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Reappraisal of the Intracranial Pressure and Cerebrospinal Fluid Dynamics in Patients with the So-called "Normal Pressure Hydrocephalus" Syndrome

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Summary

Fifty-four shunt-responsive patients were selected from a prospective protocol directed to study patients with suspected normal pressure hydrocephalus (NPH). Patients with gait disturbances, dementia, non-responsive L-Dopa Parkinsonism, urinary or faecal incontinence and an Evans ratio greater or equal to 0.30 on the CT scan were included in the study.

As a part of their work-up all patients underwent intracranial pressure monitoring and hydrodynamic studies using Marmarou's bolus test. According to mean intracranial pressure (ICP) and the percentage of high amplitude B-waves, patients were subdivided in the following categories: 1) Active hydrocephalus (mean ICP above 15 mmHg), which is in fact no tone normal pressure hydrocephalus; 2) Compensated unstable hydrocephalus, when mean ICP was below 15 mmHg and B-waves were present in more than 25% of the total recording time and 3) Compensated stable hydrocephalus when ICP was lower or equal to 15 mmHg and beta waves were present in less than 25% of the total recording time.

The majority of the patients in this study (70%) presented continuous high or intermittently raised ICP (active or unstable compensated hydrocephalus group). Mean resistance to outflow of CSF (Rout) was 38.8 mm Hg/ml/min in active hydrocephalus and 23.5 mm Hg/ml/min in the compensated group (Students t-test, $p < 0.05$). Higher resistance to outflow was found in patients with obliterated cortical sulci and obliterated Sylvian cisterns in the CT scan.

No statistically significant correlation was found when plotting the percentage of beta waves against pressure volume index (PVI), compliance or Rout. An exponential correlation was found when plotting beta waves against the sum of conductance to outflow and compliance calculated by PVI method ($r = 0.79$).

Patients with the so-called normal pressure hydrocephalus syndrome have different ICP and CSF dynamic profiles. Additional studies taking into consideration these differences are necessary before defining the sensitivity, specificity and predictive value of ICP monitoring and CSF studies in selecting appropriate candidates for shunting.

Keywords: Normal pressure hydrocephalus; intracranial pressure; B-waves; cerebrospinal fluid dynamics; compliance; hydrodynamics.

Introduction

Communicating hydrocephalus in the presence of normal cerebrospinal fluid pressure was first described in 1956 by Foltz and Ward in patients with ventricular enlargement after subarachnoid haemorrhage[21]. Later on, the syndrome of Normal Pressure Hydrocephalus (NPH) was completely outlined by Hakim and Adams in 1965[1, 2, 29, 30]. A Normal Pressure Hydrocephalus Syndrome (NPHS) can be idiopathic or secondary to subarachnoid haemorrhage, head injury, aqueductal stenosis, intracranial surgery and many other aetiologies[1, 2, 7, 13, 29, 30, 34, 36, 59, 63].

From the clinical point of view, the diagnosis of NPHS is considered in those patients with gait disturbance, progressive dementia and urinary or faecal incontinence[1, 2, 12, 34, 52]. Although the complete triad is perhaps the most frequent clinical presentation, incomplete and atypical forms of the syndrome have been reported[13, 14, 19, 36, 48, 52, 57, 59]. In spite of its apparent simplicity, NPH syndrome is a clinical complex entity, with incomplete physiopathological knowledge and too often contradictory opinions about its proper management.

The increasing availability of CT scan in clinical practice and the aging population has produced some confusion in differentiating patients with cognitive or gait disturbances due to normal aging, vascular dementia or Alzheimer disease from those clinical findings found in NPH. Normal aging and all the diseases mentioned can result in ventricular enlargement due to loss of brain substance and can mimick hydrocephalus (hydrocephalus "ex-vacuo"). Periventricular hypodensities which sometimes imitate periventricular lucency

(PVL) due to transependymal resorption of cerebrospinal fluid (CSF) are also nonspecific findings in the CT scan of patients with cognitive disorders[22, 24, 53, 58].

Different types of investigations have been proposed in order to study, select and differentiate patients who should be shunted and can improve with surgery from those not helped by placing a shunt. The majority of these studies have been directed to clearly differentiating between brain atrophy and arrested hydrocephalus from true NPH. Air encephalography, isotope or metrizamide cisternography, measurement of cerebral blood flow (CBF), CSF-tap-test, long term intracranial pressure (ICP) monitoring and a different set of hydrodynamic studies and pressure/volume tests have been carried out with variable results[6, 8, 9, 11, 13, 15, 25, 27, 37, 41, 43, 48, 52, 56, 59, 62].

ICP monitoring and hydrodynamic studies have been widely reported in NPH syndrome. Normal or low ICP, with high or low amplitude beta waves and sometimes plateau waves especially during REM sleep have been reported as typical of NPH and clearly related with a good outcome[6–9, 13, 16, 17, 27, 35, 37, 42, 49, 59]. Nevertheless, the value of long term ICP monitoring and/or CSF studies in order to select candidates to shunt is still controversial[7, 52]. The sensitivity and specificity of ICP monitoring and CSF dynamics have not been defined yet. In general, a better knowledge of the different forms of NPH is still necessary to get a better understanding of the natural evolution, clinical symptoms and signs and possible outcome in this important group of patients. Re-evaluation of the clinical value of ICP monitoring and CSF studies, should necessarily start with a better definition of the population under study.

The observation of different patterns of ICP in patients with the NPHS prompted us to analyze and describe ICP profiles and CSF studies in a series of cases studied longitudinally in order to get a better insight into the physiopathology of this complicated and, until now, not well understood syndrome.

