

# Effect of Backrest Position on Intracranial Pressure and Cerebral Perfusion Pressure in Individuals with Brain Injury: A Systematic Review



Jun-Yu Fan

**A**bstract: Head elevation is a conventional nursing procedure for brain-injured individuals with intracranial hypertension; it is performed with the intent of reducing intracranial pressure (ICP) by means of a noninvasive physical intervention. However, in certain circumstances, head elevation puts the brain-injured individual at risk for secondary cerebral injury because of impaired arterial blood pressure and compromised cerebral perfusion pressure (CPP). A systematic literature search was conducted to evaluate existing evidence regarding the effect of changing the backrest position on ICP and CPP in brain-injured individuals. Eleven articles were retrieved. In nine articles it was concluded that ICP significantly decreased at 30 degrees of head elevation compared with a flat position. Five of the nine articles showed no statistical significance in the magnitude of change in CPP from a flat position to 30 degrees of head elevation. Major limitations in the 11 articles were small sample sizes and unclear study protocols, which may have caused a failure to detect the effect of head elevation. In clinical practice, intensive care unit staff members need to cautiously perform head elevation with a thorough understanding of its physiologic effect and potential hazard. Future research should investigate the effects of therapeutic positions on different neurological and neurosurgical populations and explore the combination of head elevation and lateral side-lying positions.

Characteristics of the primary injury are not the major determinant of outcome for individuals with brain injury. It is the secondary injury, developing after the acute phase of the primary injury, that plays a crucial role in determining outcome for individuals with brain injury (Chesnut, Marshall, Piek et al., 1993; Cormio, Robertson, & Narayan, 1997; Jones et al., 1994; Narayan, 1995). The causes of secondary injury include both intracranial and extracranial factors. The intracranial factors include the cascade sequence of pathophysiological mechanisms of brain injury, biochemical mechanism, and cellular, metabolic, and chemical events (Dearden, 1998). Extracranial

factors, such as hypoxia, pyrexia, and hypotension, also evolve over time (Chesnut, Marshall, Klauber, et al; Chesnut, Marshall, Piek, et al; Dearden, 1998; Heath & Vink, 1999; Jones et al., 1994; Pietropaoli et al., 1992; Signorini, Andrews, Jones, Wardlaw, & Miller, 1999). More extensive and permanent damage is caused by the secondary injury (Gualtieri, 2002). Many studies have shown that frequent episodes of secondary insults yield poorer outcomes (Boumna, Muizelaar, Choi, Newlon, & Young, 1991; Downard et al., 2000; Jones et al., 1994; Marmarou et al., 1991; Rosner & Daughton, 1990; Rosner, Rosner, & Johnson, 1995).

After brain injury, intracranial hypertension and insufficient cerebral perfusion pressure (CPP), resulting from both primary and secondary injuries, are the major concerns during care of individuals with brain injury (Signorini et al., 1999). Intracranial hematoma, edema, vascular engorgement, and hydrocephalus are common causes of intracranial hypertension after brain injury and occur in 50%–75% of individuals with severe brain injuries (Dearden, 1998; Miller, Deardan, Piper, & Chan, 1992). These consequences are associated with 69%–95% mortality, especially in individuals with increased intracranial pressure (ICP) refractory to treatment (Alberico, Ward, Choi, Marmarou, & Young, 1987; Miller et al., 1977; Saul & Ducker, 1982; Signorini et al., 1999). Representing the pressure gradient between cerebral artery and venous vasculature, CPP is calculated by arriving at the difference between mean arterial blood pressure (MABP) and ICP. CPP is usually maintained above 70 mm Hg to meet the requirement of supplying cerebral metabolism via cerebral autoregulation (Brain Trauma Foundation, 2000b; Rosner & Daughton, 1990). Failure to maintain an adequate CPP worsens the existing ischemic zone and jeopardizes the normally perfused regions (Brain Trauma Foundation, 2000b). Several studies disclosed systemic insults such as unstable hemodynamics, hypoxia, and pyrexia, which have a critical impact on the development of secondary brain injury (Chesnut, Marshall, Klauber et al., 1993; Chesnut, Marshall, Piek, et al., 1993; Dearden, 1998; Heath & Vink, 1999; Jones et al., 1994; Pietropaoli et al., 1992; Signorini et al., 1999). The goals of treating individuals with brain injury are not only to attenuate the impact of the secondary injury

Questions or comments about this article may be directed to Jun-Yu Fan, MSN RN, by phone at 206/543-6227 or by e-mail at [jjfan@u.washington.edu](mailto:jjfan@u.washington.edu). She is a doctoral candidate at the University of Washington School of Nursing, Seattle, WA.

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through the management of intracranial hypertension but also to preserve adequate CPP and to ensure maintenance of systemic hemodynamic function during the acute stage (Gualtieri, 2002).

