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CSF dynamics and pressure-volume relationships in communicating hydrocephalus

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Twenty-nine patients consecutively admitted with a diagnosis of communicating hydrocephalus underwent 1) continuous intracranial pressure (ICP) monitoring; 2) pressure-volume studies; and 3) measurement of resistance to outflow of cerebrospinal fluid (R_{out}). The two latter calculations were made by the bolus injection and pressure-volume index (PVI) techniques. In 19 patients mean ICP never exceeded 15 mm Hg. In the other 10 patients varying degrees of mildly raised ICP was noted. The frequency of waves at $\frac{1}{2}$ to 2/min varied from 3% to 58%. The ICP pulse amplitude ranged from 0.5 to 10 mm Hg, and PVI from 4.6 to 18.2 ml. The R_{out} ranged from 2.5 to 31.4 mm Hg/ml/min, and was linearly correlated to the ICP. Thus, patients with a higher R_{out} also had a higher ICP as compared with patients with lower R_{out}, yet ICP could still be within limits considered normal. The cerebrospinal fluid dynamics (formation rate × resistance) contributed much more to the ICP than in normal individuals. It is postulated that communicating hydrocephalus represents one endpoint of a continuum, where the preceding phase is high-pressure and high-resistance hydrocephalus as, for instance, is seen after subarachnoid hemorrhage. In some patients, there is a possibility of cerebral atrophy accompanied by otherwise insignificant increased R_{out}. In this study, the PVI technique proved to be a fast and safe method of measuring R_{out}.

KEY WORDS • **communicating hydrocephalus** • **normal-pressure hydrocephalus** • **intracranial pressure** • **pressure-volume index** • **cerebrospinal fluid dynamics**

NORMAL-PRESSURE hydrocephalus (NPH), communicating hydrocephalus, and nonobstructive acquired hydrocephalus are all more or less synonymous designations of a potentially treatable condition, commonly characterized by progressive dementia and sometimes accompanied by gait disturbances and urinary incontinence. The condition may be the result of subarachnoid hemorrhage, head injury, or meningitis or may be of unknown etiology, so-called "idiopathic NPH."

Very little is known about the course of events leading to hydrocephalus with presumed normal intracranial pressure (ICP). Since cerebrospinal fluid (CSF) diversion may reverse symptoms and reduce ventricular size, it is assumed that disturbed CSF circulation is of major pathogenetic significance. It has been shown that resistance to outflow of CSF (R_{out}, the reciprocal of conductance) is increased in patients with NPH in spite of a seemingly normal ICP.[5,14] From the relationship between R_{out} and ICP defined by the steady-state equation for $ICP = I_f \times R_{out} + P_{ss}$,[40] where I_f is the formation rate of CSF and P_{ss} the pressure in the sagittal sinus, one would expect a raised ICP as the result of increased R_{out}. Other hypotheses (not necessarily mutually exclusive) have been offered.[5,15,17,22,26,47]

The present study was undertaken to clarify the seemingly contradictory interrelationships between R_{out} and ICP. A second goal was to examine whether abnormal intracranial compliance, which has been thought to play a role in other hydrocephalic conditions,[3,4] is of significance in this condition. The bolus injection method of CSF dynamics analysis described by Marmarou, *et al.*,[39,40] was employed for the following reasons: 1) the technique as described originally has not been evaluated in patients with communicating hydrocephalus, and 2) the method provides data on CSF resistance and pressure-volume relationships during the same test.

The present study was only concerned with CSF dynamics as studied by quantitative techniques and did not employ radioisotopes or x-ray contrast medium. While most studies have been concerned with the selec-

tion of patients who will benefit from CSF diversion surgery, our investigation was exclusively concerned with a pathophysiological analysis of the problem.

