LETTER TO THE EDITOR

Editor:

The role of monitoring venous gas emboli (VGE) after decompression remains uncertain. Certainly, most investigators have recognized that it has little place in establishing the diagnosis of decompression sickness (DCS) in individual cases because of variability in equipment and Doppler probe placement, the lack of objectivity in quantifying the signal, and the high rate of VGE detection in subjects without symptoms. The paper by Bayne et al. (1) emphasizes this latter point. However, the paper lacks sufficient detail to properly evaluate the results. For example, the criteria by which the Doppler diagnosis of DCS was made is not given. I am unaware of an established criteria from which to make such a diagnosis. Was it simply the presence or absence of VGE or some arbitrary VGE score level? Did any of the subjects with clinical DCS have an absence of detectable VGE? This information is critical to an analysis of the results.

In addition, the authors make no distinction between the various DCS syndromes in their results or discussion. It is unreasonable to expect a similar correlation of the Doppler results with pain-only DCS as with spinal cord DCS, for example. Although the initiating event in both may be similar (gas phase), the pathogenesis probably proceeds by very different pathways. Pain-only DCS should have the lowest correlation with VGE because symptoms probably result from the growth of stationary bubbles (I suspect that the majority of DCS in this paper was pain-only in character). In this situation, VGE serve only as an indicator of the magnitude of evolved gas phase, which may vary only randomly with pain-only symptoms. On the other hand, spinal cord DCS should have a tighter correlation with VGE because the pathogenesis *relies* on VGE in the spinal venous plexus (2). In a similar sense, chokes probably also depends on VGE because a similar syndrome in animals can be produced by infusing air intravenously, and the symptoms are infusion rate-related (3). Unfortunately, appropriate studies to evaluate the correlation of precordial VGE with spinal cord or pulmonary DCS have not been done.

Nevertheless, one cannot argue with the author's conclusions that the Doppler information has little diagnostic value in the absence of clinical information. Indeed, this must already be the consensus as I know of no group or agency routinely using Doppler information in this way. But a point to keep in mind is that a high grade of VGE may not be entirely benign, *even in the absence of symptoms,* and thus treatment based on VGE may not be as inappropriate as it now seems. Perhaps decompression schedules should be designed to avoid VGE rather than just avoiding overt symptoms as some investigators have started to do (4, 5). Much future work on the role of bubbles in DCS will be required before these issues can be answered with any confidence. For all of the above reasons, Doppler generated information continues to have substantial value in learning about the various decompression syndromes—

its role in decompression research should not be underrated because of a lack of diagnostic power.

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