Intracranial Pressure Waveform Morphology and Intracranial Adaptive Capacity
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Am J Crit Care. 2008;17: 545-554
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Intracranial Pressure Waveform Morphology and Intracranial Adaptive Capacity

By Jun-Yu Fan, RN, PhD, Catherine Kirkness, RN, PhD, Paolo Vicini, PhD, Robert Burr, MSEE, PhD, and Pamela Mitchell, PhD, CNRN

Background
Intracranial hypertension due to primary and secondary injuries is a prime concern when providing care to patients with severe traumatic brain injury. Increases in intracranial pressure vary depending on compensatory processes within the craniocerebral space, also referred to as intracranial adaptive capacity. In patients with traumatic brain injury and decreased intracranial adaptive capacity, intracranial pressure increases disproportionately in response to a variety of stimuli. However, no well-validated measures are available in clinical practice to predict the development of such an increase.

Objectives
To examine whether P2 elevation, quantified by determining the P2:P1 ratio (≥0.8) of the intracranial pressure pulse waveform, is a unique predictor of disproportionate increases in intracranial pressure on a beat-by-beat basis in the 30 minutes preceding the elevation in patients with severe traumatic brain injury, within 48 hours after deployment of an intracranial pressure monitor.

Methods
A total of 38 patients with severe traumatic brain injury were sampled from a randomized controlled trial of cerebral perfusion pressure management in patients with traumatic brain injury or subarachnoid hemorrhage.

Results
The P2 elevation was not only present before the disproportionate increase in pressure, but also appeared in the comparison data set (within-subject without such a pressure increase).

Conclusions
P2 elevation is not a reliable clinical indicator to predict an impending disproportionate increase in intracranial pressure. (American Journal of Critical Care. 2008; 17:545-554)
Intracranial hypertension sufficient to reduce cerebral perfusion pressure (CPP) contributes to secondary brain injury in persons with traumatic brain injury (TBI). A common problem for nurses monitoring patients with TBI is determining which patients with actual or potential intracranial hypertension are most likely to manifest transient or sustained increases in intracranial pressure (ICP) in response to nursing care or to internal disruption of intracranial pressure-volume compensatory systems. Relative elevation of the second peak or P2 component of the ICP pulse waveform has been suggested as an indicator that a patient is particularly likely to have a disproportionate increase in intracranial pressure (DIICP) in response to various noxious and nonnoxious stimuli. Therefore, the purpose of this study was to determine if P2 elevation, quantified by determining the P2:P1 ratio of the ICP pulse waveform, is a unique predictor of DIICP.

Background

Motor vehicle collisions, falls, and violence are the most common causes of TBI worldwide. In the United States, an estimated 1.4 million persons sustain a TBI annually, and nearly 5.3 million live with a TBI-related disability. In acute care, the goal of managing patients with severe TBI is to prevent or attenuate secondary brain injury to maximize recovery. Secondary brain injury is minimized by managing ICP and systemic blood pressure and by promoting perfusion sufficient to maintain brain and systemic oxygenation.

ICP, the sum of the pressures exerted within the craniospinal axis system, reflects the pressure-volume relationship within that axis system and the ability of the craniospinal space to accommodate changes in intracranial volume. The relationship among the volume of brain, cerebrospinal fluid, and other components of the intracranial system and resulting pressure is a nonlinear and hyperbolic function. The elastic properties of the craniospinal container determine how much added volume can be accommodated before ICP begins to increase. This compensatory ability has been called intracranial compliance (ΔV/ΔP) or intracranial elastance (ΔP/ΔV) by anesthesiologists and neurosurgeons, and has been called intracranial adaptive capacity by neuroscience nurses. When compensatory ability is exhausted, any further increase in intracranial volume results in increased ICP.

Intracranial hypertension, defined as an elevation of ICP greater than 20 to 25 mm Hg, develops in 40% to 75% of severe brain injuries (ie, injuries in which a patient’s score on the Glasgow Coma Scale is ≤8). Jones et al reported that 84% of patients with moderate and severe TBI had at least 1 episode of intracranial hypertension. Intracranial hypertension has a profound influence on patient outcome through its adverse effect on cerebral perfusion, which may result in brain ischemia and secondary brain injury.

