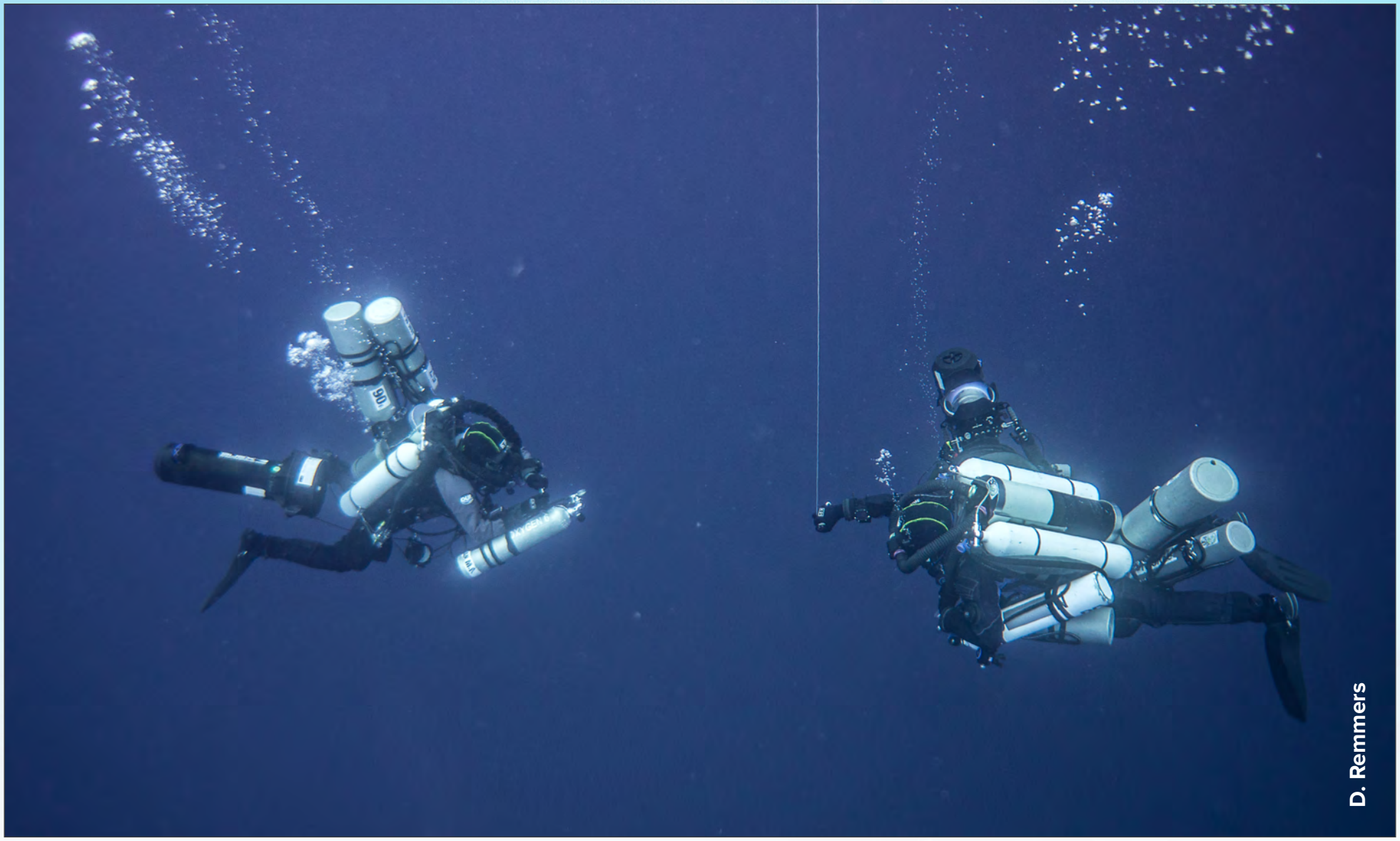
Decompression: Revisiting Old Assumptions

By Sergio Rhein Schirato

Decmompression sickness is a complex condition that is still not entirely understood.

THE first comprehensive attempt to understand the illness related to the exposure to hyperbaric environments was led by John Scott Haldane in collaboration with Arthur Edwin Boycott and Guybon Chesney Castell Damant, and published early in the 20th century as "The Prevention of Compressed-air Illness."[1] In retrospect, the methodology used in the study, the assumptions that different tissues would absorb and eliminate gas at different rates and how he modeled it, and the arguments used against the linear decompression (a method widely used at the time) are remarkable, especially if the knowledge and resources available at the time are taken into consideration. In many respects, most of Haldane's conclusions remain the basis for many procedures still in use today. With a few improvements to supersaturation values, and other refinements (or "fit-to-reality adjustments"), the differential equations used by Haldane are the same ones used in almost every computer or software available on the market today.