Clinical Material and Methods

The present study is part of a prospective protocol, that began in 1986 and in which patients with suspected Normal Pressure Hydrocephalus Syndrome (NPHS) were included. To admit a patient to this protocol, at least one of the following clinical criteria were necessary: 1) gait disturbance unexplained by other neurological disease, 2) dementia, 3) non responsive L-Dopa Parkinsonism, 3) urinary or faecal incontinence not explained by other neurological or extra-neurological disease. Apart from the clinical criteria, only patients with an Evans ratio measured in the CT scan above 0.30 were considered.

All patients were independently examined by one neurosurgeon and one neurologist. Apart from the usual neurological examination and neuropsychological studies, all patients were graded according to a modification of the Stein and Langfit scale[6, 28]: Grade 0: No neurological deficit, able to work or perform the same duties as before the disease. Grade I: Patient able to function independently at home. Grade II: Some supervision required at home. Grade III: Custodial care in spite of considerable independent function. Grade IV: No capacity for independent function. Grade V: Bedridden or vegetative.

A NPH Score to measure improvement in different disabilities was devised in our hospital to grade these patients. The scale is divided into three parts. The minimum possible score is 3 points and the maximum 15 points. The scale evaluates the three main parts of the NPH syndrome.

I) Gait Evaluation: one point if the patient is bedridden or not able to ambulate; 2, if ambulation is possible with help; 3, when independent walking is possible but is unstable or the patient falls; 4, if abnormal but stable gait is present; and 5 points when gait is normal.

II) Cognitive Functions are graded as follows: 1 point when patient is vegetative; 2, if Severe dementia is present; 3, when important memory problems exist with more or less severe behaviour disturbances; 4, when memory problems exist that are reported by patient or family; and 5, when only cognitive disturbances are found by specific tests.

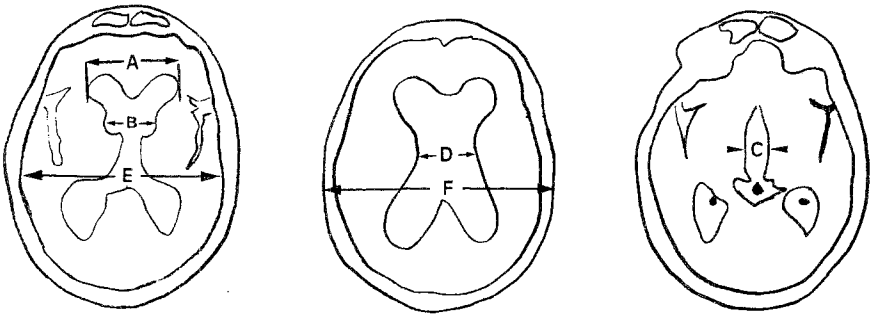

Fig. 1. Measures sites of ventricular size on CT scan. (*A*) Maximum bifrontal distance, (*B*) distance between the caudate nuclei at the level of the foramen of Monro, (*C*) maximum width of the III ventricle, (*D*) minimum width of both cella media, (*E*) maximum inner diameter of the skull at the level of the measurement of the maximum bifrontal distance, (*F*) maximal outer interparietal diameter at the level of the cella media measurement

III) Sphincter disturbances are graded 1, when the patient presents both urinary and faecal incontinence; 2, if continuous urinary incontinence is present; 3, when sporadic urinary incontinence exists; 4, only urinary urgency is present; and 5, when there are no objective nor subjective sphinter dysfunction.

The interobserver reliability of this scale is 0.92 ($n = 32$, $p < 0.01$).

All the cases were evaluated according the following scales: Pfeiffer Scale, Blessed Scale, daily life activities scale, minimental examination and Hachinski Ischaemia Score[5, 18, 20, 45, 51]. Neurological examination, grading scales and different tests are repeated at 6, 9, 12, and 24 months after shunt in those cases in which by-pass procedures are performed.

In all patients a CT scan was performed (Philips 310, GE 8800, Elscint 1800, or Elscint 2400). The following measurements and ratios were considered (Fig. 1): (A) Maximum bifrontal distance, (B) Distance between the caudate nuclei at the level of the foramen of Monro, (C) Maximum width of the III ventricle, (D) Minimum width of both cella media, (E) Maximum inner diameter of the skull at the level of the measurement of the maximum bifrontal distance, (F) Maximal outer interparietal diameter at the level of the cella media measurement (Fig. 1).

From the measurements we have described above, we obtain the following indexes: 1) Evans Index (EI): A/E, 2) Ventricular Score (VS): (A + B + C + D/E) × 100, 3) III Ventricle Index (IIIV): C/E, 4) Inverse Cella Media Index (ICMI): D/F.

In all the patients we evaluated the presence or absence of periventricular lucencies around the frontal horns of the lateral ventricles. Sylvian cisterns and convexity sulci in the uppermost slices were graded according the following classification: 1. Obliterated, 2. Normal and 3. Widened. Temporal horns and IV ventricle were also graded as normal or enlarged.

The majority of patients were also studied by magnetic resonance imaging (MRI). Both in CT scan and MRI, the presence of leukoaraiosis in the white matter was evaluated[22, 24, 55, 58]. Aqueductal stenosis was diagnosed according to MRI criteria.

As a part of their work-up, all patients underwent continuous intracranial pressure monitoring and CSF-dynamic studies. In all the cases continuous ICP monitoring was performed for at least 24 hours (Mean ± SD, 40.4 ± 15.6) including overnight recording of ICP. All patients were monitored using a fiberoptic* extradural device introduced through a pre-coronal burr hole. Hard copies of the ICP were obtained through a single channel recorder with a paper speed of 6–20 cm/hour. The patient was maintained flat throughout the recording time. Analysis of the ICP records were made following the method described by Børgesen in mmHg[7].

