Investigation of intracranial media ultrasonic monitoring model

Vytautas Petkus\textsuperscript{a}, Arminas Ragauskas\textsuperscript{a,}\textsuperscript{*}, Rytis Jurkonis \textsuperscript{b}

\textsuperscript{a} Telematics Scientific Laboratory, Kaunas University of Technology, Studentu 50-448, LT-3031 Kaunas, Lithuania
\textsuperscript{b} Institute of Biomedical Engineering, Kaunas University of Technology, Studentu 50, LT-3031 Kaunas, Lithuania

Abstract

The objectives are to investigate the peculiarities of the ultrasound pulse propagation through human extra/intracranial media by mathematical simulation and to confirm the simulation results experimentally by proving the suitability of the ultrasonic time-of-flight measurement method for human intracranial media (IM) physiological non-invasive monitoring. The mathematical model of ultrasound pulse propagation through the human extra/intracranial media is described. The simulation of various physiological phenomena were performed to determine the relationship between the characteristics of the transmitted ultrasound pulse through the human head and the acoustic properties of the IM. It is shown that non-invasive monitoring of the IM acoustic properties is possible by measuring the changes of the ultrasonic signal time-of-flight and the oscillation period. The influence made by variations in acoustic parameters of the external tissue/skull bones on the non-invasive measurement data is investigated and methods of compensation of that influence are presented. The models were applied for developing of a new non-invasive sonographic intracranial pressure (ICP) monitor (Vittamed). Comparative studies of this monitor with the invasive ICP monitor (Camino) have shown the possibility of achieving clinically acceptable accuracy of the long term non-invasive ICP monitoring of head injured patients in intensive care units. © 2002 Elsevier Science B.V. All rights reserved.

Keywords: Acoustic properties monitoring; Intracranial media; Ultrasonic simulation; Time-of-flight method; Intracranial pressure/volume

1. Introduction

Various methods of non-invasive human intracranial pressure (ICP) measurement have appeared since 1980. However, the need for new non-invasive ICP monitoring technologies that are reliable and suitable for clinical application still exists [1]. The main problem is to find suitable parameter of human cerebrospinal system which would be a stable and repeatable function related to the ICP or cerebral perfusion pressure (CPP). In addition, this function need to be independent of factors such as arterial blood pressure (ABP) or cerebral blood flow autoregulation.

Recently, a new method [2] for the non-invasive measurement of intracranial volume or pressure has been created that is based on the ultrasonic time-of-flight measurement technique. It is capable of measuring the acoustic properties of the intracranial media (IM) such as ultrasound speed and ultrasound attenuation. The aim of this study is to answer the following questions:

- what is the relationship between the characteristics of the ultrasonic signal (time-of-flight, period of oscillation) measured non-invasively by the time-of-flight technique and the acoustic properties of the IM?
- how does it reflect the ICP changes or physiological state of the human brain?
- what is the influence that the skull bones (SB)/external tissues (ET) make on the time-of-flight measurement results and how to minimise that influence?

2. Theoretical model

The idea of measuring the changes of intracranial component volumes non-invasively is based on the transmission of a broadband ultrasonic signal through the human head and monitoring such signal parameters as the time-of-flight and the oscillation period [2–4].
all intracranial components (brain tissue, cerebrospinal fluid (CSF), blood) have different acoustic properties (ultrasound speed, frequency dependent attenuation), changes of their content inside the acoustic path will influence the total acoustic characteristics of IM and the monitored parameters of the ultrasonic signal as well.

While developing a model of the human cranium as a model of the acoustic media it was assumed that the total head volume is 1300 ml consisting of 1150 ml (88.46%) of brain tissue, 75 ml (10%) of CSF, and 75 ml (10%) of blood [5]. The assumption was made that the ultrasonic signal propagates through the cranium 15 cm on a straight line and the thickness of cranial components (according to the proportions presented above) are 13.27 cm of brain tissue, 0.865 cm of CSF and 0.865 cm of blood, respectively. For evaluating the influence of the skull bone, the thickness of bone layers on each side of the head was assumed to be equal to 0.8 cm. The acoustic parameters of the media components were obtained from the references [6–8] and listed in Table 1.