Given the information that was available at the time, it is understandable that Haldane and his coworkers treated the matter as a physical (or mechanical) problem caused by bubbles forming during decompression. Having said that, it is worthwhile to note that in this study they specifically recognized that many of the animals that died did not reveal signs of bubbles during necropsy and Haldane speculated that bubbles may have formed in parts of the body they did not study.



It's long been thought that a lack of bubbles indicated a successful decompression.

Nevertheless, he laid the foundation for an idea that is still very much accepted: decompression sickness (DCS) is a mechanical problem caused by bubbles.

The purpose of this article is to discuss this and some other assumptions widely accepted as true by the diving community in light of recently published studies.

Assumption 1: Venous gas emboli are formed during decompression and filtered by the lungs, while bubbles formed in or transported to the tissues are the cause of decompression sickness.

For many years it was believed that bubbles were related to decompression sickness and that their absence would mean a successful decompression. However, with the development of Doppler ultrasound technology late in the 1970s, it became clear that even mild exposures to hyperbaric environments and subsequent decompression would lead to bubble formation in the venous circulation. Though bubbles were commonly found in the right chambers of the heart, Doppler echocardiograms showed that most of them were filtered by the lungs and were not observed in the left chambers of the heart. In theory, bubbles would be pumped from the left chambers into the systemic circulation, which would send them to the central nervous system, causing the neurological symptoms of decompression sickness. This finding led to the endless discussion about the role of cardiac or pulmonary shunts in decompression sickness since the existence of a shunt would allow the migration of bubbles from the venous to the arterial (i.e., systemic) circulation, bypassing the filtering effect of the lungs.

While this statement might hold true for large venous gas emboli most of the time, there are other facts that must be considered: (1) Patent foramen ovale (PFO), a remnant of our fetal circulation, is found in approximately one-third of the population; (2) pulmonary shunts are, among other things, a physiological response to handle the cardiac afterload, and different studies with high-performance athletes have shown that all subjects studied presented some level of pulmonary shunting as the physical effort to which they were submitted increased; (3) the central nervous system has fast inert gas kinetics,[2] meaning that bubbles eventually shunted through the heart to these tissues tend to lose gas to the media, being reduced in size and quickly collapsing. This assumption can be supported by the fact that the gold standard for PFO detection is the transesophageal echocardiogram coupled with the injection of agitated (full of bubbles) saline solution, in which gas serves as a contrasting media to the ultrasound. There are no known cases of decompression sickness-like symptoms related to the use of such contrast, even when bubbles are clearly shunted to the left atrium.

Additionally, post-dive bubbles detected by Doppler have diameters larger than 30 µm. A recent study using contrast-enhanced imaging techniques capable of detecting bubbles with diameters smaller than 10 µm indicated the presence of smaller emboli in both sides of the heart, demonstrating that:

(1) there are small bubbles in humans that are not filtered by the lungs; (2) there are small bubbles even in the absence of larger venous gas emboli; and (3) smaller bubbles follow a different timeline than larger venous gas emboli.[3] Bubbles forming in the arterial circulation have also been identified in previous studies though their role in decompression sickness, especially in the presence of neurological symptoms, is yet to be understood. Vascular bubble models, designed to study nucleation on a flat hydrophobic surface and how they expand to form bubbles after decompression, hold great promise for the improvement of decompression procedures in the future.[4]

Decompression sickness is not just a physical or mechanical issue.

Assumption 2: Mechanical damage caused by bubbles is due to decompression sickness.

Several studies over the past two decades have shown that decompression has many physiological implications, ranging from reduction in endothelial function to activation of the immune system. As discussed above, formation of bubbles is a common finding in subjects exposed to hyperbaric environments and subsequent decompression. The causal relationship between bubbles and physiological alterations, however, is yet to be proven. In recent years, the endothelial dysfunction hypothesis, which postulates that microparticles associated with endothelial damage act as nucleation sites for bubble formation, has drawn attention and gained support. This has resulted in decompression sickness being seen not as merely a physical or mechanical problem, but instead as a result of a complex biochemical process.

