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DECOMPRESSION SICKNESS: BUILDING AN UNDERSTANDING

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A NMRI researcher discusses the kind of reasoning that has been used to design decompression schedules, and presents his own view of how well the various theoretical concepts agree with proven events in a diver's body.

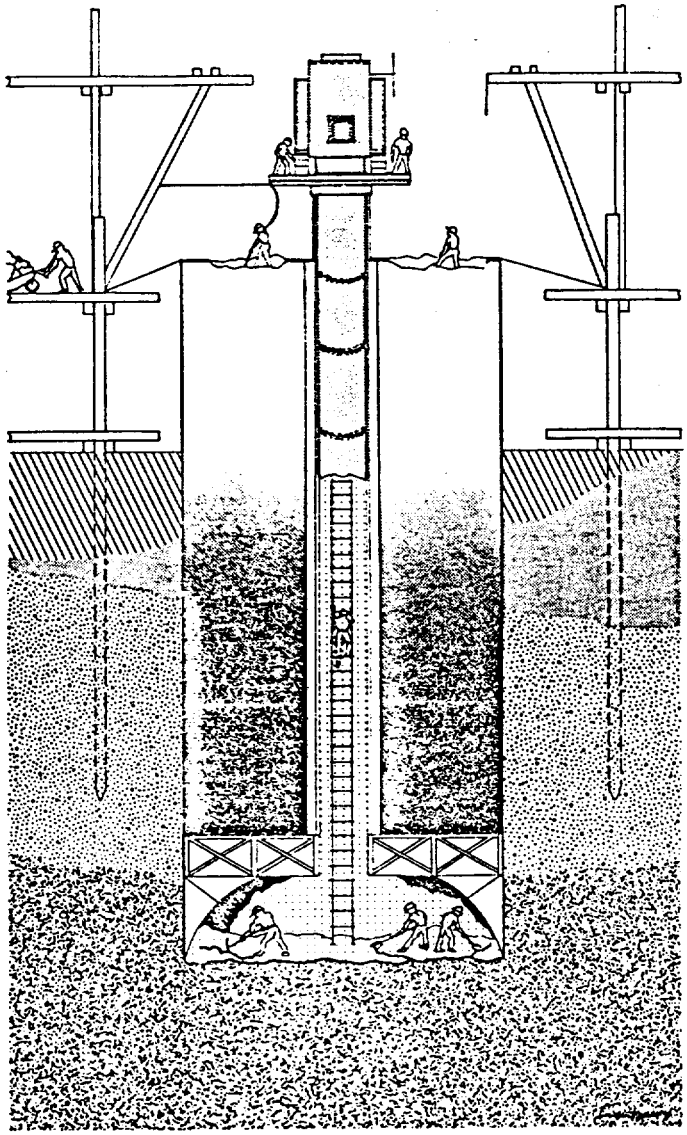
Divers are well acquainted with the U.S. Navy decompression tables. How these tables developed and how their development compares with the reasoning behind decompression tables of other navies and private organizations is not as widely known.

Decompression sickness (DCS) is a rare example of human disease in that prevention of the disease can be approached by the use of involved theoretical calculations. The theoretician follows several steps: First, he decides on the cause of the disease; he then breaks down the cause of the disease into aspects that can be solved separately; finally, he reassembles these partial solutions into a complete package that can describe the entire disease process and its prevention.

Decompression calculation procedures are thus developed from particular sets of ideas about events in the body that may cause the variety of DCS symptoms. Each set of ideas, whether based on fact, assumption, or guess, is known as a "decompression theory."

Search for the DCS Villain

Possible causes of DCS, proposed within the last century, are listed in Table 1. When workers became stricken with DCS, or "the bends," during the caisson construction of the St. Louis bridge in the 1870s, some doctors concluded that diver exhaustion was to blame. But simple physical exhaustion has failed to answer most of the questions raised by DCS, although exercise while under pressure still appears to contribute to it.



Pneumatic caissons were used widely in the late 19th century to construct bridge pilings. Work spaces were filled with compressed air to prevent water seepage. Upon returning to the surface, however, workers were often stricken with decompression sickness. Doctors called it "caisson sickness" and attributed it to physical exhaustion. Today, gas bubble theories predominate, but researchers disagree on the precise process through which the gas leads to DCS.

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"... most 'practical' diving experience does not contribute to our understanding of which (decompression sickness) theories work better than others."

More recently, several groups of diving researchers have based the prevention and treatment of DCS on drugs and on procedures that affect blood cells, blood vessel walls, or the movement of fluids among various spaces in the body. These researchers could be said to follow a *biological* approach to DCS prevention.

Today, most researchers follow a *physical* approach to DCS; that is, they believe that DCS occurs after the physical process of gas formation in the body. According to this belief, DCS can be avoided if the formation and growth of gas bubbles is avoided or, at least, minimized. Furthermore, this school of thought attributes the changes in blood cells, body fluids, and blood vessels to biochemical events that occur only *after* the bubbles appear.

Emphasis on the gas bubble as primary villain in DCS has encouraged researchers who are mathematically inclined to try to solve DCS problems without worrying about the complicated biological processes that underlie the disease (which is the usual mode of operation in medical research). The processes that describe how gases move through liquids and how bubbles form and grow in supersaturated liquids are fairly well understood. At least in simple physical situations, these processes can be described with precise mathematical expressions.

Elements of Gas Bubble Theory

Elements to be considered when one evaluates DCS bubble theories are detailed in Table 2. The major elements to be addressed are (1) *the rate of gas exchange* (i.e., at what rates and in which locations does gas move in the body during a dive?), and (2) *the symptom-provoking event(s)* (i.e., what property of the gas bubble(s) causes DCS symptoms?).