By creating a mathematical model of ultrasound pulse propagation through the human head, it was necessary to take into account the main factors that influence monitored parameters of the ultrasonic signal i.e., ultrasound attenuation, ultrasound velocity dispersion and diffraction effects. For simulation of the frequency dependent ultrasound attenuation and ultrasound velocity dispersion the spectrum decomposition method (derived by Ping He [9]) was chosen. For the simulation of diffraction effects the method of calculation of the impulse response of transmitter–receiver transducers in lossy media (derived by Lukosevicius [10]) was used. These methods were combined and developed to calculate the impulse response of the transducer system in layered attenuating media by assuming that the edge wave travels through the boundary of two different media according to Snell’s law [3].

The real input ultrasonic signal used for simulation, was copied from the transducers of a non-invasive ICP monitor using PVDF piezofilms and recorded by an oscilloscope HP54615B (Fig. 1). The central frequency of ultrasound transducers used for measurement was 1.8 MHz. The simulation of this signal propagation through the human head was performed and the change to its waveform is shown in Fig. 1.

<table>
<thead>
<tr>
<th>Medium</th>
<th>Attenuation parameters</th>
<th>Ultrasound speed c, m/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skull bone</td>
<td>11.089 dB/(cm MHz)</td>
<td>1.89</td>
</tr>
<tr>
<td>Brain tissue</td>
<td>0.8692</td>
<td>1.078</td>
</tr>
<tr>
<td>CSF</td>
<td>0.0023</td>
<td>1.9937</td>
</tr>
<tr>
<td>Blood</td>
<td>0.212</td>
<td>1.2662</td>
</tr>
</tbody>
</table>

![Fig. 1. The change of an ultrasound pulse waveform when it propagates through the human head. For comparison, the input signal is multiplied by 0.05, the signal passed through the skull bone on the one side of the head is multiplied by 0.2 and the signal passed through the whole head is multiplied by 3. All signals are shifted left in the time domain by their group delays calculated at the frequency 1.8 MHz (tgroup(bone) = 2.4753 µs, tgroup(bone + brain) = 98.8449 µs, tgroup(bone + brain + bone) = 101.8202 µs).](image)

The three most common physiological phenomena which cause an increase of ICP were simulated:

1. Brain swelling phenomenon. ICP increases during this phenomenon when the swelling brain tissue causes the increase of its volume and a decrease of the CSF volume. CSF is pushed out from the cerebral acoustic path into the spinal canal. By simulating the changes of volumes inside the parenchymal acoustic path, the brain tissue volume was increased by volume deviation from −8 to +8 ml. CSF volume was decreased by the same volume deviation and blood volume was not changed.

2. Vasodilatation phenomenon. ICP increases when the blood volume increases in the human brain by pushing out the brain tissue or CSF volume from the parenchymal acoustic path (depending on the acoustic parenchymal path chosen). Both these cases were simulated. In the first case (vasodilatation-1), blood volume was increased by volume deviation from −8 to +8 ml, brain tissue volume was decreased by the same volume deviation and CSF volume was not changed. In the second case (vasodilatation-2), the increase of blood volume by ±8 ml causes a decrease of CSF by the same volume amount. However, now the blood volume was not changed.

3. Hydrocephalus. In this case the increased volume of CSF inside the human brain causes squeezing of the cerebral veins, decreasing cerebral blood flow and blood volume inside IM. In this case, the CSF volume is increased and blood volume decreased by ±8 ml. The brain tissue volume was not changed.
The volume changes of intracranial components was chosen to be ±8 ml as such increase of the craniospinal volume can cause increase of ICP up to 25 mmHg. It is also typical for B class Lundberg’s waves, the appearance of which indicates a possible losing of cerebrovascular autoregulation [11].

