Pulse pressure waveform in hydrocephalus: what it is and what it isn’t


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Object. Apart from its mean value, the pulse waveform of intracranial pressure (ICP) is an essential element of pressure recording. The authors reviewed their experience with the measurement and interpretation of ICP pulse amplitude by referring to a database of recordings in hydrocephalic patients.

Methods. The database contained computerized pressure recordings from 2100 infusion studies (either lumbar or intraventricular) or overnight ICP monitoring sessions in patients suffering from hydrocephalus of various types (both communicating and noncommunicating), origins, and stages of management (shunt or no shunt). Amplitude was calculated from ICP waveforms by using a spectral analysis methodology.

Results. The appearance of a pulse waveform amplitude is positive evidence of a technically correct recording of ICP and helps to distinguish between postural and vasogenic variations in ICP. Pulse amplitude is significantly correlated with the amplitude of cerebral blood flow velocity (R = 0.4, p = 0.012) as assessed using Doppler ultrasonography. Amplitude is positively correlated with a mean ICP (R = 0.21 in idiopathic normal-pressure hydrocephalus [NPH]; number of cases 131; p < 0.01) and resistance to cerebrospinal fluid outflow (R = 0.22) but does not seem to be correlated with cerebrospinal elasticity, dilation of ventricles, or severity of hydrocephalus (NPH score). Amplitude increases slightly with age (R = 0.39, p < 0.01; number of cases 46). A positive association between pulse amplitude and increased ICP during an infusion study is helpful in distinguishing between hydrocephalus and predominant brain atrophy. A large amplitude is associated with a good outcome after shunting (positive predictive power 0.9), whereas a low amplitude has no predictive power in outcome prognostication (0.5). Pulse amplitude is reduced by a properly functioning shunt.

Conclusions. Proper recording, detection, and interpretation of ICP pulse waveforms provide clinically useful information about patients suffering from hydrocephalus.

Key Words • pressure • intracranial pressure • pulse pressure waveform

Intracranial pressure is more than a number. Recorded ICP consists of a series of time-varying components derived from relatively fast changes in CBV. Pulsatile changes in arterial CBV evoke the pulse pressure waveform of ICP. Changes in venous CBV due to variations in intrathoracic pressures are responsible for the respiratory component of ICP. Slower, intrinsic vasomotor changes in CBV are responsible for waves classified as B, C, plateau, and other waves.

The intracranial pulse pressure waveform attracted the attention of many scientists approximately three decades ago and has maintained their interest until the present. Relatively early it was postulated that increased pulse pressure in ventricular CSF causes dilation of the ventricles. With the advent of dynamic MR imaging, pulsatile flow of CSF has been intensively studied. Most interest has been devoted to fast CSF flow through the aqueductus cerebri, reportedly increased in hydrocephalus. However, there are conflicting reports regarding its role in prognostication following shunting, both enthusiastically supportive and highly critical. Some interesting theories on the development of communicating hydrocephalus, although still awaiting conclusive documentation, are largely based on MR imaging studies related to pulsatile CSF flow.
Pressure recording does not require strong magnets or complex machinery. Although invasive, it remains a frontline method of examination for hydrocephalus in many hospitals worldwide. Studies on the pulse pressure waveform,\textsuperscript{1,12,20,24} pioneered the understanding of CSF dynamics; however, clinical use of the data is not firmly established. A new generation of clinical neuroscientists continue in this direction, recently publishing a number of promising studies.\textsuperscript{11,17,21}

We analyzed pulse pressure amplitude during CSF infusion studies\textsuperscript{7} or overnight ICP monitoring sessions in hydrocephalic patients with or without shunts. Our intention was to summarize our subjective experience with the interpretation of ICP pulse pressure amplitude in different scenarios, in a predominantly observational manner.

