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The effect of suction catheter insertion on head injured adults

Brucia, Josephine Jacobs, Ph.D.

Case Western Reserve University (Health Sciences), 1993
THE EFFECT OF SUCTION CATHETER INSERTION
ON HEAD INJURED ADULTS

by

JOSEPHINE JACOBS BRUCIA

Submitted in partial fulfillment of the requirements
for the degree of Doctor of Philosophy

Thesis Adviser: M. Linda Workmen

Department of Nursing

CASE WESTERN RESERVE UNIVERSITY

May, 1993
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GRADUATE STUDIES

We hereby approve the thesis of

Josephine Jacobs Brucia

candidate for the PhD

degree.*

(signed) M. Aida Wohner
(chair)

Edward Rausch

Sara Doughan

Beverly F. Kleitz

date 4-22-83

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THE EFFECT OF SUCTION CATHETER INSERTION
ON HEAD INJURED ADULTS

Abstract

by

Josephine Jacobs Brucia

Endotracheal suctioning (ETS) often results in acutely increased intracranial pressure (ICP) in severely head injured adults. Because intracranial hypertension is associated with increased morbidity and mortality, ETS is a necessary but potentially dangerous nursing procedure. Insertion of the suction catheter into the endotracheal tube may be the ETS component that initiates increased ICP in the vulnerable head injured patient. The specific aims of this study were to determine in head injured adults: (a) the effect of suction catheter insertion on cerebrovascular and systemic vascular status, (b) the relationship between cerebrovascular and systemic vascular responses during suction catheter insertion with responses during actual suctioning, and (c) the effect of cerebral compliance state on cerebrovascular responses during suction catheter insertion. The independent variables were suction catheter insertion and state of cerebral compliance. The dependent variables were ICP, CPP, MAP, HR and ICP waveform configuration. The sample included 30 intubated and mechanically
ventilated severely head injured adults. All subjects were hospitalized in intensive care units of two major trauma centers, and were in the acute phase of their injury. The study was a secondary analysis of subjects' raw waveform data.

Suction catheter insertion significantly increased (p < .025) ICP, CPP, and MAP. HR was not significantly changed by suction catheter insertion. ICP and HR were significantly increased during actual suctioning compared to suction catheter insertion. MAP and CPP were not significantly changed. Subjects classified with decreased cerebral compliance prior to ETS had significantly higher (p < .05) ICP during suction catheter insertion than those subjects with normal compliance.

Further study that includes measures of subjects' cough effort, intrathoracic pressure, airway reactivity, and cerebral vasodilation during the suctioning procedure is recommended. Nursing interventions to prevent or mute airway irritant receptor stimulation include stabilizing the endotracheal tube during catheter insertion, predetermining depth of catheter insertion to avoid the carina, and administering medications to blunt irritant receptor response. Subjects with rounded or peaked ICP waveforms prior to suctioning are at increased risk of intracranial hypertension in response to suction catheter insertion.
This effort is dedicated to:

The families of the patients who made this study possible.

John whose unwavering support kept me moving forward.

Mom and Dad who always encouraged me to follow my dreams.

Bing, Brutus and Murphy who gave me perspective.
ACKNOWLEDGEMENTS

I am indebted to the Chairperson of my dissertation committee, Linda Workman, who willingly shared her time, her knowledge and clarity of thought with me. I extend my appreciation to my dissertation committee members, Beverly Roberts, Sara Douglas and Edward Ganz whose input and guidance facilitated my progress through the dissertation process. I am grateful to Ellen Rudy, who through her encouragement, mentorship and generosity made this research possible.

I wish to thank the staff at both Maryland Institute of Emergency Medical Services and Cleveland MetroHealth Center for their assistance during data collection. I am grateful to the patients and their families who agreed to participate in this study. Thanks also to Karol Lindow, for taking on the task of learning the waveform analysis program and measuring all those waves, yet a second time!

My deepest appreciation goes to my husband John who truly made this endeavor possible. Also thanks to Mom and Dad, who have fostered my success in countless ways.
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CHAPTER ONE

Background and Significance

Effect of Suction Catheter Insertion in Head Injured Adults

Every 15 seconds, someone in the United States suffers a head trauma. Every five minutes, a United States citizen dies and another becomes permanently disabled (Lewis et al., 1991). With nearly ten million head injuries yearly, craniocerebral injury is a leading cause of death and disability in the United States population (Krause et al., 1984). Although five million head injured persons survive the initial traumatic event, almost 40% will succumb during their illness (Marshall, 1986). Among those hospitalized yearly for head injury, approximately 70000 people suffer moderate to severe brain injury (Cole et al., 1991). Nearly 11,000 people yearly sustain permanent brain damage, and must live out the remainder of their lives in highly sheltered environments (Eisenberg & Levin, 1986). The economic and social costs are also very high. Head injury primarily is a disability of the young and costs approach 25 billion dollars per year (Cole et al., 1991).

During the acute phase of their injury, severely head injured adults require intubation and ventilatory support with a mechanical ventilator. Due both to their comatose state and the presence of an endotracheal tube, mechanically ventilated head injured adults cannot manage their pulmonary secretions. The intermittent removal of pulmonary secretions by endotracheal suctioning is, therefore, an
essential part of nursing care for the acutely ill head injured patient (McGinnis, 1989; Kersten, 1989).


While increased ICP during ETS is well documented, the mechanism(s) by which this response occurs is unknown. Hypoxemia, hypercapnia, Cushing ischemic reflex, vasodilatory cascade, and tracheal stimulation have been advanced as possible mechanisms for increased ICP during ETS, but have not been empirically supported. The lack of knowledge about how ETS elevates ICP has limited explication of effective interventions to prevent or blunt this potentially dangerous intracranial response to ETS. To date, studies are limited to
descriptions of possible interventions or have demonstrated inconclusive treatment effects (Crosby & Parsons, 1992; Donegan & Bedford, 1980; Parsons & Shogun, 1984; Rudy et al., 1991; White et al., 1982; Yano et al., 1986). Clearly, before effective interventions to prevent or blunt ICP increase during ETS can be tested, the processes through which ETS initiates this dangerous cerebrovascular response must be identified.

Not all head injured patients develop intracranial hypertension during ETS (Boortz-Marx, 1985; Parsons & Shogun, 1984; Rudy et al., 1991). Decreased cerebral compliance, where a small increase in intracranial volume (tissue, blood and cerebrospinal fluid) results in disproportionate ICP increase, has been recognized as an important risk factor contributing to increased ICP during ETS (Germon, 1988; Rauch, Mitchell, & Tyler, 1990). There has been limited success, however, identifying patients who have decreased cerebral compliance prior to the ETS procedure (Rauch et al., 1990). If nurses had a means to assess cerebral compliance, they could anticipate patients at risk for developing intracranial hypertension during ETS, and take actions to deter this dangerous cerebrovascular response.

During a study of ETS effects in head injured adults, ICP increased and often peaked, during introduction of the suction catheter into the endotracheal tube, **before application of negative suction pressure** (Rudy, Baun, Stone, & Turner, 1987, unpublished findings). An example of this finding is shown in
Figure 1 where catheter insertion duration is clearly differentiated from the actual suctioning component (application of negative suction pressure) of the ETS procedure.

![Figure 1](image)

**Figure 1.** Effect of suction catheter insertion on intracranial pressure. In = start of catheter insertion into the endotracheal tube. Down = maximal catheter insertion.

This observation suggests a new approach in the search for understanding of how ETS elevates ICP. If head injured adults’ cerebrovascular status is altered by mechanisms specific to each ETS component, then it would be more productive to study the individual component to better understand how the ETS procedure alters ICP. The study of suction catheter insertion as separate event of the ETS procedure will increase knowledge about how ETS alters head injured patients cerebrovascular status. If ICP increase begins during suction catheter insertion, then a new perspective about the basis for ICP response to ETS is needed.
Intuitively, tracheal stimulation best describes how catheter insertion increases ICP. To date, however, no comprehensive mechanism has been proposed and tested that explains how stimulation of the proximal airways results in intracranial hypertension. By integrating study findings about airway irritant receptors with what is known about cerebral blood flow, the current study proposed a physiologic model that explained how suction catheter insertion alters head injured patients' cerebrovascular status during ETS. The model was indirectly tested because ethical considerations for the safety of head injured patients prevented direct testing of how irritant receptor activation alters cerebrovascular status.

The threefold purpose of the current study was to (a) determine the effect of suction catheter insertion on the cerebrovascular status of head injured adults, (b) examine the relationship between head injured adults' responses to suction catheter insertion and their responses to actual suctioning (application of negative suction pressure), and (c) determine the effect of head injured adults' state of cerebral compliance before suction catheter insertion on their cerebrovascular responses during suction catheter insertion.

The significance of the study for nursing knowledge development is examined in the following sections. Cerebral compliance and its relationship to ETS is discussed. The adequacy of ICP as an indicator of cerebrovascular status is examined. Research on head injured patients' cerebrovascular responses to ETS
is analyzed for mechanisms that explain how ETS affects cerebrovascular status. A physiologic model based upon the preceding review is presented that provided the theoretical structure for this study.

Significance

Endotracheal Suctioning and Nursing

Maintaining and promoting optimal airway function has long been recognized as a fundamental concern of nursing. The profession's inclusion of airway care within nursing's domain was formalized by the North American Nursing Diagnosis Association's identification of ineffective airway clearance as a nursing diagnosis (McLane, 1987).

Endotracheal suctioning is a major nursing intervention for the diagnosis of ineffective airway clearance in critically ill patients. ETS is also the focus of nursing research (Riegel & Forshee, 1985). A national Delphi study implemented by the American Association of Critical Care Nurses (AACN) identified suctioning as a high priority domain for nursing research in the critically ill patient population (Lewandowski & Kositsky, 1983). In 1984, the Research Committee of the AACN further formalized suctioning as a priority research area by appointing the National Study Group on Endotracheal Suctioning (NSG-S). The NSG-S conducts controlled studies on endotracheal suctioning in a variety of critically ill patient populations with the goal of developing data based suctioning protocols for critical care nursing practice. The importance of the study of ETS
in critically ill head injured patients was corroborated when AACN identified the
effect of nursing activities and environmental stimuli on ICP and CPP as a clinical
research priority for 1992 (AACN News, 1992). Suction catheter insertion, as an
integral part of the ETS procedure, is both a standard nursing activity and an
environmental stimuli.

Cerebral Compliance and Endotracheal Suctioning

Because level of ICP is directly related to volume of intracranial contents
(blood, cerebrospinal fluid and tissue), any event that increases volume has the
potential to increase ICP. Although neither cerebral blood flow (CBF) or volume
(CBV) have been measured during ETS, the observed rise in ICP during ETS
strongly suggests that both CBF and CBV increase. Any activity such as ETS
increases cerebral metabolism. Because metabolism and CBF are tightly coupled,
CBF and therefore CBV, increase as cerebral metabolism increases (Berne, Winn
& Rubio, 1981; Paulson et al, 1990). As a strong and often noxious stimuli, ETS
can be expected to increase ICP through its increase of CBF and CBV.

Under physiologic conditions, a temporary increase in CBF and CBV result
in little or no increase in ICP. Regulatory and compensatory mechanisms such as
cerebral vasoconstriction (autoregulation), increased venous outflow, and
cerebrospinal fluid displacement maintain a relatively constant intracranial
environment.

Severe brain tissue injury disrupts the normal relationship between
intracranial volume and ICP. With head injury, intracranial autoregulatory and compensatory mechanisms are often damaged or overwhelmed, so that a small volume increase results in a marked increase in ICP.

The relationship between ICP and intracranial volume is expressed through the concept of cerebral compliance. Compliance is the amount of space accommodation or distensibility of the intracranial space (Langfitt, 1982). As shown in the following formula, compliance is defined as the change in volume divided by the change in pressure.

\[
\text{Compliance} = \frac{dV}{dP}
\]

The formula demonstrates the relationship of compliance to intracranial volume and pressure where compliance is inversely related to a change in pressure for a constant volume and directly related to a change in volume for a constant pressure. Normal compliance exists when an increase in intracranial volume results in minimal or no increase in ICP, as represented in the flat portion of the compliance curve in Figure 2. Decreased compliance is represented by the steep portion of the curve in Figure 2 where a small increase in CBV results in a marked rise in ICP.

Decreased compliance signals a dangerous trend of increased intracranial tissue compression and decreased cerebral blood flow that may result in brain tissue hypoxia, cerebral edema and ultimately, neuronal death (Cold, et al., 1981; Fieschi et al., 1974; Langfitt, 1982; Obrist, et al., 1984; Overgaard & Tweed).
1974). Head injured patients with decreased compliance would likely have large increases in ICP during ETS, that could extend neuronal injury and decrease their chances for meaningful recovery.

**Figure 2.** ICP response to unit volume change. P1 represents minimal pressure change per unit volume change. P2 represents a disproportionate pressure increase for the same unit volume change. Adapted from *Management of head injuries* by B. Jennett and G. Teasdale, 1981, Philadelphia: F.A. Davis p.62.

**ICP as an Indicator of Intracranial Status**

ICP has long been the major indicator of intracranial status. ICP continues to serve as the primary clinical guide for therapeutic interventions in the intensive care setting. There is a strong association between sustained or suddenly elevated ICP and devastating disability and death in the severely head injured population (Eisenberg & Levin, 1986; Lundberg, 1960; Langfitt, 1982; Marshall, 1986; McGraw, Shields, Gamel, & Greenberg, 1986; Marshall, et al., 1979; Saul, 1986; Tsutsumi et al., 1986; Yano, Ikeda, Kobayashi, & Otsuka, 1986).
The empirical indicator used to measure ICP is the magnitude of the cerebrospinal fluid pulse waveform (CSFPW). ICP magnitude (MICP) represents the mean of all pressures in the intracranial space. MICP provides information about the size of the CSFPW compared to other waveforms as measured in standard units. Although elevated MICP is associated with decreased brain compliance, low or moderately elevated MICP (10 to 20 mm Hg.), is not a dependable predictor of adequate brain compliance (Rowed, Leech, Reilly, & Miller, 1975; Sullivan, Miller & Searle, 1981). Consequently, although a sensitive indicator of decreased cerebral compliance, MICP lacks specificity. Other indicators of cerebrovascular status are needed to identify patients at risk for developing decreased compliance and its dangerous sequelae.

Two other aspects of CSFPW that have been investigated are amplitude and configuration. Amplitude refers to the height or excursion of a waveform from top to bottom (difference between highest and lowest point in the wave). ICP amplitude (ICPA) is known to vary directly with magnitude regardless of the factor responsible for the change in magnitude (Cardoso, Rowan, & Galbraith, 1983; Dardenne, Dereymaeder & Lacheron, 1969; Hamer, Alberti, Hoyer, & Wiedemann, 1977; Portnoy & Chopp, 1981; Portnoy, Chopp, Branch & Shannon, 1982). ICPA has been used an estimate of cerebral compliance, with amplitude inversely related to compliance (Avezzaat, Van Eijndhoven, & Wyper, 1979; Newell, Aaslid, Stooss, & Reulen, 1992; Rauch, Mitchell & Tyler, 1990;
Szewczykowski, Sliwka, Kunicki, Dytko, Korsak-Sliwka, 1977). Researchers have constructed the pressure-volume compliance curve based upon the relationship between ICP and MICP (Szewczykowski et al., 1977).

**Configuration** is the waveform shape or pattern. ICP configuration consistently changes to a rounded shape in response to hypoxemia, hypercapnia, and increased intraventricular volume (Cardoso, et al., 1983; Portnoy & Chopp, 1981; Portnoy et al., 1982). Because both hypoxemia and hypercapnia cause cerebral vasodilation, researchers concluded that rounded configuration represents cerebral vasodilation (Portnoy & Chopp, 1980, 1981; Portnoy, et al, 1982). ICP rounding also occurs with either experimentally induced or naturally occurring increased intraventricular volume (Bering, 1955; Portnoy & Chopp, 1980, 1981).

Each of the three preceding events increases intravascular or intraventricular volume. Because volume increase in any intracranial compartment can decrease cerebral compliance, ICP rounding also implies decreased cerebral compliance. ICP rounding, therefore, may represent both cerebral vasodilation and decreased compliance.

ICP magnitude, amplitude, and configuration each reflect conditions inside the cranial vault. Measurement of the three indicators has the potential to provide better information about head injured adults' cerebrovascular status and their responses to nursing care procedures than MICP has provided past studies.
Endotracheal Suctioning in Head Injured Adults

In the search for the physiologic basis for ICP increase during suction catheter insertion, several mechanisms can be eliminated through careful analysis of study findings. Hypoxemia, hypercapnia, Cushing ischemic reflex, vasodilatory cascade, and tracheal stimulation during ETS have been identified as possible mechanisms for increased ICP during ETS. Because any of the preceding mechanisms could occur during ETS, each is a plausible explanation for ICP increase during ETS. The following review of findings demonstrates that tracheal stimulation is the most feasible mechanism for ICP increase in response to suction catheter insertion. The specification of a mechanism is important because interventions to blunt or prevent ICP response would be different for each mechanism.

**Hypoxemia and hypercapnia.** Systemic arterial hypoxemia and hypercapnia with their local cerebrovascular vasodilatory effect were identified as possible mechanisms for increased ICP during ETS (Rudy et al., 1991; Snyder, 1983; Shapiro, 1975). In their classic study, Kety and Schmidt (1948) first demonstrated that decreased arterial oxygen tension is a powerful local vasodilator. Inhalation of 10% oxygen resulted in a 35% increase in CBF.

Elevated carbon dioxide is one of the most potent cerebral vasodilators known. Through relaxation of cerebral arterial smooth muscle, carbon dioxide causes a 2.5% increase in CBF for every 1 mm Hg. change in carbon dioxide
(Harper, 1965; Youmans, 1982).

The proposition that hypoxemia and hypercarbia are the basis for increased ICP during ETS is reasonable, because ETS results in both hypoxemia (Baun, 1977; Skelley et al., 1980) and hypercapnia (Chulay, 1988; Shapiro, 1975). Presuctioning hyperoxegenation by hyperventilation or hyperinflation, however, eliminates both hypoxemia and hypercapnia during and after ETS (Baun, 1984; Chulay, 1988; Fisher et al., 1982; Pierce & Piazza, 1987).

Rudy and colleagues (1991) oxygenated head injured adults with 100% oxygen before, between, and after suctioning. Using a pulse oximeter with a finger sensor, systemic arterial oxygen saturation was measured throughout the suctioning procedure. Although subjects maintained high oxygen saturations during and after ETS, ICP elevated during ETS. This study’s findings demonstrate that adequate systemic oxygenation throughout ETS does not prevent ICP increase. Therefore, hypoxemia cannot be the primary mechanism for ICP response to suction catheter insertion.

Fisher, Frewen and Swedlow (1982) investigated hypercapnia as the basis for increased ICP during ETS. The researchers compared ICP and end-tidal CO₂ response both to ETS and an equivalent apneic period in mechanically ventilated head injured children. Subjects were hyperventilated with high oxygen concentrations before both suctioning and apnea. No differences in end-tidal CO₂ levels were found between the ETS and apnea. Although there were no end-tidal
CO₂ differences between treatments, ICP increased significantly from baseline solely during ETS. Based upon these findings, hypercapnia is not the basis for ICP increase during ETS, however the ICP increase during ETS alone suggests that tracheal stimulation may play an important role in the phenomenon. In summary, when subjects are adequately preoxygenated and ventilated, it is unlikely that either hypoxemia or hypercarbia play a major role in ICP increase during either ETS or suction catheter insertion.

Cushing ischemic response. Parsons and Shogun (1984) proposed that the Cushing ischemic response might be responsible for the increased ICP they observed during ETS. Cushing postulated that markedly elevated ICP (ICP values equivalent to arterial pressure) caused brain tissue hypoxia. Cerebral tissue hypoxia was theorized to cause systemic responses such as arterial hypertension, bradycardia and hypopnea (Cushing, 1903). The Cushing response as a basis for increased ICP during ETS would be supported if increased ICP temporally preceded increased arterial pressure (MAP), and if ICP increased to magnitudes similar to arterial pressure values. Parsons and Shogun did not report the temporal relationship between ICP and MAP response to ETS, nor document ICP values near MAP magnitude. The only empirical evidence of temporal relationship between ICP and MAP was reported by Rudy and associates (1991), who provided preliminary evidence of an almost concurrent rise in ICP and MAP in response to ETS. No other studies reported ICP values equivalent to arterial
pressure values as a result of ETS. These findings therefore, do not support the Cushing response as the basis for ICP response during the ETS procedure.

**Vasodilatory cascade.** A process named the vasodilatory cascade has recently been identified as a basis for increased ICP in head injured patients (Rosner & Daughton, 1990). Findings suggest that cerebral vasodilation initiated by low cerebral perfusion pressure (CPP) increases ICP through an increase in CBV.

In a study of head injured adults' cerebrovascular responses to ETS, Errson and colleagues (1990) identified the vasodilatory cascade as the basis for ICP increase they observed during the ETS procedure. Findings from the study were not supportive of the vasodilatory cascade because, with the exception of one subject, MAP and ICP increased concurrently during ETS. The elevated MAP maintained CPP values at normal or higher levels, therefore, vasodilation should not have occurred. Findings from other studies corroborate increased MAP and ICP during the ETS procedure as a typical response pattern in head injured subjects (Donegan & Bedford, 1980; Parsons & Shogun, 1984; Rudy et al., 1991; White, et al., 1982). Based upon the preceding evidence, the vasodilatory cascade is not the mechanism for ICP increase throughout the ETS procedure in the majority of head injured patients.

**Tracheal stimulation.** The strongest empirical support exists for tracheal stimulation as the basis for increased ICP during ETS and suction catheter
insertion. Stimulation of proximal airways during the endotracheal suctioning procedure results in ICP increase in head injured patients (Donegan & Bedford, 1980; Fisher, Frewen, & Swedlow, 1982; White, Schlobohm, Pitts, and Lindauer, 1982; Yano et al., 1986). While investigators agree that tracheal stimulation is a major cause for ICP increase during ETS, different processes are cited for how airway stimulation results in increased ICP.

Researchers identify systemic venous change as one pathway by which ICP response is initiated (Errson et al., 1990; Fisher et al., 1982; Parsons & Shogun, 1984; Snyder, 1983; White et al., 1982; Yano et al., 1986). Mechanically ventilated patients frequently attempt to cough against the endotracheal tube when oropharyngeal and tracheal sensory receptors are stimulated (Baun, 1984). Coughing increases intrathoracic pressure, thus decreasing cerebral venous outflow. When cerebral venous outflow decreases both the volume and pressure of the cerebral capacitance vessels increase. The increase in capacitance vessel volume and pressure is exhibited by ICP elevation.

The weakness of the venous explanation is that it cannot account for ICP response to ETS in head injured patients who do not have a cough reflex. Head injured patients in deep coma, as evidenced by a Glasgow Coma Scale Score (GCS) \( \leq 8 \), often do not exhibit a cough reflex when suctioned. However findings show that subjects with GCS \( \leq 8 \) respond to ETS with elevation in ICP (Errson et al., 1990; Parsons & Shogun, 1984; Rudy et al., 1991; Yano et al.,
1986). These findings demonstrate that change in cerebrovascular status solely by the venous route does not explain ICP response during ETS in all head injured patients.

Other researchers identify systemic arterial changes as the primary mode for increased ICP during ETS (Hamill, Bedford, Weaver, & Colohan, 1981; Simard & Bellefleur, 1989; Splinter, 1990). Manipulation of the endotracheal tube initiates a strong sympathetic response by activation of the vasopressor reflex. The abrupt rise in systemic arterial pressure and heart rate is reflected in the cerebrovascular system with increased ICP.

Abrupt elevation of systemic pressure results in intracranial hypertension if cerebrovascular autoregulatory mechanisms are impaired, or MAP exceeds autoregulatory limits (Durward, Del Maestro, Amacher, & Farrar, 1983; Miller, Stanek & Langfitt, 1971; Simard & Bellefleur, 1989). Autoregulatory mechanisms are frequently impaired with severe head injury, thus permitting direct transfer of arterial pressure magnitudes to the cerebral vasculature (Cold et al., 1981; Enevoldsen & Jensen, 1978; Fieschi et al., 1974; Jennett & Teasdale, 1981; Obrist et al., 1984; Overgaard & Tweed, 1974). Severely head injured patients therefore, are vulnerable to abrupt elevation of MAP during ETS. Whether the systemic arterial pathway can explain ICP elevation during ETS in all head injured patients is not known. Because there is evidence that not all head injured subjects have impaired cerebral autoregulation, it is improbable that the
preceding mechanism explains ICP increase in all subjects (Rosner & Daughton, 1990).

In summary, there is strong empirical support for airway sensory receptor stimulation as the basis for ICP increase during ETS. However, because previous studies have not separated ICP response during actual suctioning from ICP response during catheter insertion, other mechanisms initiated by the presence of negative airway pressure during actual suctioning cannot be ruled out. Suction catheter insertion prior to the application of negative pressure provides an opportunity to study proximal airway stimulation without imposition of other mechanisms activated by other ETS procedure components. The present study investigated insertion of the suction catheter isolated from any other aspect of the ETS procedure.

Severely head injured patients vary in their ability to cough and in their degree of autoregulatory system impairment. Because of the variation both among and within patients, it is reasonable that both venous and arterial pathways transmit change in volume and pressure to the cerebrovascular system during ETS. The current study proposed a mechanism where mechanical stimulation of airway irritant receptors by a suction catheter results in both systemic arterial and venous pressure change that impacts upon cerebrovascular status during ETS.

Although irritant receptor response mechanisms formed the theoretical explanation for the conduct of this study, ethical considerations for human safety
prevented the direct investigation of these processes. The present study therefore, indirectly assessed the effects of the irritant receptor response by measuring cerebrovascular response to suction catheter insertion. A basic study assumption was that suction catheter insertion activated airway irritant receptors. Because head injury alters the normal relationship between intracranial volume and pressure, state of cerebral compliance preceding initiation of the suctioning procedure was measured using ICP configuration. MICP and CPP were measured as empirical indicators of an aggregate cerebrovascular response to all of the irritant receptor response pathways. This approach is reasonable since the three irritant receptor response mechanisms ultimately affect cerebrovascular status by increasing intracranial volume. Increased CPP was predicted because activation of the systemic vasopressor response elevated MAP (MAP - MICP = CPP).

Within the scope of the current study, the systemic vasopressor response is the only irritant receptor response mechanism that could be differentiated from the other irritant receptor responses. Based on the theoretical model proposed for this study, the vasopressor response was manifested by increased MAP that was nearly concurrent with increased MICP. Additionally, activation of the vasomotor response was manifested by increased HR.
Theoretical Basis for Suction Catheter Insertion Effects on
Cerebrovascular Status of Head Injured Adults

Physiological Responses to Suction Catheter Insertion

Insertion of a suction catheter into the trachea through an endotracheal
tube, stimulates airway epithelial irritant receptors distributed throughout the
airway mucosa, particularly near the carina (Boushey, Richardson, &
Widdecombe, 1972; Guyton, 1991; Haxhiu, Deal, Norcia, Van Lunteren, Mitra,
& Charniack, 1986; Jammes & Mei, 1979; Jenson, 1980; Levitsky, 1986; Mills,
Sellick & Widdecombe, 1969; Richardson, Herbert, & Mitchell, 1984;
Widdecombe, 1954). Stimulation of irritant receptors initiates neural transmission
of impulses via afferent fibers in the vagus to the medullary respiratory center.
Input via the vagal afferents is received primarily in the dorsal medullary groups
(predominantly inspiratory neurons) located in the nucleus tractus solitarius
(Berger, Mitchell, & Severinghaus, 1977; Guyton, 1991; Haxhiu et al., 1986;
Levitsky, 1986; Nakai & Ogino, 1984). Figure 3 displays the anatomical location
of the various neurons involved in neural responses to suction catheter insertion.

Stimulation of irritant receptors activates motor neurons in the dorsal
medullary groups (DMG) and initiates three major systemic responses (see Figure
4). First, DMG generated response is sent via vagal efferent fibers to the spinal
cord and completes a reflex arc. Local spinal reflexes integrated with the DMG-
initiated response cause smooth muscle in the extrapulmonary airways to contract
resulting in bronchoconstriction (Guyton, 1991; Levitsky, 1986; Nadel & Barnes, 1984).

Figure 3. Schematic representation of dorsal view respiratory and vasomotor motorneurons in medulla. DMG = dorsal medullary group; VMG = ventral medullary group; SN = solitary nucleus; NA = nucleus ambiguus; NRA = nucleus retroambigualis. Adapted from "Regulation of respiration" by A. Berger et al., 1977, New england journal of medicine, 297, 92-97.

As shown in Figure 4, the cough reflex is a second response to stimulation of irritant receptors, particularly with stimulation near the carina. Impulses are sent to the DMG via vagal afferent fibers. The DMG sends collateral messages to the ventral medullary groups (VMG) located in the nucleus ambiguus (see Figure 3). The VMG contain both inspiratory and expiratory motorneurons. Stimulation of inspiratory motorneurons results in impulses being sent via phrenic nerves to activate inspiratory muscles. Activation of inspiratory muscles results in a
decrease in intrathoracic pressure below atmospheric pressure initiating deep inhalation. As a consequence of this deep ventilatory inspiration, intrathoracic pressure may increase by more than 100 mm Hg. The high intrathoracic pressure created by deep inspiration constricts blood vessels in the thorax, decreasing the volume of blood the vessels can hold and receive (Guyton, 1991).

The final major event resulting from stimulation of airway irritant receptors involves the integration of cardiac and pulmonary responses to suction catheter insertion (see Figure 4). Integration of cardiac and pulmonary neural responses occur at the medullary level of the central nervous system, where both the respiratory and vasomotor centers are located. The vasomotor center is responsible for the caliber of all blood vessels in the body (Guyton, 1991). Both the vasomotor center and the DMG receive impulses transmitted from baroreceptor activation in the aortic arch and carotid sinus via vagal and glossopharyngeal afferent fibers (Haxhiu et al., 1986; Jenson, 1980; Nakai & Ogino, 1984). Additionally, spillover of nerve signals via afferent fibers is known to occur between vasomotor and respiratory centers (Jenson, 1980; Guyton, 1991).

Under normal conditions vasomotor center neurons are activated by change in either systemic arterial pressure or blood volume resulting in either vasoconstriction or vasodilation. However, DMG activation by irritant receptors may also activate the vasomotor center by transmitting collateral afferent impulses to the vasomotor neurons. In this situation neurons in the DMG initiate the
vasopressor response, rather than a peripheral change in systemic blood pressure or volume. Increased vasomotor activity excites vasoconstrictor neurons and inhibits vagal neurons, resulting in increased peripheral vascular resistance, increased systemic arterial and venous pressure, increased heart rate and stroke volume (Guyton, 1991; Jenson, 1980).

Figure 4. Theoretical model of how airway irritant receptor activation initiates a change in normal cerebrovascular state.