Clinical Material and Methods

This study group included 29 patients consecutively admitted to the neurosurgical department between September, 1981, and July, 1983, for consideration of a CSF shunting procedure for suspected NPH. The patients all demonstrated deterioration of mental faculties, and some had headache, dizziness, gait disturbances, and/or urinary difficulties. All patients exhibited ventriculomegaly, defined by linear measurements or ratios calculated from cranial computerized tomography (CT) scans in excess of the 95th percentiles, as indicated by Gyldensted[21] (that is: Evans' ratio > 0.32, or width of the third ventricle > 6.6 mm, or cella media index < 4.2). Some of the clinical features are presented in Table 1.

In the present report, "Evans' ratio" (that is, the widest diameter of the frontal horns divided by the maximum inner skull width) was used as the index of ventricular size. Details on the radiological aspects are reported elsewhere.[33] The study protocol prescribed 1) continuous overnight (24-hour) ICP monitoring; 2) pressure-volume studies; and 3) determination of resistance to outflow of CSF.

Intracranial Pressure Monitoring

Intracranial pressure was monitored by an intraventricular catheter, with the reference point being the external auditory canal. The patients were supine and unmedicated except for their usual daily medication (such as antihypertensive drugs). The ICP was measured and averaged in 5-second periods and registered continuously on a paper chart with a paper speed of 3 cm/hr in the first eight patients, but subsequently increased to 12 cm/hr. These data are referred to as mean ICP, although they may deviate slightly from the true mean (diastolic ICP + $\frac{1}{3}$ the pulse amplitude).

The analysis of ICP was executed visually, which allowed the investigator to disregard ICP elevations that were the result of external factors such as coughing, turning, and urination, while spontaneous pressure waves could be identified. Various methods were tried to obtain a reliable and reproducible estimate of the 24-hour mean ICP, and from that experience the following procedure emerged: ICP was divided in 5-mm Hg segments. For each centimeter recorded, the highest mean ICP value was selected (excluding spikes as mentioned above) and designated by the highest five multiple. So, if the highest ICP value was between 10 and 15 mm Hg and not exceeding the latter, the value 15 was given. When $\frac{1}{2}$ to 2/min waves (B-waves) were present, their baseline ICP was used. The numbers were added and divided by the number of 1-cm periods counted. This number, an estimate of the average ICP although of course slightly greater, was termed "ICP_{24}."

The appearance of pressure waves was noted and the percentage of $\frac{1}{2}$ to 2/min waves[36] was calculated as the mean of three independent counts. The pulse amplitude, which is the difference between the systolic and diastolic ICP, was extracted from the record just prior to the CSF manipulations and in the same phase that was used for the bolus test calculations. It was read with 0.5-mm Hg exactitude.

Pressure-Volume Studies

Pressure-volume studies were undertaken at the end of the monitoring period using the bolus technique of Marmarou described in detail previously.[31,39,40] In brief, while pulsatile ICP was being recorded, a bolus (1 ml/sec) of varying amounts of fluid (2 to 8 ml) was injected intraventricularly via a three-way stopcock; the following equations were employed to calculate pressure-volume index (PVI) and resistance to outflow of CSF (R_{out});[39,40]

$$PVI = \frac{\Delta V}{\log \frac{P_p}{P_o}}, \quad (1)$$

and

$$R_{out} = \frac{t \cdot P_o}{PVI \log \left(\frac{P_t}{P_p} \cdot \frac{P_p - P_o}{P_t - P_o} \right)}, \quad (2)$$

where P_o indicates baseline ICP before injection, P_p the peak ICP as a result of the injected bolus volume ΔV, and P_t the pressure at time t; all pressure values are conventionally diastolic in the same respiratory phase. An example of the results of a bolus injection is seen in Fig. 1. In two patients the bolus test failed and CSF resistance was determined with a constant-rate steady-state infusion using infusion rates of 0.51 and 1.02 ml/min, respectively. Resistance to outflow of CSF was calculated using Equation 3:[40]

$$R_{out} = \frac{P_{eq} - P_o}{\Delta Inf}, \quad (3)$$

where P_{eq} is equilibrium pressure obtained after infusion at rate ΔInf. A high degree of correlation has previously been found between resistance values obtained with the two methods.[32]

