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**PATHOLOGICAL BIOMECHANICS OF CEREBROSPINAL FLUID  
PRESSURE IN SYRINGOMYELIA: FLUID STRUCTURE INTERACTION OF AN  
IN VITRO COAXIAL ELASTIC TUBE SYSTEM**

**Bryn A. Martin<sup>1</sup>, Richard Labuda<sup>2</sup>, Thomas J. Royston<sup>3</sup>,  
John N. Oshinski<sup>4</sup>, Bermans Iskandar<sup>5</sup>, Francis Loth<sup>1</sup>**

<sup>1</sup>Department of Mechanical  
Engineering, University of  
Akron, Akron, OH

<sup>2</sup>Chiari and Syringomyelia  
Patient Education Foundation,  
Wexford, PA

<sup>3</sup>Department of Mechanical and  
Industrial Engineering,  
University of Illinois at Chicago,  
Chicago, IL

<sup>4</sup>Department of Radiology and  
Biomedical Engineering, Emory  
University, Atlanta, GA

<sup>5</sup>Department of Neurological  
Surgery, University of Wisconsin  
Medical School, Madison, WI

**ABSTRACT**

Full explanation for the formation and pathogenesis of syringomyelia (SM), a neurological pathology characterized by the formation of a cystic cavity (syrinx) in the center of the spinal cord (SC), has not yet been given. The SM pathology forms a coaxial elastic tube system with the inner tube formed by the spinal cord having a syrinx and the outer tube formed by the spinal column (dura and vertebrae). It has been assumed that abnormal cerebrospinal fluid (CSF) pressure caused by subarachnoid space (SAS) flow blockage (stenosis) is the underlying cause of syrinx formation and subsequent pain in the patient, but paucity in detailed in vivo pressure data have made theoretical explanation for the syrinx difficult. In order to understand this complex pressure environment, four in vitro models representative of various conditions associated with SM were examined. Overall, interaction of the syrinx and stenosis resulted in a diastolic valve mechanism which could have the effect of syrinx enlargement. In all experiments, the blockage was shown to increase and dissociate SAS pressure, while longitudinal pressure in the syrinx remained largely unchanged. These results provide data for validation of computational models and existing SM theories.

**Keywords:** Spinal cord, fluid structure interaction, cerebrospinal fluid, syringomyelia, Chiari malformation, hydromyelia, biofluid mechanics, subarachnoid space, syrinx, intracranial pressure, in vitro model.

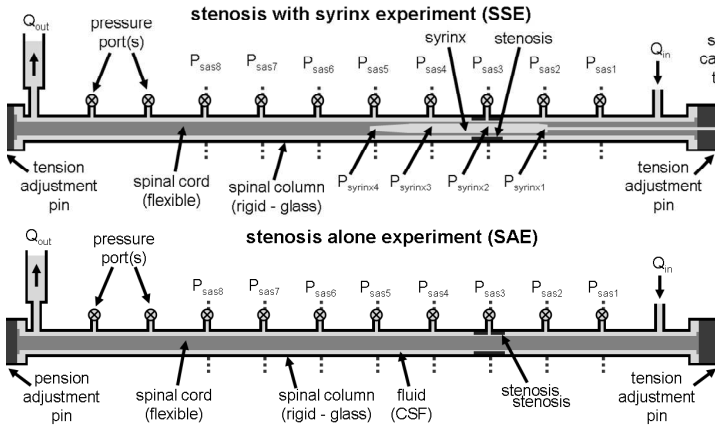
**INTRODUCTION**

Many theories have been developed towards an explanation for the pathogenesis of SM, nearly all of which place significant emphasis on the pressure environment within the spinal SAS. It has been postulated that an abnormal pressure environment caused by a CSF flow blockage in the SAS leads to syrinx pathogenesis and subsequent

pain in the patient. However, paucity and lack of detail in spinal SAS pressure measurements has left researchers with little evidence to scrutinize the existing SM theories. An in vitro study by Martin et al. documented longitudinal pressure changes in a SM model with and without a spinal stenosis [1, 2]. Pressure calculations have been documented in computational models of SM and longitudinal pressure variation in a healthy spinal canal was calculated by Loth et al. [3]. These studies have quantified the pressure environment in the SAS with SM. However, pressure is a highly complex parameter that can be examined temporally during the cardiac cycle and spatially (lateral – ventral, rostral - caudal, and transmural, across tissues). Pressure gradients are the driving force behind fluid motion in the SAS, into or out of the syrinx cavity, bulk motion of the spinal cord, and stretching or compression of the spinal cord parenchyma. Thus, we present pressure measurements on a spinal stenosis SM flow model that closely represents the in vivo case.

