Journal of Clinical Monitoring and Computing (2011) 25:203–210
DOI: 10.1007/s10877-011-9297-x

NONINVASIVE INTRACRANIAL PRESSURE MEASUREMENT USING INFRASONIC EMISSIONS FROM THE TYMPANIC MEMBRANE

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Stettin E, Paulat K, Schulz C, Kunz U, Mauer UM. Noninvasive intracranial pressure measurement using infrasonic emissions from the tympanic membrane.
J Clin Monit Comput 2011; 25:203–210

ABSTRACT. Objective. We investigated whether ICP can be assessed by measuring infrasonic emissions from the tympanic membrane. **Methods.** An increase in ICP was induced in 22 patients with implanted ICP pressure sensors. ICP waveforms that were obtained invasively and continuously were compared with infrasonic emission waveforms. In addition, the noninvasive method was used in a control group of 14 healthy subjects. **Results.** In a total of 83 measurements, the changes in ICP that were observed in response to different types of stimulation were detected in the waveforms obtained noninvasively as well as in those acquired invasively. Low ICP was associated with an initial high peak and further peaks with smaller amplitudes. High ICP was associated with a marked decrease in the number of peaks and in the difference between the amplitudes of the initial and last peaks. The assessment of infrasonic emissions, however, does not yet enable us to provide exact figures. **Conclusion.** It is conceivable that the assessment of infrasonic emissions will become suitable both as a screening tool and for the continuous monitoring of ICP in an intensive care environment.

KEY WORDS. infrasonic emission, intracranial pressure measurement, intracranial pressure monitoring.

INTRODUCTION

The assessment of intracranial pressure (ICP) is a routine method in everyday neurosurgical, neurological and intensive care clinical practice. The gold standard is the invasive monitoring of ICP using an external cerebrospinal fluid (CSF) drainage system. Different types of (epidural, intraparenchymal and intraventricular) sensors can be inserted for continuous ICP monitoring. This method is associated with surgical risks such as hemorrhage, infection in patients hospitalized for long periods, and injury to the brain parenchyma. Over the past decades, many efforts have therefore been made to find a method that allows ICP to be assessed noninvasively.

Rheography, which was introduced as early as 1943 [32] is a method of measuring changes in the capacitive resistance between electrodes placed in frontal and retromastoid regions. These changes are caused by variations in dielectric properties within the brain which result from blood and fluid shifts [14, 30].

In 1990, Reid et al. [29] found that patients with raised and normal ICP showed differences in tympanic membrane

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Received 23 January 2011. Accepted for publication 6 August 2011.

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displacement in response to the stimulation of the stapedial reflex.

Anatomically, the ear is divided into three parts. The outer ear consists of the pinna (concha), the external auditory meatus (outer ear canal), and the tympanic membrane (ear drum). The external auditory meatus is 2.5–3.5 cm long and ends in the tympanic membrane, which anatomically separates the outer ear from the middle ear and vibrates in response to sound waves as a result of its flexibility. The handle of the malleus (hammer) is attached to the medial surface of the eardrum. The malleus, the anvil (incus) and the stapes (stirrup) are three small bones that are called the auditory ossicles and can be found in the tympanic cavity. Together with the Eustachian (auditory) tube and the mastoid antrum, they form the middle ear. The base of the stirrup is flexibly attached to the oval window. Since the auditory ossicles form a lever system that helps increase the force of vibrations by a factor of 1.3 and since the surface of the tympanic membrane is 17 times larger than that of the oval window, the sound pressure at the oval window is 22 times greater than that at the eardrum. Sound waves are transmitted from the oval window to the endolymph of the membranous labyrinth. The inner ear is a bony labyrinth that is filled with perilymph and contains the membranous labyrinth. It includes the cochlea and the semicircular canals of the vestibular system. The inner ear is connected to the subarachnoid space by the cochlear duct in the region of the posterior petrous bone. ICP is transmitted via the aforementioned structures to the tympanic membrane and can be assessed on the basis of infrasonic emissions [26, 28, 31].

Infrasonic emissions are measured by recording the displacement of the tympanic membrane using a measuring probe that is placed into the external auditory meatus in an airtight manner. Tympanic membrane displacement is a result of the transmission of ICP waves via the cochlear duct to the inner ear and then via the oval window to the auditory ossicles of the middle ear, which are firmly attached to the tympanic membrane. These pressure waves displace the tympanic membrane in a characteristic way. Specific changes in tympanic membrane displacement are indicative of high or low ICP [12].