**MATERIAL AND METHODS**

Approximately 2100 clinical infusion studies were performed in 980 patients suffering from hydrocephalus of various types and origins: idiopathic NPH 47\%, post–subarachnoid hemorrhage NPH 12\%, noncommunicating hydrocephalus 22\%, and other 19\%. The mean age among patients was 65 years (range 24–94 years), and the male/female ratio was 2:1. All of the patients had attended the hydrocephalus clinic on the referral of the treating neurosurgeon, with evidence of ventricular dilation on brain

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**Fig. 1.** Left: Tracings demonstrating the principles used in calculating the pulse amplitude from time-domain (PPamp) and spectral-domain (AMP) analysis. Right: Graph demonstrating the results of comparing both methods in nearly 80 patients (ICP recorded after head injury) and showing an excellent linear relationship between amplitudes detected with both methods. b/m = beats per minute.

**Fig. 2.** Example tracings obtained during overnight monitoring of ICP using an intraparenchymal bolt in a patient with hydrocephalus. Initial ICP was approximately 4 to 8 mm Hg with a clear amplitude (AMP), which disappeared later, indicating that the transducer tip was relocated and probably exposed to atmospheric pressure because of loose bolt fixation. Further recording was unreliable. The transducer was reinserted and recordings from the following night indicated pathological increases in ICP up to 40 mm Hg. h = hours; m = minutes.
imaging (computed tomography or MR imaging) and clinical symptoms belonging to the Hakim triad. Given the nature of the patient selection, many had complex clinical problems. The group was evaluated using a constant-rate infusion study (lumbar 20%, preimplanted Ommaya reservoir 38%, shunt prechamber [G1] 40%, or open external ventricular drainage 2%) and/or overnight ICP monitoring in addition to the normal clinical and imaging assessments. Forty-four percent of the tests were performed in patients with the shunt in situ, to check its performance.

The infusion studies were performed via two lumbar needles, a shunt prechamber proximal to the valve, or a preimplanted ventricular access device. If lumbar access was required, lumbar needles (usually 21-gauge Whitacre) were used. For intraventricular access (reservoir or shunt prechamber), two needles (25-gauge butterfly) were inserted. One needle was connected via a stiff saline-filled tube to a pressure transducer and the other to an infusion pump mounted on a purpose-built trolley containing a pressure amplifier (Simonsen & Weel) and an IBM-compatible personal computer running ICM+ software (www.neurosurg.cam.ac.uk/icmplus). A strict aseptic technique was used to keep all the prefilled tubing and the transducer sterile. The skin was very carefully prepared with antisepctic solution.

After 10 minutes of baseline measurements, the infusion of normal saline or Hartmann solution was started at a rate of 1.5 ml/minute (or 1 ml/minute if the baseline pressure was higher than 15 mm Hg) and continued until a steady state ICP plateau was reached. If the mean ICP increased to more than 40 mm Hg, the infusion was stopped immediately. After ceasing the saline infusion, ICP was recorded until it returned to the previously recorded baseline level. All compensatory parameters were calculated using computer-supported methods based on physiological models of CSF circulation. Baseline ICP and CSF $R_{csf}$ (the latter calculated as the plateau ICP reached during the test minus the baseline ICP, divided by the infusion rate) characterize static conditions of CSF circulation. The elastance coefficient characterizes the ability of

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**Fig. 3.** Left: Tracings showing recorded pulse amplitudes of arterial blood pressure (ABP), ICP, and blood flow velocity (FV) in the MCA. Right: Graph demonstrating a weak but positive correlation between the ICP amplitude and the amplitude of blood flow velocity.

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**Fig. 4.** Example tracings revealing pulse amplitudes during an infusion study in a patient with NPH (a) and in another with brain atrophy (b).
the system to store an extra volume of fluid—a greater coefficient indicates that a smaller volume can be stored under the same incremental pressure conditions. During the infusion study, the ICP waveform was processed through a Fourier transform analysis to determine the pulse amplitude of ICP (AMP) as the magnitude (peak to peak) of the first harmonic component related to the heart rate. This method is an alternative to time-domain analysis, and in our experience, both methods (Fig. 1) are generally equivalent. During the study, ICP pulse amplitude or peak-to-peak pulse pressure amplitude (PPamp) increases with mean ICP. The rate of increasing amplitude per rise in ICP is called AMP/P slope, where P represents pressure.

