"GOING DIVING"

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New thoughts on decompression

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Our decompression tradition is based on the principles set by Haldane in 1908 (1). The classic or "Haldanian" models used for the calculation of present military, commercial and recreational diving tables all have the same mathematical expression: a) they figure inert gas exchanges with a series of exponential compartments, b) they specify the safe ascent criteria as a linear relation between the ambient pressure and the maximum permitted compartment tension. As a consequence, these models have produced similar decompression profiles, characterized by a rapid initial ascent on a relatively high distance. However, the drive for table development is operational pressure. Aside these traditional tables, marginal diver communities have empirically developed original decompression procedures for their needs. Refreshing experiences come from technical and cave divers who keep pushing their explorations supported by a new Internet culture. Surprising procedures come from coral or shell divers who keep diving deeper and deeper to make their livings. Apparently, all share the same strategy and use slower rates of ascent and deeper stops than would be predicted by the Haldanian models (2). An attempt is made to introduce new assumptions in the present models to obtain the typical profile of these successful decompressions.

Identifying the risk of decompression

The safety performances of decompression tables can be first defined in terms of the risk of decompression sickness (DCS) occurrence per dive exposure (3). This risk was difficult to assess until the development of diving databases in the 80's provided the volume of information required for the statistical analysis (4,5,6). DCS symptoms include a wide span of problems ranging from skin rash to articular pain and neurological symptoms which for operational reasons have for long been classified into two categories, Type I DCS and Type II DCS, according to the US Navy Diving Manual. Thus, the risk can further be defined in terms of symptoms.

Type I DCS includes simple symptoms like skin rash, articular or muscle pain. Because the symptoms are obvious, they are reported early and the treatment is initiated without hesitation. In most cases, administration of hyperbaric oxygen at 12m will rapidly resolve the symptoms. Safety wise, a Type I DCS is a "good" decompression accident because the diagnosis is easy, the onset is usually rapid, the treatment is applied rapidly, and the symptoms are treated efficiently in 95 % of the cases (7).

Type II DCS is always serious because it affects either the respiratory or the neurological systems. The symptoms which often include fatigue, headache or feeling unwell are vague and the diagnosis may be difficult at an early stage or for mild cases. The treatment is complex and requires a deep recompression, significant periods of hyperbaric oxygen breathing, fluid intake and sometimes steroids administration (7). Safety wise, a Type II DCS is a "bad" decompression accident because the diagnosis may not be easy, the treatment is often delayed, and the consequences can be dramatic.

It is interesting to note that in an earlier time, medical doctors tended to distinct more types of DCS symptoms. In the 1974 edition of the Comex Medical Book, Dr. X Fructus differentiated between vestibular hits and other neurological symptoms. At the time, he suspected that different dive profiles yield to different neurological symptoms. This came after he developed some early bounce decompression tables, called the "Cx70" tables, that were designed both for heliox surface supplied and bell diving.

The Cx70 tables were intensively used in the early North Sea developments before saturation diving finally took over bounce diving. The analysis of the safety records of the Cx70 tables (unpublished) stored in the Comex Data Base (8) showed that the risk was uneven over the exposures. The long bottom times (90 to 120 minutes) tended to induce Type I DCS in the last part of the decompression, which follows the logic of large amounts of dissolved gas to be eliminated. The intermediate bottom times (30 to 60 minutes) preferably produced neurological symptoms, either central or spinal, a common outcome with heliox bounce diving. Surprisingly, short bottom times (10 to 30 minutes) exclusively produced vestibular symptoms. Typical of their time, the Cx70 tables were characterized by a high distance between the bottom and the first stop and a rapid initial ascent. This was particularly significant for short bottom times. For instance, a particular concern was the 66m/20min table with a first stop at 12m, leading to an initial ascent of 54m done in 3 minutes (18m/min). The safety analysis of these

early deep bounce tables already indicated that the dive profile controls the bubble scenario and decides on the safety outcome of the decompression and the initial phase was slowed down in the later revisions of the tables.

The lesson learnt after the Cx70 tables is that the main risk for technical divers, who usually dive 15 to 20 minutes bottom time, is a vestibular hit. It is already a serious accident when symptoms occur at the surface, with strong nausea and vertigo. As technical diving gets deeper, it will become dramatic the day the symptoms occur in the water. With bell diving, such accidents were kept under control: the diver was recompressed in the bell or the chamber and put on mask. Unfortunately, if a technical diver got bent in the water, he would not be able to take any therapeutic action, and when trying to ascend, would likely vomit in his regulator: a critical situation. Because of the threat of symptoms occurring in the water, technical divers must use very safe tables.

