Effect of cerebrospinal fluid shunts on intracranial pressure and on cerebrospinal fluid dynamics

2. A new technique of pressure measurements: results and concepts¹

3. A concept of hydrocephalus

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SUMMARY Part 2 describes measurements of intracranial cerebrospinal fluid (CSF) pressure in 18 adult patients with CSF shunts, all pressure measurements being referred to a horizontal plane close to the foramina of Monro. All 18 patients had normal CSF pressure by lumbar puncture; however, in one patient an intracranial pressure of +280 mm was subsequently measured after pneumoencephalography. Twelve patients had pre-shunt CSF pressures measured intracranially: 11 ranged from +20 to +180 mm H₂O and one was +280 mm H₂O in the supine position. In the upright posture nine patients had values of -10 to -140 mm H₂O, while three others were +60, +70, and +280 mm H₂O. After CSF shunting in these 18 patients the pressures were -30 to +30 mm H₂O in the supine position and -210 to -370 mm in the upright position. The effect of posture on the siphoning action of these longer shunts in the erect, adult patient is a major uncontrollable variable in maintenance of intracranial pressure after shunting. Other significant variables are reviewed. In Part 3 a concept of the hydrocephalus phenomenon is described. Emphasis is placed on the pressure differential (P_a) and force differential (F_a) causing pre-shunt ventricular enlargement and post-shunt ventricular size reduction. The site of P_a , which must be very small and not to be confused with measured ventricular pressure, P , must be at the ventricular wall.

Since the advent of the concept of normal pressure hydrocephalus (NPH) (Adams, Fisher, Hakim, Ojemann, and Sweet, 1965; Hakim and Adams, 1965) cerebrospinal fluid (CSF) shunting has become commonplace in adults as well as in children (Appenzeller and Salmon, 1967; Ojemann, Fisher, Adams, Sweet, and New, 1969). Almost simultaneously there developed an increased interest in the use of lumbo-peritoneal (L-P) and ventriculo-peritoneal (V-P) shunts (Ames, 1967; Murtagh and Lehman, 1967; Dakers, Yashon, Croft, and White, 1968; Weiss and Rashkind, 1969) as alternatives to the then more common ventriculo-atrial (V-A) shunts. In the horizontal patient a longer distance between the ventricle and the shunt ter-

mination has no remarkable effect on intracranial pressure, but we felt that in the upright adult these increased distances (whether of the intraspinal CSF column in L-P shunts or of the intra-shunt CSF column in V-P and V-A shunts) might have a significant effect on intracranial CSF pressure. Forrest (1962) and de Lange, van der Gon, and de Vlieger (1968) measured pressures in shunted patients; when they elevated the head to about 30° they found some drop in ventricular pressure, but the fall was not dramatic. Rayport and Reiss (1969) considered the ventricular pressure to be sufficiently negative in the normal person to obviate any significant further reduction of CSF pressure after V-A shunting. We believe that posture may be one of the most significant variables affecting maintenance of CSF pressure in the brain of a shunted patient.

¹ This study was supported by a grant to Neurosurgical Research Unit 102.80 of the U.S. Veterans Administration.

Although excessively low intracranial pressures may lead to annoying headaches (Jackson and Snodgrass, 1955), of greater concern is the increased risk of subdural haematoma or hygroma. Although not frequently documented in the literature, this post-shunt complication is well known (Illingworth, 1970). Waga, Hashi, Ohtsubo, and Handa (1970) describe what may be the first reported chronic subdural haematoma occurring after CSF shunting for 'normal pressure hydrocephalus' (Alzheimer's disease on biopsy). Greitz, Grepe, Kalmér, and Lopez (1969) on angiograms noted three cases of subdural collections after V-A shunting for NPH. These were not removed but the authors postulated that the collections were subdural CSF. We have seen seven cases of subdural haematoma or effusion after CSF shunting for NPH. Thus, we were encouraged to investigate the CSF pressure and hydrodynamics in patients with a CSF

shunt (McCullough, Fox, Curl, and Green, 1972).

Since our criteria for shunting in these patients no longer includes the prerequisite of elevated CSF pressure, other criteria must be used. The typical patient has progressive mental deterioration and/or gait and motor disturbance after cerebral trauma, haemorrhage, meningitis or an unrecognized intracranial illness. Pneumoencephalography (PEG) reveals dilated ventricles consistent with communicating hydrocephalus—although non-communicating hydrocephalus instead may occasionally be present. CSF pressure measured by lumbar puncture is normal. Another criterion may be persistent (48 hours or more) ventricular radioactivity during isotope cisternography (Di Chiro, 1966; Bannister, Gilford, and Kocen, 1967; LeMay and New, 1970; McCullough, Harbert, Di Chiro, and Ommaya, 1970). Other authors (Appenzeller and

FIG. 1 Diagram of arrangement for measuring CSF pressure. 1: scalp, 2: skull, 3: Ommaya or Mishler reservoir, 4: 22-gauge needle, 5: air-filled tubing, 6: 0.5 cm long water column marked on tube, 7: LVDT pressure transducer, 8: air-filled tubing, 9: stopcock, 10: plastic Monoject 12 ml. syringe, 11: connection between transducer and amplifier, 12: amplifier with attached visual direct read-out pressure scale, 13: water column at pressure of 0 cm water (atmospheric) in U-tube, 14: U-tube open to the atmosphere.

