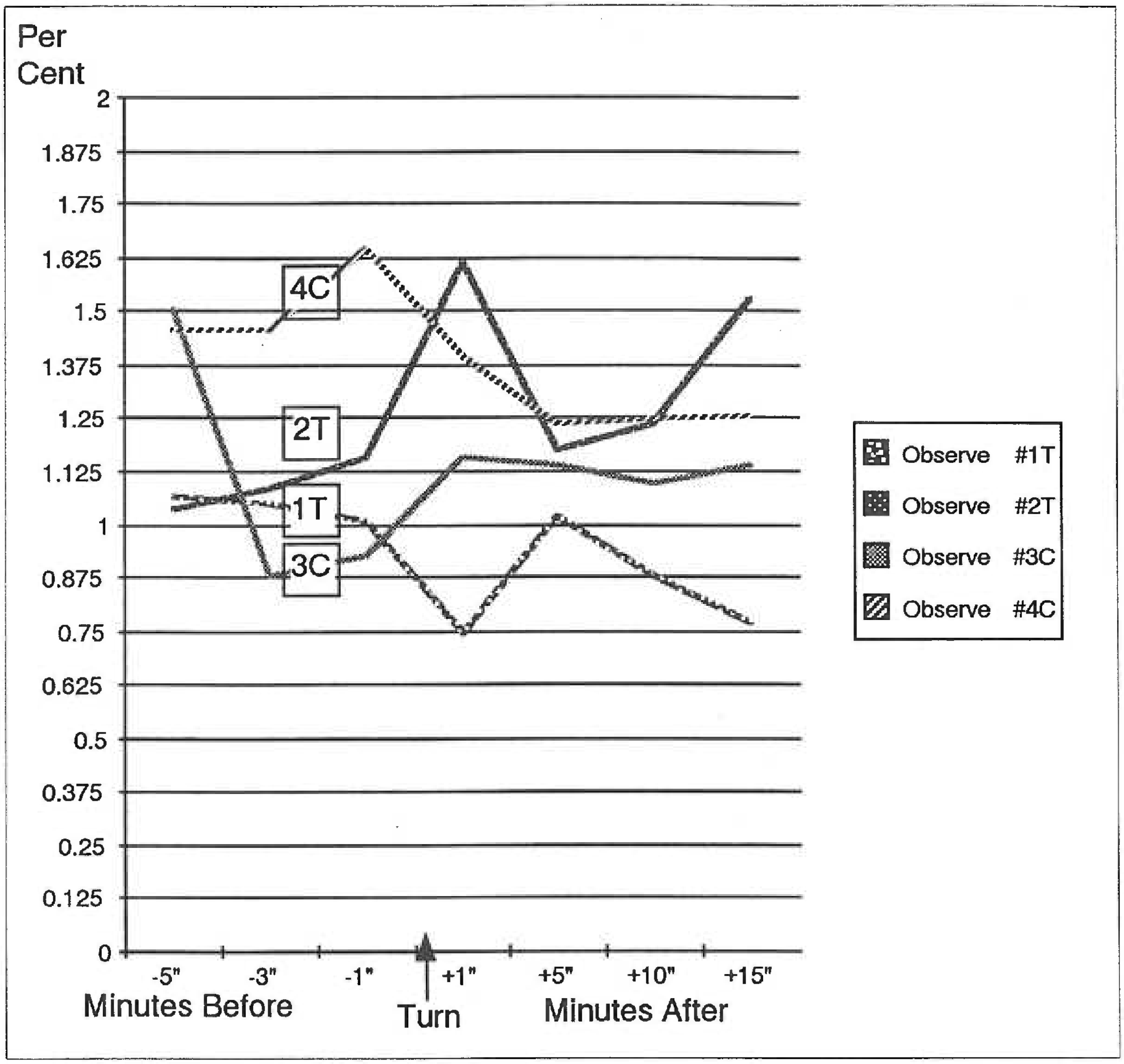


Figure 10. Cerebral perfusion pressure trends in Case 3 before and after therapeutic (T) or control (C) turns in four observation sessions.



<u>Figure 11</u>. Intracranial pressure waveform trends in Case 3 before and after therapeutic (T) or control (C) turns in four observation sessions.

Case 4

Case 4 is a 24-year-old female who sustains a depressed left temporal skull fracture following an assault with a blunt instrument. The results of the CAT Scan demonstrate an epidural and intracerebral hemorrhage in the left temporal area. Glasgow Coma Score on admission is 7 due to intubation and loss of the verbal score. Observation sessions begin five hours after normal waking time of 1100 hours and approximately 15 hours after hospitalization.

Intracranial Pressure

In the two control sessions ICP increases after the turn. A slight increase of 5 mm Hg occurs in session one whereas an increase of 4.1 mm Hg occurs in the second session. The actual rise in ICP from baseline to immediately after the turn is 8 mm Hg. However, an important observation is that this elevation is not sustained beyond nine minutes after the turn. In both therapeutic turns decreases in ICP occur yet remain in normal limits.

Cerebral Perfusion Pressure

The range of CPP for Case 4 is 52 mm Hg to the high of 74 mm Hg. In three sessions, one control and two therapeutic, CPPs rise slightly; 9 mm Hg, 1 mm Hg and 7.9 mm Hg respectively from preturn mean values. The slight drop of CPP in session two following the turn is only 4.6 mm Hg.

Intracranial Pressure Waveform

In Case 4 there are 23 abnormal waveforms. Only five waveforms evaluated for P2 amplitude are of normal height.

Preturn P2 mean (±SE) values are 116 (±0.58)% of P1 in session one. A mean value of 108 (±0.11)% follows the turn. Also a slight decrease of 10% occurs in session three comparing preturn amplitudes with those following the turn. A slight rise in percentage is present in session two, 93 (±0.15)% to 100 (±0.10)%. Session four mean (±SE) percentage values for P2 amplitude before (±0.05) and after (±0.04) the turn are 88 %.

Summary

Case 4 has decreases in ICP after therapeutic turns and increases after control. CPP rises in three turns, two therapeutic and one control but decreases in one. The majority of P2 amplitudes are abnormally high before and after turns.

The first hypothesis predicts that sustained elevations in ICP are more likely to follow control turns. Sustained elevations in ICP did not occur following either control or therapeutic turns. However, the direction of change does support the first hypothesis. The second hypothesis predicts that CPP is more likely to fall below 50 mm Hg following control turns. The only drop in CPP following any turns happens in a control type and the lowest CPP then is 60 mm Hg. The third hypothesis predicts ICPWs are more likely to be normal in configuration following therapeutic turns. This statement has weak support twice, once in single readings after therapeutic turns and once following a control turn.

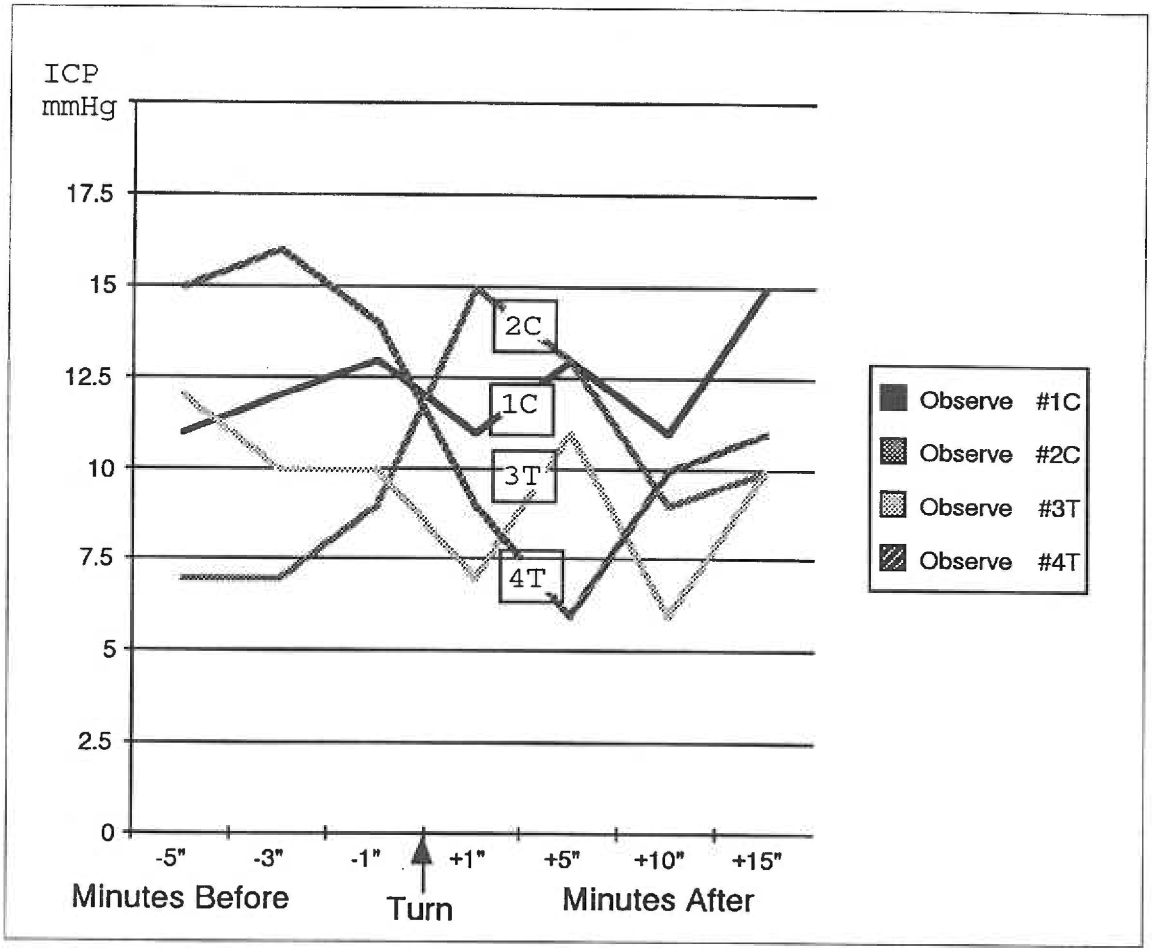


Figure 12. Intracranial pressure trends from Case 4 before and after therapeutic (T) or control (C) turns in four observation sessions.

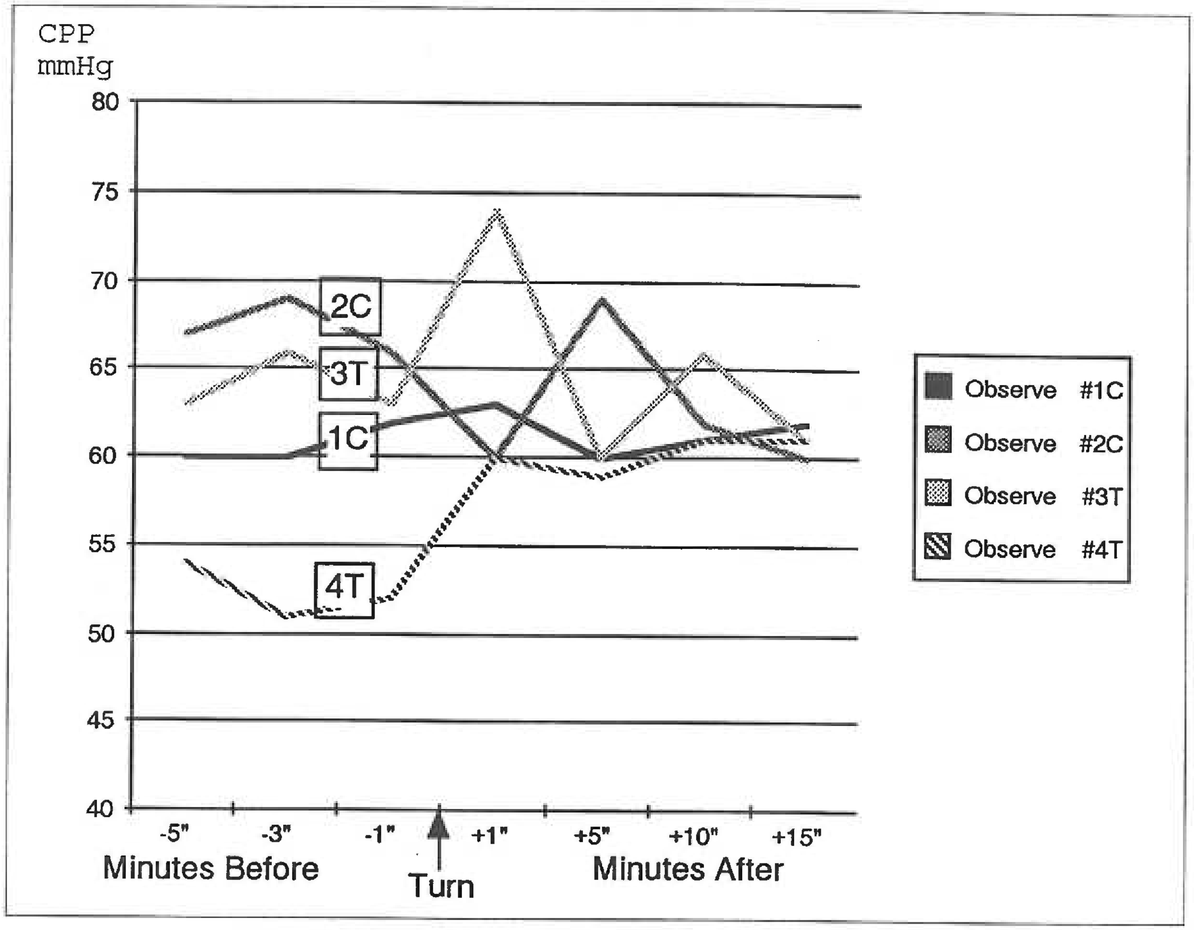


Figure 13. Cerebral perfusion pressure trends of Case 4 before and after therapeutic (T) and control (C) turns in four observation sessions.

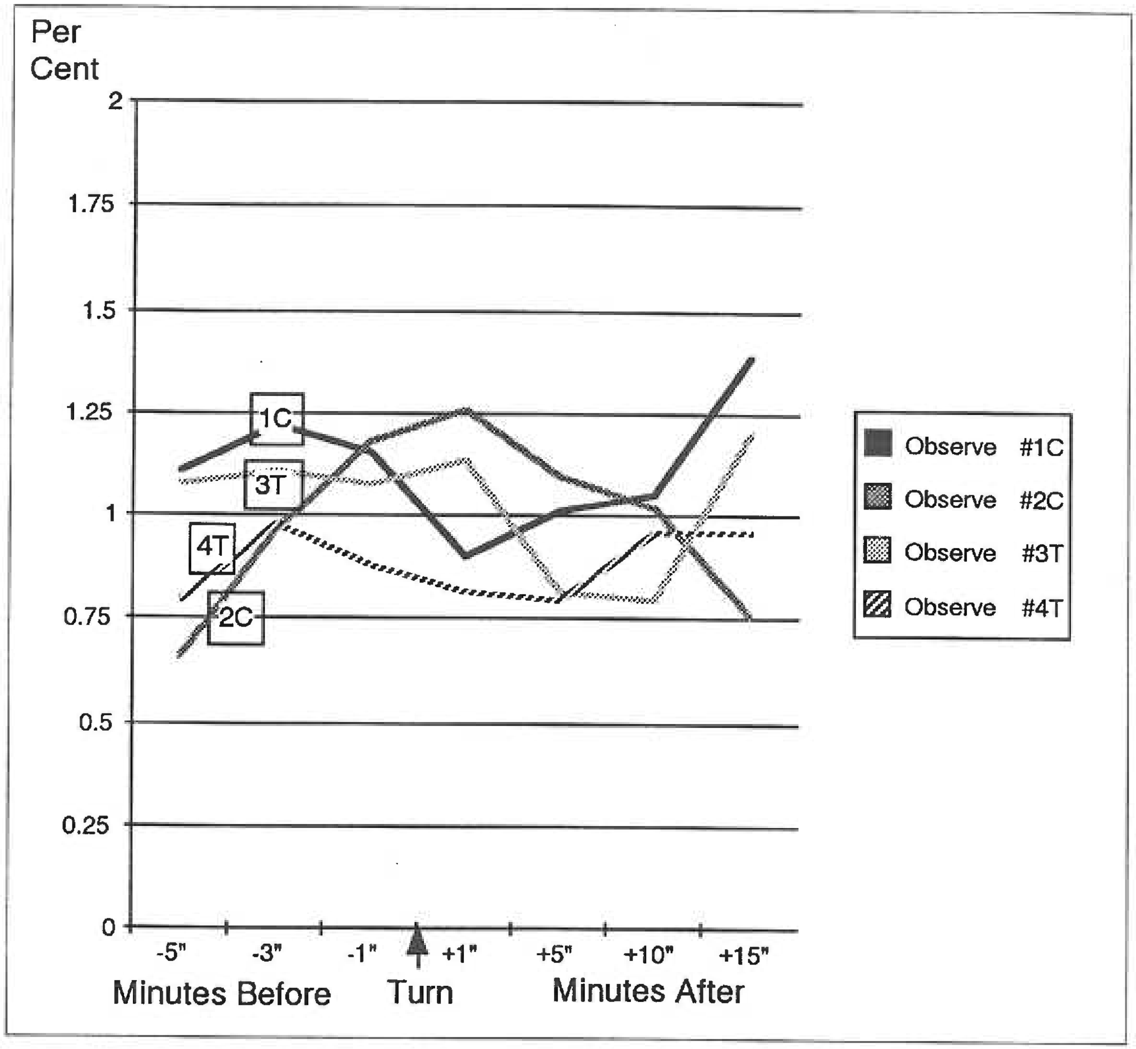


Figure 14. Intracranial pressure waveform trends in Case 4 before and after therapeutic (T) and control (C) turns in four observation sessions.

Case 5

Case 5 is an 82-year-old retired homemaker who collapses at home early in the morning. Her husband finds her on the floor of the bedroom having difficulty moving her arm as well as speaking. She arrives in the Emergency Room and the CAT scan demonstrates an intracerebral hemorrhage in the right posterior frontal area extending to the anterior parietal area. A midline shift of 4 mm right-to-left is present. A repeat scan 24 hours later shows a midline shift of 12 mm with right uncal herniation. Data collection begins within 24 hours of admission to the ICU and approximately 4 hours after her normal waking hour of 0600.

Intracranial Pressure

The range of ICP observed in all four sessions for Case 5 is from 9 mm Hg and 29 mm Hg. In session one an increase of 7 mm Hg is present immediately after the therapeutic turn. This increase is only for five minutes and returns to a value slightly greater than the first measurement. The second session demonstrates a drop of 6 mm Hg immediately after the control turn but increases to a value within 3 mm Hg of initial session values. The highest levels of ICP in the entire study occur in session three. Preturn mean (±SE) averages are 25.6 (±0.33) mm Hg and those following the turn 26.25 (±0.75) mm Hg. In the final therapeutic observation for this subject an increase of 5 mm Hg in ICP is present immediately after the therapeutic turn and then decreases. The mean (±SE) average before the turn is 9.6 (±0.88) mm Hg while those following the turn are 13.25 (±1.89) mm Hg.

Cerebral Perfusion Pressure

The range of CPP occurring in this subject is 65 mm Hg to 91 mm Hg. In session one the change in CPP immediately following the therapeutic turn is only 1 mm Hg, however, mean (±SE) average before the turn is 83 (±4.62) mm Hg and that following is 75.25 (±0.75) mm Hg. The second session (control) shows very little fluctuation with mean (±SE) averages of 75 (±1.53) mm Hg before the turn and 75.25 (±0.48) mm Hg after the turn. Immediately following the turn in session three a drop of 11 mm Hg is present with an increase to baseline within nine minutes. Preturn mean (±SE) value is 76.3 (±0.33) mm Hg while that following is 73 (±2.86) mm Hg. In session four a brief increase in CPP (8 mm Hg) occurs following the therapeutic turn. The mean (±SE) average before the fourth turn is 70 (±0.58) mm Hg with an increase to 76.75 (±0.85) mm Hg after the turn.

Intracranial Pressure Waveform

Analysis of ICPWs estimates the P2 amplitude compared to P1. All measurements in this subject demonstrate abnormally elevated amplitude of the P2 waves. A mean (±SE) increase from 170 (±0.06)% to 207 (±0.09)% is noted in session one following the therapeutic turn. In session two the mean amplitudes of P2 continue to increase from 204 (±0.10)% to 216 (±0.16)%. A slight decrease in P2 amplitude occurs in session three as before the turn P2s are 251 (±0.05)% and those after 222 (±0.19)%. In the fourth session the mean (±SE) amplitude before the turn is 176 (±0.03)% and after the turn 190 (±0.06)%.

