Intracranial Pressure Waveform Analysis: Clinical and Research Implications

Catherine J. Kirkness, Pamela H. Mitchell, Robert L. Burr, Karen S. March, David W. Newell

Abstract: *Assessment of intracranial adaptive capacity is vital in critically ill individuals with acute brain injury because there is the potential that nursing care activities and environmental stimuli to result in clinically significant increases in intracranial pressure (ICP) in a subset of individuals with decreased intracranial adaptive capacity. ICP waveform analysis provides information about intracranial dynamics that can help identify individuals who have decreased adaptive capacity and are at risk for increases in ICP and decreases in cerebral perfusion pressure, which may contribute to secondary brain injury and have a negative impact on neurologic outcome. The ability to identify high-risk individuals allows nurses to initiate interventions targeted at decreasing adaptive demand or increasing adaptive capacity in these individuals.*

Changes in the ICP waveform occur under various physiologic and pathophysiologic conditions and may provide valuable information about intracranial adaptive capacity. Simple visual assessment of the ICP waveform for increased amplitude and P2 elevation is clinically relevant and has been found to provide a rough indicator of decreased adaptive capacity. Advanced ICP waveform analysis techniques warrant further study as a means of dynamically assessing intracranial adaptive capacity.

Intracranial pressure (ICP) monitoring is common practice in the care of critically ill individuals with severe traumatic brain injury (TBI) and other conditions in which individuals have, or are at risk for, increased ICP, such as following aneurysmal subarachnoid hemorrhage. ICP monitoring is a vital component in the management of cerebral perfusion and the prevention of secondary brain injury. As well as providing a continuous measure of ICP, monitoring allows for analysis of the ICP waveform itself, which may provide valuable information about intracranial dynamics that can be used to guide nursing and medical management of individuals with acute brain injury.

This article presents an overview of the research on ICP waveform analysis and discusses its relevance for clinical nursing practice. Future areas for nursing research are also discussed. Research on the relationship between ICP and arterial blood pressure waveforms is not addressed in this article.

Background

ICP dynamics and the concepts of intracranial compliance and intracranial adaptive capacity have been reviewed extensively by others[2,3,20,29,31] and are presented only briefly here. Intracranial compliance reflects the ability of the intracranial system to compensate for increases in volume without subsequent increases in ICP. The term elastance may also be used and is the inverse of compliance. When compliance is decreased and elastance is increased, even small increases in intracranial volume result in large increases in ICP that may threaten cerebral perfusion and contribute to secondary brain injury. Decreased intracranial adaptive capacity is the nursing diagnostic category associated with decreased compliance and failure of the mechanisms that normally compensate for increases in intracranial volume.[30] Clinically, decreased intracranial adaptive capacity may be manifest by disproportionate, sustained increases in ICP in response to a variety of noxious and non-noxious stimuli.[31] Certain factors, such as previous episodes of sustained increases in ICP or high ICP, may be suggestive of decreased intracranial adaptive capacity, but currently there is no clinical means for reliably assessing adaptive capacity on an ongoing basis and predicting which individuals are at risk for adverse responses to nursing care and environmental stimuli.

ICP waveform analysis offers potential as a means of dynamic clinical assessment of intracranial adaptive capacity. The ICP waveform may provide information about intracranial dynamics reflecting compliance and/or cerebrovascular regulation that will help identify individuals at risk for adverse response to nursing care and environmental stimuli.

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The ICP Waveform

ICP waveform tracings can be obtained by using a variety of monitoring devices including intraventricular catheters and devices inserted into the subdural, epidural, or subarachnoid spaces or into the brain parenchyma. Although all methods produce waveforms that are visually similar, they may not be adequate for advanced ICP analysis techniques, such as spectral analysis. These techniques, which are described later, require that the transducers used for monitoring are responsive to an adequate range of frequencies, with a frequency bandwidth of at least 20 Hz.[19] Fiberoptic transducer tipped systems generally meet or exceed this criterion. Systems using fluid transmission of the pressure to an external transducer do not provide the accuracy needed for spectral analysis.[19]

Normal individual ICP pulse waveforms (Fig 1) generally have three characteristic peaks of decreasing height that correlate with the arterial pulse waveform and are referred to as P1 (percussion wave), P2 (tidal wave), and P3 (dicrotic wave).[11] P1 generally has a sharp peak and a fairly constant amplitude. P2 is more variable and ends on the dicrotic notch. P3 follows the dicrotic notch. There may be additional smaller peaks following the three main peaks, but these vary by individual and may not always be present. In individuals who have had a cranial bone flap removed, the ICP waveform is dampened and lower in amplitude.

