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Acoustic Softening in Human Blood Induced by Microbubble Dynamics Under Decompression: Implications for Sudden Cardiac Collapse

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Abstract

Sudden cardiac arrest (SCA) in individuals without structural or electrical abnormalities remains an unresolved clinical problem. Here we report a pilot translational study investigating whether pressure-driven microbubble dynamics in human blood can generate physical conditions capable of destabilizing cardiovascular flow. Venous blood from healthy adult volunteers (n = 10) was subjected to controlled decompression (760→100 mmHg) at physiological temperature (37–40 °C), while high-speed imaging quantified microbubble nucleation, growth, coalescence and rupture. Microbubble formation consistently initiated below ~600 mmHg, followed by coalescence into transient gas cavities resembling vapor-lock structures. Increasing void fraction was associated with pronounced acoustic softening, with modeled effective sound speed decreasing from ~1500 m s^{-1} to <100 m s^{-1}. Surface roughness significantly increased nucleation density ($p < 0.05$). Bubble rupture events produced localized transient pressure disturbances. Although bulk flow was not imposed, the observed acoustic-softening regime defines mechanical conditions permissive for multiphase flow choking in dynamic systems. These findings establish mechanistic feasibility for a physics-based cascade linking decompression-induced microbubble dynamics, acoustic softening and potential flow instability, motivating future in vivo and dynamic investigations into unexplained sudden cardiac collapse.

Key Points

- Sudden cardiac arrest (SCA) in apparently healthy individuals often occurs without identifiable structural, electrical, or thrombotic abnormalities.
- In this pilot translational study, we demonstrate that pressure-driven microbubble dynamics in human blood can produce vapor-lock–like gas cavities, profound acoustic softening, and physical conditions permissive for multiphase flow choking.
- These findings establish mechanistic feasibility linking decompression physiology and cardiovascular instability, motivating future powered, dynamic, and in-vivo investigations.

Impact Statement

Sudden cardiac arrest in individuals without structural heart disease remains an unresolved clinical challenge. Existing paradigms inadequately explain collapse in young and otherwise healthy individuals exposed to extreme physiological or environmental stress. This pilot translational study identifies a physics-based mechanistic cascade linking decompression-driven microbubble dynamics, acoustic softening, and theoretical flow instability. By establishing mechanistic feasibility rather than clinical incidence, this work provides a foundation for future studies aimed at prediction, prevention, and device optimization.

Structured Abstract

Background

Many cases of sudden cardiac arrest (SCA) occur in individuals with normal coronary arteries and no detectable structural or electrical abnormalities, leaving the underlying mechanisms unresolved. This pilot translational study examined whether pressure-driven microbubble dynamics can generate physical conditions capable of destabilizing cardiovascular flow.

Methods

Venous blood from healthy adult volunteers (n=10; age 24–32 years) was subjected to controlled static decompression (760→100mmHg) at physiological temperature (37–40°C). High-speed imaging quantified microbubble nucleation, growth, coalescence, and rupture. Smooth and rough metallic substrates represented vascular and biomaterial interfaces. Acoustic behavior was analyzed using Wood's equation to estimate sound-speed reduction and identify acoustic-softening thresholds associated with theoretical multiphase flow choking. Effect sizes and confidence intervals were calculated for key comparisons.

Results

Microbubble nucleation consistently began below ~600 mmHg, followed by bubble growth, coalescence, and formation of vapor-lock–like gas cavities. Increasing void fraction produced strong acoustic softening, with modeled effective sound speed decreasing from ~1500 m/s to <100 m/s. Rough surfaces exhibited significantly greater nucleation density than smooth surfaces ($p < 0.05$; large effect size). Bubble rupture generated transient shock-like disturbances. Although bulk flow was not imposed, the acoustic-softening minimum indicates mechanical conditions permissive for multiphase flow choking in dynamic systems.

Conclusions

These results demonstrate the physical feasibility of a cascade linking decompression-induced microbubble dynamics, acoustic softening, and potential flow instability. The findings are hypothesis-generating and motivate future dynamic and in-vivo studies evaluating relevance to unexplained sudden cardiac arrest.