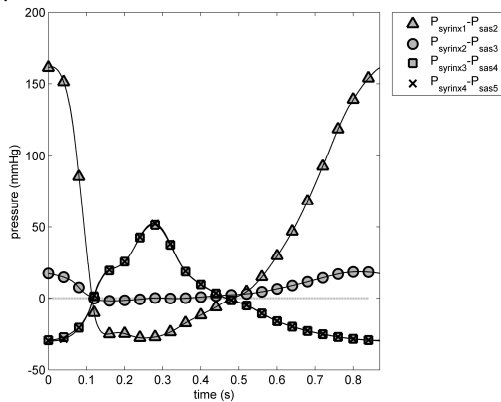
**METHODS**

Four in vitro models were constructed; a schematic diagram of two models is shown in Fig. 1. The *stenosis and syrinx experiment (SSE)* was representative of a patient who has a moderate sized syrinx that distends both in the caudal and rostral directions of a spinal stenosis (Fig. 1). The *stenosis alone experiment (SAE)* was similar to SSE, in that a spinal stenosis was present, but different in that a spinal syrinx was not present (Fig. 1). Two additional models not shown in Fig. 1 were used to examine the influence of stenosis location and removal. Stenosis location was examined in a *Chiari stenosis experiment (CSE)*, which had a stenosis located entirely rostral to the syrinx at  $P_{sas1}$ . A *stenosis removal experiment (SRE)* was conducted in which the stenosis was removed while the syrinx remained.



**Fig. 1. Model layout for each of the experiments (SSE – top), and the stenosis alone experiment (SAE – bottom).**

The spinal SAS geometry from a patient with SM was obtained using MR measurements and used for the in vitro model. Nerves were neglected and the SCs were constructed with a ~ 500 kPa Young's modulus. The fluid used was water with a ~ 1 ml/s pulsation produced by a computer controlled pump [1]. The spinal column was a glass tube with diameter, thickness, and length of 15.6, 1.2, and 48 cm, respectively having eight pressure ports in the SAS with 4 cm spacing. The SC was 48 cm with a 10 mm diameter and produced using a flexible polymer cast into an aluminum mold. An aluminum cylinder was positioned with a centering pin in the model to represent the syringe (7 mm diameter). A rubber 2cm length annular shaped stenosis was fitted into the rigid spinal canal blocking > 90% of the SAS area. Pressure transducers were located axially along the syring and SAS.



**Fig. 2. Transmural pressure differential in SSE.**

## RESULTS

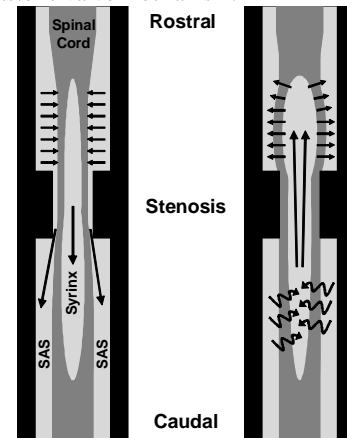
Transmural pressure (TP) is quantified by the subtraction of SAS from the syringe pressure and quantified at the four axial locations (Fig. 2). When this pressure fell below zero, the pressure in the syringe was less than in the SAS. The mean TP at the four adjacent locations was positive and negative rostral and caudal to the stenosis, respectively. The magnitude of mean TP was significantly larger rostral to the stenosis. SRE and CSE demonstrated much smaller TP fluctuation compared with SSE. The pulse pressure in CSE increased caudally while remaining relatively uniform in SRE. Thus, the presence of a stenosis and its location impacted the pressure environment greatly.

## DISCUSSION

In general, the stenosis acted to increase and dissociate the CSF pressure in the SAS to a greater degree than in the syringe. Pressure

dissociation in the SAS was greatest in SSE, and least in SRE. Maximum SC compression and distension occurred in SSE on the rostral side of the stenosis, and was far greater than with the Chiari stenosis (CSE) or with the stenosis removed (SRE). However, in CSE pulse pressures were greater throughout the entire system than when the stenosis was removed (SRE).

The axial distribution of TP was found to vary in direction and magnitude along the syring cavity and with the presence and location of a stenosis. In SSE, the stenosis caused the syringe to be compressed on one side while expanding on the other. If there is no flow blockage in the vicinity of the syringe (CSE and SRE) mean TP was small and did not have significant axial variation. SSE results indicated that the mean TP was directed into the syringe caudal to the stenosis and outward rostral to the stenosis, while the average mean TP over the entire syring remained elevated (~ 10.5 mmHg) which could result in bulk CSF movement into the syringe. The SSE acted with a diastolic valve mechanism (Fig. 1), causing greater resistance to rostral CSF flow movement than caudal CSF flow movement in the SAS. In systole, the syringe was compressed which enabled caudal CSF movement in the SAS through the stenosis. In diastole, the syringe expanded producing higher resistance to CSF flow through the SAS resulting in a diastolic valve mechanism.



**Fig. 1. Diastolic valve mechanism of the syringe and stenosis in systole (left) and diastole (right) for the SSE.**

## CONCLUSIONS

The in vitro pressure data and analyses provide valuable information about SM hydrodynamics and demonstrate the complexity of the fluid structure interaction in the coaxial elastic tube system present in SM. The syringe and stenosis interacted to function with a diastolic valve mechanism. Further research examining the influence of stenosis size and location in more sophisticated in vitro models, as well as better understanding of craniospinal tissue properties is warranted.

## REFERENCES

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