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A FLUID STRUCTRURE INTERACTION SIMULATION OF THE CEREBROSPINAL FLUID, SPINAL CORD, AND SPINAL STENOSIS PRESENT IN SYRINGOMYELIA

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ABSTRACT

Syringomyelia (SM) is a neurological disease in which a fluid-filled cystic cavity, or syrinx, forms in the spinal cord (SC) resulting in progressive loss of sensory, motor functions, and/or pain in the patient. It has been hypothesized that abnormal cerebrospinal fluid (CSF) pressure distribution and absorption in the subarachnoid space (SAS), resulting from a CSF flow blockage (stenosis), could be a key etiological factor for syrinx pathogenesis. In particular, the magnitude of the abrupt SAS pressure waves produced during coughing has been correlated with headache and pain in the patient.

To better understand the influence of coughing on the spinal SAS, four axisymmetric fluid-structure interaction (FSI) *in silico* models representative of various conditions associated with SM were constructed. Each of the models was subjected to a cough-like CSF pressure pulse.

The CSF flow stenosis was shown to attenuate and decelerate the CSF wave propagation in the SAS. The spinal SAS distensibility was also shown to have significant influence on the wave propagation. The *in silico* pressure results were found to be in agreement with a set of similar *in vitro* experiments [1].

Keywords: Syringomyelia, Chiari malformation, fluid-structure interaction, cerebrospinal fluid, Bio-fluid mechanics, subarachnoid space, syrinx, *in silico* model, computational fluid dynamics.

INTRODUCTION

As a neurological condition, SM has been recognized for over 130 years. Neurosurgeons have devised many hypotheses for the pathogenesis of SM primarily being rooted in abnormal hydrodynamics caused by CSF flow stenosis. In recent years engineers have made contributions by performing *in silico* [2, 3] and *in vitro* [1, 4, 5] simulations of SM with particular focus on biomechanical forces. While the *in vitro* models have provided detailed information on the CSF pressure and flow and spinal cord movement, they required construction of specific models for each mechanism. The *in silico* models have provided detailed information about the FSI of the spinal cord, stenosis, and CSF flow, but lacked experimental validation. Thus, the present *in silico* models were designed to be nearly identical to the *in vitro* models constructed by Martin et al. [1, 5] for validation of the computational results.

METHODS

Four in silico models were constructed in ANSYS (ANSYS Inc., Canonsburg, PA) (

Fig. 1). The model dimensions were based on an in vitro study by Martin et al. [1]. To simulate the cough excitation, a 5ms 100 mmHg pressure pulse was applied on the caudal end of the SAS (top side of

Fig. 1). The four models were the following:

1. ***stenosis and syrinx experiment model* (SSE)**: representative of a SM patient with a moderate sized syrinx and a spinal stenosis with a nearly rigid dura (modeled as glass).
2. ***stenosis and syrinx experiment with distensible spinal column* (SSED)**: similar to SSE but with flexible and thicker dura.
3. ***stenosis removed experiment* (SRE)**: similar to SSE, but with the stenosis removed.
4. ***stenosis removed experiment with distensible spinal column* (SRED)**: similar to SRE, but with a thick and flexible dura.

The axisymmetric models were constructed in ANSYS to represent the SM pathology where the CSF was contained between the spinal cord and dura mater. A cylindrical fluid-filled syrinx was located in the center of the spinal cord. A 2 cm length stenosis blocked >90% of the SAS area near the midsection of the syrinx cavity.

RESULTS

Wave propagation speed in the SAS and syrinx varied widely between each model both temporally and spatially (Fig. 2). The cough pressure excitation was administered at 0 cm (top of Fig. 2) and traveled through the spinal SAS to the rostral end (bottom of Fig. 2).

The slope of the pressure wave onset was used to calculate the SAS pressure wave speed and is compared to the *in vitro* measurements of Martin et al. [1] in Table 1.

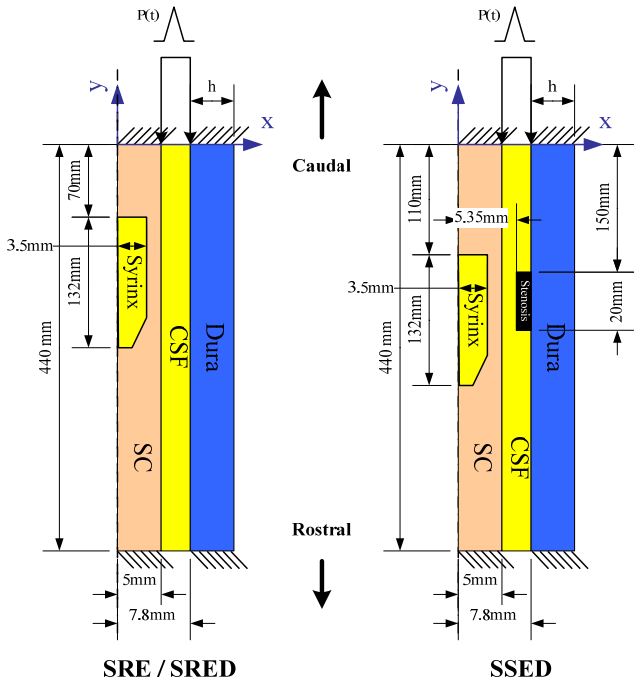

Fig. 1. Layout of the axisymmetric SRE, SRED and SSED ANSYS models. Yellow represents fluid, blue represents the spinal dura, pink represents the spinal cord, and black represents the SAS stenosis. All dimensions and properties of the models are based on Martin et al. [1].

