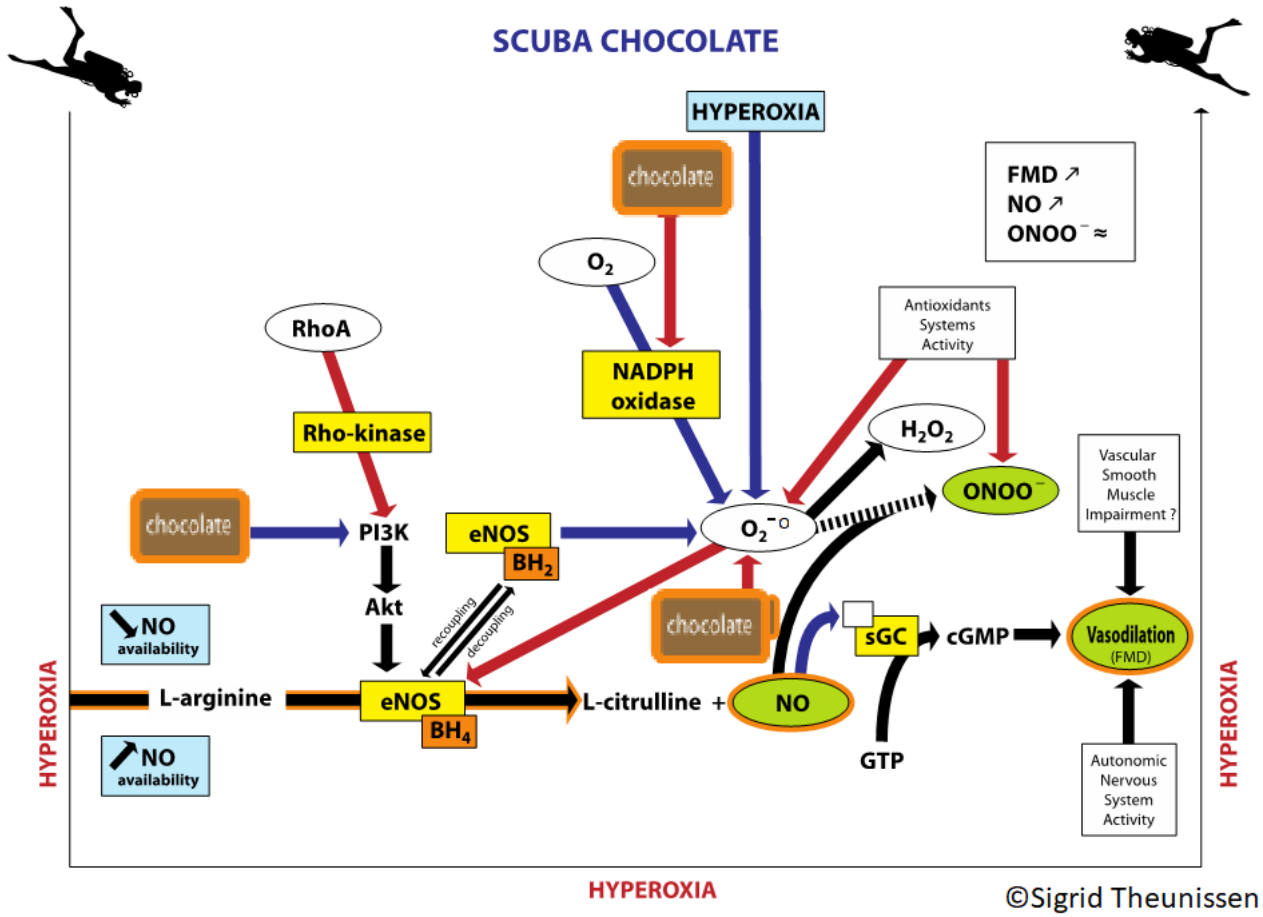
23th Capita Selecta Duikgeneeskunde

Volume 23

A comprehensive textbook of

21st Century decompression theory and DCI treatment;
from Haldane to BVM, endothelium response and Heliox treatment.

Edited by Nico A.M. Schellart



23th Capita Selecta Duikgeneeskunde,

Proceedings Published for the Symposium

21st Century decompression theory and DCI treatment; from Haldane to BVM, endothelium response and Heliox treatment.

for dive physicians, other care professionals and instructors

Edited by Nico A.M. Schellart

Academic Medical Centre, Amsterdam
Saturday 17 March, 2018

Contents

General information

23th Capita Selecta Duikgeneeskunde

Date: Saturday 18 March, 2017
Venue: Academic Medical Centre,
Meibergdreef 9 1105 AZ **Amsterdam**

Principal speaker
Adel Taher, GP, diving and hyperbaric physician, director Hyperbaric Medical Centre in Sharm el Sheik, Egypt.
Speakers
Jean-Claude Le Péchon, MSc, MEng, JCLP Hyperbarie, Paris, France.
Dr. Albrecht Salm, Dipl. Phys., SubMarineConsulting, Esslingen, Germany.
Dr. Sigrid Theunissen, Haute Ecole Bruxelles Brabant – ISEK. Environmental, Occupational & Ageing Physiology Lab. Brussels, Belgium.

Recommendation
The course is recommended by the expert group of dive medicine of the Vereniging voor Sportgeneeskunde (Soc Sports Med) and by the Nederlandse Vereniging voor Duikgeneeskunde (NVD, Dutch Soc Dive Med).

Accreditation
In general, the level of the various lectures / subjects of the meeting are (at least) in accordance with that of EDTC and ECHM, level 1 (Medical Examiner) and Level 2D (Diving Physician).
The program comprises **6 oral contact hours** and the NVAB, VSG, SCAS and NICDA (ECB representative in the Netherlands) are requested to provide 6 accreditation points for registrations as occupational physician, sports physician, sports diving physician and MED Level 1 respectively. Nearly all Dutch medical societies accept accreditation points “outside own specialism” through GAIA.
The Dutch course members who have registered for accreditation will obtain their cp’s via GAIA or will be applied for with NICDA and SCAS. All others obtain a certificate after completion of the whole course, including test.
Course members from outside the Netherlands should apply with their own accreditation office *on the basis of the requested MED Level 1 accreditation.* Diving Physician (Level 2D) is optional.

Programme committee
Nico Schellart (chair, medical physicist an diving physiologist), Tjeerd van Rees Vellinga (occupational and hyperbaric physician), Erik van der Sande (general and sport physician) and Marga Schweigmann (general and hyperbaric & diving physician), Peter Westerweel (internist-heamatologist) and ad hoc the lecturers.

Executive committee
Nico Schellart (course director) and Hans van Dam (administrative manager)

Responsibility
The Capita Selecta Duikgeneeskunde are given under the responsibility of the Academic Medical Centre, Univ. of Amsterdam (course leader Nico Schellart). The organization is by the Stichting Duik Research (SDR)[1)] and Biomed. Eng & Physics, AMC (Prof. Dr. A.G.J.M. van Leeuwen, chair).

Announcements
Ongoing announcements about future courses can be found at www.capitaselectaduikgeneeskunde.nl or are communicated by E-mail.

[1)] SDR is a non-profit organisation aimed to promote dive safety. Work for SDR is done voluntarily.

Programme

Symposium Diving Medicine; disorders of the head

Symposium 21st Century decompression theory and DCI treatment

9:00-9:30 **Welcome**

9:30-9:35 Introduction, *Nico Schellart.*

1. **9:35 -10:25 *Jean-Claude Le Péchon,*** Neo-Haldanian decompression theory and Haldanian based decompression procedures.

2. **10:25 -11:15 *Albrecht Salm*,** Dual phase decompression theory and bubble dynamics

Break

3. **11:30-12:15 *Adel Taher*,** DCI (differential) diagnostics

4. **12:15-13:00 *Jean-Claude Le Péchon,*** Selection of breathing gases for multi-day and saturation exposure and decompression in diving and tunnelling.

Lunch

5. **13:45-14:45 *Sigrid Theunissen,*** The endothelial response related to diving with specific attention to the role of anti-oxidants

6. **14:45-15:30, *Albrecht Salm,*** Implementation of dual phase decompression in tables for technical diving

Break

7 **15:45-16:45, *Adel Taher*,** Air/oxygen and Heliox DCI treatment

16:45 - 17:00 Test

Refreshments

Lecturing time includes 10 min of discussion.
Contact time: 6h00

Disclaimer: Capita Selecta Duikgeneeskunde (i.e. AMC and SDR) is bound to execute the program, but small program changes are under reserve.

The lecturers


Jean-Claude Le Péchon


Albrecht Salm


Adel Taher


Sigrid Theunissen

Jean-Claude Le Péchon graduated as biochemical engineer from INSA in Lyon in 1963 and as marine biologist from Nice University. He has been employed at the Musée Océanographique of Monaco as a research engineer and incidentally as scientific diver during the Conshelf III Experiment (the 100 msw undersea habitat organized by Jacques-Yves Cousteau in 1965). Later he joined CEMA in Marseilles (J.Y. Cousteau) to carry out research on breathing gases and to develop procedures for very deep dives (1000 m with animals; 500 m with humans) and was a test diver in the Saturation II simulated dive at 400 msw. From 1973 to 1986, he served with CG DORIS, an offshore and civil engineering commercial diving company. In 1986 he founded JCLP Hyperbarie, a global consultancy agency/ bureau specialized in matters related to life support and safety under pressure (commercial diving, tunnelling, space and hyperbaric medicine). He has been involved in more than 75 tunnelling projects with compressed air or gas mixtures, up to 6.9 bars and as well as in saturation diving technology. Although retired, he is still teaching physiology and technology of diving- and hyperbaric medicine at several universities in France and all over the world. He is a National Instructor for SCUBA diving (Air, Nitrox and Trimix) and holds a deep sea commercial diver certification since 1974. He has published many papers in magazines, books and international congresses etc., and often speaks at international congresses on different topics concerning diving, hyperbaric medicine and compressed gas work.

