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Shock-induced collapse of a bubble inside a deformable vessel

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ABSTRACT

Shockwave lithotripsy repeatedly focuses shockwaves on kidney stones to induce their fracture, partially through cavitation erosion. A typical side effect of the procedure is hemorrhage, which is potentially the result of the growth and collapse of bubbles inside blood vessels. To identify the mechanisms by which shock-induced collapse could lead to the onset of injury, we study an idealized problem involving a preexisting bubble in a deformable vessel. We utilize a high-order accurate, shock- and interface-capturing, finite-volume scheme and simulate the three-dimensional shock-induced collapse of an air bubble immersed in a cylindrical water column which is embedded in a gelatin/water mixture. The mixture is a soft tissue simulant, 10% gelatin by weight, and is modeled by the stiffened gas equation of state. The bubble dynamics of this model configuration are characterized by the collapse of the bubble and its subsequent jetting in the direction of the propagation of the shockwave. The vessel wall, which is defined by the material interface between the water and gelatin/water mixture, is invaginated by the collapse and distended by the impact of the jet. The present results show that the highest measured pressures and deformations occur when the volumetric confinement of the bubble is strongest, the bubble is nearest the vessel wall and/or the angle of incidence of the shockwave reduces the distance between the jet tip and the nearest vessel surface. For a particular case considered, the 40 MPa shockwave utilized in this study to collapse the bubble generated a vessel wall pressure of almost 450 MPa and produced both an invagination and distention of nearly 50% of the initial vessel radius on a $\mathcal{O}(10)$ ns timescale. These results are indicative of the significant potential of shock-induced collapse to contribute to the injury of blood vessels in shockwave lithotripsy.

1. Introduction

Shockwave lithotripsy (SWL) is a medical procedure to eliminate kidney stones. In typical clinical applications, approximately 2000 pressure pulses, with peak positive pressures between 30 and 110 MPa and peak negative pressures between −5 and −15 MPa, are generated by the lithotripter [1]. These pulses, or shockwaves, are focused at the location of the stones in order to pulverize them and enable their expulsion through the urinary tract. Both the effectiveness and safety of the treatment depend on, among other factors, the bubble dynamics excited by the passage of the shockwaves. Cavitating bubbles are documented to occur during treatment in both urine and surrounding tissue [2]. In the vicinity of kidney stones, cavitation erosion is thought to be an important mechanism of stone comminution and is characterized by the formation of liquid jets and the emission of shockwaves, both with the potential to inflict significant damage to nearby stones [3–5]. Unfortunately, cavitation in SWL is also implicated in the onset of renal trauma, specifically hemorrhage, which is instigated by the rupture of small blood vessels, such as capillaries, arterioles and venules, which range from 5 to 100 μm in diameter [1,6,7]. Recent experiments in the vasculature of *ex vivo* rat mesentery suggest that the growth and collapse of bubbles, along with liquid jet impact, may deform small blood vessels sufficiently as to cause them to rupture [8–10]. In order to improve the effectiveness and safety of SWL, it is therefore imperative to fully understand the mechanisms through which cavitation may contribute to both stone removal and vascular injury.

In both experiments and simulations, characterization of bubble–vessel dynamics has been difficult. Experimentally, the challenges of performing measurements and observations in tissue and blood vessels are exacerbated by small spatial and fast temporal scales. As a result, experimental work has primarily been carried out *in vitro*, typically utilizing gels and vessel phantoms to model tissue and blood vessels, respectively [7,11–13]. The first observations of *ex vivo* bubble dynamics in blood vessels and under clinical conditions were reported by Caskey et al. [14], in the case of ultrasonic cavitation of microbubbles in rat cecum. Their results were cast in the context of gene therapy and localized drug delivery and did not specifically address the potential mechanisms

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of vascular injury. These mechanisms were the focus of subsequent work by Chen et al. [8–10], who performed analogous experiments in *ex vivo* rat mesentery. Utilizing high-speed microphotography, they measured the transient bubble–vessel interactions. They concluded that in the context of SWL, three mechanisms can potentially result in the mechanical failure of blood vessels due to cavitation. These include vessel distention due to bubble growth, vessel invagination due to bubble collapse, and finally, puncturing of the vessel wall due to bubble jetting.

Identifying these same mechanisms in numerical simulations has its own set of challenges. In particular, the understanding of the rheology of tissue is incomplete. Recent efforts to study vessel rupture mechanisms due to cavitating bubbles have circumvented this issue, modeling the wall of the blood vessel in great detail and omitting the tissue in which it is embedded. These simulations, performed by Ye and Bull [15] and Miao et al. [16], were carried out in an axisymmetric geometry, with both the bubble and vessel wall immersed in an incompressible fluid. Their efforts were successful in demonstrating that ultrasonically excited microbubbles could generate sufficiently high vessel wall stresses during distention as to induce rupture. Unfortunately, due to the axisymmetry and incompressibility assumptions, the wall stresses that would arise from bubble collapse and liquid jet impact could not be addressed.

