

Growth of liquid-gas interfacial perturbations driven by acoustic waves

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Diagnostic ultrasound has been shown to cause lung hemorrhage in a variety of mammals, though the underlying damage mechanisms are still unclear. Motivated by this problem, we use numerical simulations to investigate the interaction of an ultrasound wave with the alveolar tissue-air interface. A planar, single-cycle, trapezoidal waveform propagates in tissue (modeled as water) and impinges upon an alveolus of the lung (modeled as air); to represent the alveolar surface roughness, the interface consists of a small-amplitude single-mode perturbation. Because of the sharp density gradient at the interface, we hypothesize that ultrasound waves, despite their relatively low amplitude, deposit sufficient baroclinic vorticity to drive perturbation growth. Our simulations show that the perturbation amplitude grows to sizes many times larger than the original value, well after the wave has passed. We demonstrate that conventional (linear) acoustics cannot account for such deformations; instead, the perturbation growth is driven by nonlinear effects: the baroclinic vorticity deposited along the interface, due to the misalignment of the pressure gradient (across the wave) and the density gradient (across the perturbed gas-liquid interface). Based on dimensional analysis and scaling, we observe that the perturbation amplitude and length of the interface scale with the circulation density and grow according to power laws in time. If the time interval between the pressure increase and decrease is sufficient, both deposit vorticity of the same sign, thus enhancing the perturbation growth; conversely, if the interval is too short, the vorticity deposited by the pressure increase is canceled by the decrease. A further consequence is that one may be able to control the growth of such perturbed interfaces by modulating the incoming waveform.

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I. INTRODUCTION

Diagnostic ultrasound (DUS) is one of the safest forms of medical imaging and has become ubiquitous in clinical practice. DUS-induced lung hemorrhage is the only known bioeffect of noncontrast, pulsed ultrasound (US), as bleeding has been shown to occur in mammals including mice, rats, rabbits, pigs, and monkeys [1–4]. Furthermore, this problem has been shown to occur for a wide range of frequencies from 1.5 to 12 MHz, for mechanical indices well below the accepted safe limit for diagnostic ultrasound, $MI = 1.9$ [5]. Although this problem does not appear to be of medical safety concern for humans under typical conditions, there is a need to better understand the physical mechanisms of DUS-induced lung hemorrhage, which cannot currently be explained by well-established US bioeffects mechanisms. Typically, US bioeffects are generally classified as thermal or nonthermal, with the bulk of nonthermal bioeffects being a result of acoustically driven inertial cavitation. Except for one study reporting cavitation activity [6], the bulk

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of the research suggests that inertial cavitation is not the cause of DUS-induced lung hemorrhage: Refs. [7,8] found that bleeding is not worsened by the use of ultrasound contrast agents and Ref. [9] observed that the severity of the hemorrhage increases when the hydrostatic pressure is raised. Beyond lung hemorrhage, a number of studies have explored nonlinear mechanisms as a potential cause for bioeffects. Reference [10] developed numerical models to study the effects of acoustic nonlinearity on wave propagation and heating in soft tissue. Reference [11] numerically solved a Khokhlov-Zabolotskaya-Kuznetsov (KZK)-type equation simulating high-intensity focused ultrasound (HIFU) fields in a tissue phantom with the purpose of studying the impacts of nonlinear propagation, cavitation, and boiling on lesion formation. Based on potential flow simulations of an inviscid free surface subjected to a Gaussian velocity potential, Ref. [12] suggested another damage mechanism for DUS-induced lung hemorrhage, namely, that under the right circumstances droplets capable of puncturing the air-filled sacs within the lung may be ejected from the surface by the focused US. Reference [13] experimentally demonstrated atomization of water and soft tissue at air interfaces exposed to 2-MHz HIFU, though at pressure amplitudes higher than diagnostic. Despite these efforts, the precise damage mechanism underlying DUS-induced lung hemorrhage is still unknown.

In parallel, the dynamics of accelerated interfaces separating fluids of different densities have been the subject of intensive studies in fluid mechanics. When exposed to accelerations whose sign is opposite that of the density gradient, small interfacial perturbations grow exponentially as a manifestation of the Rayleigh-Taylor (RT) instability [14]. Bubbles of light fluids “rise” into the heavy fluid while spikes of heavy fluid “fall” into the light fluid. Although the original analysis pertained to perturbation growth at early times under constant acceleration, extensions to nonlinear growth and time-varying accelerations have been performed. In the limit of instantaneous acceleration (e.g., as produced by a shock wave), small perturbations initially grow linearly in time, as predicted by Richtmyer-Meshkov (RM) analysis [15,16], regardless of the sign of the density gradient. To characterize the growth at later times, Ref. [17] developed a potential flow model for both RT and RM flows with Atwood number $A = \frac{\rho_{\text{heavy}} - \rho_{\text{light}}}{\rho_{\text{heavy}} + \rho_{\text{light}}} \rightarrow 1$, which describes bubble growth in both linear and nonlinear regimes; Ref. [18] extended this model to time-dependent Atwood numbers and acceleration profiles. In both RT and RM flows, perturbation growth can be explained by vorticity, baroclinically, i.e., due to the misalignment of the density and pressure gradients

$$\left. \frac{d\omega}{dt} \right|_{\text{baroclinic}} = \frac{\nabla \rho \times \nabla p}{\rho^2}, \quad (1)$$

where ω is the vorticity, ρ the density, and p the pressure. The majority of RT research has examined interfacial perturbation growth under constant acceleration fields; RM research is primarily concerned with shock-accelerated interfaces, where the postshock pressure is maintained constant [19]. Reference [20] bounced a fluid tank to experimentally study the Richtmyer-Meshkov instability between two liquids with an initial interface perturbation created by standing waves. A model of the interface evolution using a row of vortices was developed and shows similarly shaped growth curves to those observed in the experiments, though late-time growth rates were underestimated. There has been limited study of interfaces undergoing transient acceleration. Reference [21] experimentally studied the RT instability at diffuse, perturbed, gas-gas interfaces with Atwood numbers from $A = 0.49$ to 0.94 subjected to a rarefaction. It was found that variable acceleration had little effect on early interface perturbation growth if accounted for, however the initially diffuse interface decreased the growth rate of the instability. Reference [22] showed that multiple subsequent shocks and constant accelerations could create and subsequently diminish instabilities in stratified fluid layers. Furthermore, this work concluded that, under certain conditions, time-varying interface acceleration profiles could be treated as a series of shocks and periods of constant accelerations. Reference [23] simulated shock passage through multiple gas layers with sinusoidally perturbed interfaces to show that subsequent reshock by reflected waves causes the flow to evolve into a complex nested mushroom morphology; later Ref. [24] developed a model for hydrodynamic instabilities driven by time-dependent accelerations, which agreed well with full simulations. Reference [25] demonstrated that subsequent interactions

between reflected and transmitted shocks and rarefactions with interfaces in layered media could be used to decrease and possibly control the long-term growth of shock-accelerated interfaces, including shock-bubble interactions. Much of the past research in both RT and RM flows has focused largely on gas-gas interfaces. Reference [26] experimentally shocked helium and R22-filled bubbles in air and showed that transmitted waves may overtake one another and merge downstream as a result of nonlinear gas dynamics. Numerical simulations, in conjunction with nonlinear theory, have shown that baroclinic vorticity is generated by the wave-interface interaction and dominates the late-time dynamics of the system [27,28].

In this article, we submit that an ultrasound wave propagating in tissue and impinging upon the lung may give rise to perturbation growth along the interface, much like that observed in RT and RM flows. Despite being smooth by contrast to shocks, ultrasound waves in tissue have pressure amplitudes on the order of megapascals and wavelengths on the order of hundreds of microns to millimeters; although the strength of the waves is relatively small given the large density and sound speed, the pressure gradients are not negligible. Furthermore, the density jumps by several orders of magnitude over distances less than $1\text{ }\mu\text{m}$, across the tissue-air interface. These observations motivate our hypothesis, namely, that baroclinic vorticity generated by the misalignment of the pressure gradient across the ultrasound wave and the density gradient across the soft tissue-lung interface causes interfacial perturbations to grow, even after the passage of the wave. Ultimately, if the growth is sufficient over the relevant time scales, capillary rupture may occur. Such a phenomenon cannot be described by linear acoustics. Furthermore, the fluid mechanics of this process are expected to be different from classical RT and RM theory: By contrast to conventional RT analysis, the acceleration imparted by the pressure wave is time varying; as opposed to the classical RM process, the wave deposits vorticity over a finite-time duration. Thus, the transient nature of the problem (e.g., interface deformation during wave interaction) is expected to be important. Our objective is to describe the growth of perturbations along water-air interfaces subjected to time-varying pressure waves using numerical simulations, under conditions relevant to diagnostic ultrasound. To probe the basic mechanics, the soft tissue-lung interface is modeled as a water-air interface and the ultrasound waveform is idealized to a trapezoidal wave. The article is organized as follows. We first describe the problem under consideration and our methods. We then investigate the perturbation growth and vorticity dynamics of our baseline case. As we seek to understand the late-time growth, we then examine how the wave properties (amplitude and length) affect the dynamics. Finally, we summarize the main conclusions and suggest the next steps to be taken.