In four patients (Cases 11, 21, 22, and 28) the tests were performed under general anesthesia using N_2O, a short-acting barbiturate, and fentanyl, although it was known that the latter drug may influence resistance to CSF outflow during prolonged studies.[1] Otherwise, studies were undertaken without any medication. In two of the four above-mentioned patients (Cases 11 and 28), continuous ICP monitoring could not be completed because of severe dementia. In two patients (Cases 23 and 28) no resistance values were obtained; PVI values were obtained in all patients. At the termination of the studies, CSF was withdrawn for cell count and bacteriological studies.

Linear regression was calculated using the least-square method with significance for correlation coefficients (r) as in the Ciba-Geigy Scientific Tables.

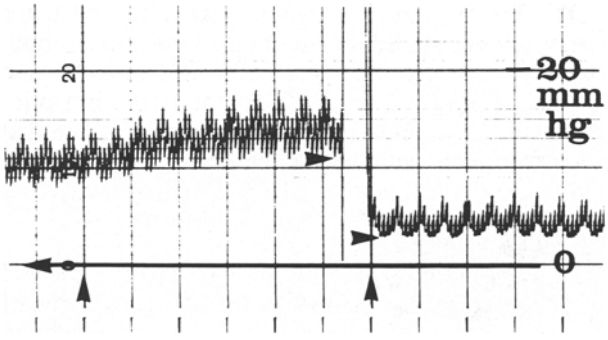

FIG. 1. Example of a tracing after a bolus injection. The recording reads from right to left, and shows the pulsatile intracranial pressure (ICP). The interval between the *vertical arrows* is 1 minute. *Horizontal arrowheads* indicate diastolic ICP values (P_o, the baseline ICP before injection, and P_p, the peak ICP after injection). These values are used in equations for pressure-volume index (PVI) and resistance to outflow of cerebrospinal fluid (R_{out}).

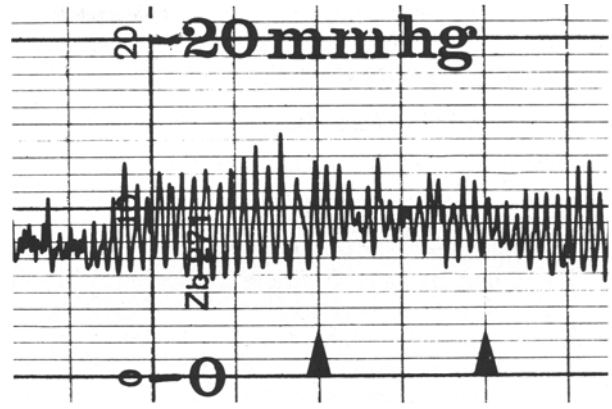

FIG. 3. Intracranial pressure (ICP) waves occurring at $\frac{1}{2}$ to 2/min (B-waves) of varying amplitude. The recording reads from right to left, and shows the mean ICP (not pulsatile). The interval between *arrowheads* is 10 minutes.

Results

Intracranial Pressure

In 19 patients mean ICP never exceeded 15 mm Hg. In two the pressure briefly rose to 20 mm Hg. Four patients had one or two trains of B-wave-like activity of high amplitude (Fig. 2), not quite similar to the ramp-waves described by Brock[8] (M Brock, personal communication, 1983); otherwise the ICP was below 15 mm Hg in these patients. Two patients (Cases 1 and 15) had markedly raised ICP (> 15 mm Hg). One of these patients (Case 1) presented with an acoustic neurinoma more than 2 years later. In the two patients in whom continuous ICP monitoring was not possible (Cases 11 and 28) the opening pressure during anesthesia was normal (5.7 and 5.2 mm Hg, respectively). The results of ICP_{24}, the opening mean pressure ($\bar{P}_o$), the PVI studies, and the frequency of B-waves, which varied from 3% to 58%, appear in Table 1. This variation between patients caused unexpected difficulty in delineating precise B-wave activity. Thus, the median variation of the three B-wave counts (the greatest value divided by the smallest) was 1.9. Because of the slow paper speed, B-wave activity could not be analyzed in the first eight patients. The amplitude of the B-waves (not the pulse amplitude during waves) varied from 2 to 16 mm Hg. Even in the same patient, the amplitude variation was considerable (Fig. 3). There was a poor correlation between the frequency of B-waves and ICP_{24} ($r = 0.52$, $0.01 < p < 0.05$) and B-waves and resistance to CSF outflow ($r = 0.46$, $p = 0.05$), and there was no correlation with PVI.