Salmon, 1967) indicate that some forms of parenchymatous degeneration of the brain without obstruction of CSF pathways may respond to CSF shunting. At present the final criterion for the diagnosis of NPH remains the clinical response to CSF shunting.

METHODS

Eighteen patients were studied as described below. Nine had ventriculo-peritoneal (V-P) shunts and nine had ventriculo-atrial (V-A) shunts. All patients had routine lumbar spinal fluid tests (none had CSF pressures over 180 mm of CSF) and PEG. Cisternography was carried out by lumbar or cisternal injection of 100 μ C of radioactive iodinated human serum albumin (131 IHSA).

Measurements of CSF pressure were performed by a new technique devised to utilize an easily calibrated external pressure transducer and a convenient amplifier with instantaneous read-out (Fig. 1). An air communication system was used in the tube to avoid the requirement of correcting for changes in vertical distance between the reference point in the head and the transducer when liquid

communication is used. The method required the use of counter-pressure with air injection into, or air withdrawal from, the transducer to prevent movement of an 0.5 cm column of water in the tube connecting the patient with the transducer. Using a 12 ml. syringe we manually injected air into the transducer if greater pressure was required to balance the increased intracranial pressure. Air was withdrawn if reduced or negative pressure was needed to balance the decreased or negative intracranial pressure. A Gulton linear variable differential transformer (LVDT) type of transducer was used. The pressure was displayed instantaneously in centimetres of water (+ or - with respect to baseline atmospheric pressure) and on a read-out attached to the amplifier.

The system is calibrated easily and accurately with a U-tube filled with water. Conversely, the standard 90 cm U-tube can be used as a model of positive or negative intracranial pressure by pre-setting the water level in the U-tube and then checking the accuracy of this pressure measuring system. This was done before and after each patient was tested; there was never an error exceeding 10%.

Before shunting, an Ommaya reservoir or a Mishler reservoir (with its cardiac end folded and ligated

FIG. 2 *Pneumoencephalogram showing relationship of bregma-interaural point with foramina of Monro, the former lying just above and behind the foramina of Monro.*

and its ventricular end attached to a Portnoy or similar ventricular catheter) was implanted under local anaesthesia. Usually the frontal horn of the right lateral ventricle was used. A few days later pressure measurements were made by sterile percutaneous puncture of the reservoir. Measurements were taken in the supine, 45° head up, sitting, and standing positions if possible. At least 10 minutes of recording time was used for each position. All pressures were corrected to a horizontal reference plane located midway between the bregma (coronal-sagittal junction) and the interaural point (Fig. 2). The bregma-interaural distances are about 12 to 14 cm in adults. This reference point was selected because of its ease of identification, its possible use as a universal refer-

ence point, and its location close to (just above and behind) the foramina of Monro. Thus, hydrostatic pressures measured above this level can be corrected by adding the *vertical* distance above the reference point, and pressures measured below this point can be corrected by subtracting the *vertical* distance below the reference point. The value of such a reference point close to the foramina of Monro was shown clearly by Bradley (1970).

A few days after standard CSF shunting (Raimondi V-P or Pudenz V-A) the identical pressure measurements were repeated. The closing pressures of these shunts at one minute after descent of the column of saline from 550 mm in the spinal manometer were all less than 25 mm water.