II. FLUID MECHANICS MODELING OF ULTRASOUND-LUNG INTERACTION

Consider a DUS pulse traveling into the lung. Since past studies have observed lung hemorrhage with frequencies ranging from 1.5 to 12 MHz and pressure amplitudes from 1.0 to 12.3 MPa [1,29–31], we consider pulses in the megahertz and megapascal ranges. The wave traverses several layers of soft tissue and fluid making up the thoracic wall ($\sim 2\text{ cm}$ thick) and pulmonary pleura ($\sim 1\text{ mm}$ thick), whose acoustic properties (density and sound speed) are close to those of water [32]. The size of the focal region is on the order of the ultrasonic wavelength λ , approximately 1 mm for a 1.5-MHz wave in tissue. After passing through the pleurae, the wave encounters a network of openly connected, air-filled sacculi with distinctly irregular surfaces: the alveoli, whose typical mean diameter in adult humans is $\ell \approx 200\text{ }\mu\text{m}$ [33]. The lung is a complex organ, as exemplified by the range of length scales and physical properties [34,35]: multiphase, viscoelastic, surface tension, and high gas volume fraction. However, dimensional arguments suggest that at sufficiently early times inertial effects dominate in the interaction of an ultrasound wave with the lung, in which case viscous, surface tension, and elastic effects are negligible. By the end of the simulations considered here, the viscous boundary layer thickness is approximately $\sqrt{\nu_{\text{water}} t_{\text{final}}} \approx 20\text{ }\mu\text{m}$, far less than both a typical alveolar diameter and the $400\text{-}\mu\text{m}$ interface amplitudes achieved at that time in our baseline case. The Weber number corresponding to the alveolar surface tension $\sigma = 9\text{ mN/m}$ [36]

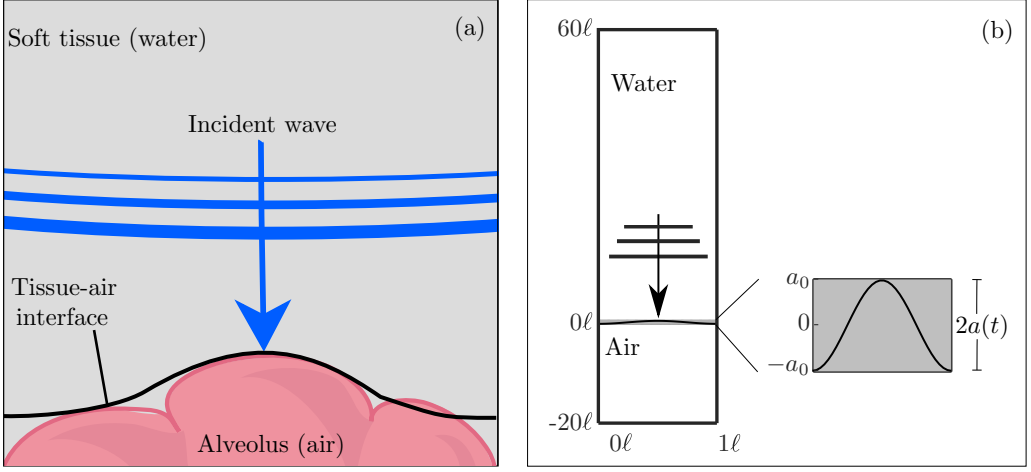


FIG. 1. (a) Schematic description of the physical problem of interest (ultrasound pulse in soft tissue impinging upon the first alveolus it encounters). (b) Computational setup of the model problem (acoustic wave in water impinging upon a sinusoidally perturbed air interface of initial amplitude a_0).

and a pressure amplitude of $p_a = 1$ MPa is $We = p_a \ell / \sigma = O(10^4) \gg 1$. For an elastic modulus $K = 5$ kPa [37], the acoustic Cauchy number is $Ca = p_a / K = O(10^2) \gg 1$.

Our interest lies in the interaction between an incident ultrasound cycle and the first alveolar tissue-air interface it encounters, as illustrated in Fig. 1. Given the complexity of the full problem, we simplify it to a tractable one on the basis of the above observations. Since viscous, surface tension, and elastic effects are negligible, the dominant mechanics are the wave propagation, its interaction with the soft tissue-lung interface, and subsequent interfacial deformations. Thus, we model the soft tissue (i.e., thoracic wall and pleura), as water and the lung as air; both substances are compressible, with appropriate density and sound speed. To simplify the representation of the alveolar surface roughness, the interface is initially represented by a single-mode sinusoidal perturbation of amplitude a_0 ,

$$y_{\text{interface}}(x, t = 0) = a_0 \sin \left(\frac{2\pi x}{\ell} - \frac{\pi}{2} \right), \quad (2)$$

where a_0 is taken to be 0.03ℓ in this work. We define the time-dependent interfacial perturbation amplitude $a(t)$ as half the peak-to-trough distance in the y direction. On the scale of an alveolus, the incoming wave is planar. More complex, corrugated interfaces can be described by combining such sinusoidal perturbations of varying amplitudes and wavelengths. Despite the three-dimensional geometry of the real problem, the essential physics are well approximated by this two-dimensional description.

Although our motivation is rooted in DUS-induced lung hemorrhage, a typical DUS pulse bears challenges when investigating the fundamental mechanics of acoustically driven perturbed liquid-air interfaces. For instance, the waveforms are often noisy, continuously vary, and come in as pulses consisting of several cycles of variable amplitude. For simplicity, we construct an idealized waveform comprising the key elements of DUS pulses expected to drive the mechanics, as illustrated in Fig. 2. By contrast to shock waves, which instantaneously and impulsively accelerate the interface and maintain a state of high pressure after their passage, an ultrasound wave continuously interacts with the interface over the finite duration of its passage; the pressure returns to its initially unperturbed ambient value thereafter. Direct application of Richtmyer-Meshkov analysis to relate the continuously varying pressure profile to baroclinic vorticity deposition is thus not straightforward. For this reason, we consider a single positive trapezoidal wave of amplitude and length relevant to DUS, consisting of a linear pressure increase followed by a constant elevated pressure, itself followed by a linear pressure

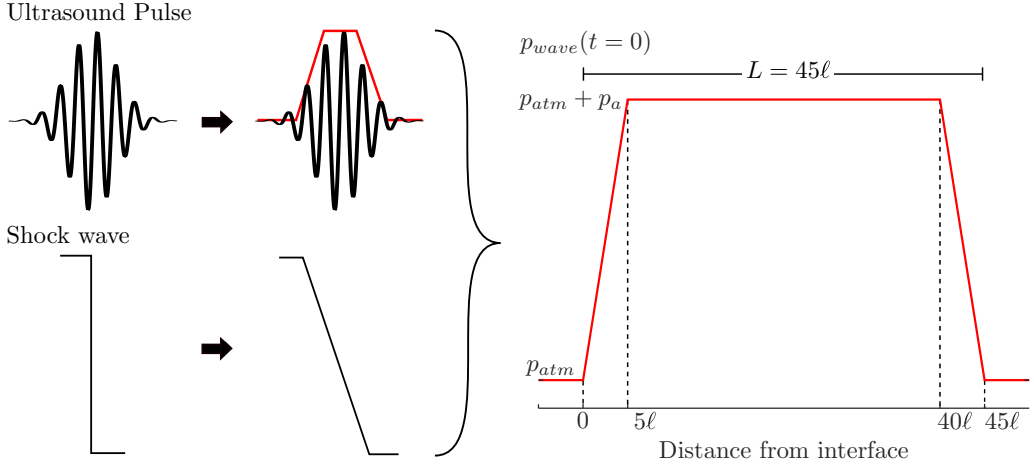


FIG. 2. Ideological progression from ultrasound pulse and shock to our baseline trapezoidal wave, which can be analyzed with Richtmyer-Meshkov-inspired analysis.

decrease back to ambient. Noting that its intensity is approximately trapezoidal, the complex multi-cycle DUS pulse is simplified to a waveform to which Richtmyer-Meshkov-inspired analysis can be applied: Though finite duration, the pressure gradients are constant and the time intervals over which vorticity is deposited (pressure increase and decrease) are clearly defined. Despite this specific choice for the waveform, we explain in Sec. IV how aspects of these results are generalizable to arbitrary waveforms with positive and negative pressure contributions. In this study, the amplitude and length of the wave are chosen to be relevant to DUS. The pressure increases from atmospheric by amplitude $p_a = 5.0\text{--}12.5$ MPa over a distance $\Delta L_a = 1$ mm, for an alveolar diameter $\ell = 200$ μm . The wave is symmetric in time such that the pressure decreases over the same ΔL_a . To keep the pulse duration consistent with DUS, we choose as a baseline a total pulse length of $L = 45\ell$ corresponding to 9 mm in soft tissue, or a $5.5\text{-}\mu\text{s}$ pulse duration, in the range relevant to previous research [1,38]. Thus, the length of the constant elevated pressure is 35ℓ . Hence, the initial pressure waveform is prescribed as

$$p(y_f, t = 0) = p_{\text{atm}} + p_a \begin{cases} 0, & y_f \leq 0 \text{ or } y_f \geq 45\ell \\ \frac{y_f}{5\ell}, & 0 \leq y_f \leq 5\ell \\ 1, & 5\ell \leq y_f \leq 40\ell \\ 1 - \frac{y_f - 40\ell}{5\ell}, & 40\ell \leq y_f \leq 45\ell \end{cases} \quad (3)$$

where $y_f = y - a_0 + 0.3\ell$ is the y location, relative to the initial location of the wave leading end. At these amplitudes and frequencies, linear acoustics describes ultrasound propagation through homogeneous tissue such that the initial x - and y -velocity components are set to $u = 0$ and $v = -\Delta p_a / \rho c$, respectively, and the initial density is $\rho_{\text{water}} + \Delta p_a / c^2$ [39], where $\Delta p_a = p(y, 0) - p_{\text{atm}}$ is the acoustic perturbation pressure.