In neurosurgical and neurological intensive care units, head elevation is a conventional nursing procedure in the care of brain-injured individuals with intracranial hypertension; it is performed with the intent of reducing ICP by means of a noninvasive physical intervention that promotes intracranial venous return and increases cerebrospinal fluid (CSF) drainage from the head (Kirkness, 1992). Recently, this traditional nursing practice of head elevation has come into question because of its potential to decrease CPP by decreasing the arterial blood pressure (Kirkness, 1992). The purpose of this systematic review is to evaluate the existing evidence regarding the effects of changing the backrest position on ICP and CPP in individuals with brain injury.

### Conceptual Basis

Head elevation, a conventional nursing procedure, is performed routinely for brain-injured individuals with intracranial hypertension. The theoretical basis is that the head is above the level of the heart on the vertical axis, and as a result, CSF is redistributed from the cranial to the spinal subarachnoid space (Kenning, Toutant, & Saunders, 1981), and it facilitates cerebral venous return (Magnaes, 1976; Magnaes, 1978; Marmarou, Shulman, & LaMorgese, 1975; Potts & Deonaraine, 1973). The redistribution of CSF in response to head elevation occurs immediately after a change in position because of free communication between the cranial and spinal subarachnoid spaces (Magnaes, 1978). The major routes for cerebral venous drainage include the internal jugular veins with the accessory systems of the external jugular veins and the vertebral venous plexi (Kenning et al., 1981). All these venous systems are valveless channels that allow cerebral venous return without interruption after head elevation. The postural impact on the systemic hemodynamics (e.g., when the patient is moved from a supine to an upright position) causes approximately 30% of the blood volume from the upper body to be suddenly displaced into the peripheral vein. In addition to the intravascular pressure in the heart, gravity contributes an additional pressure component to vessels below the heart. That is, standing up causes the compliant veins to distend. This is known as a *venous pooling*, an effect of hydrostatic pressure. Together these decrease venous return.

For a healthy individual, a sudden shift in blood volume has little effect on systemic arterial blood pressure (SABP) and CPP because various mechanisms maintain adequate cardiac output and cerebral blood flow. Maintaining SABP involves compensatory mechanisms such as baroreceptor reflexes, vasoconstriction, and the pumping effect of the skeleton muscles and the lungs that help

facilitate venous return to the heart. However, these mechanisms become less effective when an individual has prolonged bed rest. Cerebral autoregulation is the most important mechanism for maintaining CPP. It maintains a relatively constant cerebral blood flow to the brain across a range of MABP from 50 to 150 mm Hg. This is accomplished by regulation of the diameter of the resistant vessels including the major arteries in the brain parenchyma and the pial arteries (Mchedlishvili, 1980; Rogers & Stump, 1989). Impaired cerebral autoregulation is commonly associated with pathological intracranial conditions such as traumatic brain injury.

Thus, head elevation can produce a particular dilemma for healthcare providers because in certain circumstances elevating the head of the bed does not ensure decreasing ICP. On the contrary, it may put some individuals at risk for increasing ICP and cerebral ischemia due to impaired cerebral autoregulation and unstable arterial blood pressure (Rosner & Coley, 1986; Simmons, 1997). It is therefore critical to evaluate the existing evidence concerning the effects of changing the backrest position on ICP and CPP in brain-injured individuals.

### Methods

#### Sampling and Criteria

Data for this systematic review were retrieved from electronic databases such as MEDLINE, CINAHL, PsycINFO, Health STAR, and Cochrane Library, as well as from dissertation abstracts, a university library catalog, and the bibliographies of relevant studies. Keywords included *position*, *intracranial pressure (ICP)*, *cerebral perfusion pressure (CPP)*, and *head elevation*. The search was restricted to works in English published between 1980 and 2003. The following criteria were used to identify appropriate articles: (a) the representation of brain-injured patients including those with traumatic brain injury, subarachnoid hemorrhage (traumatic or aneurysm), stroke, brain tumor, and hydrocephalus; (b) therapeutic positions including supine (flat), head elevation of different degrees with or without knee-gatch, and reverse Trendelenberg; and (c) content including major outcome variables of ICP or CPP. Exclusion criteria were (a) case reports (excluded because they lack statistical inference to validate their scientific significance); and (b) studies with subjects less than 18 months of age (excluded because of the subjects' unfused skulls).

Articles were systematically analyzed and placed into the categories of author(s), year of publication, study title, research design, therapeutic backrest positions, sample size, age range (mean), pathological condition, Glasgow Coma Scale (GCS) score, ICP device, and physiologic parameters (ICP, CPP, MSAP, etc.); see Table 1. Statistical methods, major findings, effect size, strengths, and limitations of studies, and comments on each are presented in Table 2.