Nursing care activities and other external stimuli can result in transient or sustained periods of intracranial hypertension, and no clear indices exist to help determine which patients may respond adversely to such stimuli. Mitchell has suggested that these differences in responses to stimuli are a function of the ability of an individual’s craniospinal system to maintain an equilibrium between pressure and volume in the cranium, referred to as intracranial adaptive capacity. A nursing diagnosis of “decreased intracranial adaptive capacity” was proposed by Mitchell to characterize patients with “failure of normal intracranial compensatory mechanisms manifesting as DIICP” in response to a variety of noxious and nonnoxious stimuli. DIICP was defined as a response to internal or external stimuli with an increased ICP that is greater than baseline ICP by 10 mm Hg or more for 3 minutes or longer.

About the Authors

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Corresponding author: Jun-Yu Fan, RN, PhD, Department of Nursing, Chang Gung Institute of Technology, 261 Wenhwa 1st Road, Kwei-Shan, Tao-Yuan, Taiwan 333 (e-mail: jyfan@gw.cgit.edu.tw).
or an increase in ICP that triggers the protocol for medical intervention.

Since the 1970s, several invasive methods have been developed and introduced, in a limited capacity, into clinical practice to measure the compensatory ability of the craniospinal axis space. These measures include the pressure-volume index, volume-pressure response, and the dependence of pressure-volume index on the CPP level. All of these measures have been incorporated into monitoring devices.

In addition, experimental and preliminary clinical studies have shown that changes in the amplitude and configuration of the ICP pulse waveform reflect changes in craniospinal adaptive capacity (elastance) and cerebral autoregulation. Changes in ICP pulse waveforms also occur under different physiological and pathophysiological conditions. However, the use of measures that reflect intracranial compensatory ability is still limited, and further development of measures to monitor adaptive capacity at the bedside is essential.

**ICP Pulse Waveform**

Associations between the morphology of the ICP pulse waveform and the intracranial adaptive capacity (elastance) are of particular interest as background to this study. The ICP monitoring system and the study of the ICP pulse waveform were first introduced by Lundberg. The rationale behind this study of the waveform amplitude is that “with each heartbeat there is a pulsatile increase in cerebral blood volume, the equivalent of a small intracranial volume injection, and the amplitude of the ICP pulse waveform is the response of intracranial pressure to that increment of volume, and should therefore be directly related to the craniospinal elastance.”

The ICP wave has a pulsatile quality at 2 different frequencies: 1 is synchronous with the arterial pulse, and 1 is slower, in time with breathing, reflecting the cardiac and respiratory cycles, respectively.

Under normal circumstances, ICP pulse waveforms (Figure 1) generally have 3 characteristic peaks referred to as P1 (percussion wave), P2 (tidal wave), and P3 (dicrotic wave). The first peak, the P1 wave, originates from the pulsation of the choroid plexus, is sharp, and is fairly constant in amplitude. The second peak, the P2 wave, represents the rebound after the initial arterial percussion. The P2 wave also varies in shape and amplitude more than the P1 does, and it ends in the dicrotic notch. Directly following the dicrotic notch is the third peak, the P3 wave, which has a venous origin. The dicrotic notch between P2 and P3 corresponds to the dicrotic notch of the arterial pulsation.

As the craniospinal adaptive capacity decreases (e.g., because of a rapidly expanding mass effect due to an epidural hematoma), the ICP increases, as does the amplitude of the ICP waveform. At first, all components of the waveform increase simultaneously so that 3 characteristic peaks remain visible. As ICP continues to increase, distinctive changes occur in the ICP pulse waveform. Initially the amplitudes of both of the first 2 peaks (P1 and P2) of the ICP pulse waveform increase, then P2 increases to a greater extent than P1 so that P2 is predominant, and, finally, all peaks become indistinguishable. Loss of the individual component waves has been described by many investigators as “rounding” or as a “monotonous” appearance of the ICP pulse waveform (Figures 2 and 3).

