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## MR Identification of Chiari Pathophysiology by Using Spatial and Temporal CSF Flow Indices and Implications for Syringomyelia

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## MR Identification of Chiari Pathophysiology by Using Spatial and Temporal CSF Flow Indices and Implications for Syringomyelia

What key parameters should be used to evaluate symptomatic Chiari malformations? Conventional MR imaging is necessary to define the anatomic features of the brain and to describe the abnormalities of the posterior fossa and cerebellar tonsils. This is not always sufficient to show the extent or nature of the abnormalities. A need exists for techniques that can better define the causative factors, especially in complex cases with multiple abnormalities or in cases with borderline tonsillar herniation in which it is necessary to determine which patients might benefit from decompression. Previous reports have indicated little correlation between the degree of tonsillar descent and the presence or absence of syringomyelia. Additional anatomic measurements, such as posterior fossa size and geometry, have helped to some extent. However, because hydrodynamic factors have been strongly associated with the pathogenesis of syringomyelia, MR phase-contrast evaluation of CSF flow and neuraxis motion (1) has become the method of choice for the evaluation of Chiari pathophysiology and symptomatology. With the application of new MR imaging techniques for measuring CSF flow and cord motion, multiple additional physiological parameters have been reported in the literature. In this issue of the *AJNR*, Haughton et al attempt to validate peak CSF velocity as an index for the evaluation of patients who are symptomatic for Chiari malformations. Both systolic and diastolic CSF peak velocities seem increased in patients compared with healthy control volunteers. Because this parameter provides a specific numerical value, it is certainly appealing and convenient as an index for grading the degree of pathophysiology in symptomatic patients with Chiari malformations. This increased simplicity may, however, come at the cost of decreased specificity and sensitivity.

It is generally thought that the Chiari malformation leads to partial obstruction of fluid flow near the foramen magnum. Alterations in CSF flow resulting from a small posterior fossa and tonsillar obstruction lead to abnormalities in fluid pressures in the spinal canal and cord, with decreased CSF flow (mL/min) and increased CSF velocity (cm/s) near the foramen magnum. Velocity patterns in patients with Chiari malformations with and those without syringomyelia have previously been reported. The spatial and temporal changes, however, have not been well characterized. Are there specific key parameters that can be used to characterize these complex spatial and temporal dynamics? In this study, the peak systolic and diastolic velocities for patients with Chiari malformations were found to be elevated relative to those of

healthy control volunteers, but with moderate overlap. These results differ somewhat from certain other studies, but it should be noted that differences in MR imaging techniques and in patient population could be factors. In this study, measurements were obtained by using axial planes below the foramen magnum to detect local spatial variations in peak velocities. Other investigators have also used axial planes and have reported increased systolic and diastolic velocities and increased caudal but not rostral flow rates occurring in patients with Chiari and syringomyelia, with normalization achieved after surgery (2). Measurements of CSF pressures and craniospinal compliance in these studies provide additional insights into the pathophysiological mechanisms. However, these measurements are limited to particular anatomic regions and the analysis does not significantly involve the spatiotemporal nature of CSF-neuraxis dynamics and CSF pulse propagation. As discussed previously, brain stem motion has also been evaluated and found to be elevated in cases of Chiari malformation (3). In the present article, only a limited number of patients with significant cerebellar tonsillar descent or with syringomyelia were included, which makes characterization of the nature of the flow abnormalities difficult. Additional studies using a more specific patient population and comparing symptomatic cases with asymptomatic patients (rather than with normal control volunteers) would be useful.

This study also indicates the presence of spatial variations in the peak velocities, from midline to lateral. This may be due to a number of factors, including the presence of a small posterior fossa and low cerebellar tonsils, differences between the flow from the foramen of Luschka laterally, the foramen of Magendie and valleculla medially, or the presence of adhesions. Other disorders such as disk disease and spinal stenosis are associated with increased peak velocities near the site of stenosis and also have spatially inhomogeneous flows, but they do not present with symptoms of Chiari malformation or with syringomyelia. Thus, the value of using increased peak velocities or flow inhomogeneity to assess cases of symptomatic Chiari malformations does not seem to be specific.

Technically, measurements of CSF flow can be limited for a number of reasons. In this study, the use of a low temporal sampling rate may not accurately measure rapid changes in cord motion and CSF. Axial sections were chosen at a level below the tonsils at which increased turbulence and converging flows may be present, which may lead to underestimation of velocity from dephasing. Bidirectional CSF flow was

observed at that level, probably because bidirectional flows last longer in patients with Chiari malformations compared with those in control volunteers and are thus easier to observe. Also of concern is that errors in peak velocities can be produced if the epidural plexus flow adjacent to the CSF regions of interest is included in the measurements.

The presence of a small or abnormal posterior fossa may also contribute to Chiari symptomatology and dynamics, probably more to brain stem symptoms and abnormal brain stem motion than to CSF flow abnormalities. The role of adhesions in obstructing or redirecting flow may also be important, regardless of the amount of tonsillar descent. It may be worthwhile to verify whether the patients included in a study have specific abnormalities such as tonsillar adhesions, whether tonsillar motion is present, or whether the foramen of Luschka or Magendie is obstructed differently, thereby compounding differences between midline and lateral flows. In addition to CSF flow abnormalities, patients with Chiari malformations have greater motion of the medulla and brain stem than do control volunteers, which can also produce greater spatial flow variations. Theoretically, symptoms related to those anatomic regions could be secondary to regional dynamic tissue deformations. In general, clinical manifestations of Chiari malformations seem to be related to both CSF disturbances and direct compression of nervous tissue, although the structural abnormalities may be causative.

The craniospinal axis is a *spatially distributed system*, with an elongated extended geometry, such that the regional dynamic properties vary along its length. Changes occurring in one region may thus affect the dynamics in another region, reminiscent of referred pain syndromes. Craniospinal dissociation is such an example, with a loss of distal compliance due to a proximal obstruction. CSF flow abnormalities may originate from sites remote from regions of altered flow due to wave propagation effects. Present studies indicate that in syringomyelia, increased spinal pulse

pressures may lead to abnormal transmedullary pressure gradients and force movement of interstitial fluid across the spinal cord. The resulting spinal pressure waves and associated CSF flow velocities may develop increased peaks; however, other parameters, such as rate of change, propagation, or duration of these pulses, may also be important. Importantly, prolongation of CSF systole, associated with decreased spinal compliance due to mechanical obstruction, may result in abnormal pulse pressures and pulse propagation along the neuraxis, contributing to the pathogenesis of syringomyelia. Future examination of flow indices may thus need to include spatially and/or temporally separate effects, and more detailed approaches, such as multidimensional vector fields, may be useful to more accurately identify the pathophysiology (4). Combined measurements, involving both CSF flow and neuraxis motion, may thus provide the most specific and comprehensive indices with which to evaluate patients with Chiari malformations, with or without syringomyelia.

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## New Model of Minimally Invasive Experimental Spinal Cord Injury

In this issue of the *AJNR*, Dr. Purdy and colleagues present a new canine model of experimental spinal cord compression injury. They fluoroscopically manipulated a balloon catheter intrathecally from the lumbar region to the thoracic region and then inflated the balloon to create the injury. Animals underwent imaging with a 1.5-T magnet at the time of balloon inflation. This technique could have advantages over

other widely used experimental spinal cord compression injury models, such as the weight-drop method, transection, and maintained compression.

The weight-drop method results in contusion injury. Laminectomy is performed over the spinal cord region of interest, and a known weight is dropped from a known height onto the exposed spinal cord. This method is widely used in the rat because of its