Summary

Responses after Case 5 turns increase and decrease and most ICPs are abnormally high. Direction of change for CPP is also inconsistent. All P2 waves are greater than P1 and at some points double P1 size.

The first hypothesis predicts that sustained elevations in ICP are more likely to occur following control type turns. This hypothesis is rejected because ICP demonstrates sustained increases following three turns, two therapeutic and one control. Following the first therapeutic turn ICP measurements show an upward trend. The mean ICP following the second therapeutic turn shows a sustained increase of 4 mm Hg compared to those before the turn. The first control turn precedes a decreasing trend and a slight increase follows the second control turn. The second hypothesis states that CPP is more likely to remain above 50 mm Hg in the therapeutic turns, which has no support. Mean CPPs following therapeutic turns decrease by 8 mm Hg in the first, and increase in the second 6 mm Hg. There is only a slight increase of ICP following the first control of 0.75 mm Hg and the second control yields a drop of 3 mm Hg. The third hypothesis states that ICPWs are more likely to be normal in configuration following therapeutic turns. This hypothesis finds no support because P2 waves increased in amplitude following both therapeutic turns and one control turn.

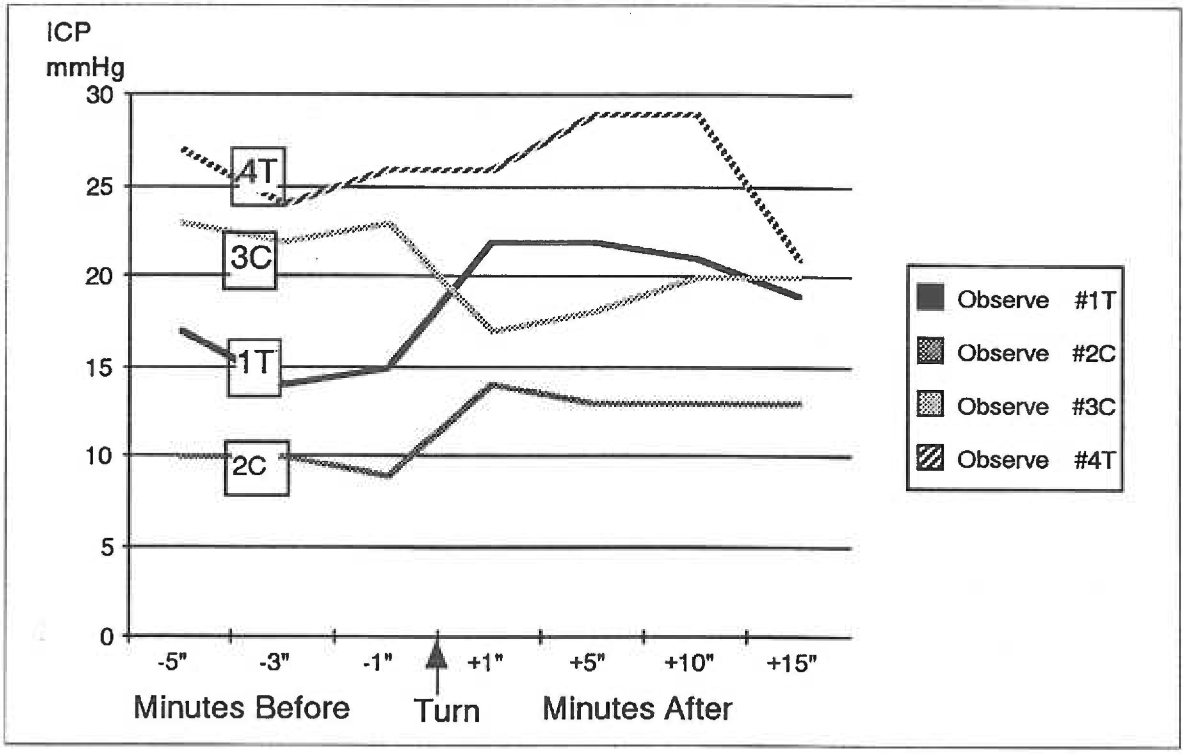


Figure 15. Intracranial pressure trends in Case 5 before and after therapeutic (T) or control (C) turns in four observation sessions.

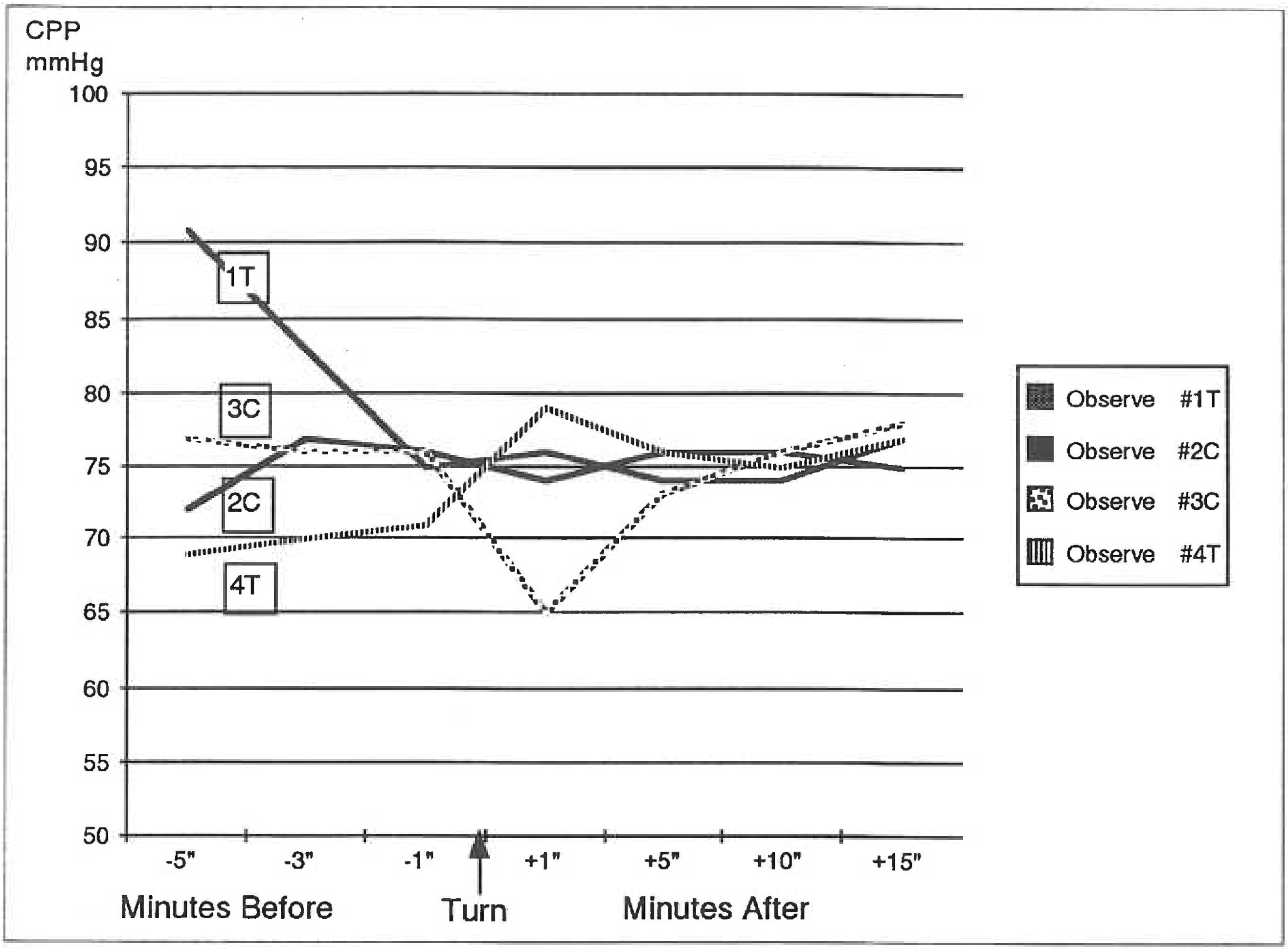


Figure 16. Cerebral perfusion pressure trends in Case 5 before and after therapeutic (T) and control (C) turns in four observation sessions.

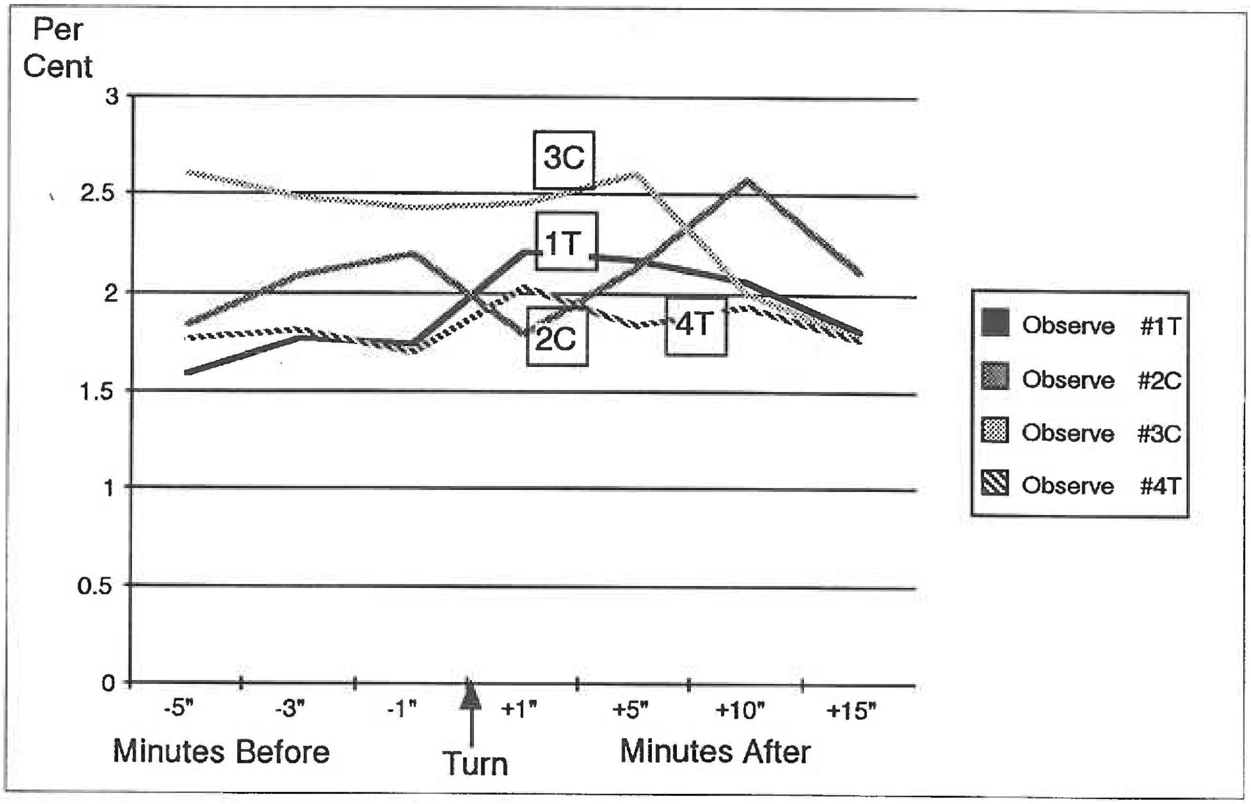


Figure 17. Intracranial pressure waveform trends in Case 5 before and after therapeutic (T) and control (C) turns in four observation sessions.

Table 7

Mean and Standard Error of ICP, CPP and ICPW in Five Cases

Case Session Turn	ICP mm Hg Mean and SE		CPP mm Hg Mean and SE		% P2 Amplitude Compared to P1 Mean and SE	
	Before	After	Before	After	Before	After
C1 S1T	15 ±0.33	13 ±1.58	64 ±0.00	58 ±2.48	134 ±0.03	140 ±0.04
C1 S2T	18 ±0.58	18 ±1.78	66 ±0.00	56 ±2.63	137 ±0.03	119 ±0.14
C1 S3C	13 ±0.33	12 ±0.71	63 ±0.88	61 ±0.48	106 ±0.03	95 ±0.02
C1 S4C	13 ±0.58	15 ±0.63	68 ±2.00	67 ±1.89	97 ±0.02	96 ±0.02
C2 S1C	3 ±0.00	3.5 ±1.26	63 ±0.33	66 ±3.93	66 ±0.03	68 ±0.03
C2 S2C	12 ±0.33	10 ±0.58	57 ±0.67	66 ±1.38	82 ±0.01	82 ±0.01
C2 S3T	10 ±0.33	11 ±0.65	65 ±0.33	74 ±3.72	122 ±0.01	109 ±0.06
C2 S4T	11 ±0.58	9 ±0.65	71 ±0.58	76 ±2.10	91 ±0.05	103 ±0.09
C3 S1T	6.3 ±0.33	4.7 ±0.95	76 ±0.00	80 ±3.71	104 ±0.02	85 ±0.06
C3 S2T	9 ±0.58	8.7 ±1.49	84 ±3.76	111±8.60	109 ±0.03	139 ±0.11
C3 S3C	4 ±0.00	5 ±0.48	70 ±2.08	70 ±1.55	110 ±0.20	113 ±0.04
C3 S4C	10.3±.88	2.2 ±0.48	87 ±5.78	83 ±4.60	152 ±0.06	128 ±0.04

Note. The letter C and a number represent each case. The letter S followed by a number indicates the observation session. The letters T or C indicates the type of turn.

Table 7

Mean and Standard Error of ICP, CPP and ICPW in Five Cases (continued)

Case Session Turn	ICP mm Hg Mean and SE		CPP mm Hg Mean and SE		% P2 Amplitude Compared to P1 Mean and SE	
	Before	After	Before	After	Before	After
C4 S1C	12 ±0.58	12.5 ±0.96	61 ±0.67	62 ±0.65	116 ±0.03	108 ±0.11
C4 S2C	7.6 ±0.67	11.7 ±1.38	67 ±0.88	63 ±2.14	93 ±0.15	100 ±0.10
C4 S3T	10.6 ±0.67	8.5 ±1.19	64 ±1.00	65 ±3.20	109 ±0.01	99 ±0.10
C4 S4T	15 ±0.58	9 ±1.08	52 ±0.88	60 ±0.48	88 ±0.05	88 ±0.04
C5 S1T	15 ±0.88	21 ±0.71	83 ±4.62	75 ±0.75	170 ±0.06	207 ±0.09
C5 S2C	23 ±0.33	19 ±0.25	75 ±1.53	75 ±0.48	204 ±0.10	216 ±0.16
C5 S3C	26 ±0.33	26 ±0.75	76 ±0.33	73 ±2.86	251 ±0.05	222 ±0.19
C5 S4T	9 ±0.88	13 ±1.89	70 ±0.58	77 ±0.85	176 ±0.03	190 ±0.06

Note. The letter C and a number identify each case. The letter S and number indicate the observation session. The letter T or C describes the type of turn.

Analysis of Five Cases as a Group

Next, the ANOVA is the statistical test used in analysis of the aggregate. The group consists of Cases One through Five. Descriptive statistics giving means before and after therapeutic and control turns are available. Then inferential statistics or two-way repeated measures ANOVA for each variable are present. However, when the five formal cases are grouped several differences are evident. Four out of the five cases need artificial airway and ventilation support. The CAT scans reports vary from temporal lobe herniation to small shifts. In addition some subjects receive very aggressive administration of osmotic diurectics while others did not. Skull fractures are present in two cases that may change pressure outputs by providing a temporary outlet. Post operative events such as increased swelling within the brain alter some pressure values while the cases treated medically escape this physiologic challenge. Thus, the five cases are not a homogeneous group.

Intracranial Pressure

Mean scores in all cases of ICP measurements before and after turns are in Table 8 and Figure 18. Scores for therapeutic and control turns for all five cases are also shown in Table 8. The mean standard deviation (±SD) before treatment conditions was 12.07 (±3.29) mm Hg that was not significantly different from the mean after, 11.53 (±4.52) mm Hg. Additionally the mean before the control turn is 12.33 (±7.05) mm Hg and the mean after the turn is 11.68

(±7.23) mm Hg, not substantially different. Finally, the interaction in Table 9 of treatment and time is also not significant. The differences between the before measures and the after measures do not appear to be a result of the type of turn.

Table 8

Means ± Standard Error of Intracranial Pressure Before and After Therapeutic and Control turns in Five Subjects

	Therapeutic	Control
Before	12.07 mm Hg ±0.43	12.33 mm Hg ±1.11
After	11.53 mm Hg ±0.82	11.68 mm Hg ±1.14

Table 9

Summary Table for ANOVA when the Dependent Variable is ICP

	Sum of Squares	df	Mean Squares	F	Significance (p)
Main Effect					
Treatment	.22	1	.22	.01	.928
Within	92.87	4	23.22		
Time	1.80	1	1.80	1.01	.373
Within	7.15	4	1.79		
Interaction Treatment by Time	.02	1	.02	.00	.959

$p = <.05$.

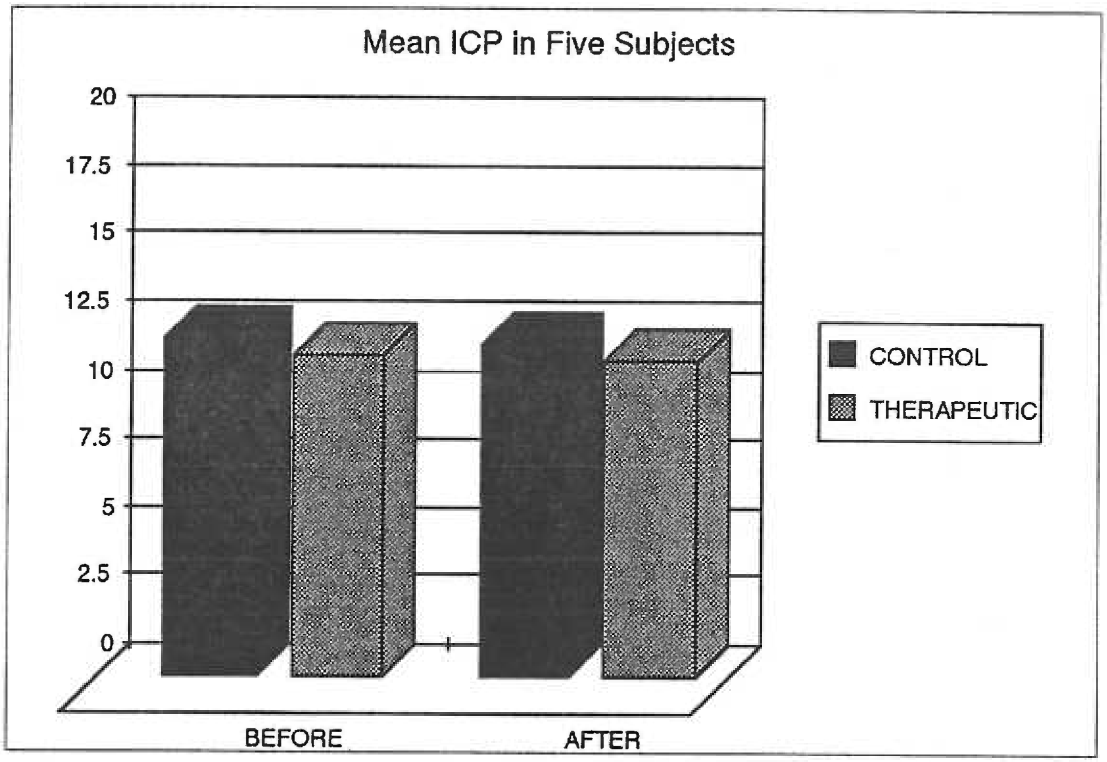


Figure 18. Means of intracranial pressure before and after turning for five subjects

Cerebral Perfusion Pressure

The results of ANOVA performed on CPPs are shown in Table 11. The main effect for treatment (therapeutic compared to control) is not significant. As shown in Table 10 and Figure 19 the mean across treatment conditions is 142.72 mm Hg not significantly different from the control mean of 137.12 mm Hg. The main effect for time (before vs. after) is also not significant. The mean total CPPs before the turn is 138.14 mm Hg and the mean total after is 141.70 mm Hg, different by only 3.56 mm Hg. Finally, the interaction of treatment and time is also not significant (See Table 11).