Once the ultrasound reaches the interface, the pressure differential (due to the geometrical perturbation) over a short distance applies a torque on fluid particles along the sharp perturbed interface, thus generating rotation (or baroclinic vorticity). Since this effect is nonlinear and thus cannot be described by linear acoustics, we solve the Euler equations, written here in two dimensions (x and y)

$$\frac{\partial \rho}{\partial t} + \frac{\partial}{\partial x}(\rho u) + \frac{\partial}{\partial y}(\rho v) = 0, \quad (4a)$$

$$\frac{\partial}{\partial t}(\rho u) + \frac{\partial}{\partial x}(\rho u^2 + p) + \frac{\partial}{\partial y}(\rho uv) = 0, \quad (4b)$$

$$\frac{\partial}{\partial t}(\rho v) + \frac{\partial}{\partial x}(\rho uv) + \frac{\partial}{\partial y}(\rho v^2 + p) = 0, \quad (4c)$$

$$\frac{\partial E}{\partial t} + \frac{\partial}{\partial x}(u[E + p]) + \frac{\partial}{\partial y}(v[E + p]) = 0, \quad (4d)$$

where t is time, ρ is density, p is pressure, u and v are the x - and y -velocity components, and E is the total energy. Gravity is neglected. A stiffened equation of state relates the pressure to the internal energy

$$E = \frac{\rho(u^2 + v^2)}{2} + \frac{p + nB}{n - 1}, \quad (5)$$

where B is an empirically determined measure of liquid stiffness. For perfect gases, such as in our treatment of air, n is the specific heat ratio and $B = 0$. The interface evolution is captured using a γ -based model [40] such that

$$\frac{\partial}{\partial t}\left(\frac{1}{n-1}\right) + u\frac{\partial}{\partial x}\left(\frac{1}{n-1}\right) + v\frac{\partial}{\partial y}\left(\frac{1}{n-1}\right) = 0, \quad (6a)$$

$$\frac{\partial}{\partial t}\left(\frac{nB}{n-1}\right) + u\frac{\partial}{\partial x}\left(\frac{nB}{n-1}\right) + v\frac{\partial}{\partial y}\left(\frac{nB}{n-1}\right) = 0. \quad (6b)$$

We initially prescribe a small, finite-thickness parameter δ to the interface [41] such that the initial volume fraction field is

$$\alpha_0 = \begin{cases} 1 & \text{(water)} \\ \exp[\ln(10^{-16})|d|^8] & \text{(mixture)} \\ 0 & \text{(air)}, \end{cases} \quad (7)$$

where $d = \frac{\delta + y_{\text{interface}}(x) - y}{2\delta}$ and $\delta = 0.08\ell$.

The dimensional fluid properties used for air are determined at 300 K and 1 atm such that $\rho_{\text{air}} = 1.18 \text{ kg/m}^3$ and $c_{\text{air}} = 347.2 \text{ m/s}$. For water, $\rho_{\text{water}} = 996 \text{ kg/m}^3$ and $c_{\text{water}} = 1648.7 \text{ m/s}$. The parameters in the stiffened equation of state are $n_{\text{air}} = 1.4$, $B_{\text{air}} = 0$, $n_{\text{water}} = 5.5$, and $B_{\text{water}} \approx 492 \text{ MPa}$ [42–44]. The density and sound speed of water, as well as the alveolar diameter, equal here to the interface perturbation wavelength, are used for nondimensionalization.

The equations are solved on a domain in the xy plane in the ranges $0 \leq x \leq 1\ell$ (periodic in x) and $-20\ell \leq y \leq 60\ell$ (outflow boundary conditions in y). The y length of the domain is chosen such that the entire wave initially fits within the domain. We use a third-order accurate discontinuous Galerkin scheme ($p = 1$) in space with the Roe solver and a fourth-order-accurate adaptive Runge-Kutta method to march forward in time [45]. To isolate the effects of a single pulse, the longest time span reasonable to observe the evolution of the system is the time between consecutive pulses, which for a typical pulse repetition frequency of 1 kHz is $t < \delta t_{\text{pulse}} = 1000 \mu\text{s}$ [30]. The grid resolution is 100 points per ℓ in x and y , except for the topmost and bottommost 10ℓ segments of the domain, where the grid is stretched geometrically to minimize artificial reflections. Given the exceedingly long time duration, we used the highest possible grid resolution based on our computing resources and time constraints. Though the solution cannot be fully converged in a pointwise sense with the Euler equations [46], the results show grid dependence of certain integral quantities. Nevertheless, the conclusions made on the basis of our results are still valid.

In this study, we determine the dependence of the time evolution of the interfacial amplitude perturbation on the wave amplitude p_a and length L of the wave. To remain clinically relevant, we consider amplitudes between 5.0 and 12.5 MPa and lengths between $L = 10\ell$ and 45ℓ , with our baseline case $p_a = 10 \text{ MPa}$ and $L = 45\ell$. As our results indicate, this baseline is convenient because the pulse amplitude is sufficiently strong to evolve the dynamics to late time within a computationally feasible time, yet not so strong as to drive the system to behave qualitatively differently than weaker waves within the diagnostic ultrasound regime.

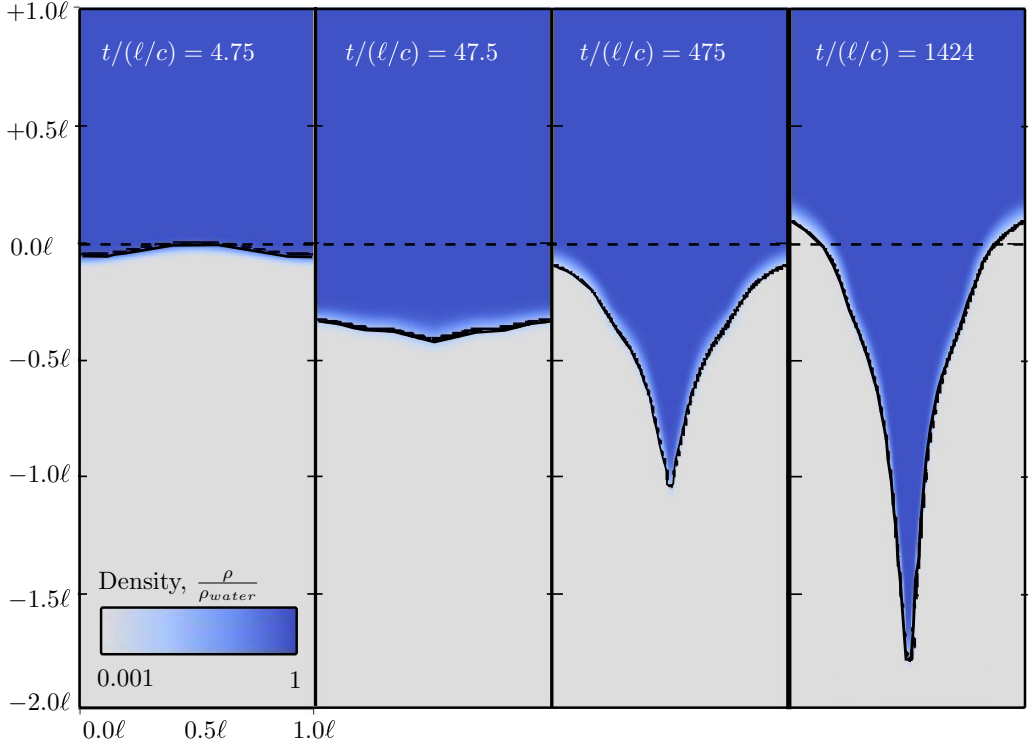


FIG. 3. Density contours at $t/(\ell/c) = 4.75, 47.5, 475$, and 1424 for the baseline $p_a = 10$ MPa, $L = 45\ell$ trapezoidal wave. The black solid line is the $\alpha = 0.5$ volume fraction isoline and the black dashed line the initial mean interface location.

As the model and problem definition developed here are motivated by diagnostic ultrasound of the lung, it is pertinent to acknowledge limitations of the present work with regard to ultrasound-lung interaction and to highlight some of the aspects of the motivating physical problem that are beyond the scope of this study. Lung tissue exhibits viscoelastic properties and complex geometries not considered here. At the blood-air barriers in the lungs and any soft tissue-air interface, viscous and elastic effects would work to retard motion and restore the shape of the interface upon strain, respectively. This could be of particular importance over the longer timescales associated with clinical ultrasound. Furthermore, any interfacial evolution leading to the creation of small-scale features would increase the importance of surface tension, which is neglected here. Each of these neglected physical effects may contribute significantly to resisting interfacial deformation at late times. Finally, while preliminary results (not shown here) indicate that gas-liquid interfaces driven by ultrasound pulse waveforms show similar behavior to those driven by the trapezoidal waves presented here, many aspects of realistic ultrasound pulses are outside the scope of this work. The ability to simulate application-specific waves over clinically relevant timescales in particular should be investigated in the future.

