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Citation for the published paper:

Qvarlander, S., Malm, J., Eklund, A. (2010)

"The pulsatility curve-the relationship between mean intracranial pressure and pulsation amplitude"

Physiological Measurement, 31(11): 1517-1528

URL: <http://dx.doi.org/10.1088/0967-3334/31/11/008>

Access to the published version may require subscription.

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The pulsatility curve – Relationship between mean intracranial pressure and pulsation amplitude

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Abstract

Amplitude of cardiac related pulsations in intracranial pressure has recently been suggested as useful for selecting patients for shunt surgery in hydrocephalus. To better understand how shunting affects these pulsations, we aim to model the relationship of mean pressure and pulsation amplitude in a wide range, including the low pressures typically found after shunt surgery. Twenty-five patients with probable INPH were examined with lumbar constant pressure infusion investigations including drainage of cerebrospinal fluid. Mean pressure and pulsation amplitude were determined for consecutive 1.5 second intervals, starting at peak pressure (ca 35 mm Hg), after infusion, continuing during spontaneous return to baseline and drainage to 0 mm Hg. The amplitude versus pressure relationship revealed a linear phase at higher pressures (14-32 mm Hg, lack of fit test: $p=0.79$), a transitional phase, and an essentially constant phase at low pressures (0-10 mm Hg, slope=-0.02, lack of fit test: $p=0.88$). Individual patients' baseline values were found in all three phases. The model and methodology presented in this paper can be used to preoperatively identify patients with potential for postoperative amplitude decrease and to predict how much the amplitude can be reduced.

Keywords: normal pressure hydrocephalus, pulse pressure, amplitudes, cerebrospinal fluid pressure, intracranial pressure, infusion test, arterial pulsations

All symbols and abbreviations are listed and explained in table 1.

1. Introduction

Idiopathic normal pressure hydrocephalus (INPH) is a syndrome characterized by enlarged cerebral ventricles and altered dynamics of the cerebrospinal fluid (CSF) system, although the exact nature of the disturbance is unknown (Malm and Eklund 2006). Gait disturbance, accompanied by cognitive decline and/or urinary incontinence are the primary symptoms (Relkin *et al* 2005). The treatment is implantation of a CSF shunt. A recent study (Brean and Eide 2008) on a Norwegian population determined that the prevalence of INPH was 21.9/100 000. While many INPH patients improve from shunt surgery, some do not, leading to the need for measurements that can predict which patients will benefit from the treatment (Marmarou *et al* 2005). Many so called predictive tests have been suggested, but their reliability is disputed, and none are universally accepted.

One commonly performed predictive test is a CSF infusion investigation (Eklund *et al* 2007). During the investigation intracranial pressure is continuously measured while the system is manipulated through infusion or drainage of Ringer solution. In the present study, focus was on the pressure pulsations related to the arterial heart pulsations. These pulsations have long been of interest to researchers who have investigated the amplitude and lag time, as well as the relation between the pressure pulsation waveform and the waveforms of cerebral blood pressure. It has even been suggested that enlarged pulsations may cause the ventricular enlargement seen in INPH (Egnor *et al* 2002, O'Connell 1943, Greitz 2004). The amplitudes of the pulsations have also been suggested as a predictive test (Eide and Brean 2006, Czosnyka *et al* 2008) but the reason behind this observation is still unknown. There are studies showing that both baseline pressure and pulsation amplitude at baseline are reduced after shunting (Petrella *et al* 2008, Eide and Sorteberg 2008). This leads to the hypothesis that there is an ideal pressure which corresponds to minimal pulsation amplitude. However, the widely accepted model of the CSF dynamics (Marmarou *et al* 1978), from which a linear relationship between pressure and pulsation amplitude can be derived (Avezaat and van Eijndhoven 1986), is empirically established only from resting pressure and up to increased pressure.