Table 10

Means ± Standard Error of Cerebral Perfusion Pressure Before and After Therapeutic and Control Turns for Five Subjects

	Therapeutic	Control
Before	69.47 mmHg±1.59	68.67 mmHg±1.28
After	73.25 mmHg±2.69	68.45 mmHg±1.01

Table 11

Summary Table for ANOVA when the Dependent Variable is Cerebral Perfusion Pressure

	Sum of Squares	df	Mean Squares	F	Significance (p)
Main Effect					
Treatment	39.20	1	39.20	1.00	.374
Within	156.63	4	39.16		
Time	15.90	1	15.90	.66	.463
Within	96.54	4	54.14		
Interaction					
Treatment by Time	20.00	1	20.00	.99	.376

p = <.05.

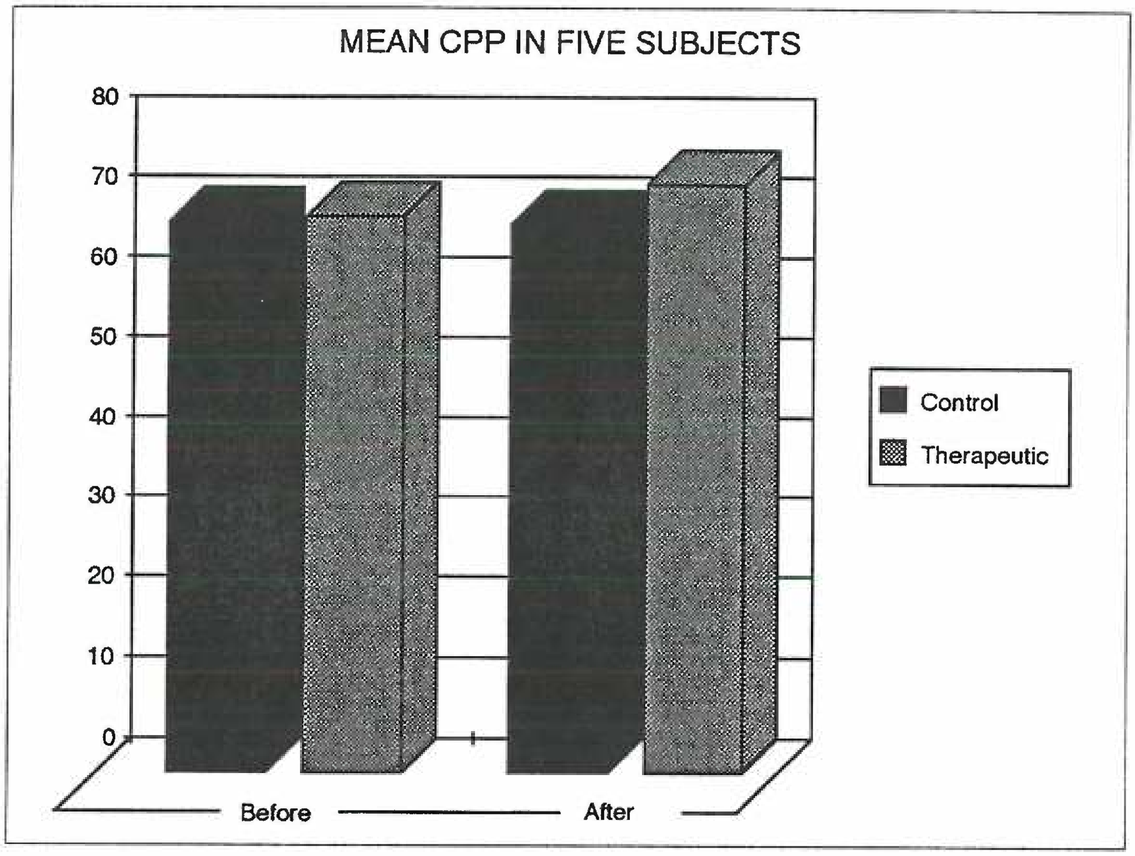


Figure 19. Mean values for cerebral perfusion pressures before and after turns in five cases.

Intracranial Pressure Waveform

The findings of ANOVA for ICPWs are present in Table 13. The ICPW has three peaks and the amplitude of the second is compared to the amplitude of the first using percentages. The main effect for treatment (therapeutic vs. control) is not significant. As shown in Table 12 and Figure 20 the mean across treatment conditions is 2.52% and that is not significantly different from the control mean, 2.51%. The main effect for time (before vs. after) also is not significant. The mean before the turn is 1.24% and the mean after the turn is 1.28% not significantly different. Finally, the interaction in Table 13 of treatment and time is insignificant, indicating that any difference detected is not due to the type of turn.

Table 12

Means ± Standard Error of Intracranial Pressure Waveform Before and After Therapeutic and Control turns for Five Subjects

	Therapeutic	Control
Before	1.24% ±0.05	1.28% ±0.14
After	1.28% ±0.07	1.23% ±0.08

Table 13

Summary Table for ANOVA of Intracranial Pressure Waveform in Five Subjects

	Sum of Squares	df	Mean Squares	F	Significance (p)
Main Effect					
Treatment	.00	1	.00	. 00	. 973
Within	.39	4	.10		
Time	.00	1	.00	.03	.878
Within	.01	4	.00		
Interaction Treatment by Time	.01	1	.01	1.25	.327

p = <.05.

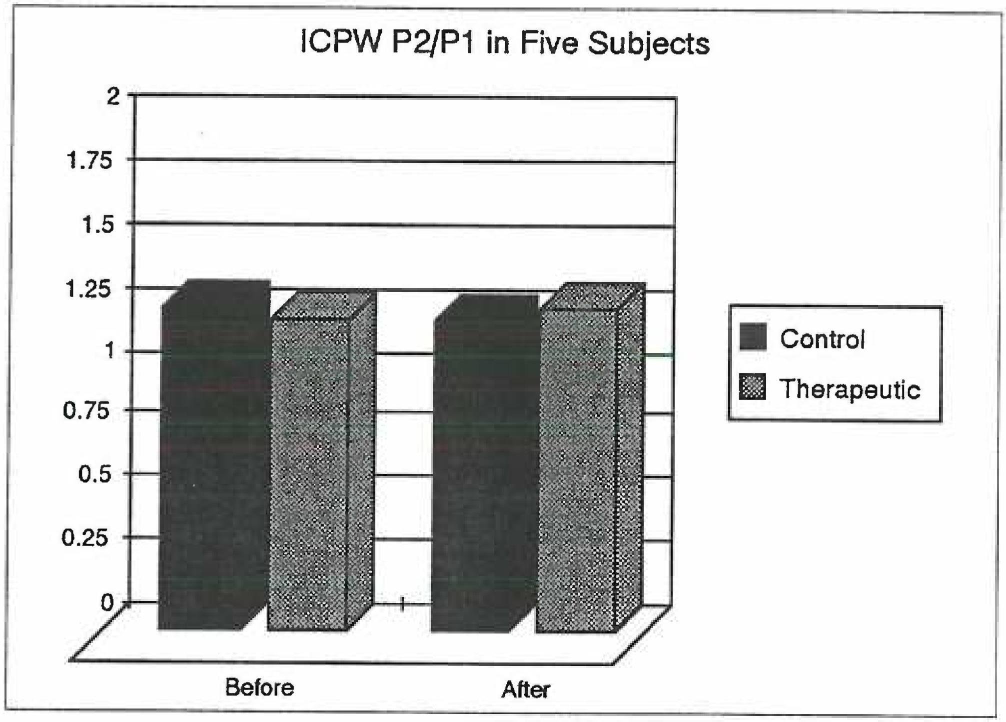


Figure 20. Mean values for the intracranial pressure waveforms before and after turns in five cases.

Analysis of Covariance

Another way to analyze variables in this study is to investigate the effect of ICPW has upon the other dependent variables. This is accomplished when ICPWs are used as covariates. Analysis of Covariance (ANCOVA) is a statistical test that combines ANOVA and regression. ANCOVA is appropriate when initial equivalency of the dependent variables is in question as is true in this study (Polit and Hungler, 1987). Covariates are useful when the investigator believes one variable may predict the direction or outcome of the other. Huck et al. (1974) describe the relationship as an increase of a specified number of points on the covariate being related to about the same increase on the dependent variable. Specifically, is it possible that when ICP levels are elevated so are P2 amplitudes? The ANCOVA findings for this study are available in Table 14 and 15.

Intracranial Pressure

Analysis of the relationship between ICPWs and ICP occurs in ANCOVA. Briefly the statistical findings of interest occur in the interaction portion of ANCOVA. Here the F-ratio statistic of 19.21 approaches statistical significance as shown in Table 14, which means ICPWs may have a direct relationship to ICP.

Cerebral Perfusion Pressure

The ANCOVA results display an insignificant relationship between ICPWs and CPP. The F statistic has a value of .12 as shown in Table 13 which is not significant. Therefore, the relationship between ICPW and CPP is not significant from a statistical standpoint, which means one can not consistently predict the direction of change for ICPWs when CPPs are changing.

Table 14

Summary Table for ANCOVA of Intracranial Pressure Waveform to Intracranial Pressure

	Sum of Squares	df	Mean Squares	F	(p)
Main Effects					
Treatment	.87	1	.37	.03	.867
Within	33.32	3	11.11		
Regression	59.55	1	59.55	5.36	.104
Time	1.38	1	1.38	1.31	.335
Within	3.14	3	1.05		
Regression	4.01	1	4.01	3.83	.145
Interaction					
Treatment by Time	4.11	1	4.11	4.09	.136
Regression	19.31	1	19.31	19.21	.022

p = <.05

Table 15

Summary Table for ANCOVA of Intracranial Pressure Waveform to Cerebral Perfusion Pressure

Significance	Sum of Squares	df	Mean Squares	F	(p)
Main Effect					
Treatment	39.34	1	39.34	.76	.449
Within	156.17	3	52.06		
Regression	.46	1	.46	.01	.931
Time	16.00	1	16.00	.50	.531
Within	96.45	3	32.15		
Regression	.10	1	.10	.00	.531
Interaction					
Treatment by Time	9.36	1	9.36	.36	.591
Regression	3.01	1	3.01	.12	.756

CHAPTER 4

DISCUSSION

Overview

Immobility following brain injury is a common practice problem in the ICU. Nurse-initiated turning is accomplished with the goal of preventing skin breakdown and pulmonary complications. Normally turning takes place as two nurses at the bedside work together to reposition the patient. One nurse places hands on the patients' shoulders and hips pulling the patient in the direction of the turn, while the other straightens sheets and places' pillows to secure the desired position. Little attention is given to the position of the head and neck during this procedure because the focus is upon relief of pressure upon skin. More importantly, turning is a form of postural drainage, used to prevent respiratory complications. However, turning is modified or discontinued when pronounced elevations in ICP or dangerous decreases in CPP follow. Consequently, the patient could remain stationary in the supine position for 24 or more hours. The risk for pneumonia and tissue hypoxia of the skin increases during these immobilized states.

The concept of intracranial adaptive capacity (ICAC) developed by Mitchell (1986) is useful when examining this practice problem because the physiologic responses of individuals to the turning procedure vary. Indirect indicators of ICAC available in the clinical setting are intracranial pressure (ICP), cerebral perfusion pressure (CPP) and intracranial pressure waveforms (ICPW).

The purpose of the study is to explore the relationship between the physiologic indicators and experimental alignment during the turn. Five brain diseased subjects make up the sample and the three physiological measurements are the variables of interest. Active alignment is suggested as a method for avoiding undesirable elevations in ICP or drops in CPP. The passive alignment represents the routine method nurses in critical care units use to turn patients. The active or experimental method secures the alignment of the head throughout the otherwise routine turn.

Comparison of responses following both types of turn becomes available with statistics. Analysis of each case employ descriptive statistics; repeated measures ANOVA are used to analyze the group data. A two-way repeated measures ANOVA compares variances in dependent variable measurements taken before and after the turn in both the control and experimental methods. The F statistic demonstrates no statistical difference in physiologic responses between the active and passive type alignment. In addition, analysis in this study explores the relationship between the dependent variables (ANCOVA). The results of ANCOVA show that the direct relationship of ICP to ICPW approaches statistical significance while the relationship of CPP to ICPW does not.

Single Case Discussion

The next section presents single case summaries along with speculations regarding response outcomes. The single cases become an aggregate and discussion regarding the group findings is presented.

Case 1

The most remarkable dependent variable for Case 1 is CPP because large drops in CPP follow turning twice. The history is one of ruptured cerebral aneurysm, which may account for the fragile fluctuations in perfusion pressure. If the cerebral vessels are diseased the possibilities that the vascular response to stimuli will be altered is great. Vasomotor centers in the brain may be unable to decipher or react to hemodynamic changes created with the turn.

The treatment plan for this case includes several medications. One medication, Nimodipine prevents vasospasm near the diseased cerebral blood vessel. Decreases in blood pressure are just one of the side effects of Nimodipine and Case 1 demonstrates blood pressure drops after three turns. Another possible explanation for large shifts in the direction of CPP is the administration of morphine sulfate and midazolam (Versed). Both medications reduce tension upon the vascular system by relaxing skeletal muscle, resulting in a drop in blood pressure and then CPP. A concurrent intravenous Mannitol drip is present. Mannitol's action to reduce overall intracerebral volume by potent osmotic diuretic affects reduces cardiac output along with systemic blood pressure. The brain has a remarkable ability to maintain constant circulation over wide ranges in blood pressure. However, during pronounced and sustained changes in cardiac output and blood pressure the capacity is decreased or lost. Therefore, large decreases in blood pressure and secondarily CPP, particularly those below 60 mm Hg, are significant in the clinical setting.

Case 2

Case 2 has a craniotomy following an arterial-venous malformation bleed and the dependent variables show interesting trends. For example, following a control session, ICP doubled briefly. The event record notes a rectal suppository for temperature control is given at the same time as the elevation. In addition, this case has a fracture of both the tibia and fibula with a cast from the left knee to the foot. Although the fractures are treated the patient continues to have pain that might account for increased muscle tone, higher blood pressures and elevated ICP in three sessions.

Case 3

Case 3 has cerebral edema with a right-to-left shift of 6 mm rather than a space occupying mass. The physiologic events in cerebral edema may explain the unusual responses in this case. The ICP values for Case 3 display minimal fluctuation, remaining within normal limits for the first three turns. Before the last turn, ICP levels are in a high normal range and the event record indicates in-line endotracheal suctioning taking place. The most provocative finding for Case 3 is a sharp increase in CPP during neurologic assessment that includes a sternal rub and loud talking. A fractured clavicle might increase the pain or annoyance following a sternal rub. Throughout the observation sessions ICP remains normal but ICPWs show concurrent elevation along with CPP. Several ICPWs are abnormally high which is perplexing given that almost all ICP levels are 10 mm Hg or below. Is it possible that pathology specific to this Case influences P2 amplitudes earlier than ICP levels?

Case 4

The ICP in Case 4 reaches 16 mm Hg before the fourth turn but returns to normal afterward. The event record lists a visit from the subject's mother and 5 year-old daughter at the time of ICP elevation. The patient has difficulty communicating because of expressive and receptive aphasia. CPP fluctuates between 51 mm Hg and 74 mm Hg and is the lowest of any case possibly due to aggressive dehydration along with a consistent low systemic blood pressure. Also, this case is 24 years old, the youngest of the five subjects. Almost 82% of the ICPWs are abnormally high. Pathology includes an epidural hematoma along with a skull fracture.

Case 5

Case 5 displays elevated ICP values in 75% of the data collection points. This finding is consistent with a CAT scan report of 12 mm right-to-left shift, an entrapped left lateral ventricle and right medial temporal lobe herniation. ICP levels immediately following control turns either decrease or remain the same. However, the ICP rises 7 mm Hg and 5 mm Hg without returning to preturn values following both therapeutic turns. Sustained elevations may have some relationship to the large shift in brain structures. CPP levels demonstrate a drop of 10 mm Hg following a control type turn in the third session. The ICPWs display the greatest P2 amplitude of all measurements and reach a 260% elevation and may reflect the terminal status of this case. Clearly, Case 5 demonstrates a decreased intracranial adaptive capacity with indirect clinical indicators. The exact parameters as defined earlier in this study for decreased ICAC do not exist but the trends are present. For instance, ICP levels following the turn never reached sustained levels of 10 mm Hg above baseline and CPPs never dropped below 50 mm Hg. All amplitudes for P2 waves were abnormally elevated substantiating the notion that ICPWs may indicate decreased ICAC. Case 5 died four hours after the last observation confirming the presence of decreased ICAC. Perhaps, the study definition for decreased ICAC or ineffective response needs to be expanded.

Discussion of Aggregate Findings

Intracranial Pressure

The first hypothesis states that with the experimental method of head alignment a sustained ICP elevation is less likely to occur. The group differences between the mean values of ICP after the therapeutic and control turns are very small and not statistically significant. The experimental method of turning did not significantly improve ICP. Again those subjects with higher baseline values demonstrate higher elevations following the turn. Perhaps one reason the findings do not support the hypothesis is that the subjects do not fit the criteria for decreased intracranial adaptive capacity. Only two cases out of the eight have baseline ICPs of 15 mm Hg. Case 5 with the highest baseline has a brief 10 mm Hg elevation of ICP following the stimulus of the turn. ICPs almost never reach sustained levels 10 mm Hg above baseline for five minutes. One possible reason for the findings of the first hypothesis is aggressive medical intervention. Nurses caring for these subjects have medical orders to treat ICP levels above 15 mm Hg. Administration of Mannitol and neuromuscular blocking agents precedes rapid reduction of ICP as noted in event records.

Cerebral Perfusion Pressure

The second hypothesis predicts that when the head is held in midline during a turning process CPP is less likely to drop below 50 mm Hg. Since CPP never falls below 52 mm Hg this hypothesis is not supported. However, the group trends before and after the turns demonstrate a slight increase in CPP following the experimental turn while the control turn was followed by a slight decrease. The direction of the trend supports the research hypothesis; however, the magnitude of change is not statistically significant. One event of note is the high elevation in CPP following the experimental turn in Case 3. A value of 130 mm Hg is reached during neurologic testing and the influence of this major elevation upon the aggregate must be considered. Therefore, CPP after experimental turns is skewed toward the high end.

Waveform Patterns

All subjects show an increase in P2 amplitude after experimental turns and a decrease following the controls. Therefore, the third hypothesis has no support. Interestingly enough, some of the ICPW findings in this study support those of Takizawa, Gabra-Sanders and Miller (1987). They document a negative correlation with ICPW and CPP when CPP fall below 70 mm Hg. Case 1 shows abnormal elevations in P2 amplitude when CPP fell below 70 mm Hg in 90 percent of the data collection points. However, a negative correlation does not consistently appear in other subjects (50% at various ranges of CPP). Case 1 is the only subject with a diagnosis of ruptured cerebral aneurysm. Perhaps this case has elements of pathology and outcomes of treatment that create this inverse relationship.

Temporal Patterning

Another area of interest includes the temporal patterning of ICP, CPP and ICPW. Marshall, Smith, and Shaprio (1978) speculate diurnal rhythms influence ICP. ICP in 67% of brain injured subjects demonstrate episodes of elevation between 0400 and 0900 hours. Was this due to an increase in brain blood volumes during the circadian cycle? Cases in the current study fall into two categories when considering circadian cycles. One category includes Case 1 and 2 because sessions begin 8 and 9 hours after usual wakening. The second group, Cases 3, 4 and 5 is observed approximately 4 and 5 hours after usual wakening. Therefore, the second group, like those in the Marshall et al. (1978) study have larger individual brain blood volumes during their observation sessions. Observations for Cases 1 and 2 begin twice as late in the circadian cycle and would have less volume and more adaptive capacity.

Comparison of Research Findings

This study can be compared to other works that report and describe the relationships between ICP, CPP and patient positioning. One study by Lipe and Mitchell (1980) demonstrates compromised venous drainage in the left lateral positions and most often in head rotation to the right. The current study shows the absence of head rotation (Cases 1-5) in both the experimental and control method. However, elevations in ICP in this present study occur more frequently in turns to the left lateral position similar to those reported by Lipe and Mitchell(1980).