III. RESULTS AND DISCUSSION

A. Dynamics of the baseline case

1. Density-based description of the perturbation growth

To exemplify the growth of a perturbation along a water-air interface driven by the trapezoidal wave of interest, Fig. 3 shows the time evolution of the density field for the baseline case. The

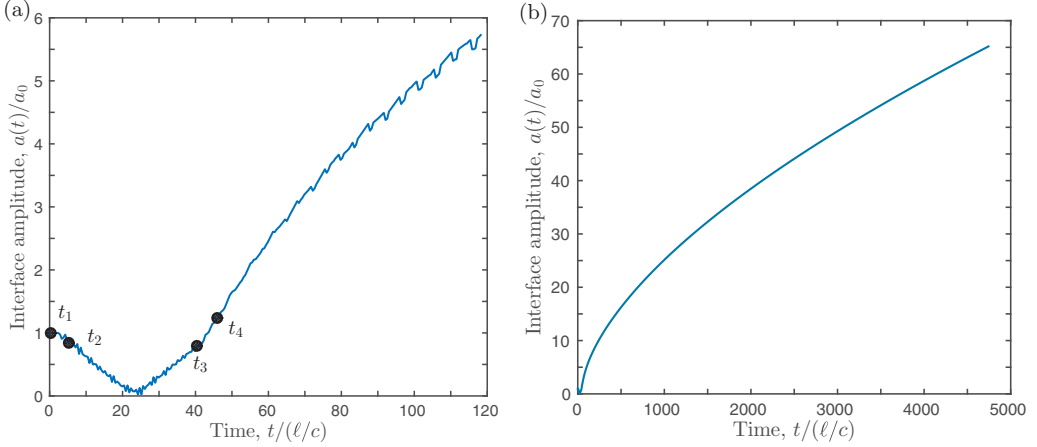


FIG. 4. Interface perturbation amplitude history $a(t)/a_0$ for the baseline $p_a = 10$ MPa, $L = 45\ell$ trapezoidal wave, for (a) $t/(\ell/c) \leq 120$ and (b) $t/(\ell/c) \leq 5000$. In (a), times at which different stages of the incoming trapezoidal pressure wave first encounter the interface are indicated as t_1 (the compression), t_2 (the constant elevated pressure p_a), t_3 (the rarefaction), and t_4 (the return to ambient pressure).

wave propagates from water (top) to air (bottom). Frame 1 shows the interface shortly after it first encounters the wave at $t/(\ell/c) = 4.75$, near the end of the compression. Upon interaction with the interface, nearly all of the acoustic energy is reflected back into the water as a rarefaction due to the significantly lower acoustic impedance of air. The transmitted wave in air is weakly focused or defocused, depending on the convex or concave nature of the curved interface. Between frames 1 and 2, the mean interface location moves in the negative y direction by 0.31 (corresponding to the mean acoustic velocity multiplied by the time between the pressure rise and decrease), as it is advected by the velocity corresponding to the elevated pressure. Between these two frames, the perturbation phase reverses as evidenced by the initial perturbation peak at $x/\ell = 0.5$ becoming a valley. From frame 2 on, bubbles of air are observed to rise into the water along the sides ($x/\ell = 0, 1$), while a water spike penetrates the air in the middle ($x/\ell = 0.5$). This bubble and spike evolution continues well after the incident wave has passed. The cumulative effect is that the interface perturbation grows from an initially smooth sinusoid to a pointed spike at late times.

We turn to a more quantitative description of the time evolution of the perturbation in Figs. 4 (amplitude) and 5 (bubble and spike locations). The bubble and spike locations are defined as the highest and lowest y coordinates, respectively, of the constant $\alpha = 0.5$ volume fraction isoline, and are meaningful only after phase reversal; the interface amplitude is calculated by taking the difference between the bubble and spike locations. The early-time behavior is characterized by several distinct events. Following the impingement of the leading end of the wave at $t/(\ell/c) = t_1 = 0.3$, the interfacial pressure rises until $t_2 = 5.3$, at which point the pressure has reached its maximum amplitude. As the initial perturbation peak moves in the negative y direction, the interface amplitude decreases to nearly zero (flat interface) at $t_p = 24$, the instant when the phase reverses. The amplitude increases thereafter as the initial peak (now the spike) continues its progress in the negative y direction. The interfacial pressure remains constant until $t_3 = 40.3$, at which point the leading end of the rarefaction reaches the interface. The pressure decreases until $t_4 = 46.1$, at which point the full wave has left the interface and the pressure is atmospheric again, as it was originally. The perturbation amplitude continues to grow well after the wave has passed, reaching many times its initial value. At these late times, the growth appears to be smooth, continuous, and monotonic in time. This large growth may have implications in the context of potential ultrasound-generated damage to the lung as the alveolar surface elongates, thus potentially giving rise to capillary rupture.

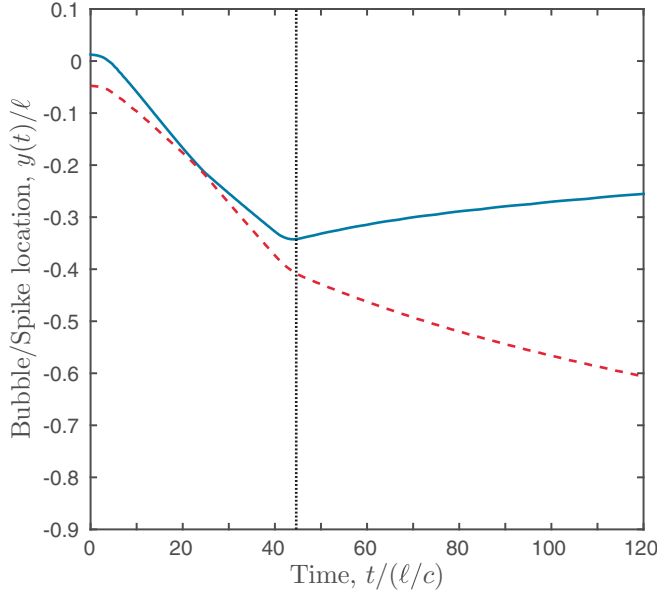


FIG. 5. The y locations of the bubble and spike for the baseline $p_a = 10$ MPa, $L = 45\ell$ trapezoidal wave, during and shortly after the wave-interface interaction. By definition, the bubble is the top (blue solid) curve and the spike is the bottom (red dashed) curve. The black dotted line shows t_Γ , as defined by the minimum bubble location.

Slightly before t_4 , the slope of the bubble and spike locations changes significantly, at $t/(\ell/c) = t_\Gamma = 44.6$. We remark that between t_2 and t_3 the bubble and spike velocities consist of the superposition of the wave velocity corresponding to the elevated pressure and the velocity induced by the baroclinic vorticity, to be discussed in greater detail in the next section. Once the magnitude of the former is sufficiently small (toward the end of the passage of the wave), the latter becomes dominant. At this time, the net bubble velocity becomes positive and rises. This time t_Γ , defined as the minimum in the bubble location after phase reversal, plays an important role in our analysis in Sec. III B.

The most important contribution of linear acoustics is the interface translation (downward on the contour plots) during the interaction with the wave; the overall interfacial perturbation evolution cannot be explained solely by this principle. The combined effects of the compression and the deformations occurring between the time the wave travels from the perturbation peak to trough would yield a perturbation amplitude change of approximately $0.01a_0$. Linear acoustics would further imply that the perturbation should no longer evolve after the wave passage. It follows that nonlinear mechanisms must drive the perturbation growth.

2. Vorticity-based description of the perturbation growth

The results presented in the preceding section demonstrate that the perturbation amplitude grows well after the incident wave has traversed the interface, driven by a mechanism that cannot be explained by conventional linear acoustics. During interaction with the interface, the pressure differential (due to the geometrical perturbation) over a short distance applies a torque on fluid particles along the interface, thus generating rotation (baroclinic vorticity). Such effects are higher order and thus negligible for acoustic waves encountering small density variations; in the present problem, however, the pressure and density change by significant amounts over short distances, thus giving rise to substantial gradients dominating otherwise first-order (acoustic) effects. For these

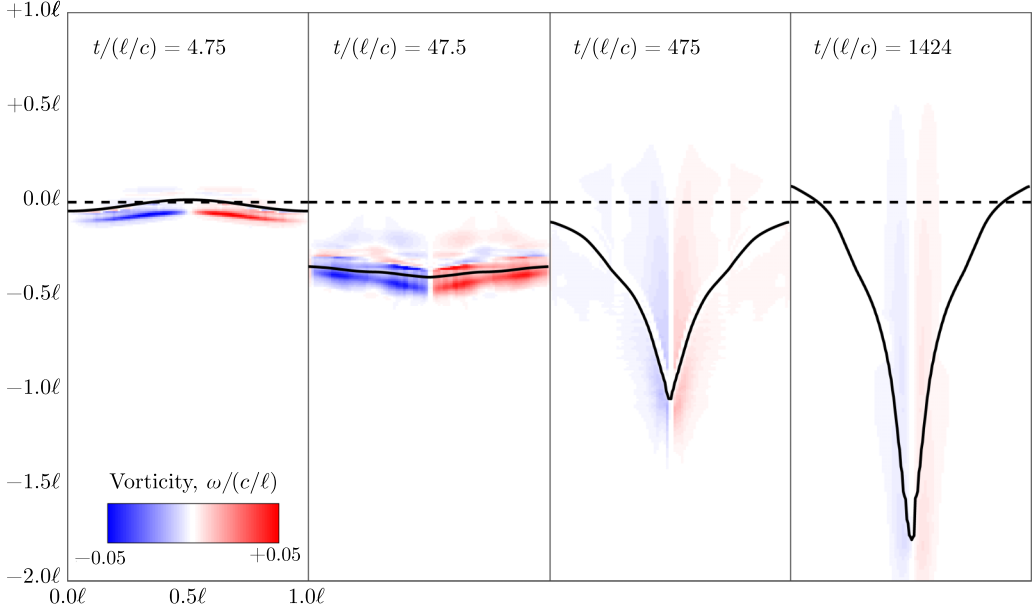


FIG. 6. Vorticity contours at $t/(\ell/c) = 4.75, 47.5, 475$, and 1424 for the baseline $p_a = 10$ MPa, $L = 45\ell$ trapezoidal wave case. The black solid line is the $\alpha = 0.5$ volume fraction isoline and the black dashed line is the initial mean interface location.

reasons, we examine the perturbation growth in terms of vorticity $\boldsymbol{\omega} = \nabla \times \mathbf{u}$, whose evolution in two dimensions is given by

$$\frac{\partial \boldsymbol{\omega}}{\partial t} + (\mathbf{u} \cdot \nabla) \boldsymbol{\omega} = -\boldsymbol{\omega}(\nabla \cdot \mathbf{u}) + \frac{\nabla \rho \times \nabla p}{\rho^2}. \quad (8)$$

The high impedance mismatch and relatively low dilatation at the wave amplitudes under consideration make the first term on the right-hand side (dilatation) essentially negligible compared to the last term (baroclinic), which is large given the nearly discontinuous density gradient and the significant pressure variations over relatively short lengths; the analysis in the Appendix quantitatively supports this claim. Figure 6 depicts vorticity contours at $t/(\ell/c) = 4.75, 47.5, 475$, and 1424 for the baseline case. At time zero, there is no vorticity in the domain. Frame 1 shows that by the end of the compression-interface interaction a vortex sheet has developed along the interface, with negative vorticity for $x/\ell \in [0.0, 0.5]$ and positive vorticity for $x/\ell \in [0.5, 1.0]$. The vorticity appears to be primarily in the air as the interface location (black line) is taken to be the $\alpha = 0.5$ volume fraction isoline. By frame 2, the initially deposited vorticity has driven the perturbation peak downward such that the phase is now reversed during the passage of the rarefaction. As a consequence of this phase reversal, most of the vorticity deposited by the rarefaction has the same sign as the vorticity generated by the compression, despite the corrugation of the interface. If the interface had remained undeformed, the vorticity deposited by the rarefaction would have been of opposite sign and would have thus acted to cancel that due to the compression. Instead, the enhanced vorticity gives rise to a clockwise (left-half domain) and counterclockwise (right-half domain) vortex pair driving the spike to form at $x/\ell = 0.5$. Over time, the vorticity contours become fainter but appear to spread over a larger region.