Albrecht Salm obtained a Ph.D. in physics and computer science at the University of Munich after his graduation at the University of Stuttgart. He was a commercial diver and Instructor for 'Hard Hat' and Saturation Diving. Since 1985 he is a PADI Master Scuba Diver Trainer #33913, and a Technical & Extended Range Instructor for SSI (Scuba Schools International, now 2nd or par to PADI), #12653. He contributes to the "CAISSON" (the quarterly GTUEM journal, GTUEM is: Gesellschaft fuer Tauch- und Ueberdruckmedizin, which reads in english: Association for Diving-& Hyperbaric Medicine), the Technical Diving Magazine (www.techdivingmag.com) and to "Diving & Hyperbaric Physicians Trainings" for the OEGTH (OeGTH is: Oesterreichische Gesellschaft fuer Tauch- und Ueberdruckmedizin. Both GTUEM and OEGTH are like smaller sisters from the UHMS). Currently he is working as a consultant with the international SubMarineConsulting Group (www.SMC-de.com)

Adel Taher, a diving instructor and diving medical specialist at the time, he was the driving force behind the multi-place, multi-lock chamber in Sharm el Sheik as he saw the need for a facility to specialize in diving related accidents. The chamber was built in the USA. He is, in addition to being director of the Hyperbaric Medical Centre in Sharm el Sheik also the founder and director of the diving chamber of Dahab. With over 200 diver-HBO treatments per year, he is without any doubt world leader and trained many physicians in hyperbaric medicine. Dr Adel Taher is member of many international medical diving committees, and lectures about his work at universities, courses, etc. all over the world. He is also the director of DAN-Egypt, a member of the UHMS and of the EUBS. He was the driving force behind the EUBS Annual Scientific Meeting in Sharm el Sheik in 2007 and a recognized invited speaker at their congresses. Diving still is his passion. He worked intensively on diver education and raising the awareness of dive professionals and divers regarding proper management of dive accidents in remote areas and the proper utilization of normobaric oxygen. The Hyperbaric Medical Centre in Sharm hosts dive medical specialists from all over the world to offer hands-on experience.

Sigrid Theunissen has graduated from the Université Libre de Bruxelles in 2004. She is professor in sports and physiology at the Haute Ecole Bruxelles-Brabant (ISEK) since 2007. She obtained her PhD at the Université de Bretagne Occidentale (UBO), France in 2013 about the effect of dark chocolate on

endothelial function after a SCUBA dive and successive breath-hold dives. Her main research theme is diving physiology and in addition she is one of the leading scientists in a hypobaric hypoxia project of ISEK, a collaboration with other institutes. She is a senior member of ISEK's Occupational Physiology laboratory. She is first author and co-author of articles in international peer-reviewed journals on oxygen physiology and author of many proceedings and poster presentations in international congresses. She is the secretary of the Belgian underwater society, member of EUBS and an enthusiastic recreational diver (2* CMAS and Nitrox). .

The symposium coordinator

Nico Schellart graduated as biologist and specialized in physiological and biomedical physics. He studied visual information processing of the retina, resulting in a PhD in 1973 (University of Amsterdam). He is an associate professor with the department of Biomedical Engineering and Physics of the AMC and was associate editor of a bio-engineering journal. He has researched information processing of the visual and auditory system of the brain in animals and humans by fundamental and clinical EEG and MEG methods. His neuroscience studies have resulted in publication of some 50 papers, 80 abstracts and 10 contributions in textbooks and been published a free electronic textbook on biomedical physics. He has studied the brain and the visual system under hypoxic and hyperoxic conditions in both the laboratory as in the field, including pre-cordial Doppler studies, and recommends HBO treatment for patients with cerebral radiation damage. He published some 20 dysbaric and HBOT studies in e.g. Cancer, J Appl Physiol and UHM, and ten different conference proceedings (like EUBS and UHMS). He is a frequently asked reviewer of journals in applied, sports and environmental medicine. Since 2011 he is course director of the CSD. He teaches diving physiology, is member EUBS and NVD, was member of UHMS (during Obama administration), and often participated with contributions in their annual meetings. Also, he has tested the technical and physiological performance of dozens of dive computers (www.diveresearch.org), and he is a recreational scuba- and a former free diver.

Preface
Reflections of the Editor

Parallel and series 'single-phase' models

Decompression physics and (patho)physiology has a history of about 150 years. In the *last century* most successful were the Neo-Haldanian models and the tables derived from these models. Typical is the assumption of parallel compartments, each with its own halftime and M-values. Most used are the Bühlmann tables and their derivatives. Also successful is the series model, applied in the DCIEM tables. This all is discussed in **Chapter 1**.

21th century models and the deep stop

Bubbles arise in the venous circulation (resulting in VGE that may cause DCS) and with DCI they also occur at the arterial side. Pathological extra vascular bubbles are of minor importance. Their existence is disputable and hard proof has never been given. Despite the fact that DCI is a phenomenon of the vessels, only recently, in the *21th century*, a series parallel model, a blood(-lung) compartment preceding the classical parallel Neo-Haldanian compartments was developed. This compartment has a very short halftime (ca. 45 s, depending on exercise). Such a model, as developed by the Editor (see Addendum 6B), behaves differently for different ascent velocities. With slow ones, its effect on inert gas release is minimal (very small extra delay), but with fast ascents, e.g. 15 m/min off-gassing is too slow and microbubbles will arise during the ascent. With shallow dives, this model predicts that the deep stop is even contra-productive (see also Addendum 2C), but with deep dives with fast ascent speeds a short (e.g. 2 min) deep stop is favourable. A very sophisticated 21th century model predicting the VGE grade for a multilevel dive, the Copernicus model, discussed in **Chapter 2** and also in **Chapter 6**, claims that with long dives the deep stop is recommended but not for short dives. With shallow dives microbubbles will hardly arise, but for deep dives they will. Hence a deep stop is possibly effective for deep dives with bounce like ascents but not for shallow dives. It should be clear that the insights about the deep stop are not equivocal. Unfortunately, prospective studies with all their different profiles and conditions are sparse, whereas retrospective research is polluted by commercial issues and hampered by an enormous number of parameters.

Dual phase models

Seemingly, due to the too high incidence of DCI in recreational and professional diving in the past, researchers developed dual phase models, which consider the gas phase in the compartments as well. This in the hope to provide more save protocols. Several were already developed in the last quarter of the last century. They are discussed in **Chapter 2**, with the emphasis on VPM and the Copernicus model. VPM versions have rather well accessible algorithms but unfortunately not Copernicus. VPM is mainly used in recreational diving, be it tech diving. Professional diving mainly applies Neo-Haldanian tables, which are adapted for each caisson and tunnelling work and which are improved (fine-tuning) by measuring the VGE grade after exposure. Such tables are discussed in **Chapter 4**. This chapter also discusses the choice of the breathing mixture with emphasis on the upper limits of the metabolic gases, oxygen and carbon dioxide, set by their toxic effects.

For decades it is a wish of researchers to personalize the table or diving algorithm of the dive computer. A decade ago the Editor developed correction tables based on age and fitness (VO2max), the demographic parameters generally accepted as affecting DCS risk. Recent models, attempt to take exercise level into account. Copernicus also uses body composition, but this is dubious since body fat or BMI are not connected causally with bubble stress (see Addendum 6A and 6B), be it that there is an indirect effect since body fat is strongly related to VO2max and age. Incorporating the latter two would be more realistic.

Bubble interface and endothelial response

Dual-phase models explicitly make use of the surface tension of bubbles. Some fifty years ago it was assumed that bubbles are covered by a fatty acid monolayer. This layer should reduce the surface tension by a factor 5 (compared to water), resulting in a strong bubble preserving effect. This assumption, still fashionable (Chapter 2), is wrong. The surface tension is only slightly smaller than that of water and it is more likely that bubbles are covered by protein (albumin), as concluded in recent studies (see e.g. Addendum 2A and 2B). A speculation might be that microparticles can attach to the protein skin.

In *the last century*, bubbles that block vessels were seen as the main cause of DCS and consequently the aetiology was predominantly organ-oriented (bends, spinal cord, vestibular…..). In the *21th century,* it became more and more clear that in addition there is also a more systemic effect. Bubbles attach to the vascular endothelium and induce an inflammation of the vessel wall. Possibly this causes to the symptom of fatigue, that nearly always occurs with DCI. In conclusion, DCI is likely (at least) dual-causal, a mixture of an organ and systemic disorder. The endothelial inflammation with platelets and neutrophil activation, increase of microparticles, occurrence of vascular dilatation, etc. is discussed in **Chapter 5**, with the emphasis on oxidative stress and the anti-oxidant cocoa.

Differential Diagnosis of DCI and 21 century DCI treatment

With non-aggressive diving, tables and computer algorithms are very save. However, DCI still happens, mostly caused by human failure but sometimes also without the violation of any safety rule. The cause of a diving accidents must be diagnosed, the sooner the better. This is extensively treated in **Chapter 3**. After the differential diagnosis, DCI treatment may be indicated. The array of treatments is discussed in the **last Chapter (7**). Nearly always they are successful, although permanent damage may occur. With tech and rebreather divers going deeper and deeper, classical treatment of DCI is not always the solution. In Chapter 7 application of Heliox will be discussed as well.

The future

Future research will offer us more understanding of the pathophysiology of DCS. After unravelling the mechanisms new drugs may be developed. A speculation is that application of normobaric 100% oxygen, at the dive site and later - mainly to reduce bubble size - and application of such new drugs makes a chamber treatment not necessary anymore. A similar future possibly holds for clinical HBOT.

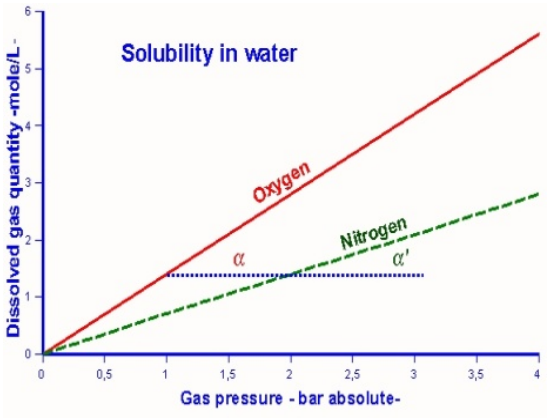
Chapter 1 Neo-Haldanian decompression theory and Haldanian based decompression procedures.

