Modern Medicine Takes **Simulation to Heart**

A fluid structure interaction simulation is performed to capture patient-specific modeling of hypertensive hemodynamics.

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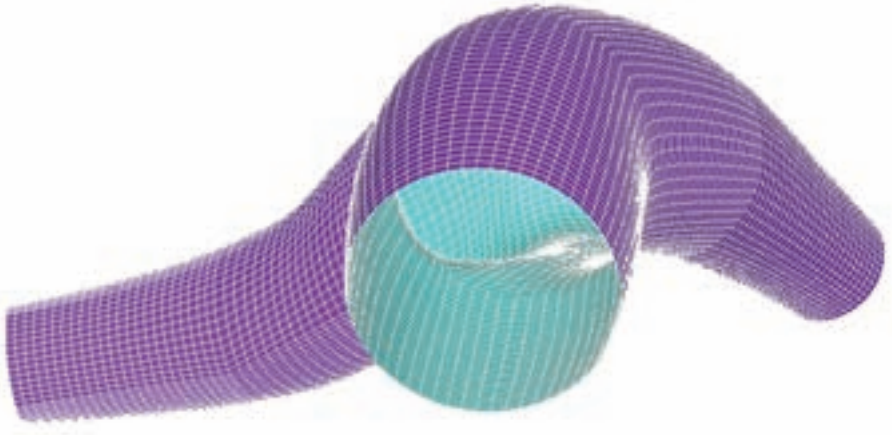
Could simulation technology more commonly associated with rocket science and race cars someday provide insight into the inner workings of the vascular system that would help doctors provide improved diagnosis treatment in clinical situations? Researchers at the University of Colorado Health Sciences Center (UCHSC) have taken the first steps toward that end, and the ANSYS fluid structure interaction (FSI) solution is proving to be a key enabling technology.

The pulmonary arteries are the blood vessels that carry oxygen-poor blood from the right ventricle of the heart to the small arteries in the lungs. For a healthy individual, the normal average pressure in the pulmonary artery is about 14 mm Hg. For individuals with pulmonary arterial hypertension (PAH), the average pressure is usually greater than 25 mm Hg. This increases the load on the right side of the heart and can lead to eventual heart failure and death.

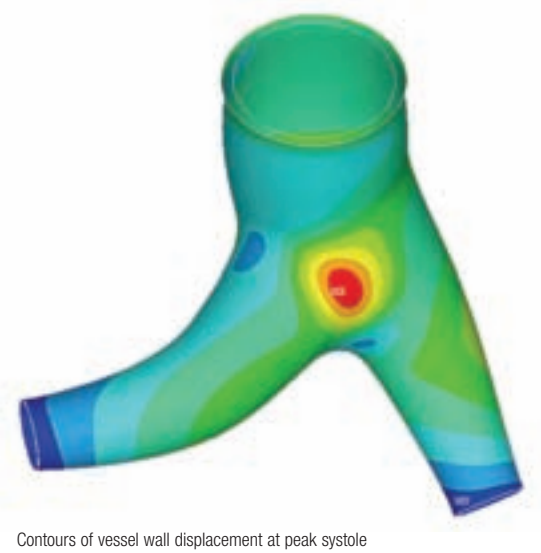
Diagnosis and evaluation of PAH typically is accomplished with a combination of cardiac catheterization (in which a plastic tube is passed through the iliac vein in the leg and weaved up the body, through the right side of the heart, and out into the main pulmonary artery) and imaging techniques such as angiography and magnetic resonance imaging (MRI). While these methods are effective in the diagnosis of vascular pathologies, they cannot currently provide enough detail or be performed with sufficient frequency to elucidate the causes of disease progression and are hard pressed to predict the outcome of clinical interventions. To date, clinicians have mainly characterized PAH by evaluating pulmonary vascular resistance (PVR), defined as the mean pressure drop divided by the mean flow rate. In considering only mean conditions, the effects of vascular stiffness are ignored; in patients with PAH, however, these effects can amount to 40 percent of the total right heart afterload. Over time, the vasculature can thicken in response to the increased pressure. Such proximal thickening and stiffening is believed to change distal flow and further increase pressures; thus, it may be part of a feedback loop by which PAH worsens.



Mesh generated using ANSYS ICEM CFD Hexa; the CFD domain is bounded by the blue cells and the shell mesh, used for the structural calculation, is shown in lavender.

At UCHSC, researchers are investigating the impact of proximal artery stiffness by using ANSYS software to simulate the transient fluid structure interaction of the blood flow and vascular walls of the pulmonary artery. By using numerical simulation, researchers can gain a better fundamental understanding of the physics involved in PAH and insight into the effects of vascular stiffness on proximal, and, perhaps more importantly, distal hemodynamics. Eventually, the regular clinical use of patient-specific

Contours of pressure on the vessel walls at peak systole



Contours of vessel wall displacement at peak systole

simulation, in which the vascular geometry is extracted from medical imaging, could provide better insight into the progression of PAH and improve predictions of the outcome of surgical intervention.

For the ANSYS FSI simulations reported here, geometry acquisition begins with bi-plane angiography of the proximal pulmonary tree performed during cardiac catheterization of an 18 month-old male patient. This provides data describing the vessel centerline and diameter. A CAD system is used to turn this skeletal data into a smooth representation of the vessel geometry. The geometry is imported into ANSYS ICEM CFD software and the Hexa meshing module is used to construct a high-quality hexahedral volume mesh. The resulting mesh uses an O-grid inflation layer from all walls so that the mesh is nearly orthogonal with excellent control over near-wall spacing. This mesh is used for the CFD component of the FSI simulation, solved using ANSYS CFX software. The quad surface elements from that same mesh are imported into ANSYS as a shell element representation of the vessel. This type of representation is a significant advantage, since it allows investigations in which the vessel wall thickness is varied without the need for geometry modifications or re-meshing. A script is used to apply variable shell thickness on a node-by-node basis to the vessel mesh.

For these studies the Arruda–Boyce hyperelastic material model is used. The model parameters were suggested by biomechanical studies of the stress–strain properties of normotensive and hypertensive pulmonary arteries from a rat model and solid-only simulations of human pulmonary arteries. Residual stress is not considered here due to the difficulty of incorporating such effects in clinical models in which direct measurements within the artery cannot be obtained. The solid model was constrained on the inflow/outflow boundaries. The remaining nodes were allowed to deform in response to applied forces.

Blood is modeled as an incompressible Newtonian fluid with constant dynamic viscosity and the flow is assumed to be laminar. Using the CFX Expression Language (CEL), it was straightforward to implement a time-varying mass flow boundary condition at the fluid inlet with a half-sinusoid profile. Exit boundary conditions were modeled using CEL and a resistive relationship in which the outlet pressure for each branch was determined by multiplying the local instantaneous flow rate by a resistance factor. [1,2]

The early results of this pilot study have confirmed the anticipated behavior of the system. Upcoming studies with improved clinical and imaging data will allow validation and refinement of the simulation methodology. Eventually, the clinical use of non-invasive, patient-specific simulation may provide better understanding of the progression of PAH and improved predictions of the potential outcomes of available treatments. ■

References

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[2] Olufsen, M.S.: Structured Tree Outflow Conditions for Blood Flow in Larger Systemic Arteries. Am. J. Physiol. (AJP) 276(1): H257-H268, 1999.