As displayed in Figure 4, the three preceding responses to stimulation of airway irritant receptors are physiologic in nature, and pose little danger to healthy adults. Transitory cerebral vasodilation occurs in response to the reflex arc and retrograde venous pressure. Precapillary cerebral vessels (small arteries and arterioles) vasoconstrict in response to increased systemic pressure, thus protecting cerebrovascular bed from large pressure increases.
Effect of Suction Catheter Insertion in Head Injured Adults

Although the preceding responses to suction catheter insertion are normally benign, when superimposed upon the pathological changes resulting from severe head injury, they have the potential to extend cerebral injury. As discussed previously, severe head injury is frequently accompanied by impaired cerebral autoregulation, resulting in aberrations of both cerebral blood flow and volume. With severe head injury the transient vasodilation resulting from irritant receptor response is superimposed upon already dilated vessels. Because cerebral autoregulation is impaired or absent, the cerebral capacitance vessels (capillaries and veins) receive unattenuated systemic arterial pressure. Direct transmission of systemic pressure results in additional cerebral vasodilation and increased capillary pressure (Portnoy et al., 1982; Simard & Bellefleur, 1989). Although the three responses act almost simultaneously on the cerebral vascular bed, for simplicity of presentation, each response's impact on cerebrovascular status of head injured adults is discussed separately.

Irritant receptor reflex arc. Completion of the irritant receptor reflex arc results in bronchoconstriction. This response is mediated through parasympathetic nervous activity, long recognized to cause bronchoconstriction (Guyton, 1991; Levitsky, 1981; Jensen, 1980; Nadel & Barnes, 1984).

It is more speculative whether there is any concurrent parasympathetic vasodilatory effect on cerebral circulation. Perivascular cholinergic fibers
accompany cerebral vessels and innervate vessel adventitia and smooth muscle media layers (Owman, Andersson, Hanko, & Hardebo, 1984). The complex relationship between sympathetic and parasympathetic systems, and the inherent difficulty in initiating and measuring a pure parasympathetic response, has limited understanding of the parasympathetic system's role in cerebral circulation. Although ethical consideration of degree of risk precludes human subject investigation, animal studies offer some insight into this phenomenon.

In a study of rats where CBF was measured after electrical stimulation of the solitary nucleus, CBF increased throughout the brain (Nakai & Ogino, 1984). The researchers speculated that activation of efferent vagal fibers in the solitary nucleus initiated cardiopulmonary responses and activated ascending vagal fibers innervating cerebral blood vessels. Therefore, stimulation of motorneurons in the solitary nucleus resulted in simultaneous cardiopulmonary responses and cerebral vessel vasodilation.

Extrapolating from Nakai and Ogino's (1984) findings, stimulation of irritant receptors by suction catheter insertion may directly dilate cerebral vessels. As discussed previously, the nucleus tractus solitarius contains the DMG that integrates responses to irritant receptor stimulation. Therefore, irritant receptor activation by suction catheter insertion could dilate cerebral vessels, thus increasing cerebral blood flow and volume. In head injured patients who are vasodilated from impaired autoregulation, further cerebral vessel dilation would be
exhibited by increased MICP, ICPA, and ICP rounding.

**Cough reflex.** The second response to stimulation of irritant receptors is initiation of the cough reflex. Large increases in intrathoracic pressure accompany the cough reflex. Increased intrathoracic pressure compresses the relatively low pressure venous vessels in the thorax. Decreased venous capacity causes translocation of blood to the heart and systemic arteries, resulting in increased arterial pressure. Compression of venous thoracic vessels also impedes cerebral venous outflow, increasing cerebral blood volume in cerebral capacitance vessels. Again the resulting vasodilation would be evidenced by increased MICP, ICPA, and ICP rounding.

**Systemic vasomotor response.** The vasomotor response impacts upon cerebrovascular status through the systemic arterial circulation. Since derangement of cerebral autoregulation usually accompanies head injury, the cerebral vasculature cannot protect itself from surges in systemic arterial pressure. Transmission of increased systemic arterial pressure to the cerebrovascular bed is reflected in sudden marked increases in ICP occurring almost simultaneously or slightly later than MAP. Although ICP is elevated, the MAP increase prevents a fall in CPP. Finally, cerebral blood volume expansion from direct transfer of arterial pressure magnitude to the cerebrovascular bed would alter ICP configuration. ICP waveform configuration would become conspicuously peaked.

In summary, suction catheter insertion by activating airway irritant
receptors, affects head injured adults' cerebrovascular status through the following process. Catheter insertion into the trachea stimulates irritant receptors. Impulses are sent via the afferent vagus to the DMG. Motoneurons in the DMG send responses via efferent vagal fibers to respiratory muscle that initiates bronchoconstriction and cough. Simultaneously, activation of ascending efferent vagal fibers result in cerebral arterial vasodilation, as evidenced by increased MICP, ICPA, and ICP rounding. The cough reflex increases intrathoracic pressure and compresses thoracic veins. Blood is shunted to systemic arterial circulation resulting in increased MAP, diminished cerebral venous outflow, and increased cerebral vascular bed volume. The effect of these circulatory changes on cerebrovascular status are manifested by increased MICP, ICPA, and ICP rounding.

DMG motoneurons simultaneously activate vasomotor motoneurons. Sympathetic fibers are activated, resulting in increased peripheral vascular resistance exhibited by increased MAP and heart rate (HR). Dilation of cerebral vessels from a combination of activation of parasympathetic fibers, metabolic acidosis, and decreased venous outflow leave the cerebral vascular bed vulnerable to increased systemic arterial pressure. Together the three irritant receptor responses would be expected to promote decreased compliance through intracranial hypertension, increased cerebral blood volume, and cerebral edema each time a suction catheter is introduced into the trachea.
The current study’s variables, empirical indicators and relationships of interest are displayed in Figure 5.

![Diagram of relationships among study variables]

**Figure 5.** Research model of the relationships among study variables.

**Specific Aims, Research Questions and Hypotheses**

Based on the theoretical and research models posited for this study, the following specific aims, research questions, and hypotheses were tested in a sample of severely head injured adults.

SA1. To determine the effect of suction catheter insertion on cerebrovascular status.

RQ1a. Is there a difference between cerebrovascular response (MICP and CPP) and vasopressor response (MAP and HR) during suction catheter insertion
compared to baseline values?

H1a. There will be an increase in MICP during suction catheter insertion compared to MICP at baseline.

H1b. There will be an increase in CPP during suction catheter suction insertion compared to CPP at baseline.

H1c. There will be an increase in MAP during suction catheter insertion compared MAP at baseline

H1d. There will be an increase in HR during suction catheter insertion compared to HR at baseline.

SA2. To compare the difference between cerebrovascular and vasopressor responses during suction catheter insertion and during actual suctioning.

RQ2. Is there a difference in cerebrovascular response (MICP and CPP) and vasopressor response (MAP and HR) during actual suctioning compared to suction catheter insertion?

H2a. There will be an increase in MICP during actual suctioning compared to MICP during suction catheter insertion.

H2b. There will be an increase in CPP during actual suctioning compared to CPP during suction catheter suction insertion.

H2c. There will be an increase in MAP during actual suctioning compared to MAP during suction catheter insertion.

H2d. There will be an increase in HR during actual suctioning compared to
HR during suction catheter insertion.

SA3. To determine the effect of state of compliance before catheter insertion on cerebrovascular response during suction catheter insertion.

RQ3. Is there a difference in cerebrovascular response (MICP) during suction catheter insertion between subjects with decreased cerebral compliance (ICP rounding) and those with normal compliance (Normal ICP configuration)?

H3a. There will be a greater increase in MICP during suction catheter insertion between subjects with decreased cerebral compliance compared to those with normal compliance.

RQ4. Do differences between cerebrovascular response and vasopressor response during suction catheter insertion and baseline hold when duration of suction catheter insertion is controlled?

H4a. There will be no difference between MICP during suction catheter insertion compared to MICP at baseline.

H4b. There will be no difference between CPP during suction catheter suction insertion compared to CPP at baseline.

H4c. There will be no difference between MAP during suction catheter insertion compared MAP at baseline

H4d. There will be no difference between HR during suction catheter insertion compared to HR at baseline.

RQ5. Do differences between cerebrovascular response and vasopressor
response during suction catheter insertion and actual suctioning hold when duration of suction catheter insertion is controlled?

H5a. There will be no difference between MICP during suction catheter insertion compared to MICP during actual suctioning.

H5b. There will be no difference between CPP during suction catheter suction insertion compared to CPP during actual suctioning.

H5c. There will be no difference between MAP during suction catheter insertion compared MAP during actual suctioning.

H5d. There will be no difference between HR during suction catheter insertion compared to HR during actual suctioning.

Definitions

The following definitions of variables and their empirical indicators were used for the current study.

Independent variables and empirical indicators

Cerebral compliance. Compliance is an expression of intracranial volume-pressure relationship. Normal compliance is the ability of the intracranial space to accommodate increased intracranial volume without a reciprocal increase in intracranial pressure. Cerebral compliance was measured by the empirical indicators, **ICP waveform configuration**, ICP amplitude and ICP slope.

ICP configuration. ICP waveform shape or pattern (see Chapter 3, Instrumentation and Appendices A and B).
Intracranial pressure amplitude (ICPA). The height or excursion of an ICP wave, from highest systolic pressure to lowest diastolic pressure (see Chapter 3, Instrumentation).

Intracranial pressure wave slope. The ratio between the change in ICP wave height and the change in time. The descending slope of the ICP wave is calculated by the formula: \( Y_2 - Y_1 \) (ICP mm Hg.) \( / \) \( X_2 - X_1 \) (seconds) = ICP mm Hg. per second. Because the ICP wave descending slope were assessed, the resulting slope values were expressed as negative values.

Irritant receptor stimulation. Mechanical stimulation of sensory receptors in the proximal airways. Irritant receptor activation was initiated by suction catheter insertion.

Suction catheter insertion. Insertion of a suction catheter into the trachea via an endotracheal tube, until the catheter cannot be advanced further, due to airway resistance or the tube being maximally advanced into the trachea.

Dependent variables and empirical indicators

Vasopressor response. Activation of vasoconstrictor motorneurons in the vasomotor center that initiate systemic increased peripheral vascular resistance and increased heart rate. The pressor response was assessed using mean arterial pressure and heart rate.

Mean arterial pressure (MAP). The average pressure in mm Hg. of the systemic arterial vascular system that delivers oxygenated blood to tissues.
Normal MAP ranges from 70 to 105 mm Hg. Average arterial pressure = 2 X diastolic + systolic pressure/3 (see Chapter 3, Instrumentation).

Heart rate (HR). The rate of heart contractions over a 60 second time period, counted as the number of R waves per minute. Normal heart rate ranges from 60 to 100 beats per minute (see Chapter 3, Instrumentation).

Cerebrovascular response. A stimulus induced transient or sustained increase in volume any of the three intracranial compartments. Cerebrovascular response was measured by MICP and CPP.

MICP. The average pressure throughout the intracranial space assessed by indirect or direct measurement of intracranial cerebrospinal fluid pressure in mm Hg. Normal MICP ranges from 0 to 10 mm Hg. Average ICP = 2 X diastolic + systolic pressure/3 (see Chapter 3, Instrumentation).

CPP. The pressure gradient between cerebral arteries and veins required for the delivery of adequate substrate to cerebral cells to carry on aerobic metabolism. Normal CPP ranges from 70 to 130 mm Hg. CPP = MAP - MICP (see Chapter 3, Instrumentation).
Terms

Endotracheal suctioning sequence (ETS sequence). A procedure designed to remove mucous and debris from the airways and endotracheal tube that consists of several components. For the purposes of this study ETS included the following steps in order of occurrence: (a) Prior to actual suctioning oxygenate subject by delivering several tidal volume breaths of 100% oxygen, (b) disconnect subject’s endotracheal tube from the mechanical ventilator, (c) insert suction catheter into endotracheal tube, advancing the catheter until resistance is met or full length of catheter is inserted, and (d) apply negative suction pressure while slowly withdrawing suction catheter from endotracheal tube in a rotating manner.

Actual suctioning. The application of negative airflow to remove mucous and debris from the airways and endotracheal tube. For this study, negative airflow was applied continuously at 16L/minute for 10 seconds through a number 14 Fr polyethylene catheter (Pharmaseal), while the catheter was slowly rotated.

Severe head injury. Adults with trauma-based open or closed injury to the head, who are in deep coma (GCS score \( \leq 8 \)), in the acute phase of their injury, and who require intensive nursing care.
CHAPTER TWO

Literature Review

The major conceptual relationships that were tested form the organization for the review of literature. As explicated in Chapter One, endotracheal suctioning was presumed to initiate a cerebrovascular response by airway stimulation from suction catheter insertion. Additionally, it was proposed that in severely head injured adults, the degree of cerebrovascular response to airway stimulation depended upon the state of cerebral compliance.

Based upon the preceding relational statements, the first section of the review focuses upon literature that addressed cerebrovascular response initiated by endotracheal suctioning. The review is structured by the way past investigations operationalized cerebrovascular response. Each operational variable is analyzed within the context of the information it provides about cerebrovascular response to ETS. Integrative findings produced by the various measurements of cerebrovascular response are presented. Where appropriate, individual study findings are also discussed. Methodological weaknesses that limit the usefulness of study findings are also included in the analysis.

The second section of the review focuses on research that specifically addressed the effect of ETS airway stimulation on cerebrovascular status in head injured persons. Literature that provided a physiological explanation for how airway stimulation may initiate a cerebrovascular response is reviewed. The final

35
section focuses on the relationship between cerebral compliance and cerebrovascular response to airway stimulation.

**Cerebrovascular Response to Endotracheal Suctioning**

A total of 13 studies were found that investigated the effect of ETS on cerebrovascular status. The population of interest was adults with head injury. Because of the relatively small number of studies, the review included investigations of children and adults with any cranial pathology that could potentially alter intracranial volume/pressure dynamics. Four of the 13 studies were descriptive in design (Boortz-Marx, 1985; Mitchell & Mauss, 1978; Shalit & Umansky, 1977; Snyder, 1983) while the remainder used quasi-experimental designs. Inferential testing in five of the quasi-experimental studies did not focus on the central issue of cerebrovascular response to ETS but still provided findings of importance to this issue (Donegan & Bedford, 1982; Fisher et al., 1982; Tsementzis et al., 1982; White et al., 1982; Yano et al., 1982). The remaining four quasi-experimental studies directly addressed the question of how ETS affects cerebrovascular status (Campbell, 1989; Ersson et al., 1990; Parsons & Shogun, 1984; Rady et al., 1991).

**Operationalization of Cerebrovascular Response to ETS**

Several studies investigated the effect of ETS on head injured patients' cerebrovascular status using ICP as a single intracranial indicator (see Table 1). The use of ICP as sole indicator of cerebrovascular response was the approach
Table 1

Studies that Used Intracranial Pressure as a Single Measure of Cerebrovascular Response to ETS

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Sample</th>
<th>Design/Purpose</th>
<th>Baseline ICP</th>
<th>Response to ETS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitchell &amp; Mauss, 1978</td>
<td>N = 9 subjects with varied cranial pathology, unreported coma depth. ETS cases = 25 in 3 subjects.</td>
<td>Descriptive. Determine what activities are associated with increased ICP.</td>
<td>None. Measured ventricular fluid drainage.</td>
<td>3 of 25 ETS events resulted in ventricular fluid drainage.</td>
</tr>
<tr>
<td>Shalit &amp; Umansky, 1977</td>
<td>N = 21 adults with varied cranial pathology, unreported coma depth. ETS cases = 28</td>
<td>Descriptive. Evaluate effect of routine bedside procedures on cerebrovascular status</td>
<td>1 case study with baseline = 20 mm Hg.</td>
<td>Case study during and after ETS = 55 mm Hg.</td>
</tr>
<tr>
<td>Snyder, 1982</td>
<td>N = 9 adults with varied cranial pathology, unreported coma severity. Respiratory care cases = 89</td>
<td>Descriptive. Evaluate effect of nursing activities on cerebrovascular status.</td>
<td>None reported.</td>
<td>None reported. Incidence of ICP $\geq$ 20 mm Hg. during &amp; after resp. care = 17 (10%).</td>
</tr>
</tbody>
</table>

(table continues)
Table 1

Studies that Used Intracranial Pressure as a Single Measure
Cerebrovascular Response to ETS

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Sample</th>
<th>Design/Purpose</th>
<th>Baseline ICP</th>
<th>Response to ETS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boortz-Marx, 1985</td>
<td>N = 4 adults with severe head injury. ETS cases = 92.</td>
<td>Descriptive. Evaluate effect of stimuli on cerebrovascular status.</td>
<td>None reported.</td>
<td>R = 1 to 42 mm Hg. M = 10.7 mm Hg.</td>
</tr>
<tr>
<td>Yano, Nishiyama, Yokota, Kato, Yamamoto, &amp; Otsuka, 1986</td>
<td>N = 9 adults with severe head injury</td>
<td>Quasi-experimental. Compare effect of IT vs. IV lidocaine on cerebrovascular status</td>
<td>M = 17.5 mm Hg. (both groups)</td>
<td>R peak values = 27 to 48 mm Hg. M = none reported.</td>
</tr>
</tbody>
</table>

Note. IT = intratracheal, IV = intravenous.
first used to assess head injured adults' cerebrovascular responses to ETS. For the present study, cerebrovascular response was defined as a stimulus induced transient or sustained increase in volume of any of the three intracranial compartments that was measured by a change in MICP and CPP.

**Ventricular fluid pressure.** In the first nursing study to investigate the effect of ETS on intracranial status, Mitchell & Mauss (1978) operationalized cerebrovascular response to ETS as the quantity of ventricular fluid drainage that dripped from a ventricular catheter into an extracranial reservoir. Quantity of ventricular fluid drainage was an indirect measure of intracranial pressure. The unit of analysis was the individual suctioning event in this descriptive study of 9 subjects.

Results from this study showed that of the 25 separate suctioning events in 3 subjects, only three events precipitated ventricular fluid drainage. Nineteen of the 25 suctioning events with no increase in ventricular drainage were completed in one subject who was hyperventilated with 100% oxygen before suctioning. Based upon the lack of response in this single subject, the researchers concluded that hyperoxygenation before ETS was an effective means to prevent increased ICP during ETS. The major weakness in this study was the use of ventricular fluid drainage as a measure of ICP response to ETS. The height of the extracranial reservoir was determined by the subject’s physician, independent of the study design. Because reservoir height was not controlled, absence of
ventricular drainage in response to ETS may have been a function of reservoir height rather than an absence of ICP response to ETS.

The Mitchell and Mauss study had a critical impact upon nursing research and practice in the neurotrauma population. The conclusion that increased ICP during ETS was prevented by presuctioning hyperventilation with high oxygen concentration, was accepted by nurse researchers, and remains a basic assumption in this area of nursing research (Boortz-Marx, 1985; Parsons & Shogun, 1984; Snyder, 1982). In part, because the hypoxemia hypothesis has not been questioned, nurse researchers have followed a path of research that has failed to identify effective ways to control ICP response to ETS.

Only recently was this assumption challenged when Rudy and associates (1991) demonstrated that high arterial oxygen saturation was not sufficient to prevent increased ICP in response to ETS (see the discussion of hypoxemia in Chapter One of this proposal). Building upon the findings of Rudy and colleagues, the present study tested an alternative explanation for increased ICP response to ETS that offers a different basis for developing nursing interventions to blunt this dangerous cerebrovascular response.

Intracranial pressure. As intracranial pressure monitoring became common clinical practice, researchers began to use ICP derived from cerebrospinal fluid pulse waves as a single indicator of cerebrovascular response to ETS (Boortz-Marx, 1985; Donegan & Bedford, 1982; Shalit & Umansky, 1977; Snyder, 1983;
White et al., 1982; Yano et al., 1982). Intracranial pressure results from the transmission of systemic arterial pressure through the cerebral vascular bed to the thin-walled pial veins. The venous pulsations are transmitted to the cerebral spinal fluid (Pornoy et al., 1982). Cerebral vasomotor tone, retrograde systemic venous pressure, cerebral tissue pressure, and cerebral spinal fluid pressure modify arterial pressure as it travels to the cerebral veins. As a measure of cerebral venous pressure, ICP represents the end product of all the preceding pressure modifiers. Although ICP is a global measure of pressure in the intracranial space, it provides a means to assess the effects of intermittent nursing procedures such as ETS, on cerebrovascular status.

Magnitude dimension of ICP response to ETS. MICP values of 10 mm Hg., 15 mm Hg., and 20 mm Hg. have been identified as the pressure levels where ICP threatens cerebral integrity (Eisenberg, 1986; Marshall, Smith, & Shapiro, 1979; Marshall, 1986; Miller, Becker, Ward, 1977; Obrist et al., 1984; Saul & Ducker, 1982; Saul, 1986). The discrepancy about what constitutes intracranial hypertension seems to be based more upon treatment philosophy than firm empirical guidelines. Clinical investigations that identified intracranial hypertension as MICP values of more than 20 mm Hg., focused upon how aggressive therapy affected outcomes in populations with sustained MICP elevations above 20 mm Hg. Researchers who identified lower ICP levels as the criteria for intracranial hypertension, focused upon whether aggressive therapy to
prevent higher ICP levels could improve outcomes.

Findings from studies that used ICP as the sole indicator of cerebrovascular response to ETS, were evaluated using the preceding definitions for intracranial hypertension (see Table 1). Of the studies in Table 1, only Yano and colleagues (1986) reported a baseline MICP values (M = 18 mm Hg.). ICP response to ETS ranged from 1 to 55 mm Hg. across all of the studies. Because of the variability in suctioning procedures, integrated findings must be interpreted with caution. Regardless of procedural differences, however, the findings offer information about ETS effect on cerebrovascular status. All studies demonstrated that ETS precipitated intracranial hypertension. Three of the four studies demonstrated that ETS precipitated MICP values far in excess of highest criteria for intracranial hypertension (20 mm Hg.) the was found in the literature.

In the study reported by Yano and associates (1986), subjects' ICP response to ETS ranged from 27 to 48 mm Hg., regardless of the fact that lidocaine had been administered to blunt the ICP response. Thus, regardless of the administration of a medication believed to attenuate cerebrovascular response to ETS, ICP increased to levels that have the potential to extend cerebral damage.

The preceding studies confirmed the widely known clinical observation that ICP increased during ETS. Unfortunately the studies offered limited knowledge to guide clinical nursing practice. None of the studies controlled factors of the actual ETS procedure such as negative suction pressure level, duration of
suctioning, presuctioning oxygenation levels, or hyperventilation maneuvers that would impact upon findings. Additionally, three of the studies used samples that had varied craniocerebral pathology and unknown levels of depth of coma (Mitchell & Mauss, 1978; Shalit & Umansky, 1977; Snyder, 1982). All the preceding factors have the potential to affect patient's ICP response to ETS. The studies demonstrated a need for better controlled studies with additional measures to provide more information about intracranial dynamics.

Multiple measures of cerebrovascular response. Although measurement of ICP offered significant information about head injured patients' transient intracranial responses to ETS, the complexity of intracranial dynamics dictated expanded measures to better assess cerebrovascular response. ICP as a single indicator did not provide enough information about cerebrovascular status to enable investigators and clinicians to identify patients who might develop malignant intracranial hypertension, or those who would experience poor outcomes. To better assess cerebrovascular response to ETS, researchers added systemic arterial pressure (MAP), heart rate (HR) and cerebral perfusion pressure (CPP) (Campbell, 1989; Donegan & Bedford, 1980; Ersson et al., 1990; Fisher et al., 1982; Parsons & Shogun, 1984; Rudy et al., 1991; Tsementis, et al., 1982; White et al., 1982). Findings from studies that used multiple measures to assess cerebrovascular status are summarized in Table 2.
Table 2

**Studies Using Multiple Measures to Assess Cerebrovascular Response to ETS**

<table>
<thead>
<tr>
<th>Authors/Year</th>
<th>Sample</th>
<th>Design/Purpose</th>
<th>Baseline Values</th>
<th>Response to ETS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parsons &amp; Shogun, 1984</td>
<td>N = 20 Children and adults with moderate to severe head injury.</td>
<td>Quasi-experimental Evaluate effect of ETS/hyper-ventilation on cerebrovascular status.</td>
<td>ICP: M = 5.5 mm Hg. MAP: M = 86 mm Hg. CPP: M = 80 mm Hg. HR: M = 100 BPM.</td>
<td>ICP: M = 10 mm Hg. MAP: M = 103 mm Hg. CPP: M = 91 mm Hg. HR: M = 121 BPM.</td>
</tr>
<tr>
<td>Campbell, 1989</td>
<td>N = 10 adults with severe head injury.</td>
<td>Quasi-experimental. Evaluate ETS/hyper-inflation effect on cerebrovascular status.</td>
<td>ICP: R = 4 to 5 mm Hg. M = 4.8 mm Hg. MAP: R = 95 to 99 mm Hg. M = 97 mm Hg. CPP: R = 90 to 94 mm Hg. M = 92 mm Hg. HR: R = 87 to 88 BPM M = 88 BPM</td>
<td>ICP: R 16 to 20 mm Hg. M = 16.2 mm Hg. MAP: R 107 to 124 M = 116 mm Hg. CPP: R 91 to 106 M = 100 mm Hg. HR: R 109 to 126 M = 119</td>
</tr>
</tbody>
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<tr>
<td>Ersson, Carlson, Mellstrom, Ponten, Hedstrand &amp; Jakobsson, 1990</td>
<td>N = 12 adults with severe head injury.</td>
<td>Quasi-experimental Compare effect of ETS vs. vent bag squeezing on cerebrovascular status.</td>
<td>ICP: R = 0 to 30 mm Hg, M = 9.7 mm Hg. MAP: Anecdotal only, CPP: R = 42 to 127 mm Hg, M = 80 mm Hg.</td>
<td>ICP: M = 34 mm Hg. MAP: Anecdotal</td>
</tr>
<tr>
<td>Rudy, Turner, Baun, Stone &amp; Brucia, 1991</td>
<td>N = 30 Cases = 60 adults with severe head injury</td>
<td>Quasi-experimental Evaluate effect of number of suction passes and size of hyperinflation breaths on cerebrovascular status</td>
<td>ICP: R = 0 to 53, M = 15.5, MAP: R = 58 to 158 mm Hg, M = 103 mm Hg. CPP: R = 51 to 138, M = 87 mm Hg. HR: R = 60 to 126, M = 93 BPM</td>
<td>ICP: R = 0 to 63, M = 21.3, MAP: R = 58 to 232, M = 118 mm Hg. CPP: R = 49 to 174, M = 93 mm Hg. HR: R = 48 to 220, M = 108 BPM</td>
</tr>
</tbody>
</table>

*(table continues)*
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<tbody>
<tr>
<td>Tsementis, Harris &amp; Loizou, 1982</td>
<td>N = 39 Children and adults with severe head injury. Group 1 = 33 cases. Minimal response to ETS. Response controlled by medication. Group 2 = 6 cases. M = ICP response &gt; 20 mm Hg. Not responsive to meds.</td>
<td>Quasi-experimental. Evaluate routine procedures effect on cerebrovascular status.</td>
<td>ICP: R = 11 to 52 mm Hg. MAP: Anecdotal data.</td>
<td>ICP: R = 31.4 to 40.5 mm Hg. M = 35.4 mm Hg. MAP: Anecdotal data.</td>
</tr>
<tr>
<td>Fisher, Frewen &amp; Swedlow, 1982</td>
<td>N = 9 children with varied cranial pathology and unknown severity of coma.</td>
<td>Quasi-experimental. Compare ETS vs. apnea effect on cerebrovascular status.</td>
<td>ICP: M = 12 mm Hg. MAP: M = 79 mm Hg. CPP: M = 67 mm Hg.</td>
<td>ICP: M = 18 mm Hg. MAP: M = 89 mm Hg. CPP: M = 72 mm Hg.</td>
</tr>
</tbody>
</table>

*(table continues)*
Table 2

**Studies Using Multiple Measures to Assess Cerebrovascular Response to ETS**

<table>
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</table>
| Donegan & Bedford, 1980 | N = 10 adults with intracranial tumors. | Quasi-experimental, Compare IV vs. IT lidocaine effect on cerebrovascular response to ETS. | ICP: $M = 18$ mm Hg.  
MAP: $M = 87$ mm Hg. | ICP: $M = 25$ mm Hg.  
MAP: $M = 99$ mm Hg. |
| White, Schlobohm Pitts, & Lindauer, 1982 | N = 15 adults with severe head injury. | Quasi-experimental, Compare medication effect on cerebrovascular response to ETS. | ICP: $M = 15$ mm Hg. (all groups)  
MAP: $M = 101$ mm Hg. (all groups). | ICP: Peak response $R = 19$ to $37$ mm Hg.  
M = 29 mm Hg.  
MAP: Peak response $R = 121$ to $130$ mm Hg.  
M = 125 mm Hg. |

*Note.* IV = intravenous, IT = intratracheal.
Magnitude dimension of ICP response to ETS. Like studies that used ICP as the only indicator for cerebrovascular response, investigations with multiple measures also demonstrated increased MICP in response to ETS (see Table 2). In contrast to the single indicator studies, the studies in Table 2 assessed baseline ICP. Baseline ICP across all studies in Table 2 averaged 14 mm Hg. with a 0 to 53 mm Hg. range. Average ICP response during suctioning was 24 mm Hg. with a 0 to 63 mm Hg. range. Average MICP during suctioning included mean values from every suction pass, thus a study with multiple suction passes would have several values incorporated.

Studies with repeated suction passes consistently demonstrated a cumulative ICP increase with each sequential suction pass (Campbell, 1989; Parsons & Shogun, 1984; Rudy, et al., 1991). Of these studies, the Parsons and Shogun study was of particular interest. Subjects demonstrated a small but cumulative MICP increase in response to ETS, although they were hyperventilated between each successive pass. This finding suggests that the mechanism responsible for ICP increase during ETS is powerful enough to exceed the vasoconstrictive effects of induced hypocapnia.

Although it has long been known that ETS increases ICP, until recently it was believed that the increases were of a transient and therefore, benign nature. Findings from more recent studies are increasingly showing that ICP response to ETS is not as transient as once believed.
Temporal dimension of ICP response to ETS. ETS causes a transient increase in intracranial pressure (Boortz-Marx, 1985; Campbell, 1989; Donegan & Bedford, 1982; Ersson et al., 1990; Fisher et al., 1982; Parsons & Shogun, 1984; Rudy et al., 1991; Shalit & Umansky, 1977; Snyder, 1983; Tsementzis et al., 1982; White et al., 1982; Yano et al., 1982). Transient increased ICP in response to stimuli such as ETS is an expected physiological response. When superimposed upon known pathological changes associated with head injury, such as tissue damage, altered cerebral blood flow, impaired autoregulation, and brain swelling, transient ICP increase may extend tissue damage. It is reasonable, therefore, that any sustained ICP increase resulting from ETS poses an even greater risk of cerebral damage to patients with head injury.

An arbitrary definition of transient and sustained ICP response was derived from the 12 studies included in this review. Transient ICP response to ETS was an ICP increase that occurred during the procedure and lasted less than one minute after suctioning. A sustained ICP response was an increase in ICP that persisted for greater than one minute after suctioning.

Six of the 12 studies measured ICP response for a least one minute after the procedure (Campbell, 1989; Ersson et al., 1990; Parsons & Shogun, 1984; Rudy et al., 1991; Shalit & Umansky, 1977; Snyder, 1983). Parsons and Shogun (1984) measured ICP at 1 minute after the end of the ETS procedure. The researchers found that ICP returned to baseline by one minute after ETS. Because
ICP response to ETS was transient and of small magnitude it was concluded that ETS posed minimal risk to head injured patients.