In addition, this study supports previous findings of Mitchell and Mauss (1978), Mitchell et al. (1981), and Shalit and Umansky (1977) in that ICP elevates in almost half of the total turns. The duration of higher values is not present the past studies but this study shows most elevations decrease shortly (4-5 minutes) after the turn. The longest elevation takes place for nine minutes. Aggressive medical treatment in this study might explain the adaptive responses to turning. Of interest is the fact that the ICP response in Case 1 with the fluid filled system demonstrates an increase in all turns. Are fluid filled monitors with external transducers more sensitive to early volume pressure changes when placed in the ventricle?

Increases in CPP following turning in the current study support the findings of Parsons and Wilson (1984), even though increases exist in only 60% of all turns. Parsons and Wilson (1984) demonstrate increased CPP in all turning directions. Quite possibly the subjects in the Parsons and Wilson (1984) had less sedation or pain medication than the present five cases. The painful stimulus of turning can cause increases in blood pressure as well as CPP.

This study also supports investigators who view the P2 wave in the ICPW as a promising indicator of adaptive capacity within the intracranial system. Thirty-nine out of a total of 140 measurements of ICP are 15 mm Hg or greater. P2 amplitudes elevate in 97% of events where ICP is 15 mm Hg or above. Takizawa et al., (1987) and Willis (1991) also report a direct relationship between ICPs and ICPWs when ICP levels are 15 mm Hg or above.

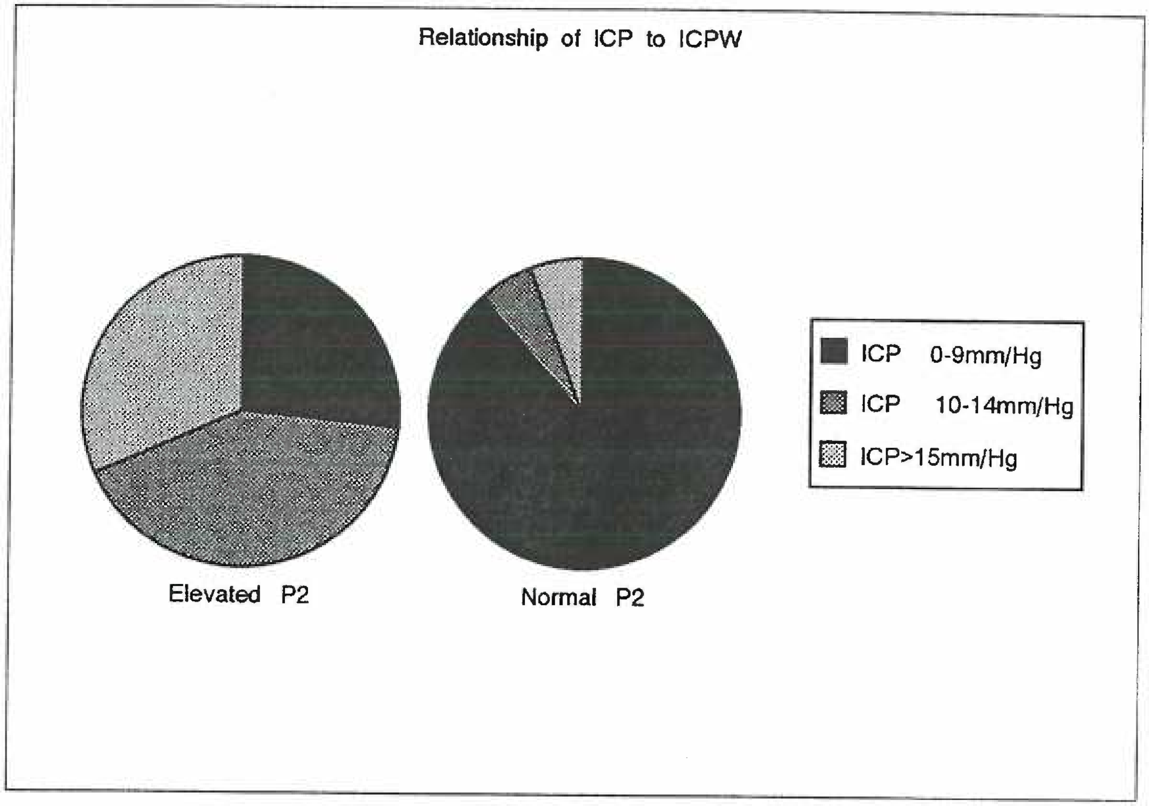


Figure 21. Three intracranial pressure ranges and presence of P2 waves 80 percent larger than P1 waves (elevated) compared to normal P2 amplitude in 140 events from five subjects. When P2 waves of normal amplitude are present ICP is often in the 0-9 mm Hg range.

Limitations, Implications and Summary

Study Limitations

Four major limitations are present. They include a small sample size, a variety of pathology, and technical limitations. The next section includes a discussion of the limitations.

Size

Perhaps size is the most significant limitation of this study. Conclusions based upon small numbers are often misleading. However, studies of this nature often have low numbers of subjects because the population from which the sample is selected are unique. For instance, subjects require specialized monitoring equipment used on an infrequent basis. In addition, potential subjects for this study were unavailable on four or five occasions due to concurrent injuries (spine) or imminent death.

Pathology

Three of the subjects have intracranial hemorrhages, one subject has an epidural hematoma and another cerebral edema. Additional subjects with subdural hematomas might have produced a more balanced reflection of data for this clinical practice problem. A possible question for future studies is: "In what ways do the physiologic responses of neurovascular disorders differ from space-occupying lesions during nurse-initiated activities?"

Technical Limitations

Several barriers exist that prevent precise collection of data. For example, the digital readouts on the personal computers at the bedside sample several ICPs and display a mean every four seconds. When the investigator observes three of these physiologic values hoping to record them simultaneously, four seconds pass. The continuous strip chart recorder validates bedside readings but precise match between the bedside reading and recorder is not always possible. The hour and minute are consistent; however, identical timing of seconds is more difficult.

Further limitation occurs with interpretation of ICPWs. For the most part, P2 evaluation is accomplished by researchers in the laboratory setting rather than the clinical environment. Clinicians have challenges with unpredictable events in the ICU that make reading P2 amplitudes difficult. For example, some waveforms show aberrant flattening due to ordinary movements on the part of patient or staff. The science of ICPW analysis is just in the early stages. Yet another technical limitation occurs when subjects need procedures such as suctioning or other treatments during the observation period. Many procedures can change pressure readings and the investigator is unable to control all stimuli that confront the subjects.

Clinical Implications

The investigator believes two pieces of clinical significance are present in the findings. First, the standard error calculations show more variability in measurements after both control and therapeutic turns. This variablity supports clinicians who report increased fluctuation in ICP and CPP following the stimuli of turning. Most clinical observations of patient response will occur shortly after the turn, perhaps even within one minute of the turn. Physiologic indicators the nurse observes in this early period often become the basis of clinical decisions for future turns. However, sustained responses, lasting 10 or 15 minutes beyond the stimulus are suggested as the best reflection of adaptive capacity.

Secondly, the practical implications from this study indicate current methods of turning are harmless when ICPs are less than 15 mm Hg. Although there are brief elevations in pressures a return to baseline appears to be the pattern. Cases 2, 3 and 4 in this study did not fit into the proposed definition of decreased intracranial adaptive capacity (ICAC) and tolerated turning satisfactorily. In Cases 1 and 5 elevated baseline ICP values remain high after turning as did similar cases in other reports (Magnaes, 1976; Shalit and Umansky, 1977). Patients presenting with higher baseline ICPs might benefit from therapeutic devices such as rotating beds to assist with mobilization while in the ICU. Also, nurses might consider a "test" type of turn to explore the patient's response to turning, looking for sustained elevations in ICP rather than brief rises. The primary nurse might consider accomplishing other tasks in the room for 15 minutes while the pressures return to baseline.

Research Implications

Future investigations might include sample selection across hospital settings and case separation by pathology. A possible question one could ask is "What type of relationship exists between intracranial responses and the type of pathology present?" One study might observe a control group with ICP lower than 15 mm Hg and compare similar treatments to subjects with ICP over 15 mm Hg. Other investigators study the magnitude of ICP and CPP responses but a new approach to duration of change is important. Further investigation of the ICPW relationship to other clinical indicators promises to add information which may explain human responses to brain injury.

Summary

In summary, this experimental study investigates active alignment of the head and neck during patient turning as an improvement over the standard method. Responses that follow both types of alignment are compared. The physiologic responses or measurements of interest include: (1) ICP values that are sustained 10 mm Hg above baseline values for five minutes following the stimuli of turning, (2) CPP levels that drop below 50 mm Hg following the turn and, (3) ICPW where the P2 amplitudes exceed P1 by 80%. A state of decreased intracranial adaptive capacity (ICAC) is reflected when the three variables demonstrate the above responses.

In all five subjects, ICP never elevates to 10 mm Hg above baseline following any of the turns. The CPPs throughout the study stay above 50 mm Hg. Therefore, if ICP and CPP are indirect indicators of ICAC most events demonstrate adequate adaptive capacity. On the other hand ICPWs frequently show ineffective responses by abnormally high P2 amplitudes. The study contains 140 measurements of ICP and 39 are above 15 mm Hg. In 97% of events where ICP is above 15 mm Hg, P2s are abnormally high. The waveform demonstrates a possible state of decreased ICAC at some point for each subject. Finally, this study suggests when normal ICP and CPP values are present the standard method for turning patients may be harmless.

References

Barlow, D. H., & Hersen, M. (1973). Single-case experimental designs: Uses in applied clinical research. Archives of General Psychiatry, 29, 319-325.

Bedford, R. F. (1985). Invasive blood pressure monitoring. In C.D. Blitt (Ed.), Monitoring in anesthesia and critical care medicine (pp.41-85). New York: Churchill Livingstone.

Boortz-Marx, R. (1985). Factors affecting intracranial pressure: A descriptive study. Journal of Neurosurgical Nursing, 17(2), 89-94.

Bornstein, R. A., & Brown, G. G. (1991). Neurobehavioral aspects of cerebral vascular disease New York: Oxford University Press.

Bruce, D. A. (1978). The pathophysiology of increased intracranial pressure Philadelphia: Upjohn.

Cordoza, E. R., Rowan, J. O., & Galbraith, S. (1983). Analysis of the cerebrospinal fluid pulse wave in intracranial pressure. Journal of Neurosurgery, 59, 817-821.

Crutchfield, J. S., Narayan, R. K., Robertson, C.S., & Michael, L.H. (1990). Evaluation of a fiberoptic intracranial pressure monitor. Journal of Neurosurgery, 72, 482-487.

Durward, Q. J., Amacher, A. L., DelMaestro, R. F., & Sibbald, W. J. (1983). Cerebral and cardiovascular responses to changes in head elevation in patients with intracranial hypertension. Journal of Neurosurgery, 59, 938-944.

Gennarelli, T. A., Spielman G. M., Langfitt, T. W., Gildenberg, P. L., Harrington, T., Jane, J. A., Marshall, L. F., Miller, J. D., & Pitts, L. H. (1882). Influence of the type of intracranial lesion on outcome from severe head injury. Journal of Neurosurgery, 56, 26-32.

Gray, W. J., & Rosner, M. J. (1987). Pressure-volume index as a function of cerebral perfusion pressure, Part 1. Journal of Neurosurgery, 67, 369-376.

Fleisher, G.R., & Ludwig, St. (1988). Textbook of pediatric emergency medicine. Baltimore: Williams & Wilkins..

Fontaine, D. K. (1989). Physical, personal, and cognitive responses to trauma. Critical Care Nursing Clinics of North America, 1(1), 11-22.

Fontaine, D. K., & McQuillan, K. (1989). Positioning as a nursing therapy in trauma care. Critical Care Nursing Clinics of North American, 1(1) 105-112.

Hobdell, E. R., Adamo, F., Carusa, J.,Dihoff, R., Neveling, E., & Roncoli, M. (1989). The efffect of nursing activities on the intracranial pressure of children, Critical Care Nursing 9(6), 75-79.

Holm, K. (1983). Single subject research. Nursing Research, 32(4), 253-255.

Hollingsworth-Fridlund, P., Vos, H. & Daily, E. K. (1988). Use of fiber-optic pressure transducer for intracranial pressure measurements: A prliminary report. Heart & Lung, 17(2), 111-118.

Hotter, A. N., (1989). The pathophysiology of shock brain. Critical Care Nursing Clinics of North America 1(1), 123-130.

Huck, S., Cormier, W., & Bounds, Jr., W. (1974). Reading statistics and research New York: Harper & Row

Jeanette, B. & and Teasdale, G. (1981). Management of head injuries. Philadelphia: F. A. Davis Company.

Johnson, S. M., Omery, A., & Nikas, D. (1989). Effects of conversation on intracranial pressure in comatose patients. Heart & Lung, 18(1), 56-63.

Kazdin, A. E. (1982). Single-case research designs: Methods for clinical and applied settings New York: Oxford University Press.

Lipe, P. H. & Mitchell, P. H. (1980). Positioning the patient with intracranial hypertension: How turning and head rotation affect the internal jugular vein. Heart & Lung, 9(6), 1031-1037.

Lundberg, N. (1960). Continuous recording and control of ventricular fluid pressure in neurosurgical practice. Acta Psychiatrica Et Neurologica Scandinavica 36(Suppl 149), 1-193.

Lundgren, J. P. (1990). Computerized EEG: Applications and interventions. Journal of Neuroscience Nursing 22, 108-112.

Magnaes, B. (1976). Body position and cerebrospinal fluid pressure Part 1: Clinical studies on the effect of rapid postural changes. Journal of Neurosurgery, 44, 687-697.

March, K., Mitchell, P. H., Grady, S. & Winn, R. (1990). Effect of backrest position on intracranial pressure and cerebral perfusion pressure. Journal of Neuroscience Nursing, 22, 375-381.

Marshall, L. F., Smith, R. W., & Shapiro, H. M. (1979). The outcome with aggressive treatment in severe head injuries. II. Acute and chronic barbiturate administration in the management of head injury. Journal of Neurosurgery, 50 26-30.

Marshall, L. F., Smith, R. W., & Shapiro, H. M. (1978). The influences of diurnal rhythms in patients with intracranial hypertension: Implications for management. Neurosurgery, 2(2),100-102.

Martins, A. N., Wiley, J. K., & Meyers, P. W. (1972). Dynamics of the cerebrospinal fluid and the spinal dura mater. Journal of Neurology, Neurosurgery, and Psychiatry, 35, 468-473.

Mendelow, A. D., Rowan, J. O., Murray, L. & Kerr, A. E. (1983). A clinical comparison of subdural screw pressure measurements with ventricular pressure. Journal of Neurosurgery, 58, 45-50.

McGillicuddy, J. E. (1985). Cerebral protection: Pathophysiology and treatment of increased intracranial pressure. Chest, 87(1), 85-93.

Millar, S., Sampson, L. & Soukup, M. (1980). American association of critical care nurses procedure mannual for critical care. Philadelphia: W.B. Saunders.

Miller, J. D. (1985). Head injury and brain ischaemia; Implication for therapy. British Journal of Anaesthesia 57(1), 120-129.

Mitchell, P. H., (1986 August (a)). Decreased adaptive capacity, intracranial: A proposal for a nursing diagnosis. Journal of Neuroscience Nursing 18, 170-175.

Mitchell, P. H., (1986 December(b)). Intracranial hypertension: Influence of nursing care activities. Nursing Clinics of North America 21, 563-576.

Mitchell, P. H., Amos, D., & Astley, C. (1986). Nursing and ICP: Studies of two clinical problems. Study 1: Effects of passive turning and lateral neck flexion. In J. D. Miller. (Eds.). Intracranial Pressure VI (pp 701-704)

Mitchell, P. H., Ozuna, J., & Lipe, H. P. (1981). Moving the patient in bed: Effects on intracranial pressure. Nursing Research, 30(4), 212-218.

Mitchell, P. H., & Mauss, N. K. (1978). Relationship of patient-nurse activity to intracranial pressure variations: A pilot study. Nursing Research, 27(1),4-10.

Moore, D. S., & McCabe, G. P. (1989). Introduction to practice of satistics. New York: Will Freeman and Co.

Nara, A. R., Burns, M. P., & Downs, W. G. (1989). Biophysical measurement series: Blood pressure Redmond, WA: SpaceLabs.

Narayan, R., Bray, R., Robertson, C. S. (1987, May). Experience with a new fiberoptic device for intracranial pressure monitoring. Presented at the Annual Meeting of American Association of Neurological Surgeons, Dallas, Texas.

Nikas, D. L. (1987). Prognostic indicators in patients with severe head injury. Critical Care Nursing Quarterly 10(3), 25-34.

Ostrup, R. C., Luerssen, T. G., Marshall, L. F., & Zornow, M. H. (1987). Continuous monitoring of intracranial pressure with a miniaturized fiberoptic device. Journal of Neurosurgery, 67, 206-209.

Parsons, C. L., & Wilson, M. (1984). Cerebrovascular status of severe closed head injured patients following passive position changes. Nursing Research, 33(2), 68-75.

Parsons, C. L., & Shogan, J. S. (1984). The effects of the endotracheal tube suctioning/manual hyperventilation procedures on patients with severe closed head injuries. Heart & Lung, 13(4), 372-380.

Polit, D. F., & Hungler, B. P. (1987). Nursing research principles and methods (3rd ed.). Philadelphia: Lippincott.

Portnoy, H. D., Chopp, M., Branch, M. S., & Shannon, M. B. (1982). Cerebrospinal fluid pulse waveform as an indicator of cerebral autoregulation. Journal of Neurosurgery, 56, 666-678.

Rea, G. L., & Rockswold, G. L. (1983). Barbiturate therapy in uncontrolled intracranial hypertension. Neurosurgery, 12 (4), 401-404.

Rockoff, M. A., Marshall L. F., & Shapiro, H. M. (1979). Barbiturate therapy in uncontrolled intracranial hypertension in man: A clinical review of sixty patients. Annals of Neurology, 6, 194-199.

Rubin, S. A. (1987). The principles of biomedical instrumentation: A beginner's guide Chicago: Year Book Medical Publishers, Inc.

Shalit, M., & Umansky, R. (1977). Effect of routine bedside procedures on intracranial pressure. Israel Journal of Medical Science, 13(9), 881-886.

Shellock, F. G. (1985). Transducer-tipped catheter. Medical Electronics, 16(6), 102-106.

Snedecor, G. W. (1956). Statistical methods. Ames: Iowa State College Press.

Snyder, M. (1983). Relation of nursing activities to increases in intracranial pressure. Journal of Advanced Nursing, 8, 273-279.

SpaceLabs, Inc. (1989). Operations Manual(i - 2-9). Redmond, WA: Space Labs.

Takizawa, H., Gabra-Sanders, T, & Miller, J. D. (1987). Changes in the cerebrospinal fluid pulse wave spectrum associated with raised intracranial pressure. Neurosurgery, 20(3), 355-361.

Tsementzis, S. A., Harris, P. and Loizou, L. A. (1982). The effect of routine nursing care procedures on the ICP in severe head injuries. Acta Neurochirurgica, 65, 153-166.

Willis, M. L. (1991). Intrepretation of intracranial pressure waveform: The predictive value of P2 elevation in the diagnosis of decreased intracranial adaptive capacity. Unpublished masters thesis, University of Washington, Seattle, WA.

Appendix A

Protocols for Data Collection, Turning
and Teaching Assistants

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

DATA COLLECTION PROTOCOL

Immediately before observation period begins:

1. Three RN's must be present in the unit that have been previously trained in the therapeutic turn.
2. The printer for ICP values and waveforms must be functioning.
3. Two extra rolls of printer paper must be present in the subject's room.
4. Zero the Camino 420 Model toward the patient monitor.
5. Zero the arterial line toward the patient monitor.
6. Calculate the position of the foramen of Monro. Place arterial line transducer upon this point.
7. Measure Head of Bed Elevation with a protractor.

AS THEY OCCUR

Record in-room events on the event record.

Five minutes prior to the turn

Record Arterial line Pressure from digital read out.
Record ICP from digital read out.
Subtract mean ICP from mean ABP using digital read out numbers.
Print ICP waveform.

Three minutes prior to the turn

Record Arterial line Pressure from digital read out.
Record ICP from digital read out.
Subtract mean ICP from mean ABP using digital read out numbers.
Print ICP waveform.

One minute prior to the turn

Record Arterial line Pressure from digital read out.
Record ICP from digital read out.
Subtract mean ICP from mean ABP using digital read out numbers.
Print ICP waveform.