Strategies for successful decompressions

Divers have known the criticality of the initial ascent in decompression for long. In fact, each divers group has generated its own strategy to increase their decompression safety. The very first adaptation is the reduction of the ascent rate. In commercial diving, most of the diving supervisors admitted they used slower rates of ascent than the 60 ft/min recommendation of the US Navy. The French Navy reduced the ascent rate from 18 to 12 m/min in the 1990 revision of their air tables.

Another common modification is the introduction of a deep stop. One of the earlier accounts I heard came from two old divers who worked for red coral in the 50's. They told they used to stop at mid-depth during their ascent from deep dives and sang a gospel. They added with a smile that of course they would adapt the number of verses to how comfortable they felt with the decompression. Obviously these two old timers knew things I ignore for I have never been able to neither imagine how physically feels the decompression stress nor conceive any physiological mechanism for it. Deep stops appeared in the early edition of the Royal Navy diving manual. The manual included a set of deep air tables to 90 meters, the particularity of which was the presence of a mid-depth decompression stop (up to now, I have been unable to identify who introduced such stops and for what reasons). Deep stops were later advertised by Richard Pyle in articles published in the "AquaCorps" and "Deep Tech" magazines (2) and supported by other influent authors (9). Today's, deep stops are so popular that most decompression software's propose one or two arbitrary deep stops during the ascent. However, other diving strategies can be identified.

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Mediterranean coral diving

Red coral jewels are a tradition around the Mediterranean and coral has been fished for centuries using an assembly of beams and nets, called "the cross", drag along the underwater walls. Since the introduction of SCUBA diving, there has been a small population of coral divers in France, mainly along the Côte d'Azur and in Corsica. Diving for coral is a tough job as divers during the season usually dive twice a day for 20 minutes, to 80-90m on air. Recently, because coral is getting scarce, divers have moved to trimix and work at around 100-130m. Coral divers use a variety of decompression procedures that they have developed empirically and are very reluctant to give away. Over the years, I have been able to collect and document several of these original procedures. It appears that coral divers normally use one set of decompression stops that they learn by heart and apply to varying exposures, such as 25 minutes at 80m, 20 minutes at 90m and so on. They usually carry their bottom mix on a twin set or tri-cylinders backpack and rely on the boat to deploy an umbilical for the rest of their ascent. They all use oxygen aggressively, usually starting at 12m in the water, sometimes at 15m for few minutes. Some of them also have a chamber on board in which they finish their ascent after surface decompression.

Coral diver tables appeared dramatically short when compared to commercial or recreational references. The reason why coral divers survive such severe decompressions must be their specific operational procedures. Coral divers have explained that at the end of the bottom time, they ascend rapidly 10-15 meters above the bottom to get read of the narcosis. Then they slow down (9-6 m/min) until they reach 40 meters where they wait for the boat to spot their bubbles. When the boat is positioned above them, they send a lift bag to the surface with the basket attached to one end. The crew sends an umbilical down using the same line. When the basket is secured, the divers proceed with the rest of their decompression. This way, the dive profile is lengthened by some 8-10 minutes spent at mid-depth, apparently essential to their decompression safety (figure 1).

Comex deep decompression studies

In 1977, Comex, a commercial diving company, planned to conduct deep diving operations on a large scale and wanted to select a pool of deep divers based on their individual susceptibility to the High Pressure Nervous Syndrome. A test protocol was set up where divers were pressurized in 15 minutes to 180m, run a battery of tests for two hours at bottom and then were decompressed. The person calculating the decompression at the time had difficulties to define a mathematical model for such a severe exposure. He finally gave up the idea of any mathematical support and simply started drawing the decompression profiles on paper. After some trials, he discovered that by plotting the rate of ascent in a logarithm scale versus depth in a linear scale, decompressions roughly appear as a straight line (figure 2). He further used this property to design a set of decompression tables. His method produced decompressions with varying rates of ascent and very deep stops. After some adjustments, he designed a schedule for the 180m/120min dive that required 48 hours of decompression and which appeared extremely safe. A total of 49 divers went through the selection tests without any symptom (10). The characteristic of the "semi log plot" is that it expands the deeper portion of the decompression and makes it possible to define rates of ascent in an area where traditional models fail to control the ascent (figure 2). Although the method was

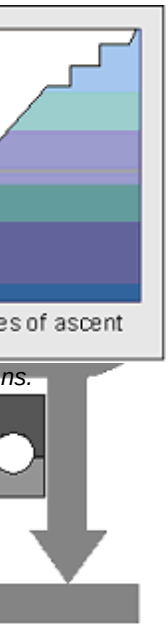
Possible ascent strategies

The classic approach of decompression is based on Haldanian models and involves assumptions that are summarized below:

1) Diving requires compressed air breathing and causes nitrogen to dissolve in the diver's tissues.
2) The critical issue is the amount of nitrogen stored in the tissues (dose) prior to the ascent.
3) The primary insult is the volume of the gas phase formed during the ascent.
4) Limb bends and neurological symptoms are not differentiated and are considered as different levels of severity of a same problem.
5) The sites for bubble formation are the tissue or the venous side of the blood circulation but no tissue is specifically identified and a series of "compartments" is considered instead.
6) The decompression strategy consists in managing the amount of gas dissolved in each compartment to control a gas phase formation and avoid DCS during the ascent (figure 3).

These assumptions are the basis of present table calculations although a large variety exists in the gas exchange models or in the criteria used to control bubbles formation. These models cannot be denied to have a certain efficiency since the present commercial air diving tables have an overall safety record around 0.5% DCS incidence (12). However, their initial ascent strategy can be questioned from the above accounts. These models work on tissue gas load. Their logic is to rapidly ascent to a stop close to the surface to create an off-gassing gradient. However, thinking in terms of bubbles instead of tissue gas load, the logic would rather be to slow down the ascent to the control bubble growth (13). Doppler detection studies with air diving up to 40m have already documented differences in ascent rate procedures (14, 15, 16). Effectively, we have noted that successful bounce decompressions tend to slow the initial ascent down by using (figure 4):

*** simply slower rates of ascent to the first stop,**
*** varying rates of ascent, from 15 m/min to as slow as 6 m/min,**
*** deeper decompression stops**
*** mid-depth stops**
*** a combination of the above.**



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It is difficult to document the efficiency of such strategies. Most of the cases reported are uncontrolled information that constitutes more anecdotes than scientific data. However, one can consider that for deep bounce tables, the criticality of the exposure replaces statistical significance. Then, there is an obvious influence of the depth. An advanced ascent strategies will not change much the safety performances of an air dive to 21 meters and data from traditional air diving are not expected to provide evidences on the matter. Finally, one can suppose that the nature of the inert gas also play a role, as helium bubbles seems to have a different behavior than the nitrogen ones. In that respect, trimix dives become difficult to analyze. To the best, one can accept the above information as a trend that could be scientifically documented in near future. This leads to reject the classic models and search for new assumptions. The arterial bubble assumption was used to propose an explanation.

The Arterial Bubble Assumption

The very idea of arterial bubbles can be tracked to page 352 of Haldane's 1908 publication where he wrote:
"If small bubbles are carried through the lung capillaries and pass, for instance, to a slowly de-saturating part of the spinal cord, they will there increase in size and may produce serious blockage of the circulation or direct mechanical damage".

Closer to our time, in paper a published in 1971, Hills (17) was able to show, using an animal model, that DCS symptoms could change from Type I to Type II by changing from continuous decompression to surface decompression. This elegant experiment demonstrated the existence of a different mechanism for the onset of Type II DCS which was later accounted for by arterial bubbles. The arterial bubbles were first detected and their possible role discussed by the scientists running Doppler detection studies (18, 19). The model was then proposed by James for the onset of CNS and spinal symptoms (20, 21). It was used to discuss the cerebral perfusion deficit in divers who had symptoms mainly referable to the spinal chord (22) and the role of a Patent Foramen Ovale in the diver's susceptibility to Type II DCS (23, 24). Finally, Hennessy published in 1989 all the physical aspects of the arterial bubbles scenario in a remarkable paper (25) that became the foundation of the Arterial Bubble assumption

The critical issue in the Arterial Bubble assumption is the filtering capacity of the lung (figure 5). The threshold radius is suspected to be the size of a blood cell. Publications on intra-vascular ultrasound techniques using a contrast agent called "Levovist" confirmed this assumption. The agent contains stable gas bubbles trapped in galactose that have a calibrated size distribution from 3 to 8 microns. The contrast agent is injected intra-venously and measurements are made few minutes later in the cerebral, renal and lower limb arteries (26). If Levovist bubbles up to 8 µ can freely cross trough the lungs, it means that decompression bubbles can pass to the arterial side, especially during the initial phase when bubbles are small. Later in the ascent, bubbles that are detected by

ultrasound Doppler on the venous side have a much larger size (20-30 microns). They obviously are trapped at the lung as conventional Doppler measurements have not permitted to detect any bubbles on the arterial side.