Postoperatively, it is expected that the new baseline pressure will be lower than the preoperative baseline pressure. Clinical studies have implied that the linear relationship is disrupted at lower pressures (Fridén and Ekstedt 1980, Szewczykowski *et al* 1977, Czosnyka *et al* 2004) and a secondary compensatory state with constant compliance has been suggested below this point (Szewczykowski *et al* 1977, Czosnyka *et al* 2004).

The aim of this study was to establish a model for how pressure and pulsation amplitude relate over a wide intracranial pressure range, including pressures above and below baseline intracranial pressure, by analysing infusion and tap tests performed on 25 patients diagnosed with probable INPH. A secondary aim was to determine where on this curve the individual patients have their baseline values, i.e. where their operating points are.

Table 1. All symbols and abbreviations in this paper are listed and explained.

Symbol/Abbreviation	Explanation
INPH	Idiopathic Normal Pressure Hydrocephalus
CSF	Cerebrospinal fluid
ΔP	Amplitude of intracranial pressure pulsation
P	Mean intracranial pressure (over 1.5 s interval)
P_0	Reference pressure
P_d	Pressure in the dural sinus
k	Elastance coefficient
ΔV	Intracranial volume change in one cardiac cycle
RPPC	Relative Pulse Pressure Coefficient
^{relax} (superscript)	Values determined from relaxation phase
^{infusion} (superscript)	Values determined from infusion phase
P_r	Baseline intracranial pressure
ΔP_r	Pulsation amplitude at P_r
_{i} (subscript)	Patient index
_{j} (subscript)	Index for each 1.5 second interval
$\overline{RPPC}$	Group average of RPPC
$\overline{P_0}$	Group average of P_0
$\widetilde{\Delta P}$	Rescaled ΔP
$\tilde{P}$	Translated P
$\overline{\widetilde{\Delta P}}$	Group average of $\widetilde{\Delta P}$
$\overline{\tilde{P}}$	Group average of $\tilde{P}$
$\Delta P_{\min}$	Minimal pulsation amplitude
$P_{\Delta P_{\min}}$	Pressure at $\Delta P_{\min}$

2. Materials and methods

2.1. Patients

Twenty-five patients (mean age 74 years, 48% women) diagnosed with “probable INPH” (Relkin *et al* 2005) were included. All patients were investigated with an infusion test which ended with a tap test (as described below). This study was approved by the regional ethical review board.

2.2. Infusion tests

Infusion tests were carried out at Umeå University Hospital with an in-house developed infusion apparatus using automated infusion protocols. The examination (figure 1) included a registration of baseline pressure at rest (approximately 20 minutes) and a constant pressure infusion protocol (Andersson *et al* 2005) with six pressure levels (each 7-10 minutes long and approximately 3 mm Hg above the previous) resulting in a total pressure increase of about 18 mm Hg. The infusion was followed by a relaxation phase and at the end of the investigation a controlled drain of CSF (tap test) was performed, allowing us to sample the amplitudes at pressures down to 0 mm Hg.

The pressure measurements were performed using two pressure transducer (PMSET 1TNF-R, BD Critical Care Systems Pte Ltd, Singapore) connected via fluid filled catheters to two needles inserted into the L3-4 interspace of the patient. The needles were inserted while the patient was in a sitting position, but the infusion test was carried out with the patient in the supine position. The zero-pressure reference level of the infusion apparatus was adjusted to the centre of the auditory meatus of the supine patient. One of the needles was used for infusion and the other for the pressure registration.

Figure 1. A lumbar constant pressure infusion protocol includes a registration of baseline pressure (I), CSF sampling while the patient is sitting up (II), a constant pressure infusion with 6 levels, a relaxation phase (IV) and a tap test phase (V).