Two or Three RN's will enter the room. The turn will begin with verbal instruction by the nurse and will terminate by the statement, "The turn is completed."

<u>One minute following the turn</u>

Record Arterial line Pressure from digital read out.
Record ICP from digital read out.
Subtract mean ICP from mean ABP using digital read out numbers.
Print ICP waveform.

<u>Five minutes following the turn</u>

Record Arterial line Pressure from digital read out.
Record ICP from digital read out.
Subtract mean ICP from mean ABP using digital read out numbers.
Print ICP waveform.

<u>Ten minutes following the turn</u>

Record Arterial line Pressure from digital read out.
Record ICP from digital read out.
Subtract mean ICP from mean ABP using digital read out numbers.
Print ICP waveform.

<u>Fifteen minutes following the turn</u>

Record Arterial line Pressure from digital read out.
Record ICP from digital read out.
Subtract mean ICP from mean ABP using digital read out numbers.
Print ICP waveform.

<u>Immediately after observation period</u>

Collect data sheets.
Collect ICP waveform paper.

MANUAL HEAD AND NECK ALIGNMENT
DURING PATIENT TURNING
IN STATES OF DECREASED
INTRACRANIAL ADAPTIVE CAPACITY

Control Turn (Standard Method) Protocol

Goal:
1. Simulate current practice standard of turning critically ill.
2. Investigator touching head and face identical to that of the therapeutic turn.
3. Head of Bed elevation to remain at 30 degrees throughout turn. Measurement by protractor prior to each turn.

From supine to lateral

1. RN #1 (the Investigator) tells the patient "We are going to turn you now."
2. RN #2 removes sheets or bed covers.
3. RN #1 places hands on each side of the head with thumbs on mandibles and fingers behind head on the occipital ridge. Hands remain passively positioned on the head throughout the turn.
4. RN #3 locates two pillows.
5. RN #2 places hands on one shoulder and one hip of patient, pulls the patient slowly toward her.
6. RN #3 places one pillow underneath back.
7. RN #3 slightly flexes knees with minimal hip flexion and places the second pillow between the patients knees.
8. RN #2 covers the patient.
9. No alignment is given to the head or neck. Passive placement of hands about the mandible and occipital ridge will take place.

From lateral to supine

1. RN #1 (the investigator) tells patient "We are going to turn you now."
2. RN #1 places hands on each side of the head with thumbs on mandibles and fingers behind head on the occipital ridge. Hands remain passively positioned on the head throughout the turn.
3. RN # 2 Removes top bed sheet, places hands on one shoulder and one hip of patient.
4. RN # 3 Removes pillows from behind back and between knees.
5. RN #2 Slowly allows the patients body to assume the supine position.
6. RN #1 Removes hands from patient head.
7. RN #2 Covers patient.

THERAPEUTIC TURN PROTOCOL.

Goal: 1. Head must be immobilized to prevent flexion-extension, rotation, lateral movement.
2. Head of Bed to remain at 30 degrees throughout turn.

Protocol for **supine to lateral turn.**

1. "Head - holder" (RN #1, the investigator) takes position at head of the patient.
2. Head holder informs the patient "the three of us are going to turn you." I will hold your head during the turn.
3. Head holder places hands on each side of the head with thumbs on mandibles and fingers behind head on the occipital ridge.
4. Hands remain on the head throughout the turn and until the head position is stabilized with bath blankets or small pillows.
5. RN #2 removes sheets or bed covers.
6. RN #2 places hands on one shoulder and one hip of the patient and pulls the patient slowly toward RN.
7. RN #3 has located 2 pillows and places one behind the back.
8. RN #3 slightly flexes knees and minimally flexes hip and places the second pillow between the patients knees.
9. RN #2 covers the patient.
10. RN #1 Assures alignment of head and neck in neutral position.

Protocol for **lateral to supine** turn.

1. "Head - holder" (RN #1,the investigator) takes position at head of the patient.
2. Head holder informs the patient "the three of us are going to turn you." I will hold your head during the turn.
3. Head holder places hands on each side of the head with thumbs on mandibles and fingers behind head on the occipital ridge.
4. Hands remain on the head throughout the turn and until the head position is stabilized with bath blankets or small pillows.
5. RN #2 removes sheets or bed covers.
6. RN #2 places hands on one shoulder and one hip of the patient.
7. RN #3 removes pillows from behind the back and between knees.
8. RN #2 slowly allows patients body to assume the supine position
9. RN #2 covers the patient.
10. RN #1 Assures alignment of head and neck and removes hands from patients head.

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Teaching Protocol for Nurse-initiated turning

Six Assistants will be trained to perform the control and therapeutic turn. Two practice sessions will precede each observation period. These session will take place on a manikin. The turn will be practiced until all three assistants can perform the two turns efficiently with minimal touching of the manikin and talking to one another. In all turns, control and therapeutic the investigator will hold the head of the subject.

Protocol for Camino Monitoring Devices

1. Document that the Camino transducer was zeroed prior to insertion.
   a. Adapt transducer-tipped catheter to Camino patient cable
   b. Note hyphen (- - -) pattern on Camino Digital Pressure module.
   c. Note digital reading of "0" evident on module screen.
   d. If not at "0", use tool from catheter kit to adjust part on posterior portion at the catheter connection to the patient cable.

THIS STEP SHOULD NOT BE REPEATED. The <u>transducer</u> is zeroed only once.

2. The Camino Digital Pressure monitor (model 420) will be <u>calibrated</u> to the bedside monitor prior to each observation session and at which time a break in the system may have occurred.

3. Document Attachment the Camino patient monitor cable to the bedside monitor.
(Space Lab PC's)

<u>Calibration from 420 Model to Patient Monitoring system.</u>

4. On the front of the Camino Digital Pressure Monitor press the asterisk labeled panel also called the cal step switch until read out is zero.

5. While maintaining the pressure on the cal step switch select ICP pressure by touching vertical rectangle on right side of screen labeled ICP. Touch the word zero which appears on the bottom of the screen. Wait for monitor to display "zeroing completed."

6. Both monitors are now calibrated.

Protocol for Measuring the P2 Amplitude Compared to P1

1. Waveform analogs from the Space Lab Strip chart record will be obtained for each observation point. (7 per observation session = 28 total for each subject)

2. Each waveform on the record stip will be analyzied for P2 amplitude in millimeters compared to amplitude of P1.

3. P2 wave is defined as the second upward inflection of the ICPW.

4. P2 amplitude is measured as the height from lowest defection (baseline) to the second upward infection. P1 amplitude is measured as the first upward infection distance from baseline. An engineers measuring device with millimeter intervals will be used to decipher amplitudes. (EKG calipers slip and/or leave the baseline whereas the ruler maintains the baseline moving to the next inflection while only the P1 or P2 heights change).

5. Strip chart recordings display an average of 10 waveforms per data point. All waveforms will be analyzed when possible. Obscuring of baseline or P2 amplitude may occur which means fewer waveforms will be analyzed for that data point.

6. Once P2 and P1 have been measured for each waveform a relationship in percentage will be calcualted. For example, when P2 is 8 millimeters in height and P1 is 10 millimeters in height, a value of **.80** will be given to that P2.

7. All waveforms present on the recording stip will be analyzed for P2 amplitude compared to P1 and then an average (Mean) value will be recorded for the appropriate data collection point. This means that each subject will have seven P2 values in each observation session or a total of 28 for four sessions.

Appendix B

Records of Background Variables
and Events

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Background Variable Record

Subject Identification number _______________

Hospital ID number ______________________Room Number ___

Date____________Time_____________

RN's assisting __________________________________

Date of Birth ______________________ Age________

Race ___________ Occupation _______________________

County of residence ________________________________

Hospital admission date and time ______________________

ICU admission date and time ___________________________

Glasgow Coma Score on admission ___________

Concurrent injuries___________________________________

CAT SCAN results ____________________date____________

____________________date ____________

____________________date____________

Previous medical history _______________________________

____

Current medical diagnoses _______________________________

____

Surgery date _____ time ______ type __________

duration _____________

medication administered ________________

Medications concurrent to study

Type Dose Route Time

____

____

____

____

____

____

____

Neurological Assessment

Date ________________

Time ________________LOC_________________Pupils__________

Motor/sensory ____

Babinski present ___Babinski absent __

Extraocular movements _______________________________________

Corneals ________________Lids ___________Ciliospinals ________

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject ____________ Date ______________ Calibration of Lines ___

A-Line transducer at foramen of Monro __________ HOB elevation _____

Clocks syncronized _____ Observation Number ___ Sequence Number _____

Turning Table Number _____ Alignment _____, _____, ______, _______

Time	BP	ICP	CPP	Waveform	Patient Position
5 min					
3 min					
1 min					
TURN					
1 min					
5 min					
10 min					
15 min					

MANUALHEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

EVENT RECORD

Time	Who	What	Whom	Purpose	Comment

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTATION

Code List for Events

Who

0 Nurse (RN)
1 Physician (MD)
2 Respiratory therapist (RT)
3 Other health professional (HP)
4 Significant other (SIG)
5 Other family member (FAM)
6 Friend or spiritual leader (FND)
7 Patient (subject)(PT)
8 Other patient (OP)

Whom

0 telephone (PH)

1 nurse (RN)
2 physician (MD)
3 respiratory therapist (RT)

4 other health professional (HP)
5 significant other (SIG)
6 other familymember (FAM)
7 friend or spiritual leader (FND)
8 patient (subject) (PT)
9 equipment or not applicable (NA)

What

0 hum, sing, whistle(hum)
1 talk (tlk)
2 touch (tch)
3 laugh (Lgh)
4 cry or moan (cry)
5 scream (scr)
6 cough or clear throat (cgh)
7 sneeze or blow nose (snz)
8 code/arrest (cde)
9 observe (obs)

Purpose

0 procedure (pro) i.e. endotracheal suctioning
1 comfort (cft)
2 assessment (ass)
3 give or seek information(inf)
4 turn(trn)
5 reposition (repo)
6 transfer (mve)
7 not applicable (na)

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Appendix C

Concent Forms

THE OREGON HEALTH SCIENCES UNIVERSITY
Consent Form for Subject

MANUAL HEAD AND NECK ALIGNMENT
DURING PATIENT TURNING
IN STATES OF
DECREASED INTRACRANIAL ADAPTIVE CAPACITY

PRINCIPAL INVESTIGATOR

Ann Alway, RN, BSN, CCRN, CNRN
(503) 370-5200, Beeper #0577

PURPOSE

I am being asked to participate in a study titled, "Manual Head and Neck Alignment During Patient Turning in States of Decreased Intracranial Adaptive Capacity" that is being conducted by Ann Alway, R.N., BSN, CCRN, CNRN. The purpose of this study is to compare the effects of two turning procedures on intracranial pressure.

PROCEDURES

Responses following two different types of head and neck alignment will be compared. Active securing of my head and neck into a neutral position will be one type of alignment. Alignment will be accomplished by hand-holding of my jaw and the back of my head. The second type of alignment will include touching of my jaw and the back of my head without purposeful alignment. The passive alignment of the head and neck will most closely reflect the routine nursing practice turn. The approximate duration of the subject's participation in this research study is eight hours.

To perform this study, Mrs. Alway will observe me one-half hour for four times approximately four hours after my normal morning wakening time. She will be recording pressures and circulation levels from monitoring devices that are already in place. These recordings will take place both before and after I am turned to a new position. Three nurses from Salem Hospital staff will assist her with the turning procedure. Additional information from my chart will be reviewed by Mrs. Alway. This information may include X-Ray reports, laboratory reports and previous medical history.

RISKS AND DISCOMFORTS

A potential risk to participation in this study is that the intermittent presence of a researcher in my room may be annoying to me or to family members who visit. Every effort to prevent any injury that could result during this study will be taken.

BENEFITS

A potential benefit of my participation in this study is that the researcher will make the information about my condition available to the nursing staff. However, I realize that I may not personally benefit from participating in this study. Mrs. Alway believes that this study will provide information that will help nurses give better care to intensive care unit patients. She believes that this study will help nurses understand more about the pressures and circulation within the brain.

ALTERNATIVE PROCEDURES

If I choose not to participate in this study I will be turned in the standard manner.

CONFIDENTIALITY

Neither my name nor my identity will be used for publication or publicity purposes. My records will be identified by a code number rather than by name.

COSTS

No research costs will be charged to me.

LIABILITY

The Oregon Heath Sciences University, as as agency of the State, is covered by the State Liability Fund. If I suffer any injury from the research project, compensations would be available to me only if I establish that the injury occurred through the fault of the University, its officers or employees. If I have further questions, I can call Dr. Michael Baird at (503) 494-8014. In the event of physical injuries resulting from this study, medical care and treatment will be available at Salem Hospital. I have not waived any legal rights or released the hospital or its agents from liability for negligence by signing this form.

OTHER

Mrs. Alway has offered to answer any questions that I might have about the study. If I have additional questions during the course of this study, or if any problems arise, I may reach Mrs. Alway at (503) 370-5200 (office). Her address is Salem Hospital, 665 Winter Street S.E., P. O. Box 14001, Salem, OR., 97309-5014

My participation in this research study is voluntary, and I may withdraw from this study at any time without affecting my relationship with or treatment at Salem Hospital. I have read and understand this consent form, and I am willing to participate in this study. After signing this consent form I will receive a copy of it.

Subject's Signature   Witness Signature   Date

THE OREGON HEALTH SCIENCES UNIVERSITY
Consent Form For Family

TITLE:

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

PRINCIPAL INVESTIGATOR

Ann Alway, RN, BSN, CCRN, CNRN
(503) 370-5200, Beeper #0577

PURPOSE

I am being asked to give consent for ________________ (subject's name) to participate in a study titled, "Manual Head and Neck Alignment During Patient Turning in States of Decreased Intracranial Adaptive Capacity" that is being conducted by Ann Alway, R.N., BSN, CCRN, CNRN. The purpose of this study is to compare the effects of two turning procedures on intracranial pressure.

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A potential risk to participation in this study is that the intermittent presence of a researcher in the room may be annoying to the subject or to family members who visit. Every effort to prevent any injury that could result during this study will be taken.

BENEFITS

A potential benefit of the subject's participation in this study is that the researcher will make the information about their condition available to the nursing staff. However, I realize that the subject may not personally benefit from participating in this study. Mrs. Alway believes that this study will provide information that will help nurses give better care to intensive care unit patients. She believes that this study will help nurses understand more about the pressures and circulation within the brain.

ALTERNATIVE PROCEDURES

If I choose not to have my family member participate in this study, they will be turned in the standard manner.

CONFIDENTIALITY

Neither the subject's name nor identity will be used for publication or publicity purposes. Their records will be identified by a code number rather than by name.

COSTS

No research costs will be charged to the subject.

LIABILITY

The Oregon Heath Sciences University, as as agency of the State, is covered by the State Liability Fund. If the subject suffers any injury from the research project, compensations would be available to them only if I establish that the injury occurred through the fault of the University, its officers or employees. If I have further questions, I can call Dr. Michael Baird at (503) 494-8014. In the event of physical injuries resulting from this study, medical care and treatment will be available at Salem Hospital. I have not waived any legal rights or released the hospital or its agents from liability for negligence by signing this form.

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Mrs. Alway has offered to answer any questions that I might have about the study. If I have additional questions during the course of this study, or if any problems arise, I may reach Mrs. Alway at (503) 370-5200 (office). Her address is Salem Hospital, 665 Winter Street S.E., P. O. Box 14001, Salem, OR., 97309-5014

Participation in this research study is voluntary, and I may withdraw from this study at any time without affecting the subject's relationship with or treatment at Salem Hospital.

I have read and understand this consent form, and I am willing to have the subject participate in this study. After signing this consent form I will receive a copy of it.

Subject's Representative's Signature Date

Relationship to Subject

Witness's Signature Date

Appendix D

Case Files

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Background Variable Record

Subject Identification number ____A___________

Glasgow Coma Score on admission ____4____

Concurrent injuries_____fractured_C-spine_______________

CAT SCAN results ____C1-C2 Separation___date____4-8________

____Rt-Left Shift 2mm___date ___4-8_______

______________________date____________

Previous medical history ________________________________

____

Current medical diagnoses _______Closed Head Injury_______

_____Ruptured Spleen_________________________________

Surgery date _4-8____ time ______ type ___Ex-lap______

Medications concurrent to study

Type	Dose	Route	Time

1. Ancef 1 gram IV Q 8hrs 0300/1100/1900
2. Mannitol 12,5 grams IV Q 8hrs 0500/1300/1800
3. Dilantin 100mg IV Q 6hrs 2400/0600/1200/1800
4. Vit. C 1 gram IV Q 8 hrs 0400/1200/2000/
5. Tagement 300mg IV Q6 0400/1200/2000
6. Versed 2 mg. IV prn
7. Codiene 30-60mg IM Q3 prn

Neurological Assessment

Date ___4-8-91_______

Time _1000______LOC_No speech___Pupils__reactive by dyscongugate___

Motor/sensory _semi purposeful withdral___less on rt leg________________

Babinski present _xx__Babinski absent __

Extraocular movements __some -limited_______________________

Corneals _____x_________Lids present____Ciliospinals __present____

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject ______A_______Date _4-8-91________Calibration of Lines _x__

A-Line transducer at foramen of Monro ___x________HOB elevation ___30__

Clocks syncronized __x__Observation Number _1_ Sequence Number __0___

Turning Table Number ___0__Alignment ___-__, ___-__, ___-__,___-___

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1414	148/ 68	7	85		SUPINE
1418	148/ 70	9	84		
1420	144/ 69	10	85		
TURN					RIGHT LAT.
1424	133/ 63	10	77		
1428	138/ 67	11	79		
1433	136/ 62	9	83		
1438	132/ 62	10	77		

EVENT RECORD, CASE A, OBSERVATION 1

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1413	MO	TALK	RT	INFO	
1416	RT	TALK	RT	INFO	
	RT	TOUCH	EQUIPMENT	EMPTY MOISTURE	FROM VENT TUBES
	RN	TALK	RN	INFO	
	MO	TCH/TLK	PT	INFO	
	RN	TALK	RN	INFO	
1418	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
	RN	TALK	PT	INFO	
	RN	TALK	RN	INFO	
1419	RN	TALK	RN TECH	INFO	
	RN	TALK	RN	INFO	
	TECH	TALK	RN	INFO	HOB 15 DEGREES
1420	RN	TLK/TCH	PT	TURN TOWARD	RIGHT SIDE
	RN	TALK	TECH	INFO	
	RN	TALK	RN	INFO	
1427	RN	TOUCH	PT	PUPIL ASSESS	
	RN	TOUCH	PT	SUCTION ORALLY	
	RN	TALK	RN	INFO	
1430	RN	TALK/TCH	PT	IV VERSED 2 MG	
	RN	TALK	RN	INFOR	
1438	RN	TLK	RN	INFO SINCE 1434	

D-3

Dependent Variable Record

Case A -OBSERVATION 2

4-8-91

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1710	123/ 61	10	70		RT LAT
1710	136/ 63	12	72		
1718	129/ 60	9	76		
TURN					Lt. Lateral
1721	127/ 62	17	69		
1525	139/ 60	10	73		
1730	141/ 62	12	73		
1735	150/ 63	13	77		

D-4

EVENT RECORD, CASE A, OBSERVATION 2

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1711	CHAP	TALK	MOM	INFO	
	MOM	TALK	CHAP	INFO	
	RN	TALK	RN	INFO	
	RN	TLK/TCH	MOM	INFO/COMFORT	
	RN	TALK	PT	INFO	
1717	RN	TALK	RN	INFOR	
	RN	TOUCH	BED	PROCEDURE	
	RN	TALK	RN	INFO	
	RN	TLK/TCH	PT	TURING	FROM SUPINE TO LEFT
1720	RN	TALK	RN	TURN COMPLETE	
1733	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	

DEPENDENT VARIABLE RECORD

Case A

Observation 3

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1933	142/ 57	15	72		Left Lateral
1935	149/ 65	16	70		
1937	144/ 58	14	72		
TURN					SUPINE
1939	133/ 57	16	64		
1944	134/ 54	11	69		
1949	125/ 52	12	63		
1954	137/ 53	10	64		