Mean resting ICP was calculated from the records every 15 minutes in periods without fluctuations. Amplitude of pulse waves in stable periods of recording and the presence of high amplitude B-waves (1/2–12/minute ICP fluctuations more than 10 mmHg above baseline and lasting at least ten minutes), were evaluated. A-waves were considered to be present when constant pressure elevations at least 20 mmHg above the resting line with abrupt onset and end, and lasting at least 10 minutes (Fig. 2) were observed.

To avoid miscalculation due to the different durations of the monitoring time, plateau and B-Waves were expressed as a percentage of the first 24 hours period of the total recording time.

In all the patients after the period of ICP monitoring, we measured the resorptive capacity of CSF using the bolus injection method described by Marmarou *et al.*[28, 39, 44, 45] and the constant rate infusion test (1 ml/min), using normal saline[37]. Marmarou's method has been used in this report to calculate Resistance to Outflow (Rout), Conductance to outflow (Cout), Pressure Volume Index (PVI) and Compliance (Ccsf). Compliance was calculated using the equation Ccsf = 0.4343* PVI/PO where PO ist the basal ICP immediately before the bolus injection. All the tests were performed by lumbar puncture with a 18 g cannula, except in suspected aqueductal stenosis in which we performed intraventricular measurements. The normal

* Manufactured by Ladd Research Industries, Inc., Burlington, Vermont.

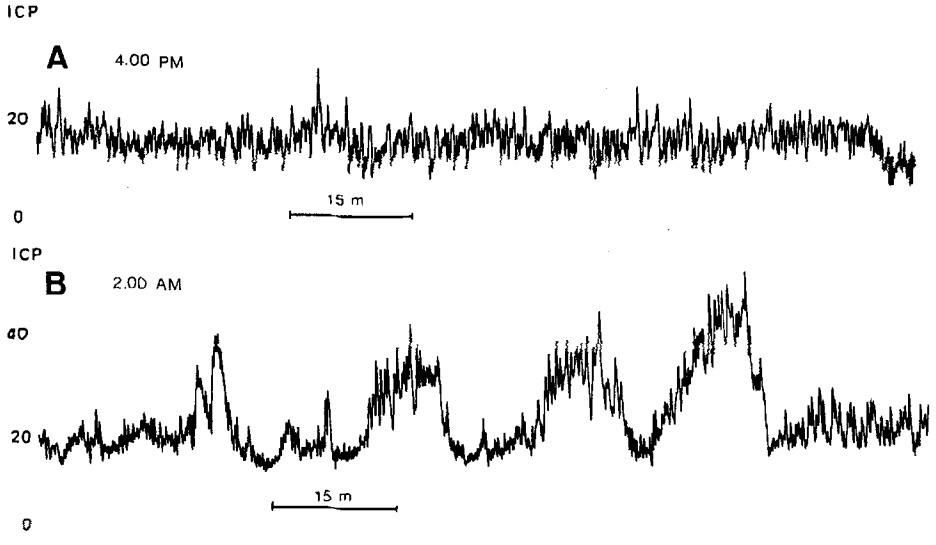

Fig. 2. Active hydrocephalus. This 68-year-old man was admitted because of a 18-months history of progressive dementia, gait disturbances and urinary dysfunction. He scored 3 points in the Stein and Langfit scale. The CT scan demonstrated a severe supratentorial ventriculomegaly with an Evans ratio of 0.46. Mean resting ICP was 35 mmHg. A) Extradural recording of ICP during the day (20 cm/hour speed). B) Plateau waves during overnight recording at the same speed as record A

values of hydrodynamic variables are those considered by Shapiro *et al.*[55]. According to the ICP recording, all the patients were included in one of the following categories: 1) Active Hydrocephalus: we considered in this group all patients with a mean ICP above 15 mmHg (Fig. 2); 2) Compensated Hydrocephalus: all the cases with a mean ICP below or equal to 15 mmHg were included in this group. Compensated hydrocephalus were divided into stable or unstable if the ICP recording showed B-waves in less or more than 25% of the recording time (Figs. 3 and 4).

Criteria for shunt operation were the following: 1) High mean ICP (> 15 mmHg), 2) Normal or low mean ICP (< or equal to 15 mmHg) with more than 10% high amplitude beta waves, 3) Normal or low mean ICP with a ROUT > 10 mmHg/ml/min and/or low PVI (less than 15 ml). All operated patients were shunted using

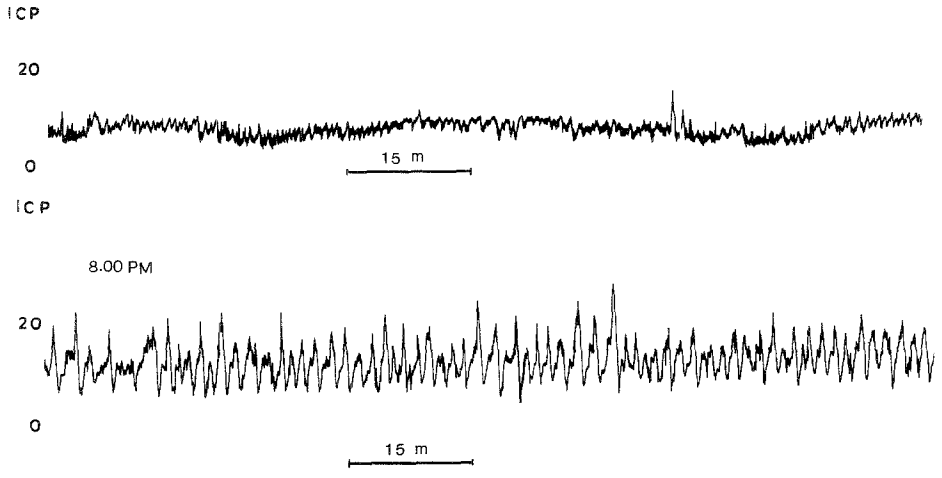

Fig. 3. Compensated Stable Hydrocephalus in a 77-year-old man who first experienced cognitive impairment two years before admission. The CT scan was evaluated as suggestive of brain atrophy because of moderate ventricular enlargement with coexistent cortical atrophy. Evans Index 0.35. The ICP extradural recording demonstrating a mean resting ICP of 8 mmHg with nocturnal Beta-waves in 18% of the total recording time (lower chart). PVI was 13.3 a Rout 19.50 mmHg/ml/min. Response to shunting was good