Pressure data during the infusion test was sampled with a rate of 100 Hz. The first version of the infusion apparatus software used an on-line analysis of data where 100 samples were evaluated at a time. If a complete heart cycle could be detected within these 100 samples, only the samples corresponding to this heart cycle were stored. This approach ensured that the complete heart cycles needed for the current study were stored, but had a disadvantage of giving concatenated 100 Hz data sets for subjects with heart rates above 60 bpm. This version was used for the first 16 patients. In an updated version of the apparatus, which was used for the final nine subjects, all 100 Hz data points were saved, resulting in a continuous time series. Feasibility for both types of data-sets for use in the pulsation analysis was confirmed against manual evaluation.

2.3. Post-processing of data

The pressure data was post-processed in MATLAB (Version 7.4.0.287, The MathWorks Inc.) using specifically developed algorithms. In order to isolate the cardiac related pulsations the signal was filtered with a high pass filter of the Butterworth type (10th order, zero phase shift), with a cut-off

frequency of 0.5 Hz. This filtered out pressure variations due to e.g. breathing and vasogenic activity. The pump used to infuse and drain CSF sometimes induced a high frequency periodic variation in the pressure signal; this noise was reduced with a low pass filter of the above mentioned type, with a cut-off frequency of 10 Hz.

Due to the concatenated state of 16 of the data sets, designing a robust algorithm to isolate individual heart beats was not trivial. Therefore the alternative approach of calculating one value of pulsation amplitude and average pressure for each consecutive 1.5 s interval was chosen. The length interval was chosen to ensure that at least one full heart cycle was always included in the interval. Amplitudes (ΔP) were calculated as the difference between the maximum and minimum pressure in each time interval. Mean pressures (P) were determined as the mean value of the unfiltered pressure in the intervals, as the slow pressure variations must be included and the effect of the pump related disturbances on the mean values was negligible.

2.4. Model development

The most established way of modelling the relationship between intracranial pressure (P) and pulsation amplitude (ΔP) is derived from the Marmarou model (Marmarou *et al* 1978), extended with a term representing a reference pressure, P_0 (Avezaat and van Eijndhoven 1986). P_0 is suggested to relate to the pressure in the dural sinuses (P_d) (Avezaat and van Eijndhoven 1986). With the assumption that the pulsatile intracranial volume change due to pulsating arterial blood flow is constant over time and pressure independent this model yields a linear equation (Avezaat and van Eijndhoven 1986):

$$\Delta P = [\exp(k \cdot \Delta V) - 1](P - P_0) \quad (1)$$

In this equation k is the elastance coefficient and ΔV the intracranial volume change over one cardiac cycle due to the pulsating arterial blood flow. The term $[\exp(k \cdot \Delta V) - 1]$ is referred to as the Relative Pulse Pressure Coefficient (RPPC) (Lenfeldt *et al* 2004), and can be interpreted as an indicator of intracranial compliance. This linear relationship, which has been empirically established from resting

pressure up to a pressure of 41 mm Hg (Avezaat *et al* 1979, Avezaat and van Eijndhoven 1986, Lenfeldt *et al* 2004) lays the foundation for the model development of this study.

2.5. Determination of RPPC and P_0

Using data from the relaxation phase of the infusion test, a least square fitted linear regression was performed for all pairs of P and ΔP corresponding to pressures at least 3 mm Hg above baseline pressure. The slope of this linear regression is an estimation of $RPPC^{relax}$, while the intercept of the regression line with the P -axis is an estimation of P_0^{relax} .

The infusion phase of the data consisted of steady state levels (figure 1), which offered the possibility of averaging over these levels in order to reduce the influence of artefacts due to e.g. coughing, patient movement (Wahlin *et al* 2010). Therefore, a single value of P was determined for each steady state level of the constant pressure infusion phase; these values were calculated as the median of the unfiltered pressure. Similarly, values of ΔP were determined as the median value of the calculated amplitudes for each steady state level. A least square fitted linear regression was performed on the median values of P and ΔP from the infusion levels in order to calculate $RPPC^{infusion}$ and $P_0^{infusion}$. This approach was not feasible when the pressure was changing continuously, such as during the relaxation phase.