EVENT RECORD

CASE A

OBSERVATION 3

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1933	RN	TALK	PT	INFO	
	RN	TALK	MOM	INFO	
1938	RN	TALK	PT	INFO	
	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
1939	RN	TLK/TCH	PT	TURN	TO SUPINE
	RN	TALK	RN	INFO	
1940	MOM	TLK/TCH	PT	COMFORT	
	RN	TALK	SO	INFOR	
1942	RN	CHANGING	IVS	PUMPS ALARMING	
1946	MOM	TALK	SO	INFO	
	SO	TALK	RN	INFO	
	RN	TALK	SO	INFO	
	RN	TALK	RT	INFO	
	RT	TALK	RN	INFO	VENTLATOR INFO
1950	RN	TALK	RT	INFO	
	RT	TALK	RN	INFO	
	RN	TALK	RN	INFO	
1954	RN	TALK	SO	INFO	
	SO	TALK	RN	INFO	

DEPENDENT VARIABLE RECORD

CASE A

OBSERVATION 4

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
2125	138/ 58	14	67		SUPINE
2127	136/ 56	15	67		
2130	138/ 57	14	67		
TURN					RT LATERAL
2134	132/ 60	17	65		
2139	143/ 59	13	72		
2144	139/ 57	10	74		
2149	146/ 65	10	77		

EVENT RECORD

CASE A

OBSERVATION 4

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
2125	RN	TALK	SO	INFO	
2129	RN	TALK	SO	INFO	
2130	MOM	TALK	PT	INFO	
	RN	TALK	MOM	INFO	
	RN	TALK	RN	INFO	
2131	RN	TLK/TCH	PT	TURN	
2132	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
2133					TURN COMPLETE
2134	RT	TCH	EQUIP	INFO	
	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
2137	RN	TALK	RN	INFO	
2139	RN	TALK	RN	INFO	
2146	RN	TALK	RN	INFO	
	RT	TOUCH	EQUIP	CHANGE VENT	TUBING
	RN	TALK	RT	INFO	

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Background Variable Record

Subject Identification number _____CASE B________

Glasgow Coma Score on admission __13/14_________

Concurrent injuries_________________________________

CAT SCAN result -LRG L. INTRACEREBRAL HEMATOMA_date_5-24-91___

______________________date _____________

______________________date_____________

Previous medical history _HYPERTENSION__ALCOHOLISM_

____

Current medical diagnoses __CVA - HEMORRHAGIC________

Surgery date 5-26__ time _1800____ type _LEFT PARIETAL CRANI-

PLACEMENT OF ICP BOLT--BOLT REPLACED 5-28-91____

Medications concurrent to study

Type Dose Route Time

1. DECADRON 4MG IV Q 6 2400,0600,1200,1800
2. DILANTIN 100MG IV Q 6 SAME
3. ANCEF 1GM Q 8HR 2400 0800-1600
4. LABETALAL 2-5MG IV FOR SBP.140
5. ZANTEC 50MG IV Q 8 2200
6. THIAMIN
7. VERSED 1-2 MG PRN 1200,1315,1500
8. MANNITOL 25 GM IV 1305
9. MS 2-4 MG IV PRN 1315, 1430,1545

Neurological Assessment

Date __5-28-91__________Time__1740_____

LOC_LETHARGIC___________Pupils___SMALL/REACTIVE

Motor/sensory NO MOVEMENT ON RT --VOLUNTARY ON LEFT_________

Babinski present XX___Babinski absent __

Extraocular movements __UNABLE TO TEST__________________

Corneals _______X_________Lids ____X______Ciliospinals ___X_____

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject ___CASE B____Date__5-28-91____Calibration of Lines __XX_

A-Line transducer at foramen of Monro __X_________HOB elevation __30___

Clocks syncronized __X_Observation Number _1__ Sequence Number _3__

Turning Table Number ___4__Alignment ___T__, ___T__, ___C_,____C__

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1155	163/ 83	13	98		SUPINE
1157	163/ 84	12	100		
1159	160/ 82	13	96		
1200TURN					TURN RT LATERAL
1201	157/ 95	19	101		
1205	180/ 92	15	109		
1210	159/ 86	14	96		
1215	150/ 84	14	93		

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

EVENT RECORD
CASE B
OBSERVATION 1

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1155					
1158	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
1200	RN	TCH/TLK	PT	THERAPEUTIC	TURN TO RT
1203	RN	TCH/	PT/EQUIP	PRO	VERSED 1 MH LABATEROL
	RT	TCH	EQUIP		
	RNX3	TALKING	AT DOOR	INFO	
	RN	TALK	RN	INFO	
1208	RN	TCH	PT	MEDICATION	IM THIAMIN
	RN	TALK	RN	INFO	
	RT	TALK	RN	INFO	
	RN	TALK	RT		
1209	RT	TALK	WIFE	INFO	
	WIFE	TALK	RN	INFO	
	RN	TALK	WIFE	INFO	
	WIFE	TALK	FAM	INFO	
1210	RN	TALK	RN	AT DOOR INFO	
	WIFE	TOUCH	BED/SHEET	COMFORT	
	WIFE	TALK	FAM	INFO	
1213	FAM	TALK	WIFE	INFO	

Dependent Variable Record

CASE B

OBSERVATION 2

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1355	143/ 78	9	91		ONRIGHT SIDE
1357	140/ 77	9	90		
1359	142/ 78	9	91		
1400 TURN					SUPINE
1401	151/ 80	9	92		
1405	141/ 72	9	87		
1410	151/ 75	9	93		
1415	145/ 74	8	91		

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

EVENT RECORD
CASE B
OBSERVATION 2

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1355	RN	TLK/TCH	PT		BEING TURN
1400	RNS	TCH	PT	TURN TO SUPINE	
	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
	RN	TALK	PT	INFO	
	RN	TALK	RN	INFO	
	RN	TALK	WIFE	INFO	
	RN	TCH	EQUIP	FOLEY EMPTIED	
1405	RT	TCH	EQUIP	ET TUBING DRAIN	
	RN	TALK	RT	INFO	
	RT	TALK	RN	INFO	
	RN	TALK	RT	INFO	
	RT	TALK	RN	INFO	
1408	RN	TALK	RT	INFO	
	RN	TALK	RN	NURSE STATION	VERY LOUD
	RN	TALK	RT	INFO	
	RT	TALK	RN	INFO	
1409	RN	TOUCH	PT	PUIPL CHECK	
	RN	TLK/TCH	PT	MOTOR ASSESS	
	RN	TALK	RN	INFO	
1410	WIFE	AT BED			

DEPENDENT VARIABLE RECORD
CASE B
OBSERVATION 3

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1555	140/ 74	10	85		SUPINE
1557	127/ 67	10	80		
1559	127/ 68	10	83		
1600 TURN					CONTROL TO LEFT LAT
1602	166/ 85	19	94		
1607	158/ 76	14	87		
1612	157/ 76	13	90		
1617	159/ 76	14	93		

EVENT RECORD, CASE B, OBSERVATION 3

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1554	RN	TOUCH	EQUIP	ZERO-- CALIBRATION	A-LINE ICP
	RN	TALK	WIFE		
	RN	TALK	WIFE	INFO	
1558	WIFE	TCH	EQUIP	MOVING CHAIR	
	RN	TALK	RN	LOUD AT STATION	
	RN	TLK/TCH	PT	PRO	TURN TO LEFT LAT
1602	RN	TCH	PT	PRO	VENT ALARM X3
	RN	TALK	RN	INFO	
1603	PT	COUGHING			
	WIFE	TOUCH	PT	COMFORT	
1606	RN	TALK	WIFE	INFO	
	WIFE	TALK	RN	INFO	
1607	RN	TALK	RN	INFO	
	RN	TLK/TCH	PT	ASSESSMENT	SEIZURE ACTIVITIY ?
	RN	TALK	RN	INFO	
	WIFE	TALK	PT	ASSESSMENT	
	RN	TLK	RN	INFO	
	RN	TOUCH	PT	ASSESSMENT	
1613	WIFE	TLK/TCH	PT	ASSESSMENT	
	RN	TLK	RN	INFO	
	WIFE	TOUCH	PT	CLEAN MOUTH	SWAB
	RN	TALK	RN	INFO	
1616	WIFE	TALK	PT	INFO	SEIZURE ?
	RN	TALK	RN	INFO	
1617	RN	TALK	RN	INFO	

DEPENDENT VARIABLE RECORD

CASE B

OBSERVATION 4

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1755	199/ 61	11	68		LEFT SIDE LYING
1757	122/ 63	10	73		
1759	132/ 65	11	75		
1800 TURN					
1801	140/ 74	9	93		SUPINE
1805	149/ 79	7	97		
1810	123/ 66	7	77		
1815	121/ 66	8	76		

EVENT RECORD
CASE B
OBSERVATION 4

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1757	RN	TALK	RN	INFO	
	RN	TALK	INTERPRE	INFO	LANGUAGE BARRIER
	RN	TALK	PT	INFO	
	INTE	TALK	PT	INFO/ASSESSMENT	
1759	RN	TALK	INTERPRE	INFO	
	INTE	TALK	WIFE	INFO	
1801	RN	TOUCH	PT	PRO	TURN TO SUPINE
	INTE	TALK	WIFE	INFO	
	RN	TALK	INTERPRE	INFO	
1803	RN	TCH	PT/EQUIP	SUCTION ET	
1805	RN	TCH	PT	PRO	TEMP IN EAR
1806	RT	TOUCH	EQUIP	ET TUBES DRAIN	
1812	RN	TALK	RN	INFO	
1815	RN	TALK	RN	INFO	

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Background Variable Record

Subject Identification number ________C_______

Glasgow Coma Score on admission ______6/7__

Concurrent injuries_FACIAL TRAUMA-LF MALLEOLAR

RT PERIORBITAL EDEMA__

CAT SCAN results __LF-PEDUNCLE_HEMORRHAGE_date___6-12-91_

______________________date _____________

______________________date_____________

Previous medical history ___HEARING LOSS 60_PERCENT_______

____

Current medical diagnoses __HEAD INJURY _________________

____

Medications concurrent to study

Type	Dose	Route	Time

1. ZINCEF 15PPMG IV Q8
2. TAGEMET 300MG IVQ6
3. VIT C 1 GRAM IV Q6
4. PAVULON 2MG IV Q 1-2HR
5. TYLENOL 10GRAINS SUPPOS Q 4HRS
6. CODEINE30-60MG IM Q 4
7. VERSED 2MG IVP PRN

Neurological Assessment

Date _____6-13-91_____

Time __1600________LOC__COMA_________Pupils_RT(INJURY) LF3/2___

Motor/sensory _EXTENSION________LUE PURPOSEFUL_______

Babinski present __XX_Babinski absent __

Extraocular movements ________________________________

Corneals _____X__________Lids ___NO_______Ciliospinals ___X ON LEFT

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

DEPENDENT VARIABLE RECORD

Subject ______C_______Date __6-13-91_________Calibration of Lines _XX

A-Line transducer at foramen of Monro _____NO____HOB elevation __30___

Clocks syncronized _X__Observation Number __1_ Sequence Number ____

Turning Table Number __4__Alignment ___T__, ___T__, ___C___,___C____

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1245	160/ 73	14	84		SUPINE
1248	162/ 72	11	85		
1250	161/ 70	10	86		
1251 TURN					THERAPEUTIC TO RIGHT
1252	168/ 76	17	85		
1257	161/ 70	10	86		
1302	159/ 74	8	90		
1307	162/ 74	6	93		

EVENT RECORD

CASE C

OBSERVATION 1

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1245	RN	TALK	RN	INFO	
	RN	TOUCH	PT	PRO- CLEAN FACE	
	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
1252	RN	TLK/TCH	PT	PRO	TURN-RT LAT
	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
1257	RN	TALK	RN	INFO	PARENTS AT BEDSIDE
	RN	TALK	RN	INFO	
	RN	TALK	PARENTS	INFO	
	SO	TALK	RN	INFO	
1304	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	

DEPENDENT VARIABLE RECORD

CASE C

OBSERVATION 2

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1445	159/ 73	8	91		
1447	168/ 73	8	91		
1449	165/ 75	9	89		
1450TURN					THERAPEUTIC SUPINE
1451	163/ 74	15	84		
1455	158/ 70	12	83		
1500	155/ 67	9	83		
1505	160/ 70	7	88		

EVENT RECORD

CASE C

OBSERVATION 2

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1448	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
	RN	TCH	PT	PRO	TEMP
1450	RN	TLK/TCH	PT	INFO	
	RN	TALK	RN		TURN COMPLETE
	RN	TALK	RN		
1453	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
1452	RN	TALK	RN	INFO	
1457	RN	TALK	STATION	CHANGE OF SHIFT	MAID NOISE

DEPENDENT VARIABLE

CASE C

OBSERVATION 3

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1645	161/ 79	6	91		
1647	166/ 74	12	101		SUCTIONING PT
1649	168/ 70	14	86		
1650TURN					CONTROL TO LEFT SIDELYING
1651	167/ 66	18	91		
1655	120/ 70	23	62		EXTENSOR RIGITY
1700	143/ 56	14	78		
1705	154/ 61	10	74		

EVENT RECORD

CASE C

OBSERVATION 3

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1644	RN	TLK/TCH	PT	PRO	CODEINE IM
	RN	TALK	RN		
1645	PT	COUGHING			
	RN	TALK	PT	INFO	
	RN	TOUCH	PT	PRO	SUCTION
1646	RN	TALK	PT	INFO	
1648	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
1650	RN	TCH/TLK	PT	PRO	TURN (C)
1654	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	
1657	RN	TALK	RN	INFO	
1700	RN	WISPER	RN	INFO	
1704	RN	TALK	RN	INFO	
	RN	TALK	RN	INFO	

DEPENDENT VARIABLE

CASE C

OBSERVATION 4

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
1908	167/ 77	9	87		
1910	157/ 72	7	86		
1912	170/ 71	5	93		
1913				CONTROL TURN	PT RESISTING RT LAT
1915	156/ 82	18	84		
1919	140/ 61	14	67		
1924	154/ 68	13	78		
1929	149/ 71	17	75		POSTURING

EVENT RECORD

CASE C

OBSERVATION 4

TIME	WHO	WHAT	WHOM	PURPOSE	COMMENTS
1908	RN	TALK	MD	INFO	
	MD	TLK/TCH	PT	ASSESSMENT	ORTHO
	RN	TALK	RN	INFO	
1913	RN	TLK/TCH	PT	PRO -TURN T	RT SIDELYING
	RN	TALK	RN	AT STATION LOUD	
1915	PT	COUGH			
1917	RN	TALK	RN	INFO	Shift Change
1920	RN	TALK	RN	INFO	
1923				PT POSTURING	Head to left
1924	RN	TALK	RN	INFO	
1927	RN	TALK	RN	INFO	
1929	RN	TALK	RN	INFO	

Background Variable Record

Subject Identification number ______**Case 1**______

Glasgow Coma Score on admission ___**none performed**______

Concurrent injuries_____**non traumatic**________________________

CAT SCAN results **Subarachnoid Bleed**__date__**8-25-91**_________

Ruptured ACA Aneurysm__date _**8-29-91**_____

______________________date____________

Previous medical history _______**hypertension**_____________________

____

Current medical diagnoses __**Subarachnoid Hemorrhage**__

Surgery date __**8/29/91**___ time __**1320**____ type **Craniotomy**

duration **1320-1750**

Medications concurrent to study

Type Dose Route Time

Versed drip IV________________________________

Mannitol drip at 2 mg/min IV ________________

Dilantin 200mg BID 0800 and 1800_____________

Nimodipine 60mg po/sl 2400\0400\0800\1200\1600\2000

Kefsol 0.5grams IV Q 8 hours

Neurological Assessment

Date __**8-30-91**_______

Time **1130-2030**______LOC__**Comatose**_______Pupils_**small/reactive**___

Motor/sensory **withdrawl of lowers/RT hand grip/ Left hand no grip**

Babinski present _**bilateral**__Babinski absent __

Extraocular movements __**unable to test**_______________

Corneals__**present**__Lids _**present**__Ciliospinals _**present**__

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject __Case 1______Date_8-30-91____Calibration of Line __XX_

A-Line transducer at foramen of Monro__X___HOB elevation 30 degrees

Clocks syncronized _X__Observation Number _1_ Sequence Number __3__

Turning Table Number_6__Alignment __T__, __T_ ,___C_, C___

Time	BP	ICP	CPP	Waveform	Patient Position
5 min 1355	135 / 52	16	64	P2 / P1 = 1.28	supine
3 min 1357	139 / 53	15	64	P2 / P1 = 1.37	
1 min 1359	139 / 53	15	64	P2 / P1 = 1.38	
TURN 1402					therapeutic
1 min 1403	137 / 49	16	64	P2 / P1 = 1.41	on to left side
5 min 1407	125 / 46	15	59	P2 / P1 = 1.47	
10 min1412	120 / 45	12	57	P2 / P1 = 1.42	
15 min1417	111 / 41	9	52	P2 / P1 = 1.30	

EVENT RECORD, CASE ONE, Obervation 1 on (8-30-91)

Time	Who	What	Whom	Purpose	Comment
1358	SO	tlk	RN	info	
	RN	tlk	RN	info	
1401	INV	tlk/tch	Pt.	info regarding turn	
	RN	tlk	SO	info	
	RN	tlk	RN	info	
	RN	tch	equ	prior to turn	
1406	RN	tch	equ	checking ventilator	
	INV	tlk	SO	info	
	SO	tlk	INV	info	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

MANUAL HEAD AND NECK ALIGNMENT
DURING PATIENT TURNING
IN STATES OF
DECREASED INTRACRANIAL ADAPTIVE CAPACITY
Dependent Variable Record

Subject __Case 1__Date___8-30-91_Calibration of Lines 1330

A-Line transducer at foramen of Monro ___X______HOB elevation __30_

Clocks syncronized __x__Observation Number 2__ Sequence Number _3

Turning Table Number 4

Alignment ___T__, __T__,___C___,___C___

Time	BP	ICP	CPP	Waveform	Patient Position
5min1556	151/54	19	66	P2/P1 = 1.34	on left side
3min1558	151/51	17	66	P2/P1 = 1.35	
1min1600	150/53	18	66	P2/P1 = 1.42	
TURN1602					therapeutic
1min1603	133/51	20	60	P2/P1 = 1.36	supine
5min1607	142/56	22	62	P2/P1 = 1.49	
10min1612	117/50	15	52	P2/P1 = 0.90	
15min1617	116/49	15	52	P2/P1 = 1.02	RN lowered HOB

EVENT RECORD, Case ONE, Observation 2

Time	Who	What	Whom	Purpose	Comment
1556	RN	tlk	INV	info	
1559	RN	tlk	INV	info	
	INV	tlk	RN	info	
	GRD	tlk	RN	info	GRD = Penitentary Guard
	RN	tlk	GRD	info	
1600	RN	tlk	RN	info	
1602	INV	tlk/tch	Pt.	turn	
	RN	tlk	RN	info	
	RN	tlk	PT	info	
	RN	tlk	INV	info	
	INV	tlk	RN	info MS 6 mg IV	(see BP at end of turn)
1609	RN	tch	Pt	rectal RX for temperature control	
	RN	tlk	INV	info	
1611	RN	tch	Pt	suctioning/ patient coughing/vent. alarming	
1613	RN	tch	EQU		
1617	RN	tlk	RN	info	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject _Case 1______Date__**8-30-91**____Calibration of Lines __1655

A-Line transducer at foramen of Monro X HOB elevation **15-20**

Clocks syncronized _x___Observation Number **3** Sequence Number _3_Turning

Table Number __5___Alignment ___T__, __T__ __C___,__C___

TIME	BP	ICP	CPP	WAVE FORM	PATIENT POSITION
5min 1755	123/ 53	14	61	P2/P1= 1.12	supine
3min 1757	120/ 53	13	64	P2/P1 = 1.01	
1MIN 1759	120/ 73	13	63	P2/P1 = 1.05	
TURN 1801					Control
1 Min.1802	127/ 49	14	62	P2/P1 = .98	
5 Min 1806	123/ 49	12	61	P2/P1 = .96	
10 Min 1811	121/ 46	11	60	P2/P1 = .89	
15 Min 1816	119/ 48	11	60	P2/P1 = 1.00	

EVENT RECORD, CASE ONE, Observation 3

Time	Who	What	Whom	Purpose	Comment
1755	RN	tlk	RN	info	
	RN	tlk	RN	info	
1800	INV	tlk/tch	Pt	turn	
	RN	tch	Pt	turn	
1802	RN	tlk	RN	info	
1804	RN	tlk	RN	info	
1806	MD	tlk	RN	info	
	RN	tlk	MD	info	
	MD	tch	Pt	info	
	MD	tlk	RN	nfo	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject _Case 1__ Date_8-30-91__Calibration of Lines 1655