The objective of this study was to investigate the clinical effectiveness of a method of assessing infrasonic emissions from the tympanic membrane which has already been used in human subjects by the University of Applied Sciences in Ulm. A number of measurements were performed by Mauer et al. [25] in a pilot study, which proved the technical feasibility of this method. Technical problems that had been identified in the pilot experiment were resolved prior to the beginning of the present study.

METHODS

Different approaches were used to induce an increase in ICP in patients with raised intracranial pressure. ICP waveforms that were obtained invasively and infrasonic emission waveforms were compared with infrasonic emission waveforms acquired in a group of healthy subjects.

For this purpose, we used a pressure sensor (HCPM005D6 pressure sensor, SensorTechnics, Puchheim, Germany). Tympanic membrane vibrations resulting from the transmission of ICP via the auditory ossicles were assessed on the basis of pressure differences. We used a differential pressure sensor with a pressure range of +5 to −5 mbar and an output range of 0.5–4.5 V.

Pressure signals are transmitted through a polyvinyl chloride (PVC) tube from the external auditory meatus to the sensor. For this purpose, an ear plug was attached to the tube and inserted in the external auditory meatus in order to provide an airtight seal. Since silicone, which is used for hearing aids and hearing protection, hardens quickly and can be easily removed, it was used for sealing.

The air in the sealed space between the sensor and the tympanic membrane is warmed by body heat and expands. The system comprises a mechanical device that allows air pressure to be regulated at any time and thus to establish normal pressure. Electrocardiographic (ECG) signals that were required for triggering were recorded in a procedure modified from Einthoven. In each group, a series of five complete RR intervals was analyzed in every case.

A MOMO multi-function monitoring system (DWL, Ueberlingen, Germany) was used for data transmission. Data from the intensive care unit (ICU) monitoring system and infrasonic emission data acquired during the examinations were transmitted by this system to the documentation computer. Since the pure signal at the ear is easily affected by noise, a hardware filter (a 5th order Butterworth low-pass filter, 2.5 kHz) was integrated into the monitoring system.

A total of 22 patients took part in the study and were placed in one of three subgroups, i.e. patients with an ICP probe and stimulation by patient position changes in bed, patients with an ICP probe and stimulation by pressure–volume tests according to Katzman and Marmarou [23, 24], and patients with an ICP probe and stimulation by positive end-expiratory pressure (PEEP) changes.

The patient group consisted of 12 men and 10 women aged between 26 and 77 years. Three patients were intubated and ventilated. A total of 52 measurements were performed. Invasive intracranial pressure monitoring was clinically indicated in all patients. Five patients were managed with an external ventricular drainage system and 17 patients with an epidural sensor. Indications for invasive ICP monitoring were normal pressure hydrocephalus in 17 cases, intracerebral hemorrhage in one case, subarachnoid hemorrhage in two cases, and intraventricular hemorrhage in two further cases. These patients do not have a critically raised ICP. It is impossible to include patients with elevated ICP in our study for the following reasons. Pretests showed that these measurements cannot be performed over a period of several hours. The measurement method is, however, based on the detection of pressure changes—in this case an increase in intracranial pressure. Inducing an ICP increase in a patient with elevated, though not critically raised, intracranial pressure would be ethically unacceptable. A change in intracranial pressure would, thus, be detected only by chance. Against this background, the ethics committee decided that the present study should be conducted primarily on patients with an ICP that is not critically elevated.

In addition, 14 healthy subjects (12 men and two women) aged between 19 and 62 years took part in the study. A total of 31 measurements were performed in the control group.

Intracranial pressure sensors had already been inserted in all patients before the measurements began. We collected electrocardiographic data and inserted the measuring probe into the external auditory meatus in an airtight manner. In the group of patients with stimulation by patient position changes, air pressure was regulated prior to the measurements and every position change. Initially, the patients lay flat on their backs. After a minimum period of 5 min of data recording, the head of the patient's bed was raised 45°. After five further minutes of data recording, the head of the bed was lowered again to the horizontal (0°) and data were recorded for five further minutes. The measuring probe was then inserted into the contralateral external auditory meatus and the entire procedure was repeated.