In addition to the individual ICP waveform pattern that occurs with each cardiac cycle, there is also a slower sinusoidal pattern of consecutive ICP waves that is related to the respiratory cycle (Fig 2).[21,27]

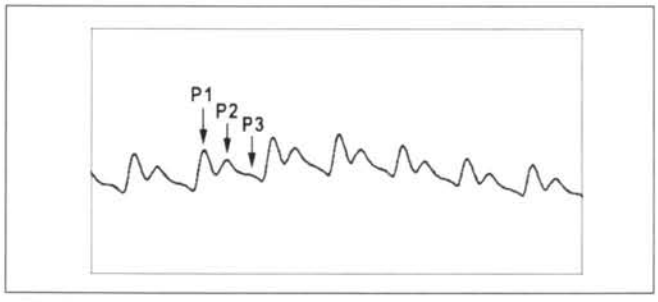

Fig 1. *Normal ICP waveform*

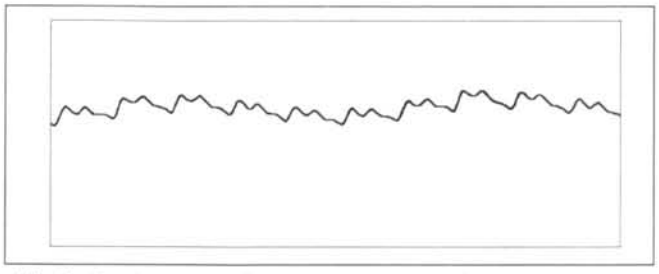

Fig 2. *Repiratory influence on ICP waveform*

Knowledge of the mechanisms of the ICP waveform can contribute to understanding the clinical application of ICP waveform analysis. The shape of the ICP waveform is determined by the complex interaction of the arterial input, intracranial contents, and venous outflow. In general, under normal physiologic conditions, the origin of the ICP waveform is thought to be primarily arterial, with retrograde venous pulsation contributing to later components.[1,8,10-12,14,17,18,21,23,25,34] Some researchers further specify that P1 and P2 are arterial in origin, and P3 is of venous origin.[14,23] Under different physiologic and pathophysiologic conditions there is greater or lesser contribution of arterial and venous input, therefore different components of the waveform are predominant. The exact mechanism of transmission of the arterial pulsation to the ICP waveform is uncertain, but transmission may occur from the intracerebral blood vessels to the brain parenchyma and cerebrospinal fluid (CSF) and/or via the choroid plexus to the CSF and brain parenchyma[6,10,25,36,42]

Changes in the ICP waveform amplitude and configuration have been attributed to both changes in intracranial compliance[4,7,11,20] and changes in cerebral blood flow regulatory mechanisms.[7,16,35,37] Both decreased intracranial compliance and impaired cerebral blood flow autoregulation contribute to decreased intracranial adaptive capacity.

ICP Changes Under Different Physiologic Conditions

Human and animal studies have examined ICP waveform changes under different physiologic and pathophysiologic conditions (Table 1). Studies that did not differentiate waveform components (P1, P2) reported findings in relation to overall waveform amplitude.

The magnitude of the ICP waveform is affected both by the state of the intracranial components and by mean ICP. As mean ICP increases, for example, from a rapidly expanding mass lesion, the amplitude of the waveform also increases.[5,14,15,27,33] Initially all components of the waveform increase, so individual peaks remain visible (Fig 3). As ICP continues to increase, P2 increases to a greater extent than P1, the peaks disappear, and P1 may become buried.

