THE EFFECT OF EXTERNAL FOLEY CATHETER SOLUTION TEMPERATURE ON INTRACRANIAL PRESSURE IN THE HEAD INJURED PATIENT

by

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TABLE OF CONTENTS

CHAPTER ONE

INTRODUCTION

Uncontrolled intracranial hypertension can be a life threatening complication of patients suffering from a cranio-cerebral insult. Uncontrolled intracranial hypertension may impair cerebral blood flow and/or compress vital centers resulting in severe neurologic dysfunction. Two priorities exist in control of increased intracranial pressure. They are: 1) early detection of elevated pressures; and 2) prevention of these elevations before they occur.

Before the introduction of intraventricular pressure monitoring by Lundberg in 1960, the first priority, detection of increased intracranial pressure was dependent upon observed and inferred clinical signs. Nurses are familiar with the traditional neurological assessment: evaluation of level of consciousness, pupillary reaction and equality, symmety and strength of movement, patterns of respiration, and changes in pulse and blood pressure. Research in intracranial pressure monitoring demonstrated that traditional clinical signs do not always correlate well with early increases in intracranial pressure and when seen are often in the late stages of increased pressure (Lundberg, 1960). In addition, observations during intracranial pressure monitoring have shown that pressure increases are usually episodic. For this reason if pressure symptoms do occur they are usually fluctuating subtle changes in level of consciousness. The use of continuous intracranial pressure monitoring allows

for early detection and more rapid treatment of increased intracranial pressure. A comparison of monitored and nonmonitored patients with increased intracranial pressure demonstrated a decrease in mortality in the monitored group (McGraw, 1976).

The second priority of increased intracranial pressure monitoring is prevention. Intracranial pressure monitoring enabled identification of factors which precipitate rises in intracranial pressure. Most of the research in this area has been focused on physiologic and metabolic mechanisms that cause intracranial hypertension, the physical effects of increased intracranial pressure, and different pathologies that cause elevations of intracranial pressure (Shapiro, 1965; Miller, 1977; Mitchell, 1978).

The specific correlation between common nursing activities and increased ICP has received little attention. Mitchell and Mauss (1978) have the only reported study that looked specifically at this relationship. In their sample of nine patients with neurological dysfunction there appeared to be a clear indication that several nursing care activities precipitated a rise in intracranial pressure. Identification of these factors, whether they are nursing interventions, physical effects, or physiologic mechanisms, is the first step in preventing uncontrolled intracranial hypertension. The need remains to identify further nursing care activities that precipitate pressure rises as well as explore alternatives in delivery of these activities.

In a pilot study of six head trauma patients, it was observed that external Foley catheter care (i.e., external cleansing of the meatus and urinary catheter with room temperature solution) is associated with

an elevation of ICP. The purpose of the study is to ask the following questions: Does external Foley catheter care cause an increase in ICP? What effect does the temperature of the Foley care solution have on the ICP?

Review of the Literature

The review of the literature provides a background of the normal physiological relationships in intracranial pressure as well as factors which are known to influence these relationships. The review of the literature covers: 1) normal intracranial pressure; 2) volume-pressure relationships; 3) intracranial pressure and cerebral blood flow; 4) intracranial pressure and brain shift; 5) nursing care activities and intracranial pressure; and 6) the skin as a sensory organ.

Normal Intracranial Pressure

Normal intracranial pressure (ICP) cannot be defined as a static phenomenon. Intracranial pressure constantly changes with respiration, vascular pulsations and a variety of activities such as straining, coughing, or changing position. Normally the pressure obtained in the spinal subarachnoid space reflects the intracranial pressure because the freely circulating cerebral spinal fluid (CSF) in the craniospinal axis has a pressure equalizing effect. In many conditions of intracranial hypertension the spinal subarachnoid pressure no longer is an accurate reflection of the ICP due to blockage of CSF flow. Therefore, intracranial pressure can only be directly assessed through intracranial measurement. This may be done by measurement of the CSF pressure in the intracranial subarachnoid

space (Vries, 1973).

The normal ventricular fluid pressure range is from 0-15 mm Hg with sustained elevations of 20 mm Hg and over considered abnormal in almost all reports (Miller, 1975). In normal individuals, transient increases of pressure above 15 mm Hg lasting less than 1-2 seconds, are commonly seen secondary to everyday activities and are considered normal variations. These pressure waves are transient because regulatory mechanisms quickly return these pressures to normal.

Abnormal pressure elevations result from loss of normal regulatory and compensatory reserve. These abnormal pressure waves are seen clinically as sustained high pressure as well as episodic waves of increased intracranial pressure.

Lundberg was the first investigator to monitor ventricular fluid pressure continuously and describe abnormal pressure waves (Lundberg, 1960). He identified three distinct wave forms: A; B; and C. The only clinically significant wave is the A wave or plateau wave. The plateau wave is a transient, episodic, elevation of pressure to a level of 50-100 mm Hg. Plateau waves usually last from 5-20 minutes and start from a base of mild to moderately elevated intracranial pressure. The waves are often associated with a transient period of neurologic dysfunction such as confusion or weakness (Miller, 1975). Unless medical treatment can be instituted, the tendency is for these waves to recur at higher amplitude and greater frequency with eventual death.

Volume-Pressure Relationships

The basic volume to pressure relationship is between the skull

and its intracranial contents. The contents of the skull are conceptually composed of three parts: approximately 80 percent brain, 10 percent cerebral spinal fluid, and 10 percent cerebral blood volume (Langfitt, 1972). The relationship between the contents and the nonexpansible calvarium are what determine intracranial pressure. An increase in volume of any of the contents may be compensated for by a decrease in volume of one or more of the other constitutents. This decrease can be accomplished by expulsion of cranial cerebral spinal fluid into the subarachnoid verterbral spaces as well as drainage of cerebral venous blood into the extracranial vascular system (Langfitt, 1968). This compensatory reserve is finite and at the point where no further displacement is possible, a much larger rise will be seen in intracranial pressure. The volume of mass, the rate at which a mass expands, and the volume of intracranial contents all affect the extent of pressure change.

Miller (1975) and Langfitt (1968) demonstrated this volume-pressure relationship by injecting volumes of 1 ml of water into the intracranial space at equal time intervals. The relationship of volume plotted against pressure shows a gradual rise of pressure until all compensatory reserve is utilized at which time a slight further increase in volume produces an exponential rise in intracranial pressure. (see Figure 1)

This unit change in pressure per unit volume is expressed as $\Delta P/\Delta V$ mmHg/ml and is called elastance (Hanlon, 1977). It is the reciprocal of compliance. In pathological conditions there is high elastance with a loss of compliance.