In contrast to the preceding findings, investigations where ICP was measured for longer than one minute after cessation of suctioning found ICP elevated above baseline values (Campbell, 1989; Ersson et al., 1990; Rudy et al., 1991; Shalit & Umansky, 1977; Snyder, 1983). Two studies that measured MICP for 10 minutes after ETS, found in most subjects ICP did not return to baseline until 8 to 10 minutes after suctioning (Campbell, 1989; Rudy et al., 1991). Ersson and colleagues (1990), who measured ICP for 15 minutes after ETS, found that subjects’ ICP had not returned to baseline by 15 minutes after cessation of the procedure. Sustained ICP increase in response to ETS was corroborated by descriptive studies where increased ICP from ETS lasted from 3 to 15 minutes after suctioning (Shalit & Umansky, 1977; Snyder, 1983).

Differences in instrumentation and procedure may account for the disparate findings between the Parsons and Shogun (1984) study and the other studies. Parsons and Shogun used nonparticipant observers to record digital data from bedside monitors at a one minute point in time, while other researchers used retrievable continuous real-time recordings over extended time periods (Campbell, 1989; Errson et al., 1990; Rudy et al., 1991). Data for a specific time point can be more accurately assessed using continuous collection techniques.

Another possible explanation for differences in findings is that Parsons and
Shogun (1984) hyperventilated subjects for at least 30 seconds after each suction pass. It was assumed that hyperventilation vasoconstricted cerebral vessels and decreased ICP. Because ICP was measured at only one minute after suctioning, the investigators failed to capture increased ICP when subjects were returned to presuctioning ventilatory patterns.

The combination of markedly elevated ICP for prolonged periods of time emphasizes that ETS is not a benign procedure. However, the mechanisms through which ETS initiates this potentially dangerous cerebrovascular response are not well understood. Because ICP increase is cumulative with repeated suctioning maneuvers, the most effective intervention would be to prevent or blunt the initial cerebrovascular response to suctioning. The current study focused on the initial cerebrovascular response to ETS, thus laying the foundation for interventions to prevent ICP increase.

Systemic arterial pressure and cerebral perfusion pressure. Since the early 1980s, measurement of systemic arterial pressure (MAP) was routinely incorporated in the study of how ETS effects cerebrovascular status in head injured patients (Campbell, 1989; Donegan & Bedford, 1980; Ersson et al., 1990, Fisher et al., 1982; Parsons & Shogun, 1984; Rudy et al., 1991; Tsemritis, et al., 1982; White et al., 1982). The utility of measuring MAP is that cerebrovascular pressure is inherently dependent upon systemic arterial pressure. Systemic arterial pressure as a measure of cerebrovascular status has traditionally
been used to assess cerebral perfusion pressure. All studies included in this
review measured systemic pressure to assess cerebral perfusion pressure
(Campbell, 1989; Ersson et al., 1990; Fisher et al., 1982; Parsons & Shogun,
1984; Rudy et al., 1991; White et al., 1982).

Cerebral perfusion pressure is the pressure required to carry adequate
Early studies of cerebral perfusion pressure identified CPP levels in the range of
40 to 50 mm Hg. as the critical lower level of CPP where autoregulation fails and
cerebral vessels become maximally dilated. If the CPP remains at this low level,
cerebral ischemia will ultimately result in neuronal death (Grubb, 1975; Miller et
al, 1971; Tsutsumi et al., 1986). To avoid cellular damage, CPP levels of 70 mm
Hg. or greater are needed to promote survival and decreased morbidity. Rosner
and colleagues demonstrated that CPP levels above 70 mm Hg. prevented large
sustained increased ICP in head injured patients (Rosner & Becker, 1984; Rosner

At the other extreme, excessive CPP threatens cerebral integrity through
capillary damage, blood-brain barrier disruption, and increased cerebral edema
(Rosner & Becker, 1984; Shapiro, 1975; Simard & Bellefleur, 1989; Youmans,
1982). Although the dangers of elevated CPP are known, an upper CPP limit is
rarely specified. Only two literature citations of an upper CPP limit of 130 mm
Hg. were found (Langfitt, 1982; McGinnis, 1988).
There is disagreement about whether CPP provides useful information about cerebrovascular status when ICP is elevated. The disparity stems from two perspectives about how CPP is derived, and how it relates to cerebral blood flow during increased ICP. Increased ICP modifies cerebral vascular resistance, thus altering cerebral blood flow (CBF). A change in CBF ultimately affects the efficiency of tissue perfusion. If CPP reflects cerebral tissue perfusion, then it provides important information about cerebrovascular status.

CPP from one perspective, is defined as the difference between carotid/vertebral artery pressure (inflow pressure) and jugular bulb pressure (outflow pressure). Because CPP is not derived from pressures within the cranial vault, it does not provide useful information about the relationship between cerebral blood flow and tissue perfusion when ICP is elevated (Langfitt, 1982; Miller et al., 1974).

Another school of thought defines CPP as the difference between systemic arterial pressure and cerebral venous pressure. ICP and cerebral venous pressure are normally equivalent. ICP is therefore, substituted for venous pressure, and CPP is calculated as the difference between MAP and ICP. From this perspective, CPP represents the pressure gradient between cerebral arterial and venous systems, thus provides a measure of cerebral tissue perfusion. CPP is conceptualized as directly proportional to CBF (Cruz, 1988).

Studies that measured CBF in patients with elevated ICP found no
consistent relationship between CPP and CBF (Cruz, 1988; Feischi et al., 1974; Obrist et al., 1984; Overgaard & Tweed, 1974). The failure to find a consistent relationship between CPP and CBF during intracranial hypertension, may reflect CBF's greater dependence on cerebral metabolism than on level of ICP (Jennett & Teasdale, 1981). In people with head injury, CBF varies regionally depending upon local metabolic need and the degree of local tissue injury (Cold et al., 1981; Enevoldsen & Jensen, 1978; Feischi et al., 1974). Because CPP reflects global pressure, it serves only as a general indicator for actual cerebral tissue perfusion in the head injured person. Because neither state of cerebral vascular resistance nor cerebral metabolic state can be inferred from CPP, it may offer minimal information about cerebral tissue perfusion (Cruz, 1988).

CPP response to ETS. As displayed in Table 2, CPP increased in response to ETS in each of the six studies that used CPP as a measure of cerebrovascular response (Campbell, 1989; Ersson et al., 1990; Fisher et al., 1982; Parsons & Shogun, 1984; Rudy et al., 1991; White et al., 1982). CPP increased in response to ETS regardless of the number of suction passes, the frequency, or volume of preoxygenation breaths (Campbell, 1989; Parsons & Shogun, 1984; Rudy et al., 1991) or medications received (White et al., 1982).

Baseline CPP values across five studies \(^1\) ranged from 51 to 138 mm Hg.

\(^1\)White and colleagues (1982) only reported CPP change values, thus were excluded from the integrative analysis.
with an 80 mm Hg. average. During suctioning, CPP averaged 87 mm Hg. and had 42 to 174 mm Hg. range. Although average CPP was not compromised by ETS, individual subjects experienced excessively high or low CPP values during the suctioning procedure. In one study, 4 cases had CPP greater than 130 mm Hg. at baseline measurement, and 13 incidents of excessive CPP were observed during ETS (Rudy et al., 1991).

Discussion of excessively large CPP values is seldom found in the medical literature, because with the exception of hypertensive encephalopathy, there are few natural or iatrogenic conditions that elevate CPP (Rosner & Becker, 1984). As demonstrated by the findings of Rudy and associates (see previous discussion), ETS can precipitate excessively elevated CPP, and therefore, may place patients at risk during this routine nursing procedure.

Because elevated CPP is directly dependent upon systemic arterial pressure, calculation of CPP may not provide any information not already available from MAP measurement. When both MAP and ICP are elevated CPP may be normal, although cerebral tissue may be severely compromised. This situation known false autoregulation, was identified by several researchers (Cold et al., 1981; Enevoldsen & Jensen, 1978; Simard & Bellefleur, 1989). Additionally, because cerebrovascular resistance and cerebral metabolic state cannot be inferred from CPP, it cannot be concluded from CPP alone that ETS precipitates hyperemia.
Although CPP may not provide definite information about cerebral tissue perfusion, it does offer general information about the relationship between systemic and cerebral vascular pressures. Additionally, CPP provides an estimate of whether cerebral perfusion pressure response to ETS is outside the accepted normal range. CPP was used as a measure of cerebrovascular response to ETS in the present study to compare findings with those of previous studies.

**Systemic arterial pressure and systemic vasopressor response.** Systemic arterial pressure typically has been used to assess CPP in previous studies of ETS in the head injured population. Systemic arterial pressure as an independent measure however, offers important information about cerebrovascular response to ETS.

Level of systemic arterial pressure is related to cerebral autoregulatory control of cerebral blood flow. Cerebral autoregulation is the intrinsic ability of the cerebral vascular bed to maintain a constant CBF over a wide range of systemic blood pressure (Berne, Winn, Rubio, 1981; Paulson, Strangaard, & Edvinsson, 1990; Youmans, 1982). Normal CBF in human adults is approximately 50 ml/100 gm/minute (Youmans, 1982).

Although there is general acceptance for the existence of cerebral autoregulation, the basic mechanisms of CBF control remain controversial. Four theories; the myogenic theory, tissue pressure theory, neurogenic theory, and the metabolic theory seek to explain underlying mechanisms of autoregulation (Berne
et al., 1981; Langfitt, 1982; Miller et al., 1971; Owman et al., 1984; Paulson et al., 1990; Youmans, 1982).

Of the preceding theories the myogenic theory is accepted as the probable basis for cerebral autoregulatory control. Myogenic theory is based upon the work of Bayliss (1902) who hypothesized that a pressure dependent basal myogenic tone exists in the resistance vessels of all vascular beds. Myogenic based autoregulation arises from smooth muscle in precapillary resistance vessels that are responsive to transmural pressure change. Myogenic response was recently identified in both small cerebral arteries and arterioles, although responses to pressure change may vary from arteries to arterioles (Baumbach & Heistad, 1983; Harper, Bolen & Rubin, 1984; Tamaki, Mayhan & Heistad, 1986). An intrinsic myogenic mechanism also is favored because cerebral vessels constrict or dilate within a few seconds after transmural pressure change occurs. An externally mediated vessel response would be less likely to respond as rapidly (Kontos et al., 1978).

Tissue pressure theory of cerebral autoregulation is based upon the concept of fluid filtration believed to occur at the capillary level in response to change in arterial pressure (Rodbard, 1971). Increased or decreased fluid filtration is posited to return CBF to normal after an arterial pressure change. Tissue pressure theory fails to explain at least two important issues in the maintenance of normal CBF. First, the tissue pressure mechanism would be too slow to allow the rapid
vessel response to arterial pressure change that is known to occur. Second, the theory is contradicted by the fact that during initial increases in intracranial pressure, CBF is usually maintained at normal levels.

Theories of a neurogenic mechanism for CBF autoregulation are based upon the knowledge that there is an extensive system of perivascular nerve fibers that appear to primarily affect inflow at the large cerebral and pial arteries (Owman et al., 1984). Additionally, nerve fibers of intracerebral origin that accompany intracerebral vessels have been identified, although their function at present is unknown (Edvinsson, Lacombe, Nielsen, & Owman, 1973). Finally, it is known that there are vasoactive neurogenic substances secreted by the endothelial cells that may mediate cerebral autoregulation (Paulson et al., 1990; Rubanyi, Romero, & Vanhoutte, 1986). While neurogenic mechanisms appear to play an important role in cerebral autoregulation, they seem to moderate, rather than control CBF and autoregulation.

The metabolic theory focuses on local CBF control. It is theorized that a change in cerebral perfusion pressure results in CBF change. CBF change alters local tissue metabolite concentration. Change in tissue metabolite concentration is posited to induce local vessel diameter change, thus maintaining local normal CBF (Herpin, 1990; Miller et al., 1971; Paulson et al., 1990). Although CBF and metabolism are closely coupled, it is less clear whether a metabolite produced by a change in CBF is active in maintaining normal CBF. It does seem likely that
local metabolite levels moderate local CBF, and therefore, may play a very localized autoregulatory role. At present, however, there is no conclusive support for a primary metabolite that controls cerebral autoregulation.

Cerebral autoregulation is effective within a MAP range of approximately 50 to 150 mm Hg., although autoregulatory limits may vary between and within individuals over time (Paulson et al., 1990). Factors known to alter the relationship between level of systemic arterial pressure and degree of cerebral vascular resistance include arterial oxygen and carbon dioxide levels (Harper, 1965; Kety & Schmidt, 1948; Paulson et al., 1990), sympathetic and parasympathetic activation (D’Alecy & Fiegl, 1972; Harper, Deshmukh, Rowan & Jennett, 1972; Edvinsson Lacombe, Nielsen, & Owman, 1983; Owman et al., 1984), and cerebral metabolic level (Berne et al., 1981; Miller et al., 1971; Paulson et al., 1990).

Under normal circumstances, as systemic pressure decreases cerebral vessels dilate to maintain a constant CBF. CBF decreases when systemic pressure is approximately 50 mm Hg. because cerebral vessels cannot further dilate to increase blood flow. Conversely, as systemic pressure increases cerebral vessels constrict to maintain constant CBF. When systemic pressure approaches 150 mm Hg. cerebral vessels are cannot further constrict to protect the cerebral vascular bed from the large systemic arterial pressure head. At these high pressure levels, as intravascular pressure increases the cerebral vessels passively dilate. In the
animal model, acute elevations of arterial pressure disrupt the blood-brain barrier and precipitate cerebral edema through extravasation of plasma proteins (Busija, Heistad, & Marcus, 1980; Langfitt, 1982; Paulson et al., 1990).

Impairment of cerebral autoregulation with severe head injury may narrow the limits of autoregulation or are totally dissociate CBF from arterial pressure levels. Severe head injury may precipitate either high or low CBF independent of systemic arterial pressure level (Cold et al., 1981; Fieschi et al., 1974; Obrist et al., 1984; Overgaard & Tweed, 1974). Typically when cerebral autoregulation is impaired the cerebral vascular bed passively reflects any change in systemic arterial pressure. Cerebral vessels dilate in the presence of increased arterial pressure and passively constrict with decreased systemic pressure.

Impaired cerebral autoregulation leaves the head injured person vulnerable to change in systemic pressure. Although extravascular factors influence intracranial pressure, ICP passively follows systemic arterial pressure when cerebral autoregulation is impaired. Low systemic arterial pressures may cause cerebral ischemia, while elevated systemic pressures may trigger fluid extravasation, resulting in increased cerebral edema and intracranial hypertension. Because ETS precipitates sudden changes in systemic arterial pressure, it is crucial to include arterial pressure (MAP) when investigating how ETS impacts upon cerebrovascular status.

**Magnitude effect of ETS on MAP.** ETS consistently increased systemic
arterial pressure across all studies included in this review (Campbell, 1989; Donegan & Bedford, 1980; Ersson et al., 1990, Fisher et al., 1982; Parsons & Shogun, 1984; Rudy et al., 1991; Tsementis et al., 1982; White et al., 1982). Additionally, several studies that included sequential suction passes demonstrated that the rise in MAP was cumulative with each additional suction pass (Campbell, 1989; Parsons & Shogun, 1984; Rudy et al., 1991, Tsementis et al., 1982).

Baseline MAP across studies averaged 91 mm Hg., and demonstrated a 58 to 158 mm Hg. range. Average MAP during ETS for all studies was 108 mm Hg. with a range of 58 to 232 mm Hg. (see Table 3).

Although the preceding systemic arterial pressure means and ranges stayed within the normal lower bounds of cerebral autoregulation, the upper MAP limit for autoregulation was exceeded. This finding is crucial to understanding the impact of ETS on cerebrovascular status, because research has traditionally focused upon the dangers of inadequate or low arterial pressure (Parsons & Shogun, 1984; Rosner & Daughton, 1989; Rosner & Coley, 1986; Rosner & Coley, 1984).

Research has traditionally focused on low arterial pressure because during the natural course of severe head injury, the risk of cerebral ischemia from arterial hypotension is much greater than that of blood-brain barrier disruption and increased cerebral edema from arterial hypertension. Similarly, research of ETS in the head injured population has concentrated on cerebral ischemia as a probable
sequelae of ETS, although ETS is more likely to disrupt of the blood-brain barrier and precipitate cerebral edema. The focus on arterial hypotension as a sequelae of ETS has therefore, limited progress in ETS research in the head injured.

As previously discussed, the normal systemic arterial pressure limits of cerebral autoregulation may not be meaningful in the severely head injured adult. There is evidence that impaired autoregulation or even vasoparalysis develop secondary to the initial tissue damage and sequelae associated with severe head injury (Bruce et al., 1973; Cold et al., 1981; Langfitt, 1982; Rosner & Becker, 1984; Obrist et al., 1984). In the head injured person, an increase in MAP that is well within the normal autoregulatory boundaries may result in cerebral vasodilation. The increased MAP from ETS as documented in the preceding studies may have been sufficient to extend neuronal damage through mechanical disruption of the blood-brain barrier and fluid extravasation into the interstitium and cerebral tissue.

MAP elevation in response to ETS may explained by one of two mechanisms. First, the Cushing response predicts that increased ICP as an indicator of cerebral ischemia will cause systemic hypertension, bradycardia, and slowed ventilatory rate (Langfitt, 1982; McGinnis, 1989). From this perspective, increased MAP is a compensatory response to ischemia. However, the Cushing theory does not fit empirical evidence that ETS increases MAP either concurrent with, or slightly before, ICP increases (Rudy et al., 1991). Additionally, the
Cushing response is associated with situations where ICP values approach the level of systemic arterial pressure. None of the studies included in this review reported ICP values near MAP values. The Cushing response, therefore, is an unlikely explanation for elevated MAP precipitated by ETS.

The alternative explanation for increased MAP in response to ETS is that MAP increases from activation of the systemic vasopressor response. Mechanical irritation of the airways as occurs with suction catheter insertion, results in an involuntary rise in blood pressure, cough, and bronchoconstriction (Haxhui et al., 1986; Jammes & Mei, 1979; Nadel & Widdicombe, 1962; Tomori & Widdicombe, 1969). Activation of the systemic vasopressor response from airway stimulation is congruent with the temporal pattern of MAP increasing concurrent with or slightly before ICP increase. Based upon the present study’s theoretical framework activation of the systemic vasopressor response from suction catheter insertion was the basis for empirical findings of increased MAP.

**Heart rate (HR).** Three of the eight studies in Table 3 measured heart rate as an additional indicator of cerebrovascular response to ETS (Campbell, 1989; Parsons & Shogun, 1984; Rudy et al., 1991). Although not explicitly identified by the investigators, the use of heart rate as an indicator of cerebrovascular response presumably was based upon Cushing’s theory. Cushing predicted that intracranial hypertension as an indicator of cerebral ischemia should result in bradycardia (Langfitt, 1982, McGinnis, 1988).
Average baseline heart rate across the three studies was 94 BPM, with a range of 60 to 126 BPM (Campbell, 1989; Parsons & Shogun, 1984; Rudy et al., 1991). During ETS HR was 116 BPM with a range of 48 to 220 BPM. Although bradycardia did occur in individual subjects, average HR increased during ETS. These findings are incongruent with the Cushing response that predicts decreased HR with elevated ICP.

An alternative explanation consistent with increased HR and MAP demonstrated across all studies, is activation of the systemic vasopressor response. Activation of the vasopressor response from tracheal stimulation during ETS would increase heart rate and MAP (Guyton, 1991; Jensen, 1980).

Past studies of the effect of ETS on cerebrovascular status head injured patients used heart rate as a measure of cerebrovascular response (Campbell, 1989; Parsons & Shogun, 1984; Rudy et al., 1991). Empirical evidence from these studies suggests however, that change in heart rate during ETS results from activation of the systemic vasopressor response. The present study specified HR as an indicator of the vasopressor response to test an alternative explanation for ETS impact on cerebrovascular status.

In conclusion, the preceding review has demonstrated that ETS results in marked and sustained increase in ICP. While continuous measurement of ICP is crucial to understanding the effect of ETS on head injured patients’ cerebrovascular status, ICP as a single measure is not sufficient to explain
observed cerebrovascular responses. Recognizing the need to better understand the effect of ETS on cerebrovascular status of head injured persons, researchers incorporated multiple measures (CPP, MAP, and HR) to assess cerebrovascular response.

The usefulness of CPP as an indicator of cerebrovascular response to ETS was questioned. MAP consistently increased in response to ETS. CPP varied directly with increased MAP. Thus when ETS is the experimental condition, CPP does not appear to offer any information not already provided by MAP measurement. To accommodate comparison between current and past study findings, the present study included CPP as a measure of cerebrovascular response.

Both MAP and HR increased in response to ETS. Although past research used MAP and HR as cerebrovascular response indicators, it was argued that they are indicators of a systemic vasopressor response activated during ETS. Although several studies suggested that ETS activated the systemic vasopressor response, it was not systematically tested. The present study tested a theoretical model that included activation of the vasopressor response by airway stimulation. The present study sought to explain how activation of the vasopressor response from airway stimulation impacted upon cerebrovascular response.

**Endotracheal Suctioning and Airway Stimulation**

Four studies included in Table 3 tested the premise that airway stimulation
results in cerebrovascular response to ETS in head injured persons (Donegan & Bedford, 1980; Fisher et al., 1982; White et al., 1982; Yano et al., 1986). Three studies concluded that airway stimulation initiated cerebrovascular response via the cough reflex (Donegan & Bedford, 1980; White et al., 1982; Yano et al., 1986). The remaining study argued against the cough reflex as the basis for observed cerebrovascular response to suctioning (Fisher et al., 1982).

Subjects for the four studies were mechanically ventilated and in the intensive care unit. Except for Fisher and colleagues (1982) who studied pediatric subjects with mixed intracranial pathology, study samples consisted of head injured subjects. Three studies tested the ability of lidocaine to attenuate the impact of ETS on cerebrovascular status, while Fisher and colleagues compared the effect of airway stimulation to the effect of apnea. All studies were quasi-experimental in design, used convenience samples, and incorporated minimal controls for the suctioning procedure.

The earliest study in this group compared the ability of intravenous (IV) lidocaine versus a placebo (IV normal saline) to attenuate cerebrovascular response to ETS in 10 head injured subjects (Donegan & Bedford, 1980). Lidocaine and saline were tested in the same subjects during temporally different suctioning events. Data time points for comparison were before medication administration (control), immediately before ETS but after medication administration, and at the highest ICP during 5 minutes after ETS. Data were
analyzed using paired t-tests between the various time points using a criterion of \( p < .05 \) for significance. Between-treatment (lidocaine vs. saline) comparisons were not reported. IV lidocaine significantly decreased ICP before ETS compared to control values, while normal saline did not. ICP values after ETS compared to control, were not significantly increased when lidocaine was administered, but were significant when saline was administered. The investigators hypothesized that IV lidocaine decreased baseline ICP by depressing metabolic brainstem activity. Depressed brainstem activity inhibited the cough reflex and therefore, attenuated cerebrovascular response to ETS.

The preceding findings must be interpreted with caution since the repeated use of paired t-tests across time points without correction increased the chance of finding significance. Although the study had methodological weaknesses, the findings demonstrated that ICP increased in response to ETS. Regardless of the fact that lidocaine may have suppressed the cough reflex, it did not prevent ICP increase with suctioning. These findings suggest that the cough reflex may not be the only mechanism through which tracheal stimulation increases ICP. The findings support a model that incorporates multiple mechanisms to explain the effect of airway stimulation on cerebrovascular status in head injured persons.

White and colleagues (1982) also identified the cough reflex as the primary mechanism for increased ICP from tracheal stimulation during ETS. The ability of five medications (fentanyl, thiopental, IV lidocaine, intratracheal [IT]
lidocaine, and succinylcholine) and a control (normal saline) to attenuate
cerebrovascular response to ETS were studied in fifteen head injured subjects of
unknown age. All subjects received the five medications and control during
temporally different suctioning events. In addition to measuring ICP and MAP as
outcome variables, a three point cough scale (no cough to vigorous cough) was
used to measure the ability of the medications to blunt the cough reflex. A one-
way analysis of variance and Duncan’s multiple range test, with a significance
criterion of $p < .05$, was used to compare the efficacy of medications to blunt
ICP and cough response to ETS.

When compared to control, only IT lidocaine and succinylcholine
significantly depressed cough and peak ICP response during ETS. MAP always
increased during ETS. Even though ICP was significantly blunted by these
medications, it still increased over baseline levels. The investigators hypothesized
that tracheal stimulation initiated the cough reflex and resulted in increased
intrathoracic pressure. Retrograde venous pressure from the intrathoracic pressure
increased cerebral venous pressure, and therefore resulted in increased ICP.

Two important points to the present study’s theoretical framework were
drawn from the preceding findings. The first point is that ETS increased ICP
regardless that two medications significantly suppressed both the cough reflex and
ICP response to ETS. Secondly, MAP continued to rise during ETS although
cough reflex was suppressed. The rise of both MAP and ICP during ETS
regardless of cough suppression, suggests that increased intrathoracic pressure is not the only mechanism responsible for cerebrovascular response to ETS. Sudden increased MAP may be attributed to the systemic vasopressor response. Airway stimulation that initiated both increased intrathoracic pressure and activation of the vasopressor response, explains MAP increase during ETS when cough is not present. The current study tested a theoretical formulation that included both increased intrathoracic pressure and activation of the systemic vasopressor response as the basis for cerebrovascular response to ETS.

Yano and associates (1986) addressed the role tracheal stimulation played in causing cerebrovascular response to ETS, by comparing the ability of IV versus IT lidocaine to attenuate ICP response to ETS in nine head injured adults. The effectiveness of the two medication routes was assessed by comparing ICP response to ETS before and after the administration of medications. Statistical tests used for analysis were not reported. Both IV and IT lidocaine did not prevent, but attenuated ICP response to ETS. IT lidocaine consistently suppressed ICP response more than IV lidocaine. The researchers hypothesized that IT lidocaine’s ability to attenuate ICP response to ETS was from suppression of the cough reflex due to the medication’s anesthetic effect upon tracheal mucosa.

The researchers’ conclusion may be too narrow in scope. In addition to its ability to blunt ICP response by suppressing the cough reflex, lidocaine may also inhibit ICP increase through effects on other neurogenic pathways. An
explanation that considers several mechanisms activated by receptors in the tracheal mucosa would explain why IT lidocaine can suppress but not prevent ICP response to ETS.

In contrast to the preceding studies, Fisher and associates (1982) argued that cough reflex and intrathoracic pressure were not the basis for cerebrovascular response to ETS. Using a sample of nine subjects, the researchers compared ICP and MAP response to ETS. Subjects were studied twice; once with tracheal suctioning, and once with a period of apnea equivalent to duration of suctioning. Treatments were compared using a within-subject repeated measures analysis of variance. A Student Newman Kuels test was used to test for significant differences ($p < .05$) in ICP and MAP during baseline, presuctioning hyperventilation, ETS, and postsuctioning hyperventilation. Both ICP and MAP were significantly increased from baseline values during suctioning, but were not significantly different during apnea. Since hyperventilation was consistently administered (25% rate increase for 60 seconds), and coughing in two subjects during ETS produced similar ICP responses to those observed during apnea, the authors deducted that neither cough reflex or intrathoracic pressure was responsible for cerebrovascular response to ETS. The investigators concluded that tracheal stimulation from suction catheter insertion was responsible for the observed effect. Although Fisher and colleagues ruled out both cough reflex and intrathoracic pressure, they did not propose an alternative explanation for how
tracheal stimulation increases ICP and MAP. The study's findings support the argument that in addition to cough reflex and increased intrathoracic pressure, tracheal stimulation may initiate cerebrovascular response by other pathways.

In summary, the preceding studies provide the following contributions that were useful to the present research. First, tracheal stimulation was identified as a major factor in cerebrovascular response to ETS. Second, findings suggest that the cough reflex, through increased intrathoracic pressure, may be one mechanism by which tracheal stimulation affects cerebrovascular status. Third, study findings suggest that neither central neural or local receptor suppression is sufficient to prevent increased ICP in response to ETS. The current study built upon these findings by testing a model that identified airway stimulation from suction catheter insertion as the major stimulus that initiates cerebrovascular response to suctioning. The present study conceptualized airway stimulation as affecting head injured adults' cerebrovascular status via several mechanisms. The following section of this review offers an explanation of how airway stimulation may affect the intracranial environment via several pathways.

**Airway Stimulation During ETS and Cerebrovascular Response**

The physiologic theory that provided structure for the current study sought to explain how tracheal stimulation resulting from suction catheter insertion altered cerebrovascular status in head injured adults. Mechanisms identified in the literature as the basis for cerebrovascular change resulting from tracheal
stimulation, either were not congruent with empirical findings or could only partially explain findings. The present study combined findings from research on pulmonary neurogenic mechanisms with research focused on cerebral blood flow to explain how tracheal stimulation affects cerebrovascular status. The following review is organized by the study’s theoretical framework, where suction catheter insertion served as a mechanical stimulus that initiated an irritant receptor response. First, studies that investigated airway irritant receptor response are reviewed. Remaining literature is organized by the three responses to irritant receptor activation: reflexive bronchoconstriction, cough reflex, and the systemic vasopressor reflex. Each irritant receptor response is discussed in light of what is known about how the response affects cerebrovascular status in head injured persons.

Activation of Irritant Receptors via Airway Stimulation

The extrapulmonary airways have receptors in the epithelial lining that respond to mechanical stimulation with reflex bronchoconstriction (Boushey et al., 1972; Guyton, 1991; Jammes & Mei, 1979; Jenson, 1980; Levitsky, 1986; Haxhiu et al., 1986; Haxhui et al., 1987; Mills et al., 1969; Nadel & Barnes, 1984; Richardson, et al., 1984; Widdecombe, 1954). It has been long known that efferent parasympathetic innervation maintains airway bronchoconstrictor tone. The pathways responsible for reflexive bronchoconstriction in response to mechanical or chemical stimulation, however, have been not been clearly specified
Widdicombe (1954) identified irritant receptors located in the extrapulmonary airways. Irritant receptors are located in the sub or intraepithelial lining of the airways and possibly in the alveoli (Levitsky, 1986; Mills et al., 1969). Irritant receptors are different from stretch receptors or deflation receptors, although reflexive bronchoconstriction is known to be integrated with inspiratory/-expiratory patterning (Boushey et al., 1972; Comroe, Forster, Dubois, Briscoe & Carlsen, 1973; Jammes & Mei, 1979; Levitsky, 1986). Irritant receptors are also differentiated from juxtapulmonary-capillary receptors (J-receptors). Unlike J-receptors that are innervated by nonmyelinated C-type fibers, irritant receptors are connected with afferent myelinated vagal fibers (Boushy et al., 1972; Levitsky, 1986; Haxhui, et al., 1986; Mills et al., 1969; Roberts et al., 1981).