Withdrawal of CSF and head elevation result in a decrease in mean ICP and ICP waveform amplitude but little change in the waveform configuration.[11] Increased ICP from increased CSF volume results in an increase in the ICP waveform amplitude, also with little change in the configuration.[10,11,17,21,42] Extreme arterial hypotension and hypertension produce changes in the ICP waveform, particularly P1. With severe arterial hypotension, there is a decrease in mean ICP and ICP waveform amplitude, especially P1.[17,21] The opposite changes occur with severe hypertension.

Conditions that result in cerebral vasoconstriction or vasodilation also result in changes in the ICP waveform. Cerebral vasospasm results in little change in mean ICP but a considerable decrease in the amplitude of all the ICP waveform components.[10,24] Severe hypercapnia or hypoxia produce an increase in ICP waveform amplitude and rounding of the waveform as ICP increases.[34] These changes may result from a decrease in cerebrovascular resistance and cerebral arteriolar vasodilation that lead to increased transfer of the arterial pulse to the ICP waveform. Hyperventilation decreases mean ICP and the amplitude of P2 and to a lesser extent P3, with P1 remaining relatively unchanged.[11] Breath holding results in significant P2 elevation as mean ICP increases.

Table 1. ICP Changes Related to Differing Physiologic Conditions

Condition	ICP Changes
Rapidly expanding mass lesion	Increase mean ICP Increase ICP waveform amplitude
Increase/decrease CSF volume	Increase/decrease mean ICP Increase/decrease ICP waveform amplitude Little change in ICP waveform configuration
Severe arterial hypotension	Decrease mean ICP Decrease ICP waveform amplitude, especially P1
Severe arterial hypertension	Increase mean ICP Increase ICP waveform amplitude
Severe hypercapnia and hypoxia	Increase mean ICP Increase ICP waveform amplitude Rounding of ICP waveform due to increase in later waveform components
Hyperventilation	Decrease mean ICP Decrease ICP waveform amplitude P2, and to a lesser degree P3 with little change in P1
Jugular vein compression	Increase mean ICP Increase ICP waveform amplitude, mainly P2 and P3

Changes in the cerebral venous system also result in ICP waveform changes. Acute bilateral jugular vein compression that restricts intracranial venous output results in increased mean ICP and increased waveform amplitude. This occurs primarily in relation to P2 and P3, although some report a lesser increase in the amplitude of P1 as well.[9,22,27,40] The increase in the P2 and P3 components of the ICP waveform may result from retrograde transmission of venous pressures to the CSF.

Spectral analysis of the ICP waveform has been explored as a technique of assessing intracranial adaptive capacity. The ICP waveform typically displayed on bedside monitors represents the waveform over time. As a prism breaks light into its constituent pure colors, spectral analysis using fast Fourier transform (FFT) represents the temporal waveform in terms of constituent component frequencies (Fig 4). The waveform is broken down into multiple sinusoidal waves over a range of frequencies or periods, each with unique amplitude and phase components.[26] The power density spectrum is a graphic representation of the relative magnitude of the variability at the different frequencies. Power, on the *y* axis, is often plotted on a logarithmic scale in decibels (dB) and frequency, on the *x* axis, in cycles per second, or Hertz (Hz). Rapidly changing rhythm patterns are associated with peaks to the right of the spectrum, while slowly changing patterns correspond to peaks to the left. The height of a peak is an index of the relative magnitude of an additive rhythm component at that frequency.

The normal ICP waveform spectrum primarily comprises low-frequency components that reflect cerebral blood flow.[13] As intracranial adaptive capacity decreases, there is an increase in high-frequency components of the spectrum.[28] The ICP high-frequency centroid (HFC) measure is the power weighted mean frequency in the range from 4 to 15 Hz.[39] Shifts in this range indicate changes in

