

Clinical Article

Intracranial pulse pressure amplitude levels determined during preoperative assessment of subjects with possible idiopathic normal pressure hydrocephalus

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Received June 2, 2006; accepted August 3, 2006; published online October 16, 2006

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Summary

Background. It was previously reported that the intracranial pulse pressure amplitudes were elevated in idiopathic normal pressure hydrocephalus (iNPH) patients responding to shunt surgery. In this study, pulse pressure amplitudes were determined in all patients referred for tentative iNPH, and patients were selected for shunt surgery based on the determination of their threshold levels of intracranial pulse pressure amplitudes.

Patients and methods. All patients referred to our department for tentative iNPH during a 12 months time period were included. Using intracranial pressure (ICP) monitoring the intracranial pulse pressure amplitudes were determined as the mean wave amplitude in consecutive 6-seconds time windows. Intracranial pulse pressure amplitudes were defined as being elevated when the mean wave amplitudes were either ≥ 4 mmHg in $\geq 70\%$, ≥ 5 mmHg in $\geq 40\%$ or ≥ 6 mmHg in $\geq 10\%$ of the ICP recording time. Shunt treatment was offered to those with elevated mean wave amplitudes. Clinical state was assessed by using a NPH Grading Scale and the Stein-Langfitt scale before ICP monitoring, and then repeated after 12 months.

Results. Among the 40 iNPH patients included during the 12 months period, the mean wave amplitudes were elevated in 24 patients (60%), while not being elevated in 16 (40%). Neither pre-operative clinical state, radiological ventricular size nor co-morbidity differed between patient groups with elevated or non-elevated mean wave amplitudes. In the shunted patients who had pre-operatively elevated mean wave amplitudes, 91% had very significant clinical change after 12 months (median change in NPH score +4). In those with non-elevated amplitudes and no shunt, clinical state was somewhat worse after 12 months (median change in NPH score -1).

Conclusions. In this one-year material, mean wave amplitudes were elevated in 60% of iNPH patients. In those with elevated mean wave amplitudes who were treated with shunt, 91% had a significant clinical response.

Keywords: Idiopathic normal pressure hydrocephalus; extra-cranial shunts; outcome; intracranial pressure; single pressure wave parameters.

Introduction

During preoperative assessment of patients with tentative idiopathic normal pressure hydrocephalus (iNPH) patients, testing cerebrospinal fluid (CSF) hydrodynamics can increase the predictive accuracy for shunt response [2, 5–7, 13–18].

Using intracranial pressure (ICP) monitoring, we recently reported that intracranial pulse pressure amplitudes were elevated in those iNPH patients improving following shunt surgery [9]. When the mean ICP wave amplitudes (i.e. pulse pressure amplitudes) were ≥ 4 mmHg in 70% of time, ≥ 5 mmHg in 40% of time, or ≥ 6 mmHg in 10% of time, the positive and negative predictive values for a significant shunt response were 82–90 and 91–100, respectively. The present study addressed the frequency of iNPH patients who presented with such elevated pulse pressure amplitude levels, and the clinical improvement in those selected for a shunt based on these pulse pressure amplitude levels. For this purpose, all patients referred to our department for tentative iNPH during a 12 months time period were included, and the proportion of patients with increased mean wave amplitude levels was determined. Patients were selected for shunt surgery by determination of the mean wave amplitudes threshold levels.

Patients and methods

Patients

All patients examined for tentative iNPH during a 12 months time period (from April 2004 to March 2005) within the Department of

Table 1. Pre-operative data at the time of ICP monitoring and changes in Stein-Langfitt and NPH scores after 12 months