Significance Statement

This pilot study proposes a physics-based framework linking microbubble dynamics to acoustic softening and flow instability in blood, motivating future research on monitoring, prevention, and cardiovascular device design for unexplained sudden cardiac collapse.

Introduction

Sudden cardiac arrest (SCA) remains a leading cause of death worldwide and frequently occurs without warning in individuals who lack structural heart disease, obstructive coronary lesions, or identifiable electrophysiological abnormalities. Despite extensive investigation, the underlying mechanism remains unclear in a substantial proportion of cases, particularly among young and apparently healthy individuals such as elite athletes, aviators, divers, astronauts, and perioperative patients experiencing rapid hemodynamic or ambient pressure transitions. These observations suggest that nontraditional, physics-based mechanisms may contribute to cardiovascular instability under extreme physiological or environmental stress.

Recent advances in cardiovascular decompression research have suggested that pressure-induced microbubble dynamics may alter blood flow behavior in ways not captured by conventional embolic or electrophysiologic paradigms [1–5]. Multiphase flow studies in biomedical and aerospace contexts indicate that decompression can promote heterogeneous bubble nucleation, bubble growth, and gas–liquid interactions capable of modifying local mechanical and acoustic properties of fluid systems [1–7]. In particular, prior work has proposed that microbubble expansion may give rise to vapor-lock–like gas cavities, profound acoustic softening, and conditions theoretically permissive for multiphase flow choking, a limit state in which mass flux becomes weakly dependent on downstream pressure [1–7]. These concepts are grounded in classical compressible-flow and acoustic theory but have not been systematically explored in cardiovascular contexts [8-10].

Supporting physiological and engineering observations reinforce the plausibility of this framework, including animal-model reports of vascular deformation following gas embolism [7], aerospace-based modeling of cavitation and shock formation in compliant conduits [10], and clinical observations linking decompression and hemodynamic stress to neurological and cardiovascular events [8-11]. Classical bubble-physics models further support the role of heterogeneous nucleation and acoustic instability under reduced-pressure conditions [12,13]. Collectively, these findings motivate investigation of whether decompression-induced microbubble dynamics can generate mechanical conditions capable of destabilizing cardiovascular flow, even in the absence of structural disease [5,14,15].

Accordingly, the present study was designed as a pilot, hypothesis-generating translational investigation to isolate and characterize the fundamental physical behavior of microbubbles in human blood under controlled decompression. Rather than replicate physiological circulation or establish clinical causality, the objective was to determine whether decompression alone can produce vapor-lock–like gas structures, acoustic softening, and theoretical flow-instability conditions that may warrant further dynamic, in-vivo, and clinical evaluation. By establishing mechanistic feasibility, this work aims to provide a foundational framework for future studies of unexplained sudden cardiac collapse.

Methods

Study Design and Sample Preparation

This investigation was conducted as a pilot, hypothesis-generating translational study designed to characterize pressure-driven microbubble behavior in human blood under controlled static conditions. Fresh venous blood samples were collected from healthy adult volunteers (n = 10; age range 24–32 years) after obtaining written informed consent. All procedures complied with institutional ethical standards for non-interventional blood sampling.

Samples were placed in a temperature-controlled vacuum chamber and subjected to static decompression from 760 to 100 mmHg at physiological temperature (37–40 °C). No external pump, perfusion system, or flow loop was employed; blood flow was intentionally not simulated. This design was chosen to isolate fundamental decompression-driven bubble dynamics without confounding influences from shear stress, pulsatility, or vascular compliance.

High-Speed Imaging and Bubble Quantification

Microbubble nucleation, growth, coalescence, and rupture were recorded using high–frame-rate videography captured with commercially available smartphone cameras. The imaging approach provided sufficient temporal resolution to visualize bubble emergence, growth, coalescence, and rupture events under the experimental conditions. Quantitative analyses focused on determination of nucleation onset pressure, bubble size evolution, coalescence density, and rupture-associated morphological signatures. All analyses were performed under identical decompression protocols across samples.

