THE ARTERIAL BUBBLE MODEL FOR DECOMPRESSION TABLES CALCULATIONS

JP Imbert, D Paris, J Hugon
Divetech, France

1. Introduction

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 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 initial rapid ascent on a relatively high distance. However, the drive for table development is operational pressure. Aside the information circuits, marginal divers' communities have empirically developed original decompression procedures for their needs. 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 and build them into a model to obtain the typical profile of these successful decompressions.

2. Background

2.1. Decompression safety

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). However, 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 of DCS.

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 "non-serious" 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 "serious" decompression accident because the diagnosis may not be easy, the treatment is often delayed, and the consequences can be dramatic.

Finally, the risk of DCS can be further appreciated in terms of neurological 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 (typically 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 profile was accentuated 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 bounce tables indicates that the dive profile controls the bubble scenario and decides on the safety outcome of the decompression. As the profile is related to the exposure, it seems that, with the existing tables:
– Short bottom time tables (10-30 min) characterized by a rapid ascent to shallow stops produce vestibular symptoms,
– Average bottom times tables (30-90 min) produce other neurological symptoms (there is even a suspicion that helium would preferably yields central and nitrogen spinal symptoms),
– Long bottom times tables (120 min) and saturation diving, associated to slow ascents, produce type I pain only DCS in the last meters of the decompression.

Therefore, we have approached the table calculation with the following vision of the problem:
– The DCS risk must be appreciated in terms of symptoms,
– Each symptom depends on a different bubble scenario,
– Each bubble scenario must be associated to a different model.

2.2. Safe decompression strategies
Divers have known for long the criticality of the initial ascent in decompression. In fact, each diver's group has generated its own empirical strategies to improve their decompression safety.

2.2.1. *Rate of ascent*
The very first adaptation is the reduction of the ascent rate when ascending to the first stop. It is a common practice in commercial diving. Companies generally use the US Navy tables as standard procedures but most of the diving supervisors admit they select slower rates than the 60 ft/min stated in the manual. Following the same idea, in the 1986 revision of the Comex air tables, the rate of ascent was reduced empirically from 15 to 12 $m.min^{-1}$. Similarly, in 1990, the French Navy reduced the ascent rate from 18 to 12 $m.min^{-1}$ in the revision of their air tables. Later, the benefit of slow ascent rates was demonstrated in recreational diving using Doppler bubble detection (9, 10).

2.2.2. *Deep stops*
Another common modification to tables is the introduction of a deep stop.
One of the earlier accounts we know comes from two old coral divers who worked in the 50's. They told they used to stop at mid-depth during their ascent from deep dives to sing a gospel. They added that they would adapt the number of verses to how comfortable they felt with the decompression. Obviously these old timers knew things we ignore for it is difficult to either imagine how the decompression stress feels physically or 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 (reason unknown). Deep stops were later advertised by Richard Pyle in articles published in the "AquaCorps" and "Deep Tech" magazines (2) and supported by other authors influent in the technical divers' community (11). Today's, deep stops are so popular that most decompression software's propose one or two arbitrary deep stops during the ascent (Deco-planner, GAP). DAN is presently conducting an evaluation program to assess the influence of deep stops with Doppler bubble detection (12).

2.2.3. *Mediterranean coral dives*

Red coral jewels are a tradition around the Mediterranean. 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 80-90 m on air. Coral divers use a variety of decompression procedures that they have developed empirically and are very reluctant to give them away. Over the years, we 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 80 m, 20 minutes at 90 m 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 12 m in the water, sometimes at 15 m for few minutes. Some of them also have a chamber on board in which they finish their ascent after surface decompression.

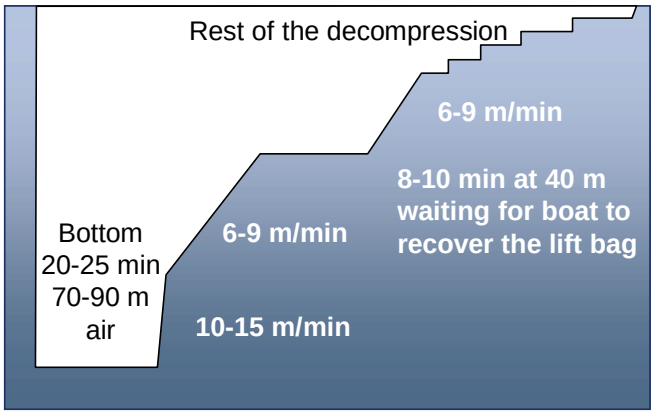

Figure 1: a typical dive profile of a coral diver

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 (fig. 1). Coral divers 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 $m.min^{-1}$) 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.