Using an animal model to study afferent pathways of pulmonary bronchoconstrictor tone, Jammes and Mei (1979) found that afferent vagotomy and procaine blockade of vagal nerves significantly decreased total lung resistance. These findings suggested that the interventions resulted in bronchodilation. The researchers next investigated which lung receptors were responsible for bronchoconstrictive tone. Histamine administered while the vagus was blocked with procaine resulted in a significant increase in lung resistance. Histamine is known to preferentially stimulate irritant receptors. By comparison, lung
resistance did not increase when phenyl diguanide, that is specific to J-receptors, was administered. The response to histamine suggested that irritant receptors were responsible for reflexive bronchoconstriction in response to irritants, while J-receptors were active in maintaining ongoing bronchoconstrictor tone. The investigators concluded that bronchoconstriction resulted exclusively from the peripheral stimulation of both J-type and irritant receptors.

Central processing of irritant receptor stimulation. Since ventilatory activity is centrally controlled in the medullary respiratory center, researchers sought to determine if irritant receptor response was centrally mediated in the same brainstem area. One group of researchers focused on the role of the ventral medullary surface in the control of reflexive airway responses to stimulation (Haxhui et al., 1986; 1987; Strohl, Norcia, Wolin, Haxhiu, Van Lunteren, & Deal, 1988). Haxhui and associates demonstrated that in the animal model, atropine abolished bronchoconstriction (demonstrating vagal paralysis), direct application of lidocaine or moderate cooling of the ventral medullary surface (VMS) diminished bronchoconstriction, and application of nicotine, carbon dioxide and N-methyl-D-aspartate (an excitatory amino acid) increased bronchoconstriction. Additionally, when tracheal constriction was induced by histamine administration, VMS cooling decreased bronchoconstriction. Bronchoconstriction returned to previous levels when the VMS returned to normal temperatures. The researchers concluded that irritant receptor stimulation
activated central neurons, and that VMS neurons mediated irritant receptor response.

In addition to bronchoconstriction, irritant receptor activation is known to initiate other responses, such as altered ventilatory pattern (Boushey et al., 1972; Richardson et al., 1984). Since there are groups of specialized neurons in the medullary respiratory center that are responsible for spontaneous generation of the inhalation/exhalation pattern, the preceding findings suggest that irritant receptor response is integrated with the medullary respiratory center that controls ventilatory activity. This hypothesis receives support from the finding there are neural projections between the VMS, the nucleus tractus solitarius, and the dorsal nuclei of the vagus (Kalia, 1981). Since respiratory neurons called the dorsal medullary group (DMG) and the vagus nerve are located in the nucleus tractus solitarius it seems likely that there is a relationship between the VMS and DMG (Levitsky, 1986; Jenson, 1980). Based upon this assumption, bronchoconstriction from irritant receptor activation would be expected to be accompanied by other motor responses known to arise from activation of neurons in the medullary respiratory center.

The preceding findings support a reflexive bronchoconstrictive response to airway irritation, initiated by irritant receptors located in airways and mediated by medullary neurons. A physiologically based relationship between reflexive bronchoconstriction and other pulmonary responses to airway stimulation was
demonstrated. All of these findings lend empirical support for the current study’s theoretical framework. The following discussion links reflexive bronchoconstriction and cerebrovascular response to airway stimulation.

**Reflexive bronchoconstriction and cerebrovascular response.** As demonstrated above, parasympathetic innervation mediates bronchoconstriction. Although pulmonary parasympathetic innervation is well established, cerebral parasympathetic innervation remains more speculative. Perivascular parasympathetic fibers are known to accompany perivascular sympathetic fibers throughout the cerebral vasculature (Owman et al., 1984). Although perivascular parasympathetic innervation exists, its role in cerebral dynamics is not understood. The fundamental question is whether cerebral parasympathetic innervation is responsible for primary cerebral vasodilation.

Findings from studies with animal models support that primary vasodilation occurs in response to both medullary ischemia and stimulation (Reis, 1984). The mechanism by which cerebral vasodilation occurs is not known. Since the medulla is well innervated with parasympathetic fibers it is possible that the increase in CBF may result from parasympathetic activation.

A study by Nakai and Ogino (1984) provided further support for primary vasodilation from parasympathetic activation. Using an animal model, the investigators studied the role that cardiopulmonary reflexes play in cerebral vessel regulation. Electrical stimulation of the medullary relay station in the solitary
nucleus resulted in marked increased CBF. The researchers hypothesized that because the solitary nucleus is the terminus for sensory vagal fibers from the heart and lung, electrical stimulation of the area may have activated ascending efferent vagal fibers that innervate the cerebral vasculature. Activation of efferent vagal fibers would be expected to vasodilate vessels and increase CBF.

Nakai and Ogino’s findings differ from previous research that demonstrated no relationship between cardiopulmonary reflexes and cerebral blood flow (Heistad & Marcus, 1976). Differences may have been methodological, since Nakai and Ogino activated vagal efferents at the medullary relay station, while other researchers had activated peripheral receptors. Nakai and Ogino speculated that when reflexes are peripherally activated, compensatory mechanisms may make it impossible to assess the role of parasympathetic activation in the cerebral vasculature.

In summary, activation of irritant receptors initiates central neural activity. It is possible that the central neural activation may also cause primary cerebral vasodilation. The reflexive cerebral vasodilation would, in part, account for increased ICP observed in response to ETS. While centrally initiated cerebral vasodilation would cause rise in MICP, vasodilation via this mechanism would not explain sudden marked increases in MICP in response to ETS, documented by Rudy and colleagues (1991). Therefore, although evidence for primary cerebral vasodilation resulting from irritant receptor activation partially explains
cerebrovascular responses to ETS, a more inclusive theoretical framework is needed to guide research in this area.

**Irritant Receptor Response and the Cough Reflex**

Mechanical irritation of the larynx has long been known to cause coughing and bronchoconstriction (Boushey et al., 1972). Only recently however, have the neural receptors that initiate the cough response been identified. Cough and bronchoconstriction induced by mechanical stimulation of the larynx are now known to be initiated by irritant receptor activation (Haxhui et al., 1987; 1988). Neural connections have been identified between the ventral medullary surface (VMS), associated with reflexive bronchoconstriction, and the ventral medullary group neurons (VMG) located in the medullary respiratory center (Kalia, 1981). It is also known that efferent vagal fibers run from the VMG to the larynx (Haxhui, et al., 1987; 1988; Kalia & Mesulam, 1980; Levitsky, 1986). Therefore, stimulation of irritant receptors in the larynx result in both bronchoconstriction and coughing.

Mechanical irritation of the carina causes intense coughing (Guyton, 1991; Kersten, 1989; Levitsky, 1986). The carina is densely populated with irritant receptors (Haxhui et al., 1986; Levitsky, 1986). Initiation of the cough reflex sends impulses to dorsal medullary group (DMG) neurons via afferent vagal fibers. Activation of the DMG causes collateral impulses to be sent to the VMG. Activation of VMG inspiratory neurons result in contraction of inspiratory
muscles, deep inspiration, and closure of the glottis. VMG expiratory neurons are then activated, resulting in forced expiration against the closed glottis.

During the cough reflex, intrathoracic pressure may increase to as high as 100 mm Hg., although it falls rapidly with expiration. The large increase in intrathoracic pressure constricts blood vessels in the thorax, thus decreasing thoracic vessel capacity to hold and receive blood (Guyton, 1991; Kersten, 1989; Levitsky, 1986).

Cough reflex and cerebrovascular response. The cough reflex is identified as a major mechanism for ICP increase in response to ETS (Donegan & Bedford, 1980; Shapiro, 1975; White et al., 1982; Yano et al., 1986). Reflexive ventilatory muscle contraction occurs, although tracheal intubation prevents severely head injured patients from coughing effectively. Although the endotracheal tube prevents the glottis from completely closing, forced expiration through a small lumen endotracheal tube, particularly when occluded by a suction catheter, results in marked increased intrathoracic pressure.

No studies were found that systematically investigated the effects of coughing on cerebrovascular status during ETS. Increased intrathoracic pressure results in increased central venous pressure that is associated with increased ICP (Hamer e: al., 1977; Dardenne et al., 1969). Ersson and colleagues (1990) anecdotally provided evidence of the relationship between central venous pressure and ICP response to ETS. In an illustration of simultaneously recorded waveform
tracings from one subject (see Errson et al., 1990, Figure 1, p. 101), CVP and ICP increase simultaneously during ETS. The basis for the relationship between increased intrathoracic pressure and increased ICP is that elevated intrathoracic pressure increases retrograde pressure on the cerebral venous bed, and decreases cerebral venous outflow. As a result of this process cerebral venous volume expands and ICP increases.

The preceding findings demonstrate that irritant receptor activation can cause both coughing and bronchoconstriction, that results in sudden ICP increase. In head injured people with an intact cough reflex, activation of the cough reflex during ETS may be a major mechanism for ICP increase. Increased intrathoracic pressure during cough may explain the sudden and marked increase in ICP observed during ETS. In deeply comatose people, however, the cough reflex is often absent, yet increased ICP during ETS has been demonstrated (Rudy et al., 1991).

An irritant receptor response that includes the cough reflex and a central neural mechanism that result in increased ICP during ETS offers an explanation of how ICP may increase even when cough is absent or suppressed by medication (Donegan & Bedford, 1980; Yano et al., 1986; White et al., 1982). The two physiologic pathways, however, do not adequately explain sudden marked ICP amplitude increase during ETS known to occur in deeply comatose patients. The following discussion presents a third mechanism that may account for ICP
increases in response to ETS.

**Irritant Receptor Response and the Systemic Vasopressor Reflex**

Activation of the systemic vasopressor reflex is often cited for increased MAP and HR observed during mechanical stimulation of the airways (Parsons & Shogun, 1984; Splinter, 1990; Ersson et al., 1990). In the head injured population, however, the vasopressor reflex as the sole mechanism for systemic and cerebral vascular responses to ETS is not congruent with systemic (increased MAP and HR) and cerebrovascular changes (increased ICP) observed during ETS.

The systemic vasopressor reflex is controlled by the vasomotor center located in the medulla in close proximity to the respiratory center (Appenzeller, 1990; Guyton, 1991; Jenson, 1980; Levitsky, 1986). Activation of the vasopressor reflex occurs when the vasomotor center increases its output of sympathetic impulses to the systemic vessels. As a result of increased sympathetic activity, both systemic blood pressure and heart rate increase (Guyton, 1991; Jensen, 1980). If irritant receptor stimulation results in increased sympathetic activity, then the vasopressor reflex would explain increased blood pressure and heart rate during ETS. As discussed previously, however, irritant receptor stimulation activates parasympathetic pathways, as demonstrated by reflexive bronchoconstriction (Haxhui et al., 1987; 1988).

It is possible that activation of the vasopressor reflex during ETS occurs
from spill-over impulses from the respiratory center (Guyton, 1991). Afferent fibers are known to extend from the vasomotor center to the respiratory center, and respiratory center efferent fibers travel to the vasomotor center (Jensen, 1980; Haxhiu et al., 1986; Nakai & Ogino, 1984). Integration of respiratory and vasomotor centers is supported by findings that pulmonary airways dilate in response to primary systemic blood pressure elevation. Conversely, airways constrict when systemic pressure decreases (Levitsky, 1986; Jenson, 1980; Haxhiu et al., 1987; 1988; Nadel & Widdicombe, 1962). When the primary stimulus is airway responsiveness rather than systemic pressure changes, bronchoconstriction is accompanied by increased systemic blood pressure (Haxhiu et al., 1987; 1988). It is concluded that the vasopressor response alone does not explain simultaneous bronchoconstriction and increased blood pressure.

No research was found that investigated the specific mechanisms that might account for both increased blood pressure and bronchoconstriction. It is possible, however, that since the respiratory and vasomotor centers are located in the same medullary area, and because neural efferent fibers are known to go from the respiratory center to the vasomotor center, activation of respiratory neurons may subsequently activate the vasomotor center. If the vasomotor center is activated by impulses transmitted from the respiratory center, then simultaneous systemic vasoconstriction, increased heart rate, and bronchoconstriction in response to ETS could be explained (Haxhiu et al., 1987; 1988). In conclusion, although a
pathway for the simultaneous activation of the vasomotor center is proposed, the
mechanism for irritant receptor activation of the vasomotor center remains
speculative at this time.

**Systemic vasopressor reflex and cerebrovascular response.** Findings from
research demonstrates consistent increases in MAP, HR, and ICP in response to
ETS in head injured patients (Campbell, 1989; Donegan & Bedford, 1982; Ersson
et al., 1990; Fisher et al., 1982; Parsons & Shogun, 1984; Rudy et al., 1991;
White et al., 1982; Yano et al, 1982). When MAP increases however, cerebral
vessels should vasoconstrict to maintain ICP level (Berne et al., 1981; Paulson et
al., 1990; Youmans, 1982). In head injured people who often have impaired
cerebral autoregulation, activation of the vasomotor center during ETS would
allow unattenuated systemic arterial pressure to enter the cerebral vascular bed
resulting in sudden marked increased ICP. Activation of the vasomotor reflex
during ETS in people with impaired autoregulation also explains the antecedent or
concurrent MAP and ICP increase.

In conclusion, activation of irritant receptors lining the airways by insertion
of a suction catheter is posited to be the major pathway by which ETS initiates a
cerebrovascular response in head injured adults. Three major physiologic
responses to irritant receptor activation were identified, each capable of altering
cerebrovascular status during ETS. Together, the three irritant receptor responses
explain increased MICP and ICP amplitude observed in response to ETS. Airway
stimulation such as that resulting from suction catheter insertion, was shown to be the component of the ETS procedure that initiates the potentially harmful cerebrovascular response in head injured adults. Clearly, it is imperative that suction catheter insertion be isolated from the other components of the ETS procedure so that its role in initiating cerebrovascular response to ETS can be determined.

**Cerebral Compliance and Cerebrovascular Response to ETS**

The following review addresses the proposed relationship between cerebral compliance and cerebrovascular response during ETS. The discussion first focuses on the conceptualization, operationalization and measurement of cerebral compliance. The final section centers on the usefulness of cerebral compliance to the investigation of cerebrovascular response to suction catheter insertion status.

**Conceptualization of Cerebral Compliance**

Interest in the relationship between intracranial volume and pressure grew from the realization that measurement of intracranial pressure alone could not differentiate between patients who had a benign course of illness and those who developed fulminating intracranial hypertension (Chopp & Portnoy, 1980; Langfitt, 1982).

The volume-pressure relationship in the intracranial space is defined by either cerebral compliance or the inverse of compliance known as cerebral elastance. Both concepts are based upon the assumption that normal volume-
pressure relationship in the intracranial space exists when there is a dynamic equilibrium between cerebrospinal fluid production and absorption (Jennett & Teasdale, 1981). Compliance is the amount of space accommodation that is available within the intracranial space (Langfitt, 1982). Compliance focuses on how a change in intracranial volume affects ICP and is expressed by the equation dV/dP. Normal compliance means that for a given amount of increase in intracranial volume there will be little or no change in ICP. The flat portion of the volume-pressure curve, as presented in Figure 1, Chapter One, represents normal compliance. Decreased compliance indicates that for a given change in volume, a disproportionate increase in ICP occurs (Langfitt, 1982).

Cerebral elastance is the resistance offered by the walls of the cerebrospinal fluid spaces. Elasticity is the change in ICP resulting from a change in CSF volume, and is expressed by the equation dP/dV (Jennett & Teasdale, 1981). Increased elastance means that there is a disproportionate increase in ICP for a given volume increase, indicating rigidity or tightness of the intracranial space. The steep portion of the volume-pressure curve in Figure 1, Chapter One, represents increased elastance.

Although cerebral compliance was first defined as CSF dynamic equilibrium, more recent conceptualization includes consideration of relationships among the three intracranial components: CSF, cerebral blood volume, and cerebral tissue (Jennett & Teasdale, 1981; Marmarou, Shulman, Rosende, 1978;
Rosner & Becker, 1984; Schettini & Walsh, 1973). Any factor that increases volume in one of the intracranial compartments has the potential to increase intracranial rigidity and decrease compliance. Decreased compliance, in turn, indicates that intracranial compensatory mechanisms are no longer functional. From this perspective, assessment of cerebral compliance may provide information to identify those head injured persons who are at risk of harmful cerebrovascular response to ETS.

**Operationalization and Measurement of Cerebral Compliance**

Because cerebral compliance characterizes the dynamic relationship among intracranial components, measures of compliance offer a way of predicting and assessing patients' cerebrovascular responses to any event that is known to increase intracranial volume. Various approaches to measurement of cerebral compliance exist. All measurement strategies assume if compliance is decreased, a change in volume in one of the intracranial compartments will cause a disproportionate increase in ICP. Measurement approaches have varied from intraventricular bolus injection tests to assessment of cerebrospinal pulse wave characteristics.

Intraventricular bolus injection tests include the volume-pressure response test (VPR) developed by Miller and Garibi (1972), and the pressure-volume index (PVI) developed by Marmarou, Shulman, and Rosende (1978). The injection tests assume that the rapid introduction of a volume of fluid challenges intracranial
compensatory mechanisms. If intracranial compensatory mechanisms are overwhelmed, then ICP will instantly increase, indicating decreased compliance. The bolus injection tests introduce risk of infection and the possibility of initiating sustained intracranial hypertension in patients who are highly vulnerable to such risks. Additionally, the assumption that the bolus tests measure intracranial elastance has been challenged. Bolus injection into a Starling resistor model of the cerebrovascular bed suggests that the rapid pressure response results from vessel compression rather than decreased space accommodation (Chopp, Portnoy & Branch, 1983). Although nurses can use the information obtained from bolus tests, the tests are not routinely administered, thus data are usually not available to assist in planning patient care. Because bolus tests involve a relatively high degree of risk, and are not an independent nursing function, this review focuses on studies that tested other means of assessing compliance more readily applicable to bedside nursing care.

Using the same assumptions of bolus injection tests, other investigators developed cerebrospinal fluid pulse amplitude analysis (CSFPAA) to assess cerebral compliance (Szewczykowski, Sliwka, Kunicki, Dytko, Korsak-Sliwka, 1977). CSFPAA uses the known linear relationship between ICP amplitude (ICPA) and mean ICP (MICP) to assess cerebral compliance. Change in cerebral blood volume resulting from each cardiac cycle is assumed to be equivalent to the rapid bolus injection. The pressure response to volume increase is assumed to be
represented by the amplitude increase in ICP wave form. Compliance is assessed by plotting a volume-pressure curve from the relationship between ICP and MICP at varied MICP levels. It is assumed that ICP increases linearly with MICP, as long as volume is constant.

The preceding approach to compliance assessment is not without risk to the patient. Jugular vein compression, used to vary ICP level so that a volume-pressure curve can be plotted, increases ICP by obstructing cerebral venous outflow. In individuals with already existing cerebral tissue damage, jugular venous compression may extend cerebral damage by initiating intracranial hypertension. Chopp and Portnoy (1980) also criticized CSFPAA because the method is based upon the erroneous assumption that the cerebral blood volume per cardiac cycle is constant. Cerebral blood volume, however, varies with each cycle (Chopp & Portnoy, 1980).

Portnoy and colleagues used systems analysis to specify information that can be derived from pulse wave analysis, and to develop a low risk method of assessing cerebral compliance (Chopp & Portnoy, 1980; Portnoy & Chopp, 1981; Portnoy, Chopp, Branch & Shannon, 1982). The researchers selected systems analysis because it does not assume a constant volume for each cardiac cycle. From a systems analysis perspective, intracranial dynamics represent an unknown. Information can be obtained about intracranial dynamics, however, by assessing how a known input signal (MAP) is altered as it passes through the intracranial
space. Change in intracranial dynamics is assessed by comparison of the input signal (MAP) to an output signal (ICP). If the output signal (ICP) remains the same, there is no change in intracranial dynamics. Conversely, a change in output signal indicates change in the intracranial system. The change in signal is assessed by comparison between input and output wave frequency, amplitude and configuration.

In investigations using animal models, Portnoy and associates (1980; 1981; 1982) consistently demonstrated that arterial and intracranial pressure are composed of identical frequencies (measured in Hertz). The identical frequencies support the derivation of ICP pulse from systemic arterial pulse. The researchers concluded that both ICP wave form amplitude and configuration are dependent upon arterial amplitude and shape.

While ICP waveform originates from MAP, it is not solely dependent upon arterial pressure. Cerebral waveforms differ from MAP in configuration, magnitude, and amplitude. The researchers attributed MAP and ICP waveform differences to impedances created by cerebral vascular resistance (Portnoy & Chopp, 1981). ICP waveforms in normal animal subjects and humans were identified by the following characteristics: (a) an initial sharp upward inflection, (b) a flattened or level peak followed by descent to baseline or, (c) after upward inflection, a gradual descent to baseline (Chopp & Portnoy, 1980, Portnoy & Chopp, 1981). Appendix A includes exemplars of normal waveforms.
Using an animal model, ICP waveform was altered by experimentally induced intracranial hypertension resulting from extravascular volume expansion, hypercapnia, and hypoxemia (Chopp & Portnoy, 1980; Portnoy & Chopp, 1981; Portnoy et al., 1982). In contrast to normal ICP waveforms, the experimental conditions resulted in the following waveform changes: (a) increased amplitude, (b) a rounded waveform that progressed to a peaked wave form and, (c) a steep descent to baseline. Exemplars of rounded waveforms are displayed in Appendix B. Under the experimental conditions, ICP waveform configuration corresponded more to arterial waveforms than during control conditions. The researchers concluded that transmission of MAP across cerebral resistance vessels was more linear during experimental conditions than during normal conditions (Portnoy et al., 1982). The researchers hypothesized that increased ICP amplitude reflected cerebral vasodilation, while increased ICP rounding and peaking represented decreased cerebrovascular tone (Portnoy & Chopp, 1981).

Portnoy and associates emphasized that the changes in ICP amplitude and configuration observed during experimental conditions were due to alteration or dysfunction of cerebral autoregulatory mechanisms, rather than a reflection of decreased cerebral compliance. These conclusions are based upon the knowledge that both hypercarbia and hypoxemia are well known to result in cerebral vasodilation. The researchers substantiated their claim with the observation that although hypercapnia and hypoxemia resulted in even more pronounced increase
in ICP amplitude and rounding than during extravascular volume expansion, simultaneous compliance testing with VPR test demonstrated better compliance during hypercapnia and hypoxemia than during extravascular volume expansion (Portnoy & Chopp, 1981).

The researchers' conclusions that increased ICP amplitude and ICP rounding exclusively reflect autoregulatory dysfunction are questioned because an increase in volume of one intracranial compartment affects total intracranial volume. Cerebral vasodilation would therefore, affect intracranial volume-pressure dynamics. Additionally, although the investigators cited results from the VPR test to support their conclusions (Portnoy & Chopp, 1981), they had previously demonstrated that the VPR decreased during intracranial hypertension, although it should increase (Chopp & Portnoy, 1980). The unreliability of the VPR during intracranial hypertension was substantiated by others (Avezaat, Van Eijndhoven, & Wyper, 1979). Thus, while Portnoy and colleagues have offered a potentially useful measure of intracranial status, it is unclear what altered ICP amplitude and configuration represents. Because increased volume in any intracranial compartment can decrease compliance, increased ICP amplitude and rounded configuration in addition to intracranial hypertension represents a reasonable measure of alteration in cerebral compliance.

Cardoso, Rowan and Galbraith (1983) also sought to identify the information that could be derived from ICP waveforms. The study investigated
the effect of head elevation, CSF withdrawal, voluntary hyperventilation and hypoventilation upon ICP amplitude and configuration in 15 adults with a variety of cerebral pathologies. ICP was continuously monitored and recorded on hardcopy during each of the study maneuvers. Waveform magnitude, amplitude and configuration during each procedure were descriptively analyzed and compared.

Although Cardoso and colleagues agreed with the Portnoy group regarding definitions and measurement of MICP and ICPA, ICP configuration was conceptualized in a related but different fashion. Cardoso and colleagues identified subwaveforms of ICP waveform. The first subwaveform (P₁), called the percussion wave, represents arterial pulsations. Although the second subwaveform (P₂), named the tidal wave, is identified in the literature as depicting retrograde venous pressure (Dardenne, Dereymaeker, Laceron, 1969; Hamer, Alberti, Hoyer, & Wiedemann, 1977), Cardoso and colleagues argued that P₂ represents state of cerebral compliance. The third subwaveform (P₃) is the dicrotic waveform, also identified as representing retrograde venous pulsations. The identification of the various subwaveforms by Cardoso and associates represents a qualitative identification of the same waveform frequencies recorded by Portnoy and associates.

Cardoso and colleagues (1983) observed that both head elevation and CSF withdrawal resulted in decreased ICP magnitude (MICP), but no change in ICP
amplitude (ICPA) or configuration. Both voluntary hyperventilation and hypoventilation altered MICP magnitude, ICPA, and configuration. Hyperventilation was found to decrease both MICP and ICPA, and to decrease $P_2$ and $P_3$ more than $P_1$. Hypoventilation had the opposite effect from hyperventilation, causing increased MICP and ICPA, while $P_2$ and $P_3$ height increased relative to $P_1$. Based upon these findings, the authors concluded that $P_1$ offers information only about arterial state, and $P_2$ represents a decrease or increase in cerebral bulk. Conclusions about change in $P_2$ were based upon the known effects of hyperventilation (cerebral vasoconstriction) and hypoventilation (cerebral vasodilation). A change in cerebrovascular volume would therefore, affect cerebral bulk that would affect cerebral compliance.

Findings from Cardoso and colleagues corroborate findings from Portnoy and associates. Both studies found that hypercapnia (increase FiCO$_2$ in the Portnoy studies and breath holding in the Cardoso study) increased MICP, ICPA and resulted in similar configuration changes. The Cardoso groups’ findings that $P_2$ and $P_3$ increased relative to $P_1$ creates the same rounded wave with increased amplitude as that identified by Portnoy and colleagues. Both research groups concluded that the observed effect of hypercapnia resulted from cerebral vasodilation.

The two groups diverge only in interpretation of findings. The Cardoso group identified ICP amplitude and configuration changes as indicating state of
cerebral compliance, while Portnoy and associates separated cerebral vasodilation from its relationship with cerebral compliance. Differences in interpretation between the two groups are not incommensurate. By attributing change in waveform to change in cerebral vascular tone Portnoy and associates are more conservative in interpreting what ICP waveform change represents. In contrast to Portnoy and associates, the Cardoso group interpreted ICP waveform change more liberally to represent state of intracranial compliance.

Although elevated ICP amplitude has been associated with decreased compliance, what constitutes elevated ICP amplitude is less clear (Gonzalez-Darder & Barcia-Salorio, 1989; Newell et al., 1992). Findings from animal studies identified an ICP amplitude between 0.3 and 2.0 mm Hg. as normal (Gonzales-Darder & Barcia-Salorio, 1989; Hamer et al., 1977; Portnoy & Chopp, 1982).

In the current study, waveform characteristics were assumed to represent cerebral compliance. Based upon the findings from the preceding literature review, ICP configuration was used to assess state of cerebral compliance before suction catheter insertion. Because decreased cerebral compliance reflects diminished ability of intracranial compensatory mechanisms to accommodate volume increase, assessment of ICP waveform characteristics provided a means to identify head injured adults predisposed to potentially damaging cerebrovascular response during suction catheter insertion.
Relationship Between Cerebral Compliance and Cerebrovascular Response to Catheter Insertion

Cerebral compliance addresses the dynamic relationship between intracranial volume and pressure. Compliance decreases when a small increase in volume results in a disproportionate increase in ICP. In people with altered volume-pressure relationship, any event that precipitates an increased intracranial volume has the potential to cause disproportionate increased ICP. In the current study, suction catheter insertion was the stimulus that through irritant receptor activation resulted in increased cerebral blood volume.

The relationship between cerebral compliance and suctioning was explored in a study that investigated the validity of two risk factors related to decreased cerebral compliance (Rauch, Mitchell, & Tyler, 1990). Increased resting MICP and increased ICP amplitude were investigated for their association with a disproportionate increase in MICP in response to ETS. The framework for the study was based upon Mitchell’s (1986) proposal that decreased intracranial adaptive capacity be used as a nursing diagnosis for people at risk of developing, or who have decreased cerebral compliance. Mitchell identified decreased intracranial adaptive capacity as present when a disproportionate increased MICP developed in response to any noxious or nonnoxious stimuli.

The unit of analysis for the study was the observation session. Analysis included 30 ETS sessions from a sample of 8 children with a variety of cerebral
pathologies. ICP tracings collected during a previous study were analyzed to determine: (a) sensitivity (association of the risk factor with disproportionate increase in MICP), (b) specificity (absence of disproportionate increase in MICP with absence of the risk factor), (c) positive predictive value (proportion of sessions with the risk factor that had a disproportionate increase in MICP) and, (d) negative predictive value (proportion of sessions without the risk factor that still displayed a disproportionate increase in MICP).

Increased resting MICP was more sensitive (54%) to disproportionate increase in MICP during ETS than increased ICP amplitude (32%). Increased ICP amplitude however, was more specific (100%) than increased resting MICP (25%). The researchers concluded that neither risk factor was sufficiently sensitive to use as a predictor for disproportionate increase in ICP. Although increased ICP amplitude had a positive predictive value of 100%, compared to resting MICP (67%), negative predictive value for increased ICP amplitude was 35%, while resting MICP was only 17%. Based on the poor negative predictive values, decreased adaptive capacity (decreased compliance) was frequently present before ETS, even in the absence of the risk factors.

Findings from Rauch and associates suggest that decreased compliance is frequently present in people with a variety of intracranial pathologies. While findings do not support the use of increased ICP amplitude and increased resting MICP as exclusive indicators for state of cerebral compliance, the addition of ICP
configuration may provide a better assessment of intracranial compliance. The current study tested the ability of ICP configuration to assess cerebral compliance before ETS.

In summary, the preceding review demonstrated that decreased compliance is frequently present in people with intracranial pathology. Tissue edema and vasodilation associated with severe head injury, place head injured adults at risk for decreased cerebral compliance. Findings demonstrated that disproportionate increases in ICP may occur whenever intracranial volume is increased in people with compromised compliance. Suction catheter insertion increases intravascular volume through activation of the irritant receptor response. Head injured adults with decreased cerebral compliance, therefore, have increased potential for a deleterious cerebrovascular response to suction catheter insertion.

The findings from this review also suggest an alternative perspective for investigating the relationship between cerebral compliance and cerebrovascular response to ETS. All tests of cerebral compliance are based upon the idea of challenging intracranial compensatory mechanisms by introducing a rapid increase in volume. Suction catheter insertion, a necessary activity, introduces a rapid increase in intracranial volume. With continuous ICP measurement during ETS, simultaneous ICP response to the increased volume can be assessed. Suction catheter insertion, therefore, provides a readily available qualitative method for nurses to evaluate head injured patients' state of compliance.
CHAPTER 3

METHOD

Design

The data for this study were collected from 1987 through 1989 for a study of the
effects of two versus three suction passes and 100% versus 135% tidal volume
preoxygenation breaths among head injured adults (Rudy et al., 1991). The
present study was a secondary analysis of raw waveform data collected during this
study. The waveforms were not measured, analyzed or used to answer research
questions for the original study.

The first and second research questions were addressed using a quasi-
experimental within-subject pre and post test design (Cook and Campbell, 1979).
The independent variable was suction catheter insertion. The dependent variables
were cerebrovascular response (MICP and CPP) and vasopressor response (MAP
and HR). The third research question was addressed by a quasi-experimental
between-group design. The nominal level independent variable for the third
research question was the presence or absence of altered cerebral compliance
before suction catheter insertion, as measured by ICP waveform configuration.
The dependent variable was cerebrovascular response (MICP) during suction
catheter insertion. Extraneous variables related to the ETS procedure that were
controlled by study design included application of negative suction pressure,
number, rate and volume of hyperoxygenation breaths preceding suction catheter
insertion, FiO₂, and level of coma (Glasgow Coma Scale score).

