Noninvasive intracranial compliance monitoring

Technical note and clinical results

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Although invasive measurement of intracranial pressure (ICP) involving high-resolution waveform analysis allows assessment of intracranial compliance (ICC), it is only feasible in a few selected neurosurgical conditions. Intracranial compliance can be assessed using the high-frequency centroid (HFC), which is the power-weighted mean frequency within the 4 to 15–Hz band of the ICP waveform. The authors have systematically tested the utility, performance, and reliability of a noninvasive monitor of ICC. The underlying principle of this device is that the ICP transmission and its infrasonic waves are transmitted through the inner ear toward the tympanic membrane. If the outer ear is sealed in an airtight fashion, motions of the tympanic membrane cause air pressure fluctuations that can be recorded using a special sensor.

The authors compared the HFC calculated from an intraparenchymal ICP sensor with that obtained simultaneously from an ipsilaterally placed noninvasive device during half of a respiratory cycle (peak to baseline) as well as for three random samples of three heart cycles. They analyzed 32 sessions in 13 patients in whom mechanical ventilation had been established. In four (11%) of 36 sessions they could not demonstrate an adequate signal.

For the peak-to-baseline cycle, the mean invasively recorded HFC was 8.05 ± 0.55 Hz (range 6.7–9 Hz) whereas the mean noninvasively recorded HFC was 8.04 ± 0.49 Hz (range 7.9–9.3 Hz). The ICP was 8.5 ± 5 mm Hg (range 2–24 mm Hg). For the three heart cycles randomly sampled, the values were 7.73 ± 0.51 Hz (range 6.7–8.6 Hz) and 7.76 ± 0.56 mm Hg (range 6.5–8.8 mm Hg), respectively.

This device allows noninvasive assessment of ICC based on the HFC waveform analysis that is equivalent to that obtained by invasive intraparenchymal recording. The monitoring device may become a valuable tool for monitoring parameters in patients in whom placement of an intracranial sensor is not feasible but assessment of ICC as an alternative to ICP measurement is desired.

**KEY WORDS** • intracranial compliance • intracranial pressure monitoring • waveform analysis

**Abbreviations used in this paper: CSF = cerebrospinal fluid; HFC = high-frequency centroid; ICC = intracranial compliance; ICP = intracranial pressure; PVI = pressure–volume index.**

The ICC curve represents the pressure–volume relationship of the brain based on the principles of the so-called Monro–Kellie doctrine. It is an indicator of the brain’s ability to tolerate or compensate for volume increases or its volume-buffering capacity. Traditionally, this curve has been derived from the volume–pressure response, or the PVI, in which a certain fluid volume is manually injected into the ventricles and the change in ICP is recorded. More recently, an automated pneumatic compliance measurement device integrated into an external ventricular drain has been devised for continuous ICC assessment.

The ICP waveform undergoes certain distinctive changes as ICP increases and ICC deteriorates. One means of grading these changes is to calculate the HFC, which is the power-weighted mean frequency within the 4 to 15–Hz frequency band of the ICP waveform. Robertson, et al. have stated the following: “Continuous monitoring of intracranial compliance by computerized analysis of the ICP waveform may provide an earlier warning of neurological decompensation than ICP per se and, unlike PVI, does not require volumetric manipulation of intracranial volume.”

In this study we report the principles, application, and first validation studies of a noninvasive device for ICC assessment based on HFC analysis of the ICP waveform. The underlying principle of this device is that the ICP and its infrasonic waves are transmitted from the subarachnoid space through the inner ear to the tympanic membrane where they can be detected using a special high-resolution transducer if the outer ear is sealed in an airtight fashion.