Several studies have indicated that P2 elevation, defined as a ratio of the amplitude of P2 relative to the amplitude of P1 (P2:P1) greater than or equal to 0.8, is associated with an increased likelihood of having DIICP, particularly in combination with a baseline ICP greater than 10 mm Hg. However,
in most of the ICP pulse waveform studies, researchers reported findings focused on the overall waveform amplitude, because of the lack of algorithms that allow each of the beat components to be detected automatically. These measurements mainly focused on the period during the episode of intracranial hypertension instead of on events preceding the episode of intracranial hypertension. Thus far, none of the measures have been implemented into clinical bedside monitoring systems as a real-time, beat-by-beat continuous measurement tool.

A precise, clinically oriented indicator that can be used in real time to assess craniospinal compensatory ability in patients with severe TBI is essential to determine which patients have nearly exhausted their compensatory ability and thus are prone to rapid and perhaps prolonged increases in ICP.

The research reported in this article is focused on the transition zone between compensated and uncompensated intracranial states. It is also based on an assumption that before the DIICP event, the ICP pulse waveform shows unique patterns that can be derived from the parameters of the ICP pulse waveform and can serve as clinical indicators of the real-time evolution of intracranial adaptive capacity in patients with severe TBI. The overall goal is early determination of which patients with severe TBI have or are at risk for decreased intracranial adaptive capacity.

Once the high-risk patients are known, nursing interventions to either increase compensatory ability or decrease compensatory demand can be tailored with the goal of preventing or minimizing secondary brain injury.

This study proposed to determine whether the ICP pulse waveform shows unique patterns on a beat-by-beat basis in the 30 minutes preceding the DIICP event. The specific research question was as follows: During the first 48 hours after deployment of an ICP monitor, does the ICP pulse waveform show a unique pattern of P2 elevation on a beat-by-beat basis in the 30 minutes preceding the DIICP event in patients with severe TBI?

**Method**

This study was an analysis of ICP data collected continuously as part of a prospective randomized controlled clinical trial to examine the impact of a highly visible display of CPP on the management and clinical outcome of patients with subarachnoid hemorrhage and traumatic brain injury (hereafter, the trial is referred to as the CPP project). The methods and outcomes of the CPP project are reported elsewhere. Approval for this secondary data analysis was obtained from the institutional review board of the University of Washington.

Anonymous data were available from 157 patients (124 male, 33 female) with TBI who were enrolled in the CPP project. Each patient had invasive ICP and arterial blood pressure (ABP) monitoring devices and electrocardiography leads in place. All signal data and information about the physiological parameters, injury severity, and clinical course were gathered while the patient was in the intensive care unit. Data from the first 48 hours after insertion of the ICP monitor were used for this analysis.

**Intracranial Pressure**

ICP was measured via a Camino fiber-optic catheter-tipped transducer and monitor (Camino Laboratories, San Diego, California). In the CPP project, the transducer was placed in the brain parenchyma of the prefrontal region 3 to 4 cm off the midline. The transducer had adequate bandwidth and frequency response for accurate spectral analysis, as well as standard numerical analysis.

**Arterial Blood Pressure**

ABP was measured via an intra-arterial catheter in the radial artery, referenced at the horizontal level of the heart. The catheter was connected to a fluid pressure transducer, and then the signal was input to the Spacelabs system (Spacelabs Healthcare, Issaquah, Washington).

**Signal Processing in the CPP Project**

The procedures for ICP and ABP signal processing within the Spacelabs monitor included the following steps (Figure 4): (1) the raw analog ICP signal went through a 12-Hz low-pass filter to remove the
high-frequency noise, (2) the signal was converted from analog to digital at a sampling rate of 112 samples per second, (3) the signal was converted from digital to analog by using linear interpolation, and (4) the converted ICP signal went through a 40-Hz low-pass filter to smooth the converted signal again, derived from the Integrated MultiParameter Modules 90470. Transducers were calibrated by bioengineering staff once a month, and the nurse on each shift zeroed the transducer.