**Table 1. Study Characteristics**

Reference	Title	Research Design	Bedside Care Activities	Sample (N)	Age (Mean)	Pathological Condition	GCS (Mean)	ICP Device	Measure Parameter
Kenning, Toutant, & Saunders, 1981	Upright patient positioning in the management of intracranial hypertension	Descriptive	Position Change Flat 45° 90° *Flexed at hip	24	7-79 (36)	9 with various cranial abnormalities; 15 with severe head injury (GCS < 8 in 14 patients)	3-15 (8.2)	Intraventricular catheter (7) Subarachnoid bolt (17)	ICP
Ropper, O'Rourke, & Kennedy, 1982	Head positioning, intracranial pressure, and compliance	Descriptive	Position Change Flat 60° *Flexed at hip	19	15-77	13 with head injury; 5 with ICH; 1 with stroke	Not reported	Subarachnoid bolt (19) 4 out of 19 also had intraventricular catheter (4)	ICP
Durward, Amacher, Del Maestro, & Sibbald, 1983	Cerebral and cardiovascular responses to changes in head elevation in patients with intracranial hypertension	Quasi-experimental design (pre-post test)	Position Change Flat 15° 30° 60° *Flexed at hip	11	Not reported	8 with acute brain injury; 3 with anoxic brain injury related to near drowning	GCS < 8	Intraventricular catheter (11)	ICP CPP MABP at head PAP PCWP CVP CO
Parsons & Wilson, 1984	Cerebrovascular status of severely injured patients following passive position changes	Quasi-experimental design (pre-post test)	Position Change Flat 35° *Flexed at hip	18	5-67	Severely head-injured patients	3T-10T	Subarachnoid bolt (18)	ICP CPP MABP at head HR
Rosner & Coley, 1986	Cerebral perfusion pressure, intracranial pressure, and head elevation	Quasi-experimental design (pre-post test)	Position Change Flat 10° 20° 30° 40° 50° *Flexed at hip	18	12-83 (36)	8 with severe head injury; 3 with ICH; 3 with tumor; 3 with hydrocephalus	3-15 (7.7)	Intraventricular catheter (18)	ICP CPP MABP at heart MABP at head CVP
March, Mitchell, Grady, & Winn, 1990	Effect of backrest position on intracranial and cerebral perfusion pressures	Within subjects experimental design (pre-post test)	Position Change Flat 30° (Flexed at hip) 30° with knee gatched Reverse Trendelenberg position	4	19-30 (23)	Traumatic head injury	4-9 4-8 (3 patients) 9 (1 patient)	Subarachnoid bolt (4)	ICP CPP CBF MABP

**Table 1. Study Characteristics (cont.)**

Reference	Title	Research Design	Bedside Care Activities	Sample (N)	Age (Mean)	Pathological Condition	GCS (Mean)	ICP Device	Measure Parameter
Feldman et al., 1992	Effect of head elevation on intracranial pressure, cerebral perfusion, and cerebral blood flow in head-injured patients	Quasi-experimental design (pre-post test)	Position Change Flat 30° *Flexed at hip	22	18–75 (35)	Head-injured patients	3–12 3–5 (3 patients) 6–8 (14 patients) 9–12 (5 patients)	Not reported	ICP CPP CBF Mean carotid pressure
Kirkness, 1992	The effect of head elevation on cerebral perfusion in patients with intracranial pathology	Experimental design (pre-post test)	Position Change Flat 30° (Flexed at hip) 30° with gatched knee flexion at 20° Reverse Trendelenberg position	7	19–73 (32)	4 with head injury; 3 with cerebrovascular pathology	4T–15 4–6T 9T 5T 7T 15 (3 patients)	Intrapatencyhymal (7)	ICP CPP CBF MABP at heart MABP at head
Schneider, von Helden, Franke, Lanksch, & Unterberg, 1993	Influence of body position on jugular venous oxygen saturation, intracranial pressure and cerebral perfusion pressure	Quasi-experimental design (pre-post test)	Position Change Flat 15° 30° 45° *Flexed at hip	25	20–79 (48)	17 with severe head injury; 5 with sub-arachnoid hemorrhage; 3 with intracerebral hemorrhage	4–8 (6)	Intraventricular catheter (7) Intrapatencyhymal (13) Epidural device (5)	ICP CPP MABP at head SjvO <sub>2</sub>
Meixensberger, Bau-nach, Amschler, Dings, & Roosen, 1997	Influence of body position on tissue-PO <sub>2</sub> , cerebral perfusion pressure, and intracranial pressure in patients with acute brain injury	Quasi-experimental design (pre-post test)	Position Change Flat 30° *Flexed at hip	22	17–71 (37)	Head-injured patients	3–5 (9 patients) 6–8 (8 patients) 9–12 (5 patients)	Not reported	ICP CPP MABP ti-PO <sub>2</sub>
Winkelmann, 2000	Effect of backrest position on intracranial and cerebral perfusion pressures in traumatically brain-injured adults	Randomized crossover experimental design (pre-post test)	Position Change Flat 30° *Flexed at hip	8	18–45 (28)	Traumatic brain injury	GCS < 8 (5)	Intraventricular catheter (2) Subarachnoid bolt (6)	ICP CPP MABP at heart