The first merit of the Arterial Bubble assumption is to introduce variability in the decompression outcome through the lung function. The first variable is individual susceptibility. It is reasonable to accept that the filtering capacity of the lung may vary from persons to persons, and for one individual, from one day to the other. It thus accounts for the inter-individual variability (age, fat content, smoking, etc.) and intra-individual variability (fatigue, hang over, etc.) which have been observed for long in DCS susceptibility. Basically, a good diver is a good bubble filter. It is a justification for divers who claim top physical fitness for severe decompressions.

The second variable is related to dive conditions. It is reasonable to speculate a possible role of CO2 on the lung filter. CO2 could decrease its filtration capacity and cause bubbles to pass to the arterial side of the circulation. Thus diving situations that produce CO2 retention or hypercapnia should be associated to a higher risk of Type II DCS. It could explain why the following situations, which are all related to high levels of CO2, have been identified as contributing factors to DCS:

* **anxiety and stress, either because of the dive conditions or of insufficient training,**
* **exhaustion or hyperventilation due to intense activity or work at the bottom,**
* **cold at bottom or during decompression,**
* **breathing resistance due to the poor performances of the regulator (bad maintenance or adjustment, insufficient flow).**

The second advantage of the Arterial Bubble assumption is that is also consistent with accidental production of arterial bubbles.

1. Diving requires breathing a compressed inert gas that dissolves in the various tissues during the bottom exposure. When the ascent is initiated, the compartments off-load the inert gas as soon as a gradient is created.

2. Bubbles are normally produced in the vascular bed and transported by the venous system to the lung.

3. The lung works as a filter and stops the bubbles in the capillaries by an effect of diameter. Gas transfer into the alveoli further eliminates the bubbles.

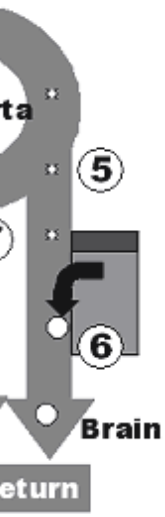
4. The critical issue is the filtering capacity of the lung system. Small bubbles may cross the lung and passes into the arterial system.

5. At the level of aorta cross, the distribution of blood is such that the bubble is likely to reach a neurological tissue such as the brain or the spinal cord.

6. The brain is a fast tissue and might be in supersaturated state in the early phase of the decompression. It acts as a gas reservoir and feeds the bubble that starts growing. The bubble may just proceed to the venous side for another cycle. It may also grow on place causing major alteration of the blood supply and finally ischemia. The consequence will soon be central neurological symptoms.

7. Similarly, arterial bubbles may also reach the spinal cord and grow on site from local gas and produce spinal neurological symptoms.

8. Much later in the decompression, bubbles may reach a significant size and exert a local pressure, specifically in dense tissues such as tendons and ligaments that excites nerve terminations and produces pain.



One scenario is a shunt at the heart or lung level that accidentally passes bubbles from the venous to the arterial side. A vast literature is now available on the subject but the latest conclusion is that a permeable patent foramen ovale (PFO) only opens in certain conditions (27, 28). A permeable PFO conveniently explains neurological accidents after recreational air diving without procedures violation. However, it does not fit the scheme for vestibular hits in deep diving. A first reason is that a deep diver generally has a long diving career and a diver with a permeable PFO would not be expected to follow it without any warning. The second is that vestibular symptoms can appear very early in the decompression, long before the massive bubble production required to overload the system.

A second scenario corresponds to pressure variations during decompression that reduce bubble diameters. Bubble trapped in the lung during a normal decompression process can suddenly pass through the capillaries and become responsible for Type II DCS symptoms. This scenario has been proposed to explain the difference in safety performances of in-water decompression versus surface decompression. Data collected in the North Sea have shown that if the overall incidence rate of the two diving methods is about the same, but that surface decompression tends to produce ten times more type II DCS than in-water decompression. The assumption is that at the moment the diver ascends to the surface, bubbles are produced that are stopped at the lung level. Upon recompression of the diver in the deck chamber, these bubbles reduce their diameter due to the Boyle's law

and pass to the arterial side, later causing neurological symptoms. A similar scenario has been proposed for type II DCS recorded after yoyo diving or multiple repetitive diving (29).

The last advantage of the Arterial Bubble assumption is that it provides an explanation for the criticality of the initial ascent phase. Bubbles associated to symptoms are not necessarily generated on site. There is an amplification process at the beginning of the ascent that may last for several cycles. Once the bubbles have reached a critical size, they are either filtered in the lung or stopped at the tissue level. It is believed that the showering process of small arterial bubbles during the first minutes of the initial ascent prepares the prognostic for further DSC symptoms. An attempt was made to turn this scenario into a decompression model.