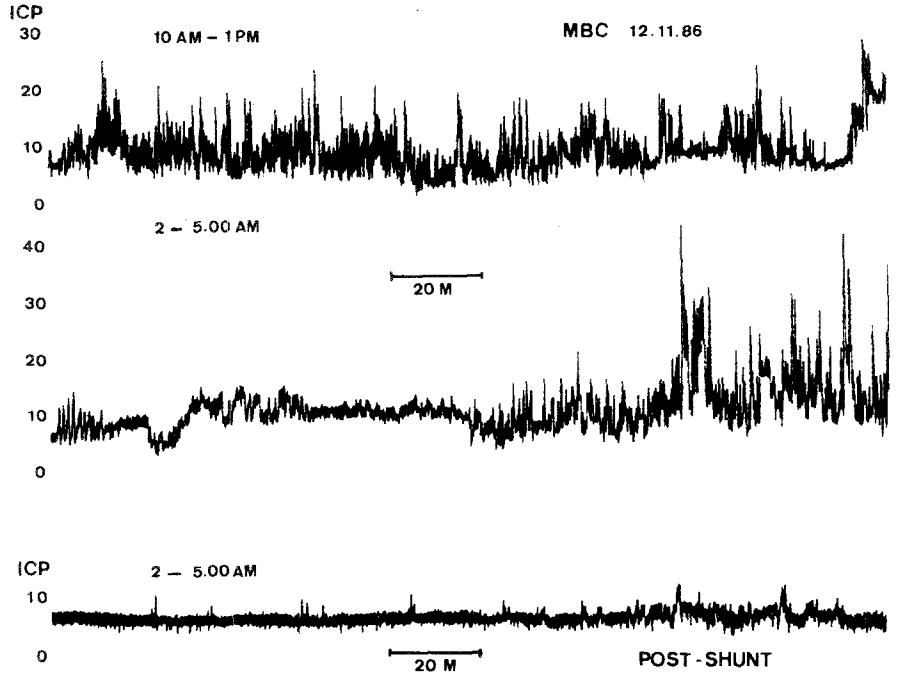

Fig. 4. Compensated unstable hydrocephalus. This 68 year-old woman was admitted to our department complaining of cognitive and gait disturbances over the preceding twelve months. Evans ratio was 0.45 in the CT scan. Mean resting ICP was 12 mmHg with beta-waves in 55% of the total recording time. Beta waves were more evident during overnight recording (middle chart). (Lower chart) ICP chart 24 hours after shunting in the same period of time than before the operation (2–5.00 a.m.). Beta waves completely disappeared in spite of similar mean resting ICP

a low profile valve, diaphragm based, with a low closing pressure range (5–20 mm H_2O)*.

After shunt placement, the patients were reviewed at 6, 9, 12, and 24 months and the clinical examination, grading, neuropsychological testing and CT scan with ventricular measurements were repeated. For the purpose of this study we selected 54 shunt responders of an entire series of 65 patients with a suspected primary or secondary NPH syndrome who were submitted to shunt. The cutoff point selected for evaluating the clinical improvement was six months after the shunt. The criteria for a shunt responder was that the patient improved at least one grade in the Stein and Langfitt scale. No attempt to evaluate the grade of improvement was done in this study. Two patients, with transient improvement and later shunt dysfunction who improved after shunt revision, were also included.

Two-tailed Student's t-test, Pearson's correlation test and linear regression using the least square method was used to compare quantitative variables. Fischer exact test and Chi-square test were used to compare quantitative variables. The Mann-Whitney U-test was used to compare not normally distributed data. The level of statistical significance was established for $p < 0.05$.

Clinical Results

Age, Sex, and Aetiology

The 54 patients considered as shunt responders ranged in age from 25 to 78 years (average ± SD, 65 years ± 11 years, with a median of 67 years). Thirty patients were men (56%) and 24 women (44%). From an aetiological point of view, hydrocephalus secondary to subarachnoid haemorrhage and idiopathic communicating hydrocephalus were the most important groups (22 and 30% respectively). A clinical summary of the group studied is set out in Table 1. According to the functional classification, 63% of the patients were completely dependent for daily life activities or bedridden (Functional grades 4 and 5) while only 6 patients (11%) presented with a functional grade of 2. Mean ± standard deviation of the scores in the different scales used to evaluate these patients are also shown in Table 1.

Clinical Symptoms

When the patients were admitted to the protocol study, the mean time for evolution of the syndrome was 28 months (range 1–216 months) with a median of 12 months (Table 2). Clinical symptoms when the patient was admitted to the protocol are shown in Table 2. Most of the patients (70%) presented the complete triad, while gait disturbances with or without sphincter dysfunction but without significant dementia was observed in only 4 patients (7%). Eighteen of the 38 patients with the complete triad, had severe dementia (scored 1 or 2 in the Cognitive Function Score).

CT Scan Findings

The 54 patients ranged in Evans Index from 0.30 to 0.48. Different ratios and indexes are shown in Table 3. Severe ventricular enlargement was observed in all the patients including the aqueductal stenosis group. In this group the mean Evans Index was 0.47

* American Heyer-Schulte Corporation. Distributed in Spain by SUMSA.S.A., Barcelona.