Jean-Claude Le Péchon

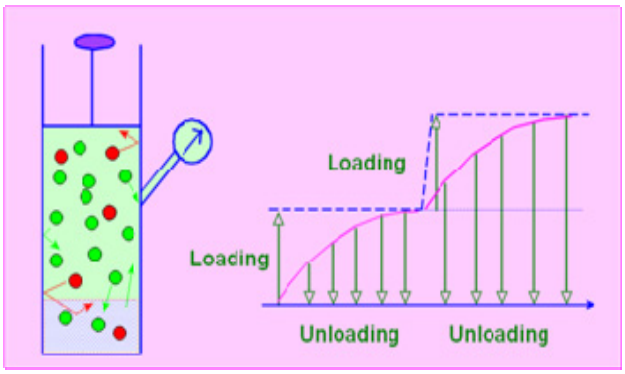
Classic principles

Gas dissolution

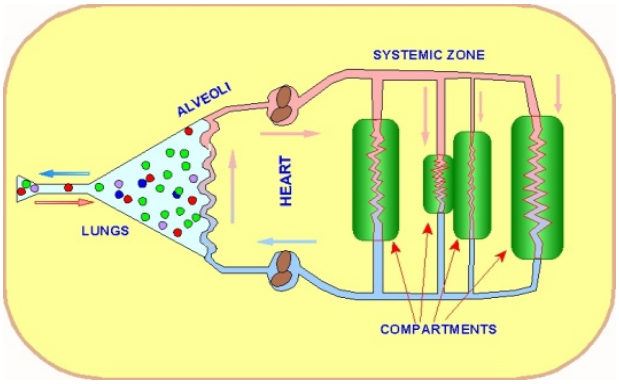
- Solubility of gases in water is directed by Henry's law which stipulates that the quantity of dissolved gas in a liquid, when equilibrium has been reached, is proportional to the partial pressure of the gas used to each that equilibrium. That quantity is also a function of temperature. Perfect gas equilibration is never the case in hyperbaric exposures



- However Henry's law is extremely useful in hyperbaric calculations to express dissolved gas quantities (mole/L, vol/L or mg/L) in pressure units which eliminates gas solubility coefficient from equations.
- When extracted, air dissolved in water and equilibrated is no more air; it is 33 % oxygen and 67% nitrogen! This is the dissolved air paradox!
- Temperature rise reduces the amount of dissolved gases in liquids.
- When a liquid is exposed to a new gas, dissolution process is directed by diffusion law (Fick's Law). The flux is proportional to the exposed surface, the difference in partial pressure of gases on both sides and the solubility coefficients of the gas in each compartment. In a capillary (alveoli or in tissues) the blood flow acts as a convector (perfusion).

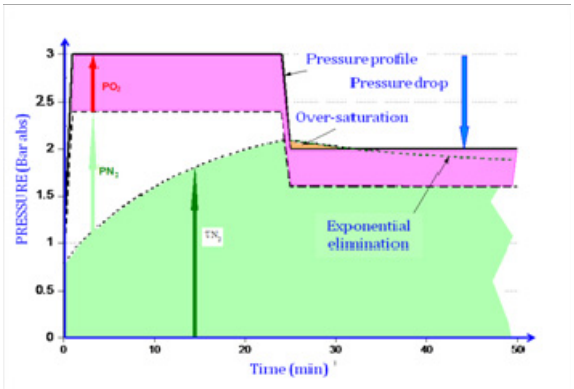


- The kinetic to reach equilibrium as a function of time is an exponential process that involves the exchange area, the flux of liquid, and the differential partial pressures of the gas between gas and liquid, or between two liquid phases. The resulting parameter is the half time of the exponential.
- When a human is considered the whole body can be divided into several zones each with a specific half-time related to perfusion, type of tissue (lipid or aqueous). They are called "tissues" when they correspond to a histological definition or "compartments" when they are only mathematical data. Uptake and elimination may not follow the same half-time values.

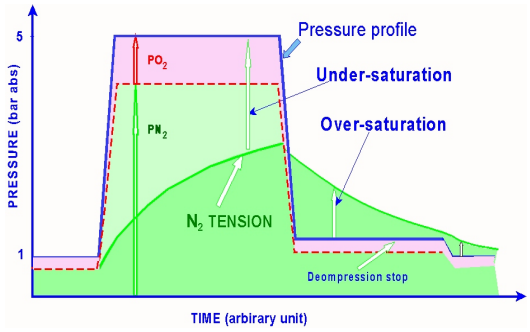


Gas elimination

- Dissolved gas elimination from a liquid may be symmetrical to uptake when conditions of flow are not changed and the pressure drop is very slow not to create significant over saturation that would induce the formation of micro bubbles from extra gas.
- The example of frizzing drinks is wrong because CO_2 being very soluble in water. The amount of dissolved CO_2 is very large and the significant pressure drop involved has nothing to compare with human decompression. Less than one second and 6 bars for champagne!



- A better example is hot water off gassing when released from the tap or when cold water is heated forming a kind of fog which is micro bubbling.
- All early decompression procedures used the concept of acceptable “silent” over saturation values that may induce bubbles (ignored in calculations) but no DCI signs. It is a purely empirical approach. Those values are designated either as:
  - ✓ Over-saturation ratios (dissolved inert gas versus total pressure),
  - ✓ Maximum inert gas content at a given pressure (M Values) or
  - ✓ Over-saturation gradient (difference between inert gas content and total pressure).



- Excursion dives: At the end of a short duration exposure to increased pressure, all compartments or tissues are not yet balanced with the inspired inert gas partial pressure. Decompression process shall take into account each compartment or tissue gas load individually.
- Saturation dives: When the exposure is long enough to reach full body equilibrium, the decompression computation needs only to consider the compartment that is the slowest in the gas exchange processes: it is called saturation decompression and it is a very long lasting operation (1 day for each 30 meter equivalent pressure drop for Heliox exposures).

Haldane tables

After a long series of experiments carried out with goats, results published in 1908, Sir Haldane, Boycott and Damant were the first to elaborate a model of gas exchanges that produced an efficient decompression procedure.

It was based on five compartments (5, 10, 20, 40 and 75 min half times) and an exponential uptake and elimination, associated with a uniform maximum over saturation ratio equal to 2:1. All the calculations were made using air pressure values rather than nitrogen partial pressures; the exact ratio with partial pressures of nitrogen is 1x0.79/2 or 1.58/1. Those tables have been implemented in the Royal Navy and for compressed air works in UK and over the world for many years.

It is only 2 years later that A. and A. Borstein who were involved in a deep compressed air tunnelling in Hamburg, established that using oxygen during decompression stops improves the safety of the procedure and they introduced immediate recompression with oxygen in case of DCS they also described bone necrosis as a consequence of poorly adapted decompression protocols.

The principle of exponential uptake of gases in various compartments is still used nowadays in most models for decompression. The principle of super-saturation ratio equal to 2:1 has been abandoned a long time ago; in particular from 1937 when Yarbrough established that short half time tissues can stand higher over-saturation ratios.

US Navy

Then Van Der Aue (USN) in 1937 had to scientifically validate surface decompression technique as used since 1917 when Damant supervised salvage operations carried out with surface decompression… He produced USN procedure for surface decompression that was safer than Yarbrough tables… Longer half time tissues have been introduced and the maximum permissible super-saturation became a function of pressure and compartment half times. In 1956 new tables were issued although not satisfactory for long exposures. In 1965 Workman published an extensive report and provided the concept of M values "*The maximum tissue pressure (M) is the greatest partial pressure of inert gas in a specific tissue which will not permit bubbles to form in the tissue at a given absolute pressure*". He denied the correspondence between histological tissues and decompression compartments and selected a purely mathematical series of half-times (Geometrical –x2- increment from 5 min to 80 min, then arithmetical (+40 min) up to 240 min. He explained in details the table calculations:

- an 8 compartment model, (5, 10, 20, 40, 80 - 120, 160, 200 and 240 min)
- M values for nitrogen and helium,
- a set of air diving tables that was implemented for US Navy use.

Workman's report: http://www.dtic.mil/dtic/tr/fulltext/u2/620879.pdf

French Ministry of labour

The French Navy had translated the 1956 USN tables and used a French metric version with some modifications for deeper dives from 1965 to 1990 when it has been revised with a 8 symmetrical compartment model and over-saturation ratios for each compartment however constant at all pressures (Tables French Navy 1990).

Following closely Workman's model, Fructus for Comex (1974) produced an air decompression table with revised lower M values obtained after a comprehensive series of human experiments. This table has been revised again in 1992 (Imbert) including validation from actual dives in the field, and incorporating a few parameters taking into account the existence of bubbles, and they became the French ministry of labour set of decompression tables, still in force nowadays. Example: Mini-Table Air decompression below.

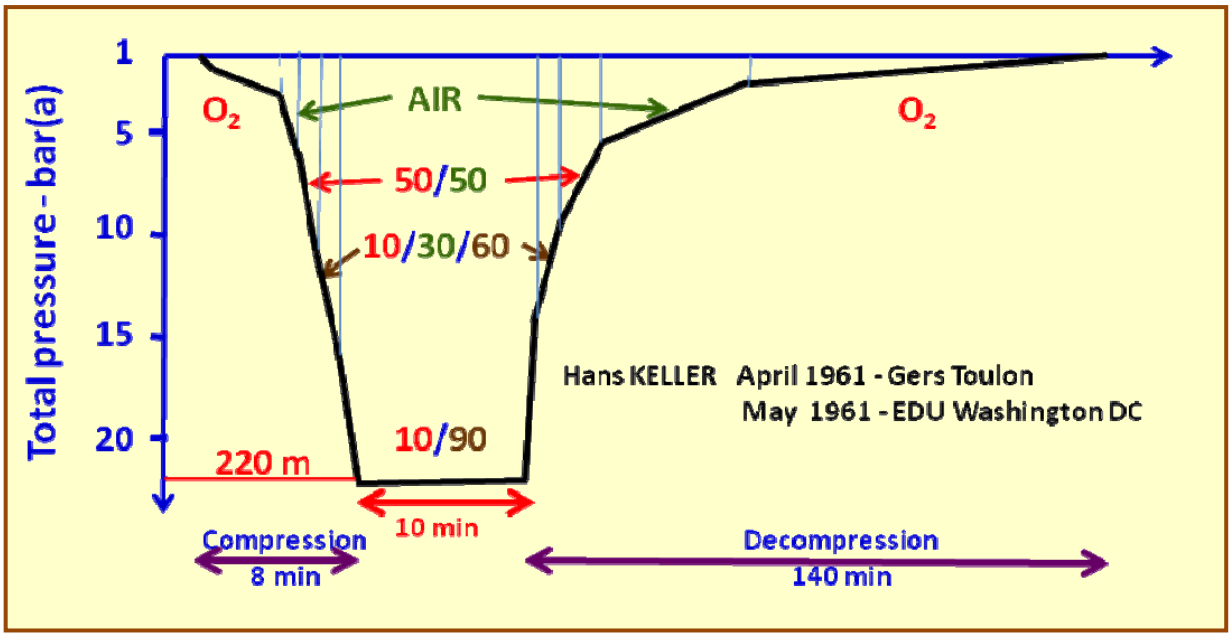
DEPTH Metres	BOTTOM TIMES (minutes)													
12	165	170	180	195	210	240	-	-	-	-	-	-	-	-
15	80	90	100	110	115	130	-	-	-	-	-	-	-	-
18	50	55	60	70	75	80	-	-	-	-	-	-	-	-
21	35	40	45	50	55	60	-	-	-	75	-	-	-	-
24	25	30	35	40	45	50	-	-	55	60	-	-	-	-
27	20	25	30	33	35			40	45	48	-	55	-	-
30	15	20	25	28	30	-	-	35	38	42	-	47	55	-
33	12	15	20	23	-	-	25	30	32	37	-	40	47	-
36	10	15	17	20	-	-	22	25	27	32	-	34	40	43
39	8	10	15	17	-	-	20	22	24	-	27	30	35	38
42	7	10	13	14	-	-	18	20	-	-	24	27	30	33
45	6	10	12	13	-	-	15	18	-	-	22	25	28	30
48	5	8	10	12	-	-	15	-	-	-	20	23	26	28
51	5	7	8	-	-	-	12	-	-	-	18	21	24	25
54	-	5	7	-	-	-	10	-	-	-	16	19	-	23
57	-	5	6	-	-	-	10	-	-	-	14	17	-	21
60	-	-	5	-	-	-	8	-	-	-	12	-	-	18
STOPS	Ascent to first stop 12 m / min (3 m each 15 seconds)													
12	-	-	-	-	-	-	-	-	-	-	-	-	-	3
9	-	-	-	-	-	-	-	-	-	-	3	3	5	5
6	-	-	-	-	-	-	3	3	3	3	5	7	10	12
3	-	3	5	7	10	15	7	12	15	20	15	20	25	25