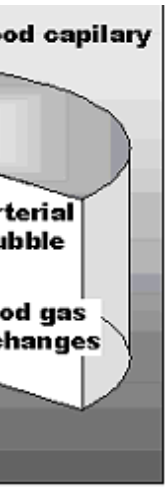
Definition of the critical case

Setting the scene for a decompression model, simplifications and restrictions must be specified:

* The divers' population targeted must correspond to "standard divers". Although such persons do not exist, it is assumed that they have a normal lung filtering function and that they have no physiological dysfunctions such as a cardiac or pulmonary shunt to accidentally pass bubbles through.
* The dive considered should also have "a standard condition" because our level of understanding does allow us to quantify the effect of fatigue, hyperventilation, cold, anxiety, etc. These conditions should be taken into account by applying the usual safety margins.
* Finally, the dive procedures must correspond to one "single square dive". Variations in the dive procedures such as yoyo diving, repetitive diving, multi-level diving introduce disturbances in the normal bubbles distribution that requires further assumptions.

From the above analysis, the critical case is defined as the arrival of an arterial bubble passing by a tissue compartment. Again, a series of simplifying assumptions is introduced (figure 6):

* The bubble was formed elsewhere. Its growth did not modify the local tissue gas load.
* The bubble is reputed to be small when compared to the tissue gas capacity, at least at the beginning of the decompression process. It does not change the tissue perfusion time response.
* Stuck in place, the bubble exchanges gases with both blood and the adjacent tissue.
* However, the bubble is stable and keeps a critical volume.



al case.

Complementary assumptions could be added that will generate a family of solutions. However, as the validation of these models is done by data fitting, the mathematical expression should remain simple and the number of parameters should be kept minimum.

See the derivation of the AB Model in Appendix.

Validation of the model

Validation of a model should follow the recommendations of the UHMS Workshop on Validation of Decompression Tables (39). The validation should range from laboratory testing to operational evaluation. Scientists refer to the convention of Helsinki for human experiment and proceed with informed consent forms. However, nowadays, ethical committees will exclude manned trails from almost any decompression study. Commercial diving, that was the main driving force during the 70's and 80's, has been ruled by laws, industry recommendations and social pressure. This normal maturation process of an industry has proved very limiting too. The last time a decompression problem was identified in the North Sea, it was solved by forbidden diving in the area of concern (40). Since then, the collapse of the offshore diving has reduced money available for diving research in many countries. The precious diving databases were lost along with any hope of official support for the validation of decompression tables. The lack of manned experiments or documented operational experience makes new models difficult to validate. The only remaining possibility for tables design is to analyze old data with new models.

AB Model-1: validation of the 1992 French air tables

Comex had used on its worksites the first version French official air decompression tables, called "MT74 Tables", that were published in 1974 by the "Ministère du Travail" (41). In 1982, the French government supported a research project for the evaluation of their safety performances using the computer treatment of the dive reports. The database analysis showed that the MT74 tables had limitations for severe exposures (12). The French government then supported a second research project to edit and validate new tables. A complete set of air tables, offering pure oxygen breathing at 6m (surface supplied), at 12m (wet bell), surface decompression, split level diving, repetitive diving, etc. was developed in 1983.

The model used to compute the tables implemented the concept of continuous compartment half-times. For the safe ascent criteria, the formulation of equation E11 was suspected but not established mathematically. An

approximation was defined empirically by fitting mathematical expressions to selected exposures stored in the Comex database. At the time, the best fit was obtained by the expression E12 below, now called "AB model-1". It is similar to the equation E11 when the last term is neglected.

E12 $$Ptis_{gas} \leq (1 + \frac{A}{T})(Pamb + B)$$

The values obtained by data fitting for the coefficients were A= 8 min and B= 0.4 bar. The AB model-1 was used to compute a set of decompression tables that was sent offshore for evaluation on selected Comex worksites. In 1986, after some minor adjustments, the tables were finally included in the Comex diving manuals and used as standard procedures (42). Later in 1992, the tables were included in the new French diving regulations under the name of "Tables du Ministère du Travail 1992" or "MT92" tables (43). The safety performances of the new AB model-1 are documented in table 1 after their offshore validation with air diving.

Table 2.
Comparison of the safety performances of the MT74 and the MT92 French air tables. The dives are classified in moderate, standard and severe exposures according to the Prt Index (5). The exposures correspond to in-water decompressions and exclude any surface decompressions. The data permit to quantify the safety performance improvement that is especially significant for type I DCS and severe exposures. Results on type II DCS are inconclusive as the general level of incidence is very low.