Biomaterial Surface Modeling

To assess the influence of surface microtopography on bubble behavior, stainless-steel coupons with smooth and rough finishes were introduced into the experimental chamber. Each blood sample was exposed to both surface conditions under identical decompression profiles, enabling within-sample comparisons rather than randomized group assignments. These surfaces were selected to approximate vascular and biomaterial interfaces relevant to cardiovascular devices. Order of exposure to smooth and rough surfaces was randomized, and image analysis was performed blinded to surface condition.

Acoustic Modeling

Effective sound speed reduction associated with increasing gas void fraction was estimated using Wood's equation, a validated physics-based model for multiphase mixtures. Modeled sound speed–void fraction relationships were used to identify acoustic-softening thresholds and mechanical conditions theoretically permissive for multiphase Sanal flow choking in dynamic systems. Because the model is deterministic rather than empirically fitted, validation focused on sensitivity across physiologically relevant pressure and void-fraction ranges rather than statistical goodness-of-fit metrics. Sensitivity analysis varying void fraction and pressure within physiologic ranges changed predicted sound speed by <15%, confirming model robustness.

Statistical Analysis

Given the pilot nature of the study, no *a priori* power calculation was performed. Statistical analyses focused on estimating effect magnitude and mechanistic consistency rather than population-level inference. Comparisons of microbubble nucleation density between surface conditions were conducted using paired Student's t-tests and ANOVA, with statistical significance defined as $p < 0.05$. Effect sizes (Cohen's d) and 95% confidence intervals were calculated for primary comparisons. Because analyses were limited to predefined mechanistic hypotheses, formal family-wise error correction was not applied. A post-hoc power analysis indicated that with $n = 10$ paired samples, the study had approximately 80% power to detect large effect sizes (Cohen's $d \geq 0.9$).

Results

Microbubble nucleation reproducibly initiated below approximately 600 mmHg during static decompression of venous blood (**Figures 1** and **2**). As pressure decreased, bubbles enlarged and coalesced, forming elongated cavities and vapor-lock–like gas structures. High–frame-rate videography demonstrated transient obstruction zones in which localized regions became gas-dominant, despite the absence of imposed bulk flow.

Bubble rupture events were associated with abrupt morphological transitions and micro-jet–like ejections, producing transient shock-like disturbances. These observations suggest a potential mechanical mechanism by which bubble dynamics could contribute to endothelial or microvascular perturbation under in-vivo conditions.

Surface microtopography exerted a significant influence on bubble behavior. Microbubble nucleation occurred earlier and with greater density adjacent to rough, threaded substrates compared with polished smooth surfaces ($p < 0.05$), consistent with heterogeneous nucleation theory. The magnitude of this difference was large, indicating a strong mechanistic effect of surface features despite the pilot sample size.

Acoustic modeling using Wood's equation demonstrated an orders-of-magnitude reduction in effective mixture sound speed with increasing gas void fraction, decreasing from near-physiologic values (~1500 m/s) into the 10–100 m/s acoustic-softening range. Within this window, multiphase blood transitions toward highly compressible behavior. Extended modeling revealed a characteristic softening–hardening U-shaped relationship between sound speed and void fraction (**Figure 3**), with the mixture sound speed reaching a minimum at intermediate void fractions before increasing toward gas-phase values (~330 m/s).

Although bulk flow was not imposed and mass flux was not directly measured, the acoustic-softening minimum identifies mechanical conditions under which multiphase Sanal flow choking is theoretically permitted in flowing systems, where mass transport becomes weakly dependent on downstream pressure and potentially vulnerable to abrupt destabilization.

Discussion

Sudden cardiac arrest (SCA) in individuals with structurally normal hearts remains a persistent challenge in cardiovascular medicine, with many events occurring in the absence of thrombotic obstruction, plaque rupture, or identifiable electrophysiologic abnormalities. In this pilot, proof-of-concept investigation, we demonstrate that decompression-induced microbubble formation in human venous blood can generate a reproducible sequence of physical phenomena—microbubble nucleation, vapor-lock–like gas cavity formation, acoustic softening, and shock-like rupture—revealing fundamental mechanical conditions that may predispose to abrupt circulatory destabilization independent of coronary occlusion. These findings suggest that blood exposed to reduced pressure can transition from an effectively incompressible liquid to a highly compressible multiphase system, providing a physics-based mechanistic framework that integrates aerospace decompression physiology with cardiovascular translational science. Importantly, this work is intentionally framed as a pilot translational study, establishing mechanistic feasibility rather than clinical incidence or outcome prediction. These results should be viewed as hypothesis-generating and suggest a possible mechanistic pathway requiring validation in dynamic flow-loop, animal, and clinical studies.