**Signal Acquisition Process in the CPP Project**

The ABP, ICP, and electrocardiographic analog physiological signals acquired from the Spacelabs bedside clinical instrumentation in the intensive care unit were linked to the DATAQ DI-720 analog-to-digital converter (DATAQ Instruments, Akron, Ohio) with 16-bit precision at a rate of 2400 samples per second per channel and then resampled at the rate of 100 samples per second. After the data were processed in the DATAQ analog-to-digital converter, they were stored in both waveform format and in files summarizing the data every 5 seconds on the hard drive of a touch-screen computer and then downloaded and copied to a CD. The accuracy and reliability of the data set processed from the DATAQ analog-to-digital converter were tested in vitro with Spacelabs simulation monitors in the hospital’s clinical engineering department and then in vivo in the neurointensive care unit. Reproduction of the nominal pressure values of the input clinical monitors was typically within 1 mm Hg.

**Sample**

**Sample Selection.** Only data from patients with TBI were included in our study. To select patients with TBI who would be representative of the range of severity of injury and of ICP variability, a sampling frame was used to list every potential patient. A 3 × 3 table was constructed with rows representing high, medium, and low ranges of median ICP for each patient from the total of each patient’s hourly files. High median range was from 18.0 to 48.0 mm Hg, medium was from 13.8 to 18.0 mm Hg, and low was from 1.1 to 13.7 mm Hg. The columns represented the low, medium, and high ranges of median absolute deviation of the daily ICP. Each potential participant’s ID number was classified into one of these cells (eg, low median ICP range: low median absolute deviation). Patients with craniectomy were excluded because of the dampening effect of craniec-

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Current study</th>
<th>CCP project</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>40.0 (22)</td>
<td>37.1 (18.1)</td>
</tr>
<tr>
<td>Score on Glasgow Coma Scale after resuscitation, mean (SD)</td>
<td>7.9 (4)</td>
<td>7.3 (3.1)</td>
</tr>
<tr>
<td>Injury mode</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor vehicle accident</td>
<td>19 (50)</td>
<td>71 (45.5)</td>
</tr>
<tr>
<td>Fall</td>
<td>7 (18)</td>
<td>32 (20.4)</td>
</tr>
<tr>
<td>Motorcycle accident</td>
<td>4 (10)</td>
<td>18 (11.5)</td>
</tr>
<tr>
<td>Bicycle accident</td>
<td>3 (8)</td>
<td>8 (5.1)</td>
</tr>
<tr>
<td>Assault</td>
<td>2 (5)</td>
<td>7 (4.5)</td>
</tr>
<tr>
<td>Pedestrian</td>
<td>2 (5)</td>
<td>14 (8.9)</td>
</tr>
<tr>
<td>Others</td>
<td>1 (3)</td>
<td>1 (0.6)</td>
</tr>
<tr>
<td>Gunshot wound</td>
<td>0 (0)</td>
<td>3 (1.9)</td>
</tr>
<tr>
<td>Hit by objects</td>
<td>0 (0)</td>
<td>2 (1.3)</td>
</tr>
<tr>
<td>Injury Severity Score on admission, mean (SD)</td>
<td>28.2 (9)</td>
<td>29.1 (9.6)</td>
</tr>
<tr>
<td>Abbreviated Injury Scale, head</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 (no visible abnormality on computed tomography scan of head)</td>
<td>1 (3)</td>
<td>3 (1.9)</td>
</tr>
<tr>
<td>1 (minor injury)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>2 (moderate injury)</td>
<td>0 (0)</td>
<td>1 (0.6)</td>
</tr>
<tr>
<td>3 (severe but not life-threatening injury)</td>
<td>5 (13.2)</td>
<td>16 (10.2)</td>
</tr>
<tr>
<td>4 (life-threatening injury but survival likely)</td>
<td>17 (44.7)</td>
<td>81 (51.6)</td>
</tr>
<tr>
<td>5 (critical injury with uncertain survival)</td>
<td>15 (39.5)</td>
<td>56 (35.7)</td>
</tr>
<tr>
<td>Score on Extended Glasgow Outcome Scale at discharge</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 (dead)</td>
<td>6 (15.8)</td>
<td>21 (13.4)</td>
</tr>
<tr>
<td>2 (vegetative)</td>
<td>2 (5.3)</td>
<td>14 (8.9)</td>
</tr>
<tr>
<td>3 (lower severe disability)</td>
<td>28 (73.7)</td>
<td>115 (73.2)</td>
</tr>
<tr>
<td>4 (upper severe disability)</td>
<td>2 (5.3)</td>
<td>7 (4.5)</td>
</tr>
</tbody>
</table>

* Values are number (%) unless otherwise indicated. Percentages may not total 100 because of rounding. 
* Data were missing for 1 person in the CPP study for injury mode.