From the last five minutes of the baseline pressure phase a median value of P (P_r) was determined and average baseline pulsation amplitude (ΔP_r) was determined as the median value of the calculated amplitudes in the same time interval.

2.6. Individual scaling and translation

In order to establish a model based on the entire group (Andersson *et al* 2008), the data from individual patients were rescaled and translated. The purpose of this was to remove inter patient variability with respect to k , ΔV and P_0 from the individual P versus ΔP curve, while maintaining the general shape of the curve. For each patient the ΔP values were rescaled according to:

$$\widetilde{\Delta P}_{ij} = \Delta P_{ij} \times \overline{\text{RPPC}} / \text{RPPC}_i . \quad (2)$$

The P values were translated according to:

$$\tilde{P}_{ij} = P_{ij} - (P_{0i} - \overline{P_0}) . \quad (3)$$

The index i represents individual patients, while j represents individual values from each 1.5 second interval. $\overline{\text{RPPC}}$ and $\overline{P_0}$ were the mean RPPC and P_0 calculated from all patients. With these two steps the linear part of each P versus ΔP curve, corresponding to increased pressure, was shifted to follow a line with an expected slope equal to the average RPPC and an expected intercept with the pressure axis at the average P_0 . Figure 2 illustrates the procedure. The relaxation and drain values were rescaled and translated using $\text{RPPC}^{\text{relax}}$ (in equation 1) and P_0^{relax} (in equation 2). In order to accurately plot baseline values with the relaxation and drain values, and determine the baseline operating point of each patient, we also rescaled and translated ΔP_r and P_r . These values were rescaled and translated using $\text{RPPC}^{\text{infusion}}$ (in equation 1) and P_0^{infusion} (in equation 2) in order to ensure that rescaling and translation of all data points were performed with the most appropriate RPPC and P_0 values.

Figure 2. In order to determine an appropriate model for the entire group of 25 patients the data of each patient was rescaled (ΔP , step II) and translated (P , step III), using their individual regression parameters ($RPPC_i$ and P_{0i} respectively, calculated in step I). After this procedure the values of each patient followed the same reference line (dashed) at higher pressures. The reference line had a slope equal to the average $RPPC^{relax}$ and intercepted the pressure axis at the average P_0^{relax} .

2.7. Merging into a group ΔP versus P curve

To facilitate averaging on the group level each patient's $\tilde{P}$ values from the relaxation phase and tap test phase were divided into pressure intervals ranging 2 mm Hg. The lowest interval started at -1 mm Hg and the highest ended at 35 mm Hg (translated pressures). For each pressure interval the median of the $\tilde{P}$ values and the median of the corresponding $\tilde{\Delta P}$ values were determined. The group means of the median $\tilde{P}$ and $\tilde{\Delta P}$ for each pressure interval, denoted $\bar{\tilde{P}}$ and $\bar{\tilde{\Delta P}}$, were then calculated. For each patient we also determined the minimal ΔP and the corresponding P , denoted ΔP_{min} and $P_{\Delta Pmin}$.

2.8. Statistics

PSAW Statistics (Release 18.0.1, SPSS Inc.) was used to calculate all statistics not included in the MATLAB algorithm. Correlations between parameters were determined as bivariate Pearson's

correlation coefficients. Parameter means were compared using paired-samples Student's t-tests. The relationship between ΔP and P was analysed using linear regression and F-tests for lack-of-fit. Statistical significance was set at $p < 0.05$.