Recent studies have shown that the exposure to high-pressure environments is sufficient to increase the production of IL-1β, an interleukin that belongs to cytokines, which is an important mediator in inflammatory responses.[5] The mechanism behind the formation of such microparticles is related to high inert gas pressure through a mechanism that causes singlet oxygen formation, a potentially toxic free radical initiated by a cycle of actin S-nitrosylation, nitric oxide synthase-2, and NADPH oxidase activation ultimately leading to microparticle formation.[6] Despite their harmful effects to the host, the production of reactive oxygen species (ROS) is part of an orchestrated physiological response of the immune system to stop bacteria and fungus. Exposure to high inert gas pressures, even in the absence of decompression, is apparently linked to an increased production of ROS. The potential to trigger this reaction depends on the gas and follows the rank: argon ~ nitrogen > helium.[7] This ranking might explain the reduced endothelial dysfunction identified after hyperbaric exposures where helium was part of the breathing mix.[8]

The mechanism behind decompression sickness appears to be more complicated than the simple growth of bubbles, and a lot remains to be understood.

Assumption 3: Decompression profiles with deep stops are safer.

With divers pushing the boundaries of deeper diving beyond military and commercial diving, and the introduction of helium in the breathing mixes in the 1990s, different decompression techniques for bounce (non-saturation) dives started to be tested. Richard Pyle, an American ichthyologist from Hawaii, was probably one of the first to publicly advocate for decompression stops deeper than those calculated by algorithms derived from Haldane´s theory. On dives ranging in depth from 40 to 70 m, he correlated catching fishes with his overall feeling after diving, and attributed feeling better to the fact that when a fish was caught, he had to stop much deeper than determined by decompression algorithms to release gas out the fish´s swim bladder. Decompression algorithms based on the control of bubble formation and growth including the Varying Permeability Model developed by David Yount, which is the most well-known algorithm based on this strategy (probably because it is open code software), require decompression stops at greater depths, corroborating Richard Pyle´s conclusions. At some point, it became well-established within the diving community that deeper stops were mandatory and even Albert Bühlmann´s ZHL 16 algorithm was adjusted; gradient factors were implemented to calculate deeper stops.

There is, however, no scientific data available to support the belief that the modification of the decompression schedule with the inclusion of deeper stops reduces the expected probability of decompression sickness. In reality, studies showed that slower ascents are related to higher counts of bubbles upon surfacing.[9] Nevertheless, whether this translates to a higher probability of decompression sickness is another matter.

In what was probably the largest study comparing the incidence of decompression sickness in bubble-based models versus dissolved gas-based models (derived from Haldane's work), the Navy Experimental Diving Unit[10] concluded that decompression schedules with deeper stops had higher incidence of decompression sickness. In this study, dive profiles with equal decompression times and to a depth of 51 m were calculated using each model. These were then compared for decompression sickness and venous gas emboli count. The deep stops schedule resulted in a significantly higher incidence of decompression sickness than the shallow stops schedule (10 cases versus three, a result significant at the 5% level of confidence). Interestingly, the bubble-based profile resulted in a higher maximum venous gas emboli grade count, as well as higher average grade count.

The reason behind the findings mentioned above might be related to the different supersaturation observed in tissues with higher half-times upon surfacing. Figure 1 illustrates total inert gas in each tissue upon surfacing, based on Albert Bühlmann´s ZHL 16 algorithm, for two profiles calculated with different gradient factors (GF) to simulate decompression schedules generated by dissolved gas- and bubble-based models.