The signal’s time-of-flight and oscillation period dependencies on the intracranial component volumes changes are shown in Fig. 2. It is seen that the signal parameters changes are linear under the physiological limits of the intracranial components volume changes i.e., 8 ml. The changes of both time-of-flight and oscillation period are in the ranges of 6–20 and 0.12–0.5 ns respectively per 8 ml craniospinal volume changes. These changes are technologically detectable using the non-invasive time-of-flight measurement technique that is capable to perform measurement of time interval with the resolution higher than 100 ps in real time [2]. The monitoring of both signal parameters (i.e., the time-of-flight and the oscillation period) can help to determine the character of the phenomena that occur in the human head and to help of choosing a more optimal acoustic path for the measurements.

3. Influence of skull bones and external tissues

The influence of SB and ET must be taken into account while performing non-invasive transintracranial ultrasonic measurements. The variation of ET acoustic properties caused by the hemodynamics or swelling in these tissues influences the change of measurement results, separately to the state of the IM.

The methods of non-invasive measuring of the acoustic properties of IM with a compensation of SB/ET influence are shown in Fig. 3. Signal delays in the SB/ET are evaluated by measuring the time-of-flight of the echo signal that is reflected from internal surfaces of the skull and extracted from the time-of-flight of the signal transmitted through the human head [2]. Further, the time-of-flight data are used for obtaining the relative values of ultrasound speed in IM. For measuring of ultrasound attenuation in IM measurement, in addition, the oscillation periods of the input, through transmitted and skull echo signals must also be monitored [4].

4. Results

A new non-invasive ultrasonic Vittamed monitor based on the models of the ultrasound speed in cerebral parenchymal acoustic path measurement was designed and tested in an intensive care unit (ICU). The example of simultaneous long term (more than 3 h) ICP monitoring with a new non-invasive Vittamed monitor and with a Camino V420 invasive monitor for a head injury patient in the ICU is shown in Fig. 4. The non-invasive ICP data are calculated from the time-of-flight measured data using the linear functions after the real-time and in situ compensation of the SB/ET influence on the measured time-of-flight data.

While attempting to prove this linear relationship between the non-invasively measured ultrasound pulse time-of-flight through the IM and ICP, the readings from the invasive ICP monitor are plotted against the readings of a non-invasive ICP monitor. Monitoring data from 28 head injured patients are shown in Fig. 5. These experimental results (Fig. 5) are the evidence that the ultrasound velocity changes are linearly dependent.
on the ICP changes. It also confirms the results of mathematical simulation of relationship between the time-of-flight and blood volume inside the transintra-cranial parenchymal acoustic path.

5. Conclusions

The investigation of the human IM physiological monitoring model allows us to develop the concepts for non-invasive ICP/CPP measurements:

- The intraventricular or supraventricular parenchymal acoustic path which crosses the human head is used for non-invasive measurements. The parenchymal arterioles that are located in this path are responsible for cerebral blood flow autoregulation [12]. The ultrasound speed inside the parenchymal acoustic path mainly depends on the blood volume inside this path.
- To measure the relative value of ultrasound speed changes inside the parenchymal acoustic path, this path is insonated by supershort ultrasonic pulses, and the time-of-flight measuring method is used.
- To compensate the influences of the human head external tissue hemodynamics in real-time and in situ, the same ultrasonic pulses and their echoes from the internal surfaces of the skull are used.
- In the case of intact cerebral blood flow autoregulation, ultrasound speed changes inside this path reflect CPP changes. In the case of stabilised ABP the ultrasound speed changes reflect ICP changes [12].

The new non-invasive sonographic ICP monitor Vittamed was created and developed following these concepts. The comparative studies of this monitor in the ICU show that it is possible to achieve an uncertainty better than ±2 mmHg for long term non-invasive ICP monitoring. This is a clinically acceptable error that must be met by invasive ICP meters used in the ICU. The linear relationship between ultrasound speed in the cerebral parenchymal acoustic path and the ICP was proved by calculating the ICP data from the non-invasively measured time-of-flight data. Such relationship was obtained for the first time during the long term monitoring in a wide range of ICP values from 0 to 50 mm Hg.

References