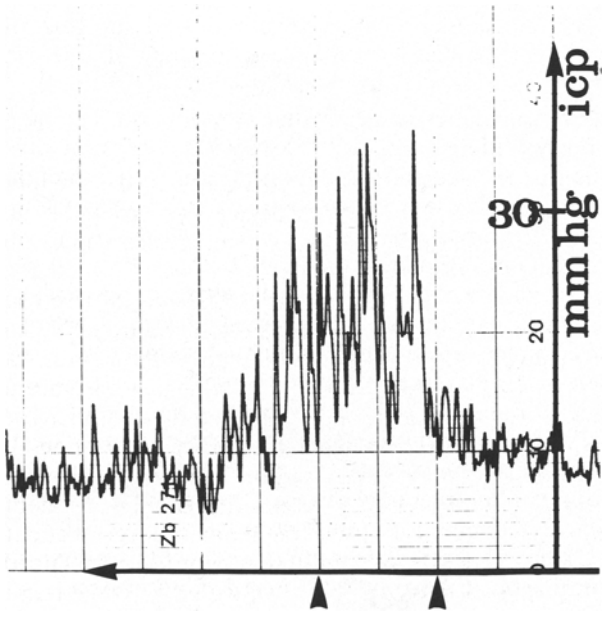

FIG. 2. The intracranial pressure (ICP) wave activity seen here was recorded in four patients (see text). The frequency was similar to that of B-waves. The recording reads from right to left, and shows the mean ICP (not pulsatile). The interval between *arrowheads* is 10 minutes.

Pulse Amplitude

The pulse amplitude varied from 0.5 to 10 mm Hg (median 4 mm Hg). There was no correlation between the amplitude and the ICP at the time the amplitude was measured ($r = 0.33$), nor with Evans' ratio or PVI.

Pressure-Volume Index

The PVI varied from 4.6 to 18.2 ml (median 8.0 ml). There was no difference in PVI relating to whether the etiology was known or unknown. The PVI correlated poorly with baseline ICP when PVI was determined (P_o) and with the size of the ventricles as defined by Evans' ratio on CT scanning (correlation coefficient for both: $r = 0.44$). There was no correlation with ICP_{24}.

TABLE 1

*Clinical features and results of ICP measurements in 29 patients with suspected communicating hydrocephalus**

