Hydrocephalus shunts and waves of intracranial pressure

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Abstract—*The majority of contemporary hydrocephalus valves are designed to introduce a low resistance to flow into the cerebrospinal fluid (CSF) drainage pathway, and an therefore intended to stabilise intracranial pressure (ICP) at a level close to the shunt's operating pressure. However, this goal cannot always be attained. Accelerated CSF drainage with vertical body posture in ventriculo-peritoneal shunts is one reason for the ICP decreasing below the shunt's operating pressure. Another possible factor has been studied: the impact of the pulsating pattern in the ICP on the operating pressure. Six popular constructions of medium-pressure valves were studied (Radionics Low-profile, Delta, Hakim Precision, Holter, Integra In-line and Hakim NMT). Valves were mounted in the testing rig in the UK. Shunt Evaluation Laboratory and perfused with de-ionised water at a rate of 0.3 ml min^{-1}, and proximal pulsating pressures of different amplitudes (from 2 to 30 mm Hg peak-to-peak) and frequencies (70–10 cycles min^{-1}) were superimposed. Laboratory findings were compared with clinical material containing recordings of ICP made in patients to diagnose reasons for ventriculomegaly. The mean operating pressure decreased in all valves when the simulated amplitude of heart pulsations increased. The rate of this decrease was dependent on the type of valve (variable from 2.5 to 5 mm Hg per increase in peak-to-peak amplitude by 10 mm Hg). The decrease was not related to the frequency of the wave. The relationship between pulse amplitude and ICP in 35 patients with blocked shunts was strong ($R = 0.48$; $p < 0.03$; slope 0.14) and in 25 patients with properly functioning shunts was non-significant ($R = 0.057$; $p = 0.765$). Two examples of decrease in mean ICP in the presence of increased vasogenic ICP waves in shunted patients are presented. The shunt operating pressure, which 'sets' the ICP in shunted patients may be influenced by the dynamics of a patient's ICP waveform.*

Keywords—*Hydrocephalus, Shunt, Intracranial pressure, CSF drainage*

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1 Introduction

CEREBROSPINAL FLUID (CSF) is derived by active secretion from cerebral arterial blood. The rate of CSF production is reported to be constant under normal conditions (DAVSON *et al.*, 1987). Produced mainly in the lateral and third ventricles, CSF flows along the aqueduct of Sylvius to reach the fourth ventricle, then passes into the subarachnoid space and is absorbed mainly to sagittal sinus blood.

Drainage of CSF into the venous compartment takes place predominantly (in humans) through arachnoid granulations that penetrate the walls of the sagittal sinus. It is important to recognise that reverse transport through the arachnoid granulations is impossible, i.e. drainage ceases if the subarachnoid ICP is less than the sagittal sinus pressure (DAVSON *et al.*, 1987). The nature of the venous drainage is linear, i.e. proportional to the pressure gradient between the CSF side of the granulation and the sagittal sinus. The inverse of the proportionality coefficient is called the resistance to CSF outflow R_{csf} and has been assessed in normal subjects as ranging from 6 to 10 mm Hg (ml min^{-1})$^{-1}$ (ALBECK *et al.*, 1997).

Undisturbed circulation of CSF is one of the fundamental mechanisms ensuring favourable conditions for the structures of the central nervous system. First, the brain and spinal cord float in the fluid, losing their relative weight according to Archimedes' law. As a result, these structures are less prone to injury in the case of mechanical shocks. Secondly, all gradients of the intracranial pressure are cancelled out by the free circulation of CSF fluid. Therefore there is neither a pressure gradient, nor a risk of volume shifts or herniation.

Obstruction of CSF flow between the third and fourth ventricles produces an accumulation of CSF in the lateral and third ventricles. This is described as non-communicating hydrocephalus and can develop owing to congenital/secondary stenosis or a mass occluding the aqueduct. Endoscopic fenestration of the floor of the third ventricle (ventriculostomy) by a trained specialist has recently become the preferred treatment for this condition.

In patients with communicating hydrocephalus, where impaired CSF flow occurs in the subarachnoid space (e.g. post meningitis, post-haemorrhagic), third ventriculostomy is unlikely to be successful, and shunting is the treatment of

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choice (LUNDKVIST *et al.*, 2001; MARMAROU *et al.*, 1978). An ideal shunt should restore the normal circulation of CSF and the normal pattern of extrachoroidal fluid flow within the brain, prevent excessive build-up of intracranial pressure and encourage restitution of the cerebral mantle, comprising both grey and white matter. As shunting is a purely mechanistic method of treatment, the biomechanics of a patient's pressure–volume compensation should ideally be examined before a shunt is implanted (CZOSNYKA *et al.*, 1988).

The majority of contemporary differential-pressure shunts have a low hydrodynamic resistance (ASCHOFF *et al.*, 1995). This reflects their intended ability to stabilise the patient's intracranial pressure (ICP) at, or below, the level of their operating pressures, which is usually fixed at 'low', 'medium' or 'high' (with the exception of a few programmable models). The stability of the ICP can be influenced by different factors. Average ICP can be lowered by the 'siphoning effect', if there is no device added to prevent this phenomenon (ASCHOFF *et al.*, 1995; CZOSNYKA *et al.*, 1988). It can also be lowered by excessive drainage secondary to slow vasogenic ('B') waves, which usually arise at night (CZOSNYKA *et al.*, 1988; DAVSON *et al.*, 1987; MOMJIAN *et al.*, 2004).