Sample and Rationale for Inclusion Criteria

The study sample consisted of 30 severely head injured adults between the ages of 15 to 79 years, admitted to either a neurologic or surgical intensive care in the participating facilities. Subjects served as their own controls. Subjects who met the following inclusion criteria were entered into the original study (Rudy et al., 1991) in the order that they were admitted to the participating clinical facilities:

1. Male or female, 15 years or older, admitted to the intensive care unit following a severe open or closed head injury with a GCS score of \( \leq 8 \) for 6 hours or longer after admission. Subjects were limited to at least 15 years of age to eliminate age related neurophysiological developmental differences that could effect individual responses to the ETS procedure. Additionally, because size of ETS suctioning catheter was controlled, adult physical size was required to accommodate the constant catheter size.

2. Presence of an intracranial monitoring device, in place for at least 4 hours. Because the sample was drawn from a critically ill patient population whose condition was in its emergent stages, a minimum of 4 hours after placement of the ICP monitoring device was established to allow subjects time to stabilize from their initial traumatic event.

3. Presence of an arterial blood pressure monitoring device. An arterial
pressure monitoring device was required for continuous data collection of arterial pressure.

4. Intubated and on a volume-limited ventilator. Because breath volume was an independent variable, subjects had to be mechanically ventilated to deliver controlled breath volume. Additionally, it was essential to control rate and number of breaths used to deliver 100% oxygen prior to ETS because arterial oxygen saturation was a dependent variable.

5. Had not received diuretics within one hour before the study’s protocol. Decreased body fluid volume from diuretics temporarily lower ICP. By temporarily diminishing fluid volume, diuretics within one hour of the study protocol could alter subjects MAP and ICP responses to ETS. Because diuretics were given intravenously, the diuretic effect would no longer be a factor after one hour.

6. Had not been suctioned or turned within 15 minutes of the study’s protocol. Past research has demonstrated both turning and suctioning cause ICP to increase. A 15 minute waiting period between either suctioning or turning and the study protocol provided sufficient time for subjects’ physiologic systems to return to resting state.

7. No change in intravenous drip rate of bronchodilating drugs or nebulized bronchodilators within 30 minutes before study protocol. Bronchodilating drugs stimulate sympathetic nerves. Stimulation of sympathetic
nerves lowers ICP through cerebrovascular constriction, that could alter subjects' responses to ETS. The 30 minute time period was sufficient for subjects' physiological systems to return to resting levels.

Of the 30 subjects included in the original sample, 23 (77%) came from the surgical intensive care unit at Cleveland MetroHealth Center (METRO) and 7 (23%) came from the neurosurgical intensive care unit at Maryland Institute of Emergency Medical Services (MIEMS). Number of MIEMS patients meeting inclusion criteria was 37 and total number of METRO patients meeting inclusion criteria was 64. Table 3 displays the reasons patients were excluded from the study.

**Sampling element and inclusion rationale.** The sampling element for the study was the head injured individual who received the endotracheal suctioning sequence. Therefore, there were 30 separate sampling elements (subjects) for the study. Subjects' endotracheal suctioning sequences met the following criteria before they were accepted into the study:

1. The beginning and end of the suction catheter insertion event was clearly identified by electrically generated real time markings, by marks made by data collectors at the time data were collected, or by taking the most conservative estimate of duration from subjects with timing marks. Because the study was based upon a theoretical framework that explained the effect of ETS on head injured adults' cerebrovascular status as a response to irritant receptor stimulation,
Table 3

Reasons for subject exclusion from original study by clinical facility

<table>
<thead>
<tr>
<th></th>
<th>MIEMS</th>
<th>METRO</th>
<th>TOTAL</th>
</tr>
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<tbody>
<tr>
<td>Total Patients</td>
<td>37</td>
<td>64</td>
<td>101</td>
</tr>
<tr>
<td>Total Sample</td>
<td>7 (23%)</td>
<td>23 (77%)</td>
<td>30</td>
</tr>
</tbody>
</table>

Reasons for not participating in study

<table>
<thead>
<tr>
<th>Reason</th>
<th>MIEMS</th>
<th>METRO</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>No family</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Family refused</td>
<td>9</td>
<td>11</td>
<td>20</td>
</tr>
<tr>
<td>Age &lt; 15 years</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>No ICP device</td>
<td>**</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Glasgow Coma Score &gt; 8</td>
<td>**</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Extubated</td>
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<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Died</td>
<td>2</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Other</td>
<td>16</td>
<td>8</td>
<td>24</td>
</tr>
</tbody>
</table>

Note: ** = Missing data.
it was critical that the stimulus (suction catheter insertion) was clearly delineated. Because the study used previously collected raw data, the critical indicator of suction catheter insertion was the temporal duration of the event. Because duration of suction catheter insertion was not controlled, it varied from subject to subject. It was crucial, therefore, that duration was determined to assess whether duration of suction catheter insertion was a covariate.

2. The beginning and end of actual suctioning was clearly identified by either electrically generated real time markings, or by timing marks made by data collectors at the time data were collected. The duration of actual suctioning was clearly marked to assess the relationship between cerebrovascular and systemic vasopressor responses to suction catheter insertion compared to actual suctioning. During actual suctioning, the application of negative suction pressure was controlled to last 10 seconds.

3. Hyperinflation breaths, suction catheter insertion, and actual suctioning were clearly differentiated. A major assumption of the study was that endotracheal suctioning consisted of several components, each with their own underlying physiologic mechanism that could affect cerebrovascular status in head injured adults. To assess how individual components affected cerebrovascular status, it was crucial that the temporal boundaries of each component of the endotracheal suctioning procedure were distinct from other components.

4. Individual intracranial pressure and arterial waveforms were clearly
identified and were not dampened due to mechanical malfunction. Past investigations have assessed cerebrovascular response by investigating ICP aggregate data (trend data) over longer times, or by assessing discrete ICP waveforms for shorter times. Unfortunately, discussions of intracranial pressure often do not differentiate ICP trend from discrete waveform pattern. This lack of clarity was found in discussions of ICP waveform in neurological nursing literature (McGinnis, 1988; Mitchell, 1986).

The inability to differentiate individual waveforms often results from intracranial pressure monitoring device malfunction. The loss of distinct waveform pattern known as dampening, signals that pressure data may be inaccurate due to mechanical problems or displacement of the intracranial or arterial catheter (Shields, 1984).

5. Waveform magnitude, amplitude, and duration were measured in standard units. Characteristics of individual waveforms were this study's focus of analysis. Accurate scale calibration for each physiologic system waveforms was essential so that meaningful comparisons could be made within and among subjects. Measurement in standard units was essential to compare findings of the present study with previous findings. The data collection system used for the study permitted both computerized and manual measurement of wave magnitude and amplitude in standard units (mm Hg.).

**Rationale for sample size.** Power analysis techniques developed by Cohen
(1988) were used to determine the effect the sample size on power. The first and second research questions focused on the effect of irritant receptor stimulation upon subjects’ cerebrovascular status. Both questions were statistically tested using a one sample paired t-test. Based on the theoretical framework posited for the study, hypotheses for question one were unidirectional and predicted that values during suction catheter insertion would be greater than preinsertion scores. Hypotheses for question two were also unidirectional, and predicted an increase during actual suctioning compared to insertion. Because the study was exploratory in nature, an overall risk of Type I error was set at alpha $p = .10$ for each of the first two research questions. To provide protection against Type I error, the Bonferroni procedure was used where the overall alpha values was divided by the number of tests (hypotheses) performed. With the Bonferroni correction, alpha was set at $p \leq .025$ for each hypothesis. The probability of detecting the experimental effect was set at a power $= .80$.

No data are available about the effect of suction catheter insertion on cerebrovascular status. Sample size was determined, therefore, using data from the Rudy et al. (1991) study. A paired t-test was calculated between the average intracranial pressure during baseline (5 minutes of data) and the average MICP during suctioning (data over 10 seconds). Baseline data preceded suction catheter insertion, while actual suctioning immediately followed catheter insertion. Based upon the present study's theoretical framework, cerebrovascular response during
actual suctioning was not expected to exceed response during suction catheter insertion. Therefore, effect size calculated from these data was a conservative estimate for the present study. Sample size was estimated using the following procedure.

First, shared standard deviation was calculated using formula 2.3.2 (Cohen, 1988, p. 44).

\[
\text{Shared } SD = \sqrt{SD_1^2 + SD_2^2}/2
\]

\[
\sqrt{11.5^2 + 10.8^2}/2 = 11.2
\]

Next, effect size was first calculated using the results of the paired t-test between baseline MICP and suctioning MICP.

Formula 2.3.3 (Cohen, 1988, p. 46).

Effect size = mean difference/standard deviation

\[
22.2 - 15.8/11.2 = .5714
\]

Third, the effect size was adjusted to compensate for double error variance contained within power tables developed for two sample t-tests.

Formula 2.3.9 (Cohen, 1988, p. 49).

Effect size = effect size/\(\sqrt{1 - \text{correlation}}\)

\[
.5714/\sqrt{1 - .801} = 1.3
\]

Finally, using linear interpolation for sample size estimation when the effect size is not included in the sample size tables.
Formula 2.4.1 (Cohen, 1988, p. 53).

\[ \text{Sample size} = \text{sample size for alpha.025/100(effect size)}^2 + 1. \]

\[ 1571^*/100(1.3)^2 + 1 = 10 \]

* This value is derived from Table 2.41, p. 55 (Cohen, 1988).

Based upon the preceding computations a sample size of 10 was needed for the paired \textit{t}-test. The study sample size was 30.

The second research question sought to determine if the response initiated by suction catheter insertion was also responsible for cerebrovascular response during actual suctioning. To test this question, cerebrovascular response during suction catheter insertion was compared the response during actual suctioning. It was hypothesized that if suction catheter insertion was the major factor, there would be no significant difference between cerebrovascular response during suction catheter insertion and during actual suctioning. Because the desired finding was to \textit{not} reject the null hypotheses, power analysis was not of relevance for the second research question.

Research question three compared cerebrovascular response during catheter insertion of subjects with decreased cerebral compliance with subjects who had adequate compliance. The third question was tested using the \textit{t}-test for independent groups. Because the third question explored the ability of a new indicator to differentiate state of cerebral compliance (waveform configuration),
Type I error is set at $p \leq .10$. Because there were no data from previous research available to determine needed sample size, an estimation technique recommended by Cohen (1988) was utilized. A large effect size of .8 was selected, because difference between group ICP responses during suction catheter insertion was anticipated to quite visible. Based upon Table 2.4.1, with an effect size of .8, power value = .80, and $alpha = .10$, a sample size of 28 was sufficient to demonstrate between group differences (Cohen, 1988, p. 54).

The independent $t$-test is robust to moderate violations of assumptions of normality and variance equality. Unfortunately it is sensitive to departures of homogeneity of variance when sample sizes are markedly different (Ott, 1984). Sample size difference of greater than 1.5 to 1 requires substitution of either a $t'$-test statistic, or a nonparametric test, such as the Wilcoxin test, for the standard $t$-test. The standard independent $t$-test was used because groups were similar in size. The normal compliance group consisted of 16 subjects, while the decreased compliance group had 14 subjects.

**Rationale for sampling design.** Because the study was based upon analysis of existing data, a nonprobability sampling strategy was used. Because the size of the accessible population was known, it was reasonable to include all sampling elements that met the inclusion criteria. Based upon the preceding analysis of sample size, the accessible population was adequate to test the study’s research questions. An additional strong endorsement for including all available subjects
that met inclusion criteria was the access to data that requires complex
instrumentation to study questions about endotracheal suctioning, in a clinical
population of limited size.

Findings from this study were generalizable to all head injured adults who
match the study inclusion criteria. All severely head injured adults in the acute
phase of their injury share similar head injury pathology. Additionally, during the
acute illness phase, the severely head injured require endotracheal suctioning
because they are intubated and mechanically ventilated. Therefore, findings from
the study were cautiously generalized to the severely head injured adult
population, in the acute phase of their injury.

**Human rights.** Because the study did not require access to subject names,
patient data were accessed solely by subject code number. Data were identified
by the same subject code number assigned during the original study. All data
were stored in a locked area. The study was exempt from review of Human
Rights Committee approval, because no contact was made with subjects.

**Potential risks and benefits.** Because the study used data collected during
the study conducted by Rudy and colleagues (1987), the only risk to subjects was
breach of confidentiality. The investigator insured that confidentiality was
maintained by accessing raw data solely by subject code number.

Subjects received no direct benefits from the study. The study benefits
future head injured adults, because it seeks to identify how endotracheal suctioning

causes potentially dangerous cerebrovascular responses. The identification of the mechanism through which endotracheal suctioning initiates a cerebrovascular response is necessary for the development of nursing interventions to prevent iatrogenic injury to severely head injured adults.

**Study Protocol**

Data were collected from both hard copy and computerized subject files available from the study by Rudy and colleagues (1987). The experimental protocol for the Rudy study included the following components:

**Hyperoxygenation - Suction Protocol**

2 Suction Sequence

| 5 minute Baseline Data | 4 LH Breaths | 10 sec. ETS | 4 LH Breaths | 10 sec. ETS | 4 LH Breaths | 10 minute Postprotocol Data |

3 Suction Sequence

| 5 minute Baseline Data | 4 LH Breaths | 10 sec. ETS | 4 LH Breaths | 10 sec. ETS | 4 LH Breaths | 10 sec. ETS | 4 LH Breaths | 10 minute Postprotocol Data |

**Figure 6.** Original endotracheal suctioning experimental protocol

The major difference between the original experimental protocol (Figure 6) and the current study protocol (Figure 7) was that the original study excluded all suction catheter insertion waveform data.
Figure 7. Current study endotracheal suctioning sequence

Figure 7 demonstrates the relation between endotracheal suctioning procedure events and the current study’s three major research questions. Only one endotracheal suctioning sequence that consisted of resting baseline, four hyperoxygenation breaths, suction catheter insertion, and actual suctioning were used for the present study. The cumulative effects from repeated catheter insertion and suctioning are avoided, by using only one endotracheal suctioning sequence per subject.

Study procedure

Entry into study. All endotracheal suctioning sequences (N = 30) were previewed for entry into the current study. Both hard copy and computer files were initially screened to determine if they met study inclusion criteria. Determination for study inclusion was judged using the following procedure:
1. Both hard copy and computerized ETS sequences were checked for temporal markings that indicated the duration of hyperoxygenation breaths, suction catheter insertion, and actual succioning. The ETS sequence was accepted if sequence components were marked on either the hard copy or computerized data. Acceptable marks included either electronic (voltage) indication of component duration on either computerized data or hard copy, or marks drawn by data collectors during data collection.

2. Both hard copy and computerized data were screened to determine if individual intracranial pressure and arterial waveforms were clearly visible, and were not dampened due to mechanical malfunction. Waveforms were screened for clarity at minutes 4 and 5 minutes before the beginning of the succioning procedure, during hyperoxygenation breaths, during suction catheter insertion, and during actual succioning. Waveforms were accepted if, for an individual endotracheal succioning sequence, the following criteria were met:

   a. Discrete waveforms were identified by the naked eye on either hard copy or computer screen, or discrete waveforms were identified by the naked eye with computerized magnification.

   b. Discrete waveforms were present at each component of the study protocol (see Figure 7).

4. Instrument calibration of ICP and MAP that preceded data collection during the ETS experimental protocols (Figure 6) were examined for:
a. Documentation that measuring instruments were checked for linearity between pressure transducers, computerized recording device, and chart recorder.

b. Vertical and horizontal scale ranges were specified.

**Data collection procedure.** To assure internal validity during initial data collection (at the clinical site), the following steps were implemented. When patients were entered into the study they were randomly assigned by a random number sheet, to receive either two or three suction passes and either 100% or 135% tidal volume for oxygenation breaths. Later analysis showed no differences between the 100% and 135% tidal volume groups on any of the dependent variables. Fifteen minutes before initiation of the experimental protocol, all subjects were placed on their backs with head of bed elevated 30 degrees. Bed elevation was measured by a protractor to assure consistent head elevation.

The ICP transducer was placed at approximately 3 cm. above the external auditory canal (about the top of the ear lobe). The transducer to monitor MAP was placed at the level of the left atrium. The transducer to monitor airway pressure was placed in line with a T-piece at the tip of the subjects' endotracheal tube. For the six subjects studied at MIEMS, the ICP transducer was at the top of the head and the transducer was calibrated at that level.

Electrocardiographic measurements of heart rate were recorded directly from the Spacelab monitoring device to the Gould TA2000 recorder. Direct
connections from the measuring devices to the Gould TA2000 recorder provided continual recordings of the MAP, MICP, and HR, during the ETS protocol (CPP is a calculated value). The pressure transducers were zeroed and calibrated against mercury and recorded on the Gould Recorder. Subject’s delivered volume was determined by a Magtrack II respirometer and set to deliver either 100% or 135% tidal volume. The wall mounted suction regulator was set to generate a suction flow rate of 16L/min. Flow rate was measured by a calibrated Fischer and Porter rotameter. The rotameter was placed between connecting tubing and the suction catheter. After measurement, the rotameter was removed.

A second primed ventilator was used to deliver 100% O₂ during the ETS procedure and to avoid problems with washout of O₂ through the ventilator tubing of the subject’s own ventilator. A second ventilator assured delivery of 100% O₂ and consistency of hyperinflation volumes. FiO₂ concentration of the patient’s inflow tubing of the ventilator was increased to 100% and tested by a calibrated oxygen analyzer to assure 100% delivered O₂. As soon as the delivered air of the ventilator was at 100% O₂ the suction protocol was initiated.

The patient was suctioned by trained staff according to the hyperoxygenation protocol. The dependent variables were monitored continuously five minutes prior to the hyperoxygenation/suctioning protocol, during the hyperoxygenation/suctioning protocol, and ten minutes post hyperoxygenation/suctioning protocol (see Figure 6).
For the present study, descriptive data were collected from subject files. Descriptive data included age, gender, size of hyperoxgenation volume (100\% or 135\% \text{V}_\text{T}), type of head injury, administration of barbiturates, mannitol, paralytic agents, or sedatives given within one hour prior to or during the study protocol, most recent \text{PaCO}_2 value, mortality and morbidity at time of discharge from acute care setting, tobacco and alcohol use.

All ICP (MICP, ICPA, ICP configuration), MAP, and HR waveforms were measured for five second duration, at the fourth and fifth minute immediately preceding initiation the endotracheal suctioning procedure. ICP, MAP, and HR were measured for the duration of hyperoxgenation breaths, during ventilator disconnection, during suction catheter insertion, and during actual suctioning. CPP was calculated (MAP - MICP) for each time point.

Waveform measurement was performed using a computerized data display and analysis program, VIEW II that was developed by Gould Electronics. Characteristics of individual waveforms could be analyzed, and mean values of both individual and group waveforms could be analyzed by the VIEW II program. Internal validity and reliability were supported by the following steps:

1. In addition to computerized measurement, the investigator measured every 10th waveform variable per case on hard copy using a magnified calibrated visual scale (calibrated to 0.1 mm. intervals) developed by Bausch and Lomb.

2. A second person measured every 10th waveform variable per case by
computer using established rules of measurement.

3. Whenever the measurement methods had a greater than 10% disparity in values, the individual measurements were compared to identify the source of the discrepancy. Whenever a disparity was identified, every 5th waveform variable of that case was measured to insure 90% reliability.

**Instrumentation**

Three physiological parameters (MAP, MICP, and HR) were recorded simultaneously before, during and following the ETS sequence by means of a calibrated Gould TA2000 recorder. The Gould recorder is a rectilinear recorder which contains amplifiers (to increase signal), filters (to eliminate noise), and a thermal array to record graphically the data being collected. The Gould TA2000 recorder, after a 15 minute warm up time, has a reported gain stability of 0.05% for 24 hours.

The recorder is part of Gould Electronics DASA (data acquisition and signal analysis) system. The recorder was used in conjunction with a data acquisitions and storage software program, called ACQ-4600, also developed by Gould Electronics. ACQ-4600 provided the capability for computer based recording and storage of the electronic signal that resulted from the conversion of physiologic pressure by a sensor-transducer (Abbey, 1990). Both MAP and ICP were converted to an electrical signal by a Statham pressure transducer that allowed the data to be stored in a computer and recorded on paper.
Arterial pressure measurement. Arterial pressure was measured through an indwelling radial arterial line using a Spectramed P23XL pressure transducer connected to the Gould recorder. Reliability of arterial pressure was assured by calibration of the Gould recorder against a column of mercury before implementation of each protocol. Validity of the measurement depends on proper placement of the arterial catheter and was evaluated by presence of proper waveforms on the recorder tracings.

Intracranial pressure measurement. Intracranial pressure was measured by an indwelling monitoring device in the intracranial space attached to a Spectramed P23XL pressure transducer. Reliability of intracranial pressure measurement was assured by calibration of the Gould recorder against a column of mercury prior to implementation of each protocol. Validity of the measurement depends upon proper placement of the intracranial device and was evaluated by the presence of a proper waveform on the recorder tracings.

Heart rate measurement. The third channel of the Gould TA2000 recorder recorded the heart rate directly from the patient’s Spacelab monitor device. Validity of the measurement was determined by comparison with the subject’s pattern and rate displayed on the bedside monitor.

Suction flow rate measurement. The suction flow rate of negative pressure was measured by a calibrated Fisher and Porter rotameter, a glass meter tube with a thallium ball. The rotameter has a reported accuracy of ± 2% full scale with a
repeatability of 0.5% full scale. The suction flow rate was controlled at 16.67 L/minute.

The Glasgow Coma Scale (GCS). The GCS is an instrument developed at the Glasgow Neurological Institute in Scotland by Teasdale & Jennett (1974) to assess level of consciousness in the closed head-injured patient. The GCS has been extensively tested for its ability to assess and determine prognoses of head injured patients in many countries of the world, including the United States. The GCS score is reliable with minimal observer deviation during testing by nurses and physicians (Teasdale, Knill-Jones & Jennett, 1974). The scale has three aspects of behavioral response that are evaluated: best verbal response, best motor response, and eye opening. The original scale ranged from a high score of 14 to a low of 3. The scale was expanded later by the addition of another possible motor response. This addition brings the highest possible score to 15 (Finklestein & Ropper, 1979). The 15 point scale is the more widely used and replicated in the literature. The expanded scale was used to assess level of consciousness for each subject in this study.

The purpose of the current study was to determine the effect of suction catheter insertion on head injured adults' cerebrovascular status, and to examine how cerebral compliance affects cerebrovascular response to suction catheter insertion. Because data for the study were derived from previously collected raw waveform data, the focus of instrumentation for the current study was the
reliability and validity of procedures for waveform measurement.

As previously discussed, data were recorded with a Gould TA2000 recorder (hard copy) and ACQ-4600 software program (computer file). Interactive with the Gould data recording system is VIEW II, a data display and analysis software program also developed by Gould Electronics (1989). The VIEW II program allows measurement, manipulation and analysis of electronic signal input stored by the ACQ program. The VIEW II program was used to measure waveform data for the current study.

Mean arterial pressure (MAP) waveform measurement. Single wave MAP was derived using the following procedure to measure stored arterial pressure signal data. Using the VIEW II program:

1. Identify and mark that point on a waveform that corresponds to the highest mm Hg. as identified by VIEW II.

2. Identify and mark that point on a waveform that corresponds to the lowest mm Hg. after the diastolic wave as identified by VIEW II.

3. Select and run the calculation function for mean value.

To calculate an average MAP from more than one arterial pressure waveform:

1. Identify and mark the beginning and end of arterial waveform during the event, such as suction catheter insertion.

2. Select and run the calculation function for mean value.

VIEW II calculates an arithmetic mean, where \( M = \) sum of samples of
arterial pressure (mm Hg.)/number of samples. Data were collected at a rate of 25 samples per second. Fidelity of values is a function of samples per second, that is influenced by wave amplitude. For example, samples with values (mm Hg.) near the upper range of arterial pressure scale (250 mm Hg.), would be less represented than samples with values near the lower range of pressure (0 mm Hg.). The mean value calculated by VIEW II is somewhat weighted towards the lower sample values. Therefore, the program produces mean values very close to MAP values calculated by the mean formula of \(2 \times \text{diastolic} + \text{systolic}/3\).

To assure reliability, arterial waveforms for 10% of MAP variables per case were measured and MAP calculated by a second trained person. MAP values (mm Hg.) calculated by each person were required to be within 10% of each other. When there was a greater than 10% difference in the two MAP measurements, the two raters compared procedures to identify the basis for difference in values.

Construct validity for MAP as a measurement of the vasopressor response was based upon the theoretical framework posited for the study (see Chapter one, Figure 4). Criterion validity was assessed by the additional measurement of arterial waveforms on hard copy. Ten percent of MAP waveforms were measured per case using the following procedure:

1. Identify the waveform on hard copy that corresponds with the computerized waveform. Identification of the same waveform was assured by
matching timepoints in the ETS sequence.

2. Measure the waveform using the Bausch and Lomb magnified scale that is calibrated to 0.1 mm Hg.

3. Identify that point on the waveform that corresponds to the highest mm Hg. on the magnified scale.

4. Identify that point on the waveform that corresponds to the lowest mm Hg. on the magnified scale after the diastolic wave.

5. Calculate the mean value using the formula: \(2 \times \text{diastolic} + \frac{\text{systolic}}{3}\).

To be accepted, values obtained from hardcopy and computer generated waveforms had to be within 10% of each other. If values are greater than 10%, the computerized measurement process and hard copy process were compared to identify the source of the value difference. When procedure error was not the basis for the difference, the hard copy value was accepted as the more accurate value.

**Heart rate (HR) measurement.** Limitations of the data recording and acquisitions system provided the capability to record either heart rate pattern input or heart rate frequency. Because only heart rate pattern was recorded, heart rate was calculated from the hardcopy using the following procedure.

1. Select the temporal point that matches the component of the ETS sequence to be assessed.

2. Select the midpoint of the component and count the number of cardiac
waves for five seconds on either side of the midpoint.

3. Multiply the number of r waves occurring during 10 seconds by six to obtain beats per minute.

To assure reliability, 10% of the heart rate variables per case were calculated by a second trained person. Heart rate per minute values were required to fall within 10% of each other. When the difference in heart rate was greater than 10%, the raters compared procedures to identify the basis for the difference in values. Construct validity for HR as a measurement of vasopressor response was based upon the study's theoretical framework (see Chapter one, Figure 4).

ICP waveform magnitude (MICP) measurement. Measurement of MICP from intracranial pressure waves was performed by exactly the same process as the measurement of MAP from arterial pressure wave data. The same procedures as specified for MAP were used to address reliability and validity of MICP measurement.

ICP waveform amplitude (ICPA) measurement. Measurement of ICPA was completed using the same electronic signal data as that was used to measure MICP. However estimation of ICPA involved a different process. The procedure to measure ICPA included the following computerized process using VIEW II.

1. Identify and mark that point on a single waveform that corresponds to the highest mm Hg. as identified by VIEW II.

2. Identify and mark that point on a single waveform that corresponds to
the lowest mm Hg. as identified by VIEW II.

3. Select and run the calculation function for the difference between the selected highest and lowest waveform points (MAX Y-MIN Y).

To assure reliability, wave amplitudes for 10% of ICPA variables per case were measured and calculated by a second trained person. The two raters ICPA values were accepted when they fell within 10% of each other. Whenever ICPA values exceeded the 10% criterion, the two raters compared measurement procedures to identify the source of the difference.

Construct validity for ICPA as a measurement of cerebral compliance was based upon the study's theoretical framework (see Chapter one, Figure 4). Criterion validity was assessed by the additional measurement of 10% of all ICPA high and low variables on hard copy. The procedure included the following steps.

1. Identify the waveform on hard copy that corresponds with the computerized waveform. Identification of the same waveform will be assured by matching time points in the ETS sequence.

2. Measure the waveform using a magnified scale that is calibrated to .01 mm Hg.

3. Identify that point on the waveform that corresponds with the highest mm Hg. on the magnified scale.

4. Identify that point on the waveform that corresponds to the lowest mm Hg. on the magnified scale.
5. Calculate the difference between the two values by subtracting the smaller from the larger value.

Hardcopy and computer generated waveform ICPA values were required to be within 10% of each other. When the difference in ICPA values were greater than 10%, both computerized and hardcopy measurement were scrutinized for the source of error. When procedure error was not identified, the hardcopy value was accepted as correct.

**ICP waveform configuration measurement.** ICP configuration was measured using both stored ICP electronic signal data and hardcopy waveforms. The purpose of measuring ICP configuration was to classify head injured adults with and without decreased compliance. Although cerebral compliance is a continuous, rather than dichotomous phenomenon, due to instrumentation limitations, subjects were identified as either having decreased or normal compliance.

**Normal compliance criteria.** Exemplar ICP waveforms representing normal compliance are displayed in Appendix A. Normal compliance waveforms met the following criteria:

1. Waveforms have initial upward inflection then descend back to baseline with a gradual slope.

2. Alternatively, waveforms have an initial upward inflection then remaining level, followed by descent with small slope to baseline (Chopp &
3. If subwaveforms are visible $P_1$ will have greater amplitude than subwaves following it.

**Decreased compliance criteria.** ICP configuration representing decreased compliance is represented by an exemplar waveforms in Appendix B are characterized by:

1. The initial upward wave inflection is rounded or peaked, followed by a steep descent (slope) to baseline.

2. If ICP waves are peaked, ICP waveform configuration is similar to the subject’s arterial waveform configuration (Portnoy & Chopp, 1981, Portnoy et al. 1982).

3. If subwaveforms are visible, subwaveforms following the $P_1$ subwaveform will be of equal or greater amplitude than $P_1$ (Cardoso et al., 1984).

**Cerebral compliance categorization and measurement.** The following procedure was used to classify ICP waveforms as representative of decreased or normal compliance:

1. Select all ICP waves that occur within a five second interval at minutes four and five immediately prior to initiation of the ETS sequence. If 50% of a wave falls within the five second interval, it will be included in the measurement.

2. Using the preceding criteria for decreased and normal compliance, compare hardcopy and/or computer stored waveforms with exemplar waveforms
in Appendices A and B.

3. Each five second interval is judged as representing normal or decreased compliance when > 50% of the waveforms are classified as normal or decreased compliance.

4. The subject is classified as having decreased or normal compliance by the majority classification of the waveform at the two time points.

In addition to the classification of subjects as having decreased or normal compliance by ICP configuration, the descending slopes of the waveforms at minutes four and five were measured to obtain a more quantitative configuration measure of cerebral compliance. The underlying assumption of the descending slope measurement was based upon previous findings that ICP peaking and rounding are accompanied by a steep descending slope. Therefore, increased slope indicates steeper descent to baseline. The VIEW II program calculates slope with the formula $Y_2 - Y_1 \text{ (ICP mm Hg.)} / X_2 - X_1 \text{ (seconds)} = \text{ICP mm Hg. per second}$. Because the ICP wave descending slope were assessed, the resulting slope values were always be negative. The procedure for measuring ICP descending slope included the following steps.

