# CSF FLOW IMAGING IN THE ACUTE PHASE OF MENINGEAL HAEMORRHAGE

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## Introduction

The study of cerebrospinal fluid (CSF) flow dynamics by cine phase-contrast MR imaging shows that CSF oscillations are synchronized with cerebral blood flow[1] and are also involved in the control of variations of intracranial pressure during the cardiac cycle. Hydrocephalus is characterized by abnormal dilatation of the ventricular system and is classified into two types: non-communicating and communicating hydrocephalus. In some cases, morphological MR imaging fails to visualize the site of obstruction. These patients fairly frequently report a history of meningitis in childhood or meningeal haemorrhage (MH). The aim of this study was to investigate the possible alterations of CSF flow dynamics during the acute phase of meningeal haemorrhage due to rupture of an aneurysm.

## Patients, Material and Methods

Seventeen patients (9 females and 8 males) with a mean age of 51 ± 10 years, with MH confirmed by CT scan were examined by MR imaging during the first 24 hours after rupture of the aneurysm (1.5 T - GE Healthcare - version 9). MR imaging comprised morphological sequences for visualization of the MH and determination of its cause and flow dynamic sequences for quantification of CSF oscillations and cerebral blood flow rates. CSF oscillations were recorded at the cerebral aqueduct, cisterna pontis and C2-C3 subarachnoid space (SAS), where a vascular sequence was also performed to quantify internal carotid artery and vertebral artery blood flow. The cine phase-contrast sequence was performed with peripheral cardiac gating, using the following parameters: TR: 30 ms / TE: 12-17 ms / FOV: 60 x 120 mm / Matrix: 256 x 128/ Slice thickness: 5 mm / Tilt angle: 30°.

Dynamic flow images were analyzed on dedicated software, developed on site, allowing calculation of flow curves over the cardiac cycle[2]. The oscillations measured in this population were compared to oscillations of a population of control subjects[1] and a difference of at least two standard deviation was used to define a hyperdynamic or hypodynamic appearance of CSF flow.

Dilatation of the ventricular system was determined on radiographs by two neuroradiologists.

## Results

No significant modification of cerebral blood flow was demonstrated in the patients of our population (636 ± 126 ml/min versus 675 ± 120 ml/min for the control population). Only 7 patients presented normal ventricular CSF flow, 2 patients presented hypodynamic flow and 8 patients presented hyperdynamic flow. Eleven patients had normal cervical CSF flow, 1 patient presented hypodynamic flow and 5 patients presented hyperdynamic flow.

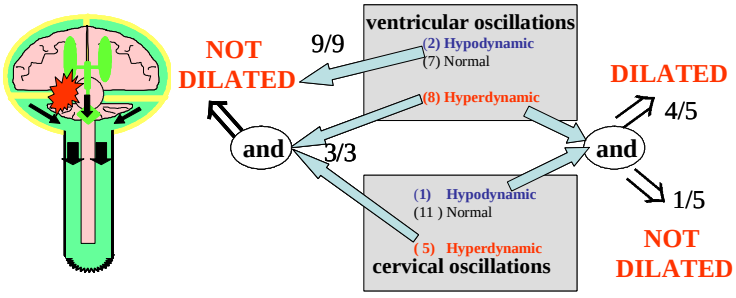
Patients with no morphological signs of ventricular dilatation presented either hypodynamic or normal ventricular CSF flow or hyperdynamic ventricular CSF flow associated with hyperdynamic cervical CSF flow. The 4 patients with ventricular dilatation all presented hyperdynamic ventricular CSF flow associated with normal or decreased cervical CSF flow (Figure 1).

## Discussion

Abnormal CSF flow dynamics were demonstrated in the absence of any modification of cerebral blood flow, suggesting the hypothesis that bleeding increases intracranial volume and induces a reduction of cerebral compliance and an increase of intracranial pressure. This increased pressure affects the amplitude of CSF oscillations, a physiological shock-absorber of variations of intracranial volume during the cardiac cycle. When bleeding causes increased resistance to flow in the subarachnoid spaces, the cerebral parenchymal pressure is transmitted to the intraventricular CSF. The increased oscillations in the ventricular system would therefore predispose to dilatation (Figure 2). This is the first study to quantify the impact of bleeding on cerebral hydrodynamics and the development of ventricular dilatation. A better understanding of these mechanisms would help to elucidate the pathophysiology of idiopathic hydrocephalus.

**References** 1 balédent O et al. Invest Radiol 2001;36(7):368-377

Figure 1. Relationship between CSF oscillations and ventricular dilatation.



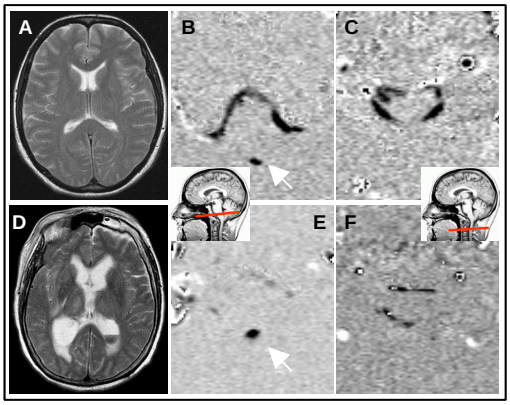




Figure 2. (A, D): FSET2. (B, E) Cine phase-contrast sequence of the cisterna pontis and the outlet of the 4th ventricle. (C, F) Cine phase-contrast sequence of C2-C3. B and D show features of hyperdynamic intraventricular flow (->). In contrast, in E and F, CSF flow in the subarachnoid spaces is altered and this patient presents a ventricular dilatation which was not demonstrated on image A, showing global hyperdynamic CSF flow (B, C).