Body position and
cerebrospinal fluid pressure

Part 1: Clinical studies on the effect of
rapid postural changes

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Cerebrospinal fluid (CSF) pressure was recorded in 149 patients and arterial blood pressure (BP) in 11 patients while moving between lateral and sitting positions. Rapid tilting initiated waves in BP and CSF filling pressure. The postural CSF pressure wave manifested itself either as a transient or as a stationary wave similar to a plateau wave. When patients sat up, transient waves had amplitudes up to 550 and stationary waves up to 1000 mm H₂O. When they lay down, transient waves had amplitudes up to 800 mm H₂O. Stationary waves were found only among patients with elevated intracranial pressure and a diseased brain. The waves were mainly caused by changes in cerebral blood volume probably reflecting the postural BP wave and brain autoregulation. Most patients with stationary and large transient waves also manifested clinical symptoms. These symptoms were aggravated when a craniospinal block developed in the sitting position, and were reduced or avoided when the tilting was performed slowly over 2 to 3 minutes.

KEY WORDS - postural changes - cerebrospinal fluid pressure - hydrostatic pressure - filling pressure - plateau waves - arterial blood pressure - autoregulation

Clinical experience has shown that postural changes in neurosurgical patients may lead to discomfort, headache, vomiting, changes in the level of consciousness, and cardiovascular and sometimes respiratory irregularities. Changes in body posture cause pressure changes in the fluid spaces of the body, in cardiac output and peripheral resistance, and in blood flow to the various vascular beds. Much work has been done to identify the postural pressure changes in the venous and arterial system. For a better understanding of these pressure changes it is necessary to distinguish between changes in the hydrostatic pressure component and changes in the filling pressure.

In the present study the pressure resulting from the weight of the vertical cerebrospinal
fluid (CSF) column is called hydrostatic pressure. The other pressure component, which is a measure of the degree of filling of the craniospinal system within the dura, is called filling pressure. This study attempts to elucidate the postural pressure changes in the CSF space, with the objective of obtaining information that might be useful in handling patients. Emphasis was placed on 1) studying the pressure dynamics in patients as they moved between the lateral and sitting positions, and 2) distinguishing between changes in hydrostatic pressure and filling pressure.

Clinical Material

We studied CSF pressure in 149 patients and arterial blood pressure in 11 adult neurosurgical patients while changing between lateral and sitting positions.

Lumbar CSF Pressure

Lumbar CSF pressure was recorded in 120 patients.

Group 1: Control. Group 1 consisted of 72 patients with neck and arm pains due to cervical spondylosis. A spinal subarachnoid block test was performed on these patients. All patients had normal CSF protein and no hindrance to CSF flow on jugular compression.

Group 2: Subarachnoid Hemorrhage. Group 2 consisted of 15 patients who had suffered a subarachnoid hemorrhage (SAH) from a saccular aneurysm. They were in clinical Grade II or III⁶ and were awaiting operation. Lumbar puncture for pressure measurement and careful CSF drainage was performed. These patients had an intravenous drip running during the tilting procedure, and mannitol was at hand.

Group 3: Hydrocephalus. Group 3 comprised 25 patients with hydrocephalus of whom seven had noncommunicating hydrocephalus. The CSF pressure was recorded before and after insertion of a Pudenz medium pressure shunt. In each case the shunt was determined from an improved clinical condition to be functioning well.

Group 4: Cervical Block. Eight patients had a complete subarachnoid block due to cervical spondylosis. In these cases CSF pressure was recorded before and after decompressive laminectomy.

Ventricular CSF Pressure

Ventricular CSF pressure was recorded in 14 patients with the following diagnoses: six patients harbored a posterior fossa tumor, four had aqueductal occlusion, and four had suffered SAH from an aneurysm.

Lumbar and Ventricular CSF Pressure

Lumbar and ventricular CSF pressure was recorded in four patients. Two of these had communicating hydrocephalus, and two had posterior fossa tumor.

Lumbar CSF Pressure and Arterial Blood Pressure

Lumbar CSF and arterial blood pressure (BP) was recorded in 11 patients, four of whom had suffered an SAH from a saccular aneurysm, four had arteriovenous malformation, and three had hydrocephalus.