This relationship may be illustrated in the head injured patient with secondary cerebral edema. As brain tissue water expands, the patient

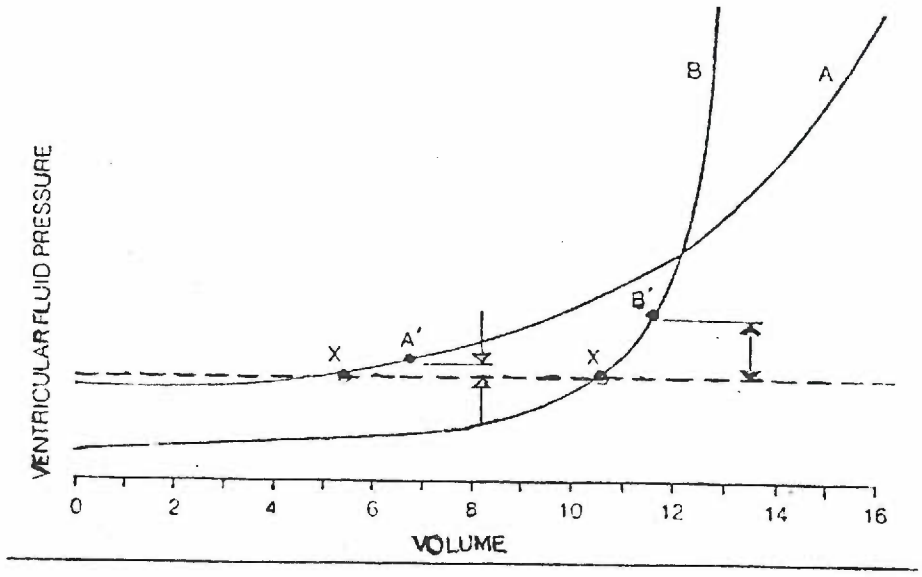

Figure 1: Two theoretical volume/pressure curves in two different patients. At the same resting pressure (X) addition of 1 unit produces a greater pressure increase on the B curve than the A curve (B' > A).

Adapted from Langfitt, T. W. Increased intracranial pressure, Clinical Neurosurgery, 1968, 16, 436-471.

moves to the right on the elastance curve with increasing elastance and decreasing compliance. Eventually a point is reached where compensatory reserve is exhausted. At this point a small increase in volume will cause a much higher rise in pressure than a porportional volume increase in the patient with normal intracranial pressure. This rapid pressure rise is often seen as the plateau or A wave described by Lundberg.

It is well documented that in some conditions transient (plateau waves) or sustained intracranial hypertension lead to neurologic dysfunction (Lundberg, 1960; Miller, 1975). This is probably due to the relationship of pressure and cerebral blood flow or pressure and brain shift.

Intracranial Pressure and Cerebral Blood Flow

Cerebral blood flow (CBF) is predominately determined by the perfusion pressure (i.e., inflow or systemic arterial pressure minus outflow or cerebral venous pressure) and/or the diameter of the cerebrovascular bed (i.e., cerebral vascular resistance).

The decrease of CBF that occurs with increased ICP can be thought of in terms of perfusion pressure changes or changes in the cerebral vascular resistance.

The following equation expresses the relationship of ICP to CBF in terms of perfusion pressure.

(1) $$CBF = \frac{\text{mean SAP} - \text{mean ICP}}{CVR}$$

(Langfitt, 1972)

SAP = systemic arterial pressure
ICP = intracranial pressure
CVR = cerebral vascular resistance
CBF = cerebral blood flow

Clinically and experimentally as ICP rises so does cerebral venous pressure. Therefore, these two pressures can be considered equal until

the veins collapse. In the above equation, if mean cerebral venous pressure is equal to mean intracranial pressure, perfusion pressure is considered to be the mean SAP minus the mean ICP. As ICP increases, perfusion pressure decreases with subsequent decrease of CBF.

If one defines perfusion pressure as mean SAP minus mean JVP the relationship of ICP to CBF is conceptualized in terms of cerebral vascular resistance.

(2) $$CBF = \frac{\text{mean SAP} - \text{mean JVP}}{CVR}$$

SAP = systemic arterial pressure
JVP = jugular venous pressure
CVR = cerebral vascular resistance
CBF = cerebral blood flow

(Langfitt, 1972)

In this equation perfusion pressure can be considered a constant because JVP is known to change very little in situations of increased ICP. Therefore, decreased CBF from increased ICP is due to increased resistance from vascular compression (Langfitt, 1965).

In conditions of increased ICP or decreased SAP the CBF will not fall until cerebral perfusion pressure is in the 40-50 mm Hg range. This regulatory mechanism is known as autoregulation and is mediated through vasodilation.

If perfusion pressure falls from increased ICP and autoregulation is intact, the arterioles will dilate to maintain cerebral blood flow. This vasodilation results in more blood entering the intracranial space, further increasing intracranial pressure.

Cerebral blood flow will remain adequate as long as CSF displacement can compensate for the increasing blood volume and brain mass. At the point on the volume-pressure curve where this is no longer possible, a slight

increase in blood volume will cause a large rise in ICP. This results in compression of cerebral vessels and ischemia with a decrease in cerebral blood flow. The ischemia elicits a vasomotor response (i.e., an increase in systemic arterial pressure) which temporarily improves cerebral blood flow but causes an additional increase in cerebral blood volume. Both pressures, SAP and ICP rise together in the form of a plateau wave or pressure wave. This cycle repeats itself until the vasopressor response fails and ICP equals mean SAP. At this point CBF ceases (Langfitt, 1965).

Intracranial Pressure and Brain Shift

It is also well documented that neurologic dysfunction is associated with conditions of brain shift (Plum & Posner, 1973). As supratentorial pressure increases, the brain reaches a point where it can no longer compensate for the expanding volume. At this point the brain herniates through the tentorial incisura resulting in compression and/or ischemia of the brain stem. Irreversible damage to the vital structure in the medulla which control respiration and cardiac function occurs (Plum & Posner, 1973).

Nursing Care Activities and Increased Intracranial Pressure

It is the goal of all personnel caring for neurological patients with diseased or injured brains to detect and control factors that might further increase intracranial volume and lead to herniation and/or cerebral ischemia. Factors have been identified that increase intracranial pressure: 1) hypercapnia; 2) hypoxemia; 3) REM sleep; 4) patient positioning; and 5) coughing and straining (Risberg, 1968; Shapiro, 975; Magnaes, 1976). These factors increase pressure by increasing

cerebral blood volume. Hypercapnia, hypoxemia, and REM sleep cause vasodilation while coughing, straining, and certain body positions cause impairment of cerebral venous drainage (Turner & McDowall, 1976). Many of these factors are associated with nursing care procedures. They should be identified and controlled.

It is well known that carbon dioxide is a potent vasodilator. Over a wide range of pCO_2, intracranial pressure increases with the maximum effect occurring at 30-50 torr (see figure 2) (Kindt & Gosch, 1972). Vasodilation effects of hypoxemia are also seen but not until pO_2 levels are less than 50 torr (Miller, 1975).

The effects of CO_2 accumulation on intracranial pressure have been seen during endotracheal suctioning. Shapiro (1975) reports that even in apneic paralyzed patients that have been hyperventilated prior to suctioning, CO_2 accumulates and is associated with significant increases in ICP.