1. Identify and mark the highest point (mm Hg.) of the descending slope.

2. Identify and mark the lowest point (mm Hg.) of the descending slope, following the diastolic wave.

3. Select and run the calculation function for slope.
To insure categorization reliability, every subject was assessed and categorized as having decreased or normal compliance by a second person, a registered nurse with critical care experience. 100% categorization agreement was required. Whenever there was disagreement, the two raters reviewed their decision process until agreement was reached.

10% of all waveform slope measurements were completed by a second trained person at the two baseline time points (minutes 4 and 5). Slope values of the two raters were required to reach 90% agreement. When slope value measurements exceeded the 10% criterion, the two raters compared procedures to find the source of the difference in values.

Data analysis and management

Data analysis and management were the responsibility of the investigator in consultation with a statistician. All descriptive and waveform data for each subject were first entered into a subject code book. Each subject’s data were accessed and identified solely by case number, thus maintaining complete subject confidentiality. Each subject’s code book consisted of a hardcopy printout recorded by the Gould TA2000 recorder of ICP, MAP, HR, and lung volume waveforms. In addition to waveform hardcopy, 18 subjects had waveform computer files that were kept on floppy disks. As previously discussed, waveform data for each subject were measured to obtain designated measurements for each variable. Measurement was accomplished both with a Bausch and Lomb
measuring magnifier for hardcopy waveforms, and with the Gould VIEW II waveform analysis program for computerized files. Intracranial and systemic arterial wave values (mm Hg.) were then used to calculate both individual waveform mean values, and average values of all waves that occurred during a specified event (for example, during suction catheter insertion). Heart rate was calculated by counting beats per minute for the duration of an event.

Two processes were used to insure accuracy of waveform measurements. First, 10% of all computerized waveforms were measured with an alternate procedure. The alternate procedure included the measurement of duplicate waveforms on hardcopy using a Bausch and Lomb magnifier. Waveform values obtained from the two measurement processes had to be within 10% of each other. The second process used a second person who measured 10% of all waveforms for every subject. The second person was a Masters prepared RN with 27 years experience in both medical-surgical and critical-care nursing. Percentage of error between first and second measurer had to be equal or less than 10% using the following formula:

\[(\text{Value A} / \text{Value B}) - 1 \times 100 = \% \text{ error}\]

Comparison of measures demonstrated at least 90% accuracy for all waveform measurements.

The second rater also categorized every subject as having either normal or decreased compliance (see the preceding discussion of Cerebral compliance
categorization and measurement). Using an iterative process between the researcher and second person, 100% agreement was obtained for subject compliance categorization. The second rater’s measurements were entered directly into each subject’s code book. Percentage of inter-rater agreement was calculated and documented in each subject’s codebook.

The researcher entered data into a computer file in preparation for statistical analysis with the Statistical Program for the Social Sciences (SPSS/PC V4.0, 1990). Computerized data were assessed for errors through an iterative procedure where all data were printed for every variable, checked for errors, corrected, rerun and checked again. After initial data cleaning, frequencies were printed for all variables. All extreme values, missing values and any other unusual values were identified by case and compared with the subject’s code book data. All categorical data were checked and corrected by category.

**Statistical procedures**

Data were analyzed using descriptive and inferential procedures. Additionally, graphical analysis were performed to visually analyze data trends over time. Frequency distributions, measures of central tendency and dispersion were calculated to describe descriptive variables and all dependent variables.

Data points used for analysis included baseline (average value of waveforms over 10 seconds, highest and lowest individual waveform during 10 seconds), hyperoxygenation (average value of waveforms during delivery of four
breaths, highest and lowest individual waveform during the four breaths),
ventilator disconnection (average waveform values during disconnection, highest
and lowest individual waveforms), suction catheter insertion (average of all
waveforms during insertion, highest and lowest individual waveform), actual
suctioning (average of waveforms during actual suctioning = 10 seconds, highest
and lowest individual waveform).

Multiple ICP, MAP, and HR waveforms occurred during each suctioning
event. Average ICP and MAP magnitude, ICP amplitude, HR beats per minute,
and CPP (calculated value) were determined for each event. At baseline, ICP
configuration and descending slopes were measured in addition to all of the
preceding measures.

Because all data generated for the dependent variables were at interval
level of measurement, parametric statistical procedures were applied. Data were
assessed for violations of statistical assumptions. Normal distribution of all
interval level variables was assessed using histograms, normal and detrended
probability plots, Lillifors test for normal distribution, tests for skewness and for
kurtosis. Extreme values were identified via boxplots, and Z-scores were
calculated for all extreme values. Any data with Z-scores \( \geq 3 \) standard
deviations were classified as outliers.

Data grouped by the presence of normal or decreased cerebral compliance
were also assessed for normal distribution using all of the preceding procedures.
Additionally, equality of variance between the two groups was assessed using spreadlevel plots and the Levine test for homogeneity of group variances.

**Inferential testing of hypotheses.** Directional hypotheses were posed when direction was predicted by the study’s theoretical model. The following research questions and their hypotheses were tested in a sample of severely head injured adults. Dependent t-tests were used to address each of the hypotheses for Research Questions one and two. To avoid an unacceptably high risk of Type I error, the Bonferroni procedure was used to correct alpha level per hypothesis. Alpha level was determined using the following formula where overall alpha was set at \( p - .10 \): \( \alpha / \text{number of tests} = p \cdot .10 / 4 = .025 \) per hypothesis.

RQ1a. Is there a difference between cerebrovascular response (MICP and CPP) and vasopressor response (MAP and HR) during suction catheter insertion compared to baseline values?

H1a. There will be an increase in MICP during suction catheter insertion compared to MICP at baseline.

H1b. There will be an increase in CPP during suction catheter suction insertion compared to CPP at baseline.

H1c. There will be an increase in MAP during suction catheter insertion compared MAP at baseline.

H1d. There will be an increase in HR during suction catheter insertion compared to HR at baseline.
Hypotheses H1a through H1d were analyzed using a dependent t-test to assess whether the dependent variables (MICP, CPP, MAP and HR) were increased with suction catheter insertion compared to prior to catheter insertion.

RQ2. Is there a difference in cerebrovascular response (MICP and CPP) and vasopressor response (MAP and HR) during actual suctioning compared to suction catheter insertion?

H2a. There will be an increase in MICP during actual suctioning compared to MICP during suction catheter insertion.

H2b. There will be an increase in CPP during actual suctioning compared to CPP during suction catheter suction insertion.

H2c. There will be an increase in MAP during actual suctioning compared to MAP during suction catheter insertion.

H2d. There will be an increase in HR during actual suctioning compared to HR during suction catheter insertion.

Hypotheses H2a through H2d were analyzed using a dependent t test to assess whether the dependent variables (MICP, CPP, MAP and HR) were increased with actual suctioning compared to suction catheter insertion.

RQ3. Is there a difference in cerebrovascular response (MICP) during suction catheter insertion, between subjects with decreased cerebral compliance (ICP rounding) and those with normal compliance (Normal ICP configuration)?

H3a. There will be a greater increase in MICP during suction catheter
insertion in subjects with decreased cerebral compliance at baseline compared to those with normal compliance.

An independent t-test with an alpha of .05 was used to test hypothesis H3a.

RQ4. Do differences between cerebrovascular response and vasopressor response during suction catheter insertion and baseline hold when duration of suction catheter insertion is controlled?

H4a. There will be no difference between MICP during suction catheter insertion compared to MICP at baseline.

H4b. There will be no difference between CPP during suction catheter suction insertion compared to CPP at baseline.

H4c. There will be no difference between MAP during suction catheter insertion compared to MAP at baseline

H4d. There will be no difference between HR during suction catheter insertion compared to HR at baseline.

RQ5. Do differences between cerebrovascular response and vasopressor response during suction catheter insertion and actual suctioning hold when duration of suction catheter insertion is controlled?

H5a. There will be no difference between MICP during suction catheter insertion compared to MICP during actual suctioning.

H5b. There will be no difference between CPP during suction catheter suction insertion compared to CPP during actual suctioning.
H5c. There will be no difference between MAP during suction catheter insertion compared MAP during actual suctioning.

H5d. There will be no difference between HR during suction catheter insertion compared to HR during actual suctioning.

Research questions four and five were not tested because linear correlations between duration of suction catheter insertion, ICP and CPP during insertion and actual suctioning, were correlated less than .60 (see Table 4). Based upon the low bivariate correlation between suction catheter duration and the cerebrovascular variables, it was not necessary to statistically control for any effect from duration. Testing the hypotheses with dependent t-tests as specified under Research Questions one and two was judged sufficient to answer both questions.
Table 4

**Bivariate Correlations Between Dependent Variables ICP and CPP During Suction Catheter Insertion and Actual Suctioning with Potential Covariate Measure, Suction Catheter Insertion Duration (N = 30)**

<table>
<thead>
<tr>
<th>Variables by Research Questions and Hypotheses</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Research Question 1</strong></td>
<td></td>
</tr>
<tr>
<td>Hypothesis 1. No difference between baseline ICP and insertion ICP.</td>
<td>-.1406</td>
</tr>
<tr>
<td>Hypothesis 2. No difference between baseline CPP and insertion CPP.</td>
<td>.0732</td>
</tr>
<tr>
<td><strong>Research Question 2</strong></td>
<td></td>
</tr>
<tr>
<td>Hypothesis 1. No difference between insertion ICP and suction ICP.</td>
<td>-.1358</td>
</tr>
<tr>
<td>Hypothesis 2. No difference between insertion CPP and suction CPP</td>
<td>.1977</td>
</tr>
</tbody>
</table>
CHAPTER 4

Results

This chapter begins with a description of study sample characteristics. Description of the study sample is summarized both for the overall sample and for subjects grouped by their state of cerebral compliance. Major study variables are summarized through measures of central tendency and dispersion. Results from testing for violations of statistical assumptions are summarized.

Data were tested for their ability to meet statistical assumptions, both for the overall sample and for the sample grouped by state of cerebral compliance. Next, findings are presented from inferential testing of hypotheses for each of the three research questions. Where appropriate, the possible influence of variables on ICP response to suction catheter insertion were tested inferentially, with alpha set at .05. Finally, results of post hoc analyses are presented.

Sample Description

Demographic Characteristics

The sample consisted of 30 severely head injured adults in the acute phase of their injury. As discussed in Chapter 3, subjects for the study were part of a previous study conducted by Rudy and others (1987). Subjects' ages ranged from 15 to 79 years old, with an average age of 31(SD = 15) years. The majority of
study subjects were between 19 to 31 years of age (n = 20), with a second peak incidence in subjects who were 51 years old (n = 3). Twenty six males (87%) and four females (13%) formed the sample.

Seven subjects (23%) were patients at the Maryland Institute of Emergency Medical Services, while the remaining 23 (77%) were patients in the Surgical Intensive Care Unit at Cleveland MetroHealth Medical Center. Twenty-eight subjects (93%) were diagnosed with closed head injuries, while the remaining two subjects (7%) had open head injuries.

Physiological Characteristics

Intracranial pressure (ICP) was measured with a Richmond bolt in 25 subjects (83%), with a Camino catheter in two subjects (7%), and with an assortment of other devices in three subjects (10%). The decision to use a given type of ICP device was the purview of the neurosurgery medical staff, therefore, type of device could not be controlled by study design. Because type of monitoring device was a potential source of extraneous variation in ICP data, baseline MICP was compared between subjects with Richmond bolts (M = 15, SD = 9 mm Hg.) and those with other devices (M = 19, SD = 15 mm Hg.). Using a Mann-Whitney U test for the comparison, no difference in baseline MICP was found between the two groups, U = 53.5, p > .05.2

2The Mann-Whitney U test was used for comparisons because of substantial group size differences, and because the smaller group’s variance (SD² = 211) was substantially larger than the larger group (SD² = 89).
Eleven subjects (37%) used tobacco products at the time of injury, 15 (50%) did not use tobacco, and tobacco use was not documented in 4 (13%) subjects. Tobacco use was of particular interest to this study because nicotine is known to excite both sympathetic and parasympathetic systems (Guyton, 1991). Subjects' tobacco use could alter airway irritant receptor activation in response to suction catheter insertion, while chronic tobacco use could alter baseline ICP though its systemic effects. To assess these possible extraneous effects of tobacco, ICP at both baseline and insertion was compared between smokers and nonsmokers. Although the smoking group had a higher average MICP at both baseline and insertion ($M = 17$ mm Hg. and $20$ mm Hg.) than nonsmokers ($M = 14$ and $17$ mm Hg.), there were no significant differences either at baseline or during insertion (baseline $U = 80$, $p > .05$, insertion $U = 80.5$, $p > .05$)\(^3\).

Subjects' Glasgow Coma Scale (GCS) scores were assessed within one hour prior to the start of the study protocol. GCS scores ranged from 3 to 9, with an average score of 6 ($SD = 2$). With the exception of one subject who had a GCS score of 9, all subjects met the study criteria for severe coma (severe coma = GCS score $\leq 8$). The sample was almost evenly divided, with 14 (47%) subjects in deepest coma (GCS scores $\leq 5$), and 16 (53%) subjects in deep but

\(^3\)The Mann-Whitney U test was used because of the differences in variances. Smaller group variance at baseline was $SD^2 = 219$ compared to $SD^2 = 35$ for the larger group. Similarly, at insertion the smaller group’s variance was 225, while the larger group had $SD^2 = 48$. 
less profound coma (GCS scores $\geq 6$).

Arterial blood carbon dioxide ($\text{PaCO}_2$) and pH data were collected from the most recent blood gas sample drawn prior to the suctioning procedure. The sample's alkalotic state, demonstrated by average $\text{PaCO}_2$ values of 30 (SD = 5) mm Hg. and mean pH values of 7.47 (SD = .05), resulted from therapeutically induced hyperventilation. Because respiratory alkalosis was induced to decrease ICP, subjects' ICP responses to suction catheter insertion could have been related to their respiratory acid-base status. Linear correlations of arterial pH and $\text{PaCO}_2$ levels with ICP at baseline and at suction catheter insertion, revealed no significant relationship between pH level and ICP ($r^2$ baseline = .01, $r^2$ insertion = .02) or ICP and $\text{PaCO}_2$ ($r^2$ baseline < .001 and $r^2$ insertion < .001).

**Description of Medications Received Before Suctioning Procedure**

The primary interest in barbiturates, paralytic medications, and sedatives was their possible effects upon subjects' ICP responses during suction catheter insertion. With the exception of barbiturates, each of the medications would be absorbed and active within an hour. Therefore, data were collected for medications administered within the hour preceding the initiation of the suctioning procedure. Because pentobarbital has a prolonged suppressive effect on cerebral metabolism, cerebral blood flow, and therefore ICP, data were collected for pentobarbital administration during the 24 hour period prior to data collection. Five subjects (17%) received mannitol, six (20%) were medicated with paralytics,
and five (17%) received sedatives prior to the study protocol. Only three subjects (10%) received barbiturates within the preceding 24 hours before initiation of the study protocol. Data for medications received within one hour prior to suctioning were not available for six subjects.

Using independent t-tests, ICP values at both baseline and suction catheter insertion were compared between subjects who received medications and those who did not receive medications. No significant differences in ICP values were found between subjects who received medications and those who did not receive medications.

Sample Description Grouped by State of Cerebral Compliance

Normal and Decreased Compliance Group Criteria

Because the third research question compared subjects with normal and decreased compliance, the sample was described by the dichotomous compliance categorization. Using preestablished criteria, subjects' baseline ICP waveforms were compared with exemplar ICP waveforms derived from the literature, that represented either normal or decreased cerebral compliance. ICP waveforms were compared for general shape of the ICP waves, the steepness of its descending slope, and the relationship of the height among ICP wave subwaveforms (see Chapter 3 for classification criteria, and Appendices A and B for exemplar

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4 Independent t-tests were used for comparisons because although group size was substantially different, group variances were similar.
waveforms). Sixteen subjects categorized with normal compliance had baseline ICP waveforms that, after initial positive inflection, displayed a plateau and gradual descent to baseline. Additionally, normal compliance ICP waveforms displayed $P_1$ subwaveforms that were slightly greater in amplitude than $P_2$ subwaveforms. Based upon ICP waveform characteristics, 14 subjects were classified with decreased cerebral compliance. Waveform criteria for decreased compliance included rounded or peaked ICP waveforms with a steep descent to baseline and predominant $P_2$ subwaveforms.

In an exploratory effort to further quantify the process of using ICP configuration to identify state of cerebral compliance, the descending slopes of all baseline ICP waves were measured. As shown in Table 5, subjects classified as having decreased compliance had steeper descending ICP slopes than did the normal compliance group, although the differences between the two groups were not statistically significant, $t(28) = 1.53, p > .05$. Group median values emphasize the differences between the two groups, where decreased group median ICP slope values are much larger than are those of the normal group.

In addition to characteristics of ICP configuration, elevated ICP amplitude ($\geq 2$ mm Hg.) has been associated with decreased cerebral compliance (see Chapter 2). As shown in Table 5, subjects classified with decreased compliance had, on average, ICP amplitudes $\geq 2$ mm Hg., while subjects with normal compliance tended to have ICP amplitudes of 2 mm Hg. or less. A comparison
between the two groups by an independent t-test demonstrated a significant
difference in ICP amplitude, \( t(25) = -2.73, p < .01 \). Most subjects met the
amplitude criteria appropriate for their compliance group. Twelve subjects (86%)
in the decreased compliance group met the elevated ICP amplitude suggestive of
decreased cerebral compliance. In the normal compliance group, 12 subjects
(75%) demonstrated low ICP amplitudes compatible with normal compliance. Chi
square supported the relationship between ICP amplitude level and state of
cerebral compliance, \( X^2 \) with Yates correction \( (1, N = 30) = 11.06, p < .05 \).

Table 5

Baseline Intracranial Pressure (ICP) Amplitude and ICP Slope Grouped by State
of Cerebral Compliance \( (N = 30) \)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal compliance group ( (n = 16) )</th>
<th>Decreased compliance group ( (n = 14) )</th>
<th>( t(df) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICP slope (mm Hg./second)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M(SD)</td>
<td>-3.7(3.8)</td>
<td>-5.9(3.6)</td>
<td>1.56(28)</td>
</tr>
<tr>
<td>Range</td>
<td>-0.2 to -12.4</td>
<td>-1.3 to -12.4</td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>-1.7</td>
<td>-6.1</td>
<td></td>
</tr>
<tr>
<td>Mode</td>
<td>-1.1</td>
<td>-1.3</td>
<td></td>
</tr>
<tr>
<td>ICP amplitude (mm Hg.)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M(SD)</td>
<td>1.7(1.6)</td>
<td>3.6(2.1)</td>
<td>-2.73(25)*</td>
</tr>
<tr>
<td>Range</td>
<td>0.5 to 5.3</td>
<td>1.0 to 9.1</td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>1.0</td>
<td>3.4</td>
<td></td>
</tr>
<tr>
<td>Mode</td>
<td>0.7</td>
<td>3.8</td>
<td></td>
</tr>
</tbody>
</table>

*\( p < .05 \) two-tailed independent t-test.
Demographic Characteristics of Compliance Groups

The two compliance groups were not significantly different in age. The normal compliance group age ranged from 15 to 64 years with an average of 29 years, while the decreased compliance group age ranged from 16 to 79, and averaged 33 years, t (25) = -.76, p > .05. The normal group was composed of 15 (94%) males and one (6%) female, while the decreased compliance group had 11 (79%) males and three (21%) females. Ten (62%) MetroHealth patients and six (38%) MIEMS patients made up the normal compliance group, while 13 (93%) METRO patients and one (7%) MIEMS patient comprised the decreased group. The two compliance groups had similar diagnoses, with 15 (94%) closed head injured and 1 (6%) open head injured person constituting the normal group. Thirteen (93%) closed head injured patients and one (7%) open head injured person made up the decreased group.

Physiological Characteristics of Compliance Groups

ICP devices were comparable between the two groups, with 13 (81%) Richmond Bolts in the normal compliance group and 12 (86%) in the decreased compliance group. Each group had one subject monitored with a Camino catheter. The normal group had two (13%) subjects with other monitoring devices, while the decreased group had one (7%) subject in that category.

Subject alcohol and tobacco use was similar across groups. Each group had four subjects with evidence of alcohol ingestion at the time of injury. Eight
of the normal group (50%) and seven of the decreased group (50%) were negative for alcohol ingestion. Five (31%) of the normal group, and six (43%) of the decreased group used tobacco products, while nine (56%) normal and 6 (43%) decreased group subjects did not use tobacco.

Group Glasgow Coma Scale (GCS) scores were not significantly different, although the normal group had a slightly lower score ($M = 5$, $SD = 2$) than the decreased group ($M = 6$, $SD = 2$), $t(26) = -0.67$, $p > .05$. $PaCO_2$ and pH levels of the two groups were quite similar. Comparison of blood gas values revealed no significant between-group differences (arterial $PCO_2$: $t(22) = .58$, $p > .05$ and pH: $t(25) = -1.00$, $p > .05$).

The normal compliance group's baseline MICP ($M = 12$, $SD = 8$ mm Hg.) was lower than the decreased group ($M = 19$, $SD = 12$ mm Hg.). Only the group with decreased compliance demonstrated intracranial hypertension at baseline. Because baseline MICP differences between groups could exaggerate any differences found at suction catheter insertion, baseline MICP was compared between the two groups using an independent t-test. No statistically significant differences were found between the normal and decreased compliance groups $t(23) = -2.00$, $p > .05$. Baseline CPP was lower for the decreased group ($M = 86$, $SD = 19$ mm Hg.) than the normal group ($M = 91$, $SD = 20$ mm Hg.) although both were within normal limits for cerebral perfusion pressure. No significant differences were found for CPP between the two groups, $t(28) = .66$, $p > .05$. 
No significant differences were found for systemic mean arterial pressure between the groups, with the normal group MAP of 103 (SD = 21) mm Hg. and decreased group MAP of 105 (SD = 14 mm Hg.), t(26) = -.40, p > .05. Heart rate was higher in the normal group (M = 96, SD = 18 BPM) than the decreased group, but not significantly different (M = 87, SD = 23 BPM), t(24) = 1.28, p > .05. Both groups’ average HR and MAP were within normal range.

**Medications Received by Compliance Group**

As displayed in Table 6, the normal compliance group had more subjects who received mannitol and sedatives within the hour before suctioning. The normal compliance group also received more barbiturates within the 24 hour period before the suctioning procedure.

**Descriptive Analysis of Sample ICP, CPP, MAP and HR Response to Each Suctioning Procedure Component**

Prior to completion of the inferential analysis, the sample’s average MICP, CPP, MAP and HR response to each suctioning procedure event was determined. As revealed in Table 7 ICP increased steadily with each subsequent suctioning event. HR also increased with each additional event, and demonstrated a dramatic increase during actual suctioning. MAP and CPP that had similar response patterns across the suctioning procedure, peaked during suction catheter insertion.
Table 6

Medication Received Before Initiation of the Suctioning Procedure by Compliance Groups (N = 30)

<table>
<thead>
<tr>
<th>Variable and attribution</th>
<th>Frequency</th>
<th>Percentage</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barbiturates-within 24 hours</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3</td>
<td>19%</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>No</td>
<td>13</td>
<td>81%</td>
<td>14</td>
<td>100%</td>
</tr>
<tr>
<td>Mannitol-within 1 hour*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>4</td>
<td>25%</td>
<td>1</td>
<td>7%</td>
</tr>
<tr>
<td>No</td>
<td>10</td>
<td>63%</td>
<td>9</td>
<td>64%</td>
</tr>
<tr>
<td>Paralytics-within 1 hour*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3</td>
<td>19%</td>
<td>3</td>
<td>21%</td>
</tr>
<tr>
<td>No</td>
<td>11</td>
<td>68%</td>
<td>7</td>
<td>50%</td>
</tr>
<tr>
<td>Sedatives-within 1 hour*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>4</td>
<td>25%</td>
<td>1</td>
<td>7%</td>
</tr>
<tr>
<td>No</td>
<td>10</td>
<td>63%</td>
<td>9</td>
<td>64%</td>
</tr>
</tbody>
</table>

*Normal group has 2 missing. Decreased group has 4 missing.
Table 7

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Oxygenated Breaths</th>
<th>Ventilator Disconnect</th>
<th>Catheter Insertion</th>
<th>Actual Suctioning</th>
</tr>
</thead>
<tbody>
<tr>
<td>MICP (mmHg.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( M(\text{SD}) )</td>
<td>16(10)</td>
<td>17(10)</td>
<td>17(11)</td>
<td>19(11)</td>
<td>22(11)</td>
</tr>
<tr>
<td>Range</td>
<td>1 to 46</td>
<td>2 to 47</td>
<td>2 to 49</td>
<td>1 to 48</td>
<td>1 to 52</td>
</tr>
<tr>
<td>CPP (mmHg.)*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( M(\text{SD}) )</td>
<td>88(19)</td>
<td>91(20)</td>
<td>89(16)</td>
<td>97(22)</td>
<td>94(18)</td>
</tr>
<tr>
<td>Range</td>
<td>50 to 135</td>
<td>50 to 150</td>
<td>62 to 126</td>
<td>54 to 166</td>
<td>58 to 147</td>
</tr>
<tr>
<td>HR (BPM)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( M(\text{SD}) )</td>
<td>92(21)</td>
<td>96(22)</td>
<td>96(22)</td>
<td>99(20)</td>
<td>113(17)</td>
</tr>
<tr>
<td>Range</td>
<td>57 to 132</td>
<td>60 to 141</td>
<td>60 to 141</td>
<td>60 to 138</td>
<td>84 to 156</td>
</tr>
<tr>
<td>MAP (mmHg.)*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( M(\text{SD}) )</td>
<td>104(18)</td>
<td>107(19)</td>
<td>106(17)</td>
<td>116(21)</td>
<td>116(18)</td>
</tr>
<tr>
<td>Range</td>
<td>60 to 161</td>
<td>61 to 159</td>
<td>68 to 140</td>
<td>54 to 178</td>
<td>56 to 166</td>
</tr>
</tbody>
</table>

* \( n = 29 \) at ventilator disconnection and catheter insertion.
Description of Response to Suctioning Procedure Grouped by State of Compliance

Because the third major research question addressed differences between subjects with normal and decreased cerebral compliance, group average MICP, CPP, MAP and HR responses to each suctioning procedure event were assessed (see Table 8). As demonstrated in Figures 8 and 9, both groups’ MICP and HR increased with each consecutive suctioning procedure event, and peaked during actual suctioning. The decreased compliance group had a higher ICP and lower HR than did the normal compliance group. During actual suctioning, however, the decreased group’s HR exceeded that of the normal compliance group. Both groups’ CPP increased across the suctioning procedure, and peaked during suction catheter insertion. MAP increased markedly during suction catheter insertion for both groups, and remained elevated during actual suctioning.

Preparation of Data for Inferential Analysis

ICP, CPP, HR, and MAP were assessed prior to suction catheter insertion at three different timepoints. The three timepoints were (a) baseline (subject resting with no direct care being given), (b) hyperoxygenation breaths, and (c) disconnection of the endotracheal tube from the ventilator. In addition to describing the sample prior to insertion of the suction catheter, the purpose of assessing the three timepoints was to determine the preinsertion event that provided the most conservative estimate (highest ICP) for within-subject comparisons.
Table 8

**Normal* and Decreased* Cerebral Compliance Groups' Intracranial Pressure (ICP), Cerebral Perfusion Pressure, Mean Arterial Pressure (MAP), and Heart Rate (HR) Response to Suctioning Procedure Events (N = 30)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Suctioning Procedure Events</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>MICP (mm Hg.)</td>
<td></td>
</tr>
<tr>
<td>Normal M(SD)</td>
<td>12(8)</td>
</tr>
<tr>
<td>Decreased M(SD)</td>
<td>19(12)</td>
</tr>
<tr>
<td>CPP (mm Hg.)</td>
<td></td>
</tr>
<tr>
<td>Normal M(SD)</td>
<td>91(20)</td>
</tr>
<tr>
<td>Decreased M(SD)</td>
<td>86(19)</td>
</tr>
<tr>
<td>MAP (mm Hg.)</td>
<td></td>
</tr>
<tr>
<td>Normal M(SD)</td>
<td>103(21)</td>
</tr>
<tr>
<td>Decreased M(SD)</td>
<td>105(14)</td>
</tr>
<tr>
<td>HR (BPM)</td>
<td></td>
</tr>
<tr>
<td>Normal M(SD)</td>
<td>96(18)</td>
</tr>
<tr>
<td>Decreased M(SD)</td>
<td>87(23)</td>
</tr>
</tbody>
</table>

Figure 8. Cerebrovascular Response: Average Intracranial Pressure (ICP) and Cerebral Perfusion Pressure (CPP) at Each Suctioning Procedure Event Grouped by Normal and Decreased Cerebral Compliance ($N = 30$)
Figure 9. Systemic Vascular Response: Mean Arterial Pressure (MAP) and Heart Rate (HR) at Each Suctioning Procedure Event Grouped by Normal and Decreased Cerebral Compliance \((N = 30)\)
Average MICP values for each of the three timepoints were compared using dependent t-tests. Average MICP was significantly elevated at disconnection compared to baseline. Ventilator disconnection was, therefore, selected as the presuction catheter insertion timepoint for within-subjects comparisons. Preparation of data collected at each of the three timepoints before suction catheter insertion, and selection of the timepoint that was used in the inferential analysis is described in Appendix C. See Table C-1 in Appendix C for results of statistical comparisons between timepoints.

Testing Statistical Assumptions for Inferential Analysis

Both independent and dependent t-tests require normally distributed data, although the t-test is robust to violations of normality (Norusis, 1990). Normal distribution of all interval level measures were assessed using histograms, normal and detrended probability plots, Lilliefors test for normal distribution, tests for skewness and for kurtosis. All data used to test hypotheses were normally distributed across the sample.

In addition to the preceding assumption, independent t-tests require that the two samples are independent and have equal variances (Norusis, 1990). In preparation for testing of the third research question hypothesis, the Levene test and spread-and-level plots were used to assess homogeneity of variance between the normal and decreased compliance groups. Group variances for MICP during suction catheter insertion were approximately equal.
Results From Inferential Testing of Hypotheses

Findings are presented in the order that research questions and hypotheses were tested. Because the present study was exploratory, and sought to identify effects of suction catheter insertion, the overall significance level was set at $p = .10$. It was recognized that the liberal experiment-wise alpha level increased the possibility of falsely rejecting null hypotheses. To protect against Type I error, the Bonferroni procedure was used to determine criteria for rejection of each null hypothesis. The overall significance level ($p = .10$) was divided by the four one-tailed hypotheses for each of the first two research questions. Using the Bonferroni procedure, the significance level was set at $\alpha = .025$, for each one-tailed hypothesis. Dependent t-tests were used to test each hypothesis of the first two research questions.

Research Question 1. Is there a difference between cerebrovascular response (MICP and CPP) and vasopressor response (MAP and HR) during suction catheter insertion compared to baseline values?

The first hypothesis stated that MICP would be increased during suction catheter insertion compared to MICP at baseline. Data obtained during ventilator disconnection were chosen as the presuction catheter insertion timepoint. The hypothesis was supported. Average MICP during suction catheter insertion ($M = 19$, $SD = 11$ mm Hg.) was significantly elevated compared to ventilator disconnection ($M = 17$, $SD = 11$ mm Hg.), $t(29) = -2.62$, $p < .025$. 