Methods

Pressure Recording

Lumbar CSF Pressure. The patients were lying flat for at least 30 minutes and after shunting for at least 2 hours before lumbar puncture. Under local anesthesia a No. 19 spinal needle was introduced between L-4 and L-5. To avoid CSF leakage and needle occlusion during tilting, the dura must be punctured only once and the needle should have a sagittal position. To achieve this all except the SAH patients were sat up for a few seconds during the puncture. The CSF pressure was then recorded with the patient in the lateral position until a stable pressure level was obtained. Shunted patients had a stabilizing period of 30 minutes regardless of whether a stable pressure level had been obtained before that time. The head and spine were levelled horizontally for recording pressure in the lateral position. When the CSF pressure was recorded in the sitting position, the patient was requested to keep erect but relaxed and to fix his eyes on a target on the wall at the same level as his head. A minitransducer and potentiometer recorder* were

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used for the pressure recording. The position of the transducer, between L-4 and L-5 in the sagittal midline, was used as zero reference level for both lateral and sitting positions.

**Ventricular CSF Pressure.** A Fischer ventricular cannula† was inserted into a frontal horn for ventriculography, CSF drainage, or CSF pressure recording. The mini-transducer was connected to the cannula through a 10-cm plastic tubing. The position of the transducer, about 1.5 cm below the top of the head in the sagittal midline, was used as zero reference level for both lateral and sitting positions.

**Arterial Blood Pressure.** Arterial blood pressure (BP) was recorded in the radial or femoral artery by continuous slow infusion of heparinized saline through the hydraulic system.† The position of the transducer, about 1.5 cm below the top of the head in the sagittal midline, was used as zero reference level for both lateral and sitting positions.

**Tilting Procedure**

**Quick Tilt.** The patient was instructed and then assisted in sitting up quickly in about 1 to 2 seconds without performing a Valsalva’s maneuver. The CSF pressure with the patient in the sitting position was recorded for about 5 minutes, but in a few patients for a shorter period of time, and in four patients for about 10 minutes. The patient rapidly resumed the lateral position, and the pressure was recorded until a stable pressure level was obtained.

**Slow Tilt.** In patients with Pattern B and D pressure recordings (see later), a slow tilt was also performed. The patient was tilted to the sitting position in the course of 2 to 3 minutes by elevating the backrest, and slowly tilted back in the same way. The rest of the procedure was as for the quick tilt.

**Results**

**Lumbar CSF Pressure on Sitting Up**

When patients sat up quickly there was a rapid primary rise in CSF pressure. Then followed a period of about 6 to 8 seconds with nearly unchanged or a small fall or rise in pressure. In the subsequent recordings four different pressure patterns could be distinguished based on a secondary CSF pressure rise and CSF pressure pulsation.

**Pattern A.** About 8 seconds after sitting up there was a secondary transient pressure rise (Fig. 1). The time from tilting to maximum pressure was relatively constant at about 15 seconds. The time of the phase of falling pressure did vary, but was usually in the range of 10 to 40 seconds. The magnitude of the pressure rise varied from scarcely visible to 550 mm H₂O, and seemed to increase with increasing CSF pressure in lateral position as seen from Fig. 1. The pressure level before and after the transient pressure rise was not always exactly the same, but usually did not differ with more than 50 mm H₂O. This pattern was found in all control patients, in 10 SAH patients, and in 21 hydrocephalic patients, but in no patient from the cervical block group. A large transient pressure rise was sometimes accompanied by headache and discomfort.

**Pattern B.** About 8 seconds after sitting up there was a secondary stationary or plateau pressure rise (Fig. 2). The secondary pressure rise ranged from 350 to 1000 mm H₂O, and remained relatively stable during the 5-minute recording. This pattern was found in two SAH patients, and in four hydrocephalic patients who had intracranial hypertension ranging from 350 to 700 mm H₂O, and clinical symptoms and signs of a diseased brain. All Pattern B patients except one felt discomfort and headache in the sitting position and during the first few seconds after lying down.