During the interface evolution, the vorticity redistributes itself along the interface. To quantify this behavior, we plot in Fig. 7 the cumulative vorticity (in y) along the interface, $\int_{-\infty}^{+\infty} \tilde{\omega}(x, y) dy$, where $\tilde{\omega} = \omega$ for $0 < \alpha < 1$ and is otherwise zero (in pure water or air). Initially, the vorticity is smooth and nearly sinusoidal, as expected [47]. During the phase reversal process, the vorticity peaks move

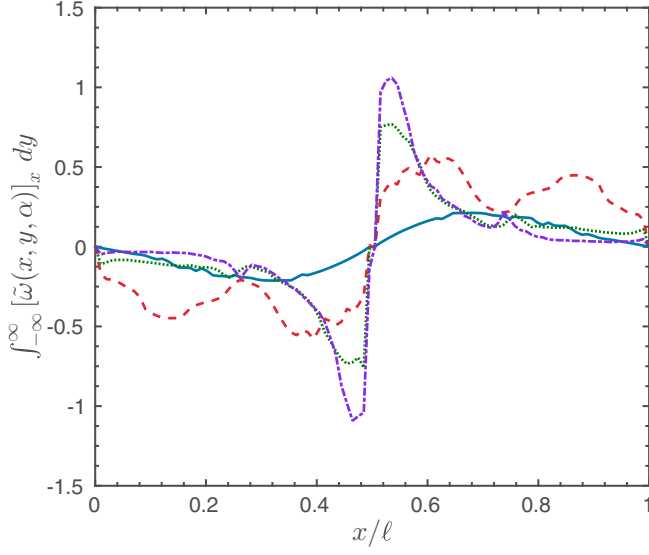


FIG. 7. Cumulative vorticity (in y) along the interface, $\int_{-\infty}^{+\infty} \tilde{\omega}(x, y) dy$, where $\tilde{\omega} = \omega$ for $0 < \alpha < 1$ and is otherwise zero (in pure water or air), at $t/(\ell/c) = 4.75$ (blue solid line), 47.5 (red dashed line), 475 (green dotted line), and 1424 (purple dash-dotted line).

toward $x/\ell = 0.5$. Given the geometry at the time when the rarefaction arrives, a second peak in the vorticity distribution is observed near $x/\ell = 0.0$ and 1.0 at $t/(\ell/c) = 47.5$. Though apparently fainter in the contour plots, the vorticity is clearly concentrated near the spike, driving the heavy fluid into the light one.

For a more quantitative global measure of vorticity, we consider the circulation produced in the right-half domain (the left is equal and opposite by symmetry) for the baseline case in Fig. 8. The same times at which different stages of the incoming trapezoidal pressure wave first encounter the interface

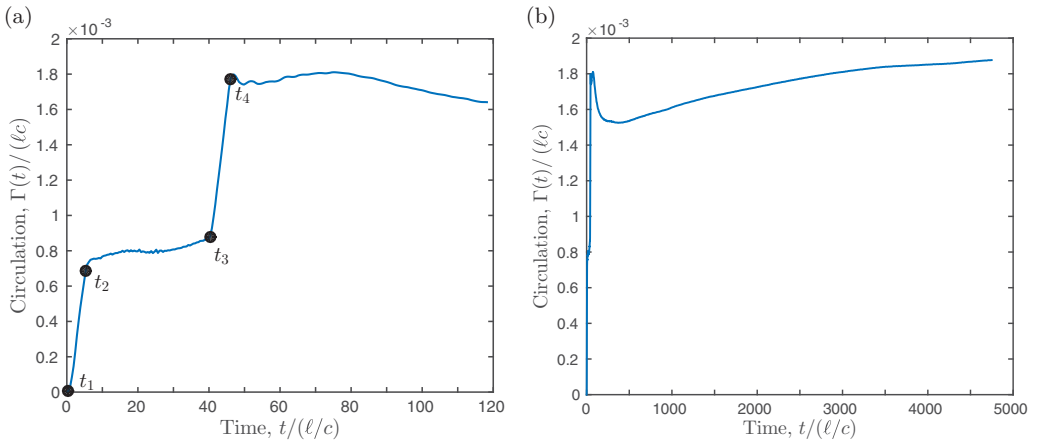


FIG. 8. Circulation history for the right-half domain for the baseline $p_a = 10$ MPa, $L = 45\ell$ trapezoidal wave case, for (a) $t/(\ell/c) \leq 120$ and (b) $t/(\ell/c) \leq 5000$. In (a), times at which different stages of the incoming trapezoidal pressure wave first encounter the interface are indicated as t_1 (the compression), t_2 (the constant elevated pressure p_a), t_3 (the rarefaction), and t_4 (the return to ambient pressure).

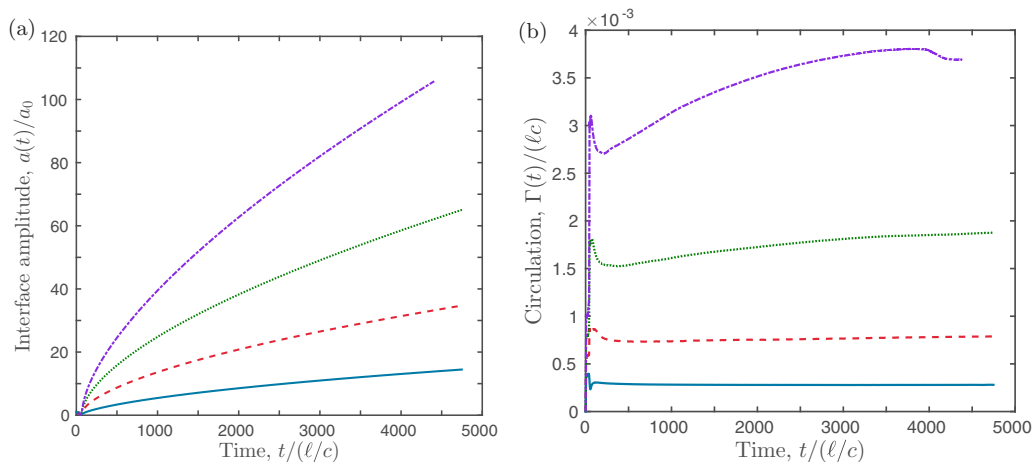


FIG. 9. Histories of the (a) interface amplitude and (b) circulation for trapezoidal wave cases with $L = 45\ell$ and $p_a = 5.0$ (blue solid line), 7.5 (red dashed line), 10.0 (green dotted line), and 12.5 (purple dash-dotted line) MPa, for $t/(\ell/c) \leq 5000$.

interface are indicated on this figure. From t_1 to t_2 (during the interaction of the compression with the interface), positive vorticity (circulation) is deposited given the direction of the density and pressure gradients. This circulation increase is linear since the pressure and density gradients are approximately constant over the interaction interval. Other than small changes due to transverse wave reflections, the circulation remains essentially constant until t_3 , at which point the leading edge of the rarefaction encounters the interface. Since the phase has reversed at t_p , the deposited vorticity is of the same sign (i.e., positive) as that deposited by the compression, such that the circulation approximately doubles by the time the trailing end of the rarefaction arrives at t_4 . Thereafter, any changes in vorticity after this point are no longer due to the primary incoming wave. The decrease in circulation observed after the wave passage is due to the intersection of the transverse reflections of the rarefaction (now compressions) near the bubble, while the late-time increase is attributed to the acceleration of the heavy fluid into the light one as the spike penetrates the air, which is a form of secondary baroclinic vorticity generation [48].

B. Dependence of the perturbation growth on the wave amplitude

1. General observations

Since the perturbation growth is driven by residual baroclinic vorticity deposited by the interaction of the ultrasound wave with the interface, it follows that the growth rate increases if more vorticity is deposited. Thus, holding all other parameters fixed, we anticipate that the growth rate of perturbation amplitude will increase with increasing pressure wave amplitude, given that the baroclinic term is proportional to the pressure difference. This behavior is confirmed by Fig. 9, which shows the amplitude and circulation histories for wave amplitudes ranging from 5.0 to 12.5 MPa; to help analyze the results, Table I lists the circulation at times t_{1-4} . As expected, after the initial transient behavior, the late-time growth rate increases with increasing pressure amplitude. The circulation values at the different t_i follow a consistent behavior, as the values increase with increasing pressure amplitude. At t_2 , we observe that the circulation deposited by the compression increases at a nearly constant rate with pressure amplitude, consistent with the fact that the time rate of change of the circulation is proportional to the baroclinic term, itself proportional to the pressure difference. From t_2 to t_3 and from t_3 to t_4 , the rise in circulation generally increases with increasing amplitude, except for the 5-MPa wave between t_3 and t_4 . In this latter case, there is a decrease in circulation, due to the fact that phase inversion has not occurred by the time the rarefaction encounters the interface.