2.2.4. *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 180 m, 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. 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 180 m / 120 min dive that required 48 hours of decompression and which appeared extremely safe (figure 2). A total of 49 divers went through the selection tests without any symptom (13). 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 (fig. 2). Although the method was deliberately empirical, it gave varying initial ascent rates that progressively turned into short deep stops. The result was outstanding when considering similar deep bounce dive trials at the Duke University (14). The French Navy, who attempted to design deep bounce tables in 150-180 meters range at the same time (unpublished) only succeeded in bringing the divers safely back to surface by using a near saturation decompression profile.

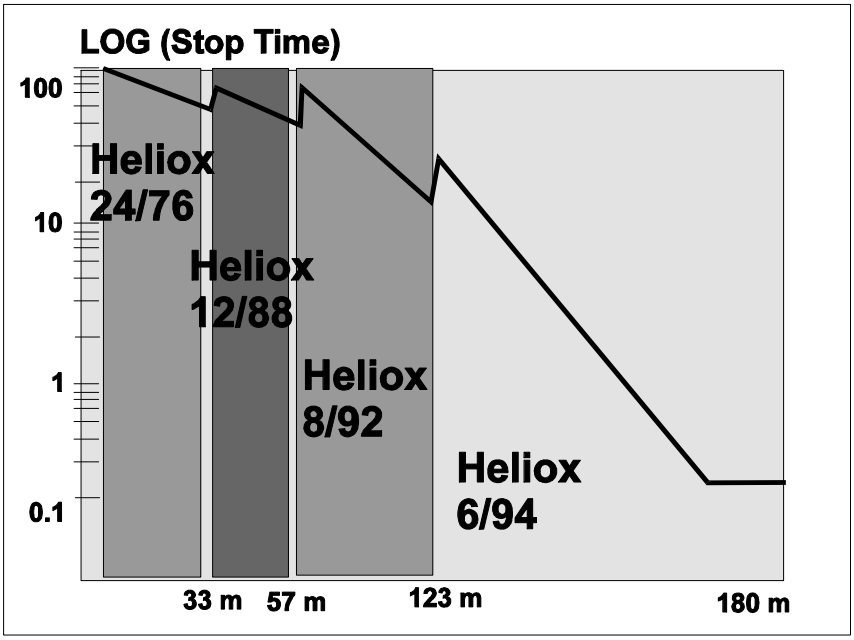

Figure 2: Semi-log plot of the 180 m / 120 min test dive carried at Comex Hyperbaric Center in 1977. In this diagram, the x-axis represents the depth. The y-axis represents the logarithm of the stop time, an information related to the rate of decompression. As the diver gets close to the surface, the stop time increases. The various decompression mixtures introduce abrupt change in the decompression rate that appears as peaks in the ascent diagram.

2.2.5 *Possible ascent strategies*
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. We consider them as operational trends that could be scientifically documented in near future. We have noted that improved bounce decompressions tend to slow the initial ascent down by using:
– simply slower rates of ascent to the first stop,
– deeper decompression stops or mid-depth stops
– a combination of the above.

These strategies are only relevant for deep bounce diving. They will not change the safety performances of an air dive to 21 meters. However, they might be important for a technical diver involved in a 20 minutes trimix dives to 75 meters dives. We have tried to identify and implement new assumptions that would support these successful decompression strategies.

3. Method
Present decompression models can be classified into two categories depending on their assumptions:
– Dissolved gas models, following the Haldane tradition, as for example the ZHL16 tables developed after the Buhlmann model (15).
– Gas phase models, such as the VPM model (16), which consider the bubble size or volume as the critical issue.
Each model has the inherent advantages and limitations of its mathematical formulation.

3.1. Dissolved gas models
These models correspond to classic approach of decompression and involve assumptions that are summarized below (fig. 3):

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 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.

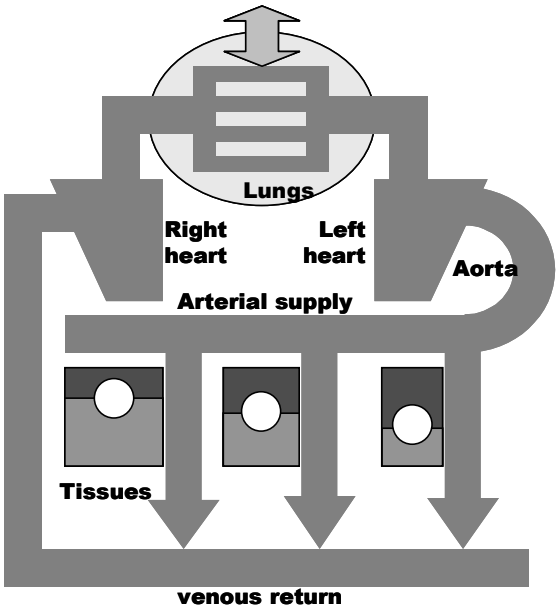

Figure 3: Dissolved gas decompression models.

