Effect of Overpressure on Dissolution and Cavitation of Bubbles Stabilized on a Metal Surface

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Abstract: Recent in vitro results[1,2] show that 1) more overpressure (30 atm vs. 3 atm) is needed to suppress damage to solids such as aluminum foil than to suspended cells and 2) that modest overpressure (10 atm) in fact increases damage to foil. We propose that result 1 arises because bubbles in the free field of the cell suspension are driven into solution by the overpressure whereas bubbles trapped in crevices on the foil are much more difficult to dissolve. Calculations of dissolution rates and bubble imaging by diagnostic ultrasound of systems both with and without applied overpressure support our hypothesis. We contend that result 2 occurs because the cavitation bubbles excited from the stabilised nuclei are driven to a more violent collapse by the overpressure. Numerical simulations of the amplitudes of the shock waves radiated by stabilised bubbles subject to lithotripsy waveforms show the same dependence on overpressure as the foil damage.

INTRODUCTION

Experimentation with in vitro systems has implicated cavitation as a mechanism by which lithotripsy shock waves cause stone comminution and cell damage. One means to suppress cavitation in an in vitro system is to apply overpressure (increase hydrostatic pressure) which dissolves the cavitation nuclei. Delius [1] observed that the overpressure to reduce cell lysis (1 atm) was significantly lower than that need to prevent gall stones from breaking (100 atm). In similar experiments, McAteer et al. [2] also found that a few atmospheres of overpressure protected red blood cells or kidney proximal tubule LLC-PK1 cells, but that 30 atm was necessary to prevent damage to foil targets. In addition, McAteer et al. found damage to foils was greater at 10 atm overpressure than no overpressure (ambient atmospheric pressure).

RESULTS

Diagnostic ultrasound images of the vials (polyethylene pipette bulbs) used for cell experiments indicated that overpressure dissolved cavitation nuclei. Figure 1 shows a sequence of B-scan frames taken of a vial filled with phosphate buffered saline (PBS; the same buffer used for cell studies in Ref. 2), during exposure to lithotripsy shock waves. In the left sequence the vial was at atmospheric pressure and the inside of the vial became echogenic after passage of a shock wave. Echogenicity indicates the presence of many gas bubbles left over from cavitation generated by the shock wave. These remnant bubbles will be nuclei for subsequent shock waves. In the right sequence the vial was pressurised and the echogenic regions were extinguished almost immediately, indicating that bubbles were quickly dissolved.

![Figure 1. B-scan of saline during SWL. On the first frame the outline of the vial is indicated, and an artifact from the shock wave appears. Left column: vial with no overpressure, the saline remained echogenic (arrows) for many seconds. Right column: vial subject to 1 atm of overpressure; there was virtually no echogenicity in the vial.](image-url)
Our companion study [2] reports that foils suffered damage up to 30 atm. We hypothesise that the surface of the foil stabilised cavitation nuclei, i.e., prevented the overpressure from eliminating small gas bubbles. The “crack and crevice” model [3] is the standard model to describe the presence of cavitation nuclei on surfaces. The adhesive forces associated with crevices protect small gas bubbles from dissolving. Ultrasound images of vials with foil could not be interpreted because reflection from the foil dominated the image. However, pitting on the foil at low overpressures indicated that cavitation occurred and a likely explanation is that nuclei were protected in surface cracks.

The second phenomenon observed experimentally was increased foil damage at moderate overpressure, of the order 10 atm. Because nuclei are stabilised on the foil, cavitation bubbles will grow after the passage of a lithotripsy shock wave in a manner similar to bubbles at atmospheric pressure. However, the collapse of the bubble, which is driven by the ambient pressure, will be accelerated because of the overpressure. The final collapse of the bubble will therefore be much more violent than occurs with no overpressure. Numerical simulations of stabilised bubbles subjected to lithotripsy waveforms were carried out. Bubble dynamics were modeled with the Gilmore-Akulichev formulation described by Church [4], with the exception that diffusion through the bubble wall was neglected. The shock wave was chosen to correspond to measured waveforms: peak positive pressure of 40 MPa and peak negative pressure of -6 MPa; all other parameters were identical to those used by Church.

In our calculations the ambient pressure of the system was varied from 1 atm to 70 atm. For overpressures in excess of 60 atm (6 MPa) the ambient pressure exceeded the peak negative pressure of the shock wave and no bubble growth was expected. In the calculations the initial bubble radius \( R_0 \) was fixed, i.e., we assumed that nuclei were perfectly stabilised. At each overpressure the response of the bubble was calculated and the maximum pressure radiated \( P_{rad} \) by the bubble in the fluid was determined at a distance of 5 mm from the bubble. The calculations were repeated for three initial bubble radii: 0.1, 1.0, and 10 \( \mu \)m. It is assumed that \( P_{rad} \) is related to the violence of the cavitation collapse. Figure 2 shows a plot of \( P_{rad} \) as a function of overpressure; superimposed is the data for foil damage. For all initial radii, \( P_{rad} \) increased with overpressure until about 30 atm and then reduced to the cut-off pressure at 60 atm. The discrepancy in the details of the experimental and numerical results arise because the model is highly idealised, however, the qualitative agreement is excellent. In addition, the calculations predicted that the collapse time of the bubble would reduce with overpressure, because the bubble had less time to grow and was more quickly crushed. Measurements using passive cavitation detection with this in vitro system at low overpressures (less than 5 atm) confirmed this prediction.

**CONCLUSIONS**

Overpressure reduced injury to cells in fluid suspension because cavitation nuclei were dissolved. However, it appeared that cracks and crevices at the surface of foil targets stabilised cavitation nuclei preventing nuclei from being crushed by the overpressure. Increased damage to foils at 10 atm occurred because the added hydrostatic pressure drove inertial cavitation bubbles into more violent collapse. For higher overpressures there was not enough negative pressure in the shock wave to make the bubbles grow significantly. We speculate that if bubbles were to be stabilised at the surface of kidney stones, then overpressure could yield more effective stone fragmentation. We gratefully acknowledge the support of The National Institutes of Health through grant PO1-DK43881.

**REFERENCES**