Several other mechanisms known to increase ICP are also present during endotracheal suctioning. One is increased intrathoracic pressure during coughing. This increased pressure is transmitted to the cerebral venous system, thus, increasing intracranial pressure (Shapiro, 1975). Initiation of the arousal response with increasing cerebral blood volume may also be a factor during endotracheal suctioning. Recent work by Bedford et al. (1979) demonstrated that patients pretreated with intravenous lidocaine showed less increase in ICP during endotracheal intubation than patients without lidocaine. The lidocaine is thought to blunt the arterial blood pressure response seen during intense stimuli.

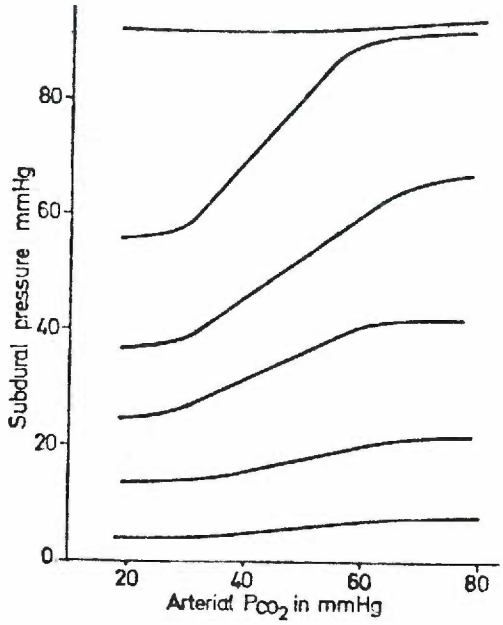

Figure 2: Profile curves demonstrating the relationship between intracranial pressure and alteration in arterial pCO_2. After decompensation there is no longer a response to pCO_2 as shown by the top line.

Kindt, G. W. and Gosch, H. H. Arterial pCO_2 effect at various levels of intracranial pressure. In M. Brock and H. Dietz (Eds.), Intracranial pressure. Springer-Verlag, 1972.

Increased mental activity as well as REM sleep are shown to increase cerebral blood flow (Risberg, 1968 and Townsend et al., 1973). Increased neuronal activity is thought to be accompanied by increased cerebral metabolism and therefore, increased cerebral blood flow. Although the magnitude of increased cerebral blood flow is not clear, even small increases in blood flow have been shown to aggravate existing elevated intracranial pressures especially in the patient with impaired autoregulation (Langfitt, 1965). Knowledge of increased cerebral blood flow during mental activity causes one to speculate how nursing interventions may decrease mental stimulation in patients with compromised intracranial pressure.

Changes of body and head position have been shown to trigger plateau waves in patients with increased intracranial pressure (Hulme, 1966; Magnaes, 1976; Shalit, 1977). Magnaes (1976) looked at the effect of lateral to sitting position changes on ICP. He noted that transient cerebral spinal fluid pressure waves were present in all patients and were considered the normal response to postural change. In patients with increased intracranial pressure the waves were of higher amplitude and often longer duration. The cerebral spinal fluid pressure elevations were aggravated by the more rapid the change in posture. The intracranial pressure changes were thought to be caused from increased cerebral blood volume secondary to blood pressure alterations as well as brain autoregulation. The clinical implication for nursing care is avoidance of rapid sitting up or lying down of the brain injured patient (Magnaes, 1976).

Forward flexion of the head and full rotation to the right or left

were observed continuously for twenty-four hours in an attempt to correlate specific nursing care activities with transient or sustained increased intracranial pressure.

Activities such as turning, suctioning, chewing, coughing, use of the bedpan and conversation were noted to increase ventricular fluid drainage. From their observations, Mitchell and Mauss (1977) suggested that other activities be investigated such as pain, conversation, arousal, and stimuli of various modalities.

The Skin as a Sensory Organ

The skin as a a conceptual major sensory receptor has gained considerable significance in its physical as well as behavioral functions. The importance of cutaneous sensation to the human organism is expressed anatomically by the large surface area of the skin containing millions of sensory receptors. In the brain also a very large proportion of the sensory cerebral cortex represents tactile sensations.

The skin has an integral role in the development of physical functions. To make any meaningful movement, the central nervous system is dependent on the sensory input it receives from the skin to make the necessary motor adjustments. This continuous stimulation of the skin is necessary in maintaining both sensory and motor function. The importance of the skin as a sensory organ can be appreciated by imagining the loss of all cutaneous sensation versus the loss of another sensory modality such as vision.

Ashley Montagu (1971) focused on the psychological aspects of the skin and built a convincing case for the role of the skin in the development

of the human being. He suggests that healthy development of an infant is dependent not only on the quantity but the quality of the cutaneous stimulation the infant receives at birth. Observations of infants who fail to thrive as a result of limited cutaneous stimulation as well as numerous animal experiments lend support to his suggestions. The quality is also considered to be more positive or beneficial when it is light and done with a warm hand than if it is firm and done with a cold hand. Personal knowledge that the touch of a cold hand is not pleasant and the touch of a warm hand is, allows us to consider that temperature may be as important in the act of touching as the sensation of pressure. Therefore, in this experiment the effect of cutaneous stimulation on ICP will be explored looking specifically at the variable of temperature.

Purpose

The purpose of this study is to assess the effect of external Foley catheter care solution temperature on intracranial pressure in head trauma patients.

Hypothesis

The use of warmed Foley care solution (112-115°F) in the head injured patient will cause less increase in intracranial pressure than the use of room temperature Foley care solution (68-72°F).

Definitions

Intracranial Pressure Monitoring

Intracranial pressure monitoring is the continuous electronic monitoring of cerebral spinal fluid pressure. It is used in patients suspected of increased intracranial pressure and provides information for diagnosis, treatment, and prevention.

Head Injury or Head Trauma

Head trauma is an open or closed craniocerebral insult that potentially may have increased intracranial pressure from a mass lesion, brain swelling, or fluid volume shift.

Autoregulation (of cerebral blood flow)

Autoregulation is the alteration in the diameter of the resistance vessels of a tissue or organ that tends to maintain constant blood flow during changes in perfusion pressure (Langfitt, 1968, p. 457).

Cerebral Blood Flow

Cerebral blood flow is the volume of blood passing through the brain or part of the brain in unit time (Gobiet, 1972, p. 372).

Perfusion Pressure

Perfusion Pressure is the difference between the pressure presented by the perfusing fluid when it enters and when it leaves the said tube or system (Gobiet, 1972, p. 372)

Intracranial Pressure

Intracranial pressure is the term used to designate the pressure of the CSF.

CHAPTER TWO

METHODS

Subjects and Setting

All subjects in this study were head injured patients 16 years and older who on admission to the surgical intensive care unit at the University of Oregon Health Sciences Center Hospital, South, responded only to noxious stimuli. A rigid protocol of treatment was followed. (Appendix A) After hemodynamic and respiratory status were stabelized, a computerized axial tomography scan (CAT) was performed to evaluate the pathology. If a hematoma was found the patient was taken to the operating room for evacuation of the hematoma as well as insertion of a subarachnoid screw for ICP monitoring in the SICU. For patients in which no surgical procedure was indicated, but increased ICP suspected, a subarachnoid screw was inserted for continuous monitoring as an aid to further diagnosis and therapy.