Case No.	Sex, Age (yrs)	Etiology	ICP_{24} (mm Hg)	$\bar{P}_o$ (mm Hg)	PVI (ml)	R_{out} (mm Hg/ml/min)	Pulse Amplitude (mm Hg)	% B-Waves	Evans' Ratio
1	F, 66	unknown	20.8	8.7	10.7	19.3	5.0	—	0.41
2	F, 65	unknown	9.7	4.0	10.6	6.9	3.0	—	0.36
3	M, 48	SAH	9.5	2.3	8.0	16.4	2.5	—	0.41
4	F, 69	unknown	9.3	3.7	6.1	6.8	5.0	—	0.39
5	M, 18	trauma + meningitis	10.8	8.5	13.3	10.3	3.0	—	0.45
6	F, 63	unknown	11.5	3.8	9.7	14.9	4.0	—	0.34
7	M, 78	unknown	16.3	8.2	7.9	27.8	5.0	—	0.39
8	F, 75	unknown	5.0	2.7	6.7	5.1	2.0	—	0.28
9	F, 37	CVA	5.9	2.3	8.2	6.3	1.0	18	0.38
10	F, 71	SAH	9.1	2.5	7.3	14.5	4.5	18	0.36
11	F, 68	unknown	—	5.7	6.9	13.9	0.5	—	0.29
12	M, 64	trauma(?)	5.2	3.2	8.2	2.5	3.5	28	0.35
13	F, 79	CVA	9.0	3.3	4.6	10.7†	5.5	16	0.30
14	F, 73	unknown	6.3	2.0	5.7	9.9	6.0	39	0.39
15	M, 76	unknown	24.4	14.3	13.6	31.4†	7.0	58	0.45
16	F, 67	unknown	9.8	2.3	6.0	9.1	4.0	26	0.32
17	F, 79	unknown	7.1	6.3	5.0	3.8	10.0	36	0.36
18	M, 53	unknown	9.4	5.0	18.2	6.9	4.5	8	0.43
19	M, 76	unknown	10.3	8.5	7.3	10.8	7.5	12	0.42
20	F, 55	SAH	6.2	3.5	7.2	11.6	1.5	9	0.34
21	F, 59	CVA	8.6	7.3	14.2	5.2	2.5	20	0.31
22	F, 52	trauma	11.4	4.3	7.8	10.0	1.0	28	0.40
23	M, 16	trauma	8.7	6.7	17.8	—	2.0	12	0.41
24	M, 79	unknown	5.9	2.3	6.3	7.6	4.0	14	0.40
25	F, 58	unknown	10.4	5.3	17.8	4.6	4.0	3	0.37
26	M, 68	unknown	10.9	4.7	7.7	12.3	5.0	16	0.34
27	M, 69	unknown	8.7	4.2	8.7	5.7	5.0	18	0.39
28	M, 8	trauma	—	5.2	13.9	—	3.5	—	0.54
29	F, 40	postirradiation	13.1	6.7	8.7	28.2	3.5	22	0.34

* ICP = intracranial pressure; ICP_{24} = average overnight ICP; $\bar{P}_o$ = mean ICP at test time; PVI = pressure-volume index; R_{out} = resistance to outflow of cerebrospinal fluid; — = not available; SAH = subarachnoid hemorrhage; CVA = cerebrovascular accident. For a definition of Evans' ratio see text.

† Values obtained by infusion test.

Resistance to Outflow of CSF

The R_{out} varied from 2.5 to 31.4 mm Hg/ml/min (median 10.0 mm Hg/ml/min). A plot of R_{out} versus the average overnight ICP (ICP_{24}) is seen in Fig. 4. A linear regression led to the equation: $y = 0.47x + 4.8$ with correlation coefficient $r = 0.80$, which is highly significant ($p < 0.01$). Correlation between R_{out} and the opening pressure (P_o) was also significant but far less strong ($r = 0.58$). There was no difference in resistance values depending on whether the etiology was known or unknown.

In Table 2 the contribution of the "CSF dynamics" (that is, the $I_f \times R_{out}$ product) to the total ICP has been computed. A theoretical formation rate of 0.35 ml/min has been employed.[12,46] The contribution has been calculated as a percentage of the ICP at the time of the test (P_o) as well as the overnight ICP (ICP_{24}). The median contribution to the former was 60%, and to the latter 37%. In 10 patients the theoretical contribution to P_o exceeded 100%, suggesting either that the formation rate estimate was too high or that the sagittal sinus pressure was negative. These patients did not contribute to the calculation of the median contribution. The variation coefficient for the PVI and R_{out} determinations (that is, the standard deviation of each determination expressed as a percentage of the mean[55]) was 13.0% and 13.7%, respectively.

Mainly on the basis of these measurements, 14 patients underwent CSF diversionary surgery. The results assessed at follow-up examination at least 3 months after surgery are given in Table 3. Three patients could not be evaluated: in Case 10 the device was removed due to infection; in Case 19 surgery was complicated by a large hematoma, leaving the patient hemiplegic; and the patient in Case 26 was an alcoholic and died from cardiopulmonary disease 3 months after surgery. Of the remaining 11, nine improved.

