LETTER TO THE EDITOR

Basic Issues in Prescribing Preventive Decompression

Editor:

Decompression sickness continues to represent the major source of nonaccidental injury in deep-sea diving, and it is most welcome to see in this *Journal* (1) a resurgence of interest in methods of formulating preventive measures. However, there also continues to be a chasm between the models used for calculation and those used to interpret symptoms. The primary mathematical issues still concern whether inert gas uptake is controlled by diffusion (2) or blood perfusion, either continuous (3) or intermittent (4), and whether, upon decompression, symptoms are provoked by the mere presence of the gaseous phase (Haldanian supersaturation (3)) or the volume of that gas, either overall volume (1) or the maximum local volume (5) that is sometimes termed "the worst possible case." On the other side of the chasm, physiological studies have approached the problem "from the other end" by trying to relate each group of symptoms that could have a common physiological mediation of the insult to a mechanism that is examined largely on the basis of clinical evidence and without quantification in terms of dive parameters. As a first step toward establishing some bridge in relating dive parameters to those describing physiological insult, it would seem helpful to obtain some concensus upon the number of symptom categories and, hence, the maximum number of models that might be needed for a truly comprehensive approach to the prevention of decompression sickness.

The original approach of the Medical Research Council (MRC) (6) in differentiating between essentially local (Type I) decompression sickness and neurological forms (Type II) was a major step in the right direction. However, more evidence indicates that spinal, cerebral, and vestibular symptoms can be selectively provoked by choice of conditions, indicating that they probably have different etiologies, with each mechanism proceeding independently. Thus a spinal "hit" is not necessarily an extension of the same process eliciting a limb "bend"—i.e., is not a chance focus of the same whole-body injury on some particular target organ.

It is therefore proposed as a basis for discussion that the MRC system be extended to the categories shown in Table 1. None of these are new from the research or clinical viewpoints, but it is felt that some degree of formalization is needed to add emphasis to the possibility that there may be as many as five mechanisms by which a decompression insult can become manifest clinically, and as many models to be considered in prescribing preventive decompression: *1*) Arterial embolism leading to stasis and tissue ischemia (2, 7); *2*) Venous occlusion (8) leading to much the same state; *3*) Infarction by the products of blood-bubble degradation to produce ischemia (8, 9); *4*) Autochthonous bubble pressing directly onto a nerve or nerve ending (5, 10); and *5*) Autochthonous gas in a noncompliant tissue occluding blood flow and producing ischemia (11, 12).

There are many variations of these basic forms of decompression insult and numerous others for specific categories, especially dysbaric osteonecrosis. With at least six categories of symptoms and at least five basic insults, there are at least thirty possible combinations. These can be drastically reduced in number, however, by asking several simple questions, some of which

tend to be overlooked by proponents of new models. For instance, it is most interesting to listen to presentations on blood-bubble interaction, but the products of such degradation are incompressible and so it is difficult to offer this model as an explanation for any acute symptoms that are resolved by recompression. Well, this is one of at least nine basic questions to be asked for each model:

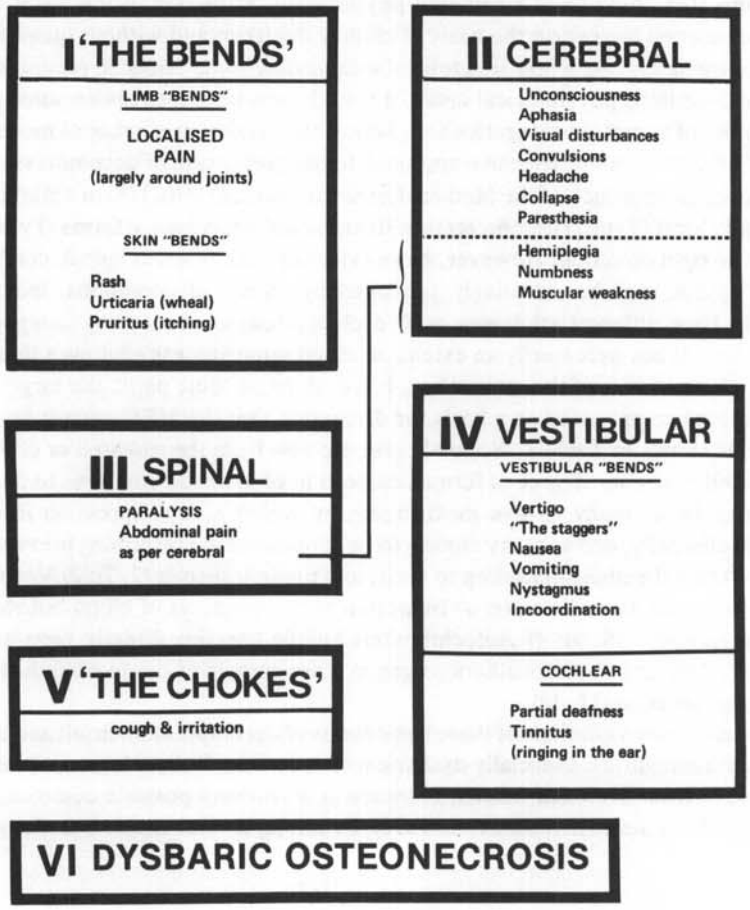
1. Are symptoms of that category reversed by recompression? If they are, then blood-bubble interaction is difficult to invoke as the primary cause.