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 (17). 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 (18).

3.2. The Arterial Bubble Assumption

The very idea of arterial bubbles can be tracked to page 352 of Haldane's 1908 publication (1) 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 (19) 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 (20, 21). The model was then proposed by James for the onset of CNS and spinal symptoms (22, 23). It was used to discuss the role of a permeable Patent Foramen Ovale (PFO) in the diver's susceptibility to Type II DCS (24, 25). Finally, Hennessy published in 1989 all the physical aspects of the arterial bubbles scenario in a paper (26) that became the foundation of the Arterial Bubble assumption (fig. 4).

The critical issue in the Arterial Bubble assumption is the filtering capacity of the lung (fig. 5). The threshold radius is suspected to be the size of a blood cell. This assumption is supported by a study on intra-vascular ultrasound techniques using a contrast agent called "Levovist" (27). The agent contains stable gas bubbles trapped in galactose that have a calibrated size distribution from 3 to 8 µm. The contrast agent is injected intra-venously and measurements are made few minutes later in the cerebral, renal and lower limb arteries. If Levovist bubbles up to 8 µm 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 grow to a larger size and remain trapped in the lung. This explains why 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 seek top physical fitness for severe decompressions.

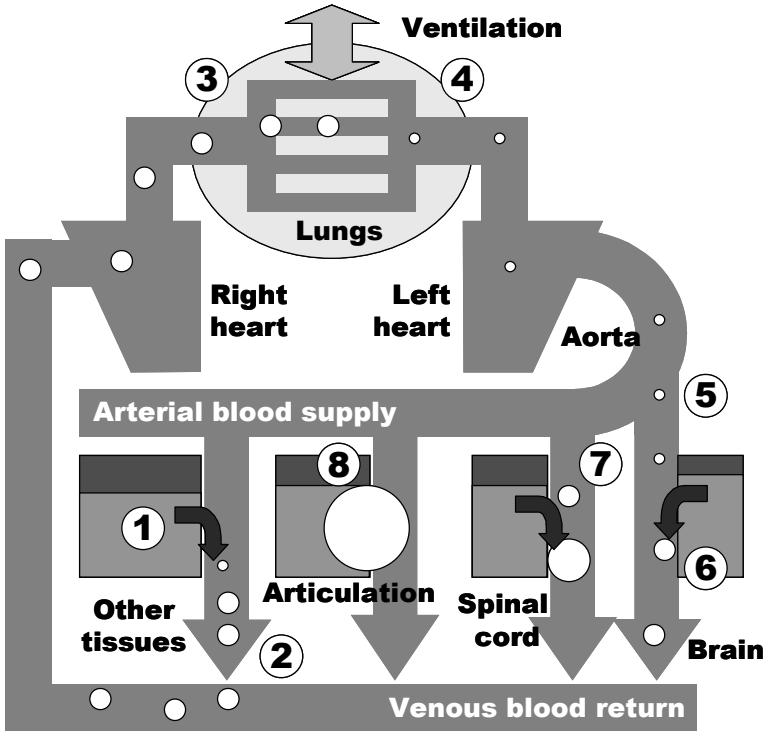

Figure 4: The Arterial Bubble Assumption

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.

The second variable is related to dive conditions. It is reasonable to speculate a possible role of CO_2 on the lung filter. CO_2 could decrease its filtration capacity and cause bubbles to pass to the arterial side of the circulation. Thus diving situations that produce CO_2 retention and 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 CO_2, 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.

One scenario considers 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 PFO only opens in certain conditions (28, 29). A permeable PFO conveniently explains neurological accidents after recreational air diving without any procedure 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 considers pressure variations during decompression that reduce bubble diameters. This way; bubble trapped in the lung during a normal decompression process could 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 (30). 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. The same scenario was proposed for type II DCS recorded after yoyo diving or multiple repetitive diving

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 DCS symptoms. An attempt was made to turn this scenario into a decompression model.

3.3. Gas phase model: derivation of the AB Model

A model is presented for the calculation of decompression tables based on the Arterial Bubble assumption. This model is later referred as the Arterial Bubble model version 2 or "AB Model-

2". Such a model obviously falls into the gas phase models category and its derivation is presented in the appendix.

The "AB Model-2" uses equation E1 for ascent criteria. The expression can be compared to a classic "M-value" associated to a corrective factor that reduces the permitted gradient for small values of the compartment time constant. Its role is similar to the "reduce gradient factor" method of calculation presented by Backer (31) or the reduction factor of the "rgbm" model (32). These compartments correspond to fast tissues that effectively direct the initial phase of the ascent. The consequence is the introduction of deeper stops than for example, a classic Buhlmann model decompression.