Mini Table Air decompression MT 1992

In the same manner Heliox tables were published as well as compressed air tables derived from the same model however with more restrictive M values to take into account the longer duration of exposure and the workload involved in those caisson works.
www.sneti.eu/pdf/tables-ministere-du-travail.pdf

Bühlmann's concept (1984)

In association with Keller he introduced the technique of multi-gas decompression after deep excursions with Heliox mixes and Trimixes during compression and decompression; Keller succeeded the very first dives in the range of 300 m (1961 in Toulon – French Navy Chamber and 1962, open sea in San Diego)!

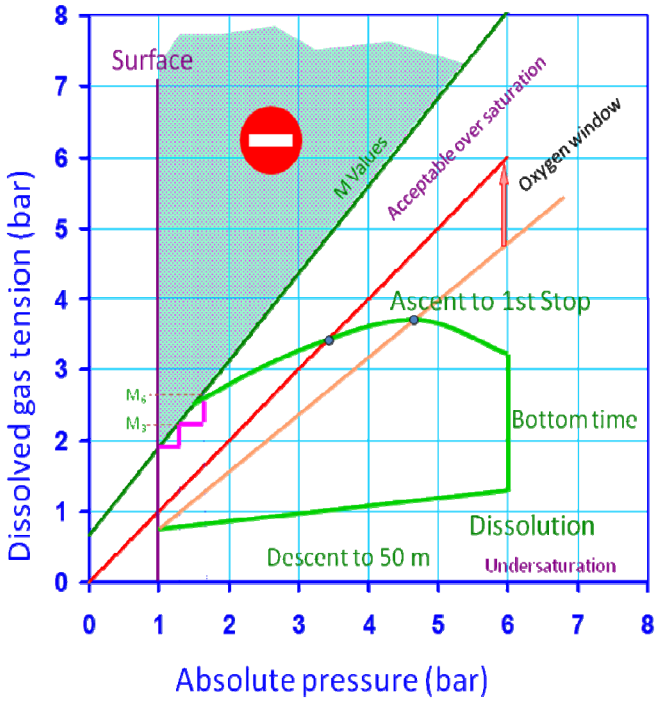


In Zurich, Bühlmann also undertook extensive series of human experiments and he established a new set of tables based on 16 histological tissues those half times have been determined experimentally. Each tissue is associated with half times and M values for cumulative helium and nitrogen. Ratio of half times for nitrogen and helium is 2.65:1. Both gases accumulate and exit compartments with relative half times. The M values for each compartment are a linear function of pressure:

M Value (Max P_{inert} gas at a given pressure P):

$$M = P/b + a,$$

where "a" and "b" are constants depending on specific half times of the tissue concerned.
This resulted in the ZH-L 16A model. Later he slightly modified several "a" constants with the aim to make the tables more conservative, resulting in the B and later the C versions of tables
He introduced also the alveolar gas as inspired inert gas partial pressure, in particular because he was concerned with altitude dives in Switzerland. Water vapour relative content in lungs being more and more important as local atmospheric pressure is reduced.



The simplicity of the calculations, and the proved quality of the tables produced made his model a nice candidate for incorporation into dive computers softwares. Many computers still distributed are based on Bülhmann's model, but generally with 8 compartments only.
Later, all kind of modifications have been implemented to take into account bubbles, and eventually to change the M Values to increase safety when special conditions exist. For instance ZHL–8 ADT MB PDIS is implemented in the recent UWATEC dive computers. In fact, a great majority of dive computers have implemented some version of the ZH-L model.
The model is purely perfusion limited and tissues are identified as organs.

Sterk calculations

Since 1975, under proprietary calculations W. Sterk, has produced The Netherlands official decompression tables, revised (2015) to improve safety and take advantage of field data and extensive bubble detections results.
Air, air and oxygen stops and Nitrox diving tables, Trimix for caisson works … are among these tables.

Hempleman's Concept

Hempleman considered mostly bends in the joints as the main DCI problem. He suggested that diffusion in a layer of joints cartilage represents the uptake of gas, and that the elimination is not symmetrical and much slower eventually due to some bubbles…
He calculated the quantity Q of gas diffusing in a single layer of tissue as a function of time: $Q = C\ t^{1/2}$ (the square root of time t). C is the gauge pressure of exposure to compressed air.
Hempleman found empirically the maximum value for Q to decompress "safely" to atmospheric pressure: $Q < 465$.
He matched his calculated table with the NO-D US Navy limits and found a perfect agreement.
He then produced a full set of table in 1968, including oxygen decompression tables. The Underwater Engineering Group published them for use by many commercial diving companies in the UK.

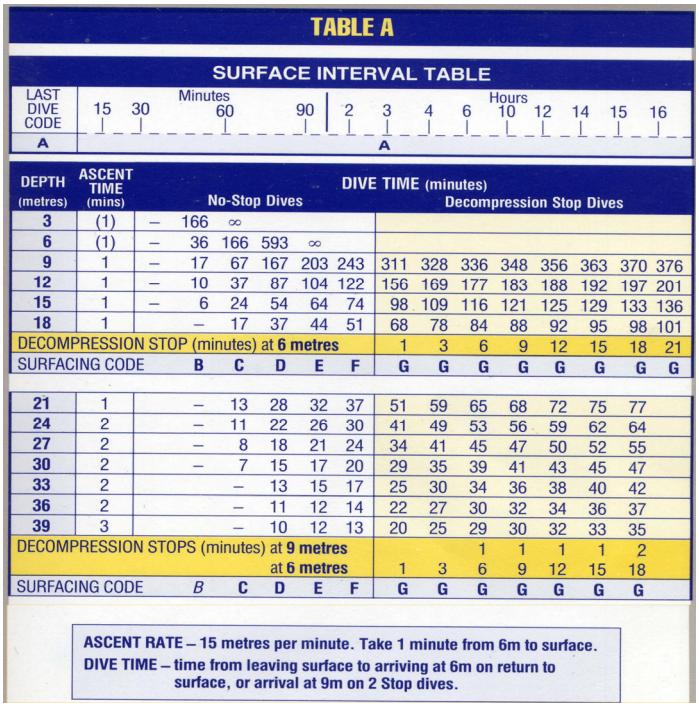


TABLE A

SURFACE INTERVAL TABLE

LAST DIVE CODE	Minutes 15	30	60	90	Hours 2	3	4	6	10	12	14	15	16
A					A								

DEPTH (metres)	ASCENT TIME (mins)	DIVE TIME (minutes) No-Stop Dives					Decompression Stop Dives							
3	(1)	–	166	∞										
6	(1)	–	36	166	593	∞								
9	1	–	17	67	167	203	243	311	328	336	348	356	363	370	376
12	1	–	10	37	87	104	122	156	169	177	183	188	192	197	201
15	1	–	6	24	54	64	74	98	109	116	121	125	129	133	136
18	1	–	17	37	44	51		68	78	84	88	92	95	98	101
DECOMPRESSION STOP (minutes) at **6 metres**								1	3	6	9	12	15	18	21
SURFACING CODE		B	C	D	E	F		G	G	G	G	G	G	G	G
21	1	–	13	28	32	37		51	59	65	68	72	75	77	
24	2	–	11	22	26	30		41	49	53	56	59	62	64	
27	2	–	8	18	21	24		34	41	45	47	50	52	55	
30	2	–	7	15	17	20		29	35	39	41	43	45	47	
33	2		–	13	15	17		25	30	34	36	38	40	42	
36	2		–	11	12	14		22	27	30	32	34	36	37	
39	3		–	10	12	13		20	25	29	30	32	33	35	
DECOMPRESSION STOPS (minutes) at **9 metres**										1	1	1	1	2	
at **6 metres**								1	3	6	9	12	15	18	
SURFACING CODE		B	C	D	E	F		G	G	G	G	G	G	G	

ASCENT RATE – 15 metres per minute. Take 1 minute from 6m to surface.
DIVE TIME – time from leaving surface to arriving at 6m on return to surface, or arrival at 9m on 2 Stop dives.