Tracings from a Pattern B patient are shown in Fig. 3. When the patient sat up, there was a primary pressure rise and after a few seconds a secondary stationary pressure rise. When he lay down, there was a fall to a new pressure level lasting for a few minutes before a spontaneous rapid fall to below the pretilting pressure level (Fig. 3 A). The level and subsequent spontaneous pressure fall was similar to the second half of the curve shown in Fig. 3 B, which probably was a plateau wave elicited by infusion of 3 ml of artificial CSF during a test of CSF absorb- tive capacity.

A slow tilt was also performed in the six Pattern B patients. When the sitting position was reached, the pressure level in five of the

†Fischer ventricular cannula manufactured by Heyer-Schulte Corporation, 5377 Overpass Road, Santa Barbara, California.
Fig. 1. Tracings showing typical Pattern A lumbar CSF pressure during rapid tilting in a hydrocephalic patient (A), and an SAH patient (B). Sitting up: Rapid primary rise in hydrostatic pressure and secondary transient rise in filling pressure. Variation in amplitude of transient wave and phase of falling pressure. Lying down: The immediate pressure fall was as expected in A, and less than expected in B.

Fig. 2. Tracings showing typical Pattern B lumbar CSF pressure during rapid tilting. A is a recording from an SAH patient; B is from a hydrocephalic patient. Sitting up: Rapid primary hydrostatic pressure rise (PPR) and secondary stationary rise in filling pressure. Lying down: The immediate pressure fall is less than expected.
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FIG. 3. Tracings showing Pattern B lumbar CSF pressure in a hydrocephalic patient. Rapid tilting (A), and infusion of 3 ml of mock CSF (B), resulted in pressure changes similar to a plateau wave. Note the paper speed.

patients corresponded to the primary pressure level during the quick tilt. In one patient the pressure level nearly reached the secondary stationary increased pressure level during the quick tilt. The patients experienced no discomfort during this procedure.

A quick-tilting procedure was again performed in the six Pattern B patients 3 months after aneurysm surgery or CSF shunting. The CSF pressure was then normal and the clinical condition markedly improved in all patients. A quick tilt was performed both from the initial CSF pressure level and from the pressure level measured when Pattern B was recorded. This pressure level was obtained by continuous infusion of mock CSF during a test of absorptive capacity. This time Pattern B was recorded in one hydrocephalic patient from the elevated pressure level. The other recordings showed Pattern A.

Pattern C. Patients having a complete cervical block had no secondary pressure rise when sitting up (Fig. 4 A). These patients had a low CSF pressure in the lateral position. The pressure was increased to 150 mm H2O by infusion of artificial CSF, and on a new tilt Pattern C was again recorded in all patients.

Pattern D. Reduced or absent arterial pulsation and no secondary pressure rise with the patient in the sitting position was found in three SAH patients. They had a CSF pressure in the lateral position of 480, 580, and 750 mm H2O respectively (Fig. 4 B). These patients experienced headache, discomfort, and faintness in the sitting position. Tachy- or bradycardia and a progressively weakening pulse indicated failing systemic circulation, and the patients had to be tilted back to the lateral position.

When performing a slow tilt, the arterial pulsation in the lumbar CSF pressure records was maintained all the time and the patient felt nearly no discomfort.

Lumbar CSF Pressure on Lying Down

Pattern A. Forty-one patients had the expected immediate pressure fall back to the pre-tilting level (Fig. 1 A). In 62 patients the immediate pressure fall was less than ex-
FIG. 4. Tracings showing lumbar CSF pressure during rapid tilting. A shows a Pattern C recording from a patient with complete cervical block. There is no secondary rise in filling pressure. B shows a Pattern D tracing from a patient with intracranial hypertension due to SAH. A reduced arterial pulsation and no secondary pressure rise indicate orthostatic craniospinal block.

expected, and the pressure then returned to the pre-tilting level usually within 10 to 40 seconds (Fig. 1 B). Eight patients had a transient pressure rise immediately after resuming the lateral position.

Pattern B. Two patients had the expected immediate pressure fall, while four patients had an immediate pressure fall less than expected (Fig. 2). The further pressure fall back to the pre-tilting level varied from 20 seconds to about 5 minutes (Fig. 3).

Ventricular CSF Pressure

In principle, the same primary and secondary CSF pressure changes were recorded during tilting as those seen in the lumbar recordings. However, the primary pressure change was in the opposite direction in these tracings compared with the lumbar records, and that made some observations more apparent.

