

Letter to the Editor

Fatty diet, active hydrophobic spots, and decompression sickness

In a study of the effect of a fatty diet on decompression bubbles, based on the responses to a questionnaire regarding daily food consumption, the approximate fat intake for each diver was calculated, taking into account the maximum recommended intake for a person doing his type of work.¹ Following hyperbaric exposure, divers were divided into two groups: 'bubblers' (a minimum of the second level on the Kisman-Masurel scale²) and 'non-bubblers'. Bubblers had higher fat consumption than non-bubblers ($146 \pm 39\%$ versus $92 \pm 18\%$). There was only a small difference in body mass index between the two groups: $26.3 \pm 3.3 \text{ kg}\cdot\text{m}^{-2}$ for bubblers and $24.9 \pm 1.9 \text{ kg}\cdot\text{m}^{-2}$ for non-bubblers. Cholesterol and triglycerides in serum were high in the bubblers ($211 \pm 39 \text{ mg}\cdot\text{dl}^{-1}$ and $230 \pm 129 \text{ mg}\cdot\text{dl}^{-1}$, respectively) compared with the non-bubblers ($188 \pm 34 \text{ mg}\cdot\text{dl}^{-1}$ and $153 \pm 111 \text{ mg}\cdot\text{dl}^{-1}$, respectively).¹ The authors concluded that a high-fat diet significantly increased the severity of decompression stress in hyperbaric air exposures. However, their explanation that the increased amount of fat in the serum contained more dissolved nitrogen, and that this was the cause of the increase in bubble production, was challenged in a subsequent letter.³

Decompression bubbles can expand and develop only from pre-existing gas micronuclei. It is known that nanobubbles form spontaneously when a smooth hydrophobic surface is submerged in water containing dissolved gas. We have shown that these nanobubbles are the gas micronuclei underlying decompression bubbles and decompression sickness.⁴ It has been suggested that hydrophobic multilayers of phospholipids on the luminal aspect of blood vessels, which we have termed active hydrophobic spots (AHS), were derived from lung surfactant.⁵ The essential components of lung surfactant required to construct the surfactant films, namely dipalmitoylphosphatidylcholine (DPPC) and surfactant proteins B and C, were found in the plasma of man and sheep, while DPPC was also found at the AHS.⁴ These findings have borne out the assumption that lung surfactants are the source of the AHS on the luminal aspect of blood vessels. These AHS seem to be stable, and their number and size increase with age as more DPPC settles. Bubbles may evolve at these AHS with decompression.⁴ The nanobubbles so formed on the surface of these lamellar layers of phospholipids in divers will expand into venous bubbles on decompression.

The main surfactant in the lung is DPPC (40%), with the presence of additional components including other phospholipids, glycerides, and cholesterol. In the cited study,¹ only serum triglycerides and cholesterol were measured, whereas it may well be that other phospholipids

and fatty acids were carried by proteins in the plasma. We suggest that, as with the different elements which compose the layers of surfactant in the lung, some of the additional fatty components carried by the blood will attach themselves to the AHS, thus contributing further to their enlargement. We hypothesise that divers who consume food that is high in fat, and as a result have more fatty components in their blood, will develop more and larger AHS, subsequently becoming bubblers with a higher risk of decompression illness.

References

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