When the measurements were performed in the group of healthy subjects, ICP changes were stimulated by changing the subject's position in a way similar to that used in the patient group. Positions were, however, changed using a tilting table. Prior to the measurements and every position change, air pressure in the system was controlled. The measurement procedure consisted of four phases. Initially, the tilting table was set at 0°. After a minimum period of 3 min of data recording, the table was tilted to 45°. The table was then tilted to a vertical position (90°). Finally, the table was lowered again to the horizontal (0°) and data were recorded for the last time over a period of at least 3 min.

RESULTS

The patient group with stimulation by position changes included 17 patients aged between 26 and 77 years. We performed a total of 31 measurements, 25 of which were suitable for analysis. Figure 1 shows typical waveforms for this group. The waveform that was obtained in the 45° condition had several peaks and clearly elevated levels in the distal waveform segment. The waveforms that were obtained in the horizontal position (higher ICP) showed a single sharp peak and a considerably flatter distal segment of the waveform. The other measurements that were performed in patients from this group confirmed these findings.

The group of healthy subjects with stimulation of ICP changes by position changes on the tilting table comprised 14 persons aged between 19 and 62 years. We performed a total of 31 measurements, 27 of which were suitable for analysis. Figure 2 shows typical waveforms for this group. When the subjects had an increased ICP during the first and last phases of the measurements, the waveforms were characterized by an initial high peak and a subsequent steep decline. When the subjects had a low ICP in the 45° and 90° conditions, the waveforms showed several peaks and elevated levels in the distal waveform segment and a lower initial peak.

A direct comparison of the waveforms obtained for the different types of stimulation revealed a number of characteristic features of the waveforms after pressure changes. An increase in ICP was associated with a decrease in the number of peaks. Moreover, the waveforms for high ICP showed regular changes in their distal segments when compared with the waveforms for a low ICP.

A comparison of healthy subjects and patients showed similar and reproducible waveforms before and after the stimulation of ICP changes by position changes (Figures 1, 2). High ICP was associated with a high peak and a subsequent steep decline in the first and last measurement phases. Low ICP was associated with a considerably elevated distal waveform segment. Both groups show similar waveforms not only before and after but also during stimulation by position changes. The figure clearly shows how ICP changed when the head of the bed was raised or the table was tilted.

Figure 3 shows two segments of measurements from one patient. The infrasonic emission waveforms have

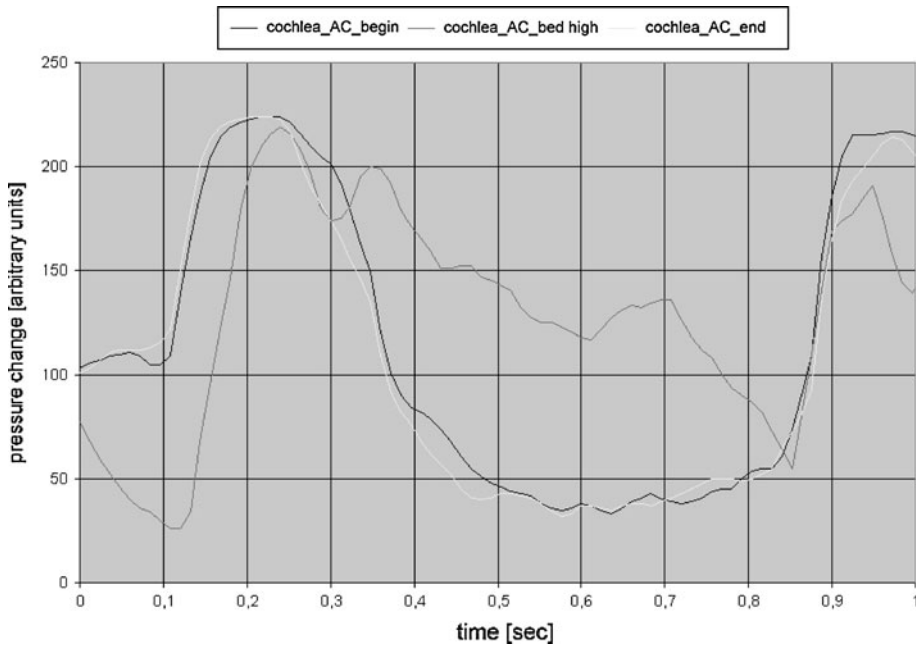

Fig. 1. Typical waveforms for a patient after stimulation by position changes.