A-Line transducer at foramen of Monro __x_____HOB elevation 20 Clocks

syncronized _x_Observation Number __4_ Sequence Number _3_

Turning Table Number 4, Alignment __T___, __T___, __C__,__C__

Time	BP	ICP	CPP	Waveform	Patient Position
5min1955	141/53	13	70	P2/P1 = 0.93	LEFT SIDE
3min1957	147/53	14	70	P2/P1 = 0.99	
1min1959	151/54	12	64	P2/P1 = 1.00	
TURN	2001				supine
1min2002	141/56	16	70	P2/P1 = 0.92	
5min2006	148/54	15	70	P2/P1 = 0.93	
10min2011	138/53	15	64	P2/P1 = 1.01	
15m2016	128/50	13	63	P2/P1 = 1.01	

EVENT RECORD, CASE ONE, Observation 4

Time	Who	What	Whom	Purpose	Comment
1954	RN	tlk	INV	info	
	INV	tlk	RN	info	
1958	RN	tlk	RN	info	
	RN	tch	EQU		
2000	INV	tlk/tch	Pt	turn	
	RN	tlk	RN	info	teaching RN
	RN	tlk	RN	info	
2004	RN	tlk	RN	info	
2006	RN	tch	Pt	suction oral cavity	
	MD	tlk	RN	info and turn on lights	
	RN	tlk	MD	info	
2008	MD	tch	Pt	assessment/auscultation/abdominal	
2009	MD	tlk	RN	info	
	RN	tlk	MD	info	
2011	MD	tlk	RN	info	
	RN	tlk	MD	info	
2013	RN	tlk	RN	info	
	RN	tch	Pt	assessment/auscultation	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Background Variable Record

Subject Identification number ___Case 2________

Glasgow Coma Score on admission _____4-5_____

Concurrent injuries____**Left leg fractured Tibia/Fibula**

CAT SCAN results: **Right temporal/occipital bleed** date: **9-4-91 at 0300**

_____________________date ____________

_____________________date____________

Previous medical history: **Left leg pain, headache 24 hours prior to admission**

____

Current medical diagnoses: **Right temperal/occipital hematoma - uncal herniation, Intubated on blow-by**

Surgery date: **9-4-91** time **_0300__** type **__Crani____**

duration **___0345 - 0630_________**

medication administered _______________

Medications concurrent to study

Type Dose Route Time

Nipride .12 micrograms per minutes

Decadron 4 mg every 6hours 0400, 1000, 1600, 2200

Dilantin 100mg every 6 hours 1800, 2400, 0600, 1200

Neurological Assessment

Date **____9-4-91_________**

Time **____1600_____**LOC**____semi-coma_____**Pupils: R 4/3.5 L. 2.5/2

Motor/sensory: **Left side decrebrate, Right side moves purposefully (voluntarily)**

Babinski present: **bilateratl** Babinski absent __

Extraocular movements **_____unable to check______________________**

Corneals **___present______**Lids **____present__**Ciliospinals **____present____**

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject ___Case 2_______Date __9-4-91_______Calibration of Lines __x_A-

Line transducer at foramen of Monro ___x__HOB elevation _x__

Clocks syncronized x Observation Number _1__ Sequence Number ___4__

Turning Table Number __6___Alignment ___C__, ___C__ ___T___,___T___

Time	BP	ICP	CPP	Waveform P2/P1	Patient Position
5 min 1411	121/50	3	63	.70	SUPINE
3 min 1413	118/48	3	62	.68	
1 min 1415	116/47	3	63	.60	
TURN 1418					ON TO RIGHT SIDE
1 min 1419	133/57	7	67	.69	
5 min 1423	138/54	1	76	.73	
10 min 1428	119/46	3	60	.61	
15 min 1433	120/44	3	59	.70	

EVENT RECORD, Case TWO, Obserrvation 1

Time	Who	What	Whom	Purpose	Comment
1418	RN	tlk	RN		
	INV	tch/tlk	Pt	turn	
	RN	tch	Pt	give rectal suppository	
1420	RN	tch/tlk	Pt	reposition head	
	RN	tlk	Priest	regarding family	
	RN	tlk	Pt	restraints	
	RN	tlk	Pt	information	
	INV	tlk	Pharmacist	medication	
1422	RN	tlk	RN	info	
	INV	tlk	RN	info	
	RN	tlk	RN/INV	info	
	INV	tlk	RN	info	
1423	RN	tlk	INVT	info	
1426	Family (SO)	tlk/tch	PT	info	
1428	SO	tlk/tch	Pt	info/comfort	
1429	DA	tlk/tch	Pt	comfort	
1431	Family	tlk	INV	info	ventilator in next room alarms
	INV	tlk	Family	info	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Dependent Variable Record

Subject _Case 2_______Date __9-4-91_______Calibration of Lines __x_

A-Line transducer at foramen of Monro ______x_____HOB elevation ___x__

Clocks syncronized __x___Observation Number _2__

Sequence Number ___4__ Turning Table Number __4___

Alignment ___C__, ___C__ ___T___,___T___

Time	BP	ICP	CPP	Waveform P2/P1	Patient Position
5 min 1627	124/48	12	56	.81	on Right side
3 min 1629	124/48	12	56	.82	on Right side
1 min 1631	130/52	11	58	.83	
TURN 1633					control turn to supine
1 min 1634	146/45	9	68	.80	supine
5 min 1638	137/56	9	69	.80	
10 min 1643	135/53	11	63	.81	
15 min 1648	136/53	11	65	.85	

EVENT RECORD, CASE TWO, Observation 2

Time	Who	What	Whom	Purpose	Comment
1628	RN	tch	Pt	give Rx MS 2 mg	
	RN	tlk	INV	info	
	INV	tlk	RN	info	loudspeaker in this room
1630	RN	tlk	RN	info	IVAC alarm
	RN	tlk	RN	info	
1633	RN	tlk/tch	Pt	info - turn to supine position	
1634	RN	tlk	Pt	info	
1639	RN	tlk	RN	IV tubing and bag check	vent alarming

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Dependent Variable Record

Subject _CASE 2___Date__9-4-91____Calibration of Lines _1800__

A-Line transducer at foramen of Monro _x____HOB elevation _30_degrees

Clocks syncronized _x___Observation Number _3__ Sequence Number _4_

Turning Table Number __4_Alignment __C___, __C___, __T___,__T___

Time	BP	ICP	CPP	Waveform P2/P1	Patient Position
5 min 1808	142/52	10	65	1.22	supine
3 min 1810	141/51	10	64	1.20	
1 min 1812	137/50	11	65	1.25	
TURN 1814					therapeutic turn to left
1 min 1815	161/66	9	84	.98	
5 min 1819	146/56	11	70	1.2	
10 min 1824	150/60	10	75	1.01	
15 min 1829	141/55	12	67	1.18	

EVENT RECORD, CASE TWO, Observation 3

Time	Who	What	Whom	Purpose	Comment
1808	RN	tlk	RN	info	
	RN	tch	Pt	glucoscan	
1813	RN	tch	Pt	turn	
	INV	tlk/tch	Pt	turn	
1816	RN	tlk	INV	info noise in hallway/pt return OR	
	INV	tlk	RN	info	
	RN	tlk	RN	info	
	RN	tlk	MD	info	
	MD	tlk	RN	info	
	MD	tch	Pt	info (cast condition)	
	MD	tlk	RN	info	
	INV	tlk	MD	info	
	INV	tlk	RN		
	RN	tch		Blow-By Machine	

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTTIVE CAPACITY

Dependent Variable Record

Subject ___Case 2______Date___9-4-91_______Calibration of Lines __x_

A-Line transducer at foramen of Monro ___x___HOB elevation _30degree__

Clocks syncronized __x___Observation Number _4__ Sequence Number _4__

Turning Table Number __4___Alignment __C_, __C__, __T__,___T__

Time	BP	ICP	CPP	Waveform P2/P1	Patient Position
5 min 1955	155/58	10	71	.87	left side
3 min 1957	151/57	11	72	.86	
1 min 1959	150/58	12	70	1.02	
TURN 2001					therapeutic turn to supine
1 min 2002	161/61	7	78	.78	
5 min 2006	152/55	10	71	1.07	
10 min 2011	160/64	8	80	1.20	
15 min2016	155/60	9	73	1.09	

EVENT RECORD CASE TWO Observation 4

Time	Who	What	Whom	Purpose	Comment
1957	RT	TCH	EQP	ventilator tubing	for moisture
2000	RN	tlk	INV	info	
	INV	tlk	RN	info	
2001	INV	tlk/tch	Pt	turn	
2002	RN	tlk	RN	turn	
	RN	tch	Pt	comfort	
	RN	tlk	RN	info	
	RN	tlk	RN	info	
2010	RN	tch	Pt	neuro assessment	pupil check
2012	RN	tlk/tch	Pt	info	
	RN	tlk	RN	info	
2014	RN	tlk	RN	info	

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Background Variable Record

Subject Identification number _______CASE 3________

Glasgow Coma Score on admission ______7_____

Concurrent injuries__**FX Left clavicle Fx Rt. Mandible**__________________

CAT SCAN results **Rt temp. skull fx with right to left shift of 6mm 9-7-91**

Previous medical history ____________________

Current medical diagnoses ____**Closed Head**

Injury________________________

____

Surgery date _____ time ______ type _________

duration ____________

medication administered _______________

Medications concurrent to study

Type Dose Route Time

Decadron 4 mg IV 2400, 0600, 1200, 1800

Versed 2-5mg IV Prn, 0745 9/7/91 2mg

MS 5 mg, 0745 9/7/91

____

____

____

Neurological Assessment

Date ___9/7/91____________

Time ___1000___LOC_____COMA_____Pupils__2.5/2, 2.5/2_____

Motor/sensory, **RUE spontaneous movement, RLE purposeful to pain, LUE questionable purposeful movement and LLE spontaneously purposeful**

Babinski present _bilateral

Extraocular movements ___**disconjugate gaze**____________

Corneals ___**present** ______Lids ____**present**______Ciliospinals ________

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

EVENT RECORD, CASE THREE, Observation 1

Time	Who	What	Whom	Purpose	Comment
0959	RN	tlk	Pt	info	
1000	INV	tlk/tch	Pt	info - turn	
	RN	tlk	RN	info	
1001	RN	tlk	RN	info	
1012	RN	tch	Pt	pro- A-line flushed	
1016	RN	tch	Pt	pro-A-line	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Dependent Variable Record

Subject ___CASE 3________Date __9-7-9__Calibration of Lines _1000_

A-Line transducer at foramen of Monro ____x___HOB elevation _30 degrees_

Clocks syncronized __x_Observation Number __2_ Sequence Number __4_

Turning Table Number __4___Alignment ___T__, ___T__, __C__,__C__

Time	BP	ICP	CPP	Waveform P2/P1	Patient Position
5 min 1155	118/62	8	77	1.04	on right side
3 min 1157	129/74	9	84	1.09	
1 min1159	138/90	10	90	1.16	
TURN 1201					therapeutic to supine
1 min 1202	120/93	8	100	1.62	
5 min 1206	135/102	6	130	1.18	
10 min1211	156/110	8	120	1.24	
15 min1216	128/82	13	93	1.53	

HEAD AND NECK POSITIONING IN STATES OF DECREASED INTRACRANIAL ADAPTATION

EVENT RECORD, CASE THREE, Observation 2

Time	Who	What	Whom	Purpose	Comment
1201	INV	tch/tlk	Pt	turn	head resisting turn
1203	RT	tch	EQP	Proc.	
	RN	tlk	RN	Info	
1204	RT	tch	EQO	clearning moisture from tubing	
1205	MD	tlk	RN	info	
	MD	tlk	Pt	neuro assessment (LOC)	
	MD	pupil	check	Dolls eye check, extremity check, sternal rub,	
	RN	tlk/tch	Pt	assessment of reflexes	
	RN	tlk	RN		
1211	RN	tch/tlk	Pt	to prevent sitting up	
1216	Pt is restless and aggitated following assessment				

Dependent Variable Record

Subject __CASE 3____Date___9-7-91_____Calibration of Lines _1345__

A-Line transducer at foramen of Monro __x___HOB elevation__30 degrees__

Clocks syncronized __x___Observation Number _3__ Sequence Number __3_

Turning Table Number __4___Alignment __T___, ___T__, __C___, __C___

Time	BP	ICP	CPP	Waveform P2/P1	Patient Position
5 min 1355	109/56	4	69	1.51	supine
3 min 1357	114/60	4	74	.88	
1 min 1359	102/55	4	67	.93	
TURN 1402					control turn tipped to left
1 min 1403	100/55	5	70	1.16	
5 min1407	98/52	5	67	1.14	
10 min 1412	99/50	5	68	1.1	
15 min 1417	105/56	5	74	1.14	

EVENT RECORD, CASE THREE, Observation 3

Time	Who	What	Whom	Purpose	Comment
1400	RN	tlk	RN	info	
1402	INV	tlk/tch	Pt	turn	
	RN	tlk	INV	info	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject __CASE 3__Date____9-7-91___Calibration of Lines __1345_

A-Line transducer at foramen of Monro _x___HOB elevation _30 degrees___

Clocks syncronized __x___Observation Number __4_ Sequence Number __3_

Turning Table Number _4_Alignment __T___, __T___, __C___,__C__

Time	BP	ICP	CPP	Waveform P2/P1	Patient Position
5 min 1555	132/58	10	77	1.46	tipped up Right to left
3 min 1557	137/73	12	88	1.46	
1 min 1559	142/79	9	97	1.65	
TURN 1602					control to supine
1 min 1603	136/61	1	96	1.40	
5 min 1607	128/64	2	83	1.24	
10 min1612	114/47	3	76	1.25	
15 min1617	109/48	3	77	1.26	

EVENT RECORD, CASE THREE, Observation 4

Time	Who	What	Whom	Purpose	Comment
1554	RN	tch	PT	Assessment	1540 MS 5mg IV
					1543 Versed 4mg IV
1556	RN	tlk	INV		
	INV	tlk	RN		
	RN	tch	Pt	pro---inline ET suction	
1559	RN	tch	Pt	repeat ET suction	
1602	INV	tlk/tch	Pt	turn	
	RN	tch	Pt	assessment	
	RN	tlk	INV	info	
	INV	tlk	RN	info	
	RN	tlk	RN	info	
1606	RN	tch	Pt	pro-assessment of bowel tones	
	INV	tlk	RN	info	
	RN	tch	Pt	assessment - pulse check	
	RN	tlk	INV	info	
1611	RN	tch	Pt	assessment	
	RN	tlk	INV	info	
	INV	tlk	RN	info	
1613	RN	tch	Pr	assessment - Babs	
1615	RN	tch	Pt	pro-check lines	
1616	RN	tlk	INV	info	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Background Variable Record

Subject Identification number _CASE 4______________

Glasgow Coma Score on admission __7___(intubated)______

Concurrent injuries__________________________________

CAT SCAN results _Epidural Left temperal/ICH________

date_9-991___________

____________________date ____________

____________________date____________

Previous medical history ___none__________________________

____

Current medical diagnoses __depressed skull

fracture__________________________

____

Surgery date _9/9/91____ time _0800-1110_____ type __Crani_______

duration ____________

medication administered _______________

Medications concurrent to study

Type Dose Route Time

Decadron 4 mg Q6hours

Dilantin 100mg IV every 8 hours

Ancef 1 gram IV q 8 hours

MS 2 mg - 5 mg IV prn pain

Versed 2-5mg prn aggitation

Neurological Assessment

Date __9-9-91_____________

Time _______________LOC____lethargic____________Pupils_________

Motor/sensory ____

Babinski present ___Babinski absent __

Extraocular movements ____________________________________

Corneals _______________Lids __________Ciliospinals ________

Dependent Variable Record

Subject ____CASE 4______Date___9-9-91_________Calibration of Lines _xx__

A-Line transducer at foramen of Monro_x___HOB elevation __30___

Clocks syncronized __x___Observation Number _1__ Sequence Number _4_

Turning Table Number __4_Alignment __C___, __C___, ___T___,___T____

Time	BP	ICP	CPP	Waveform	Patient Position
5 min1555	105/48	11	60	1.11	supine
3 min1557	103/47	12	60	1.22	
1 min1559	112/52	13	62	1.16	
TURN 1604					
1 min 1605	107/52	11	63	.90	on to left side
5 min 1609	106/50	13	60	1.01	
10 min1614	109/50	11	61	1.05	
15 min1619	108/54	15	62	1.39	

EVENT RECORD, CASE FOUR, Observation 1

Time	Who	What	Whom	Purpose	Comment
1554	Pt			awakes with a start looks around room	
				oud speaker audible	
1601	RN	unavailable for turn			
1604	RN	tlk/tch	Pt	procedures for turn	
	INV	tlk/tch	Pt	control aspect of turn	
	RN	tlk	INV	info	
	RN	tlk	Pt	info	
	INV	tlk	Pt	info	
	RN	tlk	INV	info	
	INV	tlk	RN	info	
	Rn			rattling trash bags	
1615	PT	tlk		"Where am I?"	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject __CASE 4____Date__9-9-91______Calibration of Lines __xx_

A-Line transducer at foramen of Monro ___x______HOB elevation __30___

Clocks syncronized ___x__Observation Number _2__ Sequence Number __4_

Turning Table Number _4_Alignment __C___, __C___, ___T___,____T___

Time	BP	ICP	CPP	Waveform	Patient Position
5 min1755	101/53	7	67	.66	supine
3 min1757	110/55	7	69	.97	
1 min1759	107/54	9	66	1.18	
TURN1802					control turn to Rt sidelying
1 min 1803	94/52	15	60	1.26	
5 min 1807	115/49	13	69	1.10	
10min1812	102/49	9	62	1.02	
15 min1817	93/54	10	60	.76	

EVENT RECORD, CASE FOUR, Observation 2

Time	Who	What	Whom	Purpose	Comment
1802	RN	Tlk	RN	gave Versed (2 mg) pre turn	
	RN	Tlk	INV	info	
	INV	tlk/tch	Pt	head hold for control turn	Pt assists with turn
1807	RN	tlk	INV	info	
	INV	tlk	RN	info	telephone rings
1814	RN	tlk	INV	info	
	INV	tlk	RN	info	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject __CASE 4____Date___9-9-91_____Calibration of Lines _xx__

A-Line transducer at foramen of Monro ___x_____HOB elevation __30___

Clocks syncronized __x___Observation Number _3__ Sequence Number __4_

Turn Table Number __4___Alignment __C___, _C____, ___T___,___T____

Time	BP	ICP	CPP	Waveform	Patient Position
5 min 1955	108/53	12	63	1.08	Rt Shoulder up
3 min 1957	112/54	10	66	1.11	
1 min 1959	110/54	10	63	1.08	
TURN 2000					therapeutic to supine
1 min 2001	104/56	7	74	1.14	
5 min 2005	106/51	11	60	.82	
10 min2010	109/54	6	66	.80	
15 min2015	109/54	10	61	1.2	

EVENT RECORD, CASE FOUR, Observation 3

Time	Who	What	Whom	Purpose	Comment
1954				Change of shift report	NS station noisey
2000	INV	tlk	RN	info	
2003	RN	tlk/tch	Pt	info - awaken	
	Pol	tlk	Pt	"ID attacker"	ICP 9
2004	Pt	tlk	Pol	info"I can't think"	ICP 6
	Police scrape under nails				
2006					ICP 7
2007	Pt	tlk	Police	"who is the minute"	pt. confused ICP 8
2008					ICP 6
2009					ICP 8
2010	Police still looking and scraping under fingernails				
2011	Pol	tlk	INV	info	
2012					ICP 8
2013	Pol	tlk	INV	info	ICP 8
	RN	tlk	Pol	info	
2015	RN	tch/tlk	Pt	info	Versed 2mg

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject __CASE 4_______Date__9-9-91____Calibration of Lines _XX__

A-Line transducer at foramen of Monro ____X_______HOB elevation __30___

Clocks syncronized __X__Observation Number _4__ Sequence Number __4_

Turning Table Number _4__Alignment __C___, ___C__, ___T___,__T___

Time	BP	ICP	CPP	Waveform	Patient Position
5 min 2155	108/51	15	54	.80	
3 min 2157	107/48	16	51	.98	
1 min 2159	99/44	14	52	.88	
TURN 2200					tipped onto right side
1 min 2201	107/58	9	60	.82	
5 min 2205	100/44	6	59	.80	
10 min2210	104/50	10	61	.96	
15 min2215	104/51	11	61	.96	

EVENT RECORD. CASE FOUR, Observation 4

Time	Who	What	Whom	Purpose	Comment
2154	SO family, mother, daughter age 5, sister visiting holding subjects hands				
	RN	tlk	SO	info	
	SO	lau	RN	info	
2156	RN	tlk	Daughter info		
	RN	tlk	RN	info	
	RN	tlk/tch	Pt	info	
2200	INV	tlk/tch	Pt	turning	
	INV	tlk	RN		
2202	RN	tch	Pt	change pillow case	
2203	RN	tch/tlk	Pt	Change crani dressing	
2205				dressing change continues	
2206	Pt	tlk	RN	info	
2207	RN	tch	Pt	crani dressing change continues noise at station	
	RN	tlk	RN	info	
	Mother talk		to RN	info	
2210	RN	tlk	family	info	
	SO's	tch	Pt	comfort	
2212	SO	wispering at bedside to each other			
	INV	tlk	FAM	family tlk INV	RN's talking at desk

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

MANUAL HEAD AND NECK ALIGNMENT DURING PATIENT TURNING IN STATES OF DECREASED INTRACRANIAL ADAPTIVE CAPACITY

Background Variable Record

Subject Identification number CASE 5___________

Glasgow Coma Score on admission ___none performed________

Concurrent injuries_________________________________

CAT SCAN results - 1-7-92 ICH Pt posterior frontal and anterior parietal - midline shift 4mm right to left tiny subdural collection 3-4 mm.