	All patients	Group A	Group B	Statistics	
		Non-elevated mean wave amplitudes	Elevated mean wave amplitudes	Significance	Test
<i>Demographic data</i>					
Number (%)	40	16 (40%)	24 (60%)		
Sex					
– Female	19 (52.5%)	9 (56%)	10 (42%)	ns	Chi square
– Male	21 (47.5%)	7 (44%)	14 (58%)		
Years of age	75 (60–84)	74 (60–84)	75 (66–83)	ns	ANOVA
Duration of symptoms	2 (0.5–6)	2 (0.5–5.5)	2.3 (0.8–6)		
<i>ICP recordings</i>					
Recording time (hrs)	14.2 (5.8–22.4)	13.8 (5.8–22.4)	15.0 (7.1–21.5)	ns	ANOVA
Mean ICP (mmHg)	6.9 (–3.3–16.2)	3.2 (–3.3–15.0)	8.0 (–0.9–16.2)	$P=0.008$	ANOVA
Mean wave amplitude (mmHg)	4.8 (2.2–11.5)	3.3 (2.2–4.3)	5.6 (4.5–11.5)	$P<0.001$	ANOVA
<i>Pre-operative clinical state</i>					
Stein-Langfitt	2.5 (1–5)	2 (1–4)	3 (1–5)	ns	ANOVA
NPH score	10 (4–14)	10.5 (7–14)	9.5 (4–14)	ns	ANOVA
– Gait sub-score	3 (1–4)	3 (2–4)	3 (1–4)	ns	ANOVA
– Incontinence sub-score	3 (1–5)	3 (1–5)	3 (1–5)	ns	ANOVA
– Dementia sub-score	3 (1–5)	4 (3–5)	3 (1–5)	$P=0.03$	ANOVA
<i>Pre-operative radiology</i>					
Evan's index	0.34 (0.21–0.46)	0.34 (0.28–0.41)	0.36 (0.21–0.46)	ns	ANOVA
Third ventricle index	0.09 (0.02–0.14)	0.09 (0.04–0.12)	0.09 (0.02–0.14)	ns	ANOVA
Cella media index	0.32 (0.11–0.8)	0.29 (0.22–0.35)	0.32 (0.11–0.8)	ns	ANOVA
Ventricular score	97 (44–129)	94 (78–113)	97 (44–129)	ns	ANOVA
<i>Pre-operative co-morbidity</i>					
Cerebrovascular disease; N (%)	19 (48%)	6 (38%)	13 (54%)	ns	Chi square
Cardiovascular disease; N (%)	12 (30%)	4 (25%)	8 (33%)	ns	Chi square
Arterial hypertension; N (%)	17 (43%)	6 (38%)	11 (46%)	ns	Chi square
Vertebrogenic or lower extremity disease; N (%)	8 (20%)	2 (13%)	6 (25%)	ns	Chi square
Other chronic diseases	23 (58%)	9 (56%)	14 (58%)	ns	Chi square
<i>Post-operative clinical change (12 months)</i>					
Change Stein-Langfitt score	1 (–3–4)	–1 (–1–1)	1 (–3–4)	$P<0.001$	ANOVA
Change NPH score	2 (–3–7)	–1 (–3–1)	4 (–3–7)	$P<0.001$	ANOVA
– Change Gait sub-score	1 (–2–3)	0 (–1–1)	1 (–2–3)	$P<0.001$	ANOVA
– Change Incontinence sub-score	0 (–2–3)	0 (–2–1)	1 (–1–3)	$P<0.001$	ANOVA
– Change Dementia sub-score	0 (–1–2)	0 (–1–0)	1 (–1–2)	$P<0.001$	ANOVA

Data presented either as medians with ranges in parenthesis or as numbers with percentages in parenthesis. ^a Differences between groups (A vs. B) determined either using analysis of variance (ANOVA) or Chi square test [*ns* non-significant, $P>0.5$].

Neurosurgery, The National Hospital, Oslo, Norway, were included. They followed a standardized pre- and post-operative protocol used for years in this department [7, 18]. The study was approved by the hospital authorities.

Clinical and radiological assessment

A NPH Grading Scale [9] was used for clinical assessment of the severity of gait disturbance, urinary incontinence and dementia. This NPH Grading Scale has 15 scores, ranging from the best score of 15 to the lowest score of 3. In addition, clinical grading was done using the Stein-Langfitt scale [19]. Clinical assessment was made before the ICP monitoring, and then again repeated 12 months after ICP monitoring/shunt surgery.

The presence of co-morbidity was identified before the ICP monitoring (Table 1).

Ventricular size before ICP monitoring was assessed using linear measures based on cerebral computer tomography (CT) or magnet resonance image (MRI) scanning [8, 9].

Pre-operative ICP analysis

Continuous ICP monitoring was done using a solid sensor (Codman MicroSensor™, Johnson & Johnson, Raynham, MA, USA); the ICP signals were sampled at 100 or 200 Hz and analyzed as previously described [9, 10]. For every 6-seconds time window, both mean ICP and mean ICP wave amplitude were determined; the mean wave amplitude being a mean value of the pulse pressure amplitudes during a time window [9, 10]. For each ICP recording we then determined the average values of mean ICP and mean wave amplitude, and the percentage of time with mean wave amplitudes being ≥ 4 , ≥ 5 , or ≥ 6 mmHg.