Table 1. *Clinical Summary of the Group Studied*

Number of cases	54	
Aetiology		
Idiopathic	16	30%
Subarachnoid haemorrhage	12	22%
Posttraumatic	8	15%
Meningitis/Encephalitis	1	2%
Aqueductal stenosis	12	22%
Post-craniotomy	3	6%
Posterior fossa tumour	2	4%
Functional classification		
Grade 2	6	11%
Grade 3	14	26%
Grade 4	22	41%
Grade 5	12	22%
NPH score (mean ± SD)		7.6 ± 2.8
Gait score		2.2 ± 1.3
Cognitive score		2.8 ± 1.1
Sphincter score		2.6 ± 1.3

Table 2. *Clinical Symptoms on Admission*

	n	%
Complete triad	38	70
Gait disturbances	2	4
Gait + dementia	8	15
Dementia + sphincters	4	7
Gait + sphincters	2	4

Table 3. *CT Scan Linear Measurements*

	EVANS	VSCORE	Cella media	III. ventricle
Minimum	0.30	67	0.20	0.040
Maximum	0.48	154	0.50	0.188
Mean	0.35	98.1	0.29	0.096
Median	0.34	94.6	0.27	0.090
SD	0.09	18.1	0.06	0.040

EVANS: Evans Index. VSCORE: Ventricular Score. SD: Standard deviation.

and Ventricular Score 133.1 while in the rest of the patients the mean Evans Index was 0.34 and Ventricular Score 93.5 (Students t-test, $p < 0.001$).

Periventricular frontal lucency compatible with transependymal CSF resorption was observed in only 20 patients (37%). Patients with idiopathic hydrocephalus presented PVL in 25% of the cases while this sign was present in 39% of secondary hydrocephalus. PVL was more frequent in the subarachnoid haemorrhage group (67%) than in any other group. Other periventricular hypo-intensities of the leukoaraiosis type[22, 28] were especially observed in the idiopathic group.

Obliterated cortical sulci were found in only 26 patients (48%), while in the other patients convexity sulci were considered normal for the age or cortical atrophy was found. Temporal horns were found dilated in 12 of the 16 patients with an idiopathic hydrocephalus (75%) and in 34 patients with a secondary aetiology (90%). An excellent correlation between the different ventricular indexes used in this paper was found. Correlation coefficient (r) was high in all the cases, ranging from $r = 0.69$ for comparison between Evans Index and III Ventricle Index to $r = 0.88$ when we compared Ventricular Score vs. III-Ventricle Index and Inverse Cella Media Index ($p < 0.0001$ in all the comparisons).

ICP Monitoring

Continuous recording of the ICP demonstrated in the entire group a mean ICP of 11.8 mmHg ± 7.3 mmHg (Mean ± SD). The range was between 6 mmHg and 34 mmHg with a median of 10 mmHg. Independently of the primary aetiology of the hydrocephalus, we could distinguish two groups of patients on the basis of mean ICP. Eight (15%) patients presented an elevated mean ICP (> 15 mmHg), that means they had no real Normal Pressure Hydrocephalus in spite of presenting with the typical syndrom. They were included in the Active Hydrocephalus group (Fig. 2). Most of the cases (85%), were included in the Compensated Hydrocephalus group, that is a mean ICP below or equal to 15 mmHg (Table 4) (Figs. 3 and 4).

In 30 of the patients with Compensated Hydrocephalus (65%) we observed high level beta waves in more than 25% of the recording time (Fig. 3). This group of patients was included in the Compensated Unstable Hydrocephalus group while the 16 patients (35%) with beta waves in less than or equal to 25% of the first 24 hours of recording were included in the group called Stable Compensated Hydrocephalus (Fig. 4).

Plateau waves were observed in 12 patients (22%). Most of the cases presented small plateau waves with an amplitude between 20 and 40 mmHg (Fig. 2). Only two patients had A-waves above 40 mmHg. Of the twelve patients, 11 had secondary hydrocephalus. Plateau waves were more frequent in the overnight recording. All patients with plateau waves were included in the Active Hydrocephalus (3 cases) or Unstable Compensated Hydrocephalus groups (9 cases). No patient with Stable Compensated Hydrocephalus presented plateau waves.

CSF Dynamics

Resistance to outflow was evaluated by Marmarou's bolus test. Patients included in the active hydrocephalus group had a mean resistance to resorption of 38.8 mmHg/ml/min, while the mean Rout was 23.5 mmHg/ml/min in the compensated group. Stable compensated hydrocephalus had a Rout of 14.3 mmHg/ml/min while unstable compensated hydrocephalus had a Rout of 28.5 mmHg/ml/min (Students t-Test < 0.05). There were no differences in Rout values depending on whether the aetiology was idiopathic or secondary hydrocephalus. Hydrodynamic variables are summarized in Table 5.

PVI was surprisingly higher in active hydrocephalus compared with compensated hydrocephalus (Students

Table 4. *Summary of ICP Monitoring Findings*

	n	Mean ICP	ICP Range
Active hydrocephalus	8	25.5 ± 10.4	17–35
Compensated hydrocephalus	46	9.4 ± 2.8	4–15
stable	16	8.1 ± 2.8	4–12
unstable	30	10.1 ± 2.7	6–15

Mean ICP (mean ± SD).

Table 5. *Hydrodynamic Variables in the Study Group*

	Rout	PVI	Ccsf
Active	38.8 ± 4.5 } *	24.1 ± 6.6 } *	0.33 ± 0.02 } *
Compensated	23.5 ± 2.4 }	12.7 ± 1.0 }	0.71 ± 0.1 }
unstable	28.5 ± 2.9 } *	13.7 ± 1.1	0.67 ± 0.07
stable	14.3 ± 3.4 }	12.9 ± 1.8	0.78 ± 0.09

Differences are expressed mean ± standard error of the mean. * Students t-test < 0.05.

t-test, $p < 0.05$). No significant difference existed in PVI among stable and unstable compensated hydrocephalus. Compliance calculated by Marmarou's method[44, 45, 55], gave a mean value of 0.33 in the active group and 0.71 in the compensated group ($p < 0.05$). Again no statistically significant differences were found comparing stable and unstable groups (Table 5). When mean ICP was plotted against Rout, Cout, and PVI, no statistically significant correlation was found.