While in the surgical intensive care unit all the patients were managed on a standard ICP protocol. Those patients that required assisted ventilation were maintained at moderate acute hypocapnia (pCO_2 - 27-32 torr, normal 40-45). Decadron®, 200 mg/day, was given for the first three days followed by a gradual tapering schedule. In addition, ICP was controlled for waves over 30 mm Hg or for waves of 20 mm Hg which lasted for five minutes. This control was in the form of mannitol, barbiturates, and hypothermia. The early neurologic assessment was according to the Glascow Coma scale. (see Table 1)

The Glascow coma scale is a worldwide, standardized method of rating the severity of head injury. The rating is done on the response of the patient in three different areas: eye opening, motor response and verbalization. The patient's response is assigned a number. (see Table 1) The higher the number the more responsive the individual is considered with the best response being scored a 15. This tool is useful in predicting prognosis, assessing progression of injury, as well as evaluating treatment and prevention measures (Rimel,1978).

Design

The design for this study was prospective and quasi-experimental. The independent variable was the warmed solution of the Foley care and the dependent variable was intracranial pressure. Patients were entered into the study the first morning after admission and monitored for three days.

Foley care was divided into two groups, Foley 1 and Foley 2. Those patients in group 1 received Foley care with room temperature solution followed by Foley care with warm solution. Those patients in group 2 received the opposite, Foley care with warm solution followed by Foley care with room temperature solution.

Two plans of standarized nursing care, A and B, existed in the surgical intensive care unit secondary to a separate ICP/nursing care study (Bruya, unpublished). (see Appendix B) The effects of this study were minimized by assigning group 1 or 2 to plan A or B so that four groups existed within each day: B1, A2, B2, and A1. Groups one and two were also alternated across each patient to prevent systemic bias. The design was as follows.

Table 1

The Glascow Coma Scale Response Chart

	Examiner's Test	Patient's Response	Assigned Score
Eye Opening	Spontaneous	Opens eyes on his own	4
	Speech	Opens eyes when asked to in a loud voice	3
	Pain	Opens eyes to pain	2
	Pain	Does not open eyes	1
Verbal Response	Speech	Carries on a conversation correctly and tells examiner where he is, and the year and month	5
	Speech	Seems confused or disoriented	4
	Speech	Talks so examiner can understand him but makes no sense	3
	Speech	Makes sounds that examiner can't understand	2
	Speech	Makes no noise	1
Best Motor Response	Commands	Follows simple commands	6
	Pain	Pulls examiner's hand away on painful stimuli	5
	Pain	Pulls a part of his body away on painful stimuli	4
	Pain	Flexes body inappropriately to pain	3
	Pain	Decerebrate posture	2
	Pain	Has no motor response to pain	1
			Range 3-15

Adapted from Rimel, R. Emergency management of the patient with CNS trauma, Journal of Neurosurgical Nursing. 1978, 10(4), 185-188.

	Day 1	Day 2	Day 3
1	B1	A2	B1
2	A2	B1	A2
3	B2	A1	B2
4	A1	B2	A1
.			
.			
.			
n			

This design allowed for comparison of solution temperatures and ICP within patients, as well as comparison of temporal effects between day one, day two, and day three.

Data Collection Procedure

On the first morning after entry into the study the patient was given morning nursing care according to plan A or B. Both plan A and plan B concluded with the morning bath. The patient was lying on his/her back. A resting period of ten minutes ensued with ICP being recorded at zero, five and ten minutes. During the resting period the patient was left undisturbed, curtains drawn. At the end of ten minutes, external Foley care with warm or room temperature solution was given by one of two researchers. The highest ICP noted during the activity was recorded. At the end of the Foley care another ten minute resting period followed with ICP being recorded at zero, five and ten minutes. At this time the alternate temperature Foley care was given with the same recording procedure followed by another ten minute testing period. Intracranial pressure was recorded as described above.

External Foley care was given by the following procedure modified from the University of Oregon Health Sciences Center catheter care policy. (see

Appendix C) The procedure was done in a systematic manner by each researcher.

Male

1. The kit was opened, the researcher gloved, and betadine was poured over the cotton balls.

2. The urethral meatus-glans penis was cleansed from urethra toward penis discarding the cotton ball after each stroke.

3. There were four cotton balls in each catheter care kit; two were used to cleanse the glans-penis, one was used to clean the catheter, and one was used to wipe around the urinary meatus.

4. Betadine ointment was applied around the urinary meatus.

Female

1. See step one for the male.

2. The labia and urethra were cleansed stroking from clitoris to rectum, discarding each cotton ball after use.

3. There were four cotton balls in each catheter kit; two were used to cleanse the labia and urethra, one to clean the catheter, and one to clean around the urinary meatus.

For experimental Foley care the solution was warmed to the temperature of a warm bath 112°-115°F. The solution used was Betadine (provodone iodine) which was contained in leak free aluminum packages. At the beginning of the last ten minute resting period the package was immersed in 112°-115°F water and remained there until the end of the resting period and the initiation of the Foley care. Foley care was given by one of two researchers. The researcher did the Foley care, the observation, and the recording. The two researchers alternated between days across patients. This controlled for inconsistency in procedure mechanics and at the same time provided a validity check by alternating between two researchers.

Apparatus

Intracranial pressure was monitored by use of the subarachnoid screw developed by Vries in the early 1970s. The screw is hollow and has a standard luer-lok on the distal end. A twist drill hole is made 2-3 cms from the midline on the coronal suture. The dura is nicked and the screw threaded into the hole making contact with the cerebral spinal fluid in the subarachnoid space. The screw is connected to a transducer by high pressure tubing and three-way stop cocks. The transducer converts fluid pressure into electrical current proportional to the pressure. That current is displayed on an oscilloscope or strip chart recorder for continuous readout (James, 1976; Johnson, 1977). (see Diagram 1)

Observations were recorded on a data flow sheet. (see Appendix D for a sample) Additional comments were written to the side, describing activities, noise, or any intervening variables that might affect the ICP. Patient consent was provided for under another ongoing nursing care study (Bruya, 1979).

Observer Reliability

Observer reliability was checked for two purposes: to assure that both observers were making similar recordings of intracranial pressures during the Foley care activity; and to check that the observations made by the researcher while she was giving the Foley care were similar to the observations that would be made if she were only acting as observer.

