Last Word on Point: Counterpoint: Exercise-induced intrapulmonary shunting is imaginary vs. real

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TO THE EDITOR: We completely agree with the letter writers (3) that it is important to apply quantitative techniques for the measurement of intrapulmonary shunt before drawing conclusions. Both MIGET and the 100% oxygen technique are quantitative and have consistently shown insignificant intrapulmonary shunting in most normal subjects (2, 6). Quantification of these shunts with 99mTcMAA, recently attempted (4), is problematic. This is because the 99mTcMAA technique relies heavily on assumptions about uniformity of particle size, adherence of the 99mTc to the particle, and assumptions about attenuation of counts in the face of changing respiratory muscle blood flow and pulmonary blood volume postexercise. All of these issues will bias the measurements in the direction of overestimating shunt. Additionally, 99mTcMAA has been validated only for large shunts (i.e., orders of magnitude greater than discussed here) and only against the 100% oxygen technique, which our colleagues have argued recently is invalid for detection of their arteriovenous pathways (4).

Our colleagues claim that because MIGET cannot distinguish right-to-left cardiac shunts (such as a patent foramen ovale) from intrapulmonary shunts that this somehow invalidates MIGET. All that this means is that gas exchange techniques cannot identify the physical site of a shunt, not that they cannot quantify it when present. Thus the values in Table 1(2) are a “worst case scenario” for intrapulmonary shunt, as they may also contain contributions from any intracardiac shunt.

Precapillary gas exchange is suggested as a reason why MIGET consistently measures shunt as a miniscule contribution to the AaDO2. If a vessel exchanges gas, it is by definition, NOT a shunt vessel, although diffusion-perfusion disequilibrium is possible. The effect of a shunt is to retain gases of all solubilities (not just SF6), including those that have solubilities bracketing the respiratory gases. Furthermore, O2 also exchanges upstream of capillaries (1), so this does not explain why these techniques measure a tiny shunt and associated effect on the AaDO2.

For intrapulmonary shunts to be anything but trivially important for gas exchange one has to believe that established quantitative techniques for measurement of intrapulmonary shunting are invalid: the 100% oxygen technique because of some previously undiscovered vasoconstrictive effect of oxygen on selected parts of the pulmonary circulation (4), and MIGET because of some mysterious gas exchange properties of these “shunt” vessels acting on some of the inert gases, but not respiratory gases or inert gases with similar solubilities. Finally, if many normal individuals “shunt” during exercise, as our colleagues assert, and “shunts” are potentially important determinants of cerebrovascular pathology (3), it is surprising that that athletes the world over are not experiencing cryptogenic stroke, as they exercise daily for several hours. This would appear not to be the case.

Until our colleagues can QUANTIFY flow through dilated channels using appropriately validated techniques and until those channels can be shown to have ABNORMAL O2 uptake, they have nothing more than an hypothesis. Until both standards have been met, microbubble transmission should not be called a shunt and a role in gas exchange should not be inferred.

REFERENCES

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