In shunted patients, baseline CSF pressure in the horizontal body position should result from the parallel flow of CSF through the shunt and along any residual CSF reabsorption pathway. Therefore mean ICP over a period of time (half an hour or longer) should not exceed this level (CZOSNYKA *et al.*, 1988; LUNDKVIST *et al.*, 2001). If it does, it is an indication of a shunt's under-performance, in most cases related to total or partial obstruction.

Little has been said about the pulse (heart-related) and respiratory ICP waveforms and their influence on mean ICP in shunted patients in the horizontal body position. The pulse amplitude of ICP can be, on average, 3–5 mm Hg (peak-to-peak) but, in some instances, can be as high as 10–30 mm Hg. Similarly, particularly during deep breathing, the amplitude of the respiratory wave can rise as high as 25 mm Hg. Slow waves demonstrate an amplitude, on average, of 2–5 mm Hg, but they may well exceed 40 mm Hg in pathological circumstances (AVEZAAT *et al.*, 1979; MARMAROU *et al.*, 1987). One of the fundamental physiological effects observed in cerebrospinal space is an increase in the amplitude of vasogenic components when mean ICP increases (AVEZAAT *et al.*, 1979; CZOSNYKA *et al.*, 1988; MOMJIAN *et al.*, 2004).

We have used laboratory shunt-testing methodology (UK Shunt Evaluation Laboratory) to construct a model of the CSF circulation and to evaluate the influence of waveforms of different magnitudes on the operating pressure in six popular models of the shunts. We also attempted to illustrate, using recordings of ICP taken from clinical practice, that non-linear hydrodynamic performance of the shunt can influence the physiology of normal CSF dynamics, with particular attention to vasogenic ICP waves.

2 Material and methods

2.1 *Types of valve used*

Six types of hydrocephalus shunt, representing different constructions and specific strategies for CSF drainage control, have been investigated. The shunts can be divided into three groups, according to the type of valve incorporated.

The Holter* valve consists of a curved silicone rubber layer incorporating a slit. The size of the slit determines both the hydrodynamic resistance and the opening pressure of the valve.

*Radionics, Inc., Terry Avenue, Burlington, MA 01803 USA

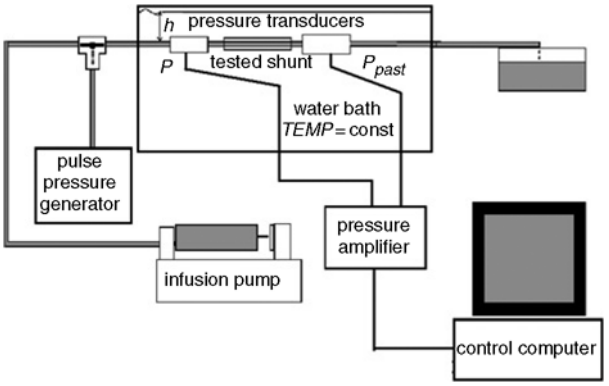

Fig. 1 *Schematic diagram of measurement system. Shunt is submerged in water bath (depth h) at constant temperature. Two pressure transducers (proximal P and distal P_{past}) measure pressure waveforms before and past shunt. Shunt is perfused at constant rate through syringe infusion pump. Sine wave pressure pattern is added before shunt from pulse pressure generator. Amplitude and frequency of this waveform can be controlled*

The Integra in-line shunt† valve contains a mitre of two flat, horizontally opposed leaflets of silicone converging on a round orifice. The mitre acts as a 'Starling resistor'. The size, shape and dimensions of the two leaflets determine the pressure characteristics.

The Radionics low-profile‡ and Delta** valves incorporate a membrane. In such valves, a mobile flexible membrane moves in response to the differential pressure across it. The pre-load force applied and the stiffness of the membrane determine the valve's pressure characteristics.

The Hakim precision†† and Hakim NMT‡‡ valves both incorporate ball-on-spring valves. These consist of a metallic spring that applies force to a ball located in an occluding orifice. The opening pressure of the valve is determined by the spring's stiffness.

All types tested were medium-pressure valves.

2.2 *The testing equipment*

The test was modified from that used in the UK Shunt Evaluation Laboratory (CZOSNYKA *et al.*, 1988), as illustrated in Fig. 1. The shunt under investigation was submerged in a water bath (depth h around 1.5 cm) at 37°C and infused with de-ionised and de-aerated water (with physical properties that represent CSF under normal conditions) by an infusion pump***. A sinusoidal pulse pressure of controlled amplitude and frequency created by the pulse pressure generator††† (the generator controls the pressure) can be added to the static pressure.

The input pressure to the shunt was measured with a luer lock pressure transducer that was submerged in the water bath. The output pressure P_{past} was measured using the second transducer of the same type. The resistance of the outlet tubing was 0.9 mm Hg $(\text{ml min}^{-1})^{-1}$.