E1 $$Ptis_{gas} \leq (1+\frac{A}{T})(Pamb+B)-\frac{A}{T}Pa_{gas}$$

3.4. Validation of the model AB Model

Validation of the model should have followed the recommendations of the UHMS Workshop on Validation of Decompression Tables (33). The full validation ranges from laboratory testing to operational evaluation. When human evaluation is concerned, scientists refer to the convention of Helsinki and proceed through a heavy ethical protocol. However, nowadays, ethical committees will exclude manned trails from almost any decompression study. The offshore industry that was the main driving force for tables' development during the 70's and 80's now relies on ROV's and has dropped diving. The diving databases ceased functioning in the 90's (32). The lack of manned experiments or documented operational experience make new models difficult to validate. The only remaining possibility for table validation is to analyze old data with new models. Part of the study was supported by the French Navy and therefore access was granted to the French Navy track records and Comex data base for the validation of the AB-model.

4. Results and discussion

4.1. Comparison with the 1992 French air tables

In the early times, Comex 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" (35). 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 (17). The French government then supported a second research project to edit and validate new tables (36). A complete set of air tables, offering pure oxygen breathing at 6m (surface supplied), at 12 m (wet bell), surface decompression, split level diving, repetitive diving, etc. was developed in 1983.

This early model already implemented the concept of continuous compartment half-times. For the safe ascent criteria, the formulation of the Arterial Bubble model was suspected but not established mathematically. Instead, 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 E2 below, now called "AB Model-1".

E2 $$Ptis_{gas} \leq (1+\frac{A}{T})(Pamb+B)$$

The parameters were defined by data fitting and yielded 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 (36). 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 (37).

Obviously, there is continuity in the mathematical expression with the proposed version of the AB Model-2 as expressed in E1. Both models have a similar mathematical expression when the last term is neglected. They overlap in the standard air diving exposures and only diverge for deep and/or long exposures where the extra term makes the difference. It is thus possible to transfer the experience of AB Model-1 to the AB Model-2 in term of safety performances over the standard exposures. The safety performances of the AB Model-1 are documented in table I under the name of MT92 and compared to the former tables under the name of MT74.

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	18	1	55	12	49	17
Percentage of cases	0.1 %	0.1 %	0.57 %	0.14 %	2.1 %	0.82 %
Number of Type II DCS	1	0	1	1	1	2
Percentage of cases	0.006 %	0.00 %	0.01 %	0.01 %	0.04 %	0.09 %

Table I: 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.

4.2. Calibration with deep experimental dives

The AB Model-2 was first calibrated by data fitting on Comex offshore and experimental dives using both air, nitrox and heliox gas mixtures. When fitted to the 180 m / 120 min experimental dive mentioned previously, the AB Model-2 succeeded in introducing the deep stop at 153 m that is very close to the actual first stop at 157 m. The AB Model-2 was also calibrated using experimental bounce dives conducted by the French Navy.

4.3. Field validation

The AB Model-2 was also evaluated by informed technical divers pushing deep exploration dives. Over the recent years, they have reached a high technical level and achieved some outstanding explorations. Their leaders have used the AB Model-2 tables in the following occasions:

– June 1996, Pascal Bernabé, 163 m at Foux du Mas de Banal, 30 minutes of descent time;
– September 1996, Pascal Bernabé, 176 m at Fontaine de Vaucluse, 20 minutes descent time;
– September 1997, Pascal Bernabé, 250 m at Fontaine de Vaucluse, 25 minutes descent time;
–September 1998, Olivier Isler, 165 m at Bourg Saint Andéol.

Pascal Bernabé also participated as a support diver to the breath-hold record set by Pipin at Cosumel in January 2000. During 15 days, he performed 10 dives between 130 and 163 m, with bottom times of 5 to 7 minutes. As Pipin was getting close to his record setting dive, he dived 153 m, 156 m, 163 m and 162 m in seven days. More recently, in October 2002, working with

the late Audrey Mestre, he made support dives to 143 m, 166 m and two dives to 170 m in a week.

Since 1999, the AB Model-2 has been used to calculate tables for coral divers. These tables use a simple gas protocol, with “heli-air” as bottom mix, air as deco mix and pure oxygen at the end of the ascent, as specified by the users. For two seasons, one crew in Corsica and a second in Tunisia have used the tables intensively. A typical coral dive would be 20-25 minutes at 100 m, and divers would repeat one dive a day as long as the weather permits it. In Tunisia, on specific sites, the tables have been used down to 140 m. The overall number of coral dives now exceeds one thousand, carried out by a dozen of coral divers, without only few articular problems reported, which is the least that could be expected. Although this is non-controlled information, the criticality of the exposure is such that a certain credit must be given to the AB Model-2.