TABLE
CLINICAL DETAILS OF 18 PATIENTS

Description of cases	Shunt distance (mm)		Intracranial pressure (mm water) at foramina of Monro			
			Supine	45°	Sitting	Standing
Case 1. M. 52 yr. Disorientation and incontinence 2 mo. post head injury	V-P 600	pre-op.	—	—	—	—
		post-op.	+ 20	- 280	- 340	—
		post-op.	0	- 270	—	—
Case 2. M. 55 yr. Apraxic-ataxic gait for 5 mo.	V-P 600	pre-op.	—	—	—	—
		post-op.	- 50	- 390	—	—
		post-op.	- 40	- 220	- 260	—
Case 3. M. 23 yr. Aphasic, hemiplegic 6 mo. post compound head injury. Lumbar CSF pressure normal before PEG	V-P 600	post-op.	- 20	- 230	- 270	- 370
		pre-op.	+ 280	+ 400	+ 270	—
		post-op.	- 70	- 130	- 140	—
Case 4. M. 45 yr. Long history of dementia, incontinence, spastic paraparesis	V-A 480	post-op.	- 30	- 170	- 150	- 290
		pre-op.	—	—	—	—
		post-op.	0	- 160	- 260	- 260
Case 5. M. 71 yr. History of several 'strokes', confusion, hemiparesis	V-P 600	pre-op.	+ 90	- 70	- 90	—
		post-op.	—	—	- 280	—
		pre-op.	+ 180	- 50	- 30	—
Case 6. M. 72 yr. Long history of dysphasia; 2 episodes of headache, lethargy, confusion	V-A 410	post-op.	—	—	- 280	—
		pre-op.	+ 100	- 50	- 40	—
		post-op.	+ 20	- 150	- 220	—
Case 7. F. 61 yr. Previous craniotomy for aneurysm. Persisting confusion, ataxic-apraxic gait	V-P 550	post-op.	—	—	- 280	—
		pre-op.	+ 150	- 40	- 120	- 20
		post-op.	0	- 200	- 235	- 235
Case 8. M. 63 yr. 5 yr ataxic-apraxic gait	V-A 400	pre-op.	+ 100	- 50	+ 70	+ 70
		post-op.	0	- 135	- 210	- 190
		pre-op.	+ 60	+ 10	- 140	—
Case 9. M. 48 yr. 2 yr of ataxic-apraxic gait	V-A 350	post-op.	0	- 260	- 250	—
		pre-op.	+ 60	- 10	+ 60	+ 50
		post-op.	0	- 260	- 250	—
Case 10. M. 58 yr. Subdural haematoma 10 yr ago; progressive dementia, seizures, ataxic-apraxic gait	V-A 400	pre-op.	+ 20	—	—	—
		post-op.	0	- 210	- 260	- 260
		pre-op.	+ 70	- 70	- 70	- 70
Case 11. M. 65 yr. Subdural haematoma 10 yr ago; progressive dementia, ataxic-apraxic gait	V-A 400	post-op.	+ 20	- 140	- 240	- 210
		pre-op.	—	—	—	—
		post-op.	+ 10	- 190	- 230	- 280
Case 12. M. 55 yr. One yr of confusion, ataxic-apraxic gait	V-P 600	post-op.	- 20	- 300	- 300	—
		pre-op.	+ 120	+ 20	- 80	—
		post-op.	0	- 250	- 310	—
Case 13. F. 25 yr. Infantile hemiplegia; 2 yr of mental deterioration and gait disturbance	V-A 400	pre-op.	+ 30	0	- 10	—
		post-op.	0	- 170	- 330	—
		post-op.	0	- 170	- 200	- 200
Case 14. M. 24 yr. Ependymoma of cerebral hemisphere, communicating hydrocephalus after surgery	V-A 450	post-op.	—	—	- 260	—
		pre-op.	—	—	—	—
		post-op.	0	- 200	- 240	—
Case 15. M. 83 yr. Subarachnoid bleed 6 weeks ago. Progressive dementia, apraxic-ataxic gait, incontinence	V-P 550	pre-op.	+ 20	- 70	- 70	—
		post-op.	0	- 200	- 340	—
		pre-op.	+ 30	0	- 10	—
Case 16. M. 52 yr. 15 yr of seizures, dementia, ataxic-apraxic gait	V-A 400	post-op.	0	- 170	- 330	—
		post-op.	0	- 170	- 200	- 200
		pre-op.	—	—	- 260	—
Case 17. F. 63 yr. Recent confusion, apraxic-ataxic gait	V-P 550	pre-op.	—	—	—	—
		post-op.	0	- 200	- 240	—
		pre-op.	+ 20	- 70	- 70	—
Case 18. M. 45 yr. Ruptured aneurysm followed by some persistent mental retardation and headaches	V-P 600	post-op.	0	- 200	- 340	—

RESULTS

The Table briefly summarizes our 18 cases, including shunt distances and recorded intracranial pressures. All patients had evidence of ventricular dilatation on PEG as well as ventricular retention of the isotope during cisternography (Table).

DISCUSSION

With the patient in a lateral decubitus, the normal CSF pressure (ventricular, cisternal, or lumbar) referred to the mid-sagittal horizontal plane ranges from 70 to 180 mm CSF (Ayer, 1920; Loman, 1934; Masserman, 1934; Masserman, 1935; Merritt, 1937; Smyth, 1938; Davson, 1967). The pre-shunt spinal tap pressures in our patients fell within that range. Pressures near the foramina of Monro, measured in 11 patients in the supine position before shunting, ranged from 20 to 180 mm water. Since the vertical hydrostatic pressure at the foramina of Monro in the supine patient is about the same as that at a lumbar puncture needle in a lateral decubitus patient, the normal CSF pressures should be nearly equivalent at these two sites of reference.

On the other hand, CSF pressures with the patient sitting are close to atmospheric at the cisterna magna (Loman, 1934; Masserman, 1935; Davson, 1967) with variations from +40 to -85 mm water (Loman, 1934). When pressure measurements are referred to the foramina of Monro or the bregma-interaural horizontal plane, about 70 mm must be subtracted, which gives normal values close to -30 to -155 mm water. In those 12 patients in whom we were able to get pre-shunt CSF pressures in the upright position, we obtained values of -10 to -140 mm water in nine patients and +60, +70, and +280 mm water in three other patients. The negative values in these nine patients appear to be within normal limits. Bradley (1970) found a CSF pressure of -165 mm water at the foramina of Monro in one sitting patient with senile dementia. This value may be at the limit of normal. Except for the patient with high pressure hydrocephalus (+280 mm water), those patients with a positive pressure while upright may have had elevated pressures due to some muscular tension causing increased venous pressure while trying to maintain an erect posture.