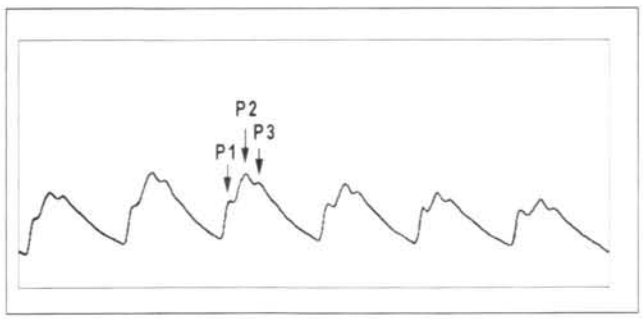

Fig 3. *Abnormal ICP waveform*

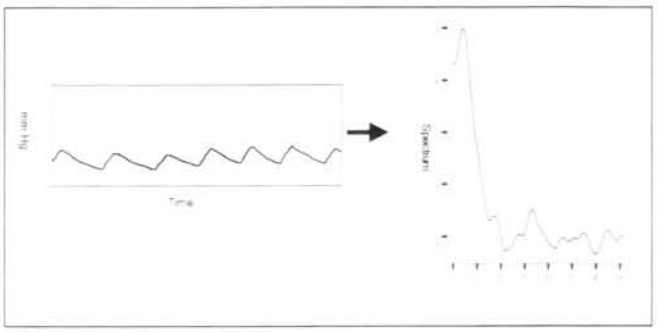

Fig 4. *ICP waveform and power density spectrum*

the relative contributions of the frequencies to the ICP waveform, reflecting changes in the physical characteristics of the brain. Although the HFC is inversely correlated with the pressure volume index, its prognostic ability and usefulness in altering clinical outcome have not been demonstrated.

Nursing Research

Nursing research on ICP waveform analysis has examined the value of various measures derived from the ICP waveform in predicting risk for disproportionate increases in ICP (DIICP). Some studies also have looked at the association between waveform measures and neurologic outcome.

Willis[41] examined the predictive value of P2 elevation in identifying individuals with decreased adaptive capacity, as reflected by episodes of DIICP. Fifteen participants with intracranial pathology were studied. The amplitudes of P1 and P2 were measured and the ratio of P2:P1 was calculated. P2 elevation was defined as a P2:P1 ratio of greater than or equal to 0.8. ICP trend data were obtained for 24 hours and evaluated for DIICP, defined as five occurrences of either ICP increases greater than 10 mm Hg over baseline for at least 5 minutes, or increases that triggered medical protocols.

In this study, P2 elevation and ICP greater than 10 mm Hg indicated decreased adaptive capacity. Odds ratios demonstrated that subjects with P2 elevation were 9 times more likely to develop DIICP than those without P2 elevation. Participants with a baseline ICP greater than 10 mm Hg were 21 times more likely to develop DIICP than those with a baseline ICP less than 10 mm Hg. P2 elevation was present in the majority of participants. No participants without P2 elevation had DIICP. Although all participants who had DIICP were correctly predicted, 50% of those predicted to have DIICP did not; thus, this method did not discriminate well between those with P2 elevation who did and did not develop DIICP.

Rauch, Mitchell, and Tyler[38] performed a secondary analysis of 30 ICP recordings from eight children to assess the predictive validity of two risk factors (wide ICP waveform amplitude and increased ICP) for DIICP. Wide ICP waveform amplitude was defined as an 8 mm Hg or wider peak-to-trough excursion, lasting for 10 minutes. ICP waveform component amplitudes were not individually determined. Increased ICP was defined as a baseline resting ICP greater than or equal to 15 mm Hg for more than 5 minutes. Data were not available to calculate CPP. DIICP was defined as a stimulus-induced increase in ICP greater than or equal to twice the resting ICP, occurring within 1 minute of the stimulus and lasting at least 3 minutes. Participants had a variety of intracranial diagnoses and ranged in age from 7 months to 9 years. Observations of ICP response to nursing care were made over 4 hours.

Wide ICP waveform amplitude was present in 7 of 30 (23%) sessions, and increased ICP at rest was present in 18 of 30 (60%) sessions. Suctioning was carried out in all sessions and turning in 17 sessions. DIICP occurred in 73% of suctioning sessions and with 23% of turns. The sensitivity of wide ICP waveform amplitude in predicting DIICP was low (32% for suctioning and 28% for turns). All wide ICP waveform amplitude occurrences were in sessions with DIICP, but 65% of occurrences of DIICP with suctioning and 33% with turning were not associated with wide ICP waveform amplitude. The sensitivity of increased resting ICP in predicting DIICP was 54% with suctioning and 28% with turning.

