Noninvasive Detection of Elevated Intracranial Pressure Using Spectral Analysis of Tympanic Membrane Pulsation Signals

R. Dhar, R. H. Sandler, K. Manwaring and H. A. Mansy

Biomedical Acoustics Research Laboratory, University of Central Florida, Orlando, Florida, USA
{rajkumar.dhar@knights., hansen.mansy@}ucf.edu, {rhsandler, manwaringmd}@gmail.com

Neurological conditions such as hydrocephalus, traumatic brain injury, intracranial hemorrhage, and meningitis can lead to intracranial pressure (ICP) elevation, which is associated with serious complications, including severe headaches, blurred vision, problems with moving or talking, seizures, and even death [1]. Due to these potential risks, prompt medical attention and reliable monitoring may be needed for patients with elevated ICP. Current diagnosis relies on invasive pressure measurement. On the other hand, expensive imaging techniques like computed tomography and magnetic resonance imaging can also be used; however, they are not as reliable as invasive methods [2]. Unfortunately, the invasive methods pose the risk of infections and also require clinical expertise to perform. Important noninvasive methods to measure ICP that have been studied include observation of optic nerve sheath diameter variation, transcranial Doppler ultrasonography, and tympanic membrane displacement [3]. This study proposes a new noninvasive and easy-to-use method for the detection of elevated ICP by leveraging an apparent hydrostatic and mechanical pressure coupling between the ICP and the eardrum (tympanic membrane). According to an animal study [4], the cochlear aqueduct connects the subarachnoid space of the brain to the perilymphatic space of the cochlea of the inner ear, thus transmitting ICP pulsation to the tympanic membrane pulsation (TMp). As a result, TMp signals may provide useful information about the ICP level. Recently, cardiac pulse-based waveforms have been used to detect ICP noninvasively [5]. In this study, TMp signals, possibly originating from cardiac pulsations are used and spectral analysis of the signals is performed for elevated ICP detection.

Five healthy volunteers participated in the study after IRB approval. TMp was measured under normal and elevated ICP conditions using a stethoscope headset (Sprague Rappaport stethoscope, ESR-112, Elite Medical Instrument Inc., Fullerton, CA 92831, USA) connected to a pressure transducer (DP103, diaphragm range dash number: 10, Validyne Engineering, Los Angeles, CA 91324, USA) with 60 cm long stethoscope tubing. The stethoscope earpieces made a tight seal with external ear canal which allows the transmission of the pressure signal to the transducer. Elevated ICP was induced by tilting the subject to a head down position using a tilt table, which is known to increase ICP [6]. At the beginning of the experiment, subjects rested on the table at a 45° head up tilt (HUT) position for 2 minutes and TMp was acquired for 30 seconds. Subsequently, the subjects were tilted to a -45° head down tilt (HDT) position and TMp was acquired for another 30 seconds.

The results suggested that TMp waveforms experienced distinctive morphological changes when ICP was increased. The rise and decay patterns of the waveforms changed when subjects were tilted to head-down position [7-8]. In addition, the high frequency peaks present in the TMp waveforms at HUT position diminished at HDT [9-10], as seen in Figure 1.

To quantify the energy in the observed high frequency peaks, the power spectral density (PSD) of TMp signals was estimated. Here, the acquired TMp signals were first filtered (pass-band 1 to 20 Hz) to remove environmental perturbations, subject movements and power-line noise. Subsequently, signals were normalized by their peak to peak value. A smooth spline was fitted to the normalized TMp waveform (see Figure 1, top row) and the resulting smooth signal was subtracted from the TMp waveform to extract a "residue" signal that primarily contained the high frequency TMp peaks (Figure 1, bottom row). In the next step, the PSD of both the TMp and its residue signal were estimated. Finally, the ratio of the spectral energy of these two signals (between 2^{nd} harmonic [starts at 2 Hz] and 20 Hz) was calculated (see Equation 1) to

