THE ‘SKIN BENDS’ HYPOTHESIS: DO EXTRAVASCULAR BUBBLES CAUSE ‘SKIN BENDS?’

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INTRODUCTION

- Bubbles appear to form in the 40-50 micron thick extravascular epidermis of the skin, not adjacent to capillaries that are located in the dermis below. Do these bubbles invade the dermis as they expand leading to: (a) the rash and itching of 'skin bends,’ (b) bloodless cutaneous lesions reported during isobaric counterdiffusion, and (c) venous gas emboli (VGE) observed after decompression or during cutaneous isobaric counterdiffusion (Bove, ed. Diving Medicine. 4th ed: Saunders; 2004:127-164)? The probability that bubbles enter capillary blood might increase if ‘skin bends’ were a histamine response resulting in greater capillary porosity. An antihistamine, on the other hand, might reduce capillary porosity and decrease the probability that bubbles enter capillary blood. These effects could be particularly important in fenestrated capillaries where discontinuous basal lamina with openings of 30-40 microns allow passage of 7.5-25 micron red and white blood cells.

METHODS

- Is this a testable hypothesis? If a dive profile were identified that reliably produced 'skin bends,’ volunteers in a double-blind human trial might make two identical dives receiving a placebo before one and an antihistamine before the other. Should bubbles that putatively cause 'skin bends' originate extravascularly, then rash, itching, and VGE detected by echocardiography might be more likely after the placebo dive than after the antihistamine dive.

DISCUSSION

- The issue of whether bubble formation is intra- or extravascular is potentially significant for understanding the mechanisms of mild (peripheral) and serious (neurological) decompression sickness (DCS) and for formulating DCS probability models that differentiate between mild and serious DCS (Vann. UHM 39(5):1038-1039). A test of the 'skin bends' hypothesis might clarify the role of VGE in serious DCS as suggested in the figures below. In Fig. 1, bubbles are most common in the skin and limbs where gas nuclei are prevalent. These bubbles seed the blood resulting in VGE that are carried by the circulation to the lungs where they are commonly filtered from the blood. Should a patent foramen ovale (PFO) or pulmonary shunt exist, however, or should small or excessive VGE pass through the pulmonary capillaries (Fig. 2), VGE might enter the arterial circulation and seed neurological tissues such as the brain that are otherwise resistant to bubble formation. Thus, cutaneous bubble formation may indirectly influence the probability of neurological DCS.

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Figure 1. Filtration of VGE that form in the limbs and skin.

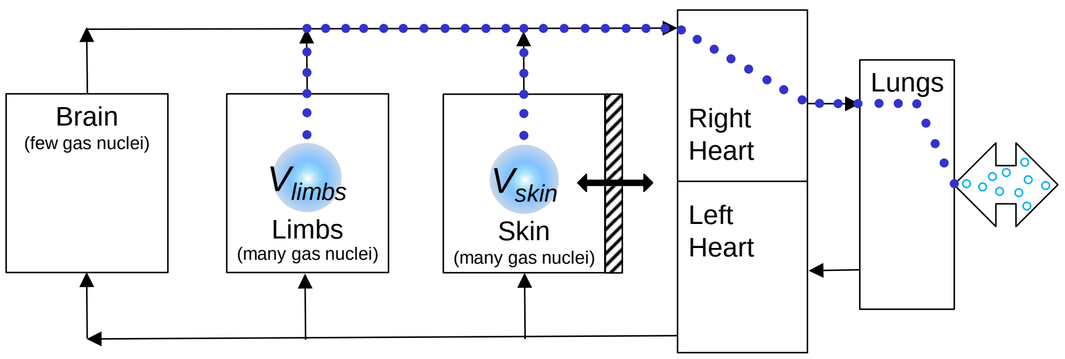

Figure 2. Arterial gas emboli (AGE) seed bubble formation in neurological tissue. AGE may result from PFO, shunt, or transpulmonary passage of small or too many VGE.