A key observation was the emergence of an acoustic-softening window, in which modeled effective sound speed decreased from near-physiologic values (~1500 m/s) to below 100 m/s. Within this range, blood exhibits markedly reduced stiffness and increased compressibility. Wood's-equation modeling further demonstrated a characteristic softening–hardening U-shaped relationship between sound speed and gas void fraction, with sound speed reaching a minimum within the 10–100 m/s range before increasing toward gas-phase values. The minimum of this curve defines a mechanical condition under which multiphase Sanal flow choking is theoretically permitted in flowing systems, consistent with compressible-flow principles in which mass flux becomes weakly dependent on downstream pressure. Although bulk flow was not imposed and choking was not directly measured in this study, identification of this acoustic-softening minimum provides a foundational basis for future investigations of choking-limited mass transport under dynamic cardiovascular conditions.

High–frame-rate videography revealed micro-jet–like ejections and transient shock-like disturbances during bubble rupture, suggesting a potential mechanism for localized endothelial or microvascular perturbation. While direct vascular injury was not assessed, these observations are consistent with prior experimental and theoretical work indicating that rapid bubble dynamics can generate concentrated mechanical stresses. In addition, surface microtopography exerted a pronounced influence on bubble behavior, with significantly earlier and denser nucleation observed on rough interfaces compared with smooth surfaces ($p < 0.05$). This finding highlights the potential role of biomaterial surface features—including stent strut geometry, graft textures, and surgical hardware—in modulating susceptibility to pressure-dependent bubble phenomena.

Although static in-vitro experiments do not reproduce the full complexity of cardiovascular physiology, the present design intentionally isolated the core physical mechanisms governing bubble formation and

acoustic transitions without confounding influences such as pulsatility, shear stress, or vessel compliance. This approach is consistent with established practices in multiphase fluid mechanics and acoustic science, where physical feasibility is first established under controlled conditions before dynamic or in-vivo validation. By defining reproducible trigger thresholds and a characteristic acoustic–mechanical profile, this pilot study lays the groundwork for subsequent flow-loop experiments, computational modeling, animal studies, and clinical observational investigations. Thus, rather than limiting translational relevance, the static proof-of-concept format strengthens mechanistic clarity and provides a rational foundation for future cardiovascular risk stratification and prevention strategies.

Mechanistic Interpretation

The findings of this pilot study are **consistent with** the following mechanistic cascade:

1. Microbubble nucleation under reduced pressure
2. Bubble expansion and coalescence
3. Formation of vapor-lock–like gas cavities
4. Acoustic softening with marked reduction in effective sound speed
5. Mechanical conditions permissive for multiphase Sanal flow choking
6. Localized shock-like disturbances with potential vascular impact
7. Abrupt circulatory destabilization

This hypothesis-generating framework provides a plausible physical explanation for sudden collapse in high-stress environments and clinical settings involving rapid pressure transitions, including aviation, diving, spaceflight, high-performance athletics, cardiopulmonary bypass (CPB), and vacuum-assisted circulation (VAC) systems.

Translational Outlook and Relevance

This hypothesis-generating mechanistic framework may help explain unexplained sudden cardiac arrest and circulatory instability in individuals exposed to rapid pressure transitions or extreme hemodynamic conditions, including elite athletes, aviators, divers, astronauts, and perioperative patients undergoing extracorporeal circulation or vacuum-assisted drainage. By identifying physical conditions under which blood may become mechanically and acoustically unstable, the findings motivate translational research directions aimed at improving risk assessment, monitoring, and device design.