*Key: GCS = Glasgow Coma Scale; ICP = intracranial pressure; CPP = cerebral perfusion pressure; MABP = mean arterial blood pressure; CVP = central venous pressure; PAP = pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; SjvO<sub>2</sub> = jugular venous oxygen saturation; ti-PO<sub>2</sub> = tissue oxygenation; HR = heart rate; RR = respiratory rate*

## Results

The initial search yielded 43 potential references, 15 of which were directly relevant to positioning and ICP or CPP. One was published before 1980 (Shalit & Umansky, 1977); the others were published between 1980 and 2003 and included 13 published articles and 1 unpublished thesis. Three (Hugo, 1992; Jones, 1994; Lee, 1989) were excluded because of the therapeutic positions used, such as head down, head rotation, and turning. Eleven references were analyzed according to the categories described above and are summarized in Tables 1 and 2 (Durward, Amacher, Del Maestro, & Sibbald, 1983; Feldman et al., 1992; Kenning et al., 1981; Kirkness, 1992; March, Mitchell, Grady, & Winn, 1990; Meixensberger, Baunach, Amschler, Dings, & Roosen, 1997; Parsons & Wilson, 1984; Ropper, O'Rourke, & Kennedy, 1982; Rosner & Coley, 1986; Schneider, von Helden, Franke, Lanksch, & Unterberg, 1993; Winkelman, 2000).

### Study Characteristics

Five articles (45.5%) were published from 1980 to 1989, and six (54.5%) were published between 1990 and 2003. Two studies (Meixensberger et al., 1997; Schneider et al., 1993) were conducted in Germany, while the other nine were conducted in the United States. A total of 178 participants were enrolled in 11 studies, and the studies' sample sizes ranged from 4 to 25. Three studies did not provide information on the ages of the subjects (Durward et al., 1983; Parsons & Wilson, 1984; Ropper et al., 1982). Subjects in the remaining eight studies ranged in age from 7 to 83 years with a mean age of 37 years. Five studies (45.5%) focused on participants with severe head injury, and six studies (54.5%) reported that more than half of the participants had severe head injury. Ten out of the 11 studies provided the range of GCS scores, from 3 to 15; Ropper et al. (1982) was the exception. GCS scores were lower than 8 for more than half of the participants (69.1%, 124/178), a finding that implied participants had severe brain injury. Most studies reported the range instead of the individual value of GCS scores; only four studies provided each participant's GCS scores, ranging from 3 to 15 with a mean GCS score of 7 (Kenning et al., 1981; Rosner & Coley, 1986; Schneider et al., 1993; Winkelman, 2000).

### ICP Device

ICP was measured by an intraventricular catheter, an intraparenchymal or a subarachnoid bolt, or epidural devices, except that four participants were measured by both intraventricular catheter and subarachnoid bolt simultaneously (Ropper et al., 1982). Two studies involving 24.7% of the patients, or 44 out of the 178, did not report what type of device was used for collecting ICP data (Feldman et al., 1992; Meixensberger et al., 1997). Subarachnoid bolt (36.0%, 64/178) was the most commonly used device, in nine studies; other devices used (listed by order of the utilization percentage) were

intraventricular catheter (25.8%, 46/178), intraparenchymal device (11.2%, 20/178), and epidural device (2.8%, 5/178).

### Design

Two studies (18.2%) used descriptive designs; six (54.5%) used quasi-experimental designs; and three (27.3%) used experimental designs.

### Therapeutic Backrest Positions

**Flat position.** All studies used a flat position as their baseline, which meant that the mattress was horizontal to the bed frame, with the patient lying supine. None of the studies mentioned whether pillows were used.

**Elevated head of bed.** The bed was adjusted so that the upper part of the bed was elevated to the chosen degree from 10 to 90 degrees. Head elevation of 30 degrees was the most commonly used position in 8 out of 11 studies. Head elevation of 35–90 degrees was used in 3 (18.2%) studies (Kenning et al., 1981; Parsons & Wilson, 1984; Ropper et al., 1982). March et al. (1990) and Kirkness (1992) used a head elevation of 30 degrees, with gatched knee flexion at 20 degrees. The purpose of the "knee-gatch" position was to prevent the patient from sliding down the bed.

**Reverse Trendelenberg.** The reverse Trendelenberg position requires elevating the head of the bed while simultaneously lowering the foot of the bed, and keeping the mattress frame straight. The beds utilized in studies by March et al. (1990) and Kirkness (1992) allowed a 15-degree angle of elevation in the reverse Trendelenberg position with the patient supine, because the investigators considered that patients in the elevated head position experienced a certain degree of flexion at the hip that might decrease the venous return to the extremities and consequently decrease cardiac output by decreasing preload.

