IT'S TOO EARLY FOR THE SHUNT DEBATE
TO THE EDITOR: The issue whether shunt does (2) or does not (1) develop during exercise and whether shunt is responsible for the increased \( A_aD O_2 \) seen in many people during exercise is developing into a methodological debate that will not be solved with the information available at this time. We do not know the in vivo bubble sizes in saline contrast echocardiography nor do we know the influence of pre-capillary gas exchange on the multiple inert gas elimination technique. The original study demonstrating shunt during exercise (3) contained one interesting subject who was a healthy and very active person but, in the several studies in which he participated, he often had the lowest \( VO_2_{max} \), despite regular training. In Dr. Stickland's study he was the only subject, out of eight, who did not demonstrate shunt and he had the lowest values for cardiac output and \( VO_2_{max} \), he had the highest pulmonary artery pressure, and he did not have an increase in \( A_aD O_2 \) during exercise. In my opinion, he perfectly demonstrates how absence of shunt during exercise can be detrimental to achieving high \( VO_2_{max} \), for without shunt, cardiac output seems to be constrained. A study comparing two groups using saline contrast echocardiography is needed: one group with increased \( A_aD O_2 \) during exercise and the other without an increase in \( A_aD O_2 \). If the latter group behaved like the single subject in Dr. Stickland’s study and the other group shunted then I think the evidence for shunt, and its importance during exercise, would be strengthened.

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REAL SHUNTS—UNPROVEN EFFECT ON GAS EXCHANGE
TO THE EDITOR: Hopkins et al. (2) leave it to the reader to decide if microbubble transmission really implies a shunt. Studies that have used the agitated saline contrast echocardiography method show that these vessels can open during exercise in some, but not all, subjects. Anatomic approaches using isolated human, baboon, and dog lungs show that solid microspheres can pass through intrapulmonary arteriovenous pathways (3). It is clear from several lines of evidence that shunting is indeed “real” (5). The salient question is what effect does shunting have on gas exchange during exercise? The answer likely depends on who is exercising and at what intensity. Shunts have been reported during submaximal exercise when gas exchange impairment is minimal or not present (1). Might the recruitment of shunts and increases in pulmonary vascular pressures partially explain the excessive widening of the \( A_aD O_2 \) seen in some endurance athletes? The magnitude of the \( A_aD O_2 \) and pulmonary arterial pressure during exercise has been shown to correlate with the presence of intrapulmonary shunts (6). However, it is important to recognize the methodological shortcomings of the saline contrast echocardiography technique. Specifically, it remains a qualitative measure (i.e., shunt vs. no shunt) despite attempts at developing a scoring system (4). While Lovering et al. (5) may “see” a shunt, much like Horton “hears” a Who, this does not mean that shunting has an effect on gas exchange. Quantitative analysis of shunting must accompany measures of gas exchange inefficiency before claims of cause-and-effect can be made.

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PATHOPHYSIOLOGICAL INSIGHT INTO SHUNTED BUBBLES
TO THE EDITOR: There may be more plasticity in the pulmonary circulation than commonly assumed (1), and the same is probably true for agitated saline bubbles (2). But it does not seem necessary to postulate particular pathways allowing for mixed venous blood to bypass the pulmonary capillaries.

Agitated saline echocardiography is commonly positive in patients with pulmonary vasodilatations on advanced chronic liver disease (3). These patients may initially present with normal arterial blood gases. Once hypoxemia develops, it is interesting that pulmonary shunt is increased, although differently according to the method of measurement. In a typical case, Crawford et al. (4) measured pulmonary shunt with radiolabeled microaggregates, sulfur hexafluoride (SF6), the less-soluble gas of the multiple inert gas elimination technique (MIGET), and pure oxygen breathing respectively at 40%, 25%, and 18% of cardiac output (4). The authors explained these discrepant results by a diffusion/perfusion disequilibrium affecting differently SF6 and oxygen (4). An alternative pro-

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posed explanation was in the insufficient discrimination by the MIGET between very low ventilation/perfusion relationships and a true shunt (5). However, hyperoxia decreases cardiac output (6), which is an additional cause of decreased pulmonary shunting. This could account for decreased bubble shunting observed in normal subjects breathing pure oxygen (1).

From the analogy to the hepatopulmonary syndrome, it is reasonable to assume that high flow-induced distention of pulmonary vessels would allow deformable bubbles to pass, yet most often with insufficient dilatation to cause hypoxemia through a diffusion/perfusion imbalance.

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LET’S FIND OUT THE SIZE OF THESE “SHUNTS”

TO THE EDITOR: How to define intrapulmonary “shunt”? Shunts for “gas exchangers” (5) are “blood that by-passes alveolar gas exchange completely,” but for “bubblers” (3) shunts are “vascular passages by which blood is diverted from its usual or normal path,” i.e., arteriovenous communications (a-vCs). The “gas exchangers” find, on heavy exercise, using 100% O2, that ≥0.5% of cardiac output bypasses alveolar gas exchange (5)—most of which is “postpulmonary.” So, intrapulmonary gas exchange shunts are “almost” imaginary.

Still, bubbles from agitated saline and from albumin microspheres (MAA) (4) traverse lungs during exercise, but rarely at rest (3). But the size range of bubbles is not well characterized—some smaller bubbles may squeeze through dilated capillaries (13 μm diameter) or alveolar corner vessels (20 μm) (2). The size of albumin microspheres can now be controlled, but even so, MAA of nominal 25 μm diameter will have 5% <21 μm diameter.

The implications for gas exchange of a-vCs <50 μm diameter are trivial (unless wall thickness was great or flow very high), because of the efficiency of diffusive exchange (1). But why not use radiolabeled (99mTc) MAA technology (25 ± 2.5 vs. 50 ± 4 μm diameter) to assess the size of a-vCs in humans undergoing strenuous exercise? The MAA shunt (as %cardiac output) can be quantitated from counts over the right kidney (postexercise) in relation to the injected counts (6). PFOs must be excluded, e.g., with bubbles.

Also, the filtering function of the lung must be considered.

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