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# Commentary: Arterial blood gases in SCUBA divers at depth

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## KEYWORDS

high pressure, diving, arterial oxygen, dense gas, depth

## A Commentary on

### Arterial blood gases in SCUBA divers at depth

By Paganini M, Zucchi L, Giacon TA, Martani L, Mrakic-Sposta S, Garetto G, McKnight JC, Camporesi  
EM, Moon RE and Bosco G (2024) *Front. Mar. Sci.* 11:1445692. doi: 10.3389/fmars.2024.1445692

In a recent interesting study (Paganini et al., 2024), arterial PO<sub>2</sub> was measured while diving to 15 m and 42 m of freshwater (mfw). It was explored whether calculation of arterial PO<sub>2</sub> at depth could use the arterial/alveolar [partial pressures of oxygen ratio (a:A ratio)] at 1 ATA, as suggested by Moon et al. (1987). The a:A ratio calculated from the baseline arterial blood gases obtained at rest, out of the water, adequately predicted the PaO<sub>2</sub> at depth. However, at the greater depth of 22 mfw, the calculation proved to be inaccurate. In addition, Weaver and Howe (1992) presented higher PaO<sub>2</sub> than that predicted by the a:A ratio.

Although the slower diffusion while breathing dense gas should increase the alveolar-arterial difference in PO<sub>2</sub> (A-a)DO<sub>2</sub>, it is well documented that breathing dense gas begets an opposite result and reduces the (A-a)DO<sub>2</sub>. During the breathing cycle, pressure reduction at inspiration causes an increase in venous return. Thus, during normal breathing, lung perfusion increases by 18% at inspiration. With the increase in gas density, breathing resistance is increased, and so there is an increase in pressure swings during the breathing cycle. This would cause an increase in tidal perfusion in the lung. When the increase in alveolar PO<sub>2</sub> during inspiration meets an increased perfusion, higher oxygenation is expected. Thus, the decrease in (A-a)DO<sub>2</sub> can be related to better timing

between perfusion and elevated  $PAO_2$ . Modeling this assumption in a single-compartment lung, Arieli and Farhi (1985) presented that at dense gas breathing,  $(A-a)DO_2$  was reduced by 2 torr. Expanding the model to nine vertical differently perfused lung compartments, Arieli (1992) calculated a decreased  $(A-a)DO_2$  by 4–5 torr. The increase in cycling perfusion of the lung at a high pressure would explain why the a:A prediction would be inaccurate at the high pressure. Therefore, I claim that in any discussion of arterial  $PO_2$  at high pressure, cycling perfusion in the lung must be considered.

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## Conflict of interest

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