Beta Waves and Hydrodynamic Variables

The percentage of beta waves in the total recording time was plotted using linear regression against Pressure Volume Index, Compliance, Conductance to outflow and Resistance to outflow. Correlation coefficients were too low to be significant ($r < 0.45$). Using B rgesen approach[10] in which beta waves are correlated with Cout and Ccsf, and where these two variables are equally weighted and simply added, we found that the plot of Beta waves versus Ccsf + Cout followed an exponential curve with a correlation coefficient of 0.79 ($r = 0.79$, $p < 0.05$).

ICP, Hydrodynamic Variables, and CT Scan

In both active and compensated hydrocephalus, linear ventricular measurements were plotted against ICP and Rout. Resistance to rearbsorption was also correlated with the width of Sylvian valleys and cortical sulci evaluated in the CT scan. The results are given in Tables 6 and 7. In the active hydrocephalus group, we could not find statistically significant correlation between ICP and Rout and linear measurements of the ventricular system in the CT scan. Nevertheless, in the compensated group, a significant negative correlation was observed when all the ventricular measurements except for the III ventricle were plotted against ICP.

Table 6. *CT Scan and Hydrodynamic Variables*

	Active	Compensated	
		r	p
VSCORE/ICP	NS	− 0.53	< 0.05
CELLAM/ICP	NS	− 0.70	< 0.001
EVANS/ICP	NS	− 0.80	< 0.001
IIIV/ICP	NS	NS	NS
VSCORE/ROUT	NS	− 0.56	< 0.05
CELLAM/ICP	NS	− 0.63	< 0.05
EVANS/ROUT	NS	− 0.58	< 0.05
IIV/ROUT	NS	− 0.41	< 0.05

NS: Not statistically significant. r = correlation coefficient.

Table 7. *CT Scan and Hydrodynamic Variables*

	Rout (Mean ± SD)	
Sylvian valleys		
obliterated	32.47 ± 17.0	NS
normal	26.05 ± 14.5	NS, S
large	15.85 ± 11.2	NS
Cortical sulci		
obliterated	27.81 ± 14.9	NS
normal	23.34 ± 10.8	NS, S
large	15.58 ± 7.25	NS

S: Statistically significant ($p < 0.05$). NS: Not statistically significant.

An inverse correlation was also found comparing ventricular size and Rout (Table 6).

Comparing resistance to outflow with the width of the cortical sulci and Sylvian cisterns in the CT scan, we found that Rout increased when the width of the cortical sulci or Sylvian fissure decreased in size (Table 7). However, these differences were not statistically significant except when comparing obliterated and augmented Sylvian fissure or cortical sulci with resistance to outflow.

Discussion

In our opinion, two different problems in suspected NPHS should be distinguished. The first would be how to find reliable diagnostic criteria of the syndrome and the second, to find and analyze those variables that influence or predict outcome after shunting. From a diagnostic point of view, clinical features and non-invasive ancillary test like CT scan and MRI help in selecting patients who are candidates for additional studies.

Our paper is not directed to analyze variables that predict successful outcome from a shunt but to analyze the ICP and hydrodynamic patterns in shunt-responsive patients in order to have a better insight into this complex syndrome. We believe that selecting only shunt-responsive patients is appropriate when trying to propose an ICP classification of NPHS. Excluding shunt failures, we are quite sure that we are selecting "true" normal pressure hydrocephalus and in this way doubtful diagnosis are excluded.

Improvement rate in presumed normal pressure hydrocephalus patients, averages 52% in the medical literature[4, 6, 7, 13, 16, 17, 19, 34, 36, 49, 50, 52, 59, 62]. This rate is quite low if we consider that a high rate of surgical complications is present in this group of patients[6, 52, 59]. Variably symptomatic or asymptomatic adult hy-

drocephalus representing stable, arrested normal pressure hydrocephalus or "ex-vacuo" ventriculomegaly could be an incidental finding on investigation of unrelated problems or found in the context of patients with dementia, gait disturbance or urinary incontinence[52]. Failure of agreement for diagnostic criteria and lack of knowledge of the spontaneous evolution of NPH makes it sometimes difficult to advise a shunt in these patients, especially when a tendency to recompensate with minimal disability has been noted in NPH[46]. To identify benign forms of NPH in which a shunt would not be necessary is a difficult problem and requires a more precise knowledge of the pathophysiology of NPH.

The most important group in our study was secondary hydrocephalus. We consider interesting the high number of cases included in the aqueductal stenosis group compared with other series in which this diagnosis was only considered in 4% of the cases[37]. The availability of high resolution CT scan and magnetic resonance imaging allowed us a better classification of patients with hydrocephalus. The fact that patients in the aqueductal stenosis group had a significantly higher Evans Index than the other patients, supports the idea of McLone that massive ventriculomegaly is more frequent when distention of the ventricles occurs early in life while sutures and fontanelle are still open[47].

In studying normal pressure hydrocephalus, ventricular size is an important variable to report in order to compare different groups of patients and make correlations with hydrodynamic variables. Although CT scan software allows the performance of very complex volumetric measurements, easily determined linear measurements are quite reliable methods for estimating ventricular size. A good correlation between the different ventricular ratios used was found in our study.