TABLE I. Circulation during the wave-interface interaction.

p_a (MPa)	$\times 10^3 [\Gamma(t_i)/\ell c]$			
	t_1	t_2	t_3	t_4
5.0	0.0	3.3	3.8	2.9
7.5	0.0	5.0	6.0	8.7
10.0	0.0	6.8	8.8	18.0
12.5	0.0	8.5	11.9	30.5

After t_4 , we observe that both the decrease and late-time rise in circulation depend on the pressure amplitude; greater amplitudes lead to greater changes. In the $p_a = 12.5$ MPa case, the circulation appears to decrease at very late times ($t/(\ell/c) \gtrsim 4000$). This behavior is caused by round-off level errors accumulating over the course of this long simulation, thus breaking the left-right symmetry of the simulation [49].

2. Late-time scaling of the perturbation amplitude

The smooth and monotonic behavior of the perturbation amplitude growth suggests that a more general scaling describing its dependence on the pressure amplitude may exist. Clearly, the perturbation growth is related to the circulation Γ . As the perturbation grows, the vorticity redistributes itself along the interface such that dependence on the interface length s and the initial wavelength ℓ are expected. Finally, given that the waves traverse the interface over a finite duration, dependence on the sound speed c is expected. Thus, the dependence of the amplitude on these variables can be formulated as a dimensional analysis problem

$$a(t) = f(\Gamma, s, \ell, c; t) \Rightarrow \frac{a(t)}{\ell} = G\left(\frac{\Gamma}{\ell c}, \frac{s}{\ell}; \frac{t}{\ell/c}\right), \quad (9)$$

where ℓ and c are used for nondimensionalization. We note that there is likely an Atwood number dependence, but since the density fields do not significantly change we ignore this parameter here. Well after the wave passage (e.g., for $t/(\ell/c) \gtrsim 500$), the circulation no longer changes significantly (except perhaps in the $p_a = 12.5$ MPa case, where the circulation changes up to approximately 30%), as there is no dominant mechanism to affect it. However, as the interface deforms and elongates [i.e., $s(t)$ increases], the vorticity gets redistributed along the interface. The circulation density Γ/s is thus a relevant quantity describing the vortex dynamics [50]. As observed in Sec. III A 2, the baroclinic vorticity is the dominant contributor to the interface perturbation growth after t_Γ . Thus, we expect the perturbation amplitude to depend on the circulation density at this time, which we define $\Gamma(t_\Gamma)/s(t_\Gamma) = \Gamma_0/s_0$. It follows that

$$\frac{a(t)}{\ell} = F\left(\frac{\Gamma_0}{s_0 c}, \frac{tc}{\ell}\right). \quad (10)$$

Finally, we hypothesize that the perturbation growth scales linearly with the circulation density such that

$$\frac{a(t)}{\ell} = \frac{\Gamma_0}{s_0 c} \mathcal{F}\left(\frac{tc}{\ell}\right). \quad (11)$$

To verify this hypothesis, we plot the perturbation amplitude $a(t)$ scaled by the circulation density at t_Γ in Fig. 10. To facilitate the comparison, the time origin has been shifted by t_p such that the instant of phase inversion has been synchronized for all cases. Two observations stand out. First, the scaled growths collapse onto what appears to be a single curve at sufficiently late times. Second, this curve appears to asymptote to a constant slope on the log-log plot, thus exhibiting a power-law behavior

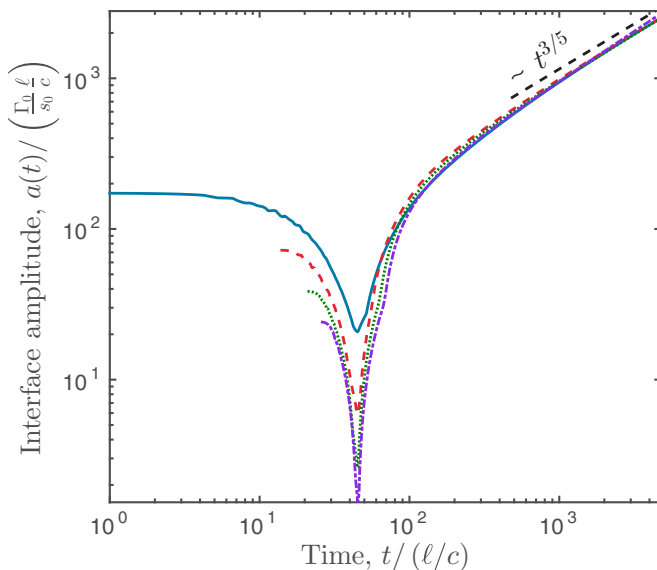


FIG. 10. Interface amplitude scaled by circulation density at t_T for the trapezoidal wave cases with $L = 45\ell$ and $p_a = 5$ (blue solid line), 7.5 (red dashed line), 10 (green dotted line), and 12.5 (purple dash-dotted line) MPa. Time is synchronized based on phase inversion. The black dashed line shows the power-law growth as $t^{3/5}$.

$\mathcal{F} = (t/(\ell/c))^n$. To compute the interface amplitude growth time exponent n , we write

$$\ln \left[a(t - t_p) / \left(\frac{\Gamma_0 \ell}{s_0 c} \right) \right] = b + n \ln[(t - t_p)/(\ell/c)], \quad (12)$$

where t_p is the phase-reversal time and b is the y intercept of the best fit line, which depends on the value of $a(t - t_p)/(\frac{\Gamma_0 \ell}{s_0 c})$ when the interface growth becomes asymptotic. By applying linear regression to the data for $(t - t_p)/(\ell/c) \geq 2000$, we determine the best-fit values for n in a least-squares sense. We find that $n \approx 3/5$, as illustrated in Table II. Though difficult to distinguish in Fig. 10, the $p_a = 12.5$ MPa case exhibits a slightly higher time exponent, with n closer to $2/3$; this discrepancy may be due to the fact that circulation, even at late times, still changes in a non-negligible fashion in this case. We note, however, that this amplitude falls beyond those typically used in clinical diagnostic ultrasound.

3. Late-time scaling of the interfacial length

The tenfold to hundredfold perturbation amplitude growth is accompanied by a corresponding increase in interfacial length s . This quantity is important for the dynamics of the vortex sheet

TABLE II. Interface amplitude growth time exponents, where $\frac{a(t)}{t} \sim t^n$.

p_a (MPa)	$n(t/(\ell/c) \geq 2000)$
5.0	0.61
7.5	0.59
10.0	0.61
12.5	0.66

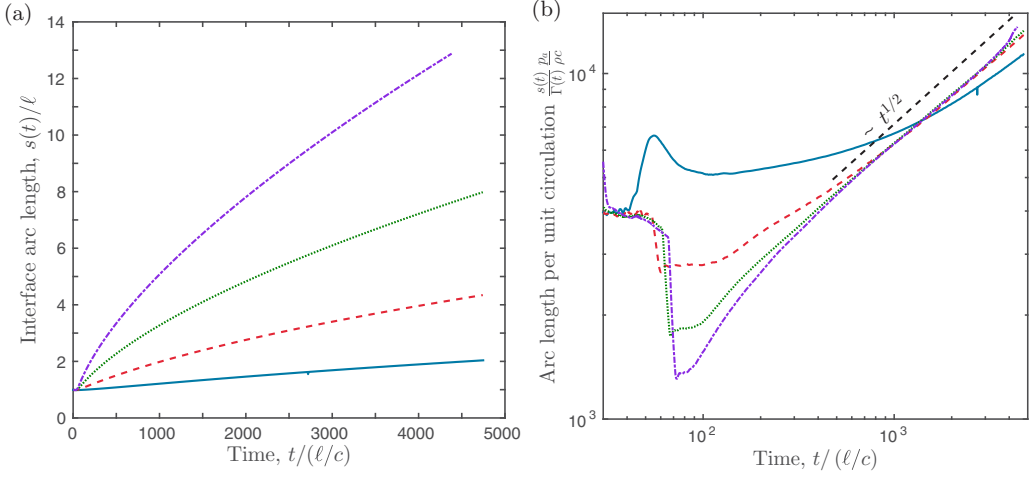


FIG. 11. Histories of the (a) interfacial length and (b) scaled interfacial length for the trapezoidal wave cases with $L = 45\ell$ and $p_a = 5$ (blue solid line), 7.5 (red dashed line), 10 (green dotted line), and 12.5 (purple dash-dotted line) MPa. Time is synchronized based on phase inversion. The black dashed line shows the power-law growth as $t^{1/2}$.

produced along the interface by the ultrasound passage [50]; vortex sheet dynamics have in fact been explored in the context of the Rayleigh-Taylor instability [51]. In such analysis, the quantity of interest is the circulation density Γ/s . We expect this quantity to depend on the wave amplitude p_a , the initial wavelength ℓ , and the density and sound speed of the liquid, ρ and c , respectively. Following a dimensional analysis process similar to that in the preceding section, we find that the inverse of the circulation density (in other words, the interfacial length scaled by instantaneous circulation) bears the following dependence on the relevant dimensionless parameters:

$$\frac{s(t)}{\Gamma(t)}c = \psi\left(\frac{p_a}{\rho c^2}, \frac{tc}{\ell}\right). \quad (13)$$

Given that the growth is baroclinic, we hypothesize that the circulation scales linearly with pressure amplitude such that

$$\frac{s(t)}{\Gamma(t)/c} = \frac{\rho c^2}{p_a} f\left(\frac{tc}{\ell}\right). \quad (14)$$

To determine the time dependence of the interfacial length, we plot in Fig. 11 the time histories of the interfacial length and scaled interfacial length. Again, time is synchronized based on phase inversion. As for the amplitude, the growth rate of the length increases with pressure amplitude. Furthermore, with the exception of the $p_a = 5$ MPa case, our simple scaling collapses the interfacial length onto a single curve after a sufficiently long time ($t/(\ell/c) \gtrsim 500$). The collapsed curve exhibits a power-law dependence on time $f = (t/(\ell/c))^m$, where $m \approx 1/2$ for $p_a = 7.5, 10$, and 12.5 MPa. This scaling confirms that the interfacial deformations are governed by the dynamics of the vortex sheet produced by the wave interaction. The result from the $p_a = 5$ MPa case does not follow the same behavior for two main reasons. First, the rarefaction encounters the interface during phase inversion, at which point the interface is essentially flat. Thus, the vorticity contribution is negligible; nevertheless, the rarefaction accelerates the interface and increases its length, thus decreasing the circulation density. As a result, the geometry at t_p is different from that observed in the other cases. Second, s/Γ has yet to achieve its asymptotic behavior. However, running the simulation for a sufficiently long time to reach this asymptotic behavior would be prohibitively expensive from a computational standpoint.