Particularly interesting are the CSF pressures measured in patients who have V-P or V-A shunts. Although we have no V-L shunt patients, the results should be similar, since the hydrostatic pressure in the upright position is the same whether it is measured via the spinal canal or via the V-P shunt. In the upright position the pressures ranged from -210 to -370 mm water at the bregma-interaural horizontal reference plane. The nine patients with V-P shunts exhibited upright pressures of -240 to -370 mm water (averaging about -290 mm). The nine patients with V-A shunts had upright pressure of -210 to -330 mm water (averaging about -260 mm). If we assume the normal CSF pressure averages about -70 mm water near the foramina of Monro, then the average CSF pressure drop *below normal* due to shunting in these patients was about -220 mm water for V-P shunts and about -190 mm water for V-A shunts in the upright patient. Such negative pressure recordings certainly indicate that the shunts are functioning.

One factor which we have not controlled is the time after getting up in the morning during which the patient has been sitting or walking before the pressure tests. Most of the patients had been upright, which may explain their low CSF pressures in the initial supine position (ranging from -30 to +30 mm water).

The findings of such low CSF pressures in ambulatory patients is explained by the siphoning action of the shunts. We put the ventricular end of various types of CSF shunts under water in a closed container filled with water (and some air to approximate the normal resiliency of the CSF membranes and the vascular space). The water continued to run out of the externalized lower distal end of the shunt until the measured air pressure inside the container achieved a negativity in mm of water approaching the vertical length of the tube (minus the closing pressure of the shunt). This took several minutes while fluid slowly dripped out of the distal end of the shunt.

de Lange (1966) once stated,

'... in our opinion the occurrence of a so-called negative pressure syndrome is based on a technical error: too frequent pumping or implantation of a pump with too low passage pressure.'

In the upright adult patient we believe a more

common cause may be related to the physics of CSF flow in a siphoning system. Our estimated average values for pressure drops of 220 mm water in upright patients with V-P shunts and 190 mm water in those with V-A shunts refer to values *below* the normal -70 mm water pressure at the foramina of Monroe in the upright non-shunted patient. We wonder whether a valve selection of 50 mm pressure as opposed to one of 10 mm pressure will influence significantly clinical results in the face of siphons (shunts) of 400 to 600 mm length causing intracranial pressure reductions of 200 mm water below normal in the upright adult patient. Indeed, the intracranial pressure variability and dependence on position, a rather uncontrollable factor in most shunted patients, lead us to inquire what scientific criterion can be used to select the correct valve.

Another variable factor is the resistance (back pressure) to flow of CSF at the distal end of the shunt. We would modify the statement by Rayport and Reiss (1969) who indicate that perfusion pressure through a V-A shunt is equal to ventricular pressure less *either* the cardiac intra-atrial systolic pressure *or* the hydrostatic opening pressure of the shunt system, whichever is the greater. Net perfusion pressure is actually equal to ventricular pressure (plus shunt hydrostatic opening pressure) less the *sum* of the intra-atrial pressure and hydrostatic opening pressure of the shunt system. Contrary to their comment, the low or negative intra-atrial pressures of the diastolic phase of the heart cycle *are* available for augmentation of the pressure gradient within the shunt assembly when they fall below the hydrostatic opening pressure. Thus, if a valve opens at 50 mm water, CSF will flow regardless if one applies positive ($> +50$ mm water at the ventricular end or negative pressure (< -50 mm water) at the cardiac or peritoneal end. The average intra-atrial pressure tends to be about 60 mm water in the supine patient but nearly atmospheric in the upright patient who is not straining (Rushmer, 1955; Best and Taylor, 1966). Thus, when the non-shunted patient stands, his CSF pressure at the foramina of Monroe may drop to -70 mm water and his average intra-atrial pressure to 0 mm water giving a net pressure difference of -70 mm water. If the implanted CSF shunt opens at 50 mm

water pressure and the hydrostatic column in the shunt of the upright patient is 400 mm from the foramina of Monroe, the net counter pressure difference is about $+350$ mm water. The result is 350 mm less 70 mm, or 280 mm water. Theoretically, in this example the intracranial pressure could drop to -280 mm water in the erect patient after V-A shunting.

A similar analysis can be carried out for V-P shunts. The average intra-abdominal pressures are extremely variable (Lam, 1939; Rushmer, 1946; Campbell and Green, 1953; Rushmer, 1955; Agostoni and Rahn, 1960; Konno and Mead, 1968). For example, intragastric pressures range from 0 mm to 150 mm water in the supine patient. Upon standing, subdiaphragmatic pressures range from -170 mm to $+120$ mm water, depending on the state of respiration. The average subdiaphragmatic pressure in the standing patient is 0 mm compared with $+400$ mm water in the pelvis. Hence, the increased resistance at the shunt termination varies with posture, site of shunt termination, obesity, and adherence of adjacent tissues. When the non-shunted patient stands and the CSF pressure at the foramina of Monroe drops to -70 mm water, his average intra-abdominal pressure at the proposed shunt site may drop to $+70$ mm water (for example) resulting again in a net pressure difference of -140 mm water. If the implanted CSF shunt opens at 20 mm water pressure and the hydrostatic column in the shunt of the upright patient is 500 mm from the foramina of Monroe, the net counter pressure is 480 mm less 140 mm, or 340 mm water. Theoretically, in this example the intracranial pressure could drop to -340 mm water in the erect patient after V-P shunting.