If the patients who improved following surgery are considered to have had NPH and those not operated on to have had atrophy (see Discussion) the medians of the PVI were 9.7 and 8.2 ml, respectively. Likewise, the medians of the pulse amplitude in the two groups were 3.5 and 4.0 mm Hg, respectively. For both parameters there was a wide range of values.

TABLE 2

*The theoretical contribution of the $I_f \times R_{out}$ product to intracranial pressure**

Case No.	$I_f \times R_{out}$ (mm Hg)	% Opening ICP (P_o)	% ICP_{24}
1	6.8	78	33
2	2.4	60	25
3	5.7	—	60
4	2.4	65	26
5	3.6	42	33
6	5.2	—	45
7	9.7	—	60
8	1.8	67	36
9	2.2	96	37
10	5.1	—	56
11	4.9	86	NA
12	0.9	28	17
13	3.7	—	41
14	3.5	—	56
15	11.0	77	45
16	3.2	—	33
17	1.3	21	18
18	2.4	48	26
19	3.8	45	37
20	4.1	—	66
21	1.8	25	21
22	3.5	81	31
23	NA	NA	NA
24	2.7	—	46
25	1.6	30	15
26	4.3	91	39
27	2.0	48	23
28	NA	NA	NA
29	9.9	—	76

* I_f = formation rate of cerebrospinal fluid (CSF); R_{out} = resistance to outflow of CSF; ICP_{24} = average overnight intracranial pressure; — = indicates product > 100% (see text); NA = not assessable.

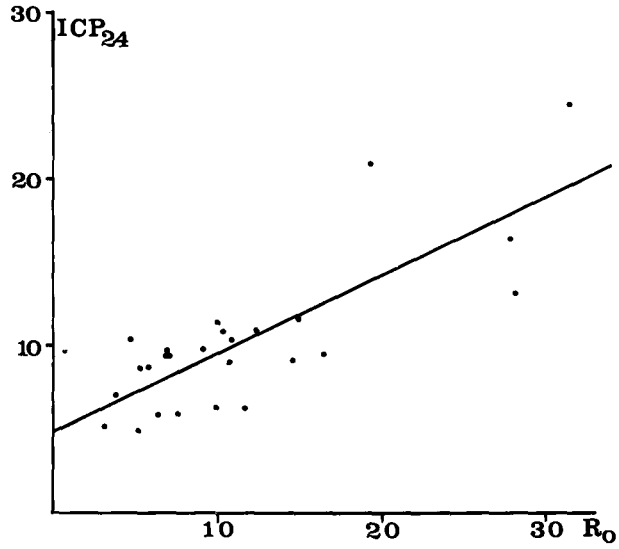

FIG. 4. Plot of resistance to cerebrospinal fluid outflow (R_o, in mm Hg/ml/min) versus the average overnight intracranial pressure (ICP_{24}, in mm Hg). The linear regression line in indicated (see text).

Discussion

The syndrome of hydrocephalus with supposedly normal ICP still presents an enigma. Most papers have been concerned with the selection of patients who might benefit from shunting.[2,6,9–11,13,14,16,20,23,27,29,34,37,38,41–43,48–50,52–54] The interpretation of data is complicated by the use of varying, often vague definitions of communicating hydrocephalus. In the literature, the distinction between hydrocephalus and cerebral atrophy (hydrocephalus *ex vacuo*) may be based on clinical ground, radiographic or isotope studies, or the diagnosis may just be taken for granted.

Since the answer to this enigma has not yet been found, no single test can at present give the ultimate answer. To define hydrocephalus by the response to CSF diversionary surgery could be a reasonable approach, but is biased by the surgeon's selection criteria. Hardly any surgeon would offer surgery known to be connected with a considerable frequency of serious complications to a patient most likely suffering from cerebral atrophy, and neither do we. By the same token, the significance of laboratory tests remains uncertain when a control group (that is, asymptomatic humans with enlarged ventricles) is not available for investigation. These considerations also apply in the interpretation of the present data. Although the rate of patient improvement in the present study suggests that the tests employed were useful in the selection of patients for shunting, it does not prove a causal role of abnormal CSF dynamics in communicating hydrocephalus.