In parallel he produced tables (1966) for compressed air workers known as Balckpool tables and introduced the principle of acclimatization, asking the new comers to enter compressed air later than the regular workers and decompress with the group. That has been a great progress, the procedure have been used in the whole Commonwealth, until the late 1990's. The stops were too long for short duration exposures and much too short for very long working times. In addition they have been used with the "decant" procedure (back to atmospheric pressure and recompression in a chamber on site). That produced a significant number of DCI and bone necrosis cases.
Presently acclimatization cannot be considered in compressed air work for TBM maintenance, since hyperbaric interventions are only necessary to repair the cutters, and they are generally not continuous. There is not time for acclimatization; tables should be safe directly for new comers.
When at last the UK authorities in 2004, removed the ban on oxygen decompression in tunnelling, they decided to still use Blackpool tables, changing only air for oxygen at the lasts stops which did not considered the advantages of oxygen on decompression time neither used deeper stops while breathing oxygen! It is a very bad decision still in force in UK!!! In the mean time Hennessy also from RNPL calculated the BSAC tables based on similar principles but taking into account

the released gas volume as bubbles. He also was the first to insist that bubble may be on the arterial side of the circulation (1988)…

Hills Thermodynamic approach

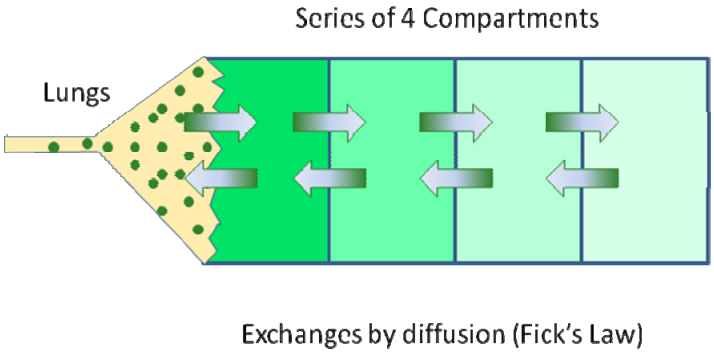
Hills (1977) tried a completely new approach using a diffusion limited algorithm. A thermal analogue was used to simulate gas exchanges, and the maximum permissible over-saturation was kept close to zero. The gas elimination driving force is mostly the inherent under-saturation created by the so called Oxygen Window. The decompression profile starts with deep stop and ends with a final sharp drop.

He nevertheless calculated free gas volume and used it as the ascent limiting factor. His model never received practical application.

Other concepts

Nishi at DCIEM in Canada developed further the model of Kidd and Stubs which was a pneumatic analogue computer for divers.

DCIEM approach is to consider a purely diffusion process into a 4 compartment model in series to compute gas uptake. Elimination is calculated following the reverse path. The decompression limiting factor is the maximum possible calculated gas volume released from solution.



The validations of the limiting factors have been established experimentally using extensively the circulating bubbles detection with the ultrasonic Doppler detector technology.

The resulting tables are currently in use in the Canadian Navy as well as in other countries, in several commercial diving companies and national recreational diving federations.

Rate of ascent to first stop

For operational and practical reasons, in the early days a conflict between US Navy hard-hat divers and swimmers resulted in a decision: ascent to first stop is 60 ft/min (18 m/min) for both groups. No physiological aspect has ever been suggested during the talks.

It appeared later that this rate of ascent is too fast for a safe decompression, nowadays it is selected between 15 and 9 m/min. Variable rates have also been introduced in modern procedures.

The main reasons for that requirement of slow ascent to the first stop being the risk of triggering micro nuclei to grow into micro bubbles during that phase. During the stops they may grow from surrounding dissolved gas and then, when total pressure decreases significantly close to the surface, Boyle-Mariotte effect will enlarge them dramatically.

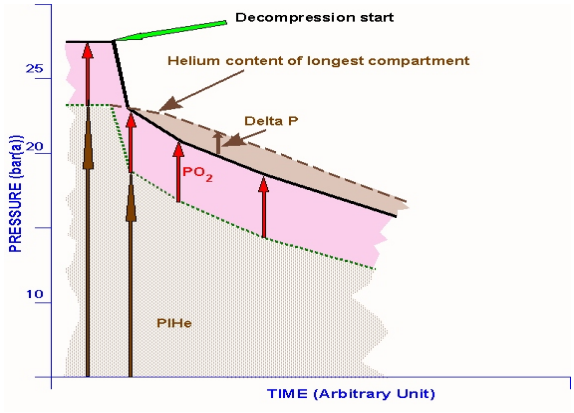
Saturation final decompression

Whatever the model, decompressing from saturation exposures is simpler since only the longest compartment is of importance to evaluate the potential over-saturation, all the faster compartments loose gas faster and cannot be the directing compartment.

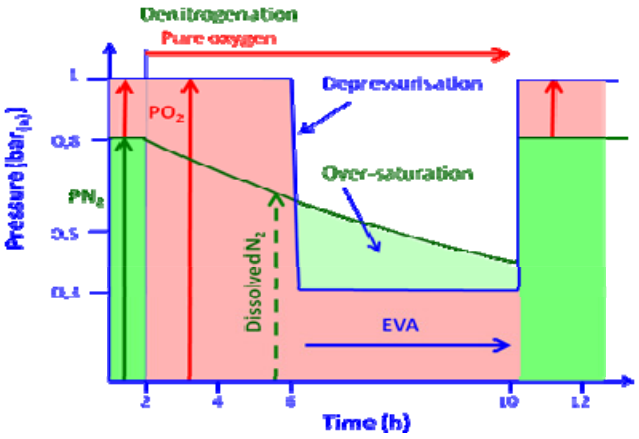
In the early days exponential decompression, with an appropriate half time for the profile, proved to be safe from 100 metres (Conshelf III – 1965). Soon it appeared that from higher pressure (> 40 b – 400 m) it was prone to induce inner ear DCI!, the fast deep pressure reduction being much too fast.

Several tentative adjustments (night rest by US navy) linear profiles, very slow last bar (10 m) resulted in various procedures. They are either purely empirical or based on gradients, and high PO_2 to keep an acceptable driving force.

The only parameter to control is either the over-saturation gradient created by the reduction of pressure or the level of circulating bubbles.



Deep Heliox saturation decompression



EVA Decompression

This is applicable to deep saturation dives as well as denitrogenation of astronauts before EVA (Extra-Vehicular Activity). On the figure, is shown a saturation decompression with semi-constant PO_2 (0.5 bar during active phases, and 0.55 bar during sleeping times). And an accepted over saturation gradient decreasing as pressure is reduced.

Some conclusions

None of these models are “physiologically” correct, probabilistic models and bubble models are not either… Even when they also produce acceptable tables they are far from reality.

In particular it is noticeable that the only bubbles currently measured are circulating bubbles although algorithms taking into account bubbles consider only tissue bubbles. The mechanism for bubbles to cross over from tissues into blood steam is still an open question.

Van Liew in 1992 specified the criteria for a good decompression algorithm… It should consider the consequences of the following points:

1. Pre-existing gas nuclei,
2. Bubbles growth is resulting from both gas diffusion and pressure decrease,
3. Pressure in a bubble is determined in adding : Ambient pressure, Pressure resulting from surface tension, tissue resistance, local hydrostatic pressure in the body and is equal the total of gas partial pressures inside (H_2O, O_2, CO_2, and N_2, He),
4. Inherent under-saturation or Oxygen Window,
5. Specific perfusion rate of each zone of tissue,
6. Small bubbles are spherical,
7. When a bubble is growing it will suck out inert gas from the close by bubbles (clamping).

References of major textbooks

Underwater physiology symposia by UHMS (I to IX) 1962-1987

“Bennett and Elliott”: The physiology and medicine of diving. Five editions. 1969-2003

Bülhmann A.A. (1984) Decompression Sickness : English translation Springer-Verlag editor

Hills B. A. (1977) Decompression sickness Volume 1; Jhon Wiley & Ssns editor

Workshops organized by UHMS (www.uhms.org) several of them are devoted to various aspects of decompression.

USN navy “historical” Workman’s report 1965
http://www.dtic.mil/dtic/tr/fulltext/u2/620879.pdf

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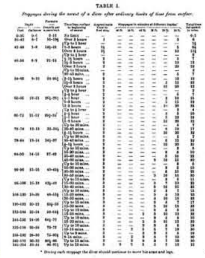
THEORY OF NO BUBBLE DECOMPRESSION

Jean-Claude LE PÉCHON



http://jclp-hyperbarie.fr

No bubbles decompressions (cont'ed...)

AGENDA

1 – CLASSICAL PRINCIPLES

2 – HALDANE'S TABLES

3 – US NAVY - WORKMAN, FRENCH MIN. of LABOUR

4 – BÜLHMANN'S CONCEPT

5 – DIFFUSION APPROACHES

6 – SPECIFIC CASES

No bubbles decompressions (cont'ed...)

1 – Classical principles

1.1 – Gas dissolution

Henry's Law :

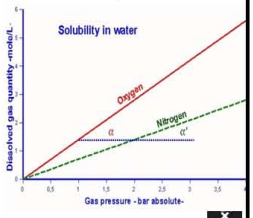
At a given temperature, the quantity of dissolved gas in a liquid is proportional to the pressure of the gas used for equilibration.

The coefficient of proportionality is specific of the gas and the liquid

Oxygen is almost twice more soluble in water than Nitrogen

Henry's law is NEVER exactly applicable in diving. However it is extremely USEFUL.

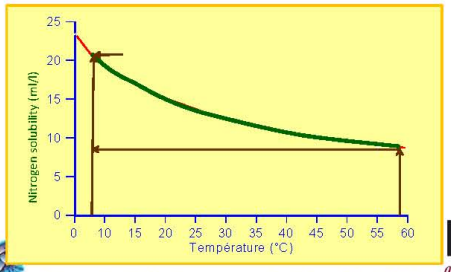
Mole/L can be changed into equivalent tension in bar.



1.1 – Gas dissolution (cont'ed...)

Effect of temperature

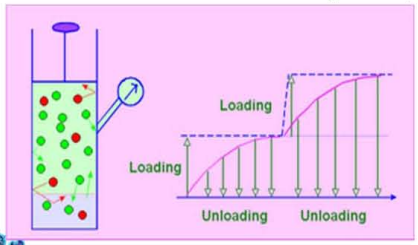
When temperature increases, the solubility of gases decreases. Except Helium...



1.1 – Gas dissolution (cont'ed...)

Dynamics of dissolution

When a gas is exposed to a gas free liquid, the uptake is exponential until saturation is achieved according to Henry's law

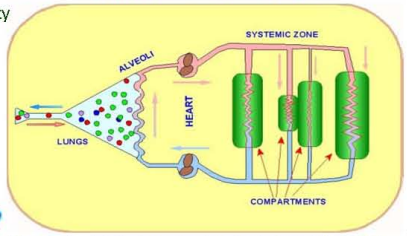


1.1 – Gas dissolution (cont'ed...)