It may be postulated that the ventriculo-sagittal sinus shunt would eliminate this siphoning problem. However, there would still exist a siphon action via the venous pathway to the heart. Indeed, neurosurgeons are very much aware of the dangers of air being sucked into the heart during surgery on the patient in a sitting position. The answer to this problem, if such negative pressures are detrimental, may be the recently developed antisiphon valve which closes when pressures less than atmospheric pressure occur inside the valve (Portnoy, Schulte, and Fox, in preparation).

With standard shunt systems currently avail-

able, if the patient is expected to be upright for much of his waking hours and his ventricles are not exceedingly large, perhaps a higher pressure valve is required and/or possibly a V-A shunt where the shunt length is less compared with a V-P shunt. If the patient is expected to be bedridden for a long time or if he has massive ventricular dilatation with lower pre-shunt CSF

pressures, then a lower pressure valve may be required. Certainly, as suggested by de Lange *et al.* (1968), flow of CSF through a shunt can be improved by elevation of the head. Furthermore, it is likely that, if a shunted adult patient is up and about and does not have CSF pressures lower than -200 mm water at the foramina of Monro when erect, the shunt is not functioning.

3. A concept of hydrocephalus

Selected patients with normal pressure hydrocephalus improve with shunting of the cerebrospinal fluid (CSF), this occurring in many instances with extremely low post-shunt intracranial pressure (McCullough, Fox, Curl, and Green, 1972). If the Force equals Pressure $\times$ Area ($F=PA$) concept as applied by Hakim and Adams (Adams, Fisher, Hakim, Ojemann, and Sweet, 1965; Hakim and Adams, 1965; Adams, 1966) is accepted (Fig. 3), then a reversal of direction of the force must occur. A modification of the concepts postulated by these pioneers in the study of normal pressure hydrocephalus is suggested.

As hydrocephalus develops, a net positive force exists in the ventricular system when CSF production in the ventricles exceeds CSF absorption from the ventricles. If CSF absorption is reduced, the positive force is increased until some mechanism of improved CSF absorption occurs as the ventricles dilate. If such compensation fails to occur, the ventricles will continue to dilate even in the presence of normal or low CSF pressure—but only if the pressure (or force per unit area) on the ventricular side is greater than the pressure on the brain parenchymal (cerebral) side as shown in Fig. 4. Fishman (1966) felt that 'the critical pressure for ventricular dilatation is the pressure gradient from ventricle to subarachnoid space—the transmural pressure gradient across cerebral mantle'. Hakim, Adams, and Fisher (1966) noted that no such pressure measurements have been made and indicated that the subarachnoid space was too thin to allow any pressure differential significant enough to effect ventricular dilatation. More recently, Hakim

(1971) discussed a concept of opposing forces resulting from the interaction of intracranial venous and CSF pressures. He theorized that a gradient in one direction may tend to cause hydrocephalus and in the other direction, pseudotumor cerebri.

Since brain parenchyma and CSF have nearly the same specific gravity, we cannot postulate differential movement during acceleration or deceleration with head movement as a cause of such a pressure differential. But CSF is incompressible, while brain parenchyma is compressible by virtue of its resiliency due to vascular perfusion, its dural envelope, and its displaceable (into systemic circulation) water content (Weed, Flexner, and Clark, 1932; Weed and Flexner, 1932; Pollock and Boshes, 1936; Fox, 1964; Davson, 1967). Therefore, the CSF space can expand at the expense of brain parenchyma when the elasticity coefficient of the ventricular wall is exceeded, as CSF is produced without adequate absorption. This differential pressure may or may not be augmented by the CSF pulse pressure as blood perfuses both the choroid plexus and the brain parenchyma, depending on whether one accepts Bering's concept (Bering, 1955; Bering, 1962; Wilson and Bertan, 1967) or disagrees with it (Dunbar, Guthrie and Karpell, 1966; Sibayan, Begeman, King, Gurdjian, and Thomas, 1970). Hakim *et al.* (1966) felt, '... it probably makes little difference in the net effect on periventricular tissues whether the force is fluctuating or steady'.

As long as the ventricular area enlarges in the presence of a persistent force differential, one can expect a proportionately lower pressure

FIG. 3 Above is diagram of inter-connected large (left) and small (right) containers of water to illustrate Pascal's Law as referred to by Hakim and Adams. See text for details. At bottom of diagram note how force generated (in centre of circles) by effect of CSF production exceeding CSF absorption is distributed over larger area in A_2 than in A_1 resulting in lower net pressure on the ventricular wall, assuming the force remains constant.