5. Conclusion

The AB Model-2 is only a slow evolution of a former proved mathematical expression. It sort of summarizes a “French decompression tradition” cumulated over 30 years professional and recreational diving. The merit of the AB Model-2 is to rely on an original bubble scenario, the Arterial Bubble assumption that was formulated into simple mathematical terms.

The advantages of the AB Models are:

– The concept of the arterial bubbles seems to be a way of designing tables with deep stops.

– 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 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 Model has a good prediction capacity as it was successfully calibrated with air and heliox from 0 to 180 m.

6. References

1. Boycott, A.E., Damant, GC, Haldane, JS. The prevention of compressed-air illness. J. Hygiene, 8, 342-443.1908.

2. Pyle R. The importance of Deep Safety Stops. Deep Tech Magazine, Issue 5.

3. Imbert JP. Decompression safety. Proceedings of the Subtec '93 Conference. Volume 31, 239-249. Aberdeen, Scotland, November 1993.

4. Imbert JP, Montbarbon S. Use of the Comex Diving Data Base. Proceedings of the EUBS Workshop on Operational dives and decompression data: collection and analysis. Amsterdam, 17 August, 1990.

5. Shields TG, Lee WB. The incidence of decompression sickness arising from commercial offshore air-diving operations in the UK sector of the North Sea during 1982/83. Report to the UK Department of Energy, 1986.

6. Giles R. Decompression sickness from commercial offshore air-diving operations on the UK continental shelf during 1982 to 1988. Report from the UK Department of Energy. 1989.

7. Imbert JP. Evolution and offshore performances of the Comex Treatment Tables. Proceedings of the UHMS Workshop on Decompression Illness Treatment. Palm Beach, Florida, 18-19 June 1995.

8. Imbert JP, Montbarbon S. Presentation of the Comex Diving Data Base. Proceedings of the EUBS Workshop on Operational dives and decompression data: collection and analysis. Amsterdam, 17August, 1990.

9. Carturan D, Boussugues A, Habib G, Gardette B, Sainty J.M. Influence of ascent rate on venous bubbles detected after recreational dives. Proceedings of the International Joint meeting on Hyperbaric and Underwater medicine, Milan, Italy, 4-8 September, 1996.
10. Carturan D, Boussugues A, Burnet H and Gardette B. Effect of ascent rate on circulating venous bubble production during decompression in air sport diving. Bull. Mesubhyp 1998; 8:1-6.
11. Wienke BR. Deep stops.Advanced Diver Magazine. Issue 12. p30-34. 2002.
12. Marroni A, Benent PB, Balestra C, Cali-Corleo R, Germonpre P, Pieri M, Bonucelli C. What ascent profile for the prevention of decompression sickness? I Recent research on the Hill/Haldane ascent controversy. Proceedings of the 28th Annual Scientific Meeting of the European Underwater and Baromedical Society, Bruges, Belgium, 4-98 September, 2002.
13. Gardette B, Lemaire C, Lamy D, Le Chuiton J. Heliox bounce dive decompression (180m). Proceedings of the 5th Annual Meeting of the EUBS. Bergen, Norway, 5-6 July, 1979.
14. Bennett PB. Proceedings of the 9th Undersea medical Society Workshop on development of decompression procedures for depths in excess of 400 feet. Duke University, USA. 21-23 February 1975.
15. Buhlmann AA. 1984. Decompression-Decompression Sickness. Berlin: Springer-Verlag.
16. Yount DE, Hoffman DC. On the use of a bubble formation model to calculate diving tables. Aviat. Space and Environ Med. :57:149-156.
17. Imbert JP, Bontoux M. Safety analysis of French 1974 air decompression tables. Proceedings of the Undersea Medical Society Workshop on Decompression in surface-based diving. Tokyo, Japan, September 12 th, 1986.
18. Marroni A, Bennett PB, Balestra C, Cali-Corleo R, Germonpre P, Pieri M, Bonucelli C. What ascent profile for the prevention of decompression sickness? II- a field model comparing Hill and Haldane ascent modalities with an eye to the development of a bubble safe decompression algorithm. Proceedings of the 28th Annual Scientific Meeting of the European Underwater and Baromedical Society, Brugges, Belgium, 4-9 September 2002.
19. Hills BA. Decompression sickness : a fundamental study of surface excursion diving and the selection of limb bends versus CNS symptoms. Aerospace Medicine, Vol 42, No 8, August 1971.
20. Brubakk AO, Grip A, Holand B, Ornaheim J, Tonjum S. Pulsed Doppler ultrasound for studying haemodynamic changes and bubbles during simulated diving. Proceedings of the VIth Annual Congress of the EUBS. Cambridge, UK, 1981.
21. Mazurel G, Hee J, Giacomini L, Guillerm R. Ultrasonic detection of circulating bubbles in ewes exposed to simulated dives deeper than 800m under isobaric conditions and with unchanged gas mixtures. Proceedings of the XI th Annual meeting of the EUBS. Goteborg, Sweden, August 1985.
22. James PB. The size distribution of gas emboli arising during decompression. A review of the concept of critical diameter of gas emboli. Proceedings of the XIII th Annual Congress of the EUBS, Lubeck, Germany, 5-8 October 1982.
23. James PB. Decompression sickness. In Clinical Neurology. Edited by Michael Swash and John Oxbury. Churchill Livingstone. Vol. 1;565, 1991.
24. Moon RE, Camporesi EM, Kisso JA. Patent foramen ovale and decompression sickness in divers. Lancet 1; 513, 1989.
25. Wilmshurt PT, Byrne JC. Relation between interatrial shunts and decompression sickness in divers. Lancet, 1302-1306, 1989.
26. Hennessy TR. On the site of origin, evolution and effects of decompression microbubbles. Proceeding of the International Symposium on supersaturation and bubble formation in fluids and organisms. Trondheim, Norway, 6-10 June 1989.