### Measurement Parameters

Two studies (Kenning et al., 1981; Ropper et al., 1982) used ICP as the sole outcome measure. The remaining nine used ICP, CPP, and MABP as outcome measurements, and additional outcome variables such as cerebral blood flow (CBF) (Feldman et al., 1992; Kirkness, 1992; March et al., 1990), jugular venous oxygen saturation (SjvO<sub>2</sub>; Schneider et al., 1993), and brain tissue oxygenation (ti-PO<sub>2</sub>; Meixensberger et al., 1997).

## Major Findings

### ICP Response to Head Elevation and Flat Position

ICP showed a statistically significant decrease with head elevation of 30 degrees in six studies (Durward et al., 1983; Feldman et al., 1992; Meixensberger et al., 1997; Rosner & Coley, 1986; Schneider et al., 1993; Winkelman, 2000), head elevation of 35 degrees in one study (Parsons & Wilson, 1984), head elevation of 45 degrees in one study (Kenning et al., 1981), and head elevation of 60

degrees in one study (Ropper et al., 1982). ICP values were maximal when the patients were placed flat compared with head elevation of different degrees in seven studies (Durward et al., 1983; Feldman et al., 1992; Meixensberger et al., 1997; Parsons & Wilson, 1984; Rosner & Coley, 1986; Schneider et al., 1993; Winkelman, 2000).

### **CPP Response to Head Elevation and Flat Position**

All but two studies (Kenning et al., 1981; Ropper et al., 1982) measured CPP as the main outcome variable. Four studies (Feldman et al., 1992; Kirkness, 1992; March et al., 1990; Schneider et al., 1993), showed no statistically significant changes in CPP from a flat position to 30 degrees of head elevation; one study (Durward et al., 1983) showed no change in CPP; two studies (Meixensberger et al., 1997; Winkelman, 2000) showed increased CPP; and two studies (Parsons & Wilson, 1984; Rosner & Coley, 1986) showed decreased CPP. Two studies (Kirkness, 1992; March et al., 1990) showed no statistically significant difference in CPP between a flat position and head elevation with knee gatched, and between flat and reverse Trendelenberg bed positions. Meixensberger et al. (1997) showed CPP was significantly higher at 30 degrees of backrest elevation ( $76.5 \pm 13.5$  mm Hg) than a flat position ( $71.5 \pm 13.2$  mm Hg). Winkelman (2000) showed a similar result:  $84.0 \pm 9.87$  mm Hg at 30 degrees of backrest elevation and  $79.9 \pm 9.72$  mm Hg at a flat position. Parsons and Wilson (1984) showed CPP was significantly lower at 30 degrees of backrest elevation than in the flat position. Rosner and Coley (1986) showed CPP was significantly higher at a flat position ( $73.0 \pm 3.4$  mm Hg) than at 30 degrees of head elevation ( $67.2 \pm 2.8$  mm Hg) resulting from a decrease in SABP from a flat position ( $94.8 \pm 3.1$  mm Hg) to 30 degrees of head elevation ( $85.4 \pm 2.4$  mm Hg). The ICP was  $22.2 \pm 2.3$  mm Hg and  $17.6 \pm 2.3$  mm Hg for the positions of flat and 30-degree head elevation, respectively. The authors also reported that CPP was maximized ( $73.0 \pm 3.4$  mm Hg) with patients lying flat even though ICP was usually highest ( $22.2 \pm 2.3$  mm Hg) at this point. Durward et al. (1983) showed that CPP did not decrease significantly until head elevation reached 60 degrees.

### **Effect Size**

*Effect size* is defined as the "magnitude of the difference between observations. It answers the question: 'Is the effect large or useful?' rather than the question 'Is there a difference?'" (Winkelman, 2001, p. 216). Effect size also provides information that allows the reader to determine whether the intervention is clinically significant or useful (Winkelman, 2001). One common criterion is based on sociobehavioral research: A small effect size is 0.2, a medium effect size is 0.5, and a large effect size is 0.8 (Cohen, 1988; Winkelman, 2001). Only Winkelman (2000) reported an effect sizes; for decreasing ICP it was 0.55, and for improving CPP it was 0.41, which showed that a head

elevation of 30 degrees is a moderately effective intervention for decreasing ICP and has a moderate trend toward improving CPP. According to the available information from each study, effect size for ICP and CPP were calculated retrospectively in four studies (Table 2). The effect size for ICP ranged from 0.74 to 7.68, showing a medium to large effect size, indicating that the head elevation of 30 degrees is beneficial to decreasing ICP. The effect size for CPP varied in range from 0.02 to 1.71, indicating that the head elevation of 30 degrees might not improve CPP.

### **Discussion**

Selecting sensitive outcome parameters to measure treatment effect is crucial in an intervention study. Failure to detect a significant effect can be attributed to (a) an inadequate and weak operationalization of the intervention; (b) a sample size too small to verify a statistically significant effect; (c) too much heterogeneity without comparable distribution of data within each stratum of the dependent variables; (d) an ineffective intervention; and (e) the lack of sensitivity of the measure in the outcome variable measurements (Lipse, 1990; Toseland & Rossiter, 1989). The following section discusses these issues related to integrity of outcome variables, therapeutic positioning, limitations, clinical application, and recommendations for further studies.