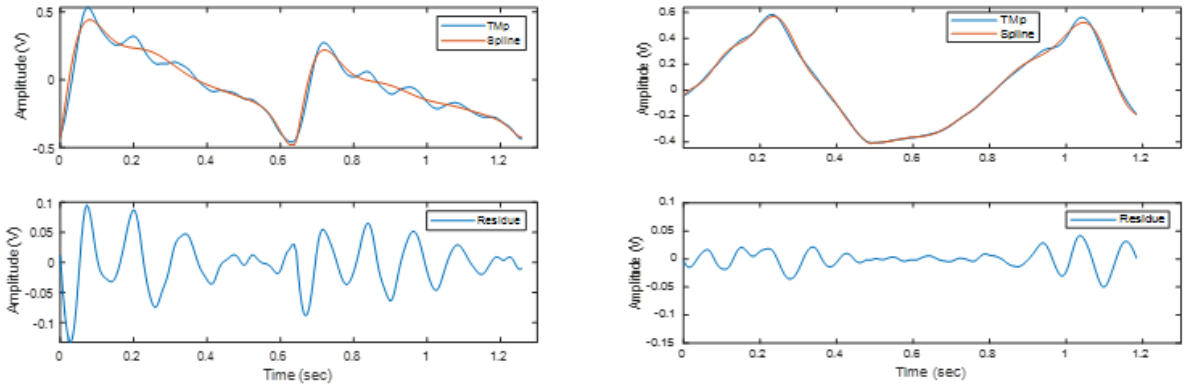

Figure 1. A representative TMp and fitted spline signals (top row) and the residue signal (bottom row) at head up tilt (left column) and head down tilt (right column) positions. The residue signal equals the difference between TMp and spline.

Subject Index	HUT	HDT	Reduction (%)
1	0.10	0.06	41.7
2	0.36	0.19	47.7
3	0.37	0.14	61.3
4	0.42	0.15	63.8
5	0.24	0.13	46.7

Table 1. Energy ratios (in the frequency band between the second harmonic and 20 Hz) at HUT and HDT and the reduction in the ratios with HDT (in percentage) for all subjects

provide a measure of the relative strength of the high frequency peaks. This energy ratio is listed in Table 1 for both the HUT and HDT positions along with the percentage ratio reduction in the HDT.

$$Energy\ Ratio = \frac{sum\ of\ PSD\ of\ residue\ signal\ between\ 2nd\ harmonic\ and\ 20\ Hz}{sum\ of\ PSD\ of\ TMp\ signal\ between\ 2nd\ harmonic\ and\ 20\ Hz} \quad (1)$$

The results of Table 1 illustrate the reduction in the energy ratio with elevated ICP in all study subjects (average 52.24%). Although the physiological mechanisms behind the energy ratio reduction are unknown, it may be related to the increased brain stiffness with elevated ICP. The consistent trend of energy ratio reduction suggests that this ratio may be useful for noninvasive detection of elevated ICP.

In the current study, the primary pressure oscillations were removed using the subtraction of a spline function. Other feasible approaches may include low-pass filtering that would remove the fundamental frequency of the primary oscillations but may not sufficiently remove its harmonics which may be in the frequency band of the residue signal. In our future work, we will compare the performances of the different approaches to detect elevated ICP noninvasively.

The proposed method of elevated ICP detection depends on the patency of the cochlear aqueduct. A study suggested that the patency of cochlear aqueduct decreases with age [11]. Therefore, this technique may be more applicable to young adults than older patients. Moreover, any other inhibition of ICP transmission to the tympanic membrane may result in lower efficacy of the suggested method. Other limitations of the study include a small number of subjects and the unavailability of direct ICP measurements. In the future, subjects with pathologic ICP elevation will be included in the study whose ICP is already being directly monitored. ICP elevation may be induced in patients using smaller tilt angles and sensitive pressure transducer may be used to detect subtle changes in the waveforms.

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