**Clinical Material and Methods**

**Measurement Principles**

Intracranial pressure fluctuations during the cardiac cycle cause infrasonic pressure waves that are below the hearing threshold, generally less than 10 Hz. They are propagated via noncompressible fluid pathways from the subarachnoid space to the perilymphatic duct (aqueductus cochleae), which serves as the primary conduit between the subarach-
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This pathway connects to the inner ear where the pressure waves eventually cause motion of the oval window and the ossicles, leading to motion of the tympanic membrane. If the outer ear is sealed in an air-tight fashion these motions of the tympanic membrane cause air-pressure fluctuations (Fig. 1) that can be recorded using a special miniature sensor, developed by Paulat and coworkers.

The device’s main unit resembles a normal desktop computer, and it is connected to a standard monitor for waveform display. It has data storage capability, and its software allows simultaneous display of other monitored parameters such as electrocardiography values and ICP. The special miniature transducer in the outer ear channel is connected to the main unit with a cable, similar to an ICP monitor.

In keeping with reports on the tympanic membrane displacement tests, the motion of the tympanic membrane can either be inward or outward depending on the motion of the stapes footplate, which is determined by the ICP. We have noted an inverse ICP tracing, mainly in patients with low ICP, which is due to this mechanism. This is, however, irrelevant for our amplitude calculations.

Monitoring Protocol

We compared HFC values simultaneously calculated from ICP waves recorded using an intraparenchymal fiber-optic ICP sensor (Camino V 420; Camino Laboratories, San Diego, CA) and the ipsilaterally placed noninvasive device. The noninvasive device was used to record measurements during half of a respiratory cycle (peak to baseline) to correct for ICP variations and waveform changes during the respiratory cycle (Period A). Additionally three samples of three cardiac cycles were randomly chosen from 10 to 15-minute recording sessions (Period B). Figure 2 shows examples of ICP waves recorded invasively and noninvasively.

To examine whether there was a correlation between HFC and ICP we compared both parameters for Periods A and B for all recordings and for each patient’s first recording (Table 1).

Patient Population

Thirty-six recording sessions in 15 patients were undertaken. Aneurysmal subarachnoid hemorrhage was present in eight patients, severe closed head injury in six, and spontaneous hypertensive intracerebral hemorrhage in one. Their mean age was 54 ± 11 years. One patient underwent one, three patients two, seven patients three, and one patient underwent four recording sessions. Correct intraparenchymal placements of all ICP probes were confirmed using computed tomography scanning. In all patients sedation and mechanical ventilation were initiated, and they were treated while in a supine 30° head-up position. No external manipulation occurred and all ventilation and infusion parameters were kept constant during the recording sessions. The need for informed consent was waived because it was an entirely noninvasive study.

![FIG. 1. The pressure transmitting window. Anatomical drawing of the structures involved in signal transmission from the CSF spaces to the outer ear.](image1)

![FIG. 2. Two examples of simultaneously obtained noninvasive and invasive ICP traces. Note the time lag of the noninvasive trace.](image2)
Obtaining the ICP Wave Noninvasively

Although several techniques have been tried, noninvasive ICP monitoring providing a continuous or numeric ICP display applicable for routine clinical use remains impossible today. It is noteworthy that some noninvasive techniques will generate a wave that more or less resembles the ICP wave obtained from direct, invasive measurements. In one such technique the dielectrical properties of the skull are used, and, in combination with cardiac and respiratory cycle analysis, waves very similar to a simultaneous invasive ICP tracing were generated. In a recent report the authors have described the use of magnetic resonance imaging for obtaining ICP values by examining CSF and cerebral blood flow volumetric flow rates; they found similarities between the magnetic resonance imaging–derived CSF pressure gradient waveforms and their corresponding invasive ICP traces.