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‡Medtronic PS Medical, 125 Cremona Drive, Goleta, California 93117-5500, USA
**Heyer-Schulte NeuroCare, 8400 Lakeview Parkway, Suite 600, PO Box 390, Pleasant Prairie, WI 53158-0390, USA
††Radionics, Inc., Terry Avenue, Burlington, MA 01803 USA
‡‡Integra SA, Biot Operation, Sophia Antipolis, F-06560 Valbonne, France
***55-2222, Harvard Apparatus, Southnatick, MA, USA
†††601A, Biotek Instruments, USA

Measurement was controlled by a standard IBM PC with software designed in-house. The pressure waveform was analysed by the computer using a spectral analysis algorithm. A 512 point discrete Fourier transform of the signal, sampled with a frequency of 20 Hz, was calculated. A discrete power spectrum was estimated from the complex series. The dominant frequency was traced using a simple maximum-seeking algorithm. The harmonic amplitude was retrieved using an interpolation algorithm (CZOSNYKA *et al.*, 1988) and converted to peak-to-peak amplitude.

This algorithm was designed to minimise the influence of mechanical disturbances such as vibrations, gravitational waves and vortices in the water bath.

2.3 *Protocol*

The proximal (P) and distal (P_{past}) pressures to the shunt were measured with the pulse amplitude changing in approximately 2 mm Hg steps from about 2 mm Hg to a maximum of 50 mm Hg of peak-to-peak values. Two frequencies were investigated: 70 cycles min^{-1} (mimicking pulse pressure) and 10 cycles min^{-1} (mimicking respiratory wave). Three samples of each valve were tested. Measurements in each valve were repeated a minimum of three times so that any transitional or unstable patterns could be investigated.

2.4 *Statistical methods*

Mean values and standard deviations from three samples and number-independent tests were used, along with standard deviations, with careful prior examination of normal distributions of the data points. For each frequency and peak-to-peak amplitude level, two sets of nine data points were tested used an unpaired t-test.

2.5 *ICP monitoring in clinical practice*

Examples of overnight ICP monitoring in patients with a shunt *in situ* illustrate the practical implications of this laboratory study. A subdural Camino ICP probe was inserted under general anaesthesia (non-dominant hemisphere), and the waveform was computer-monitored and analysed overnight. The ICP waveform was processed through Fourier transform analysis so that we could determine the pulse amplitude of ICP *AMP*, as the magnitude of the first harmonic component related to the heart rate, and *SLOW*, as the power of frequency components between 0.05 and 0.0055 Hz. This is equivalent to the period from 20 s to 3 min; the range is a little bit wider than original Lundberg B waves (30 s–2 min). The magnitude of the respiratory waves *RESP* was expressed as the power of the frequency components between 0.2 and 0.05 Hz (Fig. 2).

This method of overnight ICP monitoring is standard clinical practice, used to diagnose patients with hydrocephalus or shunt-related problems in Addenbrooke's Hospital, Cambridge, UK. The patients whose recordings are illustrated in Fig. 6 had a Codman–Hakim precision valve *in situ*. Monitoring was performed because they started to deteriorate clinically after being relatively well just after shunt implantation.

2.6 *Normal physiological pattern*

In physiological conditions, all vasogenic waves of ICP exhibit a tendency to increase when mean ICP increases (AVEZAAT *et al.*, 1979; CZOSNYKA *et al.*, 1988; MARMAROU *et al.*, 1978). This was traditionally modelled by an exponential relationship between the volume of the cerebrospinal space and mean ICP (see Fig. 3*a*). If pulsations of the volume, plotted along the x-axis (in this Figure, pulsatile changes of cerebral blood volume) occur at different levels of net intracranial volume, they produce a greater pressure response at a greater