**Data Processing and Methods of Analysis**

**ICP Pulse Waveform Analysis.** MATLAB, version 6.5 (MathWorks Co, Natick, Massachusetts), was used to analyze the morphological characteristics of the ICP pulse waveform, including determining the amplitude of P1 and P2, calculating the P2:P1 ratio,
and visualizing the pattern of the ratio over time. One of the bioengineering collaborators (R.L.B.) wrote the program to convert the digital signal data acquired from the DATAQ DI-720 analog-to-digital converter to MATLAB format. Visual examination is used clinically to identify the components of the ICP pulse waveform, but visual examination was not an efficient and precise way to study the morphology of individual ICP pulse waveforms for this large data set. Therefore, an automatic detection algorithm was developed in collaboration with another bioengineering collaborator (P.V.). This automatic detection program was based on cerebral physiology, with the assumption that the ABP signal always preceded the ICP signal because the ABP waveform was the chief input signal to the cerebral system and the ICP pulse waveform was the output response to the ABP. The investigator (J.-Y.F.) and the program designer (P.V.) found the P1 and P2 of the ICP signal corresponding to the systolic and dicrotic notch of the ABP signal, respectively.

The automatic detection program was supplemented with manual visualization for patients with overdamped or missing arterial waveforms due to unstable hemodynamic status or technical issues such as bending or kinking of the catheter. Accuracy of data collected by using combined automatic and manual detection was verified by another CPP investigator (C.J.K.) by using 3 indices of agreement that were all within clinical and statistically acceptable ranges. Finally, an operational definition of DIICP was created so that instances and episodes could be linked with waveform characteristics for statistical analysis.

**Operational Definition of DIICP**

*Baseline.* Because the ICP signal varied, the ICP baseline was not stationary over time. In order to reflect this unsteady condition, a 10-minute moving average calculation was used to serve as the baseline reference.

*DIICP Episode.* The DIICP episode was defined as a response to internal or external stimuli that met the following criteria: (1) an increase in ICP greater than 10 mm Hg over baseline ICP for 5 minutes or longer if the ICP baseline is less than 20 mm Hg, or (2) an increase in ICP to a value greater than or equal to 25 mm Hg for 5 minutes or longer if the baseline ICP was greater than 20 mm Hg. The current ICP value was compared with the moving average from the preceding 10 minutes to detect DIICP episodes.

**DIICP Event.** A DIICP event was defined as a cluster of DIICP episodes separated by less than 4 minutes. If the interval between 2 episodes was 4 minutes or less, the second episode could not be counted as an independent DIICP episode. Instead the second episode was considered as part of a DIICP event. Once a DIICP event was discovered, the first data point of the 10-minute moving average was used for this specific entire DIICP event.

**Statistical Analysis**

The overall intention of the study was to determine if any specific patterns of ICP pulse waveform occurred on a beat-by-beat basis in a time series of 30 minutes before the DIICP event. The statistical software SPSS for Windows, version 12 (SPSS Inc, Chicago, Illinois), was used for data analysis, and the significance level was set at *P* < .05.

**Cluster Data Issue.** An issue in the analysis was the different numbers of DIICP events per patient; for example, 1 patient had 12 DIICP events, whereas another patient had only 1 event. Among the identified DIICP events, some events were highly intercorrelated because they occurred in the same patient; in other words, the events were not independent. From the statistical point of view, use of all identified DIICP events from small groups of subjects (1) violated the assumption of independent observations, (2) artificially inflated the sample size, and (3) preferentially favored finding significance in patients contributing more data through narrowed confidence intervals. Therefore, the analysis was limited to the first DIICP event in each patient with at least 30 minutes of data points preceding the event. If the first DIICP event occurred either at the beginning of the first hour or within the first 29 minutes, it was excluded because not enough data points had been collected. The next available DIICP event with at least 30 preceding minutes of data points was then used for analysis.