2. Do symptoms of that category return if the subject is rapidly returned to the symptom-provoking pressure; i.e., are they pressure-reversible? An embolic mechanism is most unlikely if symptoms return with the same distribution, since obstructing bubbles are washed away (13, 14) in restoring blood flow, and it would be a remarkable coincidence for another shower of bubbles to be released and lodge in precisely the same sites as those eliciting the original symptoms.

3. Do other diseases producing potential infarcting agents elicit the symptoms of that particular category? A No makes an arterial embolic mechanism unlikely.

TABLE 1

CATEGORIES OF DECOMPRESSION SICKNESS



4. Does a general hypoxia potentiate symptoms? If it does not—as in limb bends (14)—then it is difficult to explain how any occlusion model could fail to exacerbate a situation where the tissue is already oxygen deficient.

5. Does hyperoxia ameliorate symptoms? It should do so for an ischemic insult.

6. Are the affected areas symmetrically or randomly distributed? Arterial embolism should produce a random pattern after allowing for blood flow distribution and would be difficult to implicate in Category VI, Dysbaric Osteonecrosis.

7. Does aerial decompression produce the same symptoms? The absence of bone lesions in aviators must reduce the list of models applicable to dysbaric osteonecrosis.

8. Does the time course of the manifestations fit the mechanism? For instance, it is difficult to attribute aseptic osteonecrosis to any model in which blood flow is occluded at the time of the dive.

9. Can we identify the tissue anatomically and is it compliant? It is difficult to implicate autochthonous bubbles in tissue easily pushed aside by the gas.

This is by no means a complete list of questions, but these and the suggested revision of the classification system are offered as a starting point for future discussion with a view to rationalizing model development and evaluating only those warranting full mathematical description of the insult in terms of dive parameters. Thus, studies of physiological mechanisms can play a greater role in the very practical need for designing safer decompression schedules based on more than one model—in fact as many models as are needed to prevent each category of DCS individually. It is hoped that this letter will stimulate future discussion in this area.

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REFERENCES

1. Yount DE. Application of a bubble formation model to decompression sickness in fingerling salmon. Undersea Biomed Res 1981; 8:199–208.
2. Boycott AE, Damant GCC, Haldane JS. The prevention of compressed-air illness. J Hyg (Camb) 1908; 8:342–443.
3. Hempleman HV. Investigation into the decompression tables, Report III, part A. A new theoretical basis for the calculation of decompression tables. RNPRC Report, UPS 131, London: Med Res Counc, 1952.
4. Hills BA. Intermittent flow in tendon capillary bundles. J Appl Physiol: Respir Environ Exercise Physiol 1979; 46:696–702.
5. Hills BA. A thermodynamic and kinetic approach to decompression sickness: 400-page monograph. Adelaide: Libraries Board of South Australia, 1966.
6. Medical Research Council. Bone lesions in compressed air workers with special reference to men who worked on the Clyde Tunnels (1958–1963). Decompression Sickness Panel Report. J Bone Jt Surg 1966; 48B:207–235.
7. Bert P. La Pression barometrique; récherches de physiologie expérimentale. Paris: Masson (1878). Translated by Hitchcock MA, Hitchcock FA. Columbus, Ohio: College Book Co., 1943. (Repr Bethesda MD: Undersea Medical Society, 1978.)
8. Hallenbeck JM, Bove AA, Elliott DH. Mechanisms underlying spinal cord damage in decompression sickness. Neurology 1975; 25:308–316.
9. End E. The use of new equipment and helium gas in a world record dive. J Ind Hyg Toxicol 1938; 20:511–520.
10. Nims LF. Environmental factors affecting decompression sickness. Part 1. A physical theory of decompression sickness. In: Fulton JF, ed. Decompression sickness, Chap. 8, Philadelphia: W.B. Saunders Co., 1951:192–241.
11. Kahlstrom SC, Burton CC, Phemister DB. Aseptic necrosis of bone: II. Infarction of bones of undetermined etiology resulting in encapsulated and calcified areas in diaphyses and in arthritis deformans. Surg Gynecol Obstet 1939; 68:631–641.

12. Hills BA, James PB. Spinal decompression sickness: mechanical studies and a model. Undersea Biomed Res 1982; 9:185–201.
13. Waite CL, Mazzone WF, Greenwood ME, Larsen RT. Cerebral air embolism. I. Basic studies. Rep 493, Groton, CT: US Nav Sub Med Center, 1967.
14. Grulke DC, Hills BA. Experimental cerebral air embolism and its resolution. In: Shilling CW, Beckett MW, eds. Underwater physiology VI. Proceedings of the sixth symposium on underwater physiology. Bethesda, MD: Federation of American Societies for Experimental Biology, 1978:587–594.
15. Hills BA. Mechanical vs. ischemic mechanisms for decompression sickness. Aviat Space Environ Med 1979; 50:363–367.