It is also possible to calculate ICP curves noninvasively based on transcranial Doppler ultrasonography and invasively recorded arterial blood pressure curves. A formal comparison of the noninvasively obtained ICP waves and those recorded invasively was, however, not performed in any of the aforementioned studies.


table 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± Standard Deviation</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Period A (half respiratory cycle)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All recordings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Invasive ICP</td>
<td>−0.39</td>
<td></td>
</tr>
<tr>
<td>Noninvasive</td>
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<td></td>
</tr>
<tr>
<td>1st recording</td>
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<td></td>
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<tr>
<td>Invasive ICP</td>
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<td></td>
</tr>
<tr>
<td>Noninvasive</td>
<td>−0.21</td>
<td></td>
</tr>
<tr>
<td>Period B (3 random samples)</td>
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<td></td>
</tr>
<tr>
<td>All 32 recordings</td>
<td>−0.48</td>
<td></td>
</tr>
<tr>
<td>Noninvasive</td>
<td>0.08</td>
<td></td>
</tr>
<tr>
<td>1st recording</td>
<td>−0.04</td>
<td></td>
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<tr>
<td>Invasive ICP</td>
<td></td>
<td></td>
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<tr>
<td>Noninvasive</td>
<td>−0.26</td>
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</table>

* Recording obtained in 13 cases.

Results

In two of 15 patients an adequate signal was not observed and further recording was ceased. In another two patients a sufficient signal was obtained during the first session but no signal was obtained in the next session. Overall a sufficient signal was obtained in 32 (89%) of 36 sessions.

For Period A (half of a respiratory cycle [peak to baseline]) the mean HFC ± standard deviation calculated from the invasive recording was 8.05 ± 0.55 Hz (range 6.7–9 Hz) compared with 8.04 ± 0.49 Hz (range 7–9.3 Hz) recorded noninvasively. The ICP was 8.5 ± 5 mm Hg (range 2–24 mm Hg) (Table 2).

For Period B (three random samples of three heart cycles) the HFC was 7.73 ± 0.51 Hz (range 6.7–8.6 Hz) compared with 7.76 ± 0.56 Hz (range 6.5–9.0 Hz), respectively (Table 2).

We did not find any significant negative or positive correlation between HFC and ICP (Table 1).

Discussion

The novelty of this device is its ability to capture the ICP wave noninvasively, which is the prerequisite for ICC assessment based on HFC analysis of the ICP waveform. Until now this has only been possible by using direct, invasive epidural, intraparenchymal or intraventricular recording, which requires burr hole or twist drill access to the brain.

The idea of using ICP waveform–related information to assess ICC is appealing, and this strategy was first formally assessed in a series of 55 patients with severe head injury. It becomes even more appealing when the noninvasive recording of the ICP pulsewaves becomes possible. In their study Robertson, et al., reported that an HFC of 6.5 to 7 Hz was normal and that an increase to 9 Hz coincided with a PVI reduction; the mean HFC values in their series ranged from 6.8 to 9 Hz, which is nearly identical to our range of 6.7 to 9.3 Hz, suggesting similar recording conditions and ICP waveforms. They reported associations between the length of time that the HFC was greater than 9 Hz and an increased mortality rate and between a rapid rise in HFC and deterioration likely to be caused by an intracranial hematoma.

Because of our design and patient selection, we cannot comment on such findings in our study, but they appear to be a logical topic for further comparative studies.

Intracranial Pressure and Intracranial Compliance

The exact relationship between ICP and ICC calculated from volume-pressure challenges remains unclear. Using the automated Spiegelberg method in an experimental animal model, Yau, et al., demonstrated an asymptotic relationship between ICP and ICC calculated from ventricular

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measurements at ICPs between 5 and 50 mm Hg. In an analysis in which the manual fluid bolus injection method was compared with the pneumatic method, they confirmed a statistically significant correlation. The relationship between ICP and intraparenchymal compliance was complex and biphasic. At ICPs as high as 20 mm Hg, they demonstrated a rather linear negative relationship, whereas at higher ICPs they found that the fitted curve ascended; no statistical analysis was provided, however, presumably because there were only a few measurements.

There has been no formal clinical analysis of the relationship between ICP and the HFC-derived ICC to date. In the previously discussed study by Robertson, et al.,25 this issue was not addressed, yet they stressed that deterioration of ICC can precede by several hours a rise in ICP. Our analysis of the relationship between ICP and HFC showed no correlation between these two parameters, probably because ICP was within normal limits in our patients and none experienced a delayed rise.