On the basis of a previous study [9], *non-elevated* intracranial pulse pressure amplitudes were defined as follows: Mean ICP wave amplitudes being either ≥ 4 mmHg in $<70\%$ of time, ≥ 5 mmHg in $<40\%$ of time, or ≥ 6 mmHg in $<10\%$ of time. *Elevated* intracranial pulse pressure amplitudes were defined as follows: Mean ICP wave amplitudes being either ≥ 4 mmHg in $\geq 70\%$ of time, ≥ 5 mmHg in $\geq 40\%$ of time, or ≥ 6 mmHg in $\geq 10\%$ of time.

Criteria for shunt treatment

The patients were referred to our department from different neurological departments based on tentative iNPH because of their clinical state (gait disturbance, urinary incontinence and/or dementia) and radiological evidence of ventriculomegaly. In our department the criteria for shunt treatment were mean ICP wave amplitudes being either ≥ 4 mmHg in $\geq 70\%$ of time, ≥ 5 mmHg in $\geq 40\%$ of time, or ≥ 6 mmHg in $\geq 10\%$ of ICP recording time. These threshold values were derived from a previous study of iNPH [9]; in this study these threshold values had positive and negative predictive values for a significant shunt response of 82–90 and 91–100, respectively.

A ventriculo-peritoneal (VP) shunt was implanted about 2 weeks after the ICP monitoring, using an extra-cranial HAKIM™ Programmable Valve Shunt System (Codman & Shurtleff Inc., Medos S.A. Rue Girardet 29, CH 2400 Le Locle, Switzerland). The opening pressure was set at 10–12 cm H₂O in 22 of 24 cases (ranges 10–14 cm H₂O).

Statistics

Statistical analyses were performed in SPSS, version 12.0 (SPSS Inc., Chicago, IL). Comparisons between groups were performed by one-way ANOVA with Bonferroni corrected post hoc tests. Chi square test was used to examine differences between percentages of occurrences.

Results

Patients and surgical results

During the 12 months time period we included 40 patients with tentative iNPH who had their ICP monitored (Table 1).

In 16 patients (40%) we found *non-elevated* pulse pressure amplitudes (i.e. mean wave amplitudes being either ≥ 4 mmHg in $< 70\%$ of time, ≥ 5 mmHg in $< 40\%$ of time, or ≥ 6 mmHg in $< 10\%$ of time); these 16 patients are referred to as Group A (Table 1).

In 24 patients (60%) the pulse pressure amplitudes were *elevated* (i.e. mean wave amplitudes being ≥ 4 mmHg in $\geq 70\%$ of time, ≥ 5 mmHg in $\geq 40\%$ of time, or ≥ 6 mmHg in $\geq 10\%$ of time); these 24 patients are referred to as Group B (Table 1).

The percentages of time windows with mean wave amplitudes either being ≥ 4 mmHg, ≥ 5 mmHg, or ≥ 6 mmHg for both Groups A and B are shown in Fig. 1.

Surgical complications to shunt treatment were observed in 17% of patients (i.e. shunt infection in one, subdural hematoma in one, combined shunt valve failure and abdominal hernia in one, and headache in another patient).

Pre-operative observations

As shown in Table 1 there were no differences between patients with either non-elevated (Group A) or elevated (Group B) intracranial pulse pressure amplitudes regarding age, duration of symptoms, NPH score, Stein-Langfitt grade, radiological linear measures of ventricular size, or the presence of co-morbidity (e.g. cerebrovascular disease, cardiovascular disease, hypertension). A rather high proportion of co-morbidity was found (Table 1).

Clinical change after 12 months

At follow-up after 12 months, 3 of 40 patients had died; the causes of death not being related to management of the iNPH in any of them. For the remaining