Pattern A. Pattern A was recorded in 11 patients (Fig. 5). When patients sat up, there was a primary pressure fall and a secondary transient pressure rise. When they lay down, there was a rapid rise in CSF pressure directly in an overshoot phase to 1300 mm H₂O with nearly no time lag. This pressure rise was larger than expected from the initial pressure fall, and corresponded to the tracings from the 62 patients who had an immediate pressure fall in the lumbar records that was less than expected when lying down.
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FIG. 5. Tracings showing ventricular CSF pressure during rapid tilting in a patient with aqueductal occlusion. Sitting up: Rapid fall in hydrostatic pressure and secondary transient rise in filling pressure. Change in hydrostatic pressure and filling pressure in sequence. Lying down: Nearly concomitant rise in hydrostatic pressure and filling pressure in an “overshoot” phase. (Pattern A).

FIG. 6. Tracings showing ventricular CSF pressure during rapid tilting in a patient with posterior fossa tumor. Sitting up: Large secondary stationary rise in filling pressure resulting in higher intracranial pressure on sitting than in lateral position. Lying down: Pressure level and pressure fall similar to a plateau wave. (Pattern B).

Pattern B. Pattern B was recorded in three patients with posterior fossa tumor. Two of these patients had spontaneous plateau waves in the previous pressure recording. Figure 6 is a tracing from one of these patients, showing that when the secondary stationary pressure rise was larger than the primary pressure fall, then the intracranial pressure was higher when the patient was sitting than when lying. The CSF pressure after the patient lay down was similar to the second half of a plateau wave.

Lumbar and Ventricular CSF Pressure

In lumbar and ventricular CSF pressure recordings it was seen that the primary change in lumbar and ventricular CSF pressure was in the opposite direction, while the secondary pressure change was in the same direction. The points where the tracings began to run parallel are shown in Fig. 7. The time from the start of sitting up to these points was about 2 seconds in this patient, and in all four patients between 1 and 3 seconds. The periods were a little shorter when lying down.

Blood Pressure and CSF Pressure

The postural BP pattern consisted of a fall when the patient sat up, a compensatory rise in the sitting position, and a rise when the patient lay down (Fig. 8). This pattern did not differ in patients having a transient or stationary CSF pressure rise in the sitting position.

The time sequence of BP and CSF pressure changes during a rapid-tilting procedure is shown in detail in Fig. 9. The BP fall when the patient sat up lagged behind the positional
change, while the BP rise on lying down was nearly concomitant with the positional change. The secondary transient or stationary CSF pressure rise started concomitant with the compensatory BP rise in the sitting position. The CSF pressure level during the phase of falling BP was rather stable as seen in Fig. 9 B.

Discussion

Mechanical Event and Biological Waves

The results seemed to point to the following main conclusions:

1. Rapid tilting is a mechanical event initiating biological waves in BP and CSF filling pressure.
2. The postural BP wave was relatively uniform from patient to patient.

Fig. 7. Tracings of lumbar (L) and ventricular (V) CSF pressure in a hydrocephalic patient during rapid sitting up. Primary pressure changes (PPR and PPF) in opposite directions, secondary pressure changes in same direction. Arrow with star indicated end of postural CSF redistribution.

Fig. 8. Arterial blood pressure (BP) and lumbar CSF pressure tracings during rapid tilting in one patient with SAH from an aneurysm (A), and another with an arteriovenous malformation (B). Same postural BP pattern in the patient with transient (A) and the patient with stationary (B) CSF pressure rise in sitting position.
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3. The postural CSF pressure wave manifested itself either as a transient or as a stationary wave similar to a plateau wave, and was mainly caused by changes in cerebral blood volume (CBV), probably reflecting the BP wave and brain autoregulation.