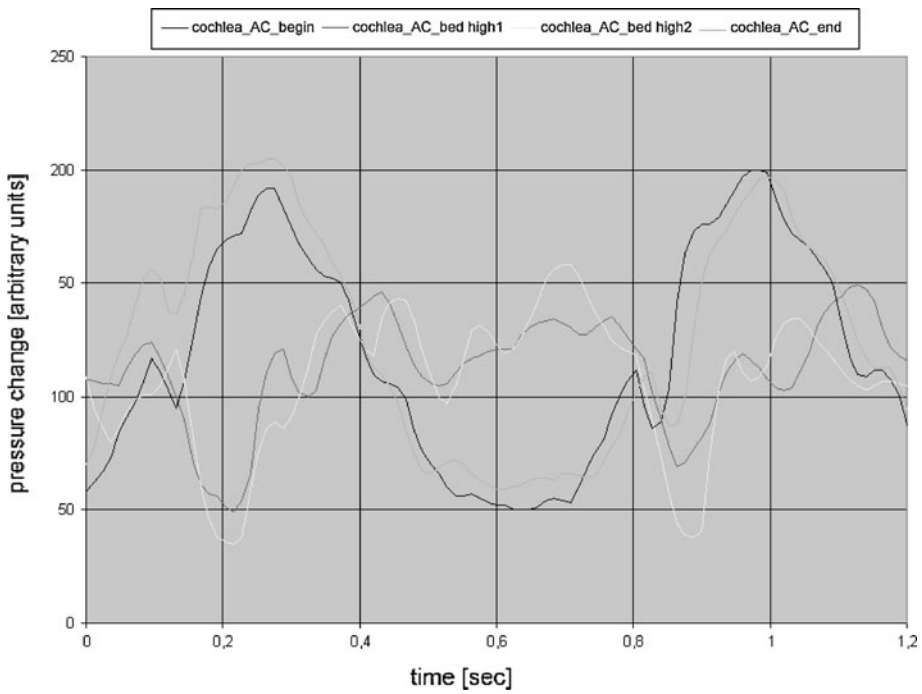

Fig. 2. Typical waveforms for a healthy subject after stimulation by position changes on a tilting table.

several peaks, especially in the distal waveform segment. The initial peak is the highest. The invasively acquired waveform, too, shows a decrease. The first peak (P1) is higher than the second one (P2). The third set of waveforms shows the waveforms obtained when ICP was raised. The infrasonic emission waveforms decline more