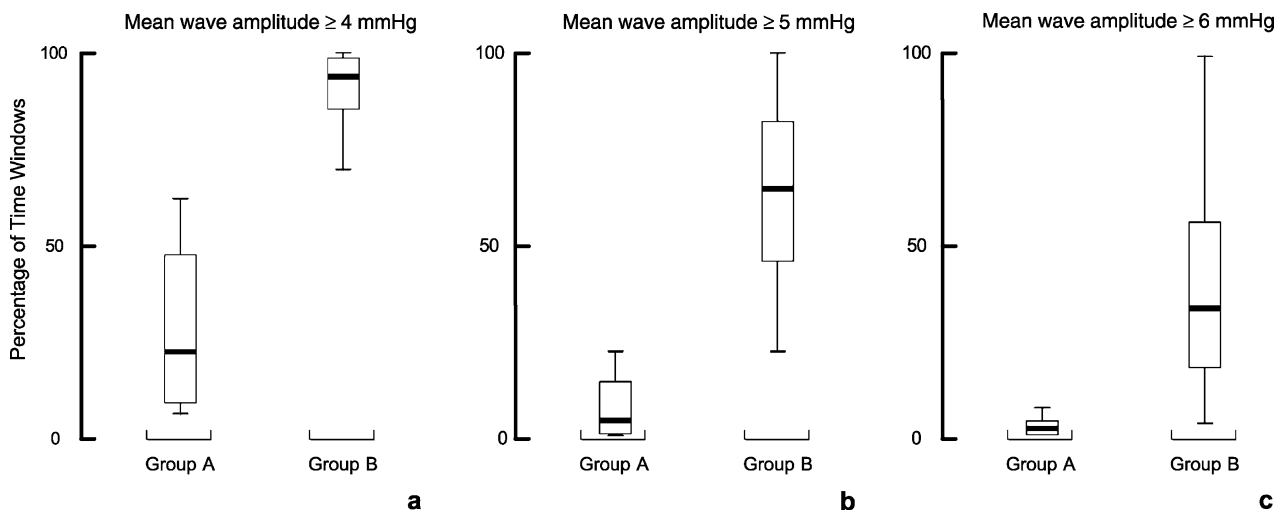


Fig. 1. Differences between patient groups that according to our criteria had non-elevated (Group A; 16 patients) or elevated (Group B; 24 patients) mean ICP wave amplitudes regarding percentage of time windows with mean ICP wave amplitudes (a) ≥ 4 , (b) ≥ 5 , or (c) ≥ 6 mmHg. Each box plot shows the median value (highlighted line within the box), the 25 percentile (lower end of the box), the 75 percentile (upper end of the box), and the ranges (lines from upper and lower ends of boxes)

14 patients with pre-operative *non-elevated* pulse pressure amplitudes (Group A) and the remaining 23 patients with pre-operative *elevated* pulse pressure amplitudes (Group B) the changes in clinical state after 12 months are presented in Table 1. In Group A the median reduction in NPH score was -1 and the median reduction in Stein-Langfitt grade -1 , while in Group B the median increase in NPH score was $+4$ and the median increase in Stein-Langfitt grade $+1$ (Table 1). Similar statistically significant differences were observed in those either not shunted or those shunted. In those not shunted the median reduction in NPH score was -1 and the median

reduction in Stein-Langfitt score -1 , while in those shunted the median increase in NPH score was $+4$ and the median increase of Stein-Langfitt score $+1$ (data not shown).

A more detailed description of the outcome categories is shown in Fig. 2a, b. In Group A one patient received a shunt since this was very strongly requested by her family, while in Group B two declined the offer of shunt surgery. The one patient in Group A receiving a shunt had an increase in NPH score of 1 (from 7 to 8); her family stated that her clinical change had been marginal. Her mean wave amplitudes were ≥ 4 mmHg in 53% of the time. The two patients in Group B rejecting shunt treatment showed a marked clinical worsening during the subsequent 12 months (reduction in NPH score of -3 and -2 , respectively); one of them becoming dependent within an institution.

Among those 22 patients in Group B who were shunted, one patient died before the 12 months control; at control before death his clinical improvement had been significant (change in NPH score of $+3$). Hence, in those 21 patients in Group B who could be assessed after 12 months; the effect of shunt treatment was very significant in 19 (91%). Three of these 19 patients (16%) improved from dependency to independency, and were transferred from institution to home. Among the 2 of 21 patients with a non-significant effect, one had a six-year history of iNPH and showed a minor clinical improvement (increase in NPH score $+2$). The other showed no clinical change; he was diagnosed with progressive supranuclear palsy.

When comparing the percentages of time windows with mean wave amplitudes either being ≥ 4 , ≥ 5 , or ≥ 6 mmHg between those not shunted in Group A and those shunted in Group B, the differences were somewhat greater (data not shown) than the differences observed for all patients in Groups A and B (Fig. 1).