In the steady-state equation[40] ($ICP = I_f \times R_{out} + P_{ss}$), ICP can be interpreted as a linear function of R_{out} with the slope I_f and y intercept P_{ss} — a relationship that is supported in the present study. Assuming that R_{out} is increased, an unchanged ICP can only be maintained by a reduction of the production rate of CSF or the sinus pressure. Without such a reciprocal change, ICP must invariably rise, although it may still be within limits considered normal. Compared with normal data using the same technique,[46] resistance was abnormally elevated in almost all patients. Raised resistance (low conductance) has also been found by others in communicating hydrocephalus, using other techniques.[5,14] Although the formation rate was not determined in the present study, there is little doubt that the CSF dynamics (represented here by the $I_f \times R_{out}$ product) contributed many times more to the ICP in these patients than in normal individuals, where only approximately 10% is contributed by the $I_f \times R_{out}$ product.[40,46]

Actual data about CSF formation rate under pathological conditions are few. Most investigators agree that the CSF formation rate in man is relatively unaffected by induced changes in ICP,[4,12,35] but there are suggestions that the production rate may be diminished under certain pathological circumstances.[18,31,44] From the above-mentioned considerations, it follows that an increase in ICP is a likely consequence of the increased resistance. The statement that ICP is not truly normal

in this clinical entity is in concert with the finding reported by others that increased ICP is suggestive of communicating hydrocephalus requiring shunt placement.[9,20,23,29,34,49] Granholm and Löfgren[19] found ICP within normal limits in patients with NPH and atrophy, but the ICP was higher in the NPH group. Also, the occurrence of B-waves is considered "diagnostic" for NPH (that is, indicative of shunt surgery),[5,9,11,43,49] although the cause of these waves remains obscure. Børgesen, *et al.*,[7] related the occurrence of B-waves to increased resistance and (low) compliance. Such a relationship was only partially supported by the present study. Surprisingly, the frequency of B-waves was lower in the present series than in Børgesen's study in spite of the inclusion of patients with greater ICP in our group.

Studies of the pulse pressure in this condition have been conducted by Di Rocco, *et al.*,[13] Maira, *et al.*,[38] and Foltz and Aine,[16] who found increased pulse amplitude in NPH. Di Rocco, *et al.*, suggested that the large pulse amplitude might be causal in the pathogenesis of communicating hydrocephalus. However, it is possible that the larger pulse amplitude might be the result of higher ICP in NPH patients as compared with patients with cerebral atrophy, although such a relationship was not clearly seen in the present study.

TABLE 3

Resistance values (mm Hg/ml/min) in relation to surgical results

Case No.	Surgery, Improvement	Surgery, No Improvement	Surgical Complications	No Surgery
1	19.3	—	—	—
2	—	—	—	6.9
3	16.4	—	—	—
4	—	—	—	6.8
5	—	—	—	10.3
6	14.9	—	—	—
7	27.8	—	—	—
8	—	—	—	5.1
9	—	—	—	6.3
10	—	—	†	—
11	—	13.9	—	—
12	—	—	—	2.5
13	—	—	—	10.7*
14	—	9.9	—	—
15	31.4*	—	—	—
16	—	—	—	9.1
17	—	—	—	3.8
18	—	—	—	6.9
19	—	—	†	—
20	—	—	—	11.6
21	—	—	—	5.2
22	10.0	—	—	—
23	†‡	—	—	—
24	—	—	—	7.6
25	—	—	—	4.6
26	—	—	†	—
27	—	—	—	5.7
28	†‡	—	—	—
29	28.2	—	—	—

* Infusion test.

† Patients could not be assessed due to complications.

‡ No resistance value available.