Periventricular hypo-intensities are frequent in old patients[22, 24, 28, 58] and not always indicative of a hydrodynamic disturbance. True periventricular lucency (PVL) indicating transependymal absorption of CSF is difficult to demonstrate. This was found in only 37% of the entire group. PVL was more frequent in patients with hydrocephalus after subarachnoid haemorrhage, a point that has been addressed by other authors[60, 61]. We have not found any statistically significant difference in the development of symptoms in patients with or without PVL. Although in depth discussion of CT scan findings in NPHS is beyond the scope of this paper, in our opinion, better defined criteria are necessary when evaluating white-matter changes in the CT scan of old patients with dementia that may allow us to differentiate transependymal CSF absorption from periventricular lucencies associated with vascular disease, dementias or normal aging[55].

Although the aetiology of the NPHS is probably multifactorial, increased resistance to absorption of CSF is supposed to be the most important single factor and has been the most constant abnormality reported by hydrodynamic studies[6–9, 27, 35–39, 42, 52, 56]. According to Børgesen, the pressure profile in NPHS presents itself as a continuum from a normal mean ICP without abnormal fluctuations to high ICP with plateau-waves and/or continuous beta-wave activity[6]. Continuous measurement of the intracranial pressure offers us one of the most reliable ways for study and selection patients with a suspected NPHS. Extradural devices allow us to monitor patients for a long time without the risk of infection and with only minor drawbacks. ICP measurements accurately reflect the intracranial dynamics. Controversial results in this field express differences in methods and lack of standardized evaluation of results rather than a true inability of ICP in distinguishing patients with normal or abnormal CSF dynamics.

High and/or low amplitude beta waves, abnormalities in pulse amplitude and plateau or plateau-like fluctuations especially during overnight recording and REM sleep have been widely reported in the NPHS[6–9, 13, 15, 27, 38, 40, 42, 43, 49, 52, 59].

Additionally, some studies have reported high mean ICP similar to that found in hypertensive hydrocephalus in patients with this syndrome[31, 38, 42]. We have used the mean ICP to classify patients into active and compensated hydrocephalus. This approach, although artificial, is useful to discriminate different groups of patients who may have different clinical, ICP and CSF dynamics profiles.

According to these ICP criteria, active hydrocephalus with a mean ICP of 25 mmHg was found in 15% of the cases. It was interesting to note, that none of these patients had an idiopathic syndrome. The average time of evolution of clinical symptoms was not statistically different in this group compared with compensated hydrocephalus (mean ICP equal to or less than 15 mmHg). Different pressure levels in patients with the same NPHS could express differences in the tensile properties of the brain parenchyma and ventricular walls when submitted to an increased CSF bulk, secondary to increased resistance to outflow. This fact, suggested an elegantly discussed by Geschwind[23], has not received enough attention in the literature on NPH.

Although the physiopathology of B-waves has not

been fully elucidated, it is quite certain that these abnormal fluctuations of ICP represent an exaggerated response to physiological increases of cerebral blood volume in patients with abnormal CSF dynamics. Therefore, it seems rational to differentiate between stable and unstable compensated hydrocephalus on the basis of beta waves. Using the percentage of beta-waves in the total recording time allowed us to distinguish two different groups of patients with a different hydrodynamic profile. About 56% of the patients in our study were included in the compensated unstable group. This fact establishes the important point that 70% of the patients with a NPHS presented in our study continuous or intermittently raised ICP. It would be very useful to know if different patterns of ICP reflect different stages of evolution of the disease or is due to biomechanical properties of the brain parenchyma. Additional studies, including a higher number of patients in which continuous measurement of ICP are used, are still necessary before determining definite clinical and physiopathological conclusions about this important point.

An increased resistance to the outflow of CSF has been the most constant factor found in different studies on NPHS[6–11, 15, 35–40, 42, 57]. Although absorptive capacity can be evaluated by several methods, the constant rate lumbar or ventricular infusion test[36, 37], the lumbo-ventricular steady-state perfusion and the bolus injection method[38, 40, 44, 45, 55] are the most widely used. Although bolus test underestimates resistance to outflow calculated by other methods[7, 9, 10, 38–40], it is a simple, fast and reliable method to measure Rout and allows us to obtain additional hydrodynamic variables such as PVI or compliance of the cranio-spinal axis.

Using the bolus test, we found that all the patients in our study had an increased resistance to absorption of the CSF when compared with the normal value of Rout reported by Shapiro *et al.* of 2.8 ± 0.8 mmHg/ml/min[55]. Mean Rout was found to be 14.3 mmHg/ml/min in the compensated stable hydrocephalus; 28.5, in the compensated unstable group, and 38.8 in patients with active hydrocephalus. Our data support the idea that although we could not find a linear correlation between Rout and ICP and between Rout and the percentage of B-waves, increased resistance to CSF is one of the most important factors determining the level of ICP and triggering the presence of beta-waves. That B-waves are more frequent in patients with a high Rout has been reported by Børgesen[10]. Nevertheless, the same author observed B-waves in patients with a Cout above 0.08 (Rout below 12.5 mmHg/ml/min) which suggests that other factors beside a low absorptive capacity influence B-wave activity[10].

In our study high amplitude B-waves were present in patients with high Rout (> 25 mmHg/ml/min) and also in those with moderate increases of Rout (10–25 mmHg/ml/min). Also B-waves were present in patients with normal, moderate or severe reduced PVI. According to this and other reports in literature[7], B-waves are not the consequence of high Rout, nor low PVI alone. Børgesen *et al.*[10] demonstrated that the combination of low compliance and low Cout (high Rout) maybe necessary to produce B-waves. Following Børgesen's approach, of weighting equally compliance and conductance to outflow, we found the same type of exponential correlation between beta waves and the sum of Cout + Compliance of the craniospinal axis. Therefore, our study supports the idea that low compliance and high resistance to outflow of CSF are necessary for B-waves to appear. We could not find any correlation when plotting the duration of beta waves against isolated compliance, PVI, Rout or Cout. Additional studies, with a clearer definition of what should be considered beta waves and what should not, are necessary to confirm these suggestions.