**Verifying the Pattern.** If a specific waveform pattern was associated with the DIICP event, the next step was to verify that this pattern was uniquely associated with and appeared only before the DIICP event. A 30-minute segment from the same patient with TBI that did not contain a DIICP event was selected for comparison. For example, in one patient, the first DIICP event emerged at the 40th minute of the second hour after the ICP monitor was deployed (DIICP group). For the same patient, a 30-minute seg-
ment before the 40th minute (matching time zone) but from a file without a DIICP event was selected as a comparison set.

**ICP Pulse Waveform Morphology Approach.** In order to examine the morphological characteristics of the ICP pulse waveform on a beat-by-beat basis 30 minutes before the DIICP event, the amplitude of P1 and P2 were compared and the P2:P1 ratio was calculated. An elevated P2 was defined as a P2:P1 of 0.8 or greater. The amplitude of each waveform component within a beat was measured, and then the P2:P1 ratio was calculated. The 1-minute P2:P1 ratios for both DIICP and comparison data sets were calculated. The analytic hypothesis was that P2 would be elevated before a DIICP event. A paired-sample t test was used to examine whether the mean 1-minute P2:P1 ratios differed significantly between the DIICP and comparison data sets.

**Results**

Data from 38 patients with TBI were used for this analysis. Seven patients with TBI did not have any episodes of DIICP during the first 48 hours after the ICP monitor was deployed and were therefore excluded from the statistical analysis.

**DIICP Events**

A total of 301 DIICP events were detected among 31 patients with TBI. The range of DIICP events in each subject was from 1 to 37; 16 of 31 patients (52%) had 6 events or fewer. Of the 301 DIICP events, 7 (2.3%) began immediately or within 15 minutes of the first hour after the ICP monitor was deployed (Figure 5); 13 (4.3%) began immediately or within 30 minutes after the ICP monitoring was resumed if the patients had undergone an urgent examination, procedure, or surgery during the treatment period. The DIICP events lasted from 5 to 439 minutes with a mean of 25.1 (SD, 41.7) minutes. A total of 81 episodes (26.9%) were less than 30 minutes long; the range was from 6 to 29 minutes and the mean duration was 15.3 (SD, 6.5) minutes. One patient was excluded because that patient had only 1 DIICP event, which occurred 14 minutes after the ICP monitor was deployed. A total of 30 patients with TBI were included in the final analysis model.

**Assessment of ICP Pulse Waveform Morphology**

The morphology of the ICP pulse waveform was examined to determine whether a unique pattern of P2 elevation (P2:P1 ratio, ≥0.8) was apparent 30 minutes before a DIICP event.

**P2 Elevation.** P2:P1 ratios for the 30 minutes before a DIICP event were examined for 8 subjects. The ratios were all greater than 0.85 in the 30 minutes preceding the DIICP event (see, eg, Figure 6). The P2:P1 ratios were almost equal to 1, and a plot showed a flat line. No sudden increase in P2 elevation occurred in the 30 minutes preceding the DIICP event. The ratios calculated for these first 8 subjects showed no discriminatory value, so no further calculations based on morphology were attempted. In summary, P2 elevation was not a distinctive predictor of DIICP events in this data set.

**Verifying Whether the P2 Elevation Was Present Only Before the DIICP Event**

A total of 60 (30 from DIICP data and 30 from comparison data) 1-minute segments of data on P2:P1 ratio were examined. All were greater than 0.8. Table 2 displays the characteristics of the differences in the P2:P1 ratio between the DIICP and comparison data sets. The DIICP data set had a significantly higher P2:P1 ratio than did the comparison data set ($t_{29} = 2.5$, $P = .02$). The difference in P2:P1 ratio between these 2 data sets ranged from -0.1 to 0.2 with a mean of 0.02 (SD, 0.05). All 30 pair differences in the P2:P1 ratio were less than 0.1 except for 2 pairs that differed by 0.1 and 0.2. Figure 7 shows the P2:P1 ratios for the DIICP and comparison data sets.

In summary, P2 was elevated at least 30 minutes before the DIICP event, but P2 elevation also appeared in the comparison data set, which did not have the DIICP event. Although the DIICP data set had a significantly higher P2:P1 ratio than did the comparison data set, the difference is not clinically important.