RESULTS

The presence of a pulse amplitude proves that the ICP waveform is being properly transmitted to the transducer, whereas its absence suggests an invalid pressure recording (Fig. 2). The ICP pulse amplitude is time-synchronized with the pulse amplitude of arterial pressure and the pulse amplitude of blood flow velocity in the MCA (Fig. 3). In patients with NPH (subgroup of 41 patients, in whom simultaneous recordings with transcranial Doppler ultrasonography were performed), the correlation between ICP pulse amplitude and the amplitude of blood flow velocity (R = 0.4, p = 0.012) was stronger than that between pulse amplitude of ICP and that of arterial pulse pressure (R = 0.23). Pulse amplitude increased proportionally to the mean CSF pressure during the infusion study. In NPH this relationship was spectacular: the mean ICP pulse amplitude increased during infusion, and all slow vasoergic waves of mean ICP produced correlated variations in amplitude (Fig. 4a). In contrast, in patients with predominant brain atrophy, the rise in ICP pulse amplitude was very sluggish and vasoergic variations were absent (Fig. 4b).

In our material, pulse amplitude was not a strong predictor of outcome after shunting (compare our findings with those in the study by Eide and Brean). We insert a

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**Fig. 5.** Graph demonstrating pulse amplitudes in patients who did and those who did not improve after shunt insertion. N = number of patients; 17:33 = 17 patients did not have improvement in their condition, 33 did have improvement.

**Fig. 6.** Graph depicting the means and 95% confidence limits of pulse amplitudes among patients with idiopathic NPH either with or without evidence of cerebrovascular disease (CVD). 53:18 = 53 patients did not have evidence of CVD, whereas 18 did.

**Fig. 7.** Example tracings obtained during an infusion study, showing pulse amplitudes before (left) and after (right) shunt insertion.
shunt predominantly in patients with increased CSF $R_{\text{out}}$ (>13 mm Hg/ml/min). The relationship between the baseline amplitude and improvement (Stein–Langfitt score ≥ 1) in a subgroup of patients with idiopathic NPH and a stable follow-up assessment (50 patients) is presented in Fig. 5. The data in this plot suggest that when amplitude is large (> 2.5 mm Hg), improvement is very likely (> 90% patients improved). When the amplitude is less than 2 mm Hg, improvement is as likely as a lack of improvement. Pulse amplitude correlated rather weakly with other compensatory parameters assessed during the infusion study. Its correlations with ICP and $R_{\text{out}}$ were positive and significant (131 patients with idiopathic NPH; $R = 0.21$ for the correlation with mean ICP, and $0.22$ for that with $R_{\text{out}}$; $p < 0.01$). The correlation with the brain elastance coefficient (or pressure volume index) was insignificant.

In idiopathic NPH there was no evidence of any association between pulse amplitude and dilation of the ventricles. Pulse amplitude measured in the ventricles was slightly lower than that measured in the lumbar space. The mean values in idiopathic NPH were $2.5 \pm 1.8$ mm Hg (mean ± standard deviation; ventricles) and $2 \pm 1.4$ mm Hg (lumbar space).

There was no difference in the pulse amplitude between men and women. The pulse amplitude did increase slightly with age (46 patients, $R = 0.39$, $p < 0.01$), but did not show any correlation with the duration or severity of symptoms of NPH (the latter measured using the NPH scale). Among patients with idiopathic NPH, pulse amplitude was much lower in those without evidence of coexisting cerebrovascular disease than in those with clear signs of vascular problems (Fig. 6). Similarly, pulse amplitude was greater, although not spectacularly so, in patients with true idiopathic NPH than in those with post–head injury or any secondary form of NPH.