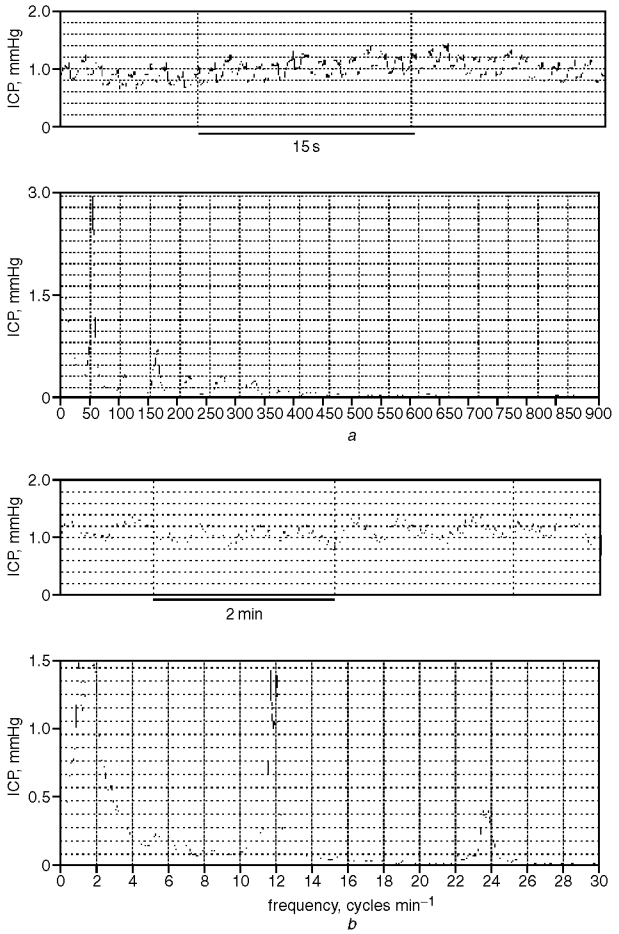

Fig. 2 *Waveforms of ICP and their spectral representations. (a) Pulse wave in time domain and results of spectral analysis (y- axis: equivalent amplitude of component, mmHg, x-axis: frequency cycles min^{-1}). Low-frequency hump represents respiratory and slow waves (combined). First sharp peak: fundamental harmonic of pulse amplitude with at least 7 higher harmonic components equally spaced along frequency axis. (b) In different time domain, respiratory wave and regular slow waves (B waves). Pulse component was filtered out. Frequency spectrum shows hump at 2 cycles min^{-1} representing slow waves. Respiratory component forms maximum at 12 cycles min^{-1} (with second harmonic)*

mean ICP level. This phenomenon can be observed in clinical practice, during the so-called infusion test (see Fig. 3*b*) (AVEZAAT *et al.*, 1979; CZOSNYKA *et al.*, 1988; MOMJIAN *et al.*, 2004).

During the test, mean ICP is increased by external constant rate infusion of normal saline into the CSF space. All vasogenic components, described in the preceding paragraph, increase proportionally to the increase in mean ICP (MOMJIAN *et al.*, 2004). One of the aims of the present study was to observe whether the non-linear properties of a hydrocephalus shunt have an influence on this physiological relationship.

3 Results

In all valves, similar patterns of P and P_{past} were recorded. For the low-frequency pressure waveform (simulating the respiratory waveform), pressure P always had an undisturbed sinusoidal shape, whereas P_{past} indicated that flow through the shunt took place only during a fraction of each oscillation (Fig. 4*a*). When peak-to-peak amplitude increased (Fig. 4*b*),

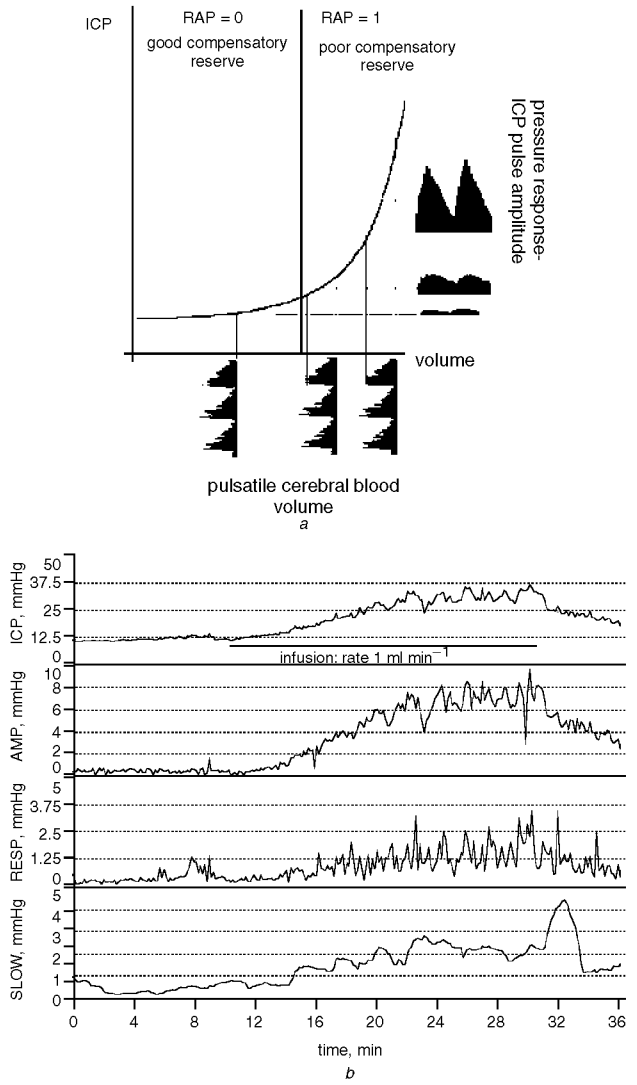

Fig. 3 *(a) In simple model, pulse amplitude of ICP (expressed along y-axis on right side) results from pulsatile changes in cerebral blood volume (expressed along x-axis) transformed by pressure–volume curve. This curve has two zones: flat zone, expressing good compensatory reserve, and exponential zone, depicting poor compensatory reserve. Pulse amplitude of ICP is low and does not depend on mean ICP in first zone. Pulse amplitude increases linearly with mean ICP in zone of poor compensatory reserve (AVEZAAT et al., 1979). RAP is a correlation coefficient between changes in mean ICP and its pulse amplitude. (b) Increasing mean ICP and amplitudes of all vasogenic waves during infusion study (AMP = pulse amplitude; RESP = respiratory wave; SLOW = slow vasogenic waves)*