In this study wide ICP waveform amplitude was not sensitive in identifying those who would have DIICP. Resting ICP level was also not a reliable predictor of DIICP. The definition of DIICP used allowed smaller absolute increases to be considered DIICP at lower mean ICP levels than at higher levels. Defining DIICP as an increase in ICP of an absolute value, for example, 10 mm Hg, would better standardize the measure.

The sample sizes in the previous two studies are small and do not represent the full range of intracranial diagnoses and severity of conditions of individuals with intracranial pathology who may be at risk for DIICP. This restricts the strength of the findings and their generalizability beyond the populations studied.

Mitchell et al.,[32] as part of a larger study, examined the use of ICP waveform analysis to predict DIICP in 37 critically ill adults with various intracranial diagnoses, including cerebrovascular pathology, traumatic brain injury, and tumor. Glasgow Coma Scale scores ranged from 3 to 15. Continuous ICP data from intraparenchymal fiberoptic monitoring devices were recorded for 1 hour and waveform analysis was carried out. Episodes of DIICP were counted over the following 24 hours. DIICP eipsodes were defined as increases greater than 10 mm Hg above baseline for more than 5 minutes or ICP increases sufficient to trigger ventriculostomy drainage or initiation of medical protocols. Waveform analysis included visual assessment for P2 elevation, defined as P2 at or exceeding P1, and spectral analysis using FFT. Factors derived from spectral analysis included low frequency power (0 to 4 Hz), high-frequency power (4 to 15 Hz), and the high-frequency centroid. Principal component analysis using the Karhunen-Loeve expansion was also carried out and allowed identification of waveform elements representing level, slope, and P1 to P3 amplitude. Outcome at discharge was categorized using the Glasgow Outcome Scale.

P2 elevation was predictive of increased frequency of DIICP. Odds ratios demonstrated that those with P2 elevation were 7.2 times more likely to have more than five episodes of DIICP and 11 times more likely to have more

than 15 episodes of DIICP, with high sensitivity (99%) for both. When P2 elevation was present, the likelihood of having DIICP was 84% for more than 5 episodes and 75% for more than 15 episodes, with an overall accuracy of 86% and 76%, respectively. However, specificity (absence of DIICP in the absence of P2 elevation) was low (less than 20%), in part related to the small number of patients with abnormal waveforms and low frequency of DIICP. DIICP did not occur in patients with a normal ICP waveform, but the presence of P2 elevation did not distinguish those with fewer episodes of DIICP from those with many.

A moderate correlation of less frequent episodes of DIICP and poorer outcome was found rather than the expected correlation of more episodes of DIICP and poorer outcome. Mean ICP was higher in participants with greater frequency of DIICP, but 44% of participants with a normal baseline ICP had DIICP.

These researchers had proposed that DIICP would reflect episodes of increased ICP and potential decreased CPP that could contribute to secondary brain injury and poorer outcome. The results suggest a more complex relationship with other intervening factors. Patients with fewer than 5 episodes of DIICP but an elevated P2 had significantly lower mean ICP, higher mean of the low frequency spectra of the ICP waveform, and a larger HFC. Individual components from the spectral analyses alone were not strong enough to predict DIICP. Incorporating a number of components into a multiple regression model that included age, mean ICP, and an index of autoregulation did provide predictive information in relation to DIICP and outcome. The complexity of performing the analysis and of the multiple regression model in real time precludes its immediate application in bedside monitoring.

Relevance for Clinical Nursing Practice

A primary goal of the nursing management of individuals with severe brain injury in the critical care phase is to prevent or minimize the occurrence of increased ICP and decreased CPP that may result in secondary brain injury and poorer neurologic outcome. Nursing care activities and environmental stimuli have the potential to result in clinically significant increases in ICP in a subset of individuals with acute brain injury who have decreased intracranial adaptive capacity. The goals then become (a) to identify risk factors for early identification of individuals with decreased adaptive capacity and (b) to understand the mechanisms, so that specific nursing interventions for preventing or minimizing care or stimuli-induced increases can be developed and tested.

Nursing interventions can be both interdependent measures designed to increase adaptive capacity, such as administration of mannitol or hypnotic drugs, and independent measures designed to decrease adaptive demand, such as therapeutic use of body position or control of environmental stimuli.