Exposures	Moderate (Prt<=25)		Standard (25<Prt<=35)		Severe (Prt>35)	
Tables	MT74	MT92	MT74	MY92	MT74	MT92
Dives	17,683	7,129	9,590	8,384	2,426	2,055
Number of Type I DCS Percentage of cases	18 0.10%	1 0.001%	55 0.57%	12 0.14%	49 2.2%	17 0.82%
Number of Type II DCS Percentage of cases	1 0.006%	0 0.00%	1 0.01%	1 0.01%	1 0.04%	2 0.09%

AB Model-2: calibration with Comex deep experimental dives

The name "AB model-2" was given to the full expression of equation E11. Used in its full extend, the expression can be compared to a normal M-value associated to a corrective factor that tends to reduce the permitted gradient for small values of T. Its role is similar to the "Reduce gradient factor" method of calculation presented by Backer (44) or the reduction factor of the rgbm model (45). These compartments correspond to fast tissues that effectively direct the initial phase of the ascent. The overall consequence is much deeper stops. The initial rate of ascent to the first stop is not part of the model control and was set arbitrarily to 9-6 m/min. The combination of the deep stops and slow ascent rates gives decompression profiles with features identified above for successful decompressions. The AB model-2 was calibrated by data fitting on a series of Comex offshore and experimental dives using both air, nitrox and heliox gas mixtures. As an example, the fit obtained for the test dive to 180m/120min already mentioned in the above paragraph is presented in figure 10. The AB model-2 succeeded in introducing the deep stop at least to 153 m that is very close to the actual one at 162m.

Discussion and conclusion

* It is admitted that the shape of the decompression generated has prevailed over the theoretical basis in the derivation of the AB model-1 and 2.
  However, a few points must be recognized in favor of the AB models:
* The concept of the arterial bubbles seems to be a way of designing tables with deep stops as we nowadays believe they should have.
* The model in its simplified form (AB model-1) has been effectively validated in controlled conditions with air diving at the time it was still possible to do so with diving data-bases.
* The mathematical formulation, based on a continuously varying compartment half-time, requires a minimum of parameters, and somehow proves the relevance of the assumptions.
* As such, the AB models have a surprising prediction capacity as they were successfully calibrated with air and heliox from 0 to 180m.

Appendix

a) Derivation of the AB Model

Decompression models are all based on the same structure (figure 7). The assumptions in the various boxes may vary from one author to another but the logic remains the same. The input parameters correspond to the operational conditions: bottom depth, bottom time, and gas protocol. They set the initial and boundary conditions for the various equations. The gas exchange model serves to evaluate the amount of available gas at the site. A second model is used to describe the gas exchanges between the bubble and its surrounding environment. Both models may interact depending on the assumptions. The safe ascent criteria is a decision on the critical

phenomena to be controlled during the ascent. It could be super-saturation as in the Haldane's model, the volume of the gas phase in the critical volume hypothesis (30), or the bubble size, the rate of growth, etc.

b) Tissue gas exchanges model

Models require parameters to be defined. The number of parameters, or degrees of freedom, increases with the complexity of the model. It is currently admitted that models with a large number of parameters are purely descriptive models and that models with a limited number parameters, corresponding to more pertinent assumptions, are more predictive.

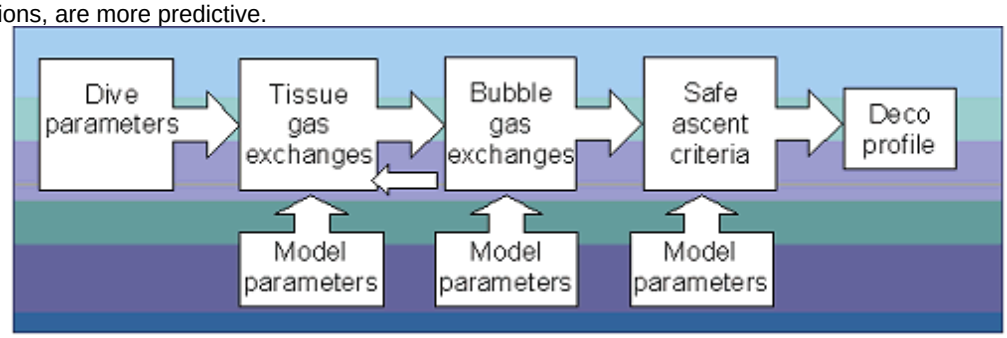


Figure 7 : structure of a decompression calculation model.