27. Besnard S, Philippot M, Hervé Ph, Porcher M, Arbeille Ph: Intravascular ultrasound contrast agent particles in the cerebral, renal and lower limb arteries – consequence on diving physiology. Proceeding of the 28th Annual Scientific meeting of the European Underwater and Baromedical Society, Bruges, Belgium, 4-8 September 2002.
28. Balestra C, P Germonpre, and A Marroni. Intrathoracic pressure changes after Valsalva strain and other maneuvers: implication for divers with patent foramen ovale. Undersea hyperb. Med, 1998. 25(3): page 171-4.
29. Germonpre P et al; Patent foramen ovale and decompression sickness in sport divers. J. Appl. Physiol, 1988, 84(5): p1622-6.
30. Imbert JP. Decompression tables versus decompression procedures: an analysis of decompression sickness using diving data-bases. Proceedings of the XVII th annual meeting of Diving and Hyperbaric Medicine, Heraklion, Crete, Greece, 20 September-3 October 1991.
31. Baker EC. 1998 Understanding M-values. Immersed. Vol. 3, No 3.
32. Wienke BR. Modeling phase volume constraints under repetitive decompression. Math; Comput. Modelling. Vol. 16, No 3, pp. 109-120, 1992.
33. Conclusions of the 37th Undersea and Hyperbaric Medical Society Workshop on Validation of Decompression Tables. Bethesda, MD. 13-14 February 1987.
34. Shields TG, Duff P, Wilcox SE. Decompression sickness from commercial offshore air-diving operations on the UK continental shelf during 1990. Report from the UK HSE. 1992.
35. Mesures particulières de protection applicables aux scaphandriers. Fascicule Spécial no 74-48 bis. Bulletin Officiel du Ministère du travail. Imprimerie du Journal Officiel, 26 rue Desaix, 75732 Paris cedex 15.
36. Imbert JP, Bontoux M. A method for introducing new decompression procedures. Proceedings of the Undersea Medical Society Workshop on validation of decompression schedules. Bethesda, Maryland, 13-14 February 1987.
37. Travaux en Milieu Hyperbare. Mesures particulières de prévention. Fascicule no 1636. . Imprimerie du Journal Officiel, 26 rue Desaix, 75732 Paris cedex 15. ISBN 2-11-073322-5.
38. Gürmen NM, Llewellyn AJ, Gilbert RA, Egi SM. Simulation of dynamic bubble spectra in tissues. IEEE Trans Biomed Eng. 2001 Feb; 48(2):185-193.)
39. Hennessy TR, Hempleman HV. An examination of the critical release gas volume concept in decompression sickness. Proc. R. Soc. B179, 299-313, 1977.
40. Egi SM and Gurmen NM. Computation of decompression tables using continuous compartment half-times. Undersea Hyper Med 2000; 27(3):143-153.
41. Imbert JP, Bontoux M. Diving data bank: A unique tool for diving procedures development. 20th Annual OTC Conference. Houston, Texas, 2-5 May 1988.
42. Ishiyama A. Analysis of gas composition of intravascular bubbles produced by decompression. Bull. Tokyo Med. Univ. 30 :25-35, 1983.
43. Van Liew HD, Bishop B, Walder-D P, Rahn H. Effects of compression on composition and absorption of tissue gas pockets. J. of Appl. Physiol. 19865; 20:927-33.

The authors thank the cave and coral divers who helped in the operational validation of the deep part of these AB model tables.