3. Results

Figure 3 shows the relationship between $\overline{\Delta P}$ and $\overline{P}$ for the 25 patients. While the relationship of $\overline{\Delta P}$ and $\overline{P}$ was linear (linear regression slope=0.61, $p < 0.01$, lack-of-fit test: $p = 0.79$) at higher pressures (14-32 mm Hg), the curve entered a transitional phase and levelled out as it approached P_0^{relax} . Below a pressure of 10 mm Hg there was essentially no change in the pulsations (figure 3), although an apparent trend towards higher pulsations at lower pressures was observed (linear regression slope= -0.02, $p = 0.10$, lack of fit: $p = 0.88$). The highest $\overline{P}$ value was excluded from linear regression analysis due to the very large standard deviation of the corresponding $\overline{\Delta P}$ (SD=1.64 mm Hg). The baseline values of some patients were in the upper, linear phase, while some patients had baseline values in the transitional or constant phase of the curve (figures 3 and 4).

Figure 3. Calculated from the 25 patients the figure shows the relationship between $\bar{\Delta P}$ and $\bar{P}$ (mean values, circles) with confidence intervals ($1.96 \cdot \text{SD}$, dotted lines). The relationship showed two distinct phases and could be modelled as linear (solid line) at high pressures and constant (dashed line) at low pressures, with the transition between the phases occurring close to P_0^{relax} . The dotted curly bracket marks the range of individual translated baseline pressures ($\tilde{P}_r$) and the X on the pressure axis marks the mean translated baseline pressure ($\bar{\bar{P}}_r$).

Figure 4. Some patients had operating points (baseline pressure) in the pressure independent phase of the ΔP versus P curve (a), with no expected reduction of amplitudes if the pressure is lowered. Other patients had operating points in the linear phase of the curve (b), with a potential amplitude decrease if the pressure is lowered. Note that the $\tilde{\Delta P}_r(\tilde{P}_r)$ points estimated before the infusion, and rescaled with $RPPC^{\text{infusion}}$ and P_0^{infusion} , do not deviate far from the curves obtained from the relaxation and drain phase. Note also that the transition phase, while not very evident in (a), was readily apparent in (b) and was not an artefact produced by the averaging of group data.

The group mean of P_r was 12.26 ± 1.87 mm Hg (mean $\pm$ SD, $N=25$), and the mean ΔP_r was 2.46 ± 1.39 mm Hg. The estimated $\Delta P_{\min}$ was 1.09 ± 0.42 mm Hg and $P_{\Delta P_{\min}}$ was 7.15 ± 2.94 mm Hg. The differences between ΔP_r and $\Delta P_{\min}$, in other words the potential decreases in pulsation amplitude, are illustrated by a histogram in figure 5. There was a high correlation between ΔP_r and the potential decrease in amplitudes ($R=0.96$, $p<0.01$), this is illustrated in figure 6. The potential decrease in pulsation amplitude also showed a significant correlation with the difference between P_r and P_0^{infusion} ($R=0.80$, $p<0.01$).

Figure 5. The potential amplitude decrease ranged from 0 mm Hg to just under 5 mm Hg for individual patients. The distribution was strongly skewed towards lower values, but over half of the patients (56%) had a potential decrease of over 1 mm Hg, which was 41% of the average ΔP_r .

Figure 6. ΔP_r showed a strong correlation ($R=0.96$) with the potential amplitude decrease, as illustrated by the linear regression line.

There was no statistically significant difference ($p=0.56$) between $RPPC^{\text{infusion}}$, 0.61 ± 0.16 , and $RPPC^{\text{relax}}$, 0.60 ± 0.12 . Neither was there any statistically significant difference ($p=0.21$) between

P_0^{infusion} , 8.7 ± 4.1 mm Hg, and P_0^{relax} , 9.4 ± 3.1 mm Hg. The standard deviation of the difference was 0.09 for RPPC values and 2.8 mm Hg for P_0 values.

4. Discussion

4.1. Pulsatility curve - model of the relationship between P and ΔP

This study empirically establishes a model for the relationship between P and ΔP in a wide pressure range, both above and below baseline pressure, and presents a methodology for assessing this relationship in individual patients. The main findings were that the relationship was linear at higher pressures and essentially constant at lower pressures, with a transition phase in between. We will denote this three-phase ΔP versus P curve the pulsatility curve.