### **Integrity of Outcome Variables**

Treatment of individuals with brain injury focuses on prevention or reduction of intracranial hypertension and maintenance of adequate cerebral perfusion to minimize secondary injury (Yanko & Mitcho, 2001). Therefore, therapies to manage intracranial hypertension and maintain CPP should be evaluated on whether they benefit patients or place them at risk (Simmons, 1997).

An extensive body of clinical research demonstrates a correlation between high ICP and a poorer outcome in individuals with severe brain injury (Becker et al., 1977; Marmarou et al., 1991; Miller et al., 1981; Narayan et al., 1981). However, no study to date has clearly shown that lowering ICP will be beneficial for individuals with severe brain injury. If it is accepted that an ICP greater than 20 mm Hg places patients at risk for pathological changes, any therapy that lowers ICP to 20 mm Hg or below and also ensures adequate CPP might be considered beneficial, thus maximizing the likelihood of recovery (Eisenberg et al., 1990; Miller et al., 1981; Narayan et al., 1981). From this point of view, ICP is a very sensitive outcome variable and should be included in each study relevant to brain-injured patients.

Another major goal in the management of severe brain injuries is to prevent or minimize secondary brain damage by maintaining adequate cerebral perfusion. The Brain Trauma Foundation (2000b, 2000c) suggests that maintaining the CPP greater than 70 mm Hg promotes adequate brain perfusion and prevents tissue ischemia.

**Table 2. Statistical Methods, Major Findings, Strengths, Limitations, and Comments**

Reference	Statistic Methods	Major Findings	Effective Size	Strengths, Limitations, and Comments
Kenning, Toutant, & Saunders, 1981	Descriptive	ICP reduced at 45° and 90° in all 43 recordings.	N/A	No level of statistical significance was given for the decreases in ICP occurring with head elevation. Only considers one aspect of ICP so the reduction of ICP that occurred may not have resulted in an improvement in CPP. It was not stated how long the subjects were in each position before the measures were taken, so it is not known if the values were reflecting an immediate response or an equilibrated response.
Ropper, O'Rourke, & Kennedy, 1982	Descriptive t-test	10 patients' ICP values were significantly reduced at 60° ( $p < .05$ ); 7 patients' ICP values did not have differences in either position; 2 patients' values were lower in flat position.	N/A	Recommend an optimal head position should be established on an individual basis rather than routinely placing all patients in a head-raised position. Examine how positioning affected ICP but did not consider the other factors that might affect CPP.
Durward, Amacher, Del Maestro, & Sibbald, 1983	Descriptive Paired t-test or ANOVA	ICP was consistently reduced for all patients at 30° backrest position compared with flat position. Highest ICP at flat and 60°. CPP did not change significantly until it decreased significantly at 60° when compared to flat (decrease of 7.9±9.3 mm Hg).	N/A	This study incorporated parameters related to cerebral and cardiac function, thus giving a more accurate measure of cerebral perfusion. In this study, mean pressure readings were used and levels of significant change were documented, which increase the significance of the result.
Parsons & Wilson, 1984	Descriptive ANOVA	Elevation of the head from flat to 35° produced significant decreases in MABP & ICP ( $p < .05$ ). Lowering the head from 35° to flat yielded significant increases in MABP, ICP ( $F$ & $p$ not reported). There was a significant change in CPP (decrease from 73.0 ± 3.4 to 67.2 ± 2.8) and CPP was never less than 50 mm Hg.	N/A	In this study, measurements were only made for 1 minute following the position changes, so further changes may occur after this time.
Rosner & Coley, 1986	Descriptive Linear regression	Data were analyzed with linear models and yielded significant relationship between SABP at head, CPP, and CVP. This equation described the relationship between ICP and head elevation with a statistical significance of $p < .10$ to .05. Head elevation resulted in a decrease in SABP at head ( $p < .001$ ) was faster than decrease in ICP ( $p < .10$ ) resulting in a net decrease in CPP ( $p < .05$ ). CPP was maximal with patients lying flat even though ICP was usually highest at this point.	ICP = 2 CPP = 1.8	The authors suggested that the compensatory increase in arterial blood pressure that occurs to maintain CPP when the head is elevated could be considered to be a result of ischemia or increased stress on the injured brain. Thus, head elevation would be considered an adverse stimulus that should be eliminated. It was not stated how long the subjects were in each position before the measurements were taken, so it is not known if the values were reflecting an immediate response or an equilibrated response. The authors' conclusions were different than those of other investigators due to different analysis.