In future studies it would be helpful to examine the relationship of HFC and ICC for better prediction of plateau waves and decreased cerebral perfusion pressure in animal experiments, although it is uncertain whether the unique human anatomy makes it possible to replicate our findings in animal research.

Anatomical and Technical Considerations

We were able to obtain a signal in almost 90% of the recordings. We were unable to determine why a signal could not be recorded in two young patients, one with a severe head injury and the other with aneurysmal subarachnoid hemorrhage, nor could we explain why only initial recordings were obtained in two patients. None of these patients suffered from mastoid bone fractures or inner-ear injury, which may be a cause for failure of signal transmission.

Transmission from the intracranial CSF spaces to the middle ear depends on the patency of the cochlear aqueduct,4 which is also a prerequisite for the tympanic membrane displacement test for ICP assessment.13,28 There is evidence reflecting the biological process of aging in the organism;33 in another investigation the yield of tympanic membrane findings were not confirmed by investigators in 101 human cadaveric temporal bones, who concluded that “there was no correlation between age and narrowest diameter, or between age and category of patency.”9 It is possible that the cochlear aqueduct was aplastic in two of our patients and that secondary changes such as hemorrhage or other obstruction caused a signal loss in the second recording in our other two cases.

A short delay between artificial CSF infusion into the subarachnoid space and the increase in intralabyrinthine pressure has been reported by authors of an animal study.2 This delay is apparent when comparing the invasive and noninvasive ICP traces in Fig. 2.

One must also consider two different signal transmission pathways from the inner ear to the tympanic membrane. Both pathways contribute to the composition of the recorded signal in the sealed outer ear. The first originates in the round window, any motion of which causes very small pressure changes in the middle ear and movement of the tympanic membrane, because the eustachian tube is normally collapsed and the middle-ear chamber sealed from external pressures. The second pathway involves direct mechanical transmission via the oval window and the ossicles of the inner ear, as previously noted.

Authors of preliminary studies have shown that mainly the low frequencies (< 1 Hz) are transmitted via the first pathway with approximately eightfold signal attenuation, whereas in the second pathway both low and high frequencies are transmitted with approximately 100-fold signal attenuation.20 In this study we recorded only signals transmitted through the second pathway, by separating high- and low-frequency signals with a high-pass filter. Anatomical conditions, such as the individual configuration of the cochlear aqueduct, perilymph composition, and ossification of malleus, incus, and stapes, may influence signal transmission and its amplification or attenuation. Although it appears that using a high-pass filter is appropriate, the contribution of the two pathways to the final composite signal is largely unknown. Further investigations are needed for a better understanding of the origin of the signal and to optimize signal detection and recording. It would also be helpful to study patients in whom the eardrums are not intact or in whom temporal bone fractures have occurred, especially because ICC is helpful in trauma.

Critical Appraisal and Prospects

We have not yet attempted to replicate results obtained from previous HFC analysis of the ICP waveform25 nor to assess further the clinical utility of this technique. This is partly because in our series the ICP was mostly within acceptable limits; one could not justify elevating ICP solely for the purpose of this study.

We wish to stress that because ICC assessment derived from HFC-based ICP waveform represents a mathematical approach to using information contained in the ICP waveform, it cannot at this time replace the traditional volume-pressure intraparenchymal access for accurate ICC measurement. Further comparative studies are needed to investigate these relationships.

Conclusions

High-frequency centroid–based ICP waveform analysis, performed using our device, has potentially valuable clinical application because it allows noninvasive ICC assessment equivalent to that obtained by invasive intraparenchymal recording. It may become a valuable tool as an alternative to ICP measurement— and/or ventricular access—based ICC calculation in patients in whom placement of an intracranial sensor is not feasible but ICC assessment is desired.

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Disclosure
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