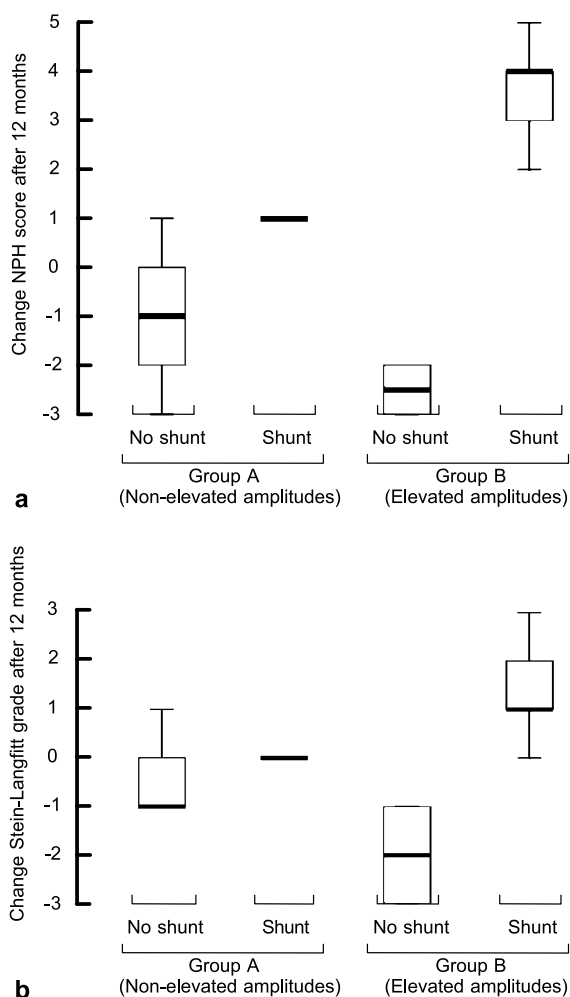


Fig. 2. The changes after 12 months in (a) NPH score and (b) Stein-Langfitt grade for patient groups that according to our criteria had non-elevated mean ICP wave amplitudes (Group A: No shunt 13 patients; Shunt 1 patient) or elevated mean ICP wave amplitudes (Group B: No shunt 2 patients; Shunt 21 patients). Each box plot shows the median value (highlighted line within the box), the 25 percentile (lower end of the box), the 75 percentile (upper end of the box), and the ranges (lines from upper and lower ends of boxes)

Discussion

Patients and surgical results

This material of 40 patients is rather small, but includes all patients referred to our department for tentative iNPH who also had their ICP monitored. We selected a 12 months time period for inclusion of new patients. The patients followed the pre- and postoperative protocol used for years in this department, except that patients now were selected for shunt surgery based on measurements of the mean wave amplitudes during ICP monitoring.

In this study we experienced surgical complications (infection, hematoma, abdominal hernia, valve failure) to the shunt implantation in 17% of the patients. This was somewhat higher than we previously observed [9]. However, our complication rate is lower than that reported in a literature review, wherein similar complications occurred in 38% [13].

ICP analysis

The method of computing the mean ICP wave amplitude has previously been described [10]; this parameter refers to an average of pulse pressure amplitudes over a time window of 6-seconds duration. It is a topic of discussion which intracranial pulse pressure amplitudes that may be regarded as *elevated* or *non-elevated*. The threshold levels used here were based on a previous study in this department [9]; retrospective analysis of ICP recordings showed that the presence of mean wave amplitudes ≥ 4 mmHg in 70% of time, ≥ 5 mmHg in 40% of time, or ≥ 6 mmHg in 10% of time had positive and negative predictive values for a significant shunt response of 82–90 and 91–100, respectively. Accordingly, in this study we defined mean wave amplitudes as elevated when they were either ≥ 4 mmHg in $\geq 70\%$ of time, ≥ 5 mmHg in $\geq 40\%$ of time, or ≥ 6 mmHg in $\geq 10\%$ of time. Mean wave amplitudes below these thresholds were referred to as non-elevated; the term “normal” was not used since exact normal values have not yet been recorded. When using our criteria of elevated/non-elevated mean wave amplitudes, 60% of patients had elevated mean wave amplitudes, while they were non-elevated in 40%.

Other authors previously have provided evidence that intracranial pulse pressure amplitudes, and not mean ICP, may be elevated in hydrocephalus [11, 12] and NPH [1].