DISCUSSION

The presence of a stenosis in the spinal SAS was found to have a major impact on the FSI present in SM. The stenosis acted to decrease pressure wave propagation speed (Table 1). But the difference in wave speed in the SAS and syrinx for each model was found to be small (Table 1).

Comparing the SAS pressure results in SSE and SSED, or SRE and SRED (Fig. 2), a flexible dura (SSED, SRED) slowed the SAS wave speed significantly. And comparing the SAS pressure results in SSE and SRE (Fig. 2), the pressure wave propagated faster in SRE than in SSE and damped quickly after the first reflection. Thus, both the presence of a stenosis and the flexibility of the dura impacted the pressure environment greatly.

CONCLUSIONS

The *in silico* analysis demonstrates the complexity of the fluid structure interaction in the spinal SAS with syringomyelia and CSF flow stenosis. The *in silico* simulations were found to have many similarities with the *in vitro* measurements performed on nearly identical models constructed by Martin et al. [1]. Further research examining the influence of more sophisticated conditions is warranted.

Table 1. Comparison of CSF wave speed results in the SAS and syrinx.

Model	SAS wave speed (m/s)		Syrinx wave speed (m/s)	
	in-silico	*in vitro*	*in-silico*	*in vitro*
SRE	367	399	440	680
SRED	25	No data	33	No data
SSE	186	155	147	118
SSED	24	24	26	25

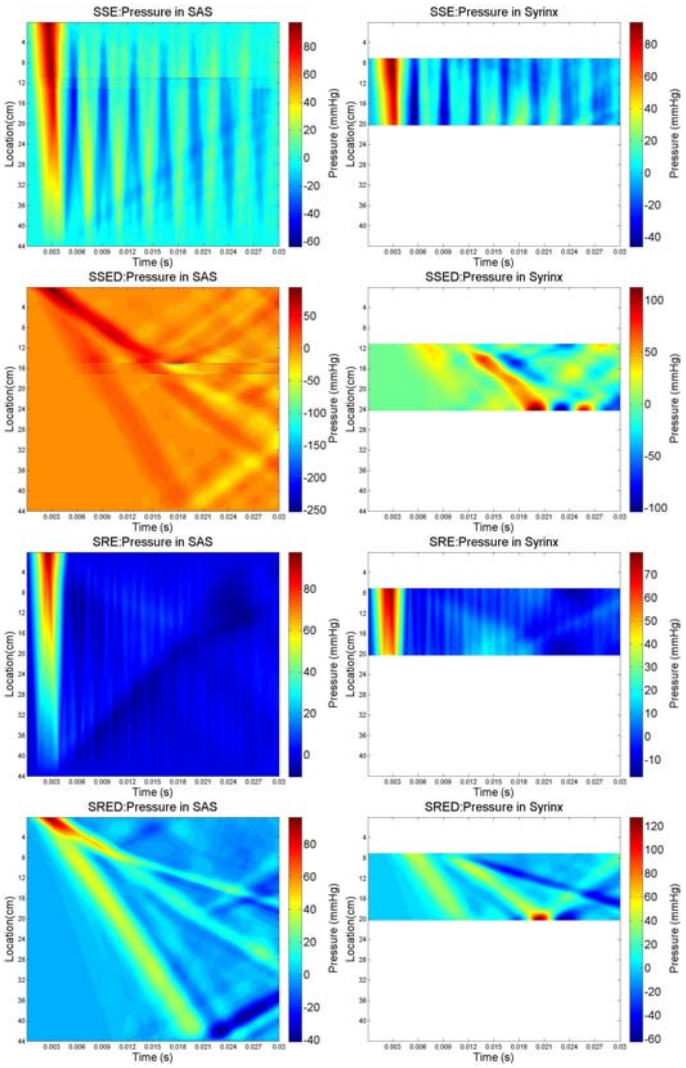

Fig. 2. Spatial vs. temporal pressure in SAS (LEFT column) and syrinx (RIGHT column).

REFERENCES

[1] Martin, B. A., and Loth, F., 2009, "The influence of coughing on cerebrospinal fluid pressure in an in vitro syringomyelia model with spinal subarachnoid space stenosis," Cerebrospinal Fluid Res, 6(1), p. 17.

[2] Bertram, C. D., Bilston, L. E., and Stoodley, M. A., 2008, "Tensile radial stress in the spinal cord related to arachnoiditis or tethering: a numerical model," Med Biol Eng Comput, 46(7), pp. 701-707.

[3] Bilston, L. E., Fletcher, D. F., and Stoodley, M. A., 2006, "Focal spinal arachnoiditis increases subarachnoid space pressure: a computational study," Clin Biomech (Bristol, Avon), 21(6), pp. 579-584.

[4] Martin, B. A., Kalata, W., Loth, F., Royston, T. J., and Oshinski, J. N., 2005, "Syringomyelia hydrodynamics: an in vitro study based on in vivo measurements," J Biomech Eng, 127(7), pp. 1110-1120.

[5] Martin, B. A., Labuda, R., Royston, T. J., Oshinski, J. N., Iskandar, B., and Loth, F., 2009, "Spinal Canal Pressure Measurements in an In Vitro Spinal Stenosis Model: Implications on Syringomyelia Theories," J Biomech Eng, In Press(June 2009).