1-8-92 Enlarged bleed in parietal area - shift now 12 mm right to left entrapment of left lateral ventrical - Right uncal herniation

Previous medical history reported as good health without previous medical problems.____

Current medical diagnoses ______________________________

____

Surgery date _____ time ______ type __________

duration _____________

medication administered ________________

Medications concurrent to study

Type Dose Route Time

Solumedrol 1000mg IVP and 300mg per hour for eight hours then 150 mgfor eight hours.

Mannitol 12.5 grams Q 6 hours

Codeine 60-90 mg IM Q 4 hours prn

Vistaril 50-75mg Im Q 4 hours prn

Vit C 1 gram IV q 8 hours

Apresoline 5 mg IM and 5mg IV Q 4 hours for systolics above 180mm/Hg

Neurological Assessment

Date __1-8-92______________Expired 2115

Time ________________LOC_____semi-coma____Pupils R F/F L 1.5/1___

Motor/sensory RUE withdrawl to nail bed pressure LUE decerbrate, lowers withdrawl to stimuli.

Babinski present bilateral

Extraocular movements ___unable to test_______

Corneals ____none_______Lids __none_____Ciliospinals _________

Dependent Variable Record

Subject _CASE 5___Date___1-8-92_____Calibration of Lines _xx__

A-Line transducer at foramen of Monro ____x___HOB elevation __30___

Clocks syncronized _x__Observation Number __1_ Sequence Number __1___

Turning Table Number __4___Alignment __T___, __C___, ___C___,___T___

Time	BP	CP	CPP	Waveform	Patient Position
5 min 1028	188/66	17	91	1.59	supine
3 min 1030	173/54	14	83	1.77	
1 min 1032	58/49	15	75	1.75	
TURN 1033					therapeutic to right
1 min 1034	167/61	22	76	2.22	
5 min 1038	168/59	22	74	2.17	
10 min1043	165/59	21	74	2.07	
15 min1048	164/58	19	77	1.82	

EVENT RECORD, CASE 5, Observation 1

Time	Who	What	Whom	Purpose	Comment
1028	RN	tlk	INV	info	
	INV	tlk	RN	info	
1029	RT	tlk	RN	info	
	RN	tlk	RT	info	
1033	INV	tlk/tch	Pt	for turn	
	RN	tlk/tch	Pt	for turn	
1036	RT	tlk	family	info	
	SO	tlk	RN	info (whisper) 3SO's = family	"Why is pressure UP?
1043	FAM	tlk	FAM	at bedside INFO a	
	INV	tlk	RN	info	
	RN	tlk	INV	info	
1044	RN	tlk	INV	info daughters still	talking at bedside
	INV	tlk	RN	info	
1048	at bedside			daughters still talking to	each other

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject ___CASE 5____Date___1-8-92_________Calibration of Lines _X__

A-Line transducer at foramen of Monro _____X______HOB elevation ___30__

Clocks syncronized _X_Observation Number _2__ Sequence Number __1_

Turning Table Number _4___Alignment ___T__, ___C__, ___C__,___T____

Time	BP	ICP	CPP	Waveform	Patient Position
5 min 1200	164/58	23	72	1.85	ON RIGHT
3 min 1202	165/58	22	77	2.09	
1 min 1204	172/60	23	76	2.20	
TURN1205					Supine
1 min 1206	165/50	17	74	1.80	
5 min 1210	171/77	18	76	2.13	
10 min1215	176/58	20	76	2.59	
15 min1220	167/55	20	75	2.12	

EVENT RECORD, CASE FIVE, Observation 2

Time	Who	What	Whom	Purpose	Comment
1205	INV	tlk/tch	PT	move to supine position	
	RN	tch	PT	turn	
	RN	tlk	RN	info	
1207	SO	tlk	SO	daughters talking softly at foot of the bed	
1214	RN	tlk	INV	info Dilantin 100mg	
	RN	tlk/tch	Pt	proc. IV RX	
	SO	tlk	SO	info-comfort	
	RN	tlk	INV	info	
	RN	tlk	FAM	info	
	FAM	tlk	RN	info	
1220	FAM	tlk	RN	info	
	RN	tlk	FAM	info	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject ___CASE 5____Date__1-8-92_____Calibration of Lines __1400_

A-Line transducer at foramen of Monro _____x______HOB elevation __30___

Clocks syncronized __x___Observation Number _3__ Sequence Number __1_

Turning Table Number _4__Alignment ___T__, __C__, ___C___,___T____

Time	BP	ICP	CPP	Waveform	Patient Position
5 min 1420	186/56	27	77	2.61	supine
3 min 1422	182/53	24	76	2.49	
1 min 1424	180/55	26	76	2.43	
TURN1425					control onto left
1 min 1426	169/47	26	65	2.46	
5 min1430	185/56	29	73	2.62	
10 min1435	190/60	29	76	2.01	
15 min1440	170/54	21	78	1.79	

EVENT RECORD, CASE FIVE, Observation 3

Time	Who	What	Whom	Purpose	Comment
1420	RN	tlk	INV	info	
	RN	tlk	RN	info	
	RN	tch	Pt	proc. to give hypo	Codeine
	INV	tlk	RN	info	
	RN	tlk	INV	info	
1422	RN	tlk	INV	info	
	RN	tch	Pt	proc. to give Lasix/Manitol	
1426	RN	tlk	INV	info	
	INV	tlk/tch	Pt	turn control	
	RN	tlk/tch	Pt	info	
	RN	tlk	RN	info	
1433	RN	tlk	INV	info preparing Manitol drip	
	RN	tlk	RN	info	
	INV	tlk	RN	info	
	RN	tlk	INV	info	
1436	RN	tch	Pt	proc. give Manitol	
	INV	tlk	RN	info	
1438	INV	tlk	RN	info	
	RN	tlk	INV	info	

(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Dependent Variable Record

Subject ___CASE 5__Date___1-8-92_______Calibration of Lines __x_

A-Line transducer at foramen of Monro ___x________HOB elevation __30___

Clocks syncronized __x___Observation Number __4_ Sequence Number __1_

Turning Table Number _4___Alignment __T___, ___C__, ___C___,___T___

Time	BP	ICP	CPP	Waveform	Patient Position
5 min 1555	150/44	10	69	1.78	onto left shoulder
3 min 1557	156/46	10	70	1.81	
1 min 1559	152/44	9	71	1.70	
TURN1600					therapeutic to supine
1 min 1601	168/55	14	79	2.04	
5 min 1605	161/50	13	76	1.84	
10 min1610	161/51	13	75	1.94	
15 min1615	165/53	13	77	1.78	

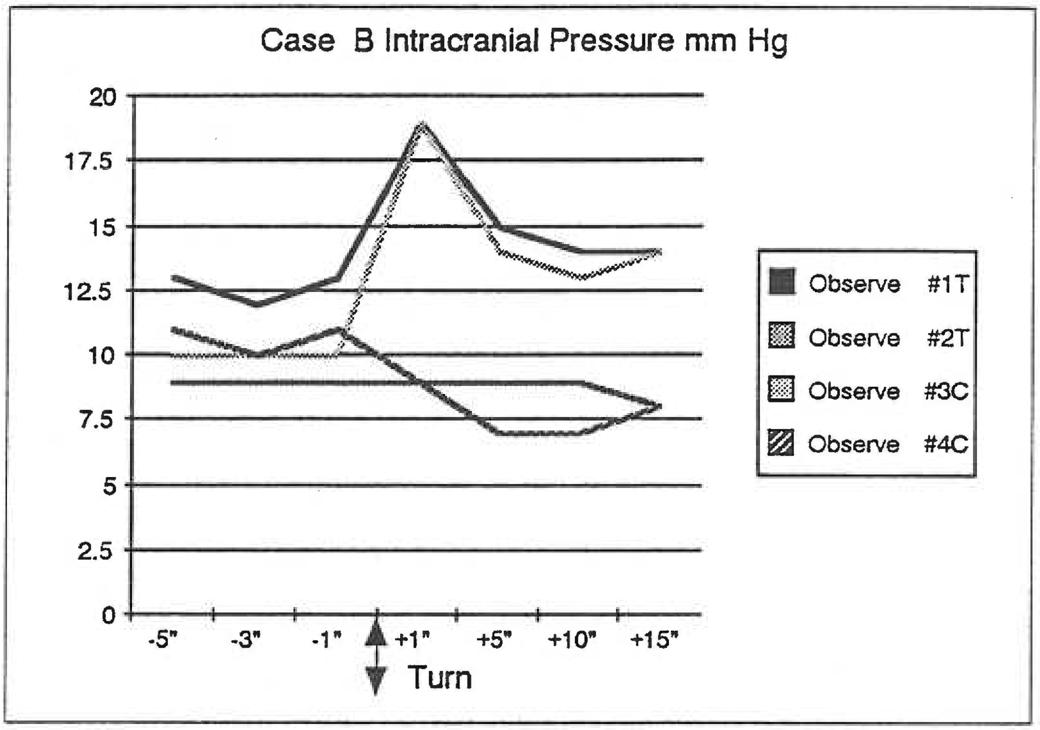
EVENT RECORD, CASE FIVE, Observation 4

Time	Who	What	Whom	Purpose	Comment
1555	INV	tlk	SO	info	daughter
1557	SO	tlk	INV		
1600	INV	tlk/tch	Pt	turn (therapeutic)	
	RN	tlk/tch	Pt	turn	
	SO	tlk	INV	info	
	RN	tlk	SO	info	
1609	RN	tlk	SO	info	
1610	Spiritual Advisor talk to family regarding patient condition				
1615		above continues			

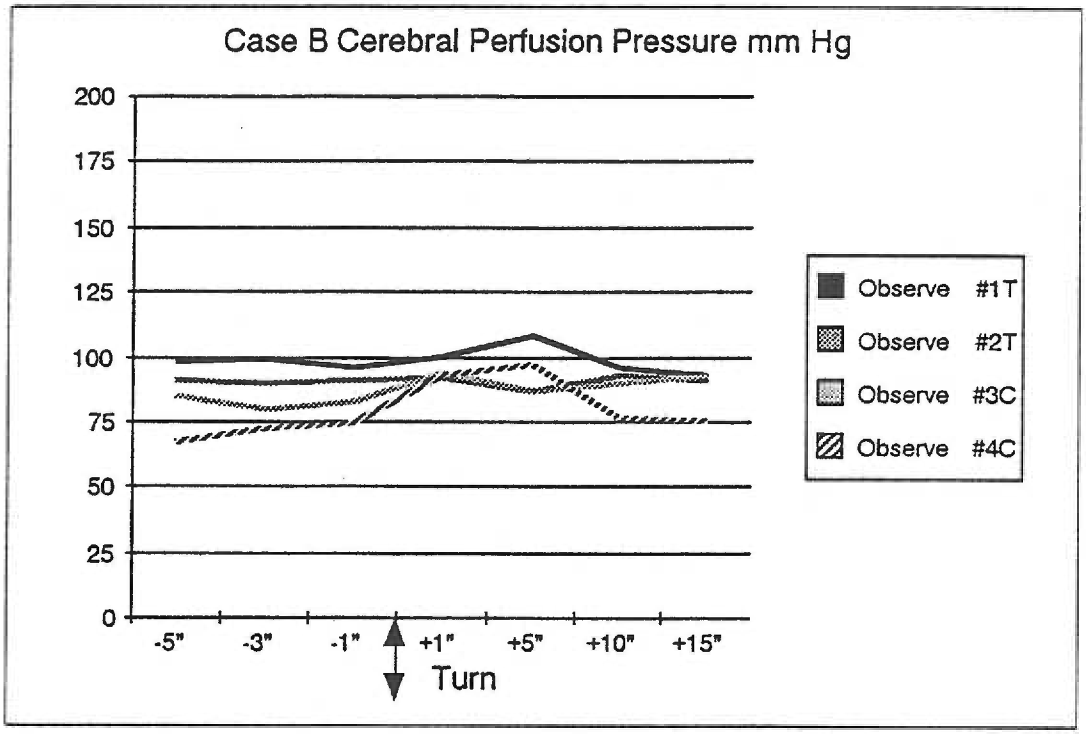
(This form is adapted from data collection forms of Felver (1989) Temporal Patterning of Physiological and Environmental Variables in Patients in an Intensive Care Unit.)

Appendix E

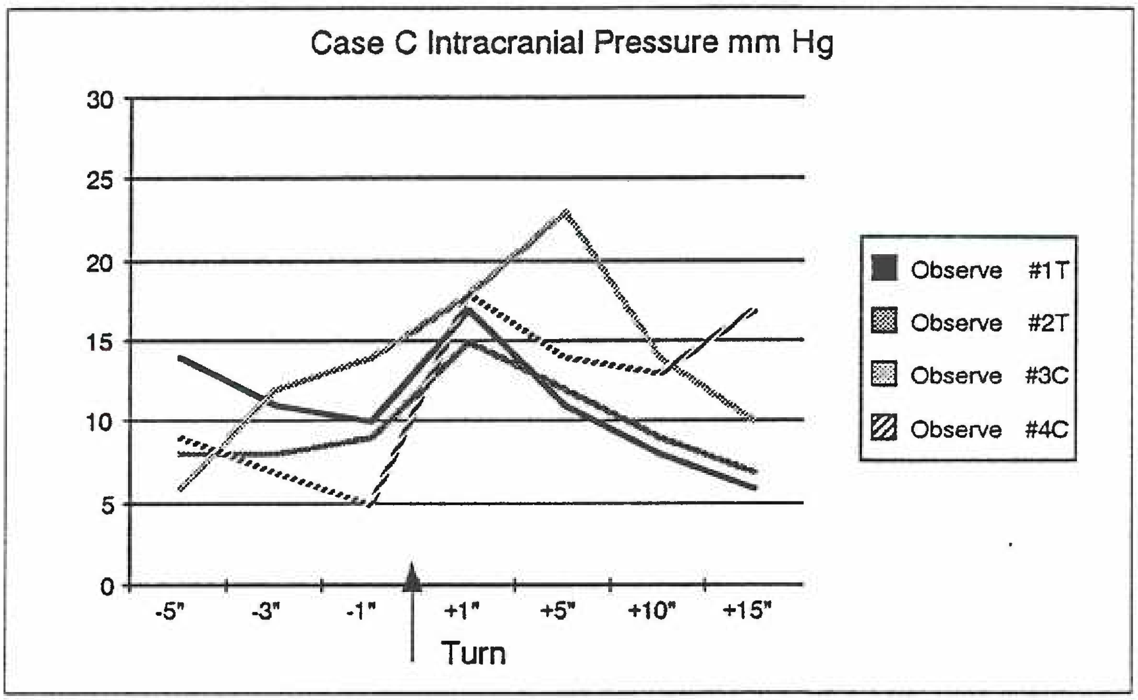
Dependent Variable Graphs for Cases B and C



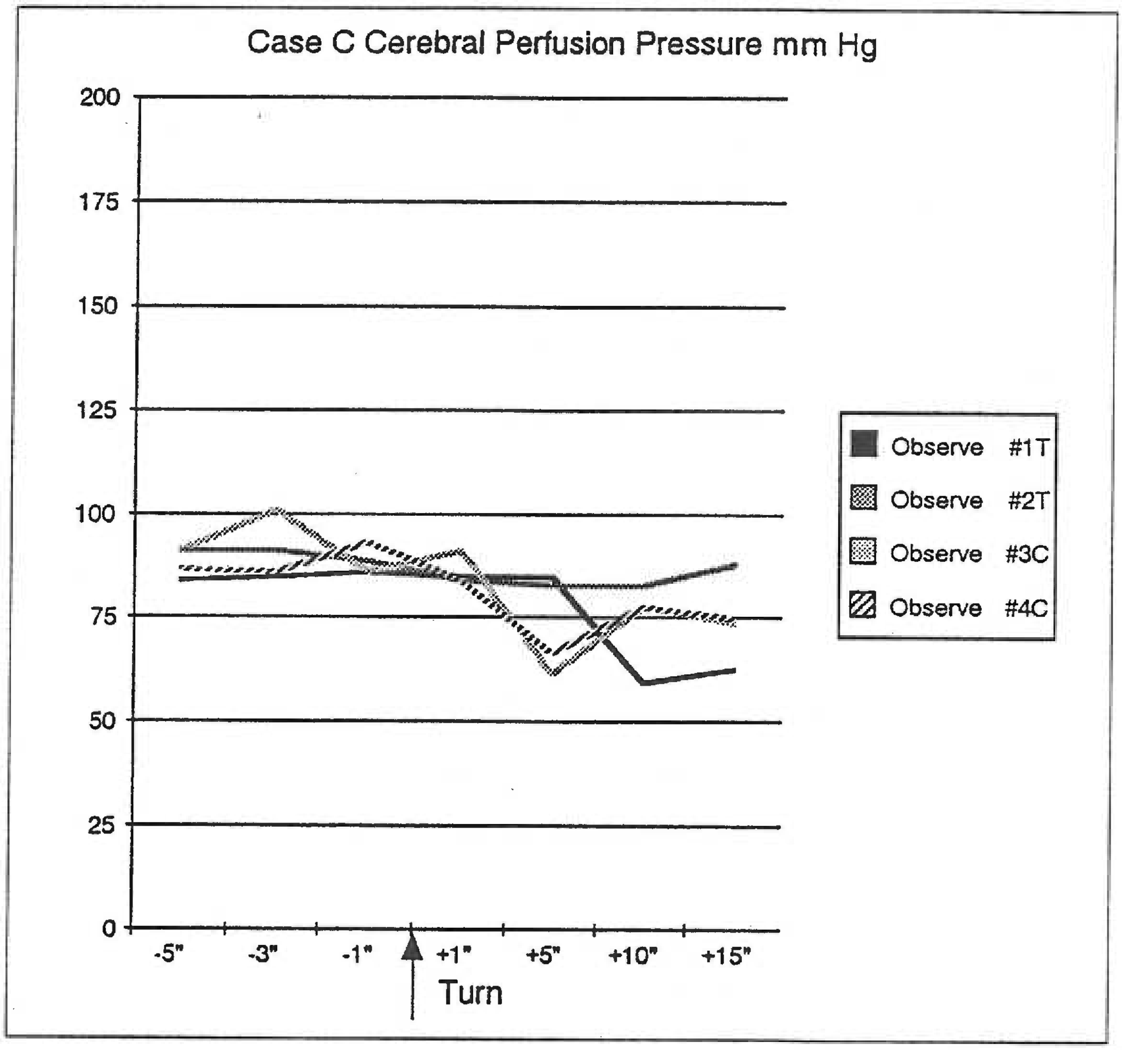
Case B Intracranial Pressure in four observation sessions.



Case B Cerebral Perfusion Pressure in four observation sessions



Case C Intracranial Pressure for four observation sessions



Case C Cerebral Perfusion Pressures in four observation sessions.