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Reply To Van Liew And Vann "Sonic Echocardiography: What Does It Mean When There Are No Bubbles In The Left Ventricle?"

Jonathan E. Elliott
*University of Puget Sound*, jkelliott@pugetsound.edu

Yujung Choi
*University of Puget Sound, Tacoma, Washington*

Steven S. Laurie
*Department of Human Physiology, University of Oregon, Eugene, Oregon*

Ximeng Yang
*Department of Human Physiology, University of Oregon, Eugene, Oregon*

Igor M. Gladstone
*Department of Human Physiology, University of Oregon, Eugene, Oregon; Department of Pediatrics, Oregon Health and Science University, Portland, Oregon*

See next page for additional authors

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Reply to Van Liew and Vann

Jonathan E. Elliott,1 Yujung Choi,2 Steven S. Laurie,1 Ximeng Yang,1 Igor M. Gladstone,1,3 and Andrew T. Lovering1

1Department of Human Physiology, University of Oregon, Eugene, Oregon; 2University of Puget Sound, Tacoma, Washington; and 3Department of Pediatrics, Oregon Health and Science University, Portland, Oregon

TO THE EDITOR: Van Liew and Vann (19) present several intravascular gas diffusion hypotheses and suspicions based on simulation data that they admit were indirectly tested and only validated by extravascular observations (1). The authors do acknowledge that “Nonbubble methods convince us that pulmonary shunts exist . . .” (19). Listed below are the data obtained using nonbubble methods.

Microspheres (25 μm) traverse the pulmonary circulation during exercise but not at rest in dogs (16). Likewise, the percentage of albumin microspheres and macroaggregates that traverse the pulmonary circulation increases during exercise in healthy humans (7, 20), all consistent with human bubble method data (3–5, 8, 9, 17). The transpulmonary passage of microspheres (60–420 μm) increases under hypoxic conditions and decreases under hyperoxic conditions in dogs (13), all consistent with human bubble method data (4, 6, 8, 9).

Van Liew and Vann ask “What if bubbles appear in the left ventricle?” (19) and propose the following.

“1) If they are larger than capillaries, they may have come through arteriovenous shunts” (19). We (and others) have come to the same conclusion based on the data listed above (3, 4, 17).

“2) If they are small, bubbles may have come through lung capillaries” (19). We respectfully suggest that this is an inappropriate interpretation. Mean pulmonary artery driving pressure during maximal exercise is 20–30 Torr (14), and previous work demonstrated the requirement of a 300-Torr pressure to force bubbles through capillaries using a firmly wedged right heart catheter (15). Van Liew and Vann speculate further: “If the blood’s passage is more rapid, as in exercise, the small bubbles may not be completely absorbed, so the inappropriate interpretation would be that there are open anastomoses” (19). We disagree. Bubbles get through intrapulmonary arteriovenous anastomoses during exercise after three cardiac cycles. With a heat rate of 180, this requires a minimum of 1 s for bubbles to traverse the pulmonary circulation. An 8-μm bubble has a life span of ~200 ms (11). Because bubble dissolution time decreases with increased pressures and flows (18, 21–23), we respectfully suggest that the inappropriate interpretation is that exercise results in conditions favoring the presence of bubbles in the left ventricle. Rather, the data suggest that exercise results in conditions least favorable for bubbles (2, 10, 12, 15).

Van Liew and Vann also propose “3) . . . it is possible that as captured bubbles shrink they may escape and pass on into the left ventricle, leading to an interpretation that shunts are open, even if there are no shunts” (19). We direct the reader to the nonphysiological conditions and physical constraints listed above, which would prevent this from occurring.

Van Liew and Vann ask, “What if bubbles do not appear in the left ventricle?” (19) and our arguments above and below address this.

Last, Van Liew and Vann ask, “Why are no bubbles seen when exercising subjects breathe 100% oxygen?” (19). They reply, “The strong potential for absorption due to lack of nitrogen in the pulmonary capillaries, pulmonary veins, and left atrium makes it likely that if they pass through the lung by any route—anastomoses, capillaries, or being freed from precapillary capture—bubbles will be absorbed before they reach the left ventricle” (19). We disagree: we do see bubbles in subjects breathing 100% O2 during exercise, just not as many (4, 9), consistent with microsphere data (13).

When in vivo data are not consistent with in silico data, we respectfully suggest that the objective should be to “rethink” the modeling data, not vice versa. The authors suggest that bubbles made of SF6 may shed some light on this topic (19). We encourage them to perform these experiments to directly test their in silico data, and we look forward to reading the results. An equally constructive experiment would be to determine if saline bubbles could be detected in the left heart of subjects with pulmonary arteriovenous malformations breathing hyperoxia.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

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