The pulsation amplitudes are affected by the compliance of the system and as such the observation of two distinct phases in the pulsatility curve should reflect an existence of two different phases or states of intracranial compliance, as well as a transitional phase between these two. Our results are supported by findings in ten patients without intracranial pathology (Szewczykowski *et al* 1977), although no transitional phase was identified in that study. The Marmarou model is only valid for the upper, linear phase (Czosnyka *et al* 2004, Juniewicz *et al* 2005) and should according to the current study only be assumed for pressures above the transitional phase. This could be of importance when measuring outflow resistance and compliance with dynamic infusion methods like the bolus method, which assume that the starting pressure is in the linear phase, since the starting pressure can be in the transition phase or even in the constant phase (figure 4).

The position of P_r on the pulsatility curve reveals in what phase the CSF system is currently working, i.e. the operating point of the system. Establishing the operating point for individual patients, by determining P_r as well as the pulsatility curve in a wide pressure range, should help to determine if compliance can be increased by lowering P_r . Patients with their operating point in the linear phase could expect increased compliance with a decreased P_r , while an operating point in the essentially constant phase implies that lowering P_r should leave compliance unaltered or even somewhat reduced. An operating point in the transitional phase leaves the effect of decreased P_r harder

to predict, but should offer a possibility for at least a slightly raised compliance. As described in this study measurement of this curve can be performed with an infusion test, as long as the equipment can be used to withdraw as well as infuse CSF and to simultaneously record pulse pressure amplitude. We found that the operating points of individual INPH patients could be identified in all three phases (figure 3).

An alternative approach to approximate the position of the operating point, if drain data is not available, is to use the difference between P_r and P_0 . It provided a reasonable estimation of how close to the constant phase a patient's operating point was. A large difference implied that the operating point was in the linear phase, with a large potential for pulsation decrease, while a small difference implied the opposite.

While the pulsatility curve and operating point provides information about which state of compliance the CSF system is currently working in, absolute values of compliance, or the elastance coefficient (k), can only be determined from this data if the cardiac related change in intracranial blood volume (ΔV) is known. This can be achieved using phase contrast magnetic resonance imaging (Wahlin *et al* 2010). Even without determining absolute values however, relative changes in compliance should be observable using the methodology presented in this paper, as long as the ΔV can be assumed to remain unchanged.

4.2. Shunting of the CSF system

The pulsatility curve offers a possibility for predicting how the pulsation amplitudes will be affected by shunt surgery, which is expected to lower the baseline pressure, i.e. move the operating point to the left in the pulsatility curve (figure 4). The individual minimum amplitude and corresponding pressure can be identified in each INPH patient and the potential amplitude decrease determined as the difference between the baseline and the minimal amplitude value (figure 5). This model and methodology can be used to predict if, and how much, ΔP can be influenced by shunt surgery, as well as how to maximize this effect with appropriate shunt settings.

The pulsatility curve implies that there is no gain in aiming for a maximal decrease in pressure, as reduction beyond a certain point would increase the amplitudes rather than reduce them further (figure 3). Setting shunts to very low opening pressures entails the risk of overdrainage, which can cause intracranial bleeding (Boon *et al* 1997).

4.3. Predictive tests

Assuming that the yet unproven hypothesis that pulsation amplitude is a major pathophysiological property of INPH (Egnor *et al* 2002, O'Connell 1943, Greitz 2004) proves valid, this model could possibly be used as a predictive test for outcome from shunt surgery.