At the bedside each researcher did the procedure in the presence of the other researcher so that similar technique could be assured. Observer reliability was tested at the bedside. Routine nursing activities were

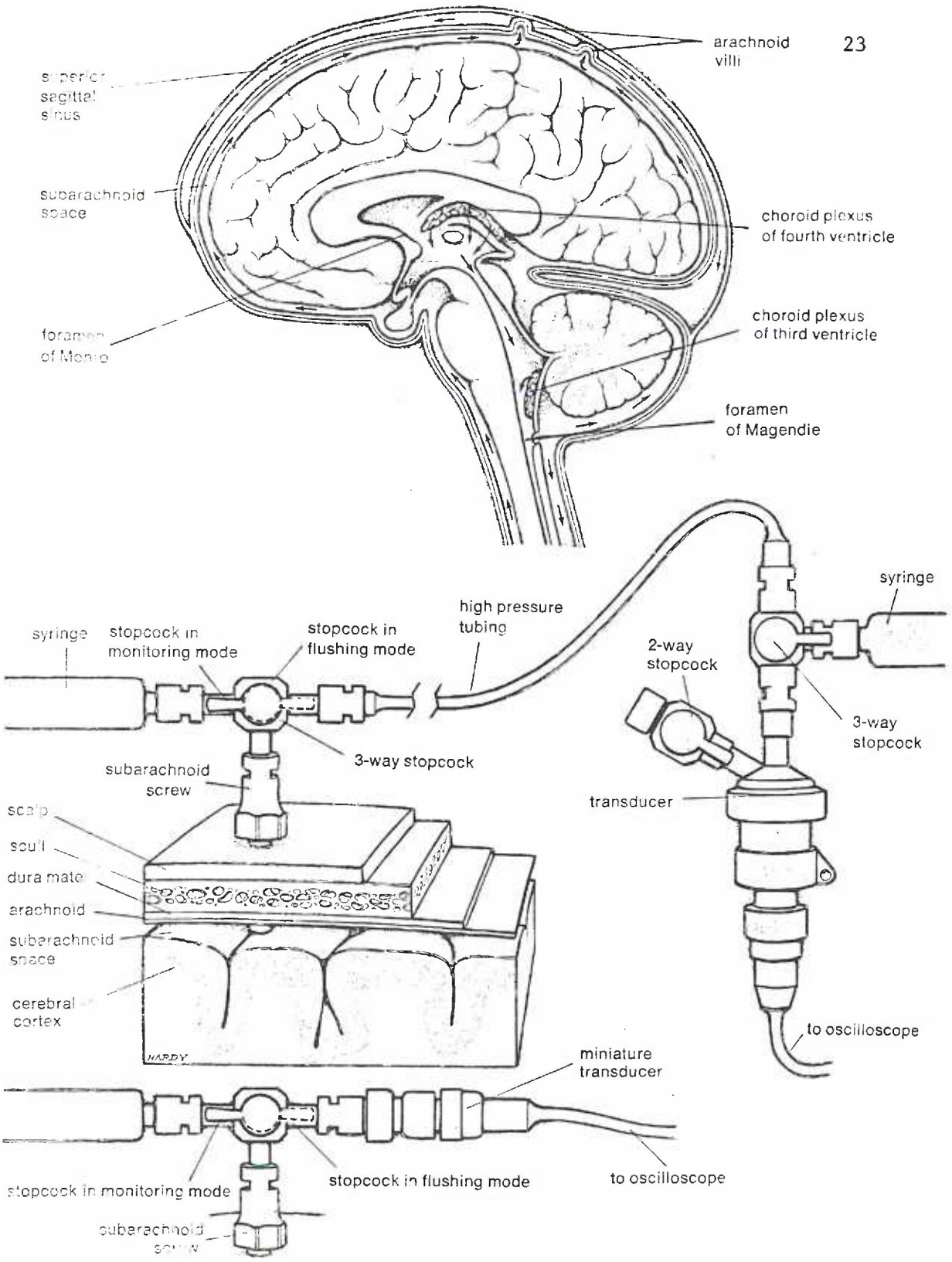

Figure 3. The subarachnoid screw

performed by the staff nurses while both researchers made simultaneous observations and recordings of ICP pressures. Comparison of these recordings achieved 95 percent agreement. The second aspect of observer reliability was tested by having one researcher do Foley care and simultaneously observe the ICP pressures, while the investigator not giving the Foley care simply observed the activity and recorded the pressures. The investigators then alternated roles. Comparison of these recordings achieved 90 percent agreement. Once agreement was shown, the remainder of the data gathering was done by only one researcher.

CHAPTER THREE

RESULTS

Characteristics of the Study Population

A convenience sample of all those patients admitted to the surgical intensive care unit during the six month data collection period who met the selection criteria composed the sample population of ten subjects. Baseline admission data for each patient are presented in Table 2. Age ranged from 16-67 with mean age of 34. Seven of the patients had surgery for intracranial hematomas secondary to head trauma. One patient had surgery for an aneurysm after a subarachnoid bleed. One patient suffered a brain contusion after a motor vehicle accident and one patient had granulomatous encephalitis, presumably tubercular in origin.

According to the Glascow coma scale (see Tabel 2) eight of the ten patients were functioning at a score of 9 or less. Essentially this score describes a patient whose only motor responses are to noxious stimulation and whose speech, if present, is confused or inappropriate. Examples of how this scale is used is shown for patients 2 and 9 in Table 3.

All ten subjects in the sample were maintained on mechanical ventilation and kept hypocapnic (PCO_2 27-32 torr) during the three days of the study. The ten subjects' initial intracranial pressures showed considerable variation. Four patients presented with initial ICP above 15mm Hg while six patients were functioning in what is considered the normal range of 0-15mm Hg.

Table 2

Individual Patient Data
On Admission to Intensive Care

Patient	Age	Sex	Diagnosis	Glascow Score	Blood Gases*	Vital Signs	Initial ICP mm/Hg
1	29	M	R. subdural Hematoma	6	PH 7.53 PCO_2 32 PO_2 98	T 100.3 P 94 BP 100/76	7
2	24	M	L. Frontal Hematoma	5	PH 7.52 PCO_2 33 PO_2 83	T 97.7 P 144 BP 160/88	12
3	34	M	L. Epidural Hematoma	5	PH 7.42 PCO_2 32 PO_2 116	T 97.5 P 92 BP 130/112	48
4	39	F	R. Subdural Hematoma	5	PH 7.58 PCO_2 29 PO_2 96	T 93.1 P 96 BP 104/85	25
5	18	M	Granulomatous Encephalitis	9	PH 7.47 PCO_2 28 PO_2 100	T 99.2 P 64 BP 130/100	15
6	40	F	Basilar artery aneurysm repair	12	PH 7.36 PCO_2 28 PO_2 156	T 101.2 P 132 BP 104/80	2
7	47	M	Subdural and Intracerebral Hematoma	13	PH 7.45 PCO_2 24 PO_2 84	T 100.4 P 130 BP 170/110	4
8	16	M	Brain Contusion	6	PH 7.46 PCO_2 32 PO_2 77	T 100.9 P 86 BP 130/86	7
9	27	M	Subdural Hematoma and Intracerebral Clot	7	PH 7.48 PCO_2 30 PO_2 137	T 98.6 P 100 BP 142/98	20
10	67	M	R. Subdural Hematoma	5	PH 7.54 PCO_2 29 PO_2 113	T 99.1 P 96 BP 140/100	13

* Blood Gases were admission gases on the ventilator

Table 3

Demonstration of the Use of the Glascow Coma Scale

Patient 2 - Glascow level	5	Patient 9 - Glascow level	7
Eye Opening		Eye Opening	
Opens eyes to pain	2	Does not open eyes to pain	1
Verbal response		Verbal response	
No verbal response	1	No verbal response	1
Best motor response		Best motor response	
Rigid extension to pain-Decerebrate	2	Pulls examiners hand away when pinched	5
	5		7

Statistical Analysis

The study tested the hypothesis that warmed Foley care solution in the head injured patient would cause less increase in intracranial pressure than the use of Foley care solution at standard room temperature.