After shunt insertion, pulse amplitude decreased. Dif-
ferences were significant both at baseline and during the infusion study (Fig. 7). Among patients with shunts, pulse amplitude was lower in those with a normally functioning shunt than in those with a blocked shunt (0.95 ± 0.4 mm Hg compared with 1.95 ± 0.61 mm Hg; p = 0.000033). Therefore, a low amplitude can be taken as one of the markers of a patent shunt in cases in which shunt functioning must be tested. In patients with shunts and slit ventricles, the baseline pressure recorded from the shunt pre-chamber does not indicate any pulse waveform. Ventricle walls are collapsed around the catheter and there is no pressure transmission. Very often, the pulse waveform appears after infusion starts and the pressure builds up and opens up the ventricles (Fig. 8).

Whereas the mean ICP reacts potently to a change in posture—it usually becomes negative in patients in an upright body position—the pulse amplitude does not usually follow postural ICP variations as it does during ICP increases originated by volume load. Therefore, when interpreting overnight ICP recordings, the tracings of AMP are useful in distinguishing between changes in pressures of different origins (Fig. 9).

Although not documented in the literature, compartmentalization of pulse amplitude is possible. There are no or very few pressure gradients between ventricles and the cranial subarachnoid space in noncommunicating hydrocephalus. However, the difference in pulse amplitude was quite consistent; it was observed in six of eight patients in whom we recorded intraventricular pressure and cranial subarachnoid pressure by using a subdural drain. The pulse pressure in the ventricles seemed to be greater than in the subarachnoid space (Fig. 10).

The slope of the AMP/P line in our group analysis (28 patients) did not correlate with the magnitude or frequency of B waves, contrary to results reported by Lenfeldt and colleagues. Similarly, we could not confirm that B waves in the overnight recordings correlated with the CSF-Rout, as previously reported by Borgesen and Gjerris. The utility of B waves in prognostication in hydrocephalus should be studied further, preferably in a multicenter trial.

Pulse amplitude in ICP recording may be weak and noisy. Proper computer detection is helpful in most cases.

**DISCUSSION**

Intracranial pressure recordings and infusion studies are routinely used at our institution in patients with complex hydrocephalus. The usefulness of physiological measurements has been recently summarized in guidelines for the diagnosis and management of NPH. In contrast to CSF Rout, the clinical value of pulse amplitude is not supported by any randomized clinical trial. Our own experience indicates that pulse amplitude helps in the interpretation of recordings in cases of disturbed CSF compensation, shunt blockage, and slit ventricles. It also helps in distinguishing postural changes and vasogenic ICP waves. A low amplitude does not predict the absence of improvement following shunt insertion, but a large amplitude is associated with a positive shunt outcome. In our clinical material there was no evidence that an increased pulse amplitude is a factor promoting ventricular dilation; that is, there was no correlation between amplitude and ventricle width. Moreover, we do not interpret pulse amplitude alone as a single parameter influencing management. It is interpreted in conjunction with the clinical picture, neuroimaging evidence, neurophysiological test results, laboratory data, and other parameters describing CSF compensation (mean pressure, CSF Rout, vasogenic waves, elastance coefficient, and so forth).

**CONCLUSIONS**

Pulse amplitude of ICP is useful in the diagnosis of hydrocephalus. An increased pulse amplitude may be used to predict improvement after shunting, although the negative
Pulse pressure waveform in hydrocephalus

predictive power is low. A decreased pulse amplitude after shunting indicates a properly draining shunt.

Disclosure

The ICM+ software is licensed by the University of Cambridge (www.neurosurg.cam.ac.uk/icmplus). Both M.C. and P.S. have a financial interest in a part of the licensing fee.

References


Neurosurg. Focus / Volume 22 / April, 2007