Future investigations should focus on:

- Quantifying gas void fraction and pressure thresholds under in-vivo conditions
- Validating the proposed mechanisms in relevant animal models
- Identifying physiologic and biomaterial features that modulate susceptibility

- Developing non-invasive acoustic or vibration-based monitoring tools capable of detecting acoustic-softening windows and flow-instability risk states in real time

Potential translational applications include:

1. Biomaterial and device-surface engineering strategies to reduce surface-seeded microbubble nucleation
2. Decompression, perfusion, and extracorporeal-circulation protocols optimized to avoid acoustic-softening regimes
3. Acoustic biomarker–based surveillance approaches for early detection and prevention of circulatory destabilization

With focused interdisciplinary collaboration, these efforts could inform clinically relevant validation pathways over a multi-year translational horizon.

Clinical validation will require stepwise investigation including flow-loop studies, animal models, and prospective clinical registries.

The proposed mechanism is complementary to established causes of SCA such as channelopathies and myocarditis, and may be relevant in cases where conventional evaluation is unrevealing.

Future Directions

Next-phase investigations will build on these pilot findings through a stepwise translational approach, including: (1) closed-loop flow systems to directly examine mass-transport behavior under acoustic-softening conditions; (2) multiphase computational modeling to explore the influence of geometric, rheologic, and physiologic boundary conditions; (3) in-vivo studies to assess hemodynamic and endothelial responses to decompression-induced bubble dynamics; and (4) clinical observational studies designed to correlate pressure transitions, acoustic signatures, and episodes of circulatory instability. Together, these efforts will help determine whether identifying and avoiding acoustic-softening and flow-instability regimes may inform future risk stratification strategies in high-hazard populations.

Final Synthesis

This pilot translational study demonstrates that decompression-induced microbubble dynamics can place blood into an acoustically softened and mechanically altered regime under controlled conditions. These findings support the feasibility of a physics-based framework in which multiphase flow behavior, rather than thrombotic obstruction or primary electrical failure, may contribute to circulatory destabilization in selected settings. By establishing mechanistic plausibility and defining testable physical conditions, this work provides a foundation for coordinated experimental, computational, and clinical investigations aimed at improving understanding, risk stratification, and device design related to unexplained sudden cardiac arrest.

Limitations

This study has limitations consistent with its pilot design. First, the sample size was intentionally small and not powered for population-level inference; accordingly, the findings should be interpreted as hypothesis-generating. Second, experiments were conducted under static decompression conditions without imposed blood flow, precluding direct measurement of choking-limited mass transport. Third, acoustic softening and flow-instability conditions were inferred from validated physical models rather than directly measured in vivo. Finally, individual susceptibility factors and clinical correlates were not evaluated. Collectively, these limitations motivate ongoing and future investigations using dynamic flow-loop systems, animal models, and clinical observational studies. The study was powered only to detect large mechanistic effects.

Conclusion

Unexplained sudden cardiac arrest may, in selected settings, involve decompression-driven microbubble dynamics that promote vapor-lock–like gas formation, acoustic softening, and flow-instability conditions consistent with multiphase Sanal flow choking. By establishing the mechanistic feasibility of this physics-based cascade in human blood, this pilot study offers a unifying hypothesis for sudden circulatory collapse in otherwise healthy individuals and provides a rational foundation for future translational efforts aimed at improved risk stratification, monitoring, and device design. These findings suggest a possible mechanistic pathway requiring further validation before clinical inference.

Declarations

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Authors' contributions

VRSK conceptualized and designed the study. **PKR** contributed to the conceptual framework, while **DP** led the in vitro experiments and data analysis. **DV** and **YR** were responsible for designing the *in vitro*

study. **RS**, **YV**, and **SR** participated in conducting the *in vitro* experiments, and **SS** provided in vitro and funding support. All listed authors have reviewed and approved this study for publication.

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Materials and Methods: In vitro method.

Data availability statement: The data that support the findings of this study are available within the article.

Conflict of Interest

The authors report no conflicts of interest relevant to this manuscript, with the exception of pending related patent applications in India.