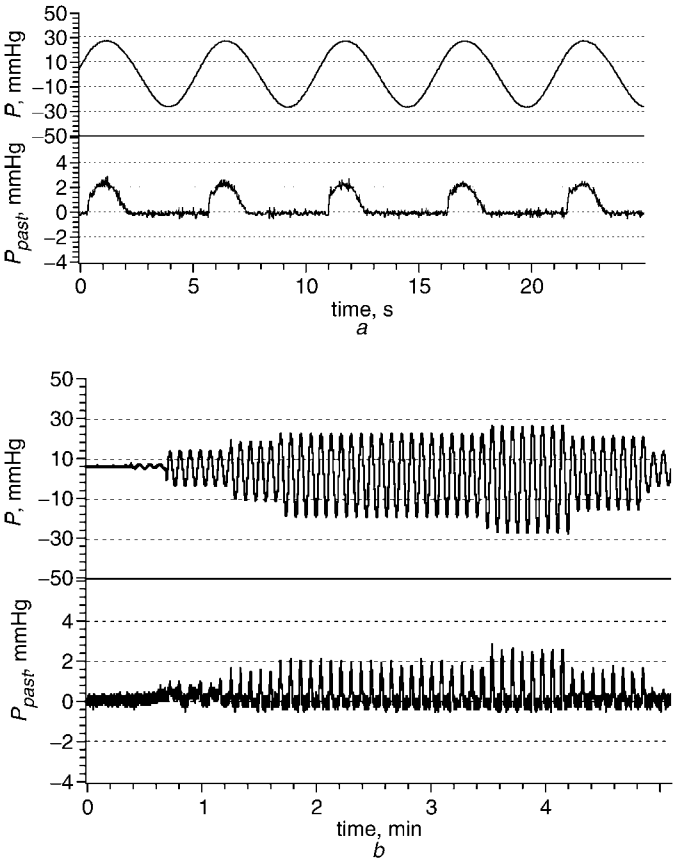

Fig. 4 *(a) Observation of pressures before (P) and after shunt (P_{past}) for slow sine-wave pulsating pressure (mimicking strong respiratory wave). This illustrates that forward flow through shunt takes place only for certain fraction of waveform's period. (b) Observation of pressures before (P) and after shunt (P_{past}) for slow sine-wave pulsating pressure (mimicking strong pulsatile valve). Valve cannot close immediately, therefore short period of backward flow is observed during each period. (both recordings in Fig. 4 are from in-line valve)*

the maximum values of P increased less than the minimum P values. Maximum values of P_{past} increased proportionally to maximum values of P.

The relationships between operating pressure (along y-axis) and the height of the pulse amplitude (70 cycles min^{-1}) for all six valves are illustrated in Fig. 5. In all types, the operating pressure decreased with pulse amplitude above a specific critical amplitude threshold. The thresholds and slope of the descending part of the relationships are summarised in Table 1. There were no significant differences between the results for the 70 cycles min^{-1} and 10 cycles min^{-1} waveforms.

A clinical illustration of the phenomenon observed in the laboratory is not easy to record. This is caused by the fact that the increase in the pulsatile waveform of the ICP is usually associated with a rise in mean ICP, in most cases provoked by an increase in cerebral blood flow and cerebral blood volume. Fig. 6*a* indicates a clear decrease in mean ICP associated with an increase in the respiratory pattern and magnitude of the slow waves, with only a moderate rise in the pulse amplitude of ICP. The patient had a functioning shunt and was asleep. Blood pressure was not recorded, but the patient was normotensive.

The second illustration is derived from an overnight ICP recording in a patient after sub-total resection of a tumour blocking the aqueduct cerebri; see Fig. 6*b*. Non-communicating hydrocephalus was diagnosed, and the patient was shunted pre-operatively (Hakim precision valve). He complained about persistent headaches. When awake, he hyperventilated voluntarily, maintaining a constant pattern of large respiratory waves (up to 40 mm Hg peak-to-peak). Baseline ICP was clearly negative, around −7 mm Hg (Fig. 6*b*). The overnight ICP during sleep was stable, with an average value of 6 mm Hg, without abnormal vasogenic waves, indicating a patent shunt.

Additionally, we analysed mean ICP and pulse amplitude in 60 previously shunted patients (CZOSNYKA *et al.*, 2002) to establish shunt patency. Patients underwent an infusion study to assess shunt functioning *in vivo*. The discrepancy between ICP achieved during the constant rate infusion and the value resulting from the shunt's opening pressure and its hydrodynamic resistance has been taken as a criterion for shunt functioning.

Thirty-five patients were classified as having under-drainage, in 25 patients, drainage was normal. The mean age was 67 years (range 37–88 years) and did not differ between

