

## Subcutaneous Cranial Migration of A Ventricula Peritoneal Shunt: Case Report

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**Abstract :** We report another case of upward shunt migration in a newborn boy suffering from hydrocephalus caused by aqueduct stenosis. Several hypothesis trying to explain this dysfunction are

presented. Measures to prevent this complication are suggested.  
**Key Word :** Ventriculo-peritoneal shunt, Migration, Pressure adjustable Valve

### INTRODUCTION

Since Scott and Jackson in 1955 (2, 6), numerous cases of upward, often intracranial, migrations of peritoneal shunts have been reported. All of these cases involved unishunt or valveless systems, that could offer no mechanical resistance to the ventricular or subdural aspiration observed under certain circumstances (1,3,4,8,9,10).

We report a case involving a ventriculo-peritoneal (VP) shunt featuring a Pressure Adjustable Valve Sophy (PAVS) presenting an unusual migration of the peritoneal part of the catheter.

### CASE REPORT

The patient was born on October the 5th, 1991, after an uneventful pregnancy. At the age of one month, the cranial perimeter began to show an abnormally rapid growth. No sign of intracranial hypertension had previously been noticed. An MRI scan performed on February the 14th, 1992, revealed a triventricular hydrocephalus, secondary to a membranous aqueduct stenosis. (Fig. 1)

At this time, the fontanel was large and bulging, but the neurological examination showed a normal

developmental level, and no functional deficit. A left VP shunt featuring a PAVS set on medium resistance, (fig. 2), was performed on March the 10th, 1992. As a post operative collapse of the fontanel was observed, the PAVS was then set on a higher resistance. The patient left the hospital on March the 21st, 1992, without any neurological deficit. Postoperative transfontanellar echography showed improvement of hydrocephalus.

He was admitted again three weeks later, on April the 14th, 1992, following progressive deterioration of consciousness, bilateral Parinaud syndrome, and showing a large fluctuant bulge behind the left ear, on the location of the valve.

CT scan revealed the recurrence of hydrocephalus, and skull X ray showed that the peritoneal catheter had moved upwards from the peritoneal cavity, now describing subcutaneous loops around the valve. (Fig 3). After checking the patency of the shunt, the catheter was repositioned in the peritoneal cavity, and a tight compressive dressing was placed over the PAVS, in order to avoid a new migration. The neurological status after six months remained satisfactory.



Fig. 1: MRI T1 weighted sagittal scan showing triventricular hydrocephalus, and a membranous aqueduct stenosis. (Feb. 2nd, 1992)



Fig. 2: Skull X-Ray showing the ventricular catheter and the pressure adjustable valve immediately after VP shunting. (March 12th, 1992)

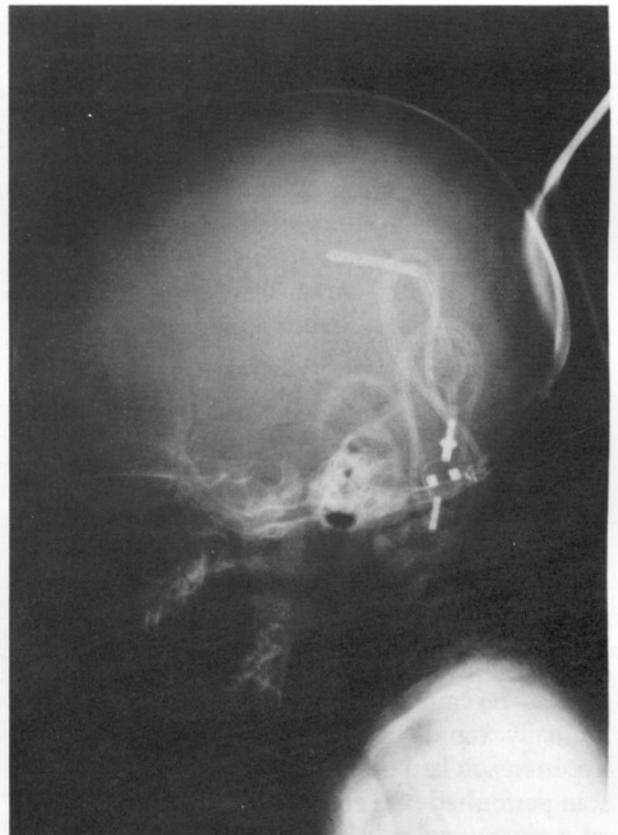


Fig. 3: Skull X-Ray showing the migrated peritoneal catheter describing loops around the PAVS. The ventricular catheter remains normally positioned. (April 15th, 1992)

## DISCUSSION

In 1989, Thauvoy and al (7,8) described a case of upward intracranial migration of a VP shunt. His etiological hypothesis was intracranial aspiration caused by negative intracranial pressure consecutive to a mechanism referred to as the "windlass effect". In this case, the impossibility of intracranial aspiration due to the volume of the valve made us search for other hypothesis

A likely theory in this case is that a high resistance pocket located at the tip of the peritoneal catheter would push the catheter upwards, step by step until the valve is reached. Although no complete shunt obstruction could be put forward, it might be that intermittent shunt dysfunction causes the same effect, delaying the onset of clinical hydrocephalus.

Upward migration of VP shunt have also been attributed to frequent flexion and extension

movements of the neck, or by frequent crying with a Valsalva effect, but no positive demonstration of these hypotheses has ever been published. Furthermore, crying and neck movements are very usual, and yet, cranial migration remains a rare complication.

Several prevention measures to prevent cranial migration of a ventriculo-peritoneal shunt can be considered :

1. The use of a valve whose diameter is larger than the burr hole created for the introduction of the ventricular catheter, as in this case a PAVS, would make intracranial aspiration of the entire shunt device impossible.

2. Safe anchoring of the valve, as described by Pang and Wilberger (1980), (5) could be a reliable alternative. Anchoring of the catheter to the abdomen is the method of choice in adults, but it should not be used with children, as normal growth would rapidly cause the rupture of the shunt.

3. Finally, subcutaneous aspiration could further be prevented by avoiding subcutaneous CSF collection during the first post operative weeks. This could easily be obtained by a long term compressive dressing.

In conclusion, we believe that, although these simple methods might be efficient in preventing this rare but life-threatening complication, the real mechanism causing this cranial subcutaneous migration remains unclear. Our etiological speculations could be better approached in an experimental animal model, that would give us better knowledge of hydrodynamic happenings during CSF drainage.

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