Dissolution in the human body

From alveoli to the deep tissues gas are transported by blood and distributed into the various organs at a rate function of perfusion...

Then into the tissues by diffusion and according to their solubility



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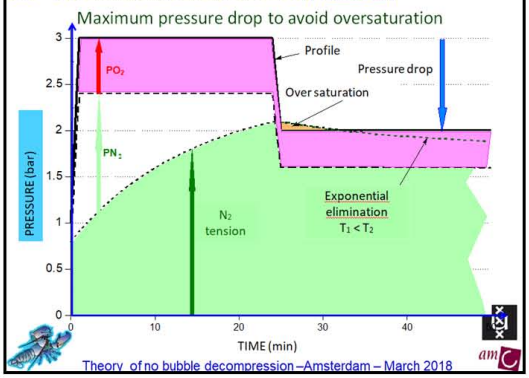
1.2 – Gas elimination from the human body

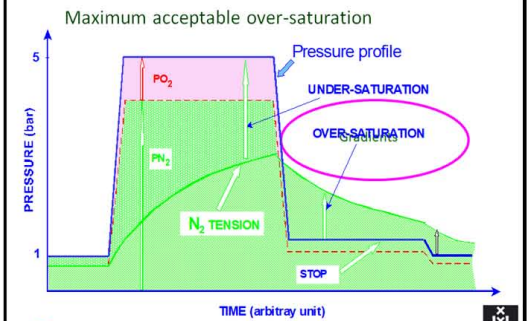
After exposure to a pressurized environment, when pressure is reduced...

Unloading gas from tissue is exponential but :

- Half time may not be the same as for uptake
- If no bubbles form

Pressure drop must be controlled to prevent free gas seeding





2 – Haldane's Tables

2.1 – Haldane's model

In 1908, from exposures of goats to compressed air Haldane, Boycott and Damant established a decompression algorithm ;

5 exponential symmetrical compartments (5, 10, 20, 40,and 75 min).

Single maximum over-saturation ratio : 2/1

Calculated : Pressure of "air" uptake/absolute pressure

Ratio applicable of all compartments and at any stop depth.

Stage decompression 32 min

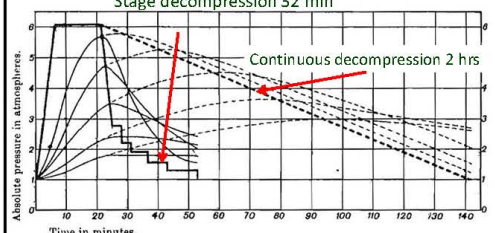


Fig. 4. Desaturation during stage decompression in 32 minutes and uniform decompression in 2 hours, after exposure for 15 minutes at 75 lbs. pressure with compression in 6 minutes. Thick lines = air pressure: continuous lines = stage decompression: dotted lines = uniform decompression. The curves from above downwards represent respectively the variations in saturation with nitrogen of parts of the body which half saturate in 5, 10, 20, 40, and 75 minutes.

Applications

Implemented in the Royal Navy

In UK, all compressed air works used those tables

In 1910, in Germany A & A Borstein introduced :

- breathing oxygen during the stops
- immediate recompression breathing oxygen in case of DCI
- description of bone necrosis as the consequence of a non adapted decompression

Until nowadays, gas uptake in most decompression algorithms is exponential as from Haldane, even when there are different numbers of compartments and different ascent criterias

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No bubbles decompressions (cont'ed...) 13 /40

3 – US NAVY - WORKMAN, FRENCH MIN. of LABOUR

3.1 – US Navy

1917, Damant initiated surface decompression with Haldane's tables

Yarbrough produced a new set of surface decompression tables that had to be improved in 1937 by Van der Aue.

With the changes :

- New compartments half times (120 min added),
- Over-saturation ratios as a function of compartment
- Over saturation ratios as fa unction of stop pressure

where used for a new set tables in the USN diving manual in 1956.

3 – US NAVY - WORKMAN, FRENCH MIN. of LABOUR (cont'ed...) 14 /40

3.2 – Workman (1965)

The extensive report published in 1965 provides new tables for

- air
- Heliox
- Oxygen decompression

Purely mathematical series of compartments

5, 10, 20, 40, 80, 120, 160, 200, 240 min

Definition of M values for both nitrogen and helium

"The maximum tissue pressure (M) is the greatest partial pressure of inert gas in a specific tissue which will not permit bubbles to form in the tissue at a given absolute pressure".

Pure oxygen at stops is considered 80 % only

3 – US NAVY - WORKMAN, FRENCH MIN. of LABOUR (cont'ed...) 15 /40

3.2 – French Ministry of labour (1974 & 1992)

History

Comex carried out an extensive research program to improve air and Heliox diving tables:

. In 1974 a new set of air table from Comex has been officially published by the French Min. of labour.

Fructus used an algorithm close to Workman's, with reduced M values. Tables had 2 versions air and oxygen decompression and fully calculated repetitive dives decompressions

. In 1992 it has been revised and extended to compressed air tables, Heliox tables, Heliox saturation to 200 m. Imbert used a modified algorithm to take into account possible free gas in bubbles. New M-values were obtained from extensive field validation between '74 and '86.

3.2 – French Ministry of labour (cont'ed...) 16 /40

M Values according to X. Fructus (Tables MT 74) in bar

Half times	5	10	20	40	80	120	240	
Surface	2,7	2,4	2,05	1,72	1,56	1,52	1,4	→ 1,52
3 m	3,3	2,9	2,5	2,12	1,91	1,85	1,7	
6 m	3,9	3,4	2,95	2,52	2,26	2,18	2,0	
9 m	4,5	3,9	3,4	2,92	2,61	2,51	2,3	→ 1,32
12 m	5,1	4,4	3,85	3,32	2,96	2,84	2,6	

Corresponding Over saturation ratio 2,95 / 1,6 = 1,84

3.2 – French Ministry of labour (cont'ed...) 17 /40

Examples of tables MT92

www.sneti.eu/pdf/tables-ministere-du-travail.pdf

AIR OXYGEN Depth : 39 meters

Bottom time (min)	Ascent to 1st Stop	AIR 21m	AIR 18m	AIR 15m	AIR 12m	AIR 9m	OXY 6m	Total dec. (min:sec)	Repet. Dive OK
10	2:45						3	5:45	OK
15	2:45						3	5:45	OK
20	2:45						7	9:45	OK
25	2:45						10	12:45	OK
30	2:30					3	15	20:30	OK
35	2:30					5	20	27:30	OK
40	2:15				3	7	25	37:15	OK
45	2:15				3	10	30	45:15	OK
50	2:15				3	10	35	50:15	OK
60	2:15				5	15	45	67:15	OK
70	2:00			3	10	20	50	85:00	NO
80	2:00			3	12	25	60	102:00	NO
90	2:00			5	15	30	70	122:00	NO
100	1:45		3	7	20	30	80	141:45	NO

MINITABLE AIR (Arrêté 15 mai 1992, annex II table n° 2)

Time after previous dive 12 hrs minimum

DEPTH meters	BOTTOM TIME (minutes)
12	165 170 180 195 210 240 - - - - - - -
15	80 90 100 110 115 130 - - - - - - -
18	50 55 60 70 75 80 - - - - - - -
21	35 40 45 50 55 60 - - 75 - - - -
24	25 30 35 40 45 50 - - 55 60 - - -
27	15 20 25 28 30 - - 35 38 42 - 47 55 -
30	12 15 20 23 - - 25 30 32 37 - 40 47 -
33	10 15 17 20 - - 22 25 27 32 - 34 40 43
36	8 10 15 17 - - 20 22 24 - 27 30 35 38
39	7 10 13 14 - - 18 20 - - 24 27 30 33
42	6 10 12 13 - - 15 18 - - 22 25 28 30
45	5 8 10 12 - - 15 - - - 20 23 26 28
48	5 7 8 - - - 12 - - - 18 21 24 25
51	- 5 7 - - - 10 - - - 16 19 - 23
54	- 5 6 - - - 10 - - - 14 17 - 21
57	- 5 5 - - - 8 - - - 12 - - 18
60	- - 5 - - - - - - - - - - -
STOPS	Ascent rate to 1st Stop : 12 m/min (3 m every 15 seconds)
12	- - - - - - - - - - - - 3
9	- - - - - - - - - - 3 3 5
6	- - - - - - 3 3 3 3 5 7 10
3	- 3 5 7 10 15 7 12 15 20 15 20 25 25

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Example of tables MT92 (cont'ed...) 19 /40

Compressed air work – with oxygen decompression

Table Air / Mention D / OXY / P = 1,95 b

WORKING TIME	TO 1st STOP	AIR 1,2 b	OXY 0,9 b	OXY 0,6 bar	TOTAL DEC.	TOTAL TIME
0h30 - 1h0	6 min			10	16 min	1h 16
1h 30	4 min		5	15	24 min	1h 54
2h 00	4 min		10	15 5 10	44 min	2h 44
2h 30	4 min		15	10 5 25	59 min	3h 29
3h 00	4 min		20	5 5 25 5 15	79 min	4h 19
3h 30	4 min		25	5 25 5 25 5 5	99 min	5h 09
4h 00	4 min		25 5 5	20 5 25 5 25	119 min	5h 59

Oxygen breathing

Air breathing

Theory of no bubble decompression –Amsterdam – March 2018

No bubbles decompressions (cont'ed...) 20 /40

4 – BÜLHMANN'S CONCEPT

He started with Hans Keller calculating extreme deep bounce dives with multiple gas shift.

After 1974 when his hyperbaric centre in Zurich became operational he carried out many human experimental compressions and decompressions up to 50 bars.

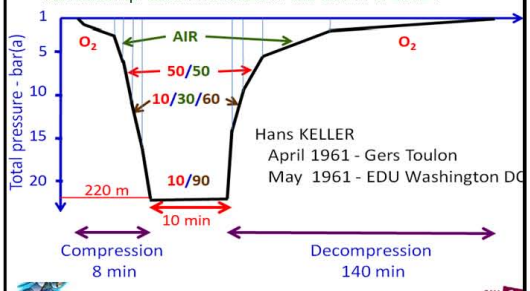
From the type of observed signs of DCI he developed the concept of multiple physiological tissues and specific over saturation values.

