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SHOCK-INDUCED BUBBLE COLLAPSE IN A VESSEL: IMPLICATIONS FOR VASCULAR INJURY IN SHOCKWAVE LITHOTRIPSY

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In shockwave lithotripsy (SWL), shockwaves are repeatedly focused at the location of kidney stones in order to break them. This process eventually leads to the onset of cavitation in tissue, which inevitably leads to hemorrhage. Approximately a thousand shockwaves, however, must be administered prior to the onset of extensive cavitation [1] and it is theorized that blood pooling due to the initial rupture of a blood vessel is the necessary precursor that provides the required cavitation nuclei [2]. We hypothesize that the shock-induced collapse (SIC) of preexisting gas bubbles is a viable contributing mechanism to the initial vascular injury. We study the mechanism numerically with an idealized problem consisting of the three-dimensional SIC of a single air bubble immersed in a cylindrical water column that is embedded in a gelatin/water mixture. The mixture serves as a tissue simulant and can be treated as a fluid due to the fast time scales, $\mathcal{O}(10)$ ns, and small spatial scales, $\mathcal{O}(1)$ μm, associated with the collapse. We thus model the problem with the compressible multicomponent flow equations and simulate it with a recently developed Godunov-type shock- and interface-capturing numerical method [3]. The method is high-order accurate, discretely conservative and non-oscillatory. A fifth-order, finite-volume, weighted essentially non-oscillatory scheme is utilized for the spatial reconstruction and a Harten-Lax-van Leer contact approximate Riemann solver is employed to upwind the fluxes. A third-order total variation diminishing Runge-Kutta algorithm marches the solution in time. We evaluate the potential for vascular injury due to the SIC of preexisting bubbles for a range of pressures, bubble and vessel diameters, shockwave angles, as well as tissue properties relevant to clinical SWL. We assess the potential for injury by comparing the finite deformations in tissue, which are obtained by tracking particles, to ultimate strains in uniaxial compression/tension experiments. We conclude that the SIC of preexisting bubbles in a blood vessel may contribute to its rupture and discuss the smallest bubble size necessary for the onset of injury.

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