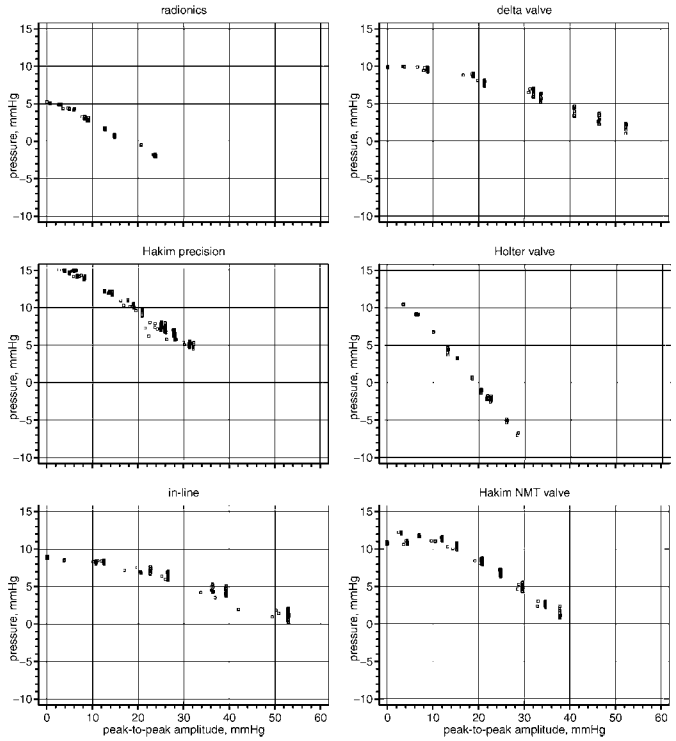

Fig. 5 *Relationships between operational pressure (y-axis) and peak-to-peak pulse amplitude (x-axis) for six types of tested valve. Excessive value of peak-to-peak amplitude has been analysed in laboratory to check whether relationship between mean pressure and its amplitude has monotonically decreasing character. For clinical purposes, only sub-range of values along x-axis can be taken into account, as, in most cases, peak-to-peak ICP pulse amplitude may be limited to 20–25 mm Hg. However, in clinical practice, peak-to-peak amplitude of B waves of 30–50 mm Hg can be recorded, and excessive respiratory waves reaching this value during voluntary deep hyperventilation can also be seen*

groups. Mean ICP was 7+/−5.6 mm Hg (mean+/−SD) in the functioning shunt group and 12.5+/−7.5 mm Hg in the non-functioning shunt group (difference significant at $p < 0.03$, using a Mann-Whitney U-test).

In patients with a functional shunt, the positive relationship between *AMP* and mean ICP (resulting from normal CSF physiology (AVEZAAT *et al.*, 1979)) should be disturbed, as an increase in pulse amplitude should decrease the shunt operating pressure and, hence, baseline ICP (resulting from the non-linear behaviour of the shunt). Indeed, in 35 patients with blocked shunts, the correlation between baseline ICP and amplitude was significant ($R = 0.48$; $p < 0.03$; slope = 0.14), whereas, in 25 patients with a properly functioning shunt, it was non-significant ($R = 0.057$; $p = 0.765$).

4 Discussion

Over the past decade, the clinical neuroscience community has witnessed many enthusiastic reports regarding technological advances in the design of better, more reliable valves. This has had a major impact on shunt prices. Many constructions, particularly programmable valves, have been advertised as devices that are able to fine-tune intracranial pressure. However, it must be remembered that these advanced valves are always inserted as part of a specific patient's complex CSF circulatory system. A shunt's hydrodynamic properties should always be considered in combination with, not in separation from, the hydrodynamic properties of the CSF space.

4.1 *Adequacy of bench test to explain shunt function in vivo*

We demonstrated that, during the bench tests, the pulse waveform can affect the operating pressure of all tested valves, and this effect is proportional to the pulse amplitude. It is very likely that a similar phenomenon affects ICP after implantation: see an example in Fig. 6*a*. The patient had a Hakim precision valve *in situ*. Pulse amplitude *AMP* calculated during the recording was the amplitude of the first harmonic of the pulse waveform of ICP. The maximum rise in this amplitude was 4 mm Hg, which was equivalent to 8 mm Hg peak-to-peak value. With an average linear distortion coefficient of the pulse waveform of ICP of 1.5 (CZOSNYKA *et al.*, 1988), this gives a real peak-to-peak amplitude of 12 mm Hg. The decrease in mean ICP recorded overnight was around 8 mm Hg, and the decrease resulting from Fig. 5 was around 5 mm Hg. However, in addition to the pulse waveform, the respiratory wave and slow waves were recorded (see Fig. 5*a*). Their phase relationships, and thus the real value of the peak-to-peak variations of ICP, are unfortunately unknown. Therefore the result of this anecdotal clinical measurement is generally in agreement with laboratory simulations. Not many patients could present with such an obviously negative association between pulse amplitude and mean ICP level, as the positive physiological relationship between pulse amplitude and mean ICP is potent (AVEZAAT *et al.*, 1979). These two mechanisms, the positive relationship resulting from the CSF physiology and the negative one resulting from the non-linear properties of the shunt, compete with each other.

We have already reported incidence of prolonged negative intracranial pressure after periods of accelerated activity of 'B' waves in patients with a shunt *in situ* (CZOSNYKA *et al.*, 1998). More evidence can be obtained by analysis of our own material, when we measured mean ICP and pulse amplitude in 60 previously shunted patients (CZOSNYKA *et al.*, 2002) to establish shunt patency. It is well known that, in a non-shunted patient, the baseline pulse amplitude of ICP is proportional to mean ICP, which reflects a decrease in brain compliance when ICP increases (AVEZAAT *et al.*, 1979). In patients with a functional shunt, the positive relationship

Table 1 Dependence of operating pressure on peak-to-peak pulse amplitude together with shunt's hydrodynamic resistance and types of construction