The damage potential of bubble jetting was considered in the axisymmetric compressible flow simulations of Freund et al. [17] and Kobayashi et al. [18], but in the context of the shock-induced collapse of a bubble near a soft tissue simulant. Freund et al. set the properties of the simulant to those of water but varied its shear viscosity in order to study how effectively the various measurements of the viscous coefficient of tissue could suppress the penetration of the liquid jet. Coefficients between 0.01 and 10 Pa·s were considered. The results of the study showed that the penetration depth of the jet into the boundary could greatly be suppressed by the higher measurements of the tissue viscosity. The resulting viscous shear stresses, however, were estimated to be sufficiently large on the surface of the tissue as to potentially damage cells. Freund et al. postulated that this mechanism could be responsible for the observed damage to the endothelium, a thin layer of cells that lines the inner surface of a vessel wall. Kobayashi et al., on the other hand, presumed that such insult to tissue could occur due to the jetting of the bubble. They characterized the injury mechanism by correlating the deformation of the tissue boundary with the impulse that is generated by the water-hammer shockwave emitted during the formation of the liquid jet. Several soft tissues and soft tissue simulants were considered, including fat, liver and a gelatin/water mixture, and were modeled by the stiffened gas equation of state by fitting the density and acoustic impedance.

In this study, we build upon the computational efforts of Freund et al. and Kobayashi et al. and analyze the three-dimensional (3D) shock-induced collapse of an air bubble inside a cylindrical water column that is embedded in a 10% by weight gelatin/water mixture. This mixture, from here on, will simply be referred to as 10% gelatin. The problem setup is designed to emulate the shock-induced collapse of a preexisting gas bubble located inside a blood vessel surrounded by tissue. At this time, the viscoelastic properties of tissue are neglected and the vessel wall is treated as a material interface between the water and 10% gelatin. To our knowledge, the fully asymmetric and compressible bubble–vessel dynamics have never previously been reported in literature, so that this represents a first effort at their analysis.

Our study is broken up as follows. In Section 2, we discuss and justify the physical model utilized in the simulation of the bubble–vessel dynamics. In Section 3, we present the numerical scheme and its adaptation to the equations of motion of the physical model. The results of the simulations are analyzed and discussed in Section 4. Parametric variations in vessel confinement, bubble proximity and shockwave angle are presented and the pressures and deformations to which the vessel wall is subjected are quantified and cast in the context of the potential for vascular injury to occur in SWL. Finally, in Section 5, we summarize the results and briefly discuss directions of future work.

2. Physical model

2.1. Problem description

Cavitation inception in SWL is at least partly due to the excitation of preexisting gas nuclei by the repeated passage of the pressure pulse that is generated by the lithotripter. Though reliable measurements of the initial population of nucleation sites in vasculature is currently unavailable, bubbles are thought to exist in blood vessels under normal physiological conditions and *in vitro* experiments suggest that their population will grow with ongoing SWL treatment [5,19]. The pressure wave emitted by the lithotripter is composed of a compressive and a tensile component. During compression, which is characterized by a shockwave and a transition into tension, preexisting gas bubbles are rapidly collapsed. This shock-induced collapse is immediately followed by a period of cavitation, which is induced by the negative pressures associated with the tensile component of the lithotripter waveform. Cavitating bubbles grow to a large size and subsequently collapse. This is often referred to as a Rayleigh collapse and is driven by the static pressure difference between the bubble contents and the surrounding fluid [20]. In a typical clinical lithotripsy treatment, the pressure pluses are delivered with a frequency between 0.5 and 3 Hz. Then, as the treatment progresses, the above described bubble dynamics become cyclical and eventually more prominent and complex, with the number of nucleation sites increasing and the interaction between neighboring bubbles becoming non-negligible.

This extremely rich and varied bubble behavior poses significant modeling challenges. Specifically, the range of spatial and temporal scales that is cumulatively spanned by shock-induced collapse, Rayleigh collapse and the interaction between adjacent bubbles makes it computationally prohibitive to attempt to comprehensively simulate the bubble dynamics inside blood vessels during the lifetime of the SWL treatment. By focusing our efforts on the analysis of the shock-induced collapse of a preexisting gas bubble, inside a small blood vessel and exposed to only one lithotripter pulse, the modeling requirements imposed by the spatial and temporal scales are significantly alleviated. The spatial scales will approximately be bound by the diameter of the small blood vessels, 5 to 100 μm, while the temporal scales will be on the order of the collapse time of comparably sized bubbles, $\mathcal{O}(10)$ ns [21]. Then, the scaling arguments put forth by previous numerical studies of this nature suggest that the modeling of diffusive effects, mass transfer and surface tension, can be neglected [17,22]. Though these arguments do not strictly apply to a collapse that is confined by the presence of a blood vessel wall and the tissue that lies beyond it, it is not expected that the confinement offered by either of these two structures will be sufficient to significantly alter the scaling analysis. This assertion is partially supported by the work of Freund [21] who generalized the Rayleigh–Plesset equation of spherical bubble dynamics to gauge the potential of the blood vessel wall and the surrounding tissue to suppress bubble growth in SWL. Freund's results suggest that across a large range of experimentally measured values of the wall and tissue elasticities, as well as tissue viscosities, bubble expansion cannot be meaningfully inhibited. Though the higher reported values of tissue viscosity were shown to be more effective, we do not model viscous effects at this time. Under these circumstances, we omit modeling diffusive effects, mass