Theory of no bubble decompression –Amsterdam – March 2018

4 – BÜLHMANN'S CONCEPT (cont'ed...) 21 /40

Hans Keller's dives

In association with H. Keller he calculated multi gas fast decompression from 220 m the 300 m in 1961 !



Theory of no bubble decompression –Amsterdam – March 2018

4 – BÜLHMANN'S CONCEPT (cont'ed...) 22 /40

Specifications of the resulting algorithm

Compartments are physiologically identified tissues

Basically there are 16 tissues concerned

Nitrogen and Helium load and unload tissues half times with a ratio **Nitrogen** v helium : 2.65

M-values : maximum inert gas content at a given pressure

It is a function of absolute Pressure (P) and tissues half times

$$M = P/b + a$$

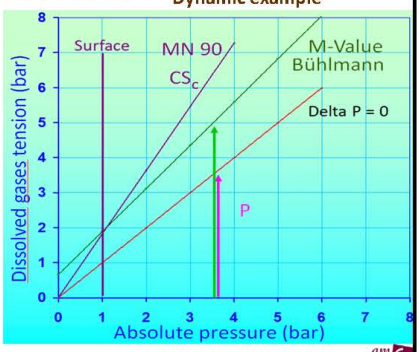
« a » and « b » constants specific of each 16 tissues

Theory of no bubble decompression –Amsterdam – March 2018

4 – BÜLHMANN'S CONCEPT (cont'ed...) 23 /40

Dynamic example

Intersection with vertical axis is ***a*** of Bühlmann and ***1/b*** is the slope of the M-Value line.

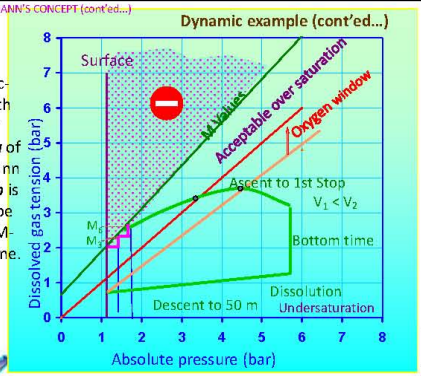


Theory of no bubble decompression –Amsterdam – March 2018

4 – BÜLHMANN'S CONCEPT (cont'ed...) 24 /40

Dynamic example (cont'ed...)

Intersection with vertical axis is ***a*** of Bühlmann and ***1/b*** is the slope of the M-Value line.



Theory of no bubble decompression –Amsterdam – March 2018

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Practical applications

Bülhmann tables are the official Swiss tables
They include calculated tables up to the altitude of 3000 m

The algorithm has been implemented in a majority of dive computers
Often with only 6 to 8 tissues and adapted M values

Some modifications to take into account bubbles growth are also added

Some computers allow individual modifications of M values to increase safety when special condition exists.

5 – DIFFUSION APPROACHES

6.1 – Hempleman's concept (RNPL Alverstoke)

Initial observation

He considered than Bends were the signs of DCI to be avoided by decompression tables.

He thought that cartilage is the site of bubble formation after slow diffusion of gas from the blood

Using a single layer diffusion model he could mimic USN NO-D curve with a simple equation :

$$Q = C\,t^{1/2}$$

Q Gas loaded at time t, to decompress back to 1 bar(a)
C is the pressure of exposure relative to surface

Diving Tables $Q = C\,t^{1/2}$

To match USN No-D limits Q should not exceed 465.

Of course this equation can not be valid for long duration exposures it was limited to 100 min
From this idea he calculated 2 series of air diving decompression tables that were formally adopted by CIRIA in UK (1968)

Air breathing at the stops

Oxygen breathing at the stops

Compressed air workers table

In 1966-72 he also produced the so called "Blackpool" tables for compressed air workers

Blackpool tables have been a great progress and have been implemented all over the Commonwealth... At least until 2000 !!!!

Blackpool tables have been used with the technique of decanting (surface decompression) with no changes !

The result has been a significant number of bone necrosis and neurological DCI cases world wide even when the number of exposures is extremely large !

The observation that new comers were more prone to DCI, lead to take into account acclimatization. New comers being exposed for shorter shifts with the same decompression profiles than "acclimatized" workers

In the present configuration of compressed air work in tunnelling, acclimatization is not considered any more since compressed air interventions are most of time not continuous.

These Blackpool tables are probably longer than needed for short duration exposures and much too short for long ones (up to 8 hours at 3 bars !!!).
In 2004 HSE lifted the stupid ban on oxygen decompression in UK

In stead of recalculating new tables to take full advantage of oxygen, they kept the same tables only shifting for oxygen at the last stops!!!!

Of course they are safer ! However there was no need for a full research project to decide this obvious change !!!

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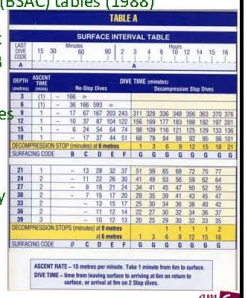
Hennessy

Also from RNPL Hennessy used a diffusion model to calculate the **B**ritish **S**ub**A**qua **C**lub (BSAC) tables (1988)

He is the first one to use a different presentation and to eliminate the 3 m stop.

He also calculated all repetitive dives tables in stead of adding an extra time to the second dive.

The French MT 92 Minitable display is copied from Hennessy with a different algorithm.



5.2 – Thermodynamic views by B. Hills (1977)

Hills proposed a thermal analogue for a diffusion only model.

A single compartment loaded by diffusion equation is used.

Limiting ascent criteria is gas phase separation.

Considering bubble condition to form and the oxygen window he calculates decompression with NO acceptable over saturation.

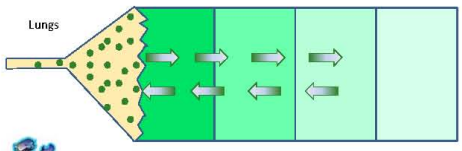
NO practical decompression tables have been derived.

5.3 - DIECM Canada

From Kidd and Stub's early pneumatic analogueNishi has developed a 4 compartment model based only on diffusion.

Ascent criteria being the maximum quantity of gas formed
The limiting values have been adjusted from detections of circulating bubbles after multiple human experimental exposures.

Series of 4 Compartments



Exchanges by diffusion (Fick's Law)

The resulting tables are the official Canadian tables (Navy and civilian)

They include Air, Air and oxygen, surface O_2 decompression and Heliox dives.

These tables are an accepted standard by IMCA, and used by many recreational organizations worldwide.

6 – SPECIFIC CASES

6.1 – The Netherland tables (Sterk Tables)

Since 1975, under proprietary calculations W. Sterk, has produced The Netherlands official decompression tables (revised 2015)

Air, air and oxygen stops and Nitrox long duration diving tables, Trimix for caisson works ... are among these tables.

6.2 – Rate of ascent to first stop

In 1956, the USNavy held a meeting to decide about a single official safe rate of ascent to the first stop (Swimmers versus Hard Hat)

Final mitigated decision : 60 ft/min (18 m/min) for both groups. No physiological background value kept by Workman with no questioning !

The 1977 édition « La Plongée »

« The rate of ascent should be as close as possible to : 17 m/min»

A new rate has been recommended (MT 92 tables) : 12 m/min

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6.2 – Rate of ascent to first stop (cont'ed...) 37 /40

In "no" bubbles models what is the rationale?

The sooner pressure is reduced the less gas uptake in not yet saturated compartments.

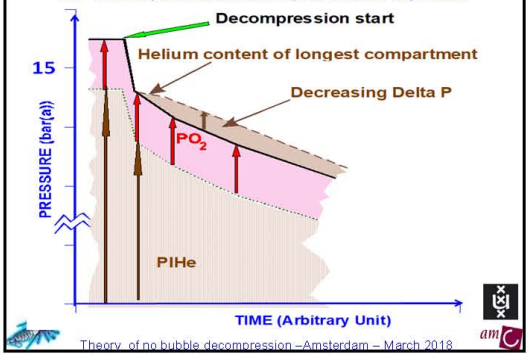
A fast ascent to the calculated first stop is better.

Unfortunately, experience have shown that there are bubbles even when they are not considered in the algorithm...

A fast ascent to the first stop may trigger gas nuclei into micro bubbles that will grow as pressure is reduced and surrounding over-saturation increases...

6 – SPECIFIC CASES (cont'ed...) 38 /40

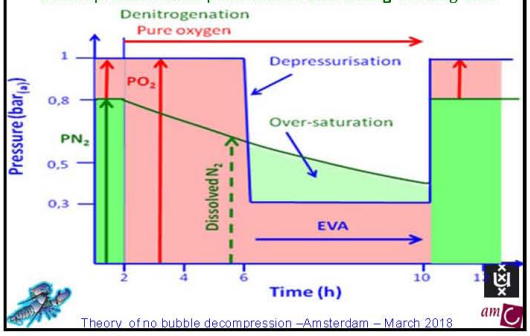
6.3 - Decompression from deep saturation exposures



6 – SPECIFIC CASES (cont'ed...) 39 /40

6.4 - Decompression from saturation for EVA

Decompression takes place **before and during** working time



40 /40

ANY FURTHER QUESTIONS OR COMMENTS

?

hyperbar@club-internet.fr

http://hyperbar.perso.sfr.fr

Chapter 2 Dual phase decompression theory and bubble dynamics

Albrecht Salm

26-2-2018

Dual Phase Decompression Theory and Bubble Dynamics

Albrecht Salm

THE

SUB
MARINE
CONSULTING

GROUP

TEL AVIV – SAN FRANCISCO – STUTTGART

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Dual Phase Decompression Theory and Bubble Dynamics

Agenda (1):
Basics of dual phase theory and its application in diving.

- What is "Dual Phase"?
- (only short) Overview on Bubble Models
- basic bubble physics, kavitation and tribonucleation,
- bubble dynamics and evolution:
- Why does a bubble exist and why does it grow or shrink?
- The critical diameter concept
- The role of bubble seeds (gas nuclei)
- How does a bubble exchange gases with its environment?
- Is a spherical bubble real or does it look more like a "cigar"?
- ...

Dual Phase Decompression Theory and Bubble Dynamics

Agenda (2):
Basics of dual phase theory and its application in diving.