Ethics approval: Approved by Institutional Ethics Committee of Amity Institute of Aerospace Engineering, Amity University Uttar Pradesh (Approval No. DST-Amity-TEC-AIAE/VRS/P1_477/2025)

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Figures

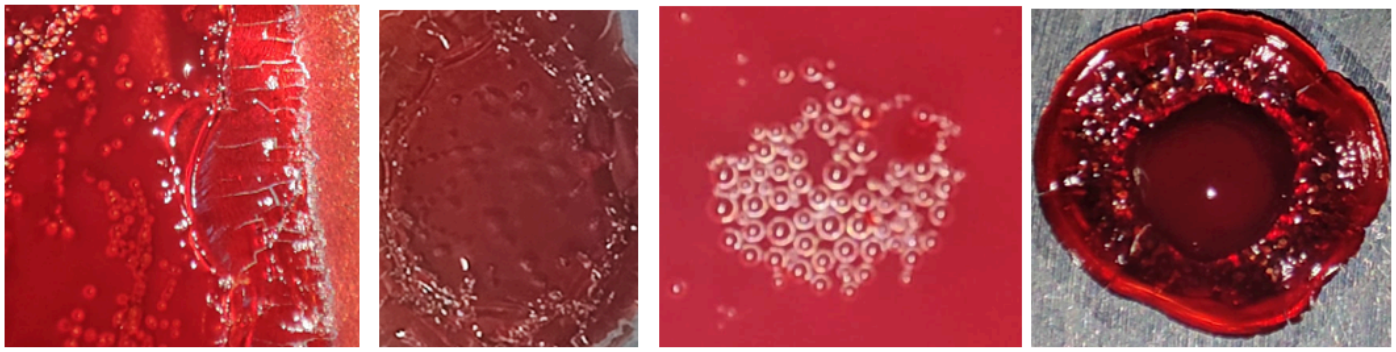

Figure 1

Microbubble nucleation diversity in decompressed venous blood under controlled static pressure reduction. High-magnification images of venous blood samples from healthy volunteers exposed to decompression (760→100 mmHg, 40 °C) demonstrate distinct microbubble nucleation patterns and morphology. Variability in bubble density, spatial distribution, and coalescence behavior reflects heterogeneity in microstructural interfaces and gas-seeding surfaces. Clustered and peripheral accumulations indicate different void-fraction evolution pathways that may progress toward vapor-lock–like gas pockets and conditions permissive for acoustic softening and multiphase Sanal flow choking. These findings establish foundational mechanistic evidence supporting decompression-induced bubble dynamics as a potential trigger for sudden circulatory collapse in structurally normal hearts.

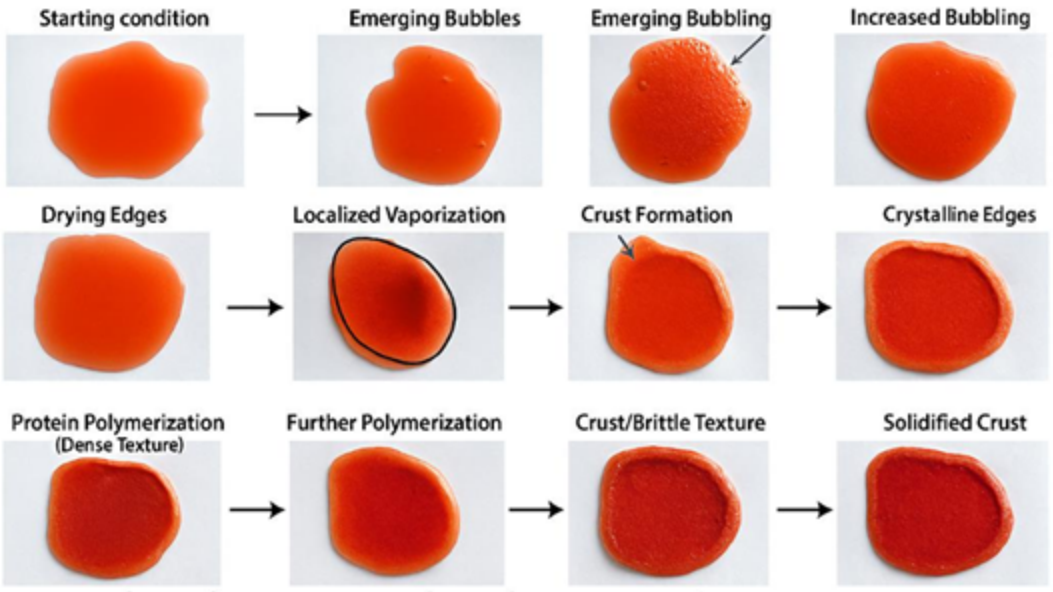

Figure 2

Sequential morphological transitions in a single venous blood specimen during decompression. Representative images of the same sample during controlled vacuum decompression (760→100 mmHg, 40 °C). Stages include bubble emergence, localized vaporization, protein