6. Appendix

6.1. Working assumptions

Setting the scene for a mathematical 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 divers considered should also be in "standard diving conditions" because our level of understanding does allow us to quantify the effect of fatigue, hyperventilation, cold, anxiety, bad equipment, 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 profile such as yoyo diving, repetitive diving, multi-level diving introduce disturbances in the normal bubbles distribution that requires further assumptions.

From the Arterial Bubble assumption presented above, the critical case is defined as the arrival of an arterial bubble in a tissue compartment. Again, a series of simplifying assumptions is introduced (fig. 5):
– 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.

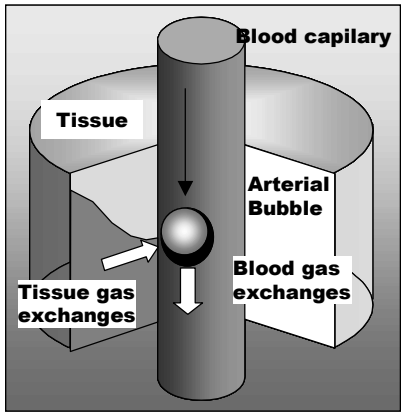

Figure 5: Definition of the critical case.

The limits of these assumptions are clearly understood:
– No information is available on the origin bubble. Arterial bubbles could in fact be just a local vascular bubble at a given stage of its development.
– The full dynamic of the bubble growth should be considered until it reaches its critical volume.
– A distribution of bubbles with various sizes should be considered instead of a single isolated bubble. This has already been solved for one single compartmental tissue array (38).

Additional 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 minimal.

6.2. Structure of the mathematical model

Decompression models are all based on the same structure presented in fig. 6. The assumptions in the various boxes may vary from one author to the other 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 are 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 (39), or the bubble size, the rate of growth, etc.

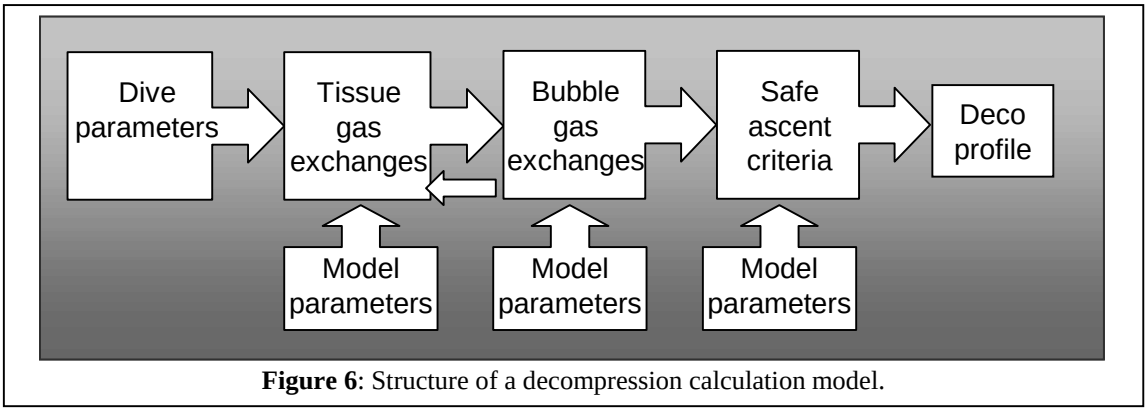

Figure 6: Structure of a decompression calculation model.

6.3. The tissue gas exchange 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 and that models with a limited number parameters, corresponding to more pertinent assumptions, are more predictive. 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 Buhlmann model (15) 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 48 parameters to run. Although these tables are among of the best air tables available, it took Dr. Buhlmann 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. The use of 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. Thus, in this model, the exponential compartments are considered as harmonics of a complex mathematical solution that are controlling the decompression one after the other. For this reason, we used the general classic expression for compartment gas exchanges:

E3 $$\frac{dPtis_{gas}}{dt} = \frac{0.693}{T}(Pa_{gas} - Ptis_{gas})$$

Where T is the compartment half-time as defined in the perfusion equation, Pa and Ptis, 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 (40). Because modern computers are fast, we decided to treat 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 ($\sqrt[10]{10}$), the series gives the following values:

10 – 12.5 – 16 – 20 – 25 – 32 – 40 – 50 – 63 – 80 – 100 minutes

Experimentally, we found that the computation becomes stable when the number of compartments 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 (41). Finally, the tissue gas exchange model only requires one parameter to be defined, corresponding to the half-time of the slowest compartment.

6.4. The bubble gas exchanges model

All gases surrounding of the bubble participate to the gas exchanges. Metabolic gases play an important role and especially CO_2 (42), 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 O_2 and CO_2 are very rapid (43). To cope with the complexity of the inert gas exchanges in the bubble, we decided to simplify the process by considering two extreme situations (fig. 7).