Model	Critical threshold, mm Hg	Rate of decrease in P per unit of increase in amplitude	Hydrodynamic resistance, mm Hg $(\text{ml min}^{-1})^{-1}$	Construction
Radionics low-profile	2	−0.335	2.5	membrane
Delta valve	7	−0.17	2.2	membrane
Hakim precision	1	−0.35	1.96	ball-on-spring
Holter	1	−0.726	4.7	slit
In-line	4	−0.17	7.5	mitre
Hakim NMT	10	−0.35	1.1	ball-on-spring

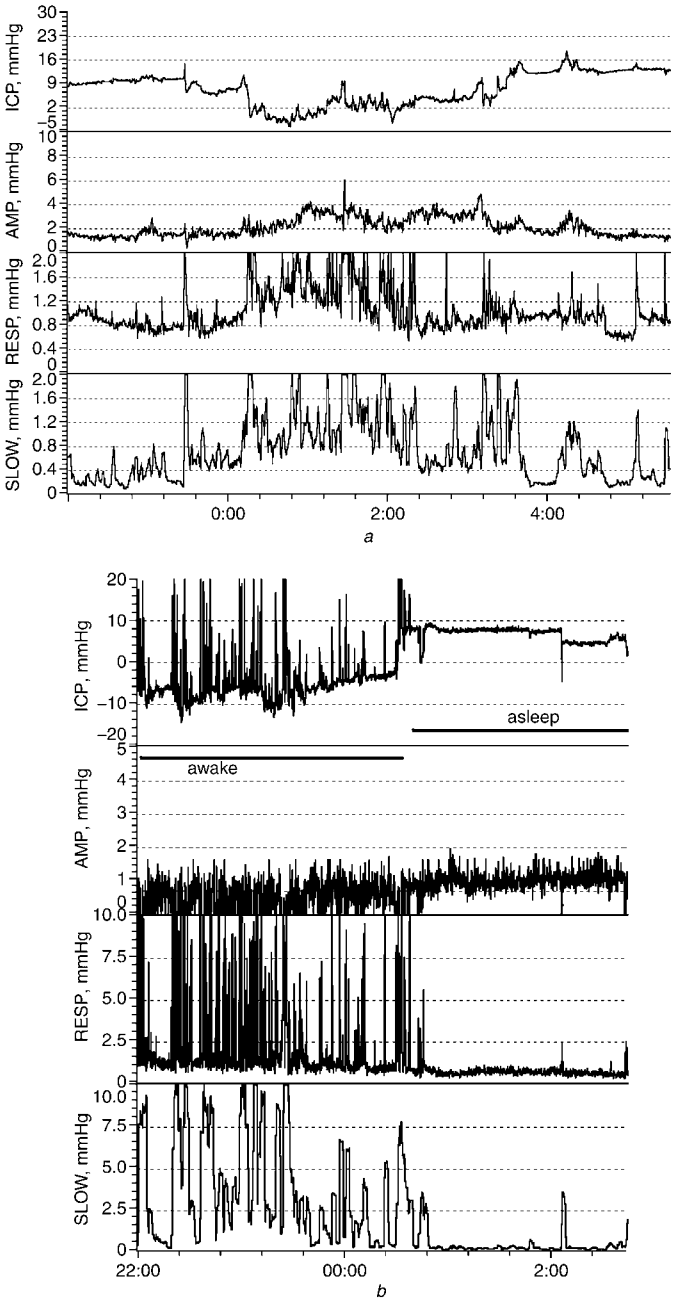

Fig. 6 *Examples of clinical recording of intracranial pressure in shunted patients (with Hakim precision valve in situ). ICP: mean ICP; AMP: magnitude of pulse waveform (first harmonic); RESP: magnitude of respiratory wave; SLOW: magnitude of slow waves. Time axis is scaled in hours and minutes*

between *AMP* and mean ICP (resulting from normal CSF physiology (AVEZAAT *et al.*, 1979)) should be disturbed, as an increase in pulse amplitude should decrease the shunt operating pressure and hence, baseline ICP (resulting from the non-linear behaviour of the shunt). Indeed, in patients with blocked shunts, the correlation between baseline ICP and amplitude was significant, whereas, in patients with a properly functioning shunt, this relationship was poor.

4.2 *Modelling interpretation*

A well-known electric model (LUNDKVIST *et al.*, 2001) of the CSF circulation can be supplemented with a simple structure representing a shunt system (see Fig. 7). Analysis of this model indicates that, when a pulse waveform of ICP is present and the shunt is open, positive and negative halves of the pulse waveform (i.e. above and below the mean ICP level, equal to or a little above the shunt opening pressure) encounter different hydrodynamic resistances. For the analysis, brain compliance has been omitted for simplicity, as it is not useful for analysis of the influence of pulse pressure on intracranial pressure. We can assume that the model represents a 'worst case' of an extremely low compliant system.

The constant current source represents production of CSF (Q_f around 0.3 ml min^{-1}), with pulsation of cerebral blood volume Q_b having zero mean value.