FIG. 4 Outline of diagrammatic ventricle (normal on left, hydrocephalic on right) inside brain parenchyma (see text). SSS: superior sagittal sinus; D & A: dura and arachnoid; SAS: subarachnoid space; Arrows in SAS on left: normal CSF flow pattern; 1 and 2: balanced pressures on either side of ependyma; 3 and 4: pressures in brain parenchyma < pressures in ventricular CSF; 5: direction of ventricular wall tension, T.

differential. Notice that we are not discussing a measured force (F) or pressure (P) *per se*, but only force differential (F_d) or pressure differential (P_d) existing on either side of the ventricular wall: in other words $F_d = P_d A$. The measured ventricular pressure, P , whether 500 mm, 70 mm, or -200 mm water, is irrelevant in terms of ventricular expansion or contraction if the same pressures exist on the brain side of the ventricular wall. The P_d need not be large, but if the concept is valid, then ventricles may enlarge with either a relative increase in ventricular P or a relative drop in brain parenchymal P (due to inadequate brain vascular perfusion?). Hence, $F = PA$ as implied here, and in the Hakim-Adams theory, must be modified. The F_d formula can explain how P_d can drop as A increases, while F_d remains fairly constant or even in-

creases; but we are unaware of any measurements of P_d across the ventricular wall in hydrocephalic animals or patients. The air pressure in the expanding balloon concept alluded to by Hakim and Adams (1965) is not contested, but the initial pressures before blowing up the balloon were atmospheric on both sides of the balloon wall. Any pressures recorded inside the expanded balloon are really P_d : balloon P plus atmospheric P minus outside atmospheric pressure. While this may be intuitively obvious, it clarifies why P cannot be the measured CSF pressure value of 180 mm water, for example, as implied by Hakim and Adams; but P , or rather P_d , must be some very small value equal to ventricular P minus brain parenchymal P .

In his discussion of ventricular expansion in these hydrocephalic patients Geschwind (1968)

stressed the structural properties of the ventricular walls and considered the Hakim-Adams theory an insufficient explanation. He postulated that the CSF pulsations (Bering, 1955; Bering, 1962) resulted in periventricular destruction of protein and lipid (Fishman and Greer, 1963). The normal CSF pressures (P) in these hydrocephalic cases were explained by the formula, $P = 2 T/R$, where T = linear surface tension in g-wt/cm and R = radius of 'spherical' container. Geschwind (1968) concluded that as the ventricles enlarge (R) and the tensile properties of their walls decrease through structural change, P necessarily must drop.

Again Geschwind, like Hakim and Adams, implies that P in their formula is the measured CSF pressure. But, in our opinion, the formula should be written $P_a = 2 T/R$, for the formula relates to the relationship between T, R, and the difference between external (brain parenchyma) and internal (ventricular CSF) pressures (P_a). We do not consider the entire cerebrum to be the 'membrane' of the container; the membrane is the ependyma and neighbouring brain substance.

If we rewrite the formula: $T = P_a R/2$, then we can see that, as the ventricular size (R) increases, T will increase until it exceeds the tensile coefficient of the juxta-ventricular brain parenchyma. The latter fibres tear, bonds break, and R increases further. Now even less P_a is required to continue this vicious cycle. Whether P increases or not depends simply on the amount and rapidity of definitive CSF absorption. But we should not make the error of equating P with P_a . Massive elevations in P will not necessarily cause signs of hydrocephalus if P is the same on both sides of the ventricular wall (Evans, Espey, Kristoff, Kimbell, and Ryder, 1951).

Insertion of a CSF shunt, then, must allow cerebral re-expansion by reversing the F_a and hence P_a gradients so that F_a and P_a are relatively negative in the ventricle. Such increased removal of CSF from the brain would improve P_a by lowering the ratio of ventricular P/brain parenchymal P. This does not require necessarily that ventricular pressure be negative to atmosphere.

This begs the question: does P_a become positive in the ventricular system of a normal pressure hydrocephalic because of a relative increase in ventricular P, or does reduced cerebral blood

flow effect a lowering of intracranial vascular pressure and consequently of brain parenchymal pressure? Salmon and Timperman (1971) noted improved cerebral blood flow with reduced intracranial pressure. Greitz (Greitz, Grepe, Kalmer, and Lopez, 1969; Greitz, 1969a; Greitz, 1969a, b) postulated that the ventricular dilatation was primary and the reduced cerebral blood flow was secondary to brain compression by the enlarging ventricles. One also can theorize that the reduced cerebral blood flow is primary (for example, atherosclerosis) and causes a relative reduction in brain parenchyma P. CSF shunting then may reverse the gradient, effecting an improved P_a . We suspect that in these situations any favourable response to shunting may often be short-lived as the atherosclerosis or primary atrophy progresses.