Rate of Gas Exchange. Gas exchange rates probably depend, in a rather complicated way, on the properties of gas molecules as well as on the properties of body tissues. Gas transport is frequently described from two simplified viewpoints, the *blood perfusion model* and the *tissue diffusion model*. The more common blood perfusion model states that gas transport is limited by rate of local blood flow because the gas molecules can exchange very quickly between small blood capillaries and the tissue spaces near them. The tissue diffusion model states that blood can supply or remove gas quickly, so that the process of diffusion of gas molecules between or into tissue cells limits the overall rate of gas exchange. Evidence from laboratory experiments has not yet resolved which simplified approach applies to human diving. The final answer may lie between these two extremes; already, several theories contain mixtures of perfusion and diffusion elements.

Symptom-Provoking Events. The second element of any gas bubble theory requires that a decision be made on what makes bubbles "bad." For much of this century, attention was focused on the idea of bubble formation. The tactic in decompression schedule calculation was to avoid a critical ratio of inert gas supersaturation (that is, the ratio of the dissolved gas partial pressure in the body to the hydrostatic pressure at the diver's particular depth). This consideration was presumed to prevent bubble formation, and it is the basis for the present U. S. Navy tables. In recent years, however, evidence has accumulated that during many dives, divers' bodies contained gas bubbles without the divers noticing any DCS symptoms. Therefore, several theories allow for the formation of bubbles but try to prevent the bubbles from growing to a certain critical size, or deforming certain tissue structures, or exceeding a certain number.

Why So Many Bubble Theories?

Gas theories for calculating decompression tables are shown in Table 3. At the risk of oversimplification, I have listed for each theory (1) the number of "tissues"

TABLE 1

POSSIBLE CAUSES OF DECOMPRESSION SICKNESS

1. Exhaustion
2. Blood cell aggregation/clotting
3. Fluid shifts
4. Chemical changes in blood vessels
5. Gas bubbles

TABLE 2

ELEMENTS OF GAS BUBBLE THEORY ABOUT DECOMPRESSION SICKNESS

1. Rate of gas exchange:
   - Blood perfusion
   - Diffusion through tissue
   - Statistical
2. Symptom-provoking event(s):
   - Initial formation of bubble
   - Growth to critical size
   - Biology following appearance
   - Location

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TABLE 3

DECOMPRESSION TABLES CALCULATED BY DIFFERENT GAS BUBBLE THEORIES

Source	*"Tissues"*	*Gas Rate*	*Bubble Event*
Haldane (1908)	5	Perfusion	Formation
US Navy (Workman, 1956)	6–9	Perfusion	Formation
Royal Navy (Hempleman, 1967)	1	Diffusion	Formation
Swiss (Bühlmann, 1969)	6	Perf/Diff	Formation
"Thermodynamic" (Hills, 1966)	2	Diffusion	Growth
Hawaii (Yount, 1978)	1	Perf/Diff	Number

(mathematically distinct sets of gas exchange rates); (2) the procedure for calculating rates depending upon the choice of blood perfusion or tissue diffusion models, or a combination of both; and (3) the choice of bubble event that leads to DCS. Obviously, theories are available to cover most of the possible combinations.

The reasons for having a wide selection of gas bubble theories of DCS are simple: Very little experimental information is available for deciding which set of assumptions matches the events occurring in a diver's body. The gaps in our knowledge exist for many reasons. The perfusion-diffusion controversy still rages despite evidence that both assumptions are incorrect. We still do not have methods to detect or measure sizes of bubbles in most parts of the body. In addition, pain, the most common symptom of DCS, is difficult to describe biologically, much less mathematically. Finally, in spite of the inherently low incidence of DCS during thousands of dives using common decompression schedules, we cannot eliminate the possibility that chance alone may account for the differences in safety reported in dives using different decompression tables. Thus, most "practical" diving experience does not contribute to our understanding of which theories work better than others.

Several projects at the Naval Medical Research Institute are aimed at plugging these gaps in our knowledge. Commander John Hallenbeck has been able to follow the progression of biological events in spinal cord DCS that occurs after bubbles become visible. Commander Thomas Berghage has amassed a large body of information on the procedures that lead to serious symptoms of DCS in small animals—information that will serve as a reference for predictions from theoretical calculations. Commanders Edward Flynn and Kristopher Greene are preparing to measure gas exchange in divers performing at different levels of exercise. Dr. Louis Homer is developing the mathematical formulations necessary to compare special combinations of perfusion and diffusion gas exchange conditions with careful animal and human measurements. My colleagues and I are measuring gas exchange over prolonged periods in many tissues of animals, and we have developed a statistical description of gas exchange rates that avoids choosing between perfusion and diffusion assumptions.

Conclusions

- Decompression theories are not scientific theories in the sense of the law of gravity; rather, they are mathematical predictions based on unverified assumptions.
- Decompression theories do provide calculation methods for summarizing the experience of divers; they also provide starting values for developing new schedules.
- Theoretical calculations of new decompression schedules usually cannot be accepted immediately; they are revised to provide adequate safety.
- It remains for researchers to establish how fast and where gas exchange occurs and the specific processes through which the gas leads to DCS.

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k. EQUIPMENT AND SUBCONTRACTS

Equipment	Estimated Cost FY 1980
Amino acid analyzer*	$40,000
EM 3630 lock/decoupler for nmr, Varian	6,000
Radial compression acessory for HPLC, Waters	3,000
2 Digital electronic balances	6,000
TOTAL	$55,000

*Beckman model 118BL analyzer with data system and spare parts kit: $38,175 and $1,580 = 39,755

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