The second hypothesis stated that CPP would be increased during suction catheter insertion compared to CPP at baseline. The hypothesis was supported. Average CPP during suction catheter insertion ($M = 97$, $SD = 22$ mm Hg.) was significantly elevated compared to during ventilator disconnection ($M = 89$, $SD = 17$ mm Hg.), $t (28) = -4.78$, $p < .025$.

The third research hypothesis stated that MAP would be increased during suction catheter insertion compared MAP at baseline. This hypothesis was also supported. Average MAP during suction catheter insertion ($M = 116$, $SD = 21$ mm Hg.) was significantly increased compared to during disconnection ($M = 106$, $SD = 17$ mm Hg.), $t (28) = -5.53$, $p < .025$.

Hypothesis four stated that HR would be increased during suction catheter insertion compared to HR at baseline. This hypotheses was not supported. No significant difference was found between average HR during suction catheter insertion ($M = 99$, $SD = 20$ BPM) and during disconnection ($M = 96$, $SD = 22$), $t(29) = -1.14$, $p > .025$.

**Research Question 2.** Is there a difference in cerebrovascular response (MICP and CPP) and vasopressor response (MAP and HR) during actual suctioning compared to suction catheter insertion?

The first hypothesis stated that there would be an increase in MICP during actual suctioning compared to MICP during suction catheter insertion. The hypothesis was supported. Average MICP during actual suctioning ($M = 22$, $SD$
= 11 mm Hg.) was significantly higher compared to during suction catheter insertion ($M = 19$, $SD = 11$ mm Hg.), $t(29) = -4.42$, $p < .025$.

Hypothesis two stated that there would be an increase in CPP during actual suctioning compared to suction catheter suction insertion. The hypothesis was not supported. No significant difference was identified when mean CPP during actual suctioning ($M = 95$, $SD = 18$ mm Hg.) was compared to mean CPP at suction catheter insertion ($M = 97$, $SD = 22$ mm Hg.), $t(28) = 1.23$, $p > .025$.

The third hypothesis stated that MAP would increase during actual suctioning compared to MAP during suction catheter insertion. The hypothesis was not supported. There was no significant difference between mean MAP during actual suctioning ($M = 116$, $SD = 18$ mm Hg.) and at suction catheter insertion ($M = 116$, $SD = 21$ mm Hg.), $t(28) = -.16$, $p > .025$.

The last hypothesis stated that HR during actual suctioning would increase compared to suction catheter insertion. The hypothesis was supported. HR during actual suctioning ($M = 113$, $SD = 17$ BPM) was significantly higher than at suction catheter insertion ($M = 99$, $SD = 20$ BPM), $t(29) = -4.82$, $p < .025$.

**Research Question 3.** Is there a difference in cerebrovascular response (MICP) during suction catheter insertion, between subjects with decreased cerebral compliance (ICP rounding) and those with normal compliance (Normal ICP configuration)?

The hypothesis for the third research question stated that there would be a
greater increase in MICP during suction catheter insertion in subjects with
decreased cerebral compliance at baseline compared to those with normal
compliance. Criteria for rejection of the third research question’s single
hypothesis was set at $p = .10$. The rationale for the relatively liberal significance
level was that the indicator used to identify state of cerebral compliance (ICP
waveform configuration) had not been previously explored. Therefore, the goal of
the third experiment was to identify any differences in ICP response between the
two compliance groups.

The hypothesis was supported. Using an independent t-test with a
significance level of $p = .10$ to test the preceding hypothesis, a significant
difference was found between the decreased compliance group’s average MICP
during suction catheter insertion ($M = 23$, $SD = 12$ mm Hg.) and the normal
compliance group’s MICP ($M = 15$, $SD = 9$ mm Hg.), $t(23) = -2.08$, $p < .10$.

In summary, suction catheter insertion significantly increased subjects’
MICP, CPP, and MAP compared to pressures that were measured before suction
catheter insertion. HR was not significantly changed by suction catheter insertion.
Subjects’ MICP and HR were significantly increased during actual suctioning
compared to during suction catheter insertion. MAP and CPP were not
significantly changed. Finally, subjects classified as having decreased cerebral
compliance had significantly higher MICP during suction catheter insertion than
those subjects who were classified as having normal compliance prior to the
suctioning procedure.

Post Hoc Descriptive Analysis

Physiologic Mechanisms of ICP Response to Suction Catheter Insertion

The major focus of the study was the impact of suction catheter insertion upon head injured adults’ cerebrovascular responses during the suctioning procedure. In addition to demonstrating the presence of a cerebrovascular response to insertion of a suction catheter, the study also sought to increase knowledge of the mechanisms through which suction catheter insertion resulted in a cerebrovascular response. The focus of post hoc analyses, therefore, was to examine the data for evidence of how suction catheter insertion may have increased head injured adults’ ICP.

As conceptualized in the study’s theoretical framework, cerebrovascular response occurred when suction catheter insertion resulted in increased intrathoracic pressure, systemic vasomotor activation, and primary cerebral vasodilation. Empirical support for the three mechanisms was sought through both descriptive and inferential analysis. Whenever inferential testing was appropriate, a significance criteria of \( p = .05 \) was observed.

Intrathoracic pressure and ICP response to suction catheter insertion. The role that increased intrathoracic pressure played in initiating a cerebrovascular response to suction catheter insertion was explored by first comparing ICP response (increased ICP) of subjects with a cough reflex to those without a cough
reflex. Second, data were analyzed for the point in time that ICP response first occurred during suction catheter insertion. An underlying assumption for each of the two analyses was that coughing, in response to suction catheter insertion, resulted in increased intrathoracic pressure.

First, it was hypothesized that subjects who experienced lighter coma would be more likely to have an intact cough reflex than subjects in deepest coma. The increase in intrathoracic pressure from coughing would increase cerebral venous pressure. Subjects with lighter coma, therefore, would have a larger ICP response to suction catheter insertion than subjects in deepest coma. The tentativeness of the preceding assumption was fully recognized in that many other factors may have affected subjects' cough ability. Using Glasgow Coma Scale (GCS) scores to determine depth of coma, subjects with GCS scores of 3 to 5 (indicating deepest coma) were compared with those who had scores of 6 to 9 (indicating lighter coma). A between-group, within-subjects repeated measures ANOVA, revealed that the two coma group's ICP values at ventilator disconnection (Deep coma group M = 18 mm Hg., Lighter coma group M = 16 mm Hg.) and at suction catheter insertion (Deep coma group M = 19 mm Hg., Lighter coma group M = 18 mm Hg.) were not significantly different, F(1, 28) = .09, p > .05. A significant within-subject effect on ICP from ventilator disconnection to suction catheter insertion was identified, F(1,28) = 3.87, p < .05. The hypothesis was not supported.
The second strategy used to clarify the role that increased intrathoracic pressure played in initiating ICP response to suction catheter insertion was to identify where, during the insertion process, ICP, MAP, and HR increased. It was hypothesized that a response that began during the later part of suction catheter insertion indicated that the carina had been mechanically stimulated by the suction catheter. A response at the beginning of suction catheter insertion indicated activation of irritant receptors in other airway tissue. Normally, stimulation of the carina produces a strong cough response. The suction catheter insertion time interval was divided into three equal sections. Each subject's ICP data were observed for their initial increase and was then categorized as occurring during the first, second or third time division.

No subjects demonstrated an increase in ICP during the first interval of the suction catheter insertion event. Five subjects experienced initial ICP increase during the second interval. Among the five subjects, ICP remained elevated through the remainder of suction catheter insertion. Eleven additional subjects experienced initial ICP increase only during the final time interval. The predominance of response onset during the last part of the suction catheter insertion interval suggests that stimulation of the carina was a major mechanism contributing to increased ICP during suction catheter insertion. Subjects who demonstrated a response midway through suction catheter insertion may have had the catheter advanced to the carina early in the interval, or may have had irritant
receptors activated that line the airways.

**Vasopressor reflex and ICP response to suction catheter insertion.** To understand better the role that systemic vasopressor response played in ICP response to suction catheter insertion, the relationship between increased ICP and MAP was explored. Although increased HR was also an indicator of the vasopressor response, it was not used in this analysis because the impact of increased MAP would have been predominant. As shown in Figure 10, both MAP and MICP increased substantially in response to suction catheter insertion.

![Graph showing ICP and MAP responses during suctioning procedure](image)

**Figure 10.** Concurrent Mean Arterial Pressure (MAP) and Mean Intracranial Pressure (ICP) Responses to Each Suctioning Procedure Event (N = 30)
The relationship between MAP and ICP responses to suction catheter insertion was further explored by graphing the concurrent average of the highest and lowest MAP and MICP responses across the suctioning procedure. It was assumed that the vasopressor effect on ICP would be minimal at lowest MAP and ICP values. This assumption is graphically supported in Figure 11, where average MICP response did not follow MAP response across the suctioning procedure.

**Figure 11.** Concurrent Average of the Lowest Mean Arterial Pressure and Mean Intracranial Pressure Responses to Each Suctioning Procedure Event ($N = 30$).
Conversely, the vasopressor effect on ICP was expected to have maximum impact at highest MAP and ICP levels. This phenomenon is demonstrated in Figure 12, where MAP and MICP had almost identical response patterns at subjects' highest MICP (17 to 27 mm Hg. across the suctioning procedure) and MAP (106 to 126 mm Hg.) values.

**Figure 12.** Concurrent Average Highest Mean Arterial Pressure and Mean Intracranial Pressure at Each Suctioning Procedure Event (N = 30)
Cerebral vasodilation and ICP response to suction catheter insertion.

Because primary cerebral vasodilation was not directly measured, its role in initiating ICP response to suction catheter insertion was inferred. Primary cerebral vasodilation was assumed to be a major contributor of ICP response if subjects did not have a functional cough reflex (GCS scores \( \leq 5 \)) and did not have a concurrent increase in MAP. The absence of cough and MAP response eliminated increased intrathoracic pressure and the vasopressor reflex as the basis for ICP response. Only two subjects met the preceding criteria.

**ICP Response Patterns During Suction Catheter Insertion**

The type of ICP response to suction catheter insertion was used to further elucidate the role that increased intrathoracic pressure, primary cerebral vasodilation, and the vasopressor response may have played in subjects' cerebrovascular responses. It was theorized that activation of the vasopressor response (increased MAP and HR) by suction catheter insertion would result in an ICP response characterized by increased ICP amplitude (ICP spiking). If ICP response to suction catheter insertion was typified by increasing ICP magnitude (steady rise above ICP baseline), then either increased intrathoracic pressure or primary cerebral vasodilation was more influential on ICP response than the vasopressor reflex.

Seventeen subjects responded to suction catheter insertion with ICP spiking, including eight subjects who experienced both ICP spiking and rising
baseline. Ten subjects demonstrated rising ICP baseline, and three subjects had no ICP response to suction catheter insertion. As shown in Table 9, subjects who experienced ICP spiking, or both ICP spiking and rising baseline, had the highest MAP (and therefore, also CPP) values during suction catheter insertion. However, subjects who responded to suction catheter insertion with a rising ICP baseline, had the highest average ICP values. Heart rate, also appeared to be related to a rising ICP baseline, as demonstrated in the high HR values of subjects with either rising ICP baseline or both rising ICP baseline and spiking.

Table 9

Average Intracranial Pressure (MICP), Cerebral Perfusion Pressure (CPP), Arterial Pressure (MAP), and Heart Rate (HR) Grouped by Intracranial Pressure (ICP) Response Patterns During Suction Catheter Insertion (N = 30)

<table>
<thead>
<tr>
<th>ICP Response Patterns</th>
<th>Spiking (n = 9)</th>
<th>Rising Baseline (n = 10)</th>
<th>Both Spike and Rise (n = 8)</th>
<th>No Response (n = 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MICP (mm Hg.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M(SD)</td>
<td>15(13)</td>
<td>24(10)</td>
<td>18(8)</td>
<td>12(7)</td>
</tr>
<tr>
<td>CPP (mm Hg.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M(SD)</td>
<td>103(27)*</td>
<td>86(18)</td>
<td>105(18)</td>
<td>94(14)</td>
</tr>
<tr>
<td>MAP (mm Hg.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M(SD)</td>
<td>119(24)*</td>
<td>111(23)</td>
<td>122(19)</td>
<td>106(18)</td>
</tr>
<tr>
<td>HR (BPM)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M(SD)</td>
<td>95(22)</td>
<td>100(25)</td>
<td>106(17)</td>
<td>94(19)</td>
</tr>
</tbody>
</table>

* n = 29.
Subjects with Mismatched ICP Amplitude and State of Cerebral Compliance

Although elevated ICP amplitude (≥ 2 mm Hg.) is associated with decreased cerebral compliance, in the current study six subjects' ICP amplitude and cerebral compliance classifications were mismatched. Four subjects classified with normal cerebral compliance had baseline ICP amplitudes of greater than 2 mm Hg., while two subjects with ICP amplitudes of less than 2 mm Hg. were identified with decreased cerebral compliance.

In an effort to identify any factors that might differentiate subjects with mismatched ICP amplitude and cerebral compliance, each of the six subjects' data were inspected. The four subjects with normal cerebral compliance and elevated ICP amplitudes had ICP waveforms that were clearly arterial in characteristic (ICP and MAP waveform configuration were similar). These four subjects constituted 57% of the total sample with arterial ICP waveform configuration. The other similarity among the four subjects was that all demonstrated tachycardia before initiation of the suctioning procedure. Although the normal cerebral compliance group had a high normal HR (M = 96, SD = 18 BPM), these four subjects had an average HR at baseline of 116(SD = 12 BPM).

The two subjects identified as having decreased cerebral compliance and small ICP amplitudes had venous ICP waveform configuration (that is, a rounded waveform dissimilar to the subject's MAP waveform). Venous ICP waveform configuration was found in 12 of the 14 subjects identified with decreased cerebral
compliance. The two subjects had relatively slow HR at baseline ($M = 71$, $SD = 11$ BPM) compared to the decreased compliance group ($M = 87$, $SD = 23$ BPM).

**Intracranial Dimensions Represented by ICP Waveform Configuration**

To shed light on the intracranial dimension represented by ICP waveform configuration, ICP waveforms used to determine subjects' state of cerebral compliance for a priori statistical testing were investigated to see if they could be further categorized either as arterial waveforms (peaked ICP waveforms with an elevated P₁ subwaveform) or as venous waveforms (rounded waveforms with elevated P₂ and P₃ subwaveforms).

Of the total sample ($N = 30$), 20 subjects had ICP waveforms that could be clearly identified as either arterial or venous in character. Seven subjects had ICP waves that were distinctly arterial in form, 12 had venous shaped ICP waves, and one subject had approximately equal arterial and venous shaped ICP waves. The data were next tested to see if there was a relationship between waves that were arterial or venous and their cerebral compliance classification. Chi square statistic demonstrated significant association between cerebral compliance and arterial/venous classifications ($X^2(1, N = 19) = 11.6, p < .05$). Five of the seven subjects with arterial shaped ICP waves were classified with normal compliance, while the 12 subjects classified with venous shaped ICP waves were classified as having decreased cerebral compliance.
CHAPTER 5

Summary and Discussion

Summary

The threefold purpose of this quasi-experimental within-subject design study was to (a) determine the effect of suction catheter insertion on the cerebrovascular status of head injured adults, (b) compare head injured adults’ responses to suction catheter insertion with their responses to actual suctioning (application of negative suction pressure), and (c) determine the effect of head injured adults’ state of cerebral compliance before suction catheter insertion on their cerebrovascular responses during suction catheter insertion.

The study was based upon the following assumptions. Insertion of a suction catheter mechanically stimulates airway irritant receptors. Cerebrovascular response to suction catheter insertion results from three physiologic processes that occurred as a consequence of irritant receptor activation. The three physiologic processes are: (a) increased intrathoracic pressure, (b) the systemic vasopressor response, and (c) primary cerebral vasodilation.

Cerebrovascular response to suction catheter insertion was assessed by the measurement of intracranial pressure (ICP) and cerebral perfusion pressure (CPP) (calculated MAP - ICP) as the dependent variables. Systemic vasomotor response to suction catheter insertion was evaluated by measurement of the dependent

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variables, mean arterial pressure (MAP) and heart rate (HR).

The study also investigated the effect of head injured adults' state of cerebral compliance on ICP response during suction catheter insertion. Cerebral compliance was conceptualized as the relationship between intracranial volume and pressure, and was operationalized as ICP waveform configuration. Cerebral compliance was normal when increased intracranial volume was accommodated without an increase in ICP. Normal compliance was represented by an ICP waveform that, after initial upward inflection, had either a small steady plateau, or immediately began a gradual descent to baseline. When compliance was decreased, any activity that increased intracranial volume resulted in increased ICP. Decreased cerebral compliance was exemplified by a rounded or peaked waveform with a steep descent to baseline.

The major finding of this study was that suction catheter insertion, isolated from other components of the suctioning procedure, significantly increased ICP, CPP and MAP. ICP continued to increase during actual suctioning compared to during catheter insertion, but CPP and MAP remained constant. The difference in ICP and MAP responses to suction catheter insertion compared to actual suctioning supports MAP increase as a consequence of airway stimulation rather than a reaction to cerebrovascular change. Based on the preceding findings, activation of the systemic vasopressor reflex (increased MAP) by insertion of a suction catheter appears to be a major mechanism contributing to increased ICP.
during the suctioning procedure.

The study also demonstrated that subjects with peaked or rounded ICP waveforms, prior to initiation of the suctioning procedure, had higher ICP levels during suction catheter insertion than subjects with normal waveform configuration. As conceptualized in the study, ICP waveform configuration was an indicator of state of cerebral compliance. Secondary analysis, however, suggested that ICP waveform configuration may also offer information about head injured adults' state of cerebrovascular reactivity.

The results of the study are interpreted in the following discussion. Discussion is organized by study findings. Findings generated from the first two research questions are discussed together for their contribution to understanding the effect of suction catheter insertion on cerebrovascular status in head injured adults. Post hoc findings are included where they contribute to understanding of the phenomenon under consideration. Findings are discussed within the context of the study's purpose, theoretical framework and within the greater background of past research findings. Alternative explanations for each of the study findings are considered. Power was recalculated whenever research hypotheses were not supported. The chapter ends with discussion of study strength, limitations, and the implications of study findings for nursing research and practice.
Discussion

Effect of Suction Catheter Insertion

The first research question considered whether, in head injured adults, suction catheter insertion resulted in a cerebrovascular response (increased MICP and CPP) and a vasopressor response (increased MAP and HR) compared to subjects' values before suction catheter insertion. It was hypothesized that all four study variables would increase in response to suction catheter insertion. The second research question compared head injured adults' cerebrovascular and vasopressor responses during actual suctioning with responses during suction catheter insertion. For the second question it was hypothesized that there would be no substantial differences between the two suctioning procedure components when cerebrovascular and vasopressor responses were compared.

Cerebrovascular response: mean intracranial pressure. MICP significantly increased during suction catheter insertion when compared to baseline ICP, and continued to increase during actual suctioning when compared to suction catheter insertion. These findings support the findings of previous research where ICP consistently increased during endotracheal suctioning (ETS) in head injured subjects (Boortz-Marx, 1985; Campbell, 1989; Crosby & Parsons, 1992; Donegan & Bedford, 1982; Ersson et al., 1990; Fisher et al., 1982; Parsons & Shogun, 1984; Rudy et al., 1991; Shalit & Umansky, 1977; Snyder, 1983; Tsementzis et al., 1982; White et al., 1982; Yano et al, 1982).
The current study differed from previous research, however, because ICP data that occurred during suction catheter insertion were measured and tested separately from ICP data of other suctioning events. By separating suction catheter insertion from other suctioning events, the study demonstrated that tracheal stimulation independently increased ICP. Until the present study, tracheal stimulation had been implicated as a major cause of increased ICP during ETS, but had not been independently tested (Donegan & Bedford, 1980; Fisher et al., 1982; White et al., 1982; Yano et al., 1986).

While the current investigation does not eliminate the possibility that other factors contributed to ICP increase during suctioning, it clearly demonstrated that suction catheter insertion increased ICP. Although ICP continued to increase during actual suctioning, because suction catheter insertion inevitably precedes actual suctioning, current study findings support suction catheter insertion as the initiatort of the well documented ICP response to ETS.

Previous research either included suction catheter insertion data as part of general suctioning data (Boortz-Marx, 1985; Crosby & Parsons, 1992; Ersson et al., 1990; Parsons & Shogun, 1984; Shalit & Umansky, 1977; Snyder, 1983; Tsementzis et al., 1982; White et al., 1982; Yano et al, 1982) or excluded suction catheter insertion data (Campbell, 1989; Donegan & Bedford, 1982; Fisher et al., 1982; Ručy et al., 1991). Although studies that excluded suction catheter insertion data demonstrated significant ICP increase during ETS, their findings do
not negate suction catheter insertion as the primary initiator of increased ICP. Mechanical stimulation from the suction catheter continues throughout actual suctioning, and thus continues to affect ICP. Additionally, it is reasonable to expect ICP to remain elevated after it is first increased from suction catheter insertion. Other studies demonstrated that ICP remained elevated for a period of time after cessation of ETS (Campbell, 1989; Ersson et al., 1990; Rudy et al., 1991; Shalit & Umansky, 1977; Snyder, 1983).

**Mechanisms activated by suction catheter insertion.** Although current study findings clearly demonstrated that airway stimulation resulted in substantial increase in ICP, this information alone is not sufficient to guide nursing research and practice. The current study was based upon a theoretical model that specified increased intrathoracic pressure, primary cerebral vasodilation, and the vasopressor response as the three major mechanisms through which suction catheter insertion resulted in increased ICP. To develop effective nursing interventions to attenuate increased cerebrovascular response during suctioning, it is important to identify the mechanisms responsible for ICP elevation. The following discussion focuses on secondary findings that provide information about how suction catheter insertion may initiate increased ICP.

**Increased intrathoracic pressure.** Two secondary findings suggest that increased intrathoracic pressure is a mechanism contributing to increased ICP during suction catheter insertion. First, it was shown that subjects with lighter
coma had significantly increased ICP during suction catheter insertion compared to subjects in deepest coma. Because subjects in lighter coma were more likely to have an intact cough reflex, increased intrathoracic pressure from cough induced by airway stimulation may have contributed to ICP increase during suction catheter insertion.

Additional support for the role of cough and increased intrathoracic pressure was provided by a descriptive analysis of when ICP increase first occurred during suction catheter insertion. The majority of subjects experienced increased ICP during the later part of the insertion process. The suction catheter is most likely to make contact with the carina when it is maximally inserted. Stimulation of the carina induces violent coughing (Kerston, 1989; Guyton, 1991; Levitsky, 1984). Therefore, reflexive cough, with its concomitant increase in intrathoracic pressure, may have played a key role in ICP increase during suction catheter insertion.

The link between increased intrathoracic pressure and cough as a basis for increased ICP during ETS has been supported by others. Tsementzis and colleagues (1982), who measured change in intrathoracic pressure with a transduced esophageal balloon, demonstrated simultaneous increases in intrathoracic pressure and ICP during ETS in severely head injured subjects. Both the intrathoracic and intracranial responses to ETS were abolished with administration of paralytic medication (pancuronium). The connection between
increased intrathoracic pressure and increased ICP was also substantiated in studies where simultaneous rise and fall of central venous pressure and ICP was demonstrated in response to intratracheal saline administration, coughing and ETS (Ersson et al., 1990; White et al., 1982).

In summary, past research and current study findings suggest that increased intrathoracic pressure initiated by airway stimulation plays a major role in increased ICP during ETS. These findings lend support to the current study's theoretical model and suggest that ICP increase during ETS may be attenuated by preventing coughing in response to airway stimulation.

**Primary cerebral vasodilation.** The study's second theoretical premise was that ICP increased as a result of primary cerebral vasodilation from airway irritant receptor activation. The study theorized that efferent parasympathetic impulses from respiratory center motor neurons located in the solitary nucleus resulted in primary cerebral vasodilation. Because the role of primary cerebral vasodilation could not be measured directly, its presence was inferred. It was speculated that if ICP increased during suction catheter insertion, after eliminating the presence of both cough and concurrent MAP elevation, primary cerebral vasodilation was present. Inspection of ICP response of subjects in deepest coma (see preceding discussion) who did not have a concurrent elevation of MAP revealed two subjects with increased ICP. Similar findings were reported by Tendentus and colleagues (1982) who implicated primary cerebral vasodilation as the basis for rapid ICP
increase in six subjects during ETS. Tsamentus reported a sudden onset of intracranial hypertension in six subjects who did not have simultaneous increased intrathoracic pressure or MAP. By comparison, current study subjects who met the criteria for primary cerebral vasodilation had sudden onset of a small increase in ICP magnitude. Current study findings offer weak support for primary vasodilation as a mechanism for increased ICP during suction catheter insertion. Differences in the magnitude of ICP response between the current study and that of Tsamentus and colleagues may be due to the interaction of time with ICP response to suctioning. Tsamentus and colleagues combined ICP data from suction catheter insertion and actual suctioning, while the current study separated the two suctioning components. ICP data from the entire suctioning event allowed more time for primary cerebral vasodilation to occur.

Vasopressor response: mean arterial pressure. Mean arterial pressure was significantly increased during suction catheter insertion compared to values during ventilator disconnection. Systemic arterial pressure peaked during suction catheter insertion, and remained unchanged throughout actual suctioning. The current study differed from previous ETS research in the head injured population in two important ways. The first difference was that the study specified that MAP increased when airway irritant receptors were activated by suction catheter insertion. Suction catheter insertion MAP data were measured and tested independently from actual suctioning data.
Current study findings concur with previous research that included suction catheter insertion data with general suctioning data, and found significant increased MAP (Crosby & Parsons, 1992; Parsons & Shogun, 1984; White et al., 1982). Other researchers demonstrated significant MAP increase regardless of the fact that suction catheter insertion MAP data was excluded from analysis (Campbell, 1989; Donegan & Bedford, 1982; Fisher et al., 1982; Rudy et al., 1991). The apparent discrepancy between the current study where MAP increased during catheter insertion but not during actual suctioning, is explained by the fact that previous studies did not capture the initial MAP increase that likely occurred during catheter insertion, and was carried over into actual suctioning.

Because suction catheter insertion was not isolated from other suctioning events in past research understanding was limited of how or why MAP increased during ETS. The current study clearly demonstrated that MAP increased when the airways were stimulated from insertion of the suction catheter. Increased MAP from airway stimulation is congruent with previous findings where tracheal intubation resulted in rapid MAP elevation (Hamill et al., 1981; Splinter, 1990).

The second major difference between the present study and previous research is conceptual in nature. The current study specified that change in MAP was an indicator of systemic vasopressor response activation, while previous researchers did not clearly designate what change in MAP represented. The lack of conceptual clarity contributed to incongruous conclusions among researchers,
both about the mechanisms of increased MAP, and about the implications of increased MAP for head injured patients. Increased MAP during ETS was identified as a compensatory mechanism in response to increased ICP, that protected cerebral integrity (Crosby & Parsons, 1992; Donegan & Bedford, 1982; Parsons and Shogun, 1982). Although White and colleagues (1982) also considered increased MAP as a compensatory response to increased ICP, they considered it potentially dangerous to head injured subjects who often experience impaired cerebral autoregulation. Other researchers did not specify the mechanism of increased MAP, but like White and colleagues, identified it as potentially harmful (Campbell, 1989; Rudy et al., 1991).

In contrast to previous studies, the current study defined the mechanism of MAP increase during suction catheter insertion as independent of cerebrovascular response. MAP consistently increases when irritant receptors are mechanically stimulated (Haxhui et al., 1986; Jammes & Mei, 1979; Nadel & Widdicombe, 1962; Tomori & Widdicombe, 1969). If MAP increase during suction catheter insertion is not merely a compensatory response to ICP increase, then it may certainly exceed levels that are safe for cerebral tissue. Excessive MAP response to suction catheter insertion may pose a serious threat to cerebral cellular integrity in head injured persons.

The potential danger of the MAP response was demonstrated in the current study where 21 subjects experienced elevated MAP, while nine subjects had MAP
level that exceeded the upper limit of cerebral autoregulation. Sudden transient
MAP surges are believed to disrupt the blood-brain barrier and to interfere with
subsequent cerebral circulatory control (Langfitt, 1982; Obrist et al., 1984).

**MAP and ICP responses to suction catheter insertion.** Elevated MAP in
response to suction catheter insertion is particularly dangerous to head injured
patients with dysfunctional cerebral autoregulation. In these patients, elevated
systemic arterial pressure enters the small thin-walled cerebrovascular bed vessels
largely unattenuated. When autoregulation is impaired, ICP passively increases as
MAP increases (Paulson et al., 1990).

For the present study, cerebral autoregulatory function was assessed by
graphing concurrent MAP and ICP values across the suctioning procedure. As
was demonstrated in Figure 10 of Chapter 4, average concurrent MAP and ICP
increased substantially during suction catheter insertion. When cerebral
autoregulation is intact, ICP remains relatively constant in response to increased
MAP. The concurrent rise in both MAP and ICP suggests that autoregulatory
function may have been impaired. The coupling between MAP and ICP elevation
became more pronounced when highest average ICP and MAP were graphed
across the suctioning procedure (see Figure 12, Chapter 4). In contrast, when
lowest average ICP and MAP values were graphed across the suctioning
procedure, ICP demonstrated minimal increase, although MAP increased markedly
during suction catheter insertion (see Figure 11, Chapter 4).
Within the context of increased MAP as vasopressor response activation from tracheal stimulation, two conclusions were drawn from the graphed ICP/MAP data. First, under certain circumstances, such as severe head injury, cerebral autoregulatory function may be overridden at lower systemic pressure values. MAP values of $\geq 150$ mm Hg. level are well established as the upper level of cerebral autoregulation (Langfitt, 1982; Paulson et al., 1990). In the graphs, however, ICP responses appear coupled with MAP values that range between 100 mm Hg. to 126 mm Hg.

Secondly, ICP level affects ICP response to MAP increase. ICP levels of $\geq 15$ mm Hg., indicate intracranial hypertension (Marshall et al., 1979; Saul & Ducker, 1982; Saul, 1986). Whenever intracranial hypertension is present, some degree of cerebral autoregulatory derangement is also present (Miller et al., 1971, Paulson, et al., 1990). In the current study, when ICP exceeded 15 mm Hg., ICP and MAP responses to the suctioning procedure remained coupled, indicating impairment of cerebral autoregulation. At ICP levels less than 15 mm Hg. ICP remained constant, while MAP increased, indicating intact cerebral autoregulation.

The preceding conclusions differ from those who perceive moderately increased MAP as compensatory for ICP elevation (Crosby & Parsons, 1992; Donegan & Bedford, 1982; Parsons and Shogun, 1982; Rosner & Becker, 1984; Rosner & Daughton, 1990). When increased MAP is conceptualized as a vasopressor response directly activated by suction catheter insertion, even
moderately elevated MAP may be excessive for the head injured person. When intracranial hypertension is present, increased MAP in response to suction catheter insertion poses an even greater threat to patient viability.