REFERENCES

- Adams, R. D. (1966). Further observations on normal pressure hydrocephalus. *Proceedings of the Royal Society of Medicine*, **59**, 1135-1140.
- Adams, R. D., Fisher, C. M., Hakim, S., Ojemann, R. G. and Sweet, W. H. (1965). Symptomatic occult hydrocephalus with 'normal' cerebrospinal-fluid pressure. treatable syndrome. *New England Journal of Medicine*, **273**, 117-126.
- Agostoni, E., and Rahn, H. (1960). Abdominal and thoracic pressures at different lung volumes. *Journal of Applied Physiology*, **15**, 1087-1092.
- Ames, R. H. (1967). Ventriculo-peritoneal shunts in the management of hydrocephalus. *Journal of Neurosurgery*, **27**, 525-529.
- Appenzeller, O., and Salmon, J. H. (1967). Treatment of parenchymatous degeneration of the brain by ventriculo-atrial shunting of the cerebrospinal fluid. *Journal of Neurosurgery*, **26**, 478-482.
- Ayer, J. B. (1920). Puncture of the cisterna magna. *Archives of Neurology and Psychiatry*, **4**, 529-541.
- Bannister, R., Gilford, E., and Kocen, R. (1967). Isotope encephalography in the diagnosis of dementia due to communicating hydrocephalus. *Lancet*, **2**, 1014-1017.
- Bering, E. A., Jr. (1955). Choroid plexus and arterial pulsation of cerebrospinal fluid. Demonstration of the choroid plexuses as a cerebrospinal fluid pump. *Archives of Neurology and Psychiatry*, **73**, 165-172.
- Bering, E. A., Jr. (1962). Circulation of the cerebrospinal fluid. Demonstration of the choroid plexuses as the generator of the force for flow of fluid and ventricular enlargement. *Journal of Neurosurgery*, **19**, 405-413.
- Best, C. H., and Taylor, N. B. (1966). *The Physiological Basis of Medical Practice*, 8th edn., pp. 771-794. Williams and Wilkins: Baltimore.
- Bradley, K. C. (1970). Cerebrospinal fluid pressure. *Journal of Neurology, Neurosurgery, and Psychiatry*, **33**, 387-397.
- Campbell, E. J. M., and Green, J. H. (1953). The variations in intra-abdominal pressure and the activity of the abdominal muscles during breathing; a study in man. *Journal of Physiology*, **122**, 282-290.