Kosteljanetz found a positive correlation between the abnormally raised Rout and ICP[39]. In the steady-state and according to Marmarou's equation, mean ICP is governed by the resistance to absorption of the CSF (Rout), the production rate of CSF (If) and the pressure in the sagittal sinus (Pss)[44, 45, 55]. It seems logical, that in our study patients included in the active hydrocephalus group had a mean Rout higher than the patients included in the compensated group. However, although patients with a mean ICP above 15 mmHg had a higher resistance to CSF outflow than patients with a compensated hydrocephalus we could not find any linear correlation between ICP and Rout in both compensated and active hydrocephalus. Our findings are similar to those reported by Børgesen, who studying only patients with a mean ICP less or equal to 12 mmHg, did not find any correlation between ICP and Cout[6].

Mean pressure volume index has been estimated by Shapiro *et al.* to be around 23.7 ± 3.7 ml[55]. PVI was surprisingly normal in active hydrocephalus (24.1 ml) and reduced in the patients with compensated hydrocephalus (13.7 ml). However, compliance was significantly lower in the active hydrocephalus compared with the compensated group. The latter finding is understood if we take into consideration the fact that PVI represents theoretically the slope of the pressure volume

curve in a semilogarithmic coordinate system[39, 40, 44, 45, 55]. PVI is the same regardless of where on the linear curve it is determined, and thus independent of the basal ICP[39, 44, 45, 55]. Nevertheless, compliance is pressure dependent and changes depending on the level of ICP.

It is difficult for us to explain why PVI was higher in patients with increased mean ICP. Pressure volume index is a complex function that depends on many other parameters like cerebral perfusion pressure, autoregulatory state of the brain vessels, cerebral blood volume, total ventricular volume, and brain parenchymal changes[26, 46, 54, 55]. Kosteljanetz has studied CSF dynamics in NPHS using the PVI method[38, 40]. In his patients, mean ICP was above 15 mmHg in only three cases and mean PVI in the entire group was 10.8 ml, very close to our results in compensated hydrocephalus[38]. Although we can hypothesize that patients with an active hydrocephalus can have a more severe structural abnormality of the brain parenchyma than patients with a compensated hydrocephalus, this assumption needs further and more complete evaluation. These abnormalities in biomechanical properties of brain tissue could help to explain why patients with a high intracranial pressure do not manifest clinical symptoms of raised ICP.

In those cases with compensated hydrocephalus, a negative correlation was found between linear ventricular measurements and ICP. This correlation was statistically significant except when plotting ICP and III ventricle measurements. That ICP increases initially and is almost normalized when the ventricles dilate, is a fact that has been demonstrated in experimental hydrocephalus[3, 32]. Our findings that increased size of cerebral ventricles is associated with a low ICP support this fact. In agreement with Kosteljanetz[39, 40], no correlation was found when plotting Rout versus linear ventricular measurements, failing to demonstrate that ventricular size is a linear function of cerebrospinal fluid absorption alone.

Patients with obliterated cortical sulci and Sylvian fissures had a higher resistance to CSF absorption than patients with normal or obliterated cortical sulci or Sylvian subarachnoid cisterns. Increased resistance to CSF was related with decreased cortical sulci or Sylvian cisterns (Table 7). These findings were only statistically significant when comparing obliterated versus increased cortical sulci or Sylvian cisterns ($p = 0.001$ for Sylvian valley and $p = 0.002$ for cortical sulci). Lack of statistical significance between the other groups is probably related to the low number of observations in our study. Børgesen and Kosteljanetz also found a low conductance to outflow in patients with small or absent cortical sulci[39, 40, 46]. Increased resistance to CSF absorption in patients with obliterated cortical pathways in the CT scan could be an aetiological factor or a consequence of the hydrocephalus. Obliteration of the convexity sulci basically depends on the transmantle gradient of pressure between the cerebral ventricles and the subarachnoid space. Transmantle gradients have been demonstrated in the laboratory and in patients with adult communicating hydrocephalus[33, 47].

In our study, we have been able to show that patients with a NPHS are not homogeneous from an ICP point of view. ICP profile is variable from cases in which mean ICP is continuously high to patients in whom resting ICP is normal but present more or less frequent intermittent fluctuations of the resting ICP (high amplitude B-Waves).

To clarify the normal pressure hydrocephalus syndrome, we need additional studies in which long term ICP monitoring should be ideally recorded in all the cases. Distinguishing between diverse groups of patients is important if we want to identify the physiopathological factors involved in the development and evolution of normal pressure hydrocephalus. In our opinion better defined criteria about estimating mean ICP and a better definition of abnormal fluctuations of ICP would be desirable and very helpful in order to compare different studies from different centers. When in all populations of patients with the syndrome of NPH a clearer hydrodynamic description could be given, then it would perhaps become possible to better select those patients for shunting who will profit from this management. Controlled studies are also an important precondition for the evaluation of the appropriate shunt system (low pressure, high pressure, flow regulated etc.) for these patients. Moreover, a better definition of the different forms of NPHS will enable us to differentiate between patients with a true NPH, patients with a clinically compensated form of the syndrome and those patients in whom hydrocephalus is only a reflection of a loss of brain substance.

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Editorial Comments

The authors decided – against suggestions of our reviewers – to include patients into their study, who had higher than normal ICP values and therefore cannot be considered to suffer from Normal Pressure Hydrocephalus.

The argument of the authors has been, that they presented only patients with typical clinical syndromes.

With some reluctance we finally have accepted the paper as it stands. But we still are convinced that it would have been better to include only patients into this interesting study, who really belong to the Normal Pressure Hydrocephalus group.

F. Loew
Chairman of the Editorial Board

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