

Venous Gas Emboli in Normal and Dehydrated Rats Following Decompression from a Saturation Dive

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Introduction: Dehydration may increase the risk for decompression sickness (DCS). Since DCS most probably is caused by endogenous gas phase formation, we hypothesized that decompression will induce more venous gas emboli (VGE) in dehydrated rats compared to controls.

Methods: Two groups of rats were pressurized to 0.5 MPa (5 ATA) on heliox for 16 h, and thereafter decompressed to atmospheric pressure at a rate of 0.3 MPa · min⁻¹. The nine control rats had free access to water ad libitum whereas the eight dehydrated rats were water-deprived for 48 h before decompression. During and after decompression, VGE was measured in the vena cava for 60 min with the Doppler technique and graded into six bubble grade (BG) categories. Body mass (BM), and food and water intake were registered daily, and venous blood samples were taken before and after pressure exposure.

Results: Serum osmolality and hematocrit increased significantly in dehydrated rats (306 ± 5.2 to 315 ± 7.3 mosmol · kg⁻¹ and 39.3 ± 4.9 to $49.6 \pm 5.2\%$) but not in controls (300 ± 8.9 to 303 ± 6.7 mosmol · kg⁻¹ and 40.3 ± 5.2 to $41.4 \pm 6.1\%$). Plasma volume decreased by 9.2% ($P < 0.05$) and 2.8% (n.s.) in dehydrated and control rats. VGE were detected in all control animals (average BG: 2.8 ± 1.9), but only in four water-deprived rats (BG: 1.6 ± 2.2). This difference was not significant.

Conclusions: Our experiments do not support the idea that dehydration increases circulatory VGE.

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