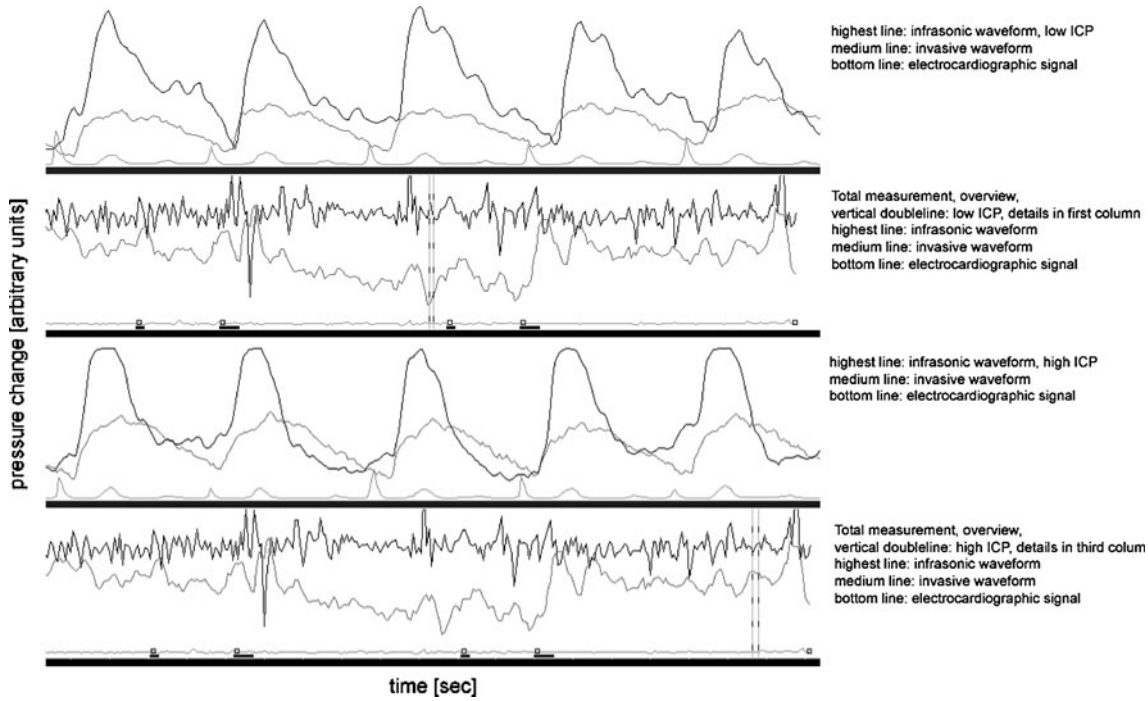

Fig. 3. Comparison of invasively and noninvasively acquired waveforms.

steeply after an initial high peak and have only a few less distinct peaks in the distal waveform segment. The invasively acquired waveform shows the typical changes associated with an increase in ICP. P2 is higher than P1.

Both waveforms change with increasing ICP. The waveform changes, however, are different. Nevertheless, the changes in infrasonic emission waveform features can be clearly seen and allow high and low ICP levels to be distinguished.

The results of our waveform comparisons can be summarized as follows:

1. A graphical analysis shows clear differences in the waveforms for high and low ICP levels. In the majority of cases, high ICP was associated with an initial high peak and a few less distinct later peaks. Low ICP was usually associated with more peaks and smaller differences between the peaks in the proximal and distal waveform segments.
2. A comparison of the waveforms obtained for identical ICP levels in the first and last measurement phases showed similar waveform features and thus reproducible waveforms before and after ICP stimulation.
3. In the patient group, the mean number of peaks was 2.8 for high ICP and 3.7 for low ICP. In 12 of 42 measurement segments, however, high ICP was associated with an equal or a higher number of peaks when compared with low ICP.

   In the group of healthy subjects, the mean number of peaks was 3.0 for high ICP and 4.3 for low ICP. In 5 of 25 measurement segments, however, high ICP was associated with an equal or a higher number of peaks when compared with low ICP.

   These results show that the number of peaks decreases with an increase in ICP.
4. In the patient group, the mean difference between the peaks in the proximal and distal waveform segments was 96.74 arbituary units for high ICP and 53.56 arbituary units for low ICP. In 5 of 39 measurement segments, however, low ICP was associated with a higher amplitude difference when compared with high ICP.

   In the group of healthy subjects, the mean difference between the peaks in the proximal and distal waveform segments was 96.76 arbituary units for high ICP and 23.20 arbituary units for low ICP. In 1 of 25 measurement segments, however, low ICP was associated with a higher amplitude difference when compared with high ICP.These results show that higher amplitude differences between the various waveform segments were associated with higher ICP levels.
5. A comparison of the methods of stimulation and the groups of subjects showed that ICP changes were reflected in the graphical representations as well as in the numbers of peaks and amplitude differences.

6. The assessment of infrasonic emissions allows ICP changes to be detected. In the absence of a zero point, however, exact figures cannot be provided.
   The waveform obtained by measuring intracranial pressure noninvasively enabled us to assess the pressure in the intracranial space on the basis of the number of peaks in patients (error of probability: 74%) and healthy subjects (error of probability: 80%) and on the basis of amplitude differences in patients (error of probability: 87%) and healthy subjects (error of probability: 96%).

DISCUSSION

In our study, we used a noninvasive and an invasive method of ICP measurement. Avezaat et al. [4] conducted a pressure wave analysis in 1979 and found a correlation between invasively acquired ICP pulse waveforms and intracranial pressure increase. When ICP is normal, the waveform shows a high peak (P1), which reflects an increase in systolic blood pressure. An increase in ICP is associated with a P2 waveform component that rises to the level of and then surpasses P1 [5, 6, 9, 11, 17]. The infrasonic emission waveform shows a number of high peaks. Both invasively acquired ICP waveforms and noninvasively obtained infrasonic emission waveforms occur with some delay relative to the R wave. The P1 component of the invasively acquired waveform results from an intracranial pressure increase that is induced by blood pressure. This is accompanied by maximum tympanic membrane displacement, which is reflected in the highest peak in the noninvasively acquired waveform [33]. The P2 component of the invasively acquired waveform is called the tidal wave and results from the difference between pressure-wave and pulse-wave velocities. It leads to a brief period of intracranial pressure stagnation or increase [1, 8, 33]. It is possible that the further peaks in the noninvasively acquired infrasonic emission waveform result from effects of the tidal wave. The exact origin, however, is unclear. P3 is the third and last component of the invasively acquired waveform and is known as the dicrotic wave.