Each patient acted as his/her own control by receiving both experimental (i.e., warmed solution) and control (i.e., room temperature) Foley care treatment, each day for three consecutive days. Observations were made on four treatment conditions each day, two resting ICP levels (R_1 and R_2) and the highest ICP level during room temperature (C) and warm (E) Foley care.

The data were subjected to a two factor analysis of variance (Days x Treatments) with repeated measures on both factors. This test provided information concerning three aspects of the data: 1) the effect of the day on the intracranial pressure; 2) the effect of the treatment on the intracranial pressure; and 3) the interaction of treatment and the day on the ICP. Summary data are found in Table 5. (Raw data in Appendix E)

Table 4

Medications Used to Control Intracranial Pressure

Patient Number	Day	Medications		
		Mannitol	Nembutal®	DMSO
1	1	none	none	none
	2	"	"	"
	3	"	"	"
2	1	none	none	none
	2	"	"	"
	3	"	"	"
3	1	X	X	X
	2		X	X
	3	X	X	X
4	1	X	X	X
	2	X	X	
	3	X	X	
5	1	X	X	X
	2		X	X
	3	X	X	X
6	1	none	none	none
	2	"	"	"
	3	"	"	"
7	1	none	none	none
	2	"	"	"
	3	"	"	"
8	1	none	none	none
	2	"	"	"
	3	"	"	"
9	1	X	X	X
	2	X	X	
	3	X	X	X
10	1	none	X	none
	2	none	none	none
	3	"	"	"

The interaction results will be considered first. The interaction effect was analyzed because uncontrolled variables such as cerebral edema, medical management, and unexpected complications, will effect the level of ICP from day to day. It is conceivable that depending on the resting ICP level, Foley care could produce different results according to the day. From the summary table the interaction effect is not significant, $F = 1.82$, $p < .05$. Therefore, the relationship between treatment and ICP is the same for each day of the study and these two factors can be considered independent.

The second piece of information obtained from the analysis of variance was the effect of the day in the course of the illness on the intracranial pressure. This proved to be insignificant, $F = .28$, $p < .05$, concluding that the ICP is essentially unchanged within days.

The last piece of information was the treatment effect. Four treatment conditions existed; two resting conditions, R_1 and R_2, a condition for warm Foley care solution (E) and a condition for room temperature Foley care solution (C). From the table it can be seen that the treatment effect was significant, $F = 3.94$, $p < .05$. This indicates that at least there is a significant difference between means but does not tell us which combination of means.

To determine which means differed and which did not a Newman-Keuls test, a multiple comparison test, was used. This test compared all possible combinations of the treatment means, $\overline{R}_1 = 13.65$mm Hg, $\overline{R}_2 = 12.81$mm Hg, $\overline{C} = 14.77$ mm Hg, $\overline{E} = 13.27$mm Hg. The results of this comparison were that the mean for room temperature solution, $\overline{C}$, was significantly larger than the other three means. The other three means, $\overline{R}_1$, $\overline{R}_2$, and $\overline{E}$ did not differ from each other. Therefore, the hypothesis was accepted concluding that

Table 5

Analysis of Variance
Four Treatment Conditions
Two Resting ICP Levels and
Control and Experimental Foley Care ICP Levels

Total	14,746.37	103		
Between Subjects	12,253.83	9		
Within Subjects	2,492.54	94		
Days	76.25	2	38.13	0.28
Days x Subjects	1,901.04	14	135.79	
Treatments	54.72	3	18.24	3.94*
Treatments x Subject	124.99	27	4.63	
Days x Treatments	69.35	6	11.56	1.82
Days x Treatments x Subjects	266.19	42	6.34	

significant p = .05

Foley care with warm solution showed a statistically significant less increase in ICP than warm Foley care.

The statistical significance of the relationship of room temperature Foley care solution to ICP was examined further in terms of ICP baselines (i.e., resting pressures) prior to the Foley care. It was speculated that patients with higher resting ICP levels might experience a larger increase in ICP than those at lower levels.

The subjects were divided into two groups according to their resting ICP baseline. One group had four patients, 3, 4, 5 and 9 with resting ICP over 15mm Hg. The other group had six patients, 1, 2, 6, 7, 8 and 10 functioning in what was considered the normal range of less than 15mm Hg.

The two groups were compared in terms of ICP difference scores for day one. The difference score was the resting ICP minus the highest ICP recorded during the Foley care. (see Table 6)

Since ICP was significantly increased by room temperature Foley care solution and not warm Foley care solution the comparison was only made for room temperature solution.

A t-test comparison was done which proved not to be significant, $t = 1.59$, df 8. Therefore, it can be concluded that the level of ICP prior to activity does not make a difference in the magnitude of elevation seen in this particular sample.

Table 6

Difference Scores for Patients with ICP less than 15 mm Hg and ICP greater than 15 mm Hg Room Temperature Solution on Day One

Patient	Resting ICP < 15mm Hg	Patient	Resting ICP > 15mm Hg
1	6	3	0
2	1	4	1
6	0	5	-2
7	2	9	1
8	2		
10	0		

CHAPTER FOUR

DISCUSSION

Results of this study lend support to the hypothesis that the level of ICP during Foley care with room temperature solution is statistically significantly higher than the resting ICP or ICP during Foley care with warm solution. More importantly, the level of ICP recorded with warm solution is not significantly different than resting ICP levels. This suggests that by warming the solution prior to Foley care, personnel caring for brain injured patients would alleviate one more factor which increases ICP.

It is also conceivable that the magnitude of this rise may be accentuated when it is not isolated from other nursing care activities. Mitchell and Mauss (1978) suggested that the cumulative aspect of nursing care might produce a higher increase in pressure than if activities were spaced with resting periods.

This study also examined the magnitude of the rise of ICP in relationship to the resting ICP baseline. It was suggested that patients with higher intracranial pressures (i.e., > 15 torr) versus those with ICP less than 15 torr would be functioning at a higher point on the volume/pressure (i.e., elastance) curve. Consequently, a greater rise of ICP would be seen for a given pressure change. Lundberg (1960) expressed this in his plateau wave description which he noted frequently arose from an elevated baseline of intracranial pressure.

Statistical analysis revealed no significant difference between these two groups. Conclusions drawn from these results are only tentative due

to the small sample size, but two explanations can be offered.