The consequence is that when the number of parameters increases, the domain of validity of the model shrinks. Classic models used in table calculations are obviously over dimensioned in terms of parameters. Typically, the Buhlman model (31) uses 16 compartments to calculate air tables. Considering the half-time and the two "a" and "b" coefficients for the safe ascent criteria of the various compartments, the model uses a total of 36 parameters to run. Although these tables are among of the best air tables available, it took Dr. Buhlman a lifetime to adjust these parameters. In addition, the model is fine as long it is used in its domain of validation that is mainly recreational diving. It could not be extrapolated for instance for very long bottom times as used in commercial diving.

The tissue gas exchange model is generally the main source of parameter proliferation because of the concept of tissue compartments. Tissue compartments are just an historical approach and their identification is not important. A series of compartments avoids the difficulty of accurately defining the process of the gas exchanges, would it be perfusion, diffusion or combined perfusion and diffusion. The exponential compartments are considered as harmonics of a complex mathematical solution that are controlling the decompression one after the other. For thisreason, we used the general classic expression:

E1 $$\frac{dPtis_{gas}}{dt} = \frac{0.693}{T}(Pa_{gas} - Ptis_{gas})$$

Where T is the compartment half-time as defined in the perfusion equation, Pa and Ptis are the arterial and tissue inert gas tensions.

The modern trend in table computation is to consider all the possible compartments and treat their half-time as a continuous variable. The difficulty then is to express the safe ascent criteria in terms of the compartment half-time, an exercise that was solved only in simple cases (32). Because modern computers are fast, the solution adopted was to keep treating tissue compartments individually but express them with a geometrical series to remove any subjectivity in their selection. We used the Renard's series, named after a French admiral who faced the standardization of ropes, sails, planks, etc. in navy arsenals, and elegantly solved the problem with a progression based on a square root of 10. For instance, with 10 values per decade (), the series gives the following values:

10 - 12.5 - 16 - 20 - 25 - 32 - 40 - 50 - 63 - 80 - 100

Experimentally, we found that the computations become stable when the number of compartment is set between 15 to 20 values per decade. This way, the description of the tissue gas exchange model only requires defining the boundaries. The fastest compartment obviously corresponds to instant equilibration and does need to be specified. The slowest compartment is defined as the one used in saturation decompressions. Based on Comex saturation experience, these values were set to 270 minutes for heliox and 360 minutes for nitrox saturation (33). Finally, the tissue gas exchange model only requires one parameter to be defined, corresponding to the half-time of the slowest compartment.

c) Bubble gas exchanges model

All gases present in the surrounding of the bubble participate to the gas exchanges. Metabolic gases play an important role and especially CO2 (34), the presence of which has been reported in bubbles as soon as Paul Bert. However, Van Liew demonstrated in his the experiments that the permeations of O2 and CO2 are very rapid

(35). If local tension of CO2 and water vapor are well documented in the surrounding tissue, the local values of the oxygen tension might become a problem, especially if various decompression mixtures are use during the ascent. As a first approximation we took the bubble oxygen pressure equal to the tissue oxygen tension and constant. However, when the decompression gas protocol is aggressive, or if pure oxygen is breathed in the last stops, this simple approach gives rapid changes of ascent rates that are incompatible with our operational experience. In an improved version of the model, the bubble oxygen partial pressure was computed as a function of the ambient pressure and the metabolic oxygen extraction (36, 37) and using the mathematical derivation of the oxygen window proposed by Egi (38).

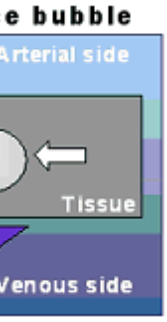
To cope with the complexity of the inert gas exchanges in the bubble, we decided to simplify the process by considering two extreme situations (figure 8). In one case, the bubble is purely vascular and remains on place. The blood flows around it and exchanges gas by convection so efficiently that there is no laminar layer and no diffusion delay at the bubble interface. In these conditions, we adopted for the bubble gas exchanges a formulation similar to the classic tissue perfusion equation. We further assumed that the blood flow draining the bubble is a small fraction of the tissue perfusion and that the blood leaves the bubble equilibrated with its gas pressure. This permitted to arbitrary express the quantity of inert gas molecules transiting through the bubble interface into the blood as:

E2 $$\frac{dn,blood_{gas}}{dt} = C\frac{0.693}{T}(Pa_{gas} - Pb_{gas})$$

Where dn,bloodgas is the number of molecules of inert gas passed from the bubble into the blood, Pagas the arterial inert gas tension, Pbgas the bubble inert gas pressure, T the compartment half-time and C a coefficient that accounts for the fraction of the tissue blood perfusion that governs these exchanges, the relative capacity of the bubble to the surrounding tissue, etc.