**Table 2. Statistical Methods, Major Findings, Limitations, and Comments (cont.)**

Reference	Statistic Methods	Major Findings	Effective Size	Strengths, Limitations, and Comments
Feldman et al., 1992	Descriptive Paired <i>t</i> -test ANOVA	Mean ICP value was significantly ( $p = .0001$ ) lower at 30° backrest position. There was no statistically significant difference in CPP ( $p = .8$ ), CBF ( $p = .657$ ) or any other parameter measured (cerebral metabolic rate of oxygen consumption, arteriovenous difference of lactate, or cerebrovascular resistance). Negative correlation between ICP and head position ( $r = -.5890$ ). The higher the ICP ( $19.7 \pm 8.3$ ) in the flat position, the greater the decrease with head elevation.	ICP = 0.74 CPP = 0.02	The authors suggested that, in general, head-injured patients with increased ICP should be maintained head elevation to 30°, which will result in a decrease in ICP without a significant decrease in CBF in the majority of patients. In this study, <i>t</i> -test score was not reported. It was not stated how long the subjects were in each position before the measurements were taken, so it is not known whether the values were reflecting an immediate response or an equilibrated response.
Kirkness, 1992	Descriptive Paired <i>t</i> -test Wilcoxon matched-pairs signed-ranks test	There were no statistically significant differences in ICP and CPP between flat position and head elevation.	CPP = 1.12	Full details described in study protocol. Small sample size ( $N = 7$ ) is the major limitation.
Schneider, von Heiden, Franke, Lanksch, & Unterberg, 1993	Descriptive Friedman test	Head elevation significantly reduced ICP from $19.8 \pm 1.3$ mm Hg at flat to $10.2 \pm 1.2$ mm Hg at 45°. There was no statistically significant change in CPP and SjvO <sub>2</sub> associated with varying head position.	ICP = 7.68 CPP = 0.17	A moderate head elevation between 15° and 30° significantly reduces ICP and, in general, does not impair cerebral perfusion. Individual responses of CPP to changes in head position were quite unpredictable.
Meixensberger, Baumach, Amschler, Dings, & Roosen, 1997	Descriptive <i>t</i> -test	The mean ICP was significantly lower ( $14.1 \pm 1.3$ mm Hg) at 30° head elevation than at 0° ( $19.9 \pm 8.3$ mm Hg). CPP was slightly higher at 30° ( $76.5 \pm 13.5$ mm Hg) than at 0° ( $71.5 \pm 13.2$ mm Hg). MABP was unaffected by head elevation. Regional ti-PO <sub>2</sub> was unaffected by body position.	ICP = 0.97 CPP = 0.37	A moderate head elevation of 30° reduces ICP without jeopardizing regional cerebral microcirculation.
Winkelman, 2000	Descriptive Repeated-measures ANOVA	ICP and CPP changes occurred immediately after elevation of the head from flat to 30° ( $F(2) = 20.21, p = .002$ ). During equilibrium, ICP was significant lower and CPP was significantly higher for a 30°-backrest elevation than for a flat position ( $F(2) = 8.323, p = .02$ ).	ICP = 0.55 CPP = 0.41	Fully described study protocol. Small sample size ( $N = 8$ ) is the major limitation; therefore, could not generalize about the result.

Key: ICP = intracranial pressure; CPP = cerebral perfusion pressure

Many studies considered ICP as the factor to predict patients' outcome but found that it was not enough to measure the cerebral perfusion (Brain Trauma Foundation, 2000a, 2000b; Hilton, 2000; Iacono, 2000). Both ICP and CPP parameters are therefore key determining factors for treatment of brain-injured patients and should be included in each brain injury study. Low CPP (< 60 mm Hg) would cause brain ischemia, especially in brain-injured patients who have had hypotensive episodes (systolic blood pressure < 90 mm Hg) during the first several hours or days after their primary injury (Brain Trauma Foundation, 2000b). Carefully monitoring the MABP is crucial because MABP is a component of CPP (equal to MABP minus ICP), and it also can reflect the adequacy of blood supply to the brain, along with its oxygen substrate (Hilton, 2000; Yanko & Mitcho, 2001).

Some evidence exists that ICP and CPP do not accurately reflect cerebral blood flow (CBF) and autoregulatory capacity (Feldman et al., 1992; Hilton, 2000). CBF was added as an outcome variable in three studies (Feldman et al., 1992; Kirkness, 1992; March et al., 1990). Results showed no statistically significant change between 30 degrees of backrest elevation and a flat position. This may be due to a sample size too small to have relevance for this issue. Jugular venous oxygen saturation (SjvO<sub>2</sub>) and brain tissue PO<sub>2</sub> (ti-PO<sub>2</sub>) were outcome variables in the studies conducted by Schneider et al. (1993) and Meixensberger et al. (1997). The results demonstrated no significant change in SjvO<sub>2</sub> or regional ti-PO<sub>2</sub> associated with various head positions. Although no evidence was available to validate CBF, SjvO<sub>2</sub>, and ti-PO<sub>2</sub> as reliable outcome variables, it is highly recommended that studies using these physiologic measures be conducted to clarify their roles in clinical practices.

