even up to 15 minutes after decompression. The presence or absence of surfactants and salts in solution makes no difference, because there are no preexisting bubbles. Therefore, it is unlikely that in divers or dolphins there are preexisting bubbles, for all would also have dissolved under the pressure.

If the dish is moved or shocked, bubbles form. This is consistent with the notion that cavitation in the fluid is the ultimate source of heterogeneous nucleation in decompression. Turbulence in the fluid itself can cavitate it, as with focused high pressures of sound. But, most commonly, the movement of a solid through the water or the rubbing of two solid surfaces underwater generates the same acuminate concentrations of energy. The pulling apart of two wetted areas in contact produces a huge local stress and can produce microcavitation (*3*).

A cavity once formed, however small, is filled with a gas-vapor mixture under the partial pressures available, and only the surface tension redissolves the bubble if the pressure is maintained. Large as that tension can be with the high curvature of the bubble, redissolving takes time, but it is aided by a sharp increase in hydrostatic pressure on the system (*4*). A shaken bottle of beer loses its ebullience on opening the longer that opening is delayed after shaking. A day later one can scarcely tell if the beer was shaken.

Consider some of the sources of cavitation in man: the slap of heart valves, the flow of blood around excrescences on the lips of those valves, the turbulence of common heart murmurs and of blood flowing over atheromatous plaques. There is the crepitation of bits of broken-off cartilage in the knee joints, the audible grate of the intervertebral processes in neck and lumbar regions, and the rubbing together of bony spurs. Sounds are produced when a partly compressed artery suddenly opens during the rise of the pulse pressure wave or when an artery is compressed between muscles or bones during a powerful limb movement (*5*). Every knuckle crack is a cavitation, and so on.

We must look anew at the anatomy of the dolphin and whale in whom we suspect no turbulent or cavitational causes, for bubbles abound. We suggest that the heart sounds of the dolphin are practically inaudible; that the rise and fall of the pressure wave in the peripheral vessels is slower than in land animals; that the heart valves are uncommonly smooth; that there is no atheromatosis; and that no artery is in a position to be partly occluded by any skeletal motion. In short, we suppose that whales and dolphins owe their immunity to the bends more to the smooth shaping of their form by evolution than to any physiological or biochemical trick yet to be found.

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Mackay and Lettvin *et al.* have proposed interesting explanations of how dolphins avoid the bends. The latter suggest that the physiology and anatomy of dolphins is less generative of bubble nuclei than that of humans. The former suggests that bubbles are generated, but are effectively crushed by pressure at depth. The weight of available evidence does not support either of these mechanisms.

There are neither theoretical nor empirical indications that physiological flows in dolphins are less turbulent than those in man. It is theoretically doubtful that velocities attained in either venous system are sufficient to produce Reynolds' cavitation even in a local region of constriction. Should bubbles be generated by tribonucleation, that is, by the collapse and opening of peripheral vessels, then it might be argued that dolphins should be more bubble-prone than man. The cetacean heart is not radically different from that of other mammals, and despite a blubber layer and longer acoustic path its sounds are audible with a stethoscope on the chest just as human heartbeats are. Chest sound recordings of our experimental subject dolphin Blue and a human of similar size revealed similar sound pressure levels and spectral characteristics.

The differences in decompression between species are less likely to be related to differences in vascular turbulence, analogous to shaken beer, than to differences in body weight, fat content, or gas solubility coefficients (*1*).

Mackay suggests that the repetitive diving of dolphins could effectively crush any bubble nuclei that might otherwise grow to symptomatic size during ascent. Any effective crush depth, however, is expected to be unreasonably deep. For rats, a predive conditioning pressure spike of 200 m reduced subsequent decompression deaths (a rather extreme threshold point) by only 8 percent (*2*).

In another study (*3*), however, decompression deaths were almost 40 percent when a 3-minute interval at the surface was interposed between 5-minute dives by mice to about 100 m; there were no deaths when the surface interval was eliminated. The authors proposed that the repetitive dives acted as an effective "bubble amplifier" (*3*). This experimental result remains inexplicable by conventional decompression theory but clearly indicates that repetitive dives to 100 m do not effectively crush nuclei. Our experimental dolphins willingly made 23 and 25 dives to 100 m in rapid succession.

Consideration of a prophylactic benefit of repetitive crushing of nuclei cannot ignore the famous investigation of Paulev (*4*): repetitive dives to 20 m over a 5-hour period can result in decompression sickness in man. Why dolphins are not similarly affected is not yet known. Differences in anatomy such as the extensive networks called rete mirabile and large venous space (*5*), or differences in biochemistry such as the lack of Hageman (*6*) factor and a more potent heparin might contribute to dolphin resistance to bends (*7*). But these proposals, like those of Mackay and those of Lettvin *et al.*, are speculation. Perhaps the mechanism is an evolutionary development akin to the still little understood bends acclimation mechanism in man (*8*).

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