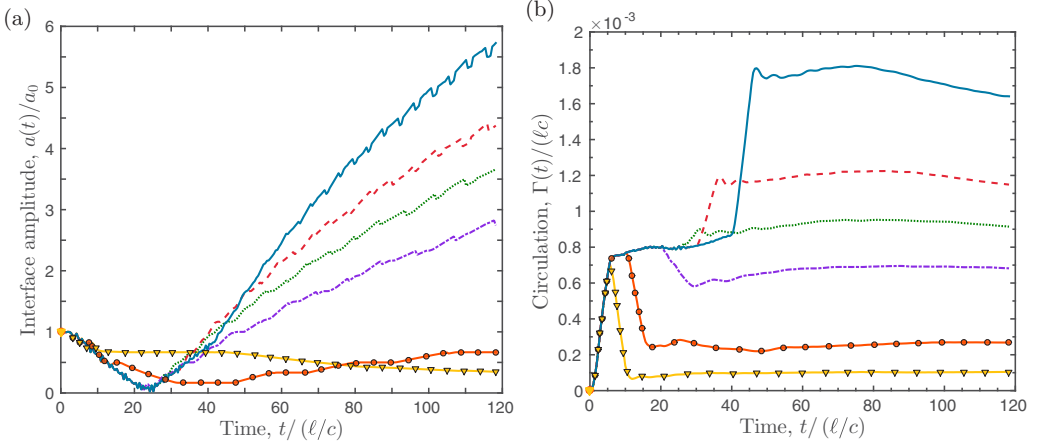


FIG. 12. (a) Interface amplitude and (b) circulation histories for the $p_a = 10$ MPa trapezoidal wave case with $L = 45\ell$ (blue solid line), 35ℓ (red dashed line), 30ℓ (green dotted line), 20ℓ (purple dash-dotted line), 15ℓ (orange line with circles), and 10ℓ (yellow line with triangles).

We highlight the different dependence of the perturbation amplitude and interfacial length on circulation. The growth of the former is dictated by the geometry and the amount of circulation at the end of the interaction. Thus, the instantaneous circulation at that time is sufficient to describe the growth. On the other hand, the interfacial length depends on the details of the vortex sheet dynamics. Thus, the interfacial length is sensitive to the instantaneous circulation and in fact local vorticity.

Finally, we remark that grid independence is not fully achieved on the current meshes, i.e., the actual value of the time exponents may change slightly as the grid is refined. For instance, for the $p_a = 10$ MPa wave, the interfacial length per unit circulation time exponents are approximately $m = 0.49, 0.46$, and 0.43 for 75, 100, and 125 points per wavelength, respectively. Given the limited computational resources and exceedingly long simulation times required to achieve an asymptotic state, a more comprehensive resolution study is not feasible. Furthermore, at these extremely late times in inviscid simulations of interfacial flows, round-off level errors are known to grow and give rise to asymmetric growth, despite the symmetry of the problem [49,52]. Nevertheless, achieving an asymptotic growth state does not appear to depend on grid spacing at the resolutions under consideration.

C. Dependence of the perturbation growth on the wave duration

The results from the previous sections indicate that the interface morphology during the interaction of the rarefaction with the interface plays a key role in the dynamics. If the interface has undergone phase inversion by the time the rarefaction has arrived, vorticity of the same sign as that due to the compression is deposited, thus enhancing the growth. On the other hand, if the rarefaction arrives before the phase has inverted, vorticity of the opposite sign is deposited and counteracts the vorticity initially deposited by the compression. In the limit where the rarefaction immediately follows the compression, zero net vorticity would be deposited only if the interface has not deformed, thus leading to no growth. The amount of baroclinic vorticity deposited by the rarefaction depends on the interface morphology at the time of interaction, described by the sine of the angle between density and pressure gradients, such that the effect of the rarefaction on the interface perturbation growth depends heavily on the time-dependent features of the wave. To examine this behavior, we hold the pressure amplitude constant at $p_a = 10$ MPa and vary the time (or length L) between the compression and rarefaction. The corresponding amplitude and circulation histories are shown in Fig. 12. For the two longest waves $L = 35\ell$ and 45ℓ , the rarefaction encounters the interface after

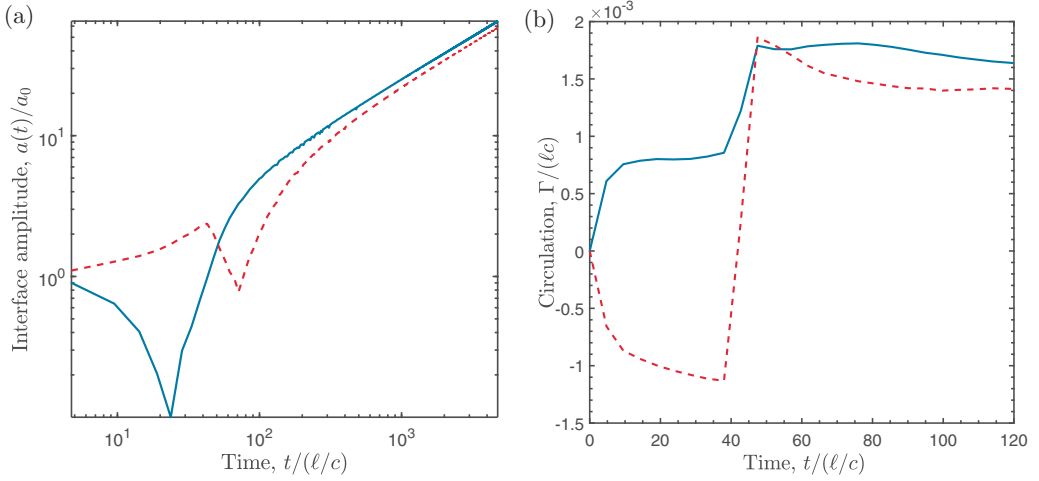


FIG. 13. (a) Interface amplitude and (b) circulation histories for the $p_a = +10$ (blue solid line) and -10 (red dashed line) MPa trapezoidal wave cases with $L = 45\ell$.

phase inversion. In these cases, the rarefaction deposits additional vorticity of the same sign as that due to the compression (e.g., positive vorticity in the right side of the domain) and enhances growth. For the $L = 30\ell$ case, the rarefaction impinges upon the interface shortly after the interface phase reversal, when the interface is nearly flat. As a result, the pressure and density gradients are nearly aligned and little additional circulation is generated. Thus the growth is driven purely by the circulation deposited by the compression. For shorter waves ($L \leq 25\ell$), the rarefaction encounters the interface before the perturbation reverses phase, thus reducing the circulation. Among cases for which the interface inverts phase before encountering the rarefaction, the larger the instantaneous perturbation amplitude at the time of the rarefaction, the greater the circulation generated, since the average angle between the density and pressure gradients is greater. The same is true when comparing cases for which the interface inverts its phase before encountering the rarefaction. We note that the staircaselike behavior observed in the curves at late times is an artifact of the postprocessing software.

These results suggest that by appropriately modulating the incoming wave the perturbation growth could be controlled. This observation is particularly important for waves for which the pressure returns to its ambient value after the wave passage, which is the case for acoustic waves in general by contrast to the conventional shock-accelerated (Richtmyer-Meshkov instability) problem.

D. Dependence of the perturbation growth on the wave polarity

The results shown thus far indicate that interface evolution is driven by the acoustic wave and deposited vorticity during the wave-interface interaction and at late times is governed by the residual baroclinic vorticity. It is reasonable to ask whether the dynamics and morphology of the interface, during the wave-interface interaction, depend on the polarity of the wave. Because the rate of baroclinic vorticity generation is proportional to the pressure gradient of the acoustic wave, it logically follows that the sign of the deposited vorticity also depends on the sign of the incoming waveform. Hence the interface dynamics are expected to depend on the polarity of the driving wave during the interaction but exhibit similar asymptotic growth at late times.