### **Therapeutic Positioning**

Positioning can influence physiologic variables for brain-injured patients in the critical care setting. Head elevation is a conventional nursing intervention used to control increased ICP in patients with brain injury. Appropriate therapeutic position or optimal level-of-head elevation has been widely investigated. In most studies, head elevation up to 30 degrees, along with fixed head and neck alignment, limited hip flexion, stability of CPP, and other cerebrovascular parameters, decreased ICP. Thus, head elevation up to 30 degrees is highly recommended as a therapeutic position for increased ICP patients.

### **Limitations**

The randomized controlled trial (RCT) is thought to be the scientific gold standard and the most rigorous method for accepting a clinical treatment as the standard of care. Difficulties in conducting an RCT in intensive care units include the unpredictable and debilitating conditions of critically ill individuals. In this report, all studies were crossover designs, with patients as their own

control, except two studies (Kenning et al., 1981; Ropper et al., 1982), and only three studies were randomized crossover experimental designs (Kirkness, 1992; March et al., 1990; Winkelman, 2000).

Small sample size is the major limitation in these studies. The sample size in four studies ranged from 4 to 11 (Durward et al., 1983; Kirkness, 1992; March et al., 1990; Winkelman, 2000). Although the remainder were from 18 to 25, Winkelman (2001, p. 216) claimed that "non-significant results do not mean that there is no difference between the positions; rather it means that the researcher cannot rule out the chances or sampling variability as an explanation for the observed difference." Larger sample size and study of specific populations, such as traumatic brain injury or subarachnoid hemorrhage, are highly recommended for future investigations.

The study protocol was not fully described for either the experimental or the control groups. For example, (Feldman et al., 1992; Kenning et al., 1981; Parsons & Wilson, 1984; Rosner & Coley, 1986) did not state how long the subjects stayed in each position before the measurement was taken, so it was not possible to define whether the values reflected an immediate response or an equilibrated response. Only 3 (Durward et al., 1983; Kirkness, 1992; Winkelman, 2000) out of the 11 fully detailed their study protocol. Regarding appropriate time points for measurements, decreased ICP is expected to occur immediately (Winkelman, 2000), but might vary according to different subjects, hemodynamic status, and position manipulation. Multiple time-point measurements are recommended to capture whether the effect of decreasing ICP is transient. This systematic review revealed that all but one (Kenning, 1981) reported collecting the data during the procedure, and the timing in these 10 studies varied. Six measured only at one time point either right after the change in position (Feldman et al., 1992; Parsons & Wilson, 1984; Ropper et al., 1982; Rosner & Coley, 1986) or 10–20 minutes after the change in position (Meixensberger et al., 1997; Schneider et al., 1993). The single time-point measurement could not capture both immediate and equilibrated response; in order to capture these responses, multiple time-point measurements are necessary. In four studies, measurements were taken more than twice: The first measurement was taken at five minutes after the change in position, and 1–3 additional measurements were taken within 60 minutes (Feldman et al., 1992; Kirkness, 1992; March et al., 1990; Winkelman, 2000).

### **Clinical Applications**

Although aimed at providing empirically based guidance on positioning practice with brain-injured patients, all the studies yielded somewhat inconclusive and occasionally conflicting findings, which restricted broader evidence-based clinical applications. On the basis of the studies reviewed, two clinical recommendations for therapeutic positioning can be made: (a) consider using head

elevation up to 30 degrees to significantly reduce ICP but without significantly changing CPP, and (b) monitor CPP during head-elevation positioning because head elevation may simultaneously decrease CPP by decreasing MABP.

## Recommendations for Future Studies

A comprehensive multisystem physiologic perspective on outcome variables such as cerebral, hemodynamic, and systemic oxygenation is needed (Sullivan, 2000). These variables should be studied with large sample sizes in order to validate statistically and clinically meaningful effects. Future research should examine the effects of therapeutic positions in different neurological and neurosurgical populations such as people with cerebrovascular disorders or brain tumors, the elderly, and children in order to generalize the findings. In clinical practice, most patients with traumatic brain injury have combined multisystem injuries such as lung contusions or multiple bone fractures or underlying diseases such as chronic obstructive pulmonary disease or cardiovascular disease. Therefore, use of supine and head-elevated positions may not be sufficient to address these patients' clinical conditions. Further investigations should evaluate the effect of lateral side-lying positions on ICP and CPP for brain-injured individuals who have multisystem involvement.

## Summary

Reviewing current therapeutic positioning research has provided a template of evidence-based data from which preliminary recommendations may guide present nursing practice in the therapeutic positioning of patients with brain injury. A backrest elevation of 30 degrees is a therapeutic intervention for ICP in brain-injured patients. Ongoing and future studies are needed to generate the conclusive, empirically based determination of best practices for positioning critically ill neurology and neurosurgery patients.

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