Let us reflect on how current predictive methods relate to the pulsatility curve. According to the Davson equation (Davson *et al* 1973) a change in outflow resistance will affect the operating point. An increased outflow resistance raises the baseline pressure and thus shifts the operating point to the right. The RPPC describes the steepness of the linear phase of the curve, and when the operating point is in the linear phase, it is a major contributor to how much shifting the operating point can influence the amplitudes. According to the pulsatility curve and operating point concept, RPPC and outflow resistance independently affect how the compliance can be altered by a shunt. An elevated outflow resistance has been shown to have prognostic power for selecting shunt responders (Boon *et al* 1997), but there are also studies showing a lack of prognostic power (Malm *et al* 1995). Similarly, one study (Anile *et al* 2010) showed a good prognostic power for a high intracranial elastance (IE) slope, which is closely related to RPPC, while another study demonstrated a lack of prognostic power for the slope of pulsation amplitude versus pressure (Delwel *et al* 2005). These inconclusive results concerning the predictive power may be explained by these parameters independently providing only partial information, which suggests a need to establish a more extensive view of the CSF system and its operating point in order to predict outcome from shunt surgery.

The range of $\Delta P_{\min}$ in this study was small compared to the range of ΔP_r , which lead to the close correlation between ΔP_r and the potential decrease in amplitudes (figure 6). Evidence has previously been presented (Eide and Brean 2006, Eide and Sorteberg 2008, Eide and Brean 2010, Czosnyka *et al* 2008, Petrella *et al* 2008) for the suggestion that large amplitudes are a good predictor

of positive outcome from shunting. This supports our idea that determining the potential amplitude decrease may be a way to identify shunt responders. Czosnyka et al. also observed that low pulsation amplitudes were not a good predictor of poor outcome from shunting, as some patients with low amplitudes still improved. In the context of the model presented in this study these patients may still have a potential for reduction of amplitudes due to having even smaller minimal amplitudes.

4.4. Stability of measured parameters

The results of this study are based on measurements made during infusion studies including a tap test phase, which entail significant external influence on the system. The assumption was made that our findings about the CSF physiology are valid, even though the process of measurement was quite different from any physiological situation. The comparison of $RPPC^{infusion}$ and $P_0^{infusion}$ with $RPPC^{relax}$ and P_0^{relax} was performed as an attempt to validate this assumption. The good agreement between $RPPC^{infusion}$ and $RPPC^{relax}$ implies that the elastance coefficient (k) and the cardiac related volume change (ΔV) are stable during external influence on the CSF system. Although $P_0^{infusion}$ and P_0^{relax} were not significantly different, the relatively large standard deviation of their differences implies that P_0 is a less stable parameter. This indicates that P_0 was influenced by physiological reactions to the unphysiological external manipulation, possibly through variations in the venous pressure. The use of the model developed in this study as a way to predict shunt response, or determine an appropriate opening pressure for a shunt, is based on the assumption that the P_0 and $RPPC$ parameters remain unaffected after a shunt is inserted. For $RPPC$ this is likely, as shunts in their current design add very little to k , instead improving compliance through a lowering of the intracranial pressure, and should not change ΔV . The effect on P_0 , however, needs to be further investigated. We plan to investigate the stability of both $RPPC$ and P_0 after shunt surgery in a future study which will follow up the 25 patients of this study postoperatively.

5. Conclusions

Two distinct phases, as well as a transitional phase, were observed in the relationship between ΔP and P ; a close to pressure independent phase at low pressures gradually transitioned into a linear phase at higher pressures. The transitional phase appears close to the reference pressure P_0 . The pulsatility curve and methodology can be used to identify patients with a potential for amplitude decrease and to predict how much the amplitudes can be reduced by inserting a shunt with an appropriate, and individually adjusted, opening pressure. The adjustable shunts available today offer the possibility of validating this model in practice, and evaluating its potential as a predictive test in light of the yet unproven hypothesis that pulsation amplitudes are a component of the INPH pathophysiology.

Acknowledgement

This project was financed by VR, VINNOVA and the SSF through their common initiative: “Biomedical engineering for improved health”, as well as by the EU through ERDF: Objective 2, Northern Sweden.

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