First, the exact location of these patients on the volume/pressure curve is not known. Since we do not know if any of these patients were actually on the vertical portion of the curve it is possible that the maximum ICP elevation seen with room temperature external Foley care is not demonstrated in this sample.

Secondly, the lack of discrepancy in magnitude of rise at different ICP resting levels may be explained by an uncontrolled medication variable. Patients 3, 4, 5 and 9 (with resting ICP > 15 torr) were being treated for increased ICP according to medical protocol with intravenous mannitol, nembutal ®, and/or DMSO. The other patients were not receiving these medications.

Barbiturates are known to depress the activity of all excitable tissues, but at different rates and serum concentrations. Although the central nervous system is more quickly depressed than the peripheral tissues, at anesthetic levels, a generalized depression will be seen (Goodman & Gilman, 1975). The subjects in this study were maintained within an anesthetic range of serum nembutal ® levels 2.5-3.5mg percent to decrease metabolic activity of the brain. In ICP management an initial dose of 5mg/Kg IV was given followed by 1mg/Kg/hr continuous IV infusion. At these barbiturate levels it is possible that there was a decrease in skin sensitivity which provides an explanation for the lack of significant difference between the two groups.

In the future, consideration of the drug choice in ICP management may have a direct relationship to the ICP changes seen with cutaneous stimulation. This may be of some importance with the present use of DMSO

for ICP control. Presently this drug is being used experimentally and although the exact mechanism of action is not known, it is not thought to depress excitable tissues (de la Torre, et al., 1972).

In summary, the results of this study show that the temperature of the solution used in external Foley care is a factor in the elevation of ICP and warrants warming of Foley care solution prior to the activity.

Limitations

The major limitation of this study was the dependency on numerical values for recording ICP elevations. Interpretation of ICP change was based on the highest score observed during the Foley care activity. The duration of this increase was monitored by observing the patient for ten minutes following the activity. In this study only brief episodes (less than one minute) of increased pressure were ever observed during the activity with immediate resumption of resting pressures.

Although the duration of pressure elevation was brief in this study, this parameter is of importance in interpreting the seriousness of pressure elevations. According to Lundberg, the brain is able to handle brief episodes of high pressure. It is the high pressures of long duration (2-20 minutes) or plateau waves that threaten cerebral blood flow and neurological function (Lundberg, 1960). Ideally a continuous write out of wave form along with numerical values would provide a more precise means of data recording and answer the questions of duration as well as level of elevation.

The other limitation of this study was failure to calibrate the monitor prior to each morning's care. This limitation is offset by the fact that

the monitors are calibrated on a routine basis in the ICU which should assure linearity. In addition, the scores used in this study were not absolute scores but a comparison of difference scores. This allowed these scores to have a relative comparison due to the fact that they all started from the same zero point.

CHAPTER FIVE

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

Preventing increased intracranial pressure for the patient at risk is a major goal of nursing practice in the intensive care unit. The first step toward this goal is to identify those aspects of standard nursing practice that may potentially cause rises of ICP and subsequently devise alternative methods of practice.

The study explored one such aspect of nursing practice, external Foley catheter care. Rises of intracranial pressure had been noted during Foley care in a previous pilot study which gave impetus for the study of this particular activity. Research on touch, temperature and on the skin as a sensory organ has given some support to the role of temperature as a variable in the responses seen during handling (Montague, 1971). Therefore, this study looked at the temperature of the solution in external Foley care procedures.

The subjects were ten patients with head injuries admitted to the surgical intensive care unit of the University of Oregon Health Sciences Center. All patients were suspected of increased intracranial pressure and were being electronically monitored via a subarachnoid screw. All patients with elevations of 20mm Hg for five minutes or more were treated according to protocol with mannitol, nembutal ® and DMSO.

Control for solution temperature was built into the quasi-experimental

modified time-series design. Solution temperature of 112-115°F was used for the experimental Foley care and room temperature solution was used as the control. Intracranial pressure was monitored for a ten-minute resting period prior to each Foley care, during the activity, and for a ten-minute resting period after the activity. Control and experimental Foley care were done on each patient for three consecutive days.

A two factor analysis of variance with repeated measures on both factors was applied to the data with resulting statistical significance for the treatment effect. A Newman-Keuls test showed the mean for the room temperature solution values to be significantly larger than the other means, thus accepting the hypothesis.

Conclusions

The findings of this study indicate that the elevations of ICP in head injured patients seen during external Foley catheter care are influenced by the temperature of the solution. Measurable differences between ICP elevations with warm and room temperature solution suggest that in this study population it would be advantageous to warm the solution. Although the exact mechanism for this difference is unclear, and implications for clinical practice can only be made on a tentative basis, it appears Foley care solution should be warmed.

In this study, it was possible that the effect of cutaneous stimulation for those patients most at risk was obscured by treatment with barbiturates. Additional study of the effects of cutaneous stimuli on patients not receiving barbiturates is worth exploring.

Recommendations

On the basis of this study, it is suggested that the following recommendations for further study be considered.

1. Use of a polygraph machine as well as numerical values to facilitate a more accurate degree of evaluation of ICP pressures and their duration.
2. Future studies to investigate the effects of other specific nursing activities on ICP.
3. Additional research on the effect of cutaneous stimulation in the brain injured patient using patients who are not receiving barbiturates.

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APPENDICES

APPENDIX A

Increased ICP Management Protocol

INCREASED ICP MANAGEMENT PROTOCOL

I) General:
- A) Maintain serum osms about 300 mi osm
- B) BP below 140 systolic
- C) ↑ HOB 30°
- D) Initial Decadron 200 mg/day in 4 doses for adults, taper $\bar{p}$ 3 days
  " 1.5 mg/Kg/day kids

II) Initial Rx
- A) Establish airway
- B) CT scan to evaluate pathology
- C) Control ventilation if P_aCO_2 is not >27 <32. If not, intubate, paralyse as needed and control to $27<P_aCO_2<32$. Supplement O_2 to keep P_aO_2 over 90 Torr, PEEP to keep FiO_2 under 50%.
- D) Place Richmond Screw as indicated. (Write order to contact Margaret Bruya when screw is placed)

III) If ICP
- A) Progressively rises above 20 Torr
- B) Has waves over 20 Torr for greater than 5 minutes
- C) Waves over 20 Torr in response to stimulation (suctioning, etc.) which does not subside within 5 minutes
- D) Any pressure over 30 Torr----------THEN IV)

IV) (First 24 hrs. post injury)
- A) Mannitol 0.25mg/Kg IV or Lasix o/5 mg/Kg IV

  --if this is shortlived or ineffective--

- B) Nembutal 5mg/Kg IV followed by Nembutal 1mg/Kg/hr IV infusion Shoot for Nembutal level of 2.5-3.5mg%

V) After the first 24 hrs
- A) Mannitol 0.5mg/Kg IV

  --if this is ineffective or must be repeated more often than q3h or osm is greater than 320mosm--

- B) Nembutal as above, if not done before

  --if this does not work--

- C) ↑ barbiturate infusion until a) pressure is controlled, b) EEG is flat or shows burst suppression, c) cardiac index falls below 2.5L/min/M^2 (Swan-Caloric Technique) measurement, d) QRS > .12 sec.