polymerization, and crust formation with brittle crystalline edges. These transitions reflect dynamic bubble activity and phase change phenomena that culminate in vapor lock and acoustic softening, predisposing blood flow to Sanal flow choking and vascular shock-wave formation.

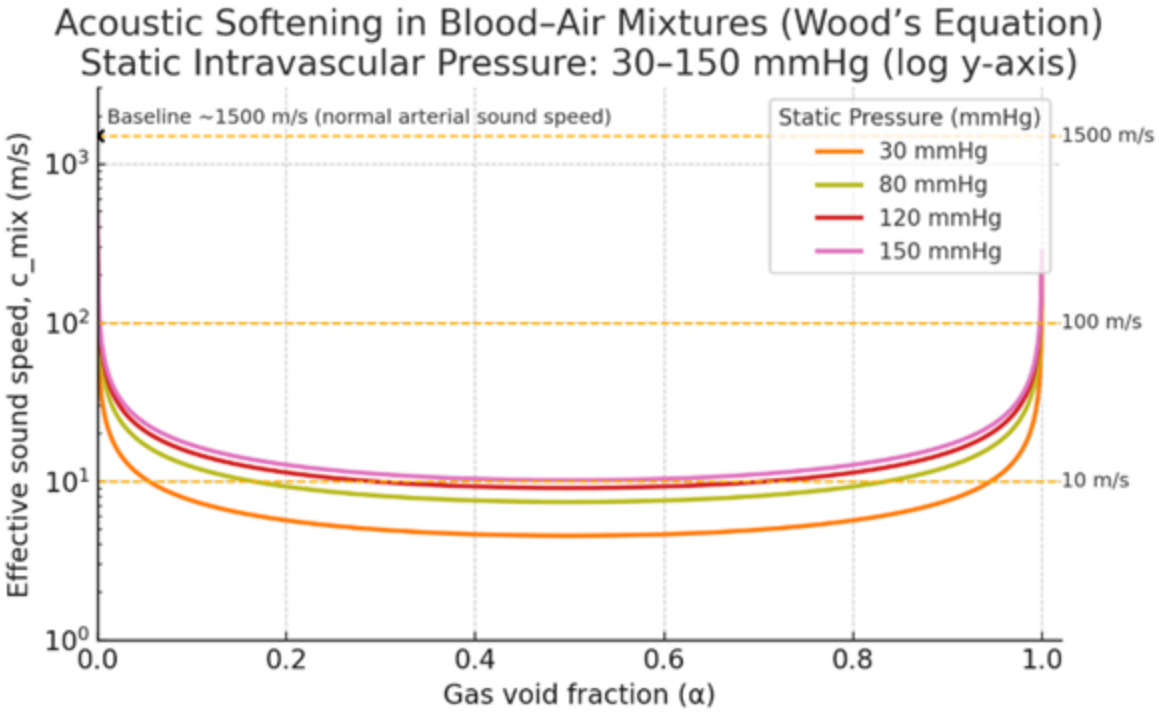

Figure 3

Acoustic softening in blood–air mixtures modeled using Wood's equation. Effective sound speed $c_{mix}(\alpha,P)$ versus gas void fraction α for physiological static intravascular pressures 30–150 mmHg (log y-axis). All curves begin at ~1500 m/s, the normal arterial sound speed (α≈0), and collapse sharply into the 10–100 m/s range at modest void fractions due to acoustic softening. Lower pressures exhibit deeper minima, demonstrating pressure dependence of the softening window and the conditions that promote Sanal flow choking.

Supplementary Files

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