The current study conceptualized and tested the response of two additional vascular dependent variables. Heart rate was the second vasopressor response indicator, while CPP was the second cerebrovascular response indicator. Both variables were included in the study for the additional information they could provide about the effects of suction catheter insertion on cerebrovascular status in head injured adults. It was hypothesized that both HR and CPP would increase in response to suction catheter insertion. The following discussion considers findings related to HR and CPP response within the context of the information provided by each indicator about the effects of suction catheter insertion and actual suctioning.

**Vasopressor response: heart rate.** HR increased during catheter insertion, but was not statistically significant. By contrast, HR significantly increased during actual suctioning when compared to HR during catheter insertion. Because the current study measured and tested HR data that occurred exclusively during suction catheter insertion, past findings are of limited usefulness in interpreting current study findings. Past research has either excluded HR suction catheter insertion data (Campbell, 1989; Rudy et al., 1991), or included it as part of general suctioning data (Crosby & Parsons, 1992; Parsons & Shogun, 1984). Although previous research demonstrated significant increase in HR during ETS,
the findings offer no information about HR response to suction catheter insertion. To better understand the absence of a significant HR response to suction catheter insertion two aspects of HR response were considered.

The first concern was why HR response during suction catheter insertion did not achieve significance. Using Cohen's (1988) process for power calculation, power was recalculated to evaluate the statistical basis for the lack of significant heart rate effect.

1. Shared standard deviation was calculated using formula 2.3.2 (Cohen, 1988, p.44).

\[ \text{Shared SD} = \sqrt{SD_r^2 + SD_s^2/2} \]

\[ \sqrt{21.895^2 + 20.345^2/2} = 21.163 \]

2. Effect size was calculated using formula 2.3.3 (Cohen, 1988, p. 46).

\[ \text{Effect size} = \frac{\text{mean difference}}{\text{standard deviation}} \]

\[ 96.3 - 99.2/21.2 = -0.132 \]

3. The effect size was adjusted to compensate for double error variance contained within Cohen's power tables that were developed for two sample t-tests. Formula 2.3.9 provided the needed correction for a dependent t-test (Cohen, 1988, p. 49).

\[ \text{Effect size} = \frac{\text{Effect Size}}{\sqrt{1-\text{correlation}}} \]
\[ -0.132 / \sqrt{1 - 0.788} = 0.2867 \]

4. Using Table 2.3.5 (Cohen, p. 36), a power of .19 was obtained.

Although HR did increase during suction catheter insertion compared to during ventilator disconnection, the low power of the paired t-test did not allow rejection of the unidirectional null hypothesis. A major contributor to low test power was the small alpha criteria (0.025) for each hypothesis. Because the alpha was quite stringent, beta error probability (0.81) was large, therefore the probability that the null hypothesis would be erroneously supported was increased. As demonstrated by the small effect size (0.29), although the phenomenon existed (that is, heart rate increased during suction catheter insertion), the degree to which it was present was not of adequate magnitude for the statistical test to capture, within the constraints of the small alpha level. Although the relatively small sample size was also a factor contributing to low power, the sample was adequate to achieve significance in the other vasomotor variable (MAP).

Another explanation for the small HR response to suction catheter insertion is suggested in Figure 13, where average HR response to each suctioning procedure event is graphed. Average HR increased during delivery of the hyperoxygenation breaths, and remained at that level during ventilator disconnection. Manual delivery of the hyperoxygenation breaths and endotracheal tube disconnection from the ventilator may have inadvertently diluted the effect of
the independent variable, suction catheter insertion, by providing airway stimulation. Therefore, difference in HR during suction catheter insertion compared to before treatment (ventilator disconnection) was diminished.

**Figure 13.** Average heart rate (HR) at each suctioning procedure event. ($N = 30$)

The second issue of importance for HR response to suction catheter insertion is that although it was small, the phenomenon (HR increase) was present. As specified in the study’s theoretical framework, insertion of a suction catheter into the endotracheal tube resulted in an irritant receptor response. Previous research has shown that stimulation of irritant receptors results in bronchoconstriction, increased MAP and decreased HR. (Appenzeller, 1990;
Boushey et al., 1972; Guyton, 1991; Jenson, 1980; Levitsky, 1986; Haxhiu et al.,
1986; Haxhui et al., 1987; 1984; Widdecombe, 1954). Additionally, it is known
that reflexive bronchoconstriction is mediated by afferent parasympathetic vagal
fibers (Haxhui et al., 1988). Vagal stimulation from activation of airway irritant
receptors by a suction catheter, however, cannot explain the increased MAP and
HR observed in both current and past ETS studies. In the current study, it was
theorized that the excitatory part of the vasomotor center was activated by
spillover from respiratory motor neuron activation as part of the irritant receptor
response. Activation of the excitatory center may account for both increased MAP
and HR during suction catheter insertion. Further study is required to understand
the role of the vasomotor center during suction catheter insertion.

It is possible that other neurogenic mechanisms may account for increased
HR during suction catheter insertion. The irritant receptor response is similar to
the diver reflex, in that both involve activation of upper airway receptors, and
both result in increased MAP and decreased HR. Both reflexes function to
preferentially redistribute blood flow to the brain. Frankel and colleagues (cited
in Appenzeller, 1990) demonstrated that the diver reflex was overcome by
excessive stimulation of thoracic wall and pulmonary stretch receptors, resulting in
hyperventilation, tachycardia and hypertension. Additionally, hyperventilation,
tachycardia and hypertension consistently occur immediately after cessation of
both the diver response and irritant receptor response (Appenzeller, 1990). When
a suction catheter is inserted into the airway in many head injured patients, they
immediately respond with attempted deep inhalation followed by a strenuous effort
to cough out the foreign object (suction catheter). Thus, it may be that activation
of pulmonary stretch receptors contribute to the increase in both HR and MAP
consistently shown to occur during suction catheter insertion and actual suctioning.

During actual suctioning, HR significantly increased while MAP remained
unchanged compared to during suction catheter insertion. It is possible that
arterial pressure remained constant during actual suctioning because the continued
elevation in HR maintained intravascular volume by increasing stroke volume and
cardiac output. Finally, the current study may have captured evidence of a
different phenomenon affecting HR during suction catheter insertion compared to
HR during actual suctioning.

Cerebrovascular response: cerebral perfusion pressure. CPP (calculated
value, MAP - ICP) was the second variable used to assess cerebrovascular
response. CPP was significantly increased during suction catheter insertion
compared to during ventilator disconnection, but remained unchanged during
actual suctioning. The finding agrees with past ETS research where CPP
increased in response to ETS (Campbell, 1989; Crosby & Parsons, 1992; Ersson
et al., 1990, Fisher et al., 1982; Parsons & Shogun, 1984; Rudy et al., 1991). In
contrast to previous findings, however, the current research demonstrated that
stimulation from insertion of the suction catheter alone, was sufficient to
significantly elevate cerebral perfusion during the suctioning procedure. Because concurrent CPP and ICP increase requires substantially increased MAP, and because past research had shown CPP increased during ETS, the current study questioned whether CPP measurement would offer any additional information not already provided by MAP measurement.

While ICP provided information about the general pressure throughout the intracranial contents, CPP contributed information specifically about the level of intracranial intravascular pressure. MAP, a measure of systemic arterial pressure, provided information about cerebral (carotid) arterial pressure. CPP, as a derivative of the relationship between ICP and MAP, provided further information about the cerebral arterial to venous pressure gradient. Through its known relationships with other intravascular variables, CPP provided indirect information about state of cerebral vascular resistance (CVR), cerebral blood flow (CBF), and cerebral blood volume (CBV) during suction catheter insertion. The relationship between CPP and CBF is illustrated using Ohm’s Law of Electricity where Current Flow = Potential Difference/Resistance. Substitution of cerebral circulatory variables for the preceding electrical variables results in the equation: CBF = CPP/CVR. Based on the relationships represented in the equation, elevated CPP during suction catheter insertion, may have been accompanied by increased cerebral blood flow and decreased cerebrovascular resistance to accommodate the increased flow.
Caution is in order when extrapolating CBF and CVR states from CPP data, because head injury alters normal cerebrovascular relationships. Studies that measured CBF in patients with elevated ICP found no consistent relationship between CPP and CBF (Cruz, 1988; Feischi et al., 1974; Obrist et al., 1984; Overgaard & Tweed, 1974). The failure to find a consistent relationship between CPP and CBF during intracranial hypertension, may reflect CBF’s greater dependence on cerebral metabolism than on level of ICP (Jennett & Teasdale, 1981). In people with head injury, CBF varies regionally depending upon local metabolic need and the degree of local tissue injury (Cold et al, 1981; Enevoldsen & Jensen, 1978; Feischi et al., 1974). Because cerebral metabolic state can not be inferred from CPP, assumptions about CBF based upon CPP remain tentative.

Based on known relationships between CPP and CBV, elevated CPP suggested that CBV increased during suction catheter insertion. As blood flows more rapidly through the cerebral vessels there is less time for cellular exchange of nutrients and metabolic end-products. Decreased CVR increases cerebral blood volume to insure adequate delivery of nutrients and oxygen to cerebral cells (Mulvany & Aalkjaer, 1990; Rubanyi et al, 1986). The relationship among CBF, CBV, and rate of CBF is expressed by the Stewart-Hamilton equation where CBV = CBF/mean transit time (Meier & Zierler, 1954). Although the increase in CPP during suction catheter insertion suggests that global CBF and CBV increase, regional differences dependent on degree of tissue injury, must be taken into
consideration.

Although average CPP increased during suction catheter insertion, only three subjects experienced excessive CPP response (CPP ≥ 130 mm Hg.) to suction catheter insertion. This finding concurs with CPP responses reported by other suctioning researchers (Campbell, 1989; Crosby & Parsons, 1992; Ersson et al., 1990; Parsons & Shogun, 1984; Fisher et al., 1982; Rudy et al., 1991). Elevated CPP promotes increased cerebral edema through extravasation of fluid into interstitial spaces (Rosner & Becker, 1984; Shapiro, 1975; Simard & Bellefleur, 1989; Youmans, 1982). Based on the 130 mm Hg. criteria for elevated CPP, suction catheter insertion does not appear to threaten patient safety through the sequelae of excessive cerebral perfusion. However, because there is a dearth of studies on what constitutes excessive CPP, it is possible that much lower CPP levels may promote cerebral edema in the severely head injured person. Thus, it is prudent to maintain a narrow range of CPP during suctioning.

Increased CPP requires either increased MAP or decreased ICP. The relationship between CPP, MAP, and ICP in the current study is illustrated in Figure 14. The predominant influence of MAP on CPP response pattern is evident, except during actual suctioning where MAP remained constant and ICP increased. Based on the parallel MAP and CPP response patterns, of the three physiologic processes proposed to initiate cerebrovascular response in the current study, the systemic vasopressor response appears instrumental in elevating CPP.
Figure 14. Cerebral Perfusion Pressure (CPP), Mean Arterial Pressure (MAP), and Intracranial Pressure (ICP) Across the Suctioning Procedure (N = 30)

CPP decreased in five subjects during suction catheter insertion. Of those five subjects, four had CPP levels near 70 mm Hg. before and during suction catheter insertion. Four additional subjects had CPP levels less than 70 mm Hg. at baseline, although their values improved somewhat during suctioning. With the exception of one case, all subjects with low CPP also had arterial hypotension throughout the entire suctioning procedure.

Other studies in the head injured population also reported individual cases
throughout the entire suctioning procedure.

Other studies in the head injured population also reported individual cases of low CPP before and during general suctioning (Ersson et al., 1990; Fisher et al., 1982; Rudy et al., 1991). CPP levels of 70 mm Hg. or less endanger cerebral viability through cerebral hypoxia (Rosner & Becker, 1984; Rosner & Coley, 1986; Rosner & Daughton, 1990). Based upon current and previous findings, head injured patients who have low perfusion levels prior to suctioning are at high risk for sustaining inadequate cerebral perfusion during suction catheter insertion.

The Effect of Cerebral Compliance on ICP Response to Suction Catheter Insertion

After determining that suction catheter insertion significantly elevated ICP, the study sought to determine if state of cerebral compliance, prior to the suctioning procedure (during resting baseline), affected magnitude of ICP response during suction catheter insertion. It was hypothesized that subjects with decreased compliance would have higher ICP in response to suction catheter insertion than subjects with normal compliance. There was a significant difference in ICP between normal and decreased cerebral compliance groups, with the decreased compliance group exhibiting higher ICP levels. The finding is consistent with past studies that demonstrated elevated ICP in severely head injured patients with decreased cerebral compliance, as measured by the Pressure-Volume Index (Maset, Marmarou, Ward & Young, 1986), the Volume-Pressure Response test
(Rowed, Leech, Reilly & Miller, 1975), and the Amp-Pressure curve (Szewczykowski et al., 1977).

For the current investigation, state of cerebral compliance was assessed by measurement of ICP waveform configuration (shape, amplitude and slope). The degree of isomorphism between cerebral compliance and ICP configuration remains equivocal, in part because cerebral compliance measurement is fraught with problems of accuracy and interpretation, and because of the conceptual distance between ICP configuration and cerebral compliance. Methodological validity therefore, remains an important issue that potentially weakens the study's claim that decreased compliance contributes to increased ICP during suction catheter insertion. To address the validity of ICP configuration as an indicator of cerebral compliance, study ICP configuration data were reanalyzed for information about the underlying cerebral dynamics they represented.

**ICP waveform configuration.** Subjects who demonstrated rounded and/or peaked ICP waveforms with a steep descent to baseline, or if visible, had ICP P₂ and P₃ subwaves with higher amplitudes than the P₁ subwaves, were identified as having decreased cerebral compliance. Subjects grouped with normal compliance had ICP waveforms that, after initial upward inflection, displayed a plateau and gradual descent to baseline, or if visible, had predominant P₁ subwaves. The preceding criteria were derived from past research (Cardoso et al., 1983; Chopp & Portnoy, 1980; Portnoy & Chopp, 1981; Portnoy et al., 1982).
As presented in Chapter 4, subjects' ICP waveforms used to determine state of cerebral compliance were further categorized either as arterial waveforms (peaked ICP waveforms with an elevated P₁ subwaveform) or as venous waveforms (rounded waveforms with elevated P₂ and P₃ subwaveforms). Significant dependence was found between venous shaped (rounded) ICP waveforms and the decreased cerebral compliance classification, while those with arterial waveform configuration were associated with normal compliance classification.

The relationship between rounded ICP waveforms and decreased compliance is supportive of past research where predominantly venous shaped ICP waveforms, similar to right atrial waves in shape, were theorized to result from elevated pressure and volume in the low pressure cerebrovascular bed, indicative of decreased cerebral compliance. Increased vascular volume from experimentally induced hypercapnia and hypoxemia was shown to precipitate ICP waveform rounding and elevation of P₂ and P₃ subwaveforms (Cardoso et al., 1983; Portnoy & Chopp, 1981).

The possibility that arterial shaped ICP waveforms may represent an intracranial dimension other than decreased compliance has been suggested by findings of other researchers. Portnoy and associates (1982) demonstrated that when cerebrovascular tone is diminished, arterial pressure transfer to the cerebral vascular bed is less modified, resulting in ICP waves that mimic systemic arterial
waves in shape.

The preceding data, combined with the finding that subjects classified with decreased cerebral compliance had significantly higher ICP during suction catheter insertion, suggests that regardless of the phenomenon represented, head injured patients with venous shaped ICP waveforms are at greater risk for experiencing potentially dangerous ICP response during suctioning. ICP waveform configuration analysis may offer information about both the state of compliance and cerebrovascular tone. The information regarding both intracranial dimensions derived from analysis of ICP waveforms increases the clinical efficacy of waveform analysis for nurses. The increased specification of information derived from ICP waveform changes provides a means for nurses to individualize care of head injured adult patients. For example, patients with predominantly arterial waveforms may not be capable of responding to hyperventilation maneuvers to blunt ICP response to suctioning and other stimuli, while patients with predominantly venous waveforms may benefit from hyperventilation.

In summary, the determination of cerebral compliance by ICP waveform configuration requires further study. However, current study findings demonstrated that patients with rounded ICP waveforms were at risk for potentially dangerous ICP response to suction catheter insertion. ICP waveform configuration is a viable, easily used assessment process nurses can implement in their bedside practice.
ICP amplitude. In addition to ICP waveform shape, ICP amplitude was assessed for its relationship to state of cerebral compliance. Study findings indicated that the decreased compliance group had a significantly larger average baseline ICP amplitude than the normal compliance group. The preceding findings concur with past research, where significant inverse linear correlations were found between ICP amplitude and cerebral compliance (Bering, 1955; Cardoso, Reddy, & Bose, 1988; Gonzalez-Darder & Barcia-Salorio, 1989; Hamer et al, 1977; Newell et al., 1992; Szewczykowski et al., 1977).

ICP slope. The descending slope of ICP waves was the final aspect of ICP configuration that was explored for its relationship with state of cerebral compliance. It was hypothesized that head injured subjects classified as having decreased cerebral compliance would have steeper descending slopes that those with normal compliance. Average baseline ICP descending slope was larger for the decreased compliance group compared to the normal compliance group, although not significantly different (See Table 5, Chapter 4). The effort to identify differences in ICP descending slope related to state of cerebral compliance was entirely exploratory. No previous investigations of ICP slope were found, although it was identified as an aspect of ICP waveform configuration (Cardoso et al., 1983; Chopp & Portnoy, 1980; Portnoy & Chopp, 1981; Portnoy et al., 1982).

Because calculation of slope is the dividend of amplitude over time, it
offers a numerical value of the interaction between ICP amplitude and the shape of the ICP wave. Although ICP slope did not offer different information about head injured adults' cerebrovascular status than that provided by ICP amplitude and shape, it is a more inclusive, and more quantitative measure of change in ICP waveform configuration. The ability to quantify change in configuration may limit measurement error associated with the more qualitative assessment of overall waveform shape. Finally, although it was beyond the scope of the current study to identify a specific criteria for elevated and normal ICP slope, such a value may offer a means to identify patients at increased risk for experiencing a deleterious ICP response to stimuli such as suctioning.

Conclusions

In conclusion, study findings support that increased ICP during endotracheal suctioning is a predominately neurogenic-based response, initiated by insertion of the suction catheter into the endotracheal tube. Increased intrathoracic pressure and the systemic vasomotor response, appear to be the major mechanisms resulting in elevated ICP during suction catheter insertion. During actual suctioning compared to suction catheter insertion, additional mechanisms such as increased cerebrovascular volume and edema may also contribute to the continued ICP increase. Finally, decreased cerebral compliance as evidenced by ICP waveform rounding, was identified as a factor that contributed to higher ICP levels during suction catheter insertion. The preceding findings have implications
for both nursing research and practice.

**Study Strengths and Limitations**

**Strengths**

Although the study was a secondary analysis of data collected from a previous study, the investigator collected the majority of the original data. Therefore, unlike most secondary analyses, the strengths and limitations imposed by the quality of the data collection process were known to the current researcher. Additionally, because the waveform data collected during the original study were in a continuous real time format, waveform data specific to the current study’s purpose were readily accessible. The following discussion focuses on strengths of the current study.

1. The study was driven by a comprehensive theoretical framework designed to explain and predict relationships among study variables. The relationships between study variables and the physiologic constructs they represented were clearly specified.

2. The individual components of the suctioning procedure that were the focus of this within-subject study, were clearly delineated in real time. Additionally, the time interval for baseline data was reduced to approximate the duration of other suctioning procedure events. The near equality of duration for suctioning procedure events reduced the introduction of baseline bias during statistical testing.
3. The data were derived from continuous real time waveform data that were displayed on both hard copy and computer. Thus, by using the original raw waveform data, the current study was not dependent upon data that had been previously reduced or in any way manipulated by others. Additionally, the availability of the original continuous waveform data allowed measurement replication by a second person to assess data reliability.

Limitations

The study was a secondary analysis, that by its nature imposes limits on study design. The focus of the following discussion is current study limitations, rather than those of the original study from which the data were derived.

1. Although irritant receptor response was identified as the physiologic basis for the phenomena that were the focus of the study, it was not directly measured. Increased intrathoracic pressure, primary cerebral vasodilation, and bronchoconstriction were identified as the major pathways through which the irritant receptor response resulted in increased ICP, yet none of the three phenomena were measured. The effect of attempted coughing by subjects in response to insertion of the catheter was indirectly assessed, although coughing may have had a substantial influence on the study’s dependent variables. The failure to directly assess these physiologic processes mandated that study findings often remained in the realm of conjecture, and thus limited the ability to specify interventions to prevent ICP increase during ETS.
2. Because the original study was not designed to investigate ICP waveform configuration, the waveform recorder was not adjusted to maximize visual waveform analysis. Adjustment of the recorder may have improved the reliability of the waveform measure.

3. The effect of various therapeutic measures on study outcome variables was not controlled by the study design. Although data were collected and descriptively analyzed for medications administered, interactions between medications and study variables were not assessed. The major threat that medications posed was that they had the potential to diminish ICP response to suction catheter insertion, and therefore, threatened internal validity. Significance effects were found regardless of any effect medications may have had on cerebrovascular and vasopressor responses. It is also recognized that the presence of medications could affect the ability to generalize findings to head injured patients who may receive different therapeutic regimens.

4. A major weakness in the study's conceptualization was the use of ICP waveform configuration as an indicator of state of cerebral compliance. Although this aspect of the study was exploratory in nature, the conceptual distance between ICP waveform shape and cerebral compliance threatens internal validity and therefore the credibility of study findings.

5. Although the focus of this discussion is limitations of the current study, a critical limitation for the study that arose from the original study was that ICP
measurement was derived from several different ICP monitoring devices. Because the different devices measured ICP from different intracranial spaces (for example, subarachnoid versus ventricular pressure), the influence of intracranial location on ICP configuration can not be eliminated.

**Recommendations for Future Research**

The findings of this study offer a new direction for suctioning research in severely head injured adults. Previous nursing studies of suctioning in the head injured have focused on the act of suctioning, without determining how the suctioning procedure initiated the well documented potentially dangerous cerebrovascular response. The current study demonstrated that insertion of the suction catheter initiated significantly increased ICP. Based upon study findings, future research must focus on preventing or blunting the neurogenic-based response to catheter insertion. The focus on a mechanically induced neurogenic process that resulted from catheter insertion diverges from previous suctioning research. Previous investigations studied suctioning in general, and did not specify how the response may have occurred. Because the study requires a change in focus for suctioning research, replication is needed to insure the validity of its findings. While replication would increase the strength of the current study’s claims, future studies could be strengthened by incorporating the following recommendations.

1. Subjects’ cough effort in response to suction catheter insertion should be
measured. Cough effort could be measured by observation (White et al., 1982) or by a more quantitative estimate, such as measuring diaphragmatic muscle activity.

2. Future study of the effect of suction catheter insertion should include measurement of intrathoracic pressure, airway reactivity, and cerebral vasodilation. It is recognized that many methods for measurement of each of the three mechanisms are invasive and are outside the domain of nursing practice. However, through multidisciplinary research or additional specification of subject selection criteria could provide means of better assessing the preceding physiologic phenomena. Intrathoracic pressure could be more closely approximated by limiting subject selection to those who have a central venous pressure line. Airway reactivity could be estimated by noninvasive measurement of airway pressure. Cerebral vasodilation poses the greatest challenge for measurement. However, dilation could be indirectly assessed by CBF studies. Additionally, noninvasive cerebral doppler techniques hold great promise as a tool for estimation of cerebrovascular dynamic for nurse researchers.

3. To understand better how suctioning impinges upon the cerebral vasculature, systemic arterial systolic and diastolic pressures should be measured and assessed separately. By separating the systolic and diastolic components, the effects of forward arterial pressure and retrograde venous pressure on intracranial dynamics could be assessed.

4. The possible effects of therapeutic measures on subjects' responses to
suctioning requires better control. It was noted in the current study, for example, that subjects with normal cerebral compliance received more medications than those with decreased compliance. A tool such as the Therapy Intensity Level Index, created by Lawrence Marshall and modified by Maset and associates (1986), would provide a means to quantify level of therapy so that if necessary its effects could be statistical controlled.

Although study replication will increase study generalizability, concurrent interventional research is recommended. Recommended nursing interventions pose no risk and may benefit head injured patients. The findings from the current study point to a new, potentially fruitful area for nursing research. Nursing actions to minimize irritant receptor stimulation, such as stabilizing the endotracheal tube during suctioning, using the smallest diameter suction catheter feasible for effective suctioning, and inserting the suction catheter a specified minimal distance beyond the length of the endotracheal tube must be studied under controlled conditions to evaluate their efficacy in attenuating patients cerebrovascular responses to suctioning. Multidisciplinary investigations of the effects of anesthetic medications, such as lidocaine, on blunting the irritant receptor response to suction catheter insertion are also needed.

The assessment of ICP waveform configuration requires further study to determine the intracranial dimension represented by change in ICP configuration, and how it may best be used by nurses to assess head injured patients. Further
study of ICP descending slope and amplitude may offer more quantitative
measures of change in configuration. Finally, it is recommended that other
measures of cerebral compliance and cerebral vasoreactivity be included in future
study of ICP configuration.

Recommendations for Nursing Practice

The findings from this study reemphasize, that while endotracheal
suctioning is a necessary nursing action for intubated severely head injured adults,
it is not a benign procedure. The focus of nursing practice recommendations
derived from the current study is on minimizing or avoiding airway reflex
activation.

Although research is needed, there are simple actions that nurses can
implement immediately to reduce the risk of detrimental cerebrovascular response
to suctioning. First, the patient's endotracheal tube should be stabilized
throughout the ETS procedure to prevent movement. Currently, standard suction
catheter size used in adult patients is usually a number 14 (French) catheter.
However, because the head injured population is youthful and generally are in
good health prior to injury, copious or tenacious secretions are often absent.
Therefore, a smaller suction catheter may be sufficient to maintain a patent
airway, and may lessen airway stimulation upon insertion. It is recommended that
the suction catheter be inserted only slightly beyond the end of the endotracheal
tube, to insure continued tube patency while minimizing irritant receptor
stimulation. To determine how far to insert the catheter, the length of endotracheal tube from a standard reference (such as from the lip or adaptor) must be known. Suction catheters with measurement lines like those currently used with neonates would be useful in determining insertion depth.

During suctioning, CPP and MAP should be maintained within normal range, if possible. Although the effects of low CPP and MAP have been emphasized in the past, current study findings suggest increased CPP and MAP threaten patient viability by promoting increased intracerebral volume and diminished cerebrovascular tone. Although specific values based on study findings are tentative, MAP values greater than 100 mm Hg. may contribute to loss of cerebral vasoreactivity and pose a potential danger in severely head injured patients.

While nurses routinely monitor severely head injured patients' ICP magnitude, the current study suggests that assessment of ICP configuration offers additional information about intracranial status. Patients who exhibit rounded ICP waves may be at greater risk of developing increased ICP in response to suctioning. Special precautions to minimize ICP increase, such as administering medications to lower ICP at optimal presuctioning times, minimizing stimulation prior to and during suctioning, and using hyperinflation breaths for presuctioning hyperoxygenation in select patients, may blunt patient ICP response to suctioning.

In summary, findings from the present study offer simple interventions
nurses can implement immediately to minimize potentially dangerous
cerebrovascular responses to suctioning in severely head injured adults. Although
suctioning is a necessary nursing procedure in intubated head injured adults, it is
imperative that nurses take action to minimize danger in the vulnerable head
injured patient population.
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Appendix A

Exemplars of Normal ICP Waveform Configuration

Appendix B

Exemplars of Abnormal ICP Waveform Configuration

![Graphs showing different waveforms with annotations](image)
Appendix C

Preparation of Data for Inferential Analysis

Data Reduction

Two baseline measurements, each of five seconds duration, were taken within one minute before initiation of the hyperoxygenation breaths. Data from the two timepoints were compared using a dependent t-test. Because no significant differences were found, the data from the two timepoints were combined for a total of 10 seconds baseline data.

Although baseline provided the best estimate of subjects' resting vascular values, safe clinical practice required that ETS was preceded by several highly oxygenated breaths (hyperoxygenation event). Elevated oxygen levels cause cerebral vasoconstriction, and therefore, had the potential to lower ICP. Waveform data collected during the hyperoxygenation event were measured by both a computerized program and by manual measurement. The computerized method included waveform data that occurred during the delivery of each hyperoxygenation breath as well as data that occurred between each delivered breath. The manual method included waveform data that occurred only during the delivery of the four breaths.

To determine whether the two measurement methods were equivalent, 10 randomly selected subjects' ICP data were measured by both computer and manual methods. Using dependent t-tests for within subject comparisons, ICP data
derived from the two methods were compared. Although average MICP measured by computer ($M = 19.6$ mm Hg.) was equivalent to that derived by the manual method ($M = 20.0$ mm Hg.), the difference was statistically significant, $t (9) = 2.45, p < .05$. From a clinical perspective, however, the MICP differences are irrelevant and would not result in different intervention decisions. Therefore, data derived by both methods were included in analysis.
Table C-1

Heart Rate (HR), Arterial Pressure (MAP), Intracranial Pressure (MICP), and Cerebral Perfusion Pressure (CPP) at Baseline, Hyperoxygenation, and Ventilator Disconnection (N = 30)

<table>
<thead>
<tr>
<th>Variable</th>
<th>T1*</th>
<th>T2b</th>
<th>T3c</th>
<th>Timepoints compared</th>
<th>t(df)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MICP (mm Hg.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M(SD)</td>
<td>16(10)</td>
<td>17(10)</td>
<td>17(11)</td>
<td>T1 - T2</td>
<td>-1.90(29)</td>
</tr>
<tr>
<td>Range</td>
<td>1 to 46</td>
<td>2 to 47</td>
<td>2 to 49</td>
<td>T1 - T3</td>
<td>-2.43(29)*</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>T2 - T3</td>
<td>-1.52(29)*</td>
</tr>
<tr>
<td>CPP (mm Hg.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M(SD)</td>
<td>88(19)</td>
<td>91(20)</td>
<td>89(16)</td>
<td>T1 - T2</td>
<td>-2.01(29)*</td>
</tr>
<tr>
<td>Range</td>
<td>50 to 135</td>
<td>50 to 150</td>
<td>62 to 126</td>
<td>T1 - T3</td>
<td>.09(29)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>T2 - T3</td>
<td>-1.98(29)</td>
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<tr>
<td>HR (BPM)</td>
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<td></td>
</tr>
<tr>
<td>M(SD)</td>
<td>92(21)</td>
<td>96(22)</td>
<td>96(22)</td>
<td>T1 - T2</td>
<td>-1.55(29)</td>
</tr>
<tr>
<td>Range</td>
<td>57 to 132</td>
<td>60 to 141</td>
<td>60 to 141</td>
<td>T1 - T3</td>
<td>-1.60(29)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>T2 - T3</td>
<td>.57(29)</td>
</tr>
<tr>
<td>MAP (mm Hg.)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M(SD)</td>
<td>104(18)</td>
<td>107(19)</td>
<td>106(17)</td>
<td>T1 - T2</td>
<td>-2.59(29)*</td>
</tr>
<tr>
<td>Range</td>
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<td>61 to 159</td>
<td>68 to 140</td>
<td>T1 - T3</td>
<td>-1.08(29)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>T2 - T3</td>
<td>-1.36(29)</td>
</tr>
</tbody>
</table>

*T1 = baseline.

*T2 = hyperoxygenation breaths.

*T3 = ventilator disconnection.

*p < .05, two-tailed paired t-tests.