**P2 elevation is not a reliable clinical predictor of decreased intracranial adaptive capacity.**
Discussion

The purpose of this study was to determine whether ICP pulse waveforms show any unique pattern on a beat-by-beat basis in the 30 minutes preceding a DIICP event during the 48 hours after deployment of an ICP monitor, with emphasis on elevation of the P2 waveform component.

ICP Pulse Waveform Analysis

In terms of overall waveform amplitude, our findings are consistent with the results of many previous studies: a sustained increase in amplitude of the ICP pulse waveform occurs as ICP increases. However, when specific components of the ICP pulse waveform are examined, our finding of P2 elevation differs from that reported by Contant et al., who found that the P2 amplitude did not increase 10 to 60 minutes before transient episodes of intracranial hypertension. Contant et al provided no further information about changes in the shape of the ICP pulse waveform and the sampling time period, so elucidating where the differences may occur is difficult.

P2 elevation (P2:P1 ratio, ≥0.8). Because of the technical difficulty, few researchers have investigated the ICP morphology on a beat-by-beat basis in real time. Our finding that P2 was elevated is consistent with the results of Willis and of Mitchell et al., who reported a P2 elevation preceding the DIICP. Our finding is also consistent with the result reported by Willis that P2 elevation is not a clinically reliable factor for determining which patients have decreased intracranial adaptive capacity. We were disappointed to find that P2 elevation before DIICP was not a unique characteristic, even if P2:P1 ratios were significantly higher in the DIICP data set than in the comparison data set.

Similar to Willis, we found that P2 elevation could not be used to discriminate patients with TBI who had few DIICP events from patients with TBI who had many events. Although P2 elevation was significantly higher in patients with DIICP than in the comparison data set, this difference was statistically but not clinically significant, and the P2:P1 ratio was greater than 0.8 in both. The small difference (mean, 0.02; SD, 0.05) in the P2:P1 ratio between DIICP and non-DIICP data makes it unlikely that the choice of a ratio cutoff other than 0.8 would be any more useful for discriminating. Therefore, this particular ratio is not a useful discriminator and its clinical application is limited.

We recommend examining different measures of the ICP pulse waveform in future studies. For example, longitudinal beat-by-beat analysis of

Table 2

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracranial pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DIICP</td>
<td>20.5 (4.4)</td>
<td>11.2-30.0</td>
</tr>
<tr>
<td>Comparison</td>
<td>16.0 (3.2)</td>
<td>8.5-22.3</td>
</tr>
<tr>
<td>Difference (DIICP – comparison)</td>
<td>4.6 (3.3)</td>
<td>0.1-12.4</td>
</tr>
<tr>
<td>P2:P1 ratio</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DIICP</td>
<td>1.0 (0.1)</td>
<td>0.9-1.2</td>
</tr>
<tr>
<td>Comparison</td>
<td>1.0 (0.1)</td>
<td>0.8-1.1</td>
</tr>
<tr>
<td>Difference (DIICP – comparison)</td>
<td>0.02 (0.05)</td>
<td>-0.1-0.2</td>
</tr>
</tbody>
</table>

* P < .001.

b P < .05.
Waveform variability might directly reflect the complex intracranial adaptive capacity, because the ICP pulse waveform is the output product of the craniospinal system.

Conclusion

The purpose of our study was to investigate whether the ICP pulse waveform shows a pattern of P2 elevation on a beat-by-beat basis in the 30 minutes preceding the DIICP event, during the 48 hours after the an ICP monitor is deployed. The results indicated that the P2 elevation not only was present before the DIICP event but also appeared in the comparison data set. Accordingly, the P2:P1 ratio is not a reliable clinical indicator for predicting the development of DIICP events. Future studies are warranted to search for composite indicators that reflect intracranial adaptive capacity and incorporate other physiological parameters. Researchers could examine the use of those parameters at the bedside to determine which patients are at risk for sustained increase in ICP and examine the association of those parameters with neurological outcome.

ACKNOWLEDGMENTS

We thank Dr Martha J. Lentz for her guidance during the data analysis and her statistical assistance with this study.

FINANCIAL DISCLOSURES

The study from which the data were obtained was funded by grant R01 NR04901 from the National Institute of Nursing Research to Pamela H. Mitchell and Catherine Kirkness, principal investigators.

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