To examine this behavior, we consider a wave with the same parameters as the baseline case, but with $p_a = -10$ MPa, i.e., a wave with opposite polarity, hereafter referred to as the negative wave. The corresponding amplitude and circulation histories are shown in Fig. 13, along with the baseline case (i.e., positive wave) for comparison. For the negative wave, during the initial rarefaction, the perturbation amplitude increases and negative vorticity is deposited along the interface in the

right-half plane. The perturbation amplitude continues to grow throughout the constant pressure period in the direction opposite that observed in the case of the positive wave, but consistent with the sign of the vorticity. This growth is entirely positive and is enhanced by the initially deposited vorticity. At $t/(\ell/c) = 42.7$, during the compression, the perturbation amplitude begins to decrease. For the negative wave, the angle between the interface density and wave pressure gradients is greater for the compression than for the earlier rarefaction. Consequently, the compression generates vorticity of opposite sign (i.e., positive), but with greater magnitude than that of the initial rarefaction. Thus, after the passage of the negative wave, net positive vorticity remains along the interface, as is the case for the positive wave. At $t/(\ell/c) = 71.2$ the phase inverts and the bubble and spike continue moving in their respective directions, driven by vorticity. The asymptotic behavior of the perturbation amplitude is very close to that of the positive wave: The interface amplitude growth time exponent is also approximately $n = 0.61$. While the circulation and interface dynamics are clearly dependent on the polarity of the wave during the wave-interface interaction, the late-time dynamics are not. One qualitative difference of note between the positive and negative trapezoidal wave cases is that, independently of pressure amplitude, the negative trapezoidal wave is not expected to drive the phase inversion during the wave-interface interaction, because of the direction the interface peaks and troughs move due interaction with the wave. Based on these results, it is reasonable to expect that arbitrary pressure waveforms interacting with perturbed interfaces give rise to different interfacial dynamics during the interaction and for some time thereafter; however, the late-time behavior is expected to be dominated by the residual vorticity deposited during the interaction.

IV. CONCLUSION

We investigated the interaction of an acoustic wave propagating in a liquid and impinging upon a perturbed liquid-gas interface, as a model for ultrasound interaction with alveoli, in the context of lung hemorrhage. For waveform parameters (amplitude, length, and polarity) relevant to the application, we observed that acoustic waves in water lead to interface perturbation phase inversion, followed by perturbation growth to amplitudes many times larger than the initial value well after the wave passage. We further characterized the dependence of the perturbation growth on the wave amplitude and length. We demonstrated that the mechanism driving the perturbation growth is the torque generated by the misalignment of the pressure and density differentials during the wave interaction, manifested by the production of baroclinic vorticity along the interface. This effect, usually higher order in acoustics, is dominant in our problem due to the substantial pressure difference over a short length (megapascals over millimeters) and the nearly discontinuous density profile across the interface. Although the symmetric nature of the wave may suggest that vorticity deposited by the compression should be exactly canceled by that produced by the rarefaction, such an argument overlooks the transient nature of the process, namely, the fact that the baroclinic torque drives the interface to deform during the passage of the compression, during the time when the pressure amplitude is kept high and during the passage of the rarefaction. As a result, the alignment between the pressure and density gradients is different during the passage of the rarefaction, compared to that produced by the compression. This result can be generalized to state that, waves interacting with an interface over a finite duration and producing interface deformation may generate net baroclinic vorticity, whether the wave is symmetric in time or not.

Depending on whether phase inversion has occurred by the time the rarefaction reaches the interface, one of two perturbation growth regimes is observed. If the phase has inverted, vorticity of the same sign as that deposited by the compression is produced, thus leading to enhanced growth; conversely, if the phase has not yet inverted, vorticity of the sign opposite that deposited by the compression is produced, thus leading to reduced growth. Varying the amplitude and length of the wave can shift the dynamics from one regime to the other. Although the early-time behavior depends on the wave properties (length, amplitude, and polarity) and interface morphology, the late-time perturbation growth obeys a power-law scaling in time at sufficiently high pressure amplitudes, irrespective of the wave polarity. By considering the evolution of the interfacial length and the vorticity

redistribution along the interface, we find this result to be consistent with a vortex-sheet-based description of the interface dynamics. The dependence of the results on the wavelength suggests that perturbation growth may be controlled by modulating the waveform.

This work is a step toward a fundamental understanding of the effects of acoustically generated vorticity along liquid-gas interfaces. Despite selecting a simplified yet relevant waveform (trapezoidal, symmetric in time, returns to ambient conditions after pressure rise) to facilitate the analysis, portions of the results are generalizable to more complex, continuous ultrasound waveforms. Specifically, our observation that transient waves may be capable of depositing lasting vorticity, driving interface evolution long after the wave passage, may be generalizable to waves capable of appreciably deforming the interface during the wave-interface interaction. A potential consequence of this is that significant strains may be imposed upon the interface, given the potentially large, vorticity-driven perturbation growth. However, to directly relate these findings to ultrasound-induced lung hemorrhage, further studies with more comprehensive descriptions of the tissue-lung rheology (viscoelastic properties) and geometry are required, along with the use of application-specific waveforms.

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APPENDIX: ORDER OF MAGNITUDE ANALYSIS OF VORTICITY DYNAMICS

To quantifiably compare the various mechanisms by which vorticity changes in the flow, we perform an order of magnitude analysis on each term of the vorticity transport equation (8). Initially, there is no vorticity. Given the present problem setup, the only mechanism that can produce vorticity is the baroclinic torque, which is clearly nonzero during the interaction of the ultrasound wave with the interface since the pressure (wave) and the density (interface) gradients are misaligned. For this reason, we focus on the relative magnitude of each term during the interaction time $\Delta t_a \approx 5$ as the compression encounters the interface. Since the average perturbation amplitude during the interaction $0.96a_0$ is sufficiently close to a_0 , we assume the interface remains static and undeformed throughout the interaction such that the density gradient is approximately constant. We treat the divergence and magnitude of curls or gradients of quantity f as $\sim \Delta f / \Delta L$, where ΔL is the problem's characteristic length scale. Since the flow is driven by the acoustic wave $\Delta p = \Delta p_a$, $\Delta u = \Delta u_a$, and $\Delta \rho = \Delta \rho_a$, where the subscript a denotes acoustic quantities. The quantities are related according to

$$\Delta p_a = \pm \Delta u_a \rho c = c^2 \Delta \rho_a. \quad (\text{A1})$$

We define the angle between the density and pressure gradients as θ . Since $a_0/\ell \ll 1$ (indicating small misalignment between $\nabla \rho$ and ∇p), we can approximate $\sin \theta \approx \theta$. It thus follows that the magnitude of the baroclinic term is

$$\left\| \frac{\nabla \rho \times \nabla p}{\rho^2} \right\| = |\nabla \rho| |\nabla p| \sin \theta = O\left(\frac{|\Delta \rho_I|}{|\Delta L_I|} \frac{|\Delta p_a|}{|\Delta L_a|} \frac{1}{|\rho|^2} |\theta| \right), \quad (\text{A2})$$

where $\Delta \rho_I$ is the density jump across the interface, Δp_a is the pressure amplitude of the wave, ΔL_I is the characteristic length of the interface (thickness), and ΔL_a is the characteristic length of the wave (wavelength). Approximating the vorticity as of equal magnitude to the baroclinic term, the

dilational term can be estimated as

$$\|-\boldsymbol{\omega}(\nabla \cdot \mathbf{u})\| = O\left(\frac{|\Delta u_a|}{|\Delta L_a|} \frac{|\Delta \rho_I|}{|\Delta L_I|} \frac{|\Delta p_a|}{|\Delta L_a|} \frac{1}{|\rho|^2} |\theta|\right). \quad (\text{A3})$$

Making use of Eq. (A1), the relative magnitude of the baroclinic to dilational terms is

$$\frac{\left\| \frac{\nabla \rho \times \nabla p}{\rho^2} \right\|}{\|-\boldsymbol{\omega}(\nabla \cdot \mathbf{u})\|} \sim O\left(\frac{|c|}{|\Delta u_a|}\right) = O\left(\frac{|\rho|}{|\Delta \rho_a|}\right). \quad (\text{A4})$$

To evaluate the above expressions for comparison with our computational results, we consider our base trapezoidal wave case where $p_a = \Delta p_a = 10$ MPa. The length scale associated with the acoustic wave is the initial length of the pressure compression $\Delta l_a = 5\ell$. The initial interface length scale ΔL_i , defined as the thickness of the mixed layer from $\alpha = 0.05$ to 0.95 volume fraction of water, is estimated as $\Delta L_i \approx 0.05\ell$. We approximate the order of θ based on its average value along a half-wavelength of the interface for $a_0 = 0.03\ell$ such that the average value of $|\theta| \approx 0.12$. Evaluating the expression (A4) we find that $|c|/|\Delta u_a| = O(10^2)$ and thus expect that the relative contribution of baroclinic to compressible or advective vorticity generation is approximately $O(10^2)$ at $t/(\ell/c) = \Delta t_a \approx 5$, near the end of the compression.

To compare our computational results to the analysis we consider the integral of the vorticity and vorticity generation terms over the right-half domain

$$\Gamma = \int_{A_{rh}} \omega dA_{rh}, \quad (\text{A5})$$

where $\int_{A_{rh}} dA_{rh} = \int_{-\infty}^{+\infty} \int_{\ell/2}^{\ell} dy dx$. Only the right-half domain is considered because the total circulation over the whole domain is zero due to symmetry. Circulation is chosen as the quantity of comparison as it is a global quantity, which better captures the overall vorticity dynamics than the vorticity at any single point. As a single quantity rather than a field, it is also simpler to compare the computational and analytical results. The relative order of magnitude relationships obtained in (A4) are spatially independent and expected to hold when integrated over the right-half domain. Accordingly, we evaluate the vorticity generation terms from our computational results integrated over the right-half domain. At $t \approx 5.0$ we find that

$$\frac{\int_{A_{rh}} \left[\frac{\nabla \rho \times \nabla p}{\rho^2} \right] dA_{rh}}{\int_{A_{rh}} [-\boldsymbol{\omega}(\nabla \cdot \mathbf{u})] dA_{rh}} = O(10^2). \quad (\text{A6})$$

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