In the second case, the bubble is purely extravascular. The bubble exchanges gas with the surrounding tissue by diffusion. We used the classic assumption of a linear gradient in a surrounding shell and obtain a second general expression for the number of inert gas molecules diffusing through the bubble interface into the tissue.

E3 $$\frac{dn,tis_{gas}}{dt} = \frac{1}{K}(Ptis_{gas} - Pb_{gas})$$



Where dn,tisgas is the number of molecules of inert gas diffusing from the tissue into the bubble, Ptisgas the tissue inert gas tension, Pbgas the bubble inert gas pressure, K a coefficient that accounts the diffusibility of the gas, the thickness of the layer, the surface of the bubble, etc.

Finally, we imagined an intermediate situation where the bubble is at the interface between the blood and the tissue and exchanges gas through the two above mechanisms. The importance of the exchange varies with the relative area of the bubble exposed to each media. The ratio between the two exposed areas of the bubble is called (and varies from 0 to 1. The inert gas mass balance of the bubble becomes:

E4 $$\frac{d(PbVb)}{dt} = \frac{1}{R\tau}(\alpha\frac{dn,tis_{gas}}{dt} + (1-\alpha)\frac{dn,blood_{gas}}{dt})$$

Where R is the gas constant, (the absolute temperature and Vb the volume of the bubble.
Safe ascent criteria.
The ascent criteria simply seeks the stability of an arterial bubble, with a critical size, stuck at the interface of the blood vessel and exchanging gas with both the blood and the tissue. We translated this statement by specifying that the overall mass balance of the arterial bubble remains unchanged in these conditions:

E5 $$\frac{d(PbVb)}{dt} = Pb\frac{dVb}{dt} + Vb\frac{dPb}{dt} = 0$$

At a constant ambient pressure, corresponding to the situation of a decompression stop, the stability of the bubble requires two conditions. The first one is that the bubble volume remains constant $\frac{dVb}{dt} = 0$ and the second that the various pressures are balanced at the surface of the bubble $\frac{dPb}{dt} = 0$.

This last condition means that the sum of all the internal gas pressures equals the external ambient pressure plus the stabilization pressures (surface tension, skin elasticity, tissue compliance). This is written as:

E6 $$Pb_{gas} + Pb_{O2} + Pb_{H2O} + Pb_{CO2} \leq Pamb + Pstab$$

Where Pbgas, PbO2, PbH2O, PbCO2 are respectively the pressures of the inert gas, oxygen, water vapor and CO2 inside the bubble, Pamb the ambient pressure and Pstab the sum of the various stabilization pressures. Assuming PbO2 is constant and equal to the tissue oxygen tension and introducing B, a coefficient of obvious definition, we obtained a simpler form:

E7 $$Pb_{gas} \leq Pamb + B$$

In these conditions, the overall of gas transfers between the bubble and its surrounding is balanced. The same amount of molecules of inert gas must enter and leave the bubble during a unit of time. There is no gas accumulation inside the bubble. Equations E5 and E4 become:

E8 $$\alpha \frac{dn,blood_{gas}}{dt} = -(1-\alpha)\frac{dn,tis_{gas}}{dt}$$, and yields:

E9 $$\frac{\alpha}{K}(Ptis_{gas} - Pb_{gas}) = -(1-\alpha).C\frac{0.693}{T}(Pa_{gas} - Pb_{gas})$$

Finally, equation E10 and E8 are combined to eliminate Pbgas. After defining a coefficient A as:

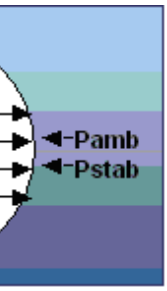
E10 $$A = 0.693.\frac{(1-\alpha)}{\alpha}.C.K$$

The final expression of the safe ascent criteria becomes:

E11 $$Ptis_{gas} \leq (1+\frac{A}{T})(Pamb + B) - \frac{A}{T}Pa_{gas}$$

Equation E11 sets the condition for a safe ascent to the next stop according to the initial hypothesis: an arterial bubble, exchanging gas with blood and tissue that keeps a critical size during the ascent. It is a function similar to an M-value. With the tissue compartment tension evolution defined in E1, it permits the classic computation of a decompression stop time. This computation model is later referred as the Arterial Bubble model or "AB" model.

The primary interest of the ascent criteria in E11 is that it is expressed as a function of T and thus can be defined continuously for each compartment. This definition is also extremely efficient in terms of parameters, as it only requires determining (when PbO2 is taken as a constant) the values of the A and B coefficients. The total number of model parameters reduces to three coefficients that are the longest tissue half-time and the two A and B coefficients.



volved in the
bble surface.

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