Mean ICP alone does not predict whether an individual will have DIICP. It is important to remember that low mean ICP does not rule out decreased adaptive capacity. Simple ongoing visual assessment of the ICP waveform for increased amplitude, elevated P2, and rounding of the waveform can provide at least nonspecific information suggesting decreased intracranial adaptive capacity and altered intracranial dynamics. P2 elevation is a rough estimate of risk for DIICP but is not specific enough to distinguish between high- and low-risk patients. DIICP episodes have also been shown to occur without P2 elevation. Prior recent episodes of DIICP are suggestive of decreased adaptive capacity and risk for further DIICP.

Nurses must be aware of care-related activities and environmental stimuli that may result in increased ICP and decreased CPP in individuals with acute brain injury. Individuals should be monitored for changes in the ICP waveform occurring in relation to care activities or environmental stimuli. Care must be individualized based on ongoing assessment of adaptive capacity and response to therapeutic interventions. Close monitoring is particularly important during events that pose greater risk for alterations in ICP, CPP, cardiovascular, or respiratory parameters, such as with noxious stimuli or during patient transfers and diagnostic testing. If a patient is identified as being at risk for, or has already demonstrated increases in ICP in response to nursing care or environmental stimuli, this information must be communicated to all those caring for the patient. The particular events that result in ICP increases in that individual, interventions to prevent or minimize the increases, and the response to these interventions should also be communicated.

Implications for Future Nursing Research

The usefulness and clinical applicability of advanced ICP waveform analysis techniques require further study but have the potential to be of value in predicting individuals with acute brain injury who have decreased adaptive capacity and are at risk for the deleterious effects of increased ICP and decreased CPP. Of particular interest for further study are measures of the transfer of the ambulatory blood pressure (ABP) waveform to the ICP waveform, which may reflect cerebral autoregulation. Adaptive capacity is a dynamic state, and the development of continuous assessment techniques that provide immediate feedback on patient response to nursing care and environmental stimuli will be important. ICP waveform analysis is potentially well suited to this.

Nursing care activities and environmental stimuli that may increase ICP have been identified, but further research is necessary to understand the variability in ICP response of individuals and to further develop predictors of those with decreased adaptive capacity

who will respond adversely. Much of the previous nursing research on ICP response to care and environmental stimuli has reported only group results. Future study designs should incorporate time series analysis techniques that allow examination of individual responses over time. Further study to examine within individual variability in ICP and cerebrovascular parameters and the potential link of variability and adaptive capacity would be of value. Complexity and nonlinear analysis techniques, such as approximate entropy, which assess regularity and predictability of a time series, offer exciting opportunities for examining these issues.

In addition to further development and testing of nursing interventions to prevent or minimize ICP increases that may threaten CPP, interventions that may decrease ICP, such as touch, need to be explored. The effect of nursing interventions and environmental stimuli on other indicators of cerebral perfusion and oxygenation, such as jugular venous oxygen saturation, also requires further study.

The impact of short-term increases in ICP and decreases in CPP on outcome is not clear. It would be of interest to further examine whether the effect of these occurrences is cumulative and whether repeated perturbations of the intracranial system influence the ability to maintain adaptive capacity, particularly in critically ill individuals with an already unstable cerebrovascular system. Even beyond preventing increases in ICP and decreases in CPP outside normal limits, it may be significant to promote stability of these measures. How stability should be defined and how it can best be achieved remains to be determined.

Summary

Ongoing assessment of intracranial adaptive capacity is critical in the nursing management of individuals with acute brain injury. Nursing care activities and environmental stimuli that challenge intracranial adaptive capacity have already been identified through nursing research. The ability to better predict which individuals with decreased intracranial adaptive capacity will respond adversely to these events will allow the design and testing of nursing interventions for minimizing or preventing care- or environmental stimuli-induced increases in ICP and decreases in CPP that may ultimately affect neurologic outcome.

Visual assessment of the ICP waveform for increased amplitude and P2 elevation provides a rough indicator of decreased adaptive capacity. Advanced methods of ICP waveform analysis that examine the transfer between the ABP and ICP waveforms require further development but may provide a clinically important means of dynamically assessing adaptive capacity and obtaining immediate feedback on response to care that can be used to guide nursing practice.

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