The R_{out} represents the resistance of CSF outflow, which in hydrocephalus is increased above the norm (from 6 to 10 mm Hg (ml min^{-1})$^{-1}$ (ALBECK *et al.*, 1997)). The voltage source p_{SS} expresses saggital sinus pressure. The last branch of the electrical model represents the technical parameters of the implanted shunt. It contains a diode (valve), with a constant voltage source connected in series, to represent opening pressure p_C, and two resistances R_v and R_D, expressing resistance to flow of a valve and distal catheter, respectively. The total resistance of a shunt is, hence, $R_S = R_V + R_D$.

The mean ICP value when pulsation is not present (i.e. when $Q_b = 0$) is

$$ICP = Q_f \frac{R_S R_{out}}{R_S + R_{out}} + \frac{p_{SS} R_S + p_C R_{out}}{R_S + R_{out}}$$

It can easily be checked that the above pressure remains within the range

$$Q_f R_S + p_C \leq ICP \leq Q_f R_{out} + p_{SS}$$

The upper ICP pressure results directly from the Davson formula (DAVSON *et al.*, 1987) and refers to the situation when the shunt is permanently closed. The lower limit concerns the case where the shunt's resistance to flow is much less than the outflow resistance, so that, practically, CSF drainage takes place only through the shunt.

The value of ICP remains a little above the shunt opening pressure p_C, i.e. very close to its lower limit. Therefore, when pulsation is added, the shunt becomes closed during the negative part of the pulse waveform.

Let us assume, for simplicity, that the waveform is described by the sinusoidal function of period T

$$Q_b(t) = Q_b \sin \frac{2\pi}{T} t$$

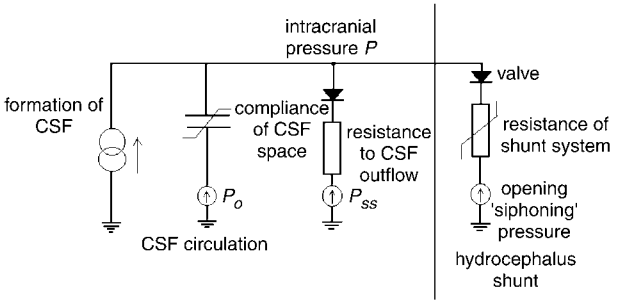

Fig. 7 *Electrical model representing CSF compensation after implantation of shunt. Diode and voltage source (opening pressure) represent mechanisms of valve. Non-linear (in most valves) resistance: hydrodynamic resistance of open valve plus resistance of long peritoneal drain*

When the shunt is open (during the positive part of the sinusoidal function), $ICP(t)$, which is now a function of time, becomes

$$ICP(t) = \left(Q_f + Q_b \sin\frac{2\pi}{T}t\right)\frac{R_S + R_{out}}{R_S + R_{out}} + \frac{p_{SS}R_S + p_C R_{out}}{R_S + R_{out}} \quad \text{for} \quad 0 < t < \frac{T}{2}$$

When the shunt is closed, the CSF flows only through the natural (partially impaired) flow path, and ICP approaches its upper limit

$$ICP(t) = \left(Q_f + Q_b \sin\frac{2\pi}{T}t\right)R_{out} + p_{SS} \quad \text{for} \quad \frac{T}{2} < t < T$$

Taking the average of $ICP(t)$ over the period T and denoting the amplitude of ICP as $AMP = R_{out}Q_b$, we can express mean ICP as the function of AMP

$$\langle ICP\rangle = Q_f\frac{R_S R_{out}}{R_S + R_{out}} + \frac{p_{SS}R_S + p_C R_{out}}{R_S + R_{out}} - AMP\frac{1}{\pi}\frac{R_{out}}{R_S + R_{out}}$$

This formula explains why, with an increasing AMP, mean ICP decreases.

In real life, this formula can probably be identified with great difficulty. It does not take into account the unknown 'vasogenic component' of ICP (MARMAROU *et al.*, 1987). Also, when taking measurements in the shunt's pre-chamber (CZOSNYKA *et al.*, 1998) or Ommaya reservoir, we usually assume that the CSF volume is ideally balanced, i.e. there is no prior over-drainage (related to patient posture: patients usually walk into the treatment room or sit before the measurement).

The formula also does not indicate any presence of the threshold amplitude above which mean ICP starts to fall with increasing pulsations. The non-linear distortion of the relationship between the mean pressure and increasing amplitude, as seen in Fig. 5, is probably caused by limited sensitivity of the measurement system and the influence of compartmental compliances not taken into account in the modelling structure.

However, in our opinion, the practical role of this study is important. It explains why, no matter how sophisticated the shunt system, static conditions for CSF drainage are always influenced by dynamic or transitional phenomena that render any idea of accurate control of mean ICP wishful thinking.

We can presume that almost all shunt types exhibit similar phenomena. At the time of performing the study, we did not have three samples of strata and Codman valves. The mechanism of the Codman Hakim programmable valve is the same as the Codman Hakim precision (non-programmable) valve. Both shunts represent ball-on-spring mechanisms, tested in the study and confirmed as being sensitive to pressure pulsations. The strata valve was tested recently, and the results confirmed this inference.

In conclusion, from the point of view of cerebral hydrodynamics, a hydrocephalus shunt represents a strong, non-linear element. The influence of its non-linearity may have an impact, not only on the constant drainage of CSF, but also on cerebrospinal physiology, disturbing or reversing the proportional relationship between pulsations of ICP and mean ICP level.

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