In one case, the bubble is purely vascular and remains in place. The blood flows around it and exchanges

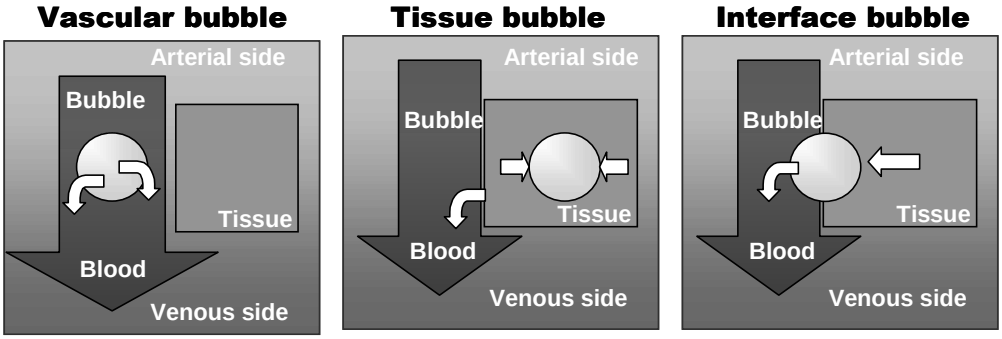

Figure 7: Possible bubble gas exchange situations

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:

E4 $$\frac{dn,blood_{gas}}{dt} = C\frac{0.693}{T}(Pa_{gas} - Pb_{gas})$$

Where $dn,blood_{gas}$ is the number of molecules of inert gas passed from the bubble into the blood, Pa_{gas} the arterial inert gas tension, Pb_{gas} 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 obtained a second general expression for the number of inert gas molecules diffusing through the bubble interface into the tissue.

E5 $$\frac{dn,tis_{gas}}{dt} = \frac{1}{K}(Ptis_{gas} - Pb_{gas})$$

Where dn,tis_{gas} is the number of molecules of inert gas diffusing from the tissue into the bubble, $Ptis_{gas}$ the tissue inert gas tension, Pb_{gas} 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 α and varies from 0 to 1. The inert gas mass balance of the bubble becomes:

E6 $$\frac{d(PbVb)}{dt} = \frac{1}{R\tau}(\alpha\frac{dn,tis_{gas}}{dt} + (1-\alpha)\frac{dn,blood_{gas}}{dt})$$

Where R is the gas constant, τ the absolute temperature and Vb the volume of the bubble.

6.4. The 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:

E7 $$\frac{d(PbVb)}{dt} = Pb\frac{dVb}{dt} + Vb\frac{dPb}{dt} = 0$$

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 volume remains constant $\frac{dVb}{dt} = 0$ and the second that the various pressures are balanced its surface $\frac{dPb}{dt} = 0$.

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:

E8 $$Pb_{gas} + Pb_{O2} + Pb_{H2O} + Pb_{CO2} \le Pamb + Pbstab$$

Where Pb_{gas}, Pb_{O2}, Pb_{H2O}, Pb_{CO2} are respectively the pressures of the inert gas, oxygen, water vapor and CO_2 inside the bubble, Pamb the ambient pressure and Pbstab the sum of the various stabilization pressures.

Assuming Pb_{O2} is constant and equal to the tissue oxygen tension and introducing B, a coefficient of obvious definition, we obtained a simpler form of the criteria:

E9 $$Pb_{gas} \le Pamb + B$$

In these conditions, the overall of gas transfers between the bubble and its surrounding are balanced. For each gas, the same amount of molecules enters and leaves the bubble during a unit of time. There is no gas accumulation inside the bubble. Equations E6 and E5 give:

E10 $$\alpha\frac{dn,blood_{gas}}{dt} = -(1-\alpha)\frac{dn,tis_{gas}}{dt}$$, and yields:

E11 $$\frac{\alpha}{K}(Ptis_{gas} - Pb_{gas}) = -(1-\alpha).C\frac{0.693}{T}(Pa_{gas} - Pb_{gas})$$

Finally, equation E9 and E11 are combined to eliminate Pb_{gas}. After defining a coefficient A as:

E12 $$A = 0.693.\frac{(1-\alpha)}{\alpha}.C.K$$

The final expression of the safe ascent criterion becomes:

E13 $$\boxed{Ptis_{gas} \le (1+\frac{A}{T})(Pamb + B) - \frac{A}{T}Pa_{gas}}$$

Equation E13 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 E3, it permits the classic computation of a decompression stop time. The rate of ascent to the first stop is not part of the model control and is set arbitrarily to 9 m/min.

The AB Model-2 provides deeper stops than for a classic decompression model. The combination of the deep stops and slow ascent rates gives original decompression profiles with features identified above for successful decompressions.