Mean ICP was normal in our patients, remaining ≤ 15 mmHg, except for one patient wherein the average value of the trend plot was 16.2 mmHg. Mean ICP tended to be higher in the group with elevated mean wave amplitudes (Group B) with median values of mean ICP of 3.2 mmHg in Group A and 8.0 mmHg in Group B (Table 1), which are well within normal ranges in both groups. Hence, mean ICP was not useful for differentiation of patients. It should be noted that several authors have previously reported elevations of mean ICP (or mean cerebrospinal fluid pressure by lumbar puncture) in those improving after shunt treatment [15–17]. These studies explored the occurrence of short-lasting elevations in mean ICP (i.e. B-waves).

Pre-operative observations

Before ICP monitoring we recorded duration of symptoms, clinical severity of iNPH, radiological ventricular size and the presence of co-morbidity. None of these parameters differed between those with elevated or non-elevated mean wave amplitudes (Table 1).

Clinical change after 12 months

In patients with non-elevated mean wave amplitudes and no shunt, some worsening of the clinical state had occurred during the 12 months course. These results compare with previous observations of no change, or even a worsening, in clinical state in the shunted iNPH patients who had non-elevated mean wave amplitudes [9]. Hence, in the present patients with non-elevated mean wave amplitudes, the natural course without a shunt compared to the course after shunting in individuals with non-elevated ICP wave amplitudes.

Among the 21 shunted patients with elevated mean wave amplitudes, 19 patients (91%) experienced a marked clinical improvement (median change in NPH score +4); clinical improvement was slight in one; with the last individual showing no clinical improvement. These results concur with our previous observations of marked clinical improvement in those iNPH patients with elevated mean ICP wave amplitudes [9]. In comparison, a recent study reported clinical improvement in 75% of iNPH patients after a mean follow-up of 18 ± 13 months [15]. An improvement rate of 59% was found after 1 year in the Dutch NPH study [3]. According to the literature review, an overall clinical improvement of 59% was reported, though lasting clinical improvement was observed in only 29% [13]. When using lumbar drainage for 3 days to predict shunt responsiveness, Marmarou *et al.* [14] increased their predictive accuracy for shunt response in iNPH to 88%. On this background, the present results are promising, although our patient number is rather small.

We found a high percentage of co-morbidity (e.g. cerebrovascular and/or cardiovascular disease, and hypertension) in those with elevated mean wave amplitudes. Hence, co-morbidity such as cerebrovascular disease did not prevent shunt response in iNPH. This observation concurs with recent data [15], while others [4] reported cerebrovascular disease to be a predictor of poor outcome after shunt treatment.

Conclusions

We report a material of 40 patients examined within our department for tentative iNPH during a 12 months

period. When defining pulse pressure amplitudes as being elevated when mean wave amplitudes were either ≥ 4 mmHg in $\geq 70\%$ of time, ≥ 5 mmHg in $\geq 40\%$ of time, or ≥ 6 mmHg in $\geq 10\%$ of time, elevated amplitudes were recognized in 60% of patients, and non-elevated amplitudes in 40%. In those iNPH patients selected for shunt surgery based on the presence of elevated mean wave amplitudes, significant clinical improvement was observed in 91%. This one-year patient material is rather small, but the results concur with previous observations that determining percentage of time with given levels of intracranial pulse pressure amplitudes predicts clinical improvement after shunting iNPH patients.

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Comments

The data presented in this paper demonstrates a relatively straightforward method upon which to select idiopathic normal pressure hydrocephalus patients for shunt surgery. The clinical decisions as to whether to insert a shunt in these patients can be difficult so the differences in outcome shown between those who had elevated pulse pressure amplitudes and those who did not may better inform that judgment.

The number of patients in this study is small and therefore it would be wise not to treat the results with some caution. However this work should inform a calculation to determine the number of patients required for a statistically powered study that could determine a sound evidence base for this methodology.

Iain Chambers

These investigators previously reported that patients with Idiopathic Normal Pressure Hydrocephalus responding to shunt surgery showed elevated intracranial pulse pressure amplitudes. Now they selected patients for shunting on the basis of these increased amplitudes that were found for 24 of 40 patients with a tentative diagnosis of INPH. The improvement ratio 12 month after surgery for 22 of the 24 patients receiving a shunt was 86%. This study would have gained strength if all 40 patients had received a shunt permitting calculation of positive and negative predictive values of the method. Those patients not shunted were followed for 12 months as well giving some data on the natural history of this group. Their score on a 12 point scale after one year was only one point less demonstrating that they did not deteriorate.

The data presented in this study further underline the diagnostic value of measuring pulse pressure amplitudes.

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