Reduction in CSF Pulsatility with Altered Intracranial Compliance by Craniectomy in Communicating Hydrocephalus

S. Rashid[1], J. P. McAllister[2], M. Schuhmann[3], and M. Wagshul[4]

[1]Biomedical Engineering, Stony Brook University, Stony Brook, NY, United States, [2]Primary Children's Medical Center, University of Utah, Salt Lake City, UT, United States, [3]Klinik für Neurochirurgie, Eberhard Karls Universität, Tübingen, Germany, [4]Gruss MRRC, Albert Einstein College of Medicine, Bronx, NY, United States

Introduction Communicating hydrocephalus (CH) is a neurodegenerative disease characterized by dilated cerebral ventricles and hyperdynamic pulsatile CSF flow in the cerebral aqueduct, evident from MRI (*1*). Currently, its etiology is poorly understood. Numerous studies have shown that CH is accompanied by reduced intracranial compliance, (*2*), which has been suggested as a causative factor involved in the development of CH (*3*). It follows that a procedure such as decompressive craniectomy, which increases intracranial compliance, and may provide a useful technique for studying the effects of compliance on CH characteristics. We hypothesize that increased compliance, produced by craniectomy, will reduce ventricular dilation as well as elevated CSF pulsations in a rat model of CH.

Methods CH was induced in adult Sprague-Dawley rats by injecting kaolin into the basal cisterns (*4*). CH was characterized by measuring pulsatile CSF stroke volume (SV) at the aqueduct using a cardiac gated phase contrast MR scan (TE/TR = 6/10, 128x128 matrix, ST = 1 mm, FOV = 3 cm, velocity encoding = 1-2 cm/s). SV was calculated as the net volume of CSF flowing in one direction over a cardiac cycle. CH was also characterized by measuring ventricular volume (VV) using a balanced 3D TrueFISP MR scan (TR/TE = 2/4, 128x128x100 matrix, ST = 3.2 mm, FOV = 3 cm) and a T_2 weighted 2D FSE sequence (TR/TE = 36/3074, ETL = 8, 256x256 matrix, 40 slices, ST/gap = 0.6/0.1 mm, FOV= 3 cm). Volume was calculated by segmenting the CSF encompassing the lateral and third ventricles and aqueduct.

Bilateral craniectomies (4x10 mm each) were made over the parietal bone two weeks after induction of CH. VV and SV were measured prior to and immediately after craniectomy. VV and SV were compared before and after craniectomy. The dura was left intact in all animals, although small tears were present in some. All animals were intubated and ventilated on isoflurane/O_2 during MRI.

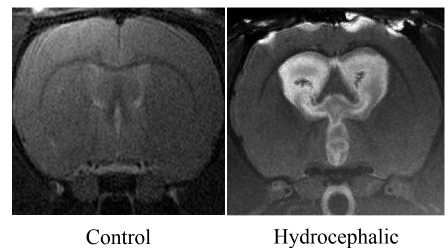

Figure 1. T_2 weighted axial slices of anterior lateral ventricles at the Foramen of Monroe of a control (left) and a CH (right) animal. Dilated ventricles are clearly evident in the CH animal but almost barely visible in the control.

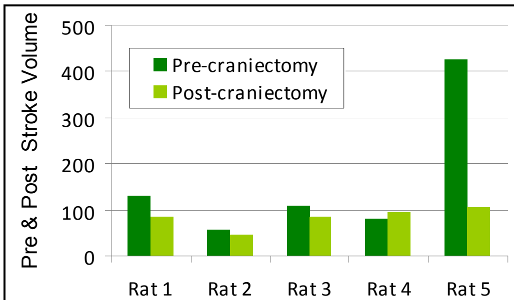

Figure 2. Bar graph showing SV before craniectomy (blue) and after craniectomy (purple) for the first group of animals (post-induction group). The animal at the right was the most severely hydrocephalic and had the

Results Animals that failed to develop CH were excluded. Fig. 1 shows T_2 weighted axial slices of lateral ventricles at the Foramen of Monroe of a control and a CH animal. Of the five animals used in analysis, most exhibited only small changes in VV after craniectomy. In contrast, four of the animals had considerable reduction in SV post craniectomy. The change varied over a wide range (22-75%), with the most severely hydrocephalic animal showing the largest decrease (Fig. 2). Moreover, the ratio of post-craniectomy to pre-craniectomy SV plotted against VV showed a linear trend ($R^2 = 0.98$, $p < 0.02$), with the exception of one animal that had no change in SV after craniectomy and was treated as an outlier (Fig. 3).

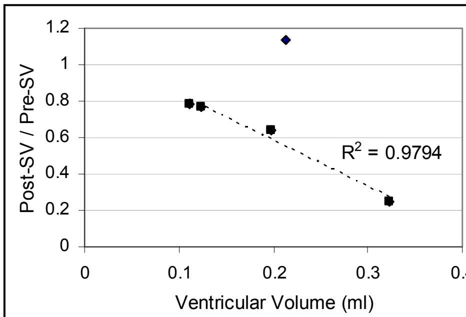

Figure 3. Graph of the ratio of post-craniectomy to pre-craniectomy SV vs. ventricular volume for the first group (post-induction group). Except for one outlier (which showed no change in SV post craniectomy), there was a linear relation in percentage of SV reduction with ventricular size.

Discussion This was a novel and preliminary study testing the effect of altered intracranial compliance on pulsatile CSF flow. Our results indicate that CSF pulsatility is closely linked to compliance, and that increased compliance reduces pulsatile CSF flow. The relationship between pulsatility decrease and ventricular dilation also indicates that this effect is more pronounced for more severe forms of CH. So, increased intracranial compliance may help ameliorate functional and behavioral deficits in CH, although this is purely speculative at this point. Further study is needed to draw solid conclusions, and the effect of altering compliance by other techniques (such as CSF infusion and shunting) on pulsatility and ventricular dilation also needs to be investigated.

References

1. W. G. Bradley, Jr. *et al.*, *Radiology* 198, 523 (Feb, 1996).
2. J. M. Gonzalez-Darder, J. L. Barcia-Salorio, *Acta Neurochir (Wien)* 97, 166 (1989).
3. D. Greitz, *Neurosurg Rev* 27, 145 (Jul, 2004).
4. J. Li *et al.*, *Exp Neurol* 211, 351 (Jun, 2008).