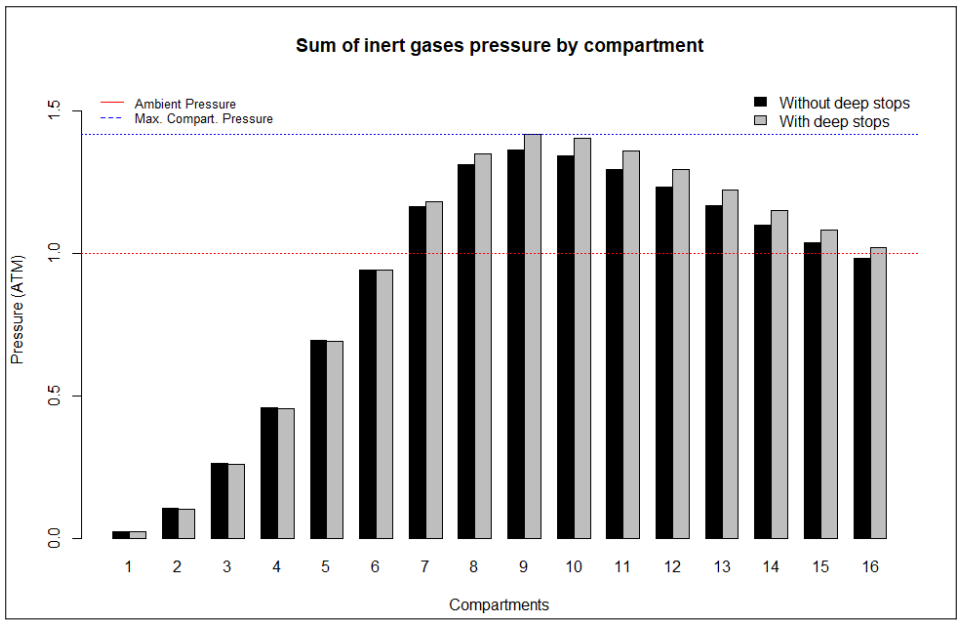


Figure 1

Both profiles were calculated to provide similar decompression times for a dive to 51 m of depth and a bottom time of 30 minutes. The profile with deeper stops generated higher supersaturation values in the slower compartments upon surfacing, while the profile without deeper stops generated higher supersaturation values in the faster compartments, which, presumably, tolerate higher inert gas pressures.

A better way to compare these two decompression schedules would be to compare the supersaturation in a given compartment produced by each one by subtracting the ambient pressure from the total inert gas pressure in a compartment (all calculations were made using an ambient pressure of 1 atmosphere). In Figure 1, only compartments 7 to 15 have internal inert gas pressures higher than ambient pressure upon surfacing, meaning that compartments 1 to 6 and 16 had total inert gas pressures below ambient pressure upon surfacing. The comparison between the two profiles can be seen in Figure 2.

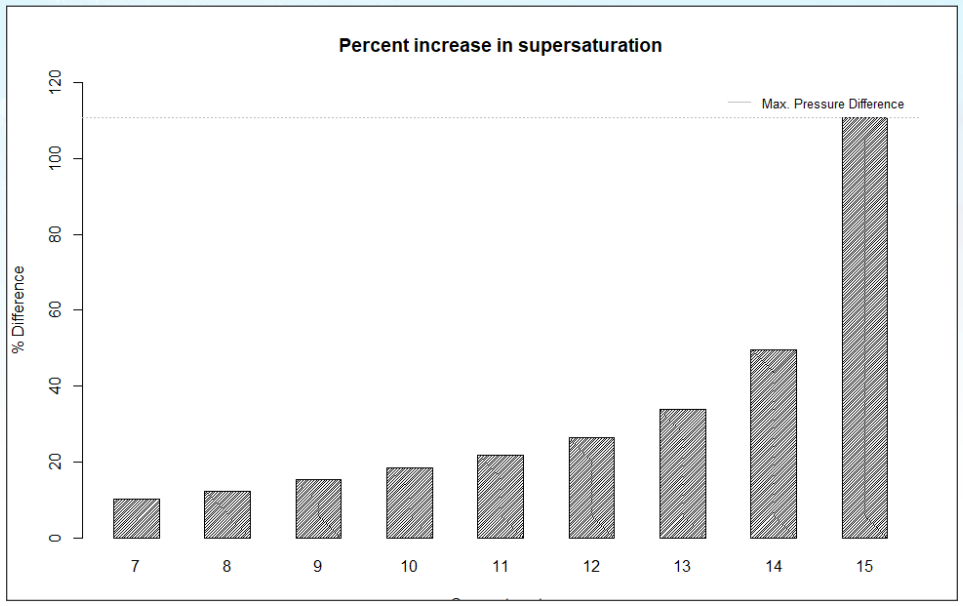


Figure 2

The decompression schedules with deeper stops generated supersaturation values as high as 2.15 times the supersaturation produced by the profile with shallower stops. This difference might be a possible explanation for the conclusions from the above-mentioned studies.

CONCLUSION

Over the past decades, many beliefs about decompression and decompression sickness have permeated the diving community, many of them based on ideas not supported by scientific evidence. Recent studies have demonstrated that in some cases, evidence points in the opposite direction. Decompression sickness is a multifactorial condition

> Decompression is an area where you discover that, the more you learn, the more you know that you really don't know what is going on. For behind the 'black-and-white' exactness of table entries, the second-by-second countdowns of dive computers, and beneath the mathematical purity of decompression models, lurks a dark and mysterious physiological jungle that has barely been explored.
>
> Karl E. Huggins, 1992

Efficient decompression is as much an art as it is a science.

that involves the activation of many biochemical pathways, and the mechanisms behind it are still not fully understood. There is still a long way ahead and each new study helps to add another piece to this complicated puzzle.

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