- Basics of the "Varying Permeability Model" (VPM)
- VPM in a nut-shell
- The shortcomings of VPM
- "Colors" of VPM
- BVM(3) (Bubble Volume Model, USN)
- modern Hybrid-Deco-Models: "Copernicus"
- ...

Bubble Models

Description of the free gas phase (= bubbles)

3 prominent representatives:

→ VPM (Varying Permeability Model; deterministic)
- „best fit" via USN, RNPL, TEKTITE
- Implementations in various mix gas computers & free-/share ware programs

→ RGBM (Reduced Gradient Bubble Model; deterministic)
- cryptic ..., „VPM like"
- licence models for Suunto® & Mares® computers
- relatively great bubbles method ... (© ALBI)

→ BVM(3) (Bubble Volume Model; probabilistic)
- USN / 3 compartments (HalfTime = 1; 26; 316 min.)

Bubble Models

Description of the free gas phase (= bubbles)
The prominent deterministic representatives:

→ these are all DUAL PHASE
→ i.e.: FREE phase (gas bubbles)
→ AND the
→ LIQUID phase (gas in solution)
→ this is the perfusion part of the model:
→ Start: saturation
→ End: de-saturation

→ only difference to Haldane, Workman, etc:
→ „1 equation" for „safe ascent"
→ constant for all compartments / half-times
→ constant for all depths

Sources:

→ VPM: D.E. Yount, D.C. Hoffman, On the Use of a Bubble Formation Model to Calculate Diving Tables. Aviation, Space, and Environmental Medicine, February, 1986, 57: 149 – 156

→ RGBM: [71] "Reduced Gradient Bubble Model in depth", Bruce R. Wienke, Best Publishing Company, 2003, ISBN 1-930536-11-9

→ BVM(3): Gerth WA, Vann RD. Probabilistic gas and bubble dynamics models of decompression sickness occurrence in air and N2-O2 diving. Undersea Hyperb Med. 1997;24:275-92.

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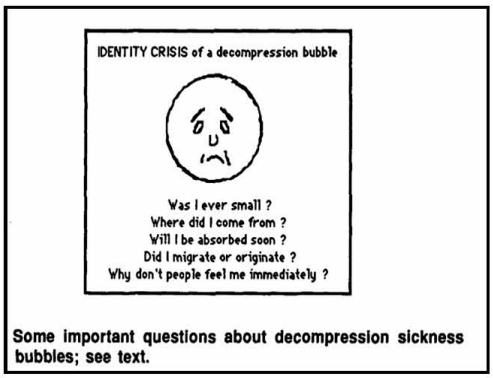
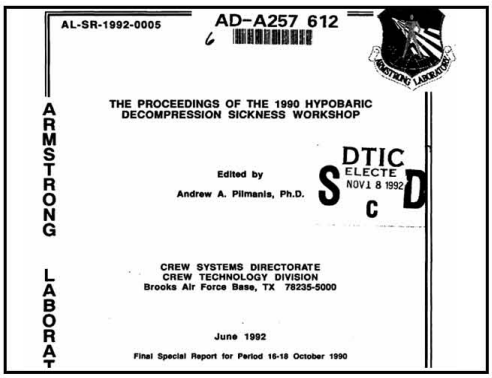


Some important questions about decompression sickness bubbles; see text.



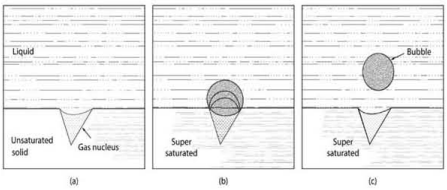


Figure 10.6 Depiction of a gas micronucleus resident in a crevice on a blood vessel wall. In the unsaturated state (a), the nucleus is quiescent. When the underlying tissue becomes supersaturated (b), gas diffuses into the nucleus and causes it to grow until such time that a bubble breaks off (c), after which the cycle will be repeated.

Source: Edmonds et al., 5.th edition, 2016, p. 129

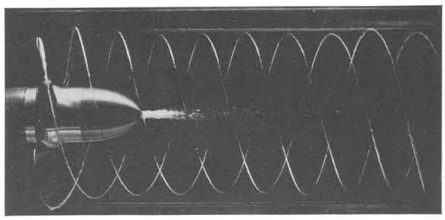
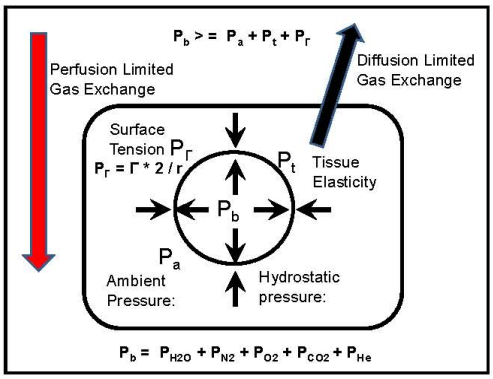


Abb. VII, 54. Kavitationsblasen hinter einem rotierenden Schiffspropeller (Versuchsanstalt für Wasserbau und Schiffbau, Berlin)



The „real" physics:

only the partial pressures of the gases:

$P_b = P_{H2O} + P_{N2} + P_{O2} + P_{CO2} + P_{He}$

only the basic pressure components:

$P_b >= P_a + P_t + P_r$

with:

$P_a = P_{hydrostatic} + P_{dynamic/blood}$

$P_t = H * V$

$P_r = \Gamma * 2 / r$

r : radius of the bubble
V : volume of bubble (= displaced tissue)
H : tensor of bulk modulus for tissue elasticity
Γ : surface tension constant

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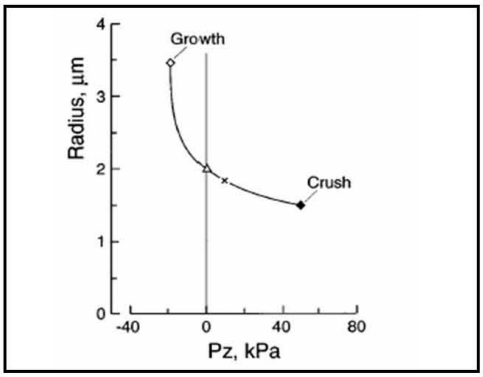
Generic example for a small bubble:

Γ = 50 mN/m (=50 dyn/cm)
r = 1 µm, then:
P_Γ = 1 Bar
and thus the internal bubble pressure:
ca. 2 Bar at P_a = 1 Bar and P_t = 0.

Water / Air e.g.: Γ = ca. 72 mN/m at ca. 37° C;
Blood / Air e.g.: Γ = ca. 58 mN/m at ca. 37° C;
Quicksilver (Hg) / Air: Γ = ca. 485 mN/m at ca. 20° C
Ether / Air: Γ = ca. 20 mN/m at ca. 20° C

For a valid doppler-reading required:
r > 40 µm and / or v > 20 cm/s
depending on site (pre-cordial, sub-clavia, …) &
measuring frequency (1- 6 MHz)



The critical radius r_c

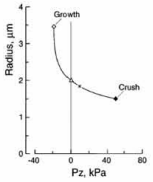
Metastable state, bubble either:
→ crushes
→ expands

$P_b = P_a + P_t + P_\Gamma$

P_t = 0, thus:

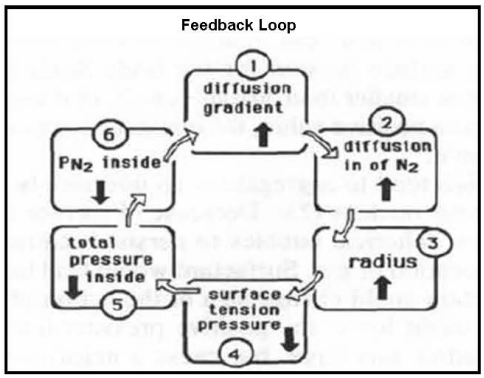
$P_b = P_a + P_\Gamma = P_a + \Gamma * 2 / r$

$r_c = \Gamma * 2 / \{P_b - P_a\} = \Gamma * 2 / P_{SS}$



Supersaturation $P_{SS} = P_b - P_a$

For P_{SS}, please see as well slides # 24, 31/32, 38/39



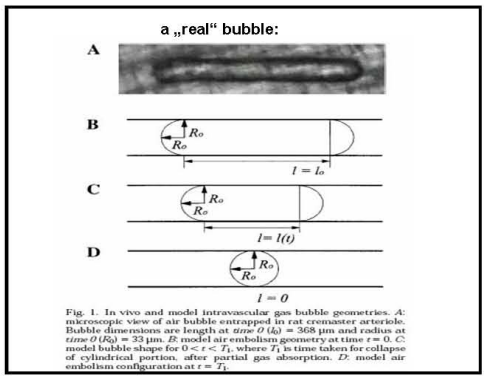
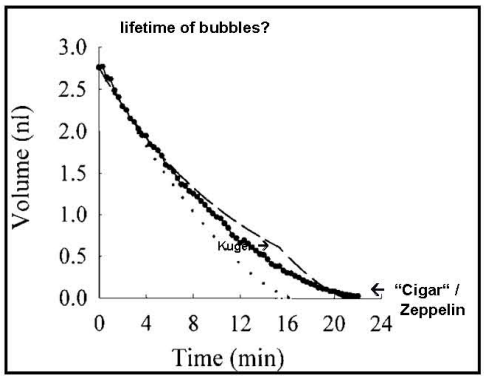


Fig. 1. In vivo and model intravascular gas bubble geometries. *A*: microscopic view of air bubble entrapped in rat cremaster arteriole. Bubble dimensions are length at *time 0* (l_0) = 368 µm and radius at *time 0* (R_0) = 33 µm. *B*: model air embolism geometry at time t = 0. *C*: model bubble shape for $0 < t < T_1$, where T_1 is time taken for collapse of cylindrical portion, after partial gas absorption. *D*: model air embolism configuration at $t = T_1$.



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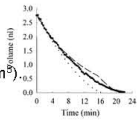
lifetime of bubbles?



Measured and predicted absorption times for bubble having initial volume of 2.8 nl (=2.8 mm^3).

In vivo measurement (large dotted line),

Prediction using initial in vivo bubble dimensions (dashed line), and

Prediction assuming spherical bubble of same initial volume (small dotted line) are shown.

Sources:

Franklin Dexter, MD, PhD, and Bradley J. Hindman, MD: Recommendations for Hyperbaric Oxygen Therapy of Cerebral Air Embolism Based on a Mathematical Model of Bubble Absorption, Anesth Analg 1997;84:1203