tribution of scores we used the Wilcoxon test with adjustment for ties, one-tailed interpretation. Several findings emerged at high levels of confidence. However, the same findings emerged from the simpler technique of comparing the proportion of animals in treatment groups (versus saline) that met criteria for FMB or SPMB.


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## Dolphins and the Bends

Ridgway and Howard (*1*) conclude that "the mechanism by which dolphins avoid decompression sickness on dive schedules known to produce the syndrome in man is not yet completely understood." The formation of bubbles that cause the problems of decompression sickness probably depends on the existence of tiny gas nuclei on which the bubbles form. A single brief excursion to a depth of 200 m reduces bends in rats during a subsequent dive (*2*), and ultrasonic observations suggest that repeated excursions to lesser depths could also reduce the incidence of bends by forcing minute bubble nuclei back into solution. Observations of decompressed samples of gelatin suggest how such changes might come about (*3*). Thus the periodic swimming up and down of dolphins could effectively crush out bubble nuclei.

There is considerable interest in the mechanism of any nonthermal effect of high-frequency sound waves on the body, such as the effect on eye pressure and glaucoma (*4*), especially because of the increasing use of medical ultrasonic equipment. If stable, tiny bubbles exist in normal animal tissue—and the difference in the effects of decompression on whales and men suggest that they do—then in a man exposed to high-frequency sound these bubbles would vibrate and could cause tissue alteration (*5*).

The difference between whales and humans may not be large since one analysis of data from human dives (*6*) suggests that a dive to 100 m allows a safe pressure reduction of 2 to 3 atm, which approximates the conditions described in (*1*). The suggestion that whales must avoid the bends while other creatures do not may not be strictly true. Although most fish do not rapidly cycle between the surface and feeding depths as dolphins do, we have observed goldfish to be relatively inert to decompression death when many bubbles are present (*7*), but marine mammals may sometimes suffer decompression sickness (*8*).

Even if dolphins are not totally able to avoid bubble formation, they may be able to avoid damaging massive bubbling. Decompression bubbles generally tend first to appear in lipid-rich tissue (*7, 9*), and if such tissue is in the acoustic pathway of a dolphin approaching a dangerous condition, the animal's high-frequency sounds could give warning to guide surfacing. Decompression bubbles, a very small fraction of a wavelength in diameter, are detectable in intact tissue by ultrasound (*7*). The partial opacity of the dolphin lower jaw to sound could, for example, prevent it from hearing clearly, because the jaw is in the sound pathway and is fat-filled. Guinea pigs have been successfully decompressed by keeping bubble size limited to about twice the diameter of capillaries (*9*); such bubbles were detected with 150-kHz sound, to which dolphins respond. In man symptomless bubbles occur, but the subjects are not aware of them in the way a dolphin might be.


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Ridgway and Howard (*1*) showed that the partial pressure of dissolved $N_2$ in the muscles of dolphins that dive repeatedly to 100 m is about three times that in dolphins who remain at the surface. With this partial pressure in the tissues, a rapid ascent, or decompression, can produce the bends in man but not in dolphins or whales. That is, bubbles can form in blood, joint fluids, and at other sites, and they do so more easily in older persons.

In many respects, accounting for the difference between species is much like explaining what happens in a newly opened bottle of beer. If a bottle has been at rest for a day or so and is carefully opened, no bubbles appear in the resting beer. If the bottle has been shaken recently, or foreign matter is dropped into the fluid, bubbles evolve. The beer always bubbles when the flow is turbulent but not when the flow is smooth and laminar.

At a pressure of 4 or 5 atm, any bubbling in the charged beer must be due to heterogeneous nucleation, that is, the further filling of preexistent bubbles with the dissolved gases. Such pressure is not by itself sufficient to rend the attractive forces that hold water together. Accordingly, in the static case, we look to two sources for ebullition: free-floating "microbubbles" and vapor pockets at boundaries with solids. In respect of the latter, no solid surface in contact with beer is uniformly smooth microscopically. There are reentrant undercuts, patches that are nonwettable, sharp edges and points at grain boundaries, and all manner of favorable geometries to promotes separation of fluid from solid. At these places, particularly if there is a concavity of high curvature facing the beer, bubbles can form much more easily than in the fluid itself. [Surfactants such as foaming agents that reduce the surface tension of beer inhibit bubble growth in the presence of a preexisting trapped bubble (*2*). Surface-active chemicals, such as citrates, are used routinely to make long-lived club soda. But no surfactant can inhibit the growth of bubbles in solution.]

An experiment in our laboratory showed that shards of glass, grits, sand, and detritus of all sorts, when put into a dish of water before it is compressed to 4 or 5 atm, are not heterogeneous nucleators when the pressure is relieved after about 30 minutes. No bubbles appear



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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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