  --if pressure is <u>still</u> elevated--

- D) Hypothermia to 32°C
- E) If ICP rises on barbiturates or during hypothermia and osmolality is <320mi osm, give Mannitol 0.5mg/Kg

APPENDIX B

Morning Care Plan A and B

Morning Care Plan A and Plan B

Plan A: Morning nursing care activities that were not spaced with resting periods

Order of Nursing Care Activities

1. Vital Signs

   Blood pressure
   Pulse
   Temperature
   Respirations

2. Suctioning/bagging and oral care

3. Bath

   Face Neck
   Shoulders
   Chest
   Right arm
   Left arm
   Right leg
   Left leg
   Back

Plan B: Morning Nursing Care Activities that were spaced with ten minute resting periods

1. The plan was the same as above with ten minute periods between each of the three activities.

APPENDIX C

Foley Care Procedure From

University of Oregon Health Science Center

Procedure Manual

UNIVERSITY OF OREGON HEALTH SCIENCES CENTER

University Hospital / Department of Nursing

NURSING PROCEDURE

SUBJECT: CATHETER CARE - RETENTION CATHETER
Procedure done at least twice daily

RESPONSIBILITY	ACTION
Nurse	1. Obtain: A. Disposable catheter care kit B. Acetone C. Bag or paper for discard D. Urinary drainage tubing and collecting bag
	Male Patient
	2. Cleanse urethral meatus-glans penis, retracting foreskin and cleanse well. Foreskin should not be left in retracted position.
	3. Cleanse catheter from urethra toward catheter connector removing all crusting and drainage. Discard cotton ball sponge after each stroke.
	4. Wipe dry.
	5. Remove old tape marks with adhesive solvent. (CAUTION: avoid contact with urethra.) Retape catheter medially or laterally of super-pubic area.
	6. Apply Iodine ointment around urinary meatus.
	Female Patient
	7. Cleanse inner and outer labia with cotton balls stroking from clitoris to rectum (discarding each cotton ball after use). Cleanse directly over clitoris and urethra on last stroke.

RESPONSIBILITY	ACTION
Nurse	Female Patient (cont.)

8. Cleanse catheter from urethra toward catheter connector, removing crusting and drainage. Discard cotton ball sponge after each stroke.

9. Wipe dry.

10. Remove old tape mark with acetone (CAUTION: avoid contact with urethra.) Retape catheter to leg.

11. Apply Iodine ointment around urinary meatus.

12. Change drainage tubing system at least once a week.
    NOTE: Avoid unnecessary tubing system changes because opening the system increases the possibility of infection to the urinary tract.

13. Open sterile prepackaged drainage system and collecting bag and clamp tubing.

14. Wipe connection with Iodofor swab then firmly grasp end of retention catheter, pinch and quickly detach from tubing.

15. Reattach new drainage system.
    CAUTION: DO NOT CONTAMINATE END OF TUBING WHEN ATTACHING TO CATHETER.

16. Unclamp tubing and attach collecting bag to bedside.

17. Measure urine in used collecting bag, discard urine and dispose of tubing and bag.

APPENDIX D

Data Flow Sheet

Data Collection Sheet: INTRACRANIAL PRESSURE/FOLEY CARE

Patient number _____ Day 1 2 3 Protocol A B Foley care 1 2

Sex M F Age _____ Date of Admission _____ Hospital Day _____

Surgery _____ Screw Placement _____

Nurse _____ ICP machine _____ Initial ICP _____

Glascow score _____ Observer _____ Ventilated Yes/No

	Time	ICP Pressure	Comments
Resting Period	0"	_____	HOB*
	5"	_____	Medications:
	10"	_____	

Foley Care _____
Type
Temperature

Resting Period	0"	_____	
	5"	_____	Lab:
	10"	_____	PH _____
			PCO_2 _____

Foley Care _____
Type
Temperature

			PO_2 _____
			Na^+ _____
Resting Period	0"	_____	K^+ _____
	5"	_____	Hct _____
	10"	_____	Hgb _____

* HOB = Head of Bed

APPENDIX E

Raw Data for Foley Care ICP Levels

Raw Data of ICP Levels
For Four Treatment Observations

Treatment	Patient	Days		
		One	Two	Three
R_1	1	3	10	-
	2	14	9	-
	3	22	16	12
	4	26	28	20
	5	31	30	53
	7	4	-	-
	8	4	4	4
	9	19	9	11
	10	6	3	10
R_2	1	6	15	-
	2	10	14	-
	3	22	19	12
	4	25	29	18
	5	26	36	39
	6	4	0	4
	7	2	-	-
	8	3	2	2
	9	18	6	10
	10	6	3	2
C	1	9	17	-
	2	11	13	-
	3	22	17	13
	4	27	30	20
	5	29	36	54
	6	4	1	8
	7	4	-	-
	8	6	3	7
	9	20	9	11
	10	6	4	3
E	1	7	7	-
	2	13	9	-
	3	24	19	14
	4	26	30	19
	5	23	28	56
	6	5	0	4
	7	4	-	-
	8	5	4	0
	9	18	8	11
	10	6	4	1

AN ABSTRACT OF THE THESIS OF

JULIE HAWKINS CARTER

for the Master of Nursing

Date of receiving this degree: June 8, 1979

Title: THE EFFECT OF EXTERNAL FOLEY CATHETER SOLUTION TEMPERATURE ON INTRACRANIAL PRESSURE IN THE HEAD INJURED PATIENT

Approved: [redacted]

Margaret Bruya, R.N. M.N., Associate Professor, Thesis Advisor

Since the clinical use of direct intracranial pressure monitoring in the 1960s, research has explored many factors that increase ICP. Aspects of nursing care pose potential factors and have received limited attention. Therefore, further research is demanded, especially of specific nursing care activities.

In a pilot study, external Foley catheter care was noted to be associated with a rise of ICP. This study attempted to examine this relationship and explore an alternate method for this nursing care activity. This was done by manipulating the temperature of the Foley care solution.

Ten head trauma patients received Foley care with warm (112-115° F) and room temperature (68-72° F) solution for three consecutive days. Foley care was isolated from other nursing activities by providing a ten minute resting period before and following each Foley care procedure. Observations

were made on four treatment levels. Two resting ICP levels, R_1 and R_2, and ICP levels with warm, E, and room temperature Foley care, C.

An analysis of variance showed a statistically significant difference ($p<.05$) for the treatment effect. A Newman-Keuls test compared the means of each treatment level which demonstrated that the mean for room temperature Foley care solution ($\bar{C}$) had a significantly higher elevation of ICP than with warm Foley care solution ($\bar{E}$). Moreover, warm solution showed no significant difference from the resting ICP baselines. The conclusion drawn from this study is that the solution used during external Foley care should be warmed to a temperature of 112-115° F.