- Dakters, J. G., Yashon, D., Croft, T. J., and White, R. J. (1968). Cerebrospinal fluid diversion. Lumbar subarachnoid peritoneal placement with valve regulation. *Archives of Surgery*, **96**, 56–57.
- Davson, H. (1967). The cerebrospinal fluid-pressure. In *Physiology of the Cerebrospinal Fluid*, pp. 337–382. Churchill: London.
- Di Chiro, G. (1966). Observations on the circulation of the cerebrospinal fluid. *Acta Radiologica, Diagnosis*, **5**, 988–1002.
- Dunbar, H. S., Guthrie, T. C., and Karpell, B. (1966). A study of the cerebrospinal fluid pulse wave. *Archives of Neurology*, **14**, 624–630.
- Evans, J. P., Espey, F. F., Kristoff, F. V., Kimbell, F. D., and Ryder, H. W. (1951). Experimental and clinical observations on rising intracranial pressure. *Archives of Surgery*, **63**, 107–114.
- Fishman, R. A. (1966). Occult hydrocephalus. *New England Journal of Medicine*, **274**, 466–467.
- Fishman, R. A., and Greer, M. (1963). Experimental obstructive hydrocephalus. *Archives of Neurology*, **8**, 156–161.
- Forrest, D. M. (1962). Flow characteristics of the Spitz-Holter valve. *Developmental Medicine and Child Neurology*, **4**, 295–297.
- Fox, J. L. (1964). Development of recent thoughts on intracranial pressure and the blood-brain barrier. *Journal of Neurosurgery*, **21**, 909–967.
- Geschwind, N. (1968). The mechanism of normal pressure hydrocephalus. *Journal of Neurological Science*, **7**, 481–493.
- Greitz, T. (1969a). Cerebral blood flow in occult hydrocephalus studied with angiography and the xenon 133 clearance method. *Acta Radiologica, Diagnosis*, **8**, 376–384.
- Greitz, T. (1969b). Effect of brain distension on cerebral circulation. *Lancet*, **1**, 863–865.
- Greitz, T. V. B., Grepe, A. O. L., Kalmér, M. S. F., and Lopez, J. (1969). Pre- and postoperative evaluation of cerebral blood flow in low-pressure hydrocephalus. *Journal of Neurosurgery*, **31**, 644–651.
- Hakim, S. (1972). Observations on the biomechanics of hydrocephalus. In *Cisternography and Hydrocephalus*, pp. 25–56. Edited by J. C. Harbert. Thomas: Springfield, Ill.
- Hakim, S., and Adams, R. D. (1965). The special clinical problem of symptomatic hydrocephalus with normal cerebrospinal fluid pressure. Observations on cerebrospinal fluid hydrodynamics. *Journal of Neurological Sciences*, **2**, 307–327.
- Hakim, S., Adams, R. D., and Fisher, C. M. (1966). Occult hydrocephalus. (Letter to the editor.) *New England Journal of Medicine*, **274**, 467.
- Illingworth, R. D. (1970). Subdural haematoma after the treatment of chronic hydrocephalus by ventriculocaval shunts. *Journal of Neurology, Neurosurgery and Psychiatry*, **33**, 95–99.
- Jackson, I. J., and Snodgrass, S. R. (1955). Peritoneal shunts in the treatment of hydrocephalus and increased intracranial pressure. A 4-year survey of 62 patients. *Journal of Neurosurgery*, **12**, 216–222.
- Konno, K., and Mead, J. (1968). Static volume-pressure characteristics of the rib cage and abdomen. *Journal of Applied Physiology*, **24**, 544–548.
- Lam, C. R. (1939). Intra-abdominal pressure. A critical review and an experimental study. *Archives of Surgery*, **39**, 1006–1015.
- Lange, S. A. de (1966). *Surgical Treatment of Progressive Hydrocephalus*. North-Holland: Amsterdam.
- Lange, S. A. de, Denier van der Gon, J. J., and Vlieger, M. de (1968). Preliminary study on the ventricular pressure in hydrocephalic patients. *Psychiatria, Neurologia, Neurochirurgia*, **71**, 267–273.
- LeMay, M., and New, P. F. J. (1970). Radiological diagnosis of occult normal-pressure hydrocephalus. *Radiology*, **96**, 347–358.
- Loman, J. (1934). Components of cerebrospinal fluid pressure as affected by changes in posture. *Archives of Neurology and Psychiatry*, **31**, 679–681.
- McCullough, D. C., Fox, J. L., Curl, F., and Green, R. C. (1972). Effect of CSF shunts on intracranial pressure and CSF dynamics. In *Cisternography and Hydrocephalus*, pp. 335–342. Edited by J. C. Harbert Thomas: Springfield, Ill.
- McCullough, D. C., Harbert, J. C., Di Chiro, G., and Omaya, A. K. (1970). Prognostic criteria for cerebrospinal fluid shunting from isotope cisternography in communicating hydrocephalus. *Neurology (Minneapolis)*, **20**, 594–598.
- Masserman, J. H. (1934). Cerebrospinal hydrodynamics. IV. Clinical experimental studies. *Archives of Neurology and Psychiatry*, **32**, 523–553.
- Masserman, J. H. (1935). Cerebrospinal hydrodynamics. V. Studies of the volume elasticity of the human ventriculo-subarachnoid system. *Journal of Comparative Neurology*, **61**, 543–552.
- Merritt, H. H., and Fremont-Smith, F. (1937). *The Cerebrospinal Fluid*. Saunders: Philadelphia.
- Murtagh, F., and Lehman, R. (1967). Peritoneal shunts in the management of hydrocephalus. *Journal of the American Medical Association*, **202**, 1010–1014.
- Ojemann, R. G., Fisher, C. M., Adams, R. D., Sweet, W. H., and New, P. F. J. (1969). Further experience with the syndrome of 'normal' pressure hydrocephalus. *Journal of Neurosurgery*, **31**, 279–294.
- Pollock, L. J., and Boshes, B. (1936). Cerebrospinal fluid pressure. *Archives of Neurology and Psychiatry*, **36**, 931–974.
- Portnoy, H. D., Schulte, R. R., and Fox, J. L. (In preparation.) An anti-siphon valve for hydrocephalic shunts.
- Rayport, M., and Reiss, J. (1969). Hydrodynamic properties of certain shunt assemblies for the treatment of hydrocephalus. Part I. Report of a case of communicating hydrocephalus with increased cerebrospinal fluid production treated by duplication of shunting device. Part 2. Pressure-flow characteristics of the Spitz-Holter, Pudenz-Heyer, and Cordis-Hakim shunt systems. *Journal of Neurosurgery*, **30**, 455–467.
- Rushmer, R. F. (1955). *Cardiac Diagnosis. A Physiologic Approach*, pp. 34–40. Saunders: Philadelphia.
- Rushmer, R. F. (1946). The nature of intraperitoneal and intrarectal pressures. *American Journal of Physiology*, **147**, 242–249.
- Salmon, J. H., and Timperman, A. L. (1971). Effect of intracranial hypotension on cerebral blood flow. *Journal of Neurology, Neurosurgery, and Psychiatry*, **34**, 687–692.
- Sibayan, R. Q., Begeman, P. C., King, A. I., Gurdjian, E. S., and Thomas, L. M. (1970). Experimental hydrocephalus. Ventricular cerebrospinal fluid pressure and waveform studies. *Archives of Neurology*, **23**, 165–172.
- Smyth, G. E., and Henderson, W. R. (1938). Observations on the cerebrospinal fluid pressure on simultaneous ventricular and lumbar punctures. *Journal of Neurology and Psychiatry*, **1**, 226–237.
- Waga, S., Hashi, K., Ohtsubo, K., and Handa, H. (1970). Bilateral chronic subdural hematomas developing after ventriculo-atrial shunt for 'normal pressure hydrocephalus'. A case report. *Brain Nerve (Tokyo)*, **22**, 325–329.

- Weed, L. H., and Flexner, L. B. (1932). Cerebrospinal elasticity in the cat and macaque. *American Journal of Physiology*, **101**, 668–677.
- Weed, L. H., Flexner, L. B., and Clark, J. H. (1932). The effect of dislocation of cerebrospinal fluid upon its pressure. *American Journal of Physiology*, **100**, 246–261.
- Weiss, S. R., and Raskind, R. (1969). Twenty-two cases of hydrocephalus treated with a silastic ventriculoperitoneal shunt. *International Surgery*, **51**, 13–19.
- Wilson, C. B., and Bertan, V. (1967). Interruption of the anterior choroidal artery in experimental hydrocephalus. *Archives of Neurology*, **17**, 614–619.