Pressure-volume studies in patients with NPH have not attracted wide attention.[7,51] Tans and Poortvliet[51] found diminished PVI in the majority of patients with adult hydrocephalus. We found highly abnormal pressure-volume conditions in the entire group compared to the normal values for PVI (approximately 25 ml) reported by Shapiro, *et al.*[46] However, our patients were much older than the normal individuals studied by Shapiro, *et al.*, and aging of the brain in itself may influence pressure-volume relationships. Sklar, *et al.*,[47] found an increased elastance slope (corresponding to decreased PVI) in patients with communicating hydrocephalus, and hypothesized that the abnormal elastance slope could be of importance in the pathogenesis. Our finding of diminished PVI should be compared with the findings of Shapiro, *et al.*,[45] who found increased PVI in pediatric hydrocephalus and suggested that the increased compliance (that is, less stiffness) might facilitate the development of hydrocephalus. These results suggest that the development of NPH is fundamentally different from that of infantile hydrocephalus.

The finding of diminished PVI in the present group of patients with enlarged ventricles makes it less likely that the neural axis compliance in this entity is a function of craniospinal volume, as was inferred from the study of Shapiro, *et al.*,[46] in normal subjects. There was indeed a statistically significant but weak correlation between PVI and ventricular size, with a correlation coefficient in the same order of magnitude as reported by Sklar, *et al.*,[47] but we would by no means infer any causal relationship from these data. The diminished PVI might be a result of qualitative or quantitative changes in the compartment that accommodates the injected bolus (which probably is the venous pool[40]) or due to changes in the periventricular tissue.[17] As far as any proposed causal relationship between pressure-volume conditions or pulsatile force and hydrocephalus is concerned,[3,47] that contention was neither supported nor refuted by the present study.

In experimental hydrocephalus, ICP is initially increased as a result of obstructed CSF outflow, after which it approaches normal levels as the ventricles dilate.[4,25,28] The development of communicating hydrocephalus subsequent to subarachnoid hemorrhage probably takes the same course. In the acute phase ICP is elevated, mainly as a result of increased resistance to CSF absorption even in the absence of frank hydrocephalus.[31] Later, both ICP and R_{out} approach normal, likely via an intermediate phase of high-pressure hydrocephalus with increased resistance.[18] One of the patients reported here (Case 20; reported as Case 9 elsewhere[31]) supports this contention. She was studied in the acute phase after subarachnoid hemorrhage, at which time ICP and resistance were elevated (R_{out} = 85 mm Hg/ml/min) in spite of the absence of hydrocephalus. The present study was undertaken 4 months later, when R_{out} was 11.6 mm Hg/ml/min.

It is possible that ICP and R_{out} may approach normal such that therapy aimed at a normalization of R_{out} is in vain; the condition may then be indistinguishable from primary cerebral atrophy. The lack of therapeutic success in that situation may also be the result of irreversible brain damage secondary to low cerebral blood flow.[24] It is speculative whether subclinical subarachnoid hemorrhage, spontaneous or traumatic, is responsible for "idiopathic" communicating hydrocephalus in some cases or if other mechanisms are in play. It is conceivable that some cases of idiopathic NPH may represent atrophic brain disease accompanied by otherwise insignificant increased resistance to CSF outflow.[30] This statement is in accordance with the finding that almost all patients in the present study had increased resistance.

In conclusion, the present work supports the concept that abnormal CSF dynamics play an important role in communicating hydrocephalus, while the significance of abnormal pressure-volume relationships remains speculative. The resistance to CSF outflow is a major determinant of the ICP which, strictly speaking, is not normal. Patients with communicating hydrocephalus represent a continuum ranging from those with unequivocally elevated ICP and high resistance through those with intermediate values to nearly normal pressure and resistance, which may represent a stage that each patient passes through. Based on these considerations, one may question whether the normally accepted upper limit for ICP (approximately 15 mm Hg) is too high, and whether the term "normal-pressure hydrocephalus" is indeed appropriate.

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