CSF Redistribution, Total CSF Volume, and CBV

Postural changes result in a redistribution of CSF within the craniospinal space and altered hydrostatic CSF pressure components. The hydrostatic change in ventricular and lumbar CSF pressure will be in the opposite direction because the pressures are recorded on each side of the hydrostatic indifferent point (HIP), and the magnitude of the hydrostatic pressure change will depend on the distance from the HIP. Changes in CSF filling pressure, however, will result in an equal deflection in the same direction in ventricular and lumbar tracings. This occurred 1 to 3 seconds after the start of sitting up or lying down indicating the postural redistribution of CSF to be completed within this time. In other words, from that point on, recorded pressure reflected changes in filling pressure. This is also what would be expected, that in a watery system with free communication CSF redistribution is mainly completed when the new body position is reached.

In most patients with transient CSF pressure waves the pre-tilting pressure level was reached within 40 seconds of lying down, which indicated a limited postural change in total CSF volume. During the first 20 to 30 seconds in the sitting position, a period that is important for the interpretation of the tracings, total CSF volume must be considered unchanged. Consequently, transient waves and at least the first part of stationary waves...
waves mainly reflected changes in blood volume, or more precisely in CBV because the waves were not seen in the lumbar recordings from patients having complete cervical block. The rapidity of the secondary pressure changes also supports this explanation.

**CSF Pressure Wave Reflecting BP Wave and Brain Autoregulation**

The most constant finding was the start of a CSF pressure wave concomitant with the two phases of rising BP. The most likely explanation is that rapid BP changes may cause volume changes in the intracranial arteries as a mechanical result of the sudden change in endovascular pressure, as well as volume changes from the arterioles and downstream before autoregulation has time to become effective. The further shape of the CSF pressure wave seemed to reflect the autoregulatory response.

Transient CSF pressure waves were found in all control patients and must be considered the normal postural CSF pressure wave. In patients with elevated intracranial pressure, the amplitude of the waves was larger probably reflecting the less compliant craniospinal space. When patients lay down, the transient wave manifested itself as the "overshoot" phase in the intracranial recordings or "less than expected immediate pressure fall" in the lumbar recordings.

Stationary CSF pressure waves, of which two were similar to plateau waves, were found only among patients having elevated intracranial pressure and a diseased brain. Stationary waves most likely reflected the vasomotor instability of a diseased brain. Stationary waves as a passive mechanical effect of elevated intracranial pressure per se seemed less reasonable because all except one patient with stationary waves at a later stage when the clinical condition had improved markedly, showed transient postural waves when the CSF pressure was artificially elevated.

Spontaneous plateau waves are assumed to be triggered by an initial small elevation in intracranial pressure probably due to accumulation of CSF, and they are accompanied by a rise in CBV. The postural trigger seems to be different. Rapid postural changes put a simultaneous stress on the blood circulatory system, on craniospinal CSF redistribution and transmission of pressure, and on brain autoregulation. From some tracings, for instance, those in Fig. 2 B, it is tempting to assume the following sequence: rapid tilting causes a transient CSF pressure wave that, in patients with impaired autoregulation, may trigger a plateau wave.

During the phase of falling BP the CSF filling pressure in most patients remained relatively unchanged, in some showed a limited fall, and in a few, a limited rise. In other words, CBV was more constant during falling BP compared with the two phases of rising BP. One explanation might be that the first BP rise was a direct continuation of the BP fall and that the second BP rise was the steepest, two circumstances that may put extra stress on the brain vasoregulatory system. Another explanation might be that the regulatory response is not equal for a fall or rise in BP.

**Orthostatic Craniospinal Block**

Pattern D with loss of CSF pressure pulsation and no secondary CSF pressure rise in the lumbar recordings strongly indicated a craniospinal block with the patient in the sitting position. The CSF pulsation disappeared nearly immediately on tilting. Thus the block was probably caused by the craniospinal redistribution of CSF. A craniospinal block reduces intracranial compliance, and the intracranial postural CSF pressure wave must be assumed to be augmented accordingly. This will increase the risk of progressive impaction at the tentorial notch or foramen magnum, and the failing systemic circulation in these patients indicated pressure on the caudal brain stem.

**Clinical Implications**

In some patients with elevated intracranial pressure rapid sitting up or lying down led to clinical symptoms, which occurred concomitant with large transient or stationary CSF pressure waves. The symptoms were aggravated when a craniospinal block developed with the patient in the sitting position, and they were reduced or avoided when the tilting was performed slowly over the course of 2 to 3 minutes.

**References**


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