Unlike the invasively acquired pressure waveform, the noninvasively obtained waveform does not show an association between high ICP and an elevation of the distal waveform segment, which can even be completely absent in the case of a considerable increase in ICP. By contrast, low ICP is likely to be associated with one or more high peaks in the distal segment of the waveform. Low ICP shows a larger total number of peaks than high ICP.

The exact origin of the peaks in the infrasonic emission waveform is unclear. The aforementioned observations suggest, however, that an increase in ICP influences the entire endolymphatic and perilymphatic system and thus also the auditory ossicles. The higher the ICP level, the less freely the tympanic membrane (i.e. the end organ) can vibrate. As a result, the tympanic membrane responds only to peak levels and transmits only a few strong impulses. As early as 1964, Hommerich demonstrated a relationship between hearing loss or a reduced ability of the tympanic membrane to vibrate and increased ICP [18, 19, 35].

A direct comparison of infrasonic emission waveforms from healthy subjects and patients reveals that the waveforms for high and low ICP levels show a wider range of variation in their individual courses. This may be explained by the normal ICP of the healthy subjects. Reference data from healthy subjects are important for two reasons. Firstly, healthy subjects allow us to gain basic experience with the method in a laboratory setting. Secondly, the method serves to identify patients with pathological ICP. This, however, necessitates the availability of physiological data.

Patients were included in this study only if their medical condition was associated with an elevated ICP. Although the patients had different types of conditions such as normal pressure hydrocephalus, intraventricular bleeding, subarachnoid bleeding, and intracerebral bleeding, they all presented with an increased ICP and poor intracranial compliance irrespective of their diagnoses.

A further reason for the interindividual range of variation is the notable difference in age between the two groups. The mean age was approximately 58 years in the patient group and only approximately 28 years in the group of healthy subjects. Increasing age is associated with a decrease in the elasticity of the tympanic membrane [13] and progressive ossification of the cochlear duct [21]. A study on a larger number of patients should be conducted in an attempt to establish an age-related factor that quantifies and compensates for age-dependent changes.

A comparison of the waveforms for healthy subjects and patients shows that the waveforms for healthy subjects on average have more peaks. This applies in particular to waveform segments obtained for low ICP. As early as 1989, Phillips and Marchbanks measured tympanic membrane displacement in response to acoustic stimulation of the stapedial reflex in subjects who fell into two different age groups and found that the ability of the tympanic membrane to vibrate decreased with age [27].

In a total of 6 measurements (4 in the patient group, 2 in the healthy subject group), the waveform for high ICP showed the highest peak in its distal segment. The waveform for low ICP showed an unexpected elevation in its proximal segment and a minor decrease in its distal

segment. The number of peaks, the change in the number of peaks after stimulation, and the amplitude difference corresponded with previous average results. This difference can be the result of a variation in the location of the tympanic membrane. Insufficient ventilation of the tympanic cavity through the Eustachian tube can lead to negative pressure causing the tympanic membrane to move inward. This causes a change in the ability of the tympanic membrane to vibrate [10].

Differences in the results of the assessment of infrasonic emissions can also be the result of inflammatory changes or scarring in the region of the tympanic membrane, diseases such as otosclerosis, or tympanic membrane perforation [10].

The study group included 17 patients with a suspected diagnosis of normal pressure hydrocephalus (NPH). Diagnostic investigations include invasive procedures such as the insertion of an intracranial pressure monitoring device that records ICP over two nights and a pressure–volume test, which was performed by 8 patients. As a result of poor intracranial compliance, autoregulatory mechanisms in patients with NPH are largely or entirely unable to compensate for ICP changes caused by breathing, heart beat or external factors [2, 7, 15, 16, 20, 34]. During recording, the measuring system was found to be easily affected by movement noise, e.g. sounds, yawning, snoring, or voluntary and involuntary movements of the body such as breathing movements of the thorax. For this reason, measurements over extended periods of time, for example for the detection of pathological waveforms in patients with normal pressure hydrocephalus at night [3, 11, 22], are more difficult since involuntary patient movements can cause too many measuring artefacts during sleep. This method is more suitable for measurements in patients requiring long-term intubation in the ICU or for measurements over short periods, for example during pressure–volume tests. When the aforementioned 8 patients underwent pressure–volume tests, the measurements confirmed the invasively obtained results and demonstrated changes in ICP. Changes in waveform features reliably indicated changes in ICP. The assessment of infrasonic emissions from the tympanic membrane, however, does not yet enable us to provide exact figures.

CONCLUSIONS

The waveform obtained by measuring intracranial pressure noninvasively enables us to assess the pressure in the intracranial space on the basis of the number of peaks and amplitude differences in patients and healthy subjects. This method does not allow us to measure absolute ICP levels. Absolute measurements, however, may be unnecessary since this method is likely to provide a measure of compliance, which can provide more important information than absolute ICP levels.

Since it is currently unlikely that absolute ICP levels can be established, one possible approach is the development of computer software that analyzes and evaluates the shape and number of peaks of ICP curves and then, for example, uses a green, yellow or red light to indicate normal ICP levels, a possible increase in ICP, or a pathological increase in intracranial pressure.

REFERENCES

1. Aaslid R, Markwalder TM, Nornes H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg 1982; 57: 769–774.
2. Adams RD, Fisher CM, Hakim S, Ojemann RG, Sweet WH. Symptomatic occult hydrocephalus with "normal" cerebrospinal-fluid pressure. A treatable syndrome. N Engl J Med 1965; 273: 117–126.
3. Arnold H, Lars R. Plateau waves; production in the rat and simulation by means of a mechanical model. Intracranial pressure IV, Springer: Heidelberg, 1980, p. 525.
4. Avezaat CJ, van Eijndhoven JH, Wyper DJ. Cerebrospinal fluid pulse pressure and intracranial volume-pressure relationships. J Neurol Neurosurg Psychiatry 1979; 42: 687–700.
5. Dardenne G, Dereymaeker A, Lacheron JM. Cerebrospinal fluid pressure and pulsatility. An experimental study of circulatory and respiratory influences in normal and hydrocephalic dogs. Eur Neurol 1969; 2: 193–216.
6. Dereymaeker A, Stevens A, Rombouts JJ, Lacheron JM, Pierquin A. Study on the influence of the arterial pressure upon the morphology of cisternal CSF pulsations. Eur Neurol 1971; 5: 107–114.
7. Dommasch D, Mertens H. Physiologie und Pathophysiologie der Liquordynamik. In: Brock M, ed. Cerebrospinalflüssigkeit-CSF. Thieme Verlag: Stuttgart; 1980. p. 212–224.
8. Doyle DJ, Mark PW. Analysis of intracranial pressure. J Clin Monit 1992; 8: 81–90.
9. Dunbar HS, Guthrie TC, Karpell B. A study of the cerebrospinal fluid pulse wave. Arch Neurol 1966; 14: 624–630.
10. Franzen A. Kurzlehrbuch Hals-Nasen-Ohren-Heilkunde, Elsevier, Urban & Fischer Verlag: München; 2007. p. 1–64.
11. Gaab MR. Die Registrierung des intrakraniellen Druckes. Grundlagen, Techniken, Ergebnisse und Möglichkeiten, in Med. Habilitationsschrift. Julius-Maximilians-Universität Würzburg; 1980.
12. Gaihede M, Felding JU, Elbrond O. Biomechanical characteristics of the middle ear system measured by a new method. III: Comparisons with tympanometric measurements. Acta Otolaryngol 1995; 115: 522–527.
13. Gaihede M, Koefoed-Nielsen B. Mechanics of the middle ear system: age-related changes in viscoelastic properties. Audiol Neurootol 2000; 5: 53–58.

14. Ghajar J. Intracranial pressure monitoring techniques. New Horiz 1995; 3: 395–399.
15. Hakim S. Some observations on CSF pressure: hydrocephalic syndrome in adults with ''normal'' CSF pressure. Thesis No. 957. Bogota, Columbia, Javeriana University School of Medicine, 1964.
16. Hakim S, Adams RD. The special clinical problem of symptomatic hydrocephalus with normal cerebrospinal fluid pressure. Observations on cerebrospinal fluid hydrodynamics. J Neurol Sci 1965; 2: 307–327.
17. Hamer J, Alberti E, Hoyer S, Wiedemann K. Influence of systemic and cerebral vascular factors on the cerebrospinal fluid pulse waves. J Neurosurg 1977; 46: 36–45.
18. Hommerich KW. Hearing disorders and disturbances of the olfactory system in intracranial diseases. Arch Ohren Nasen Kehlkopfheilkd 1964; 183: 86–124.
19. Hommerich KW. Intracranieller Druck und Cochlearfunktion, in Med. Habil.-Schrift. Heidelberg: Dr. Alfred Hütig Verlag Heidelberg; 1963. p. 87.
20. Kontos HA, Wei EP, Navari RM, Levasseur JE, Rosenblum WI, Patterson JLJ. Responses of cerebral arteries and arterioles to acute hypotension and hypertension. Am J Physiol 1978; 234: H371–H383.
21. Marchbanks RJ, Reid A. Cochlear and cerebrospinal fluid pressure: their inter-relationship and control mechanisms. Br J Audiol 1990; 24: 179–187.
22. Marmarou A, Bullock R, Young H. The contribution of raised ICP and hypotension to reduced cerebral perfusion pressure in severe brain injury. In: Nagai H, Kamiya K, Ishii S, eds. Intracranial pressure IX. Tokyo, New York: Springer-Verlag; 1994. p. 302.
23. Marmarou A, Shulman K, LaMorgese J. Compartmental analysis of compliance and outflow resistance of the cerebrospinal fluid system. J Neurosurg 1975; 43: 523–534.
24. Marmarou A, Shulman K, Rosende RM. A nonlinear analysis of the cerebrospinal fluid system and intracranial pressure dynamics. J Neurosurg 1978; 48: 332–344.
25. Mauer U, Paulat K, Galler M, Kunz U. Nichtinvasive Messung des intracraniellen Drucks mit Hilfe der Infraschallemission des Trommelfells. In: Weinrich D, ed. Artikel zu Wehrmedizin und Wehrpharmazie 2003. vol. 16. Bonn; 2003. p. 25–27.
26. Paulat K, Mauer U, Galler M. Nichtinvasive Messung des intracraniellen Druckes mit Hilfe der Infraschallemission des Trommelfells, in Fachhochschule Ulm, Ulm: Ulm, 2000, p. 95.
27. Phillips AJ, Marchbanks RJ. Effects of posture and age on tympanic membrane displacement measurements. Br J Audiol 1989; 23: 279–284.
28. Reid A, Marchbanks RJ, Bateman DE, Martin AM, Brightwell AP, Pickard JD. Mean intracranial pressure monitoring by a non-invasive audiological technique: a pilot study. J Neurol Neurosurg Psychiatry 1989; 52: 610–612.
29. Reid A, Marchbanks RJ, Burge DM, Martin AM, Bateman DE, Pickard JD, et al. The relationship between intracranial pressure and tympanic membrane displacement. Br J Audiol 1990; 24: 123–129.
30. Russegger L, Ennemoser O. Atraumatic measurement of intracranial pressure. Wien Klin Wochenschr 1990; 102: 543–547.
31. Schiebler TH, Schmidt W, Arnold G. Organum vestibulum cochleare, Ohr, Hör- und Gleichgewichtsorgan. In: Schiebler TH, ed. Lehrbuch der gesamten Anatomie des Menschen Cytologie, Histologie, Entwicklungsgeschichte, makroskopische und mikroskopische Anatomie unter Berücksichtigung des Gegenstandskataloges, überarb. und erg. 2nd edn. Aufl. Berlin-West Heidelberg New York: Springer; 1981. p. 723–738.
32. Schuhfried F. Rheography. Arztl Forsch 1961; 15: I/455–I/462.
33. Steinhausen M. Herz, in Medizinische Physiologie. Gustav Fischer; Stuttgart: 1993. p. 31–76.
34. Turkheimer E, Cullum CM, Hubler DW, Paver SW, Yeo RA, Bigler ED. Quantifying cortical atrophy. J Neurol Neurosurg Psychiatry 1984; 47: 1314–1318.
35. Walker CB. Analogies and differences of the second and eighth nerves and end-organs: generalizing, preliminary, anatomic considerations, especially with reference to choked disc, glaucoma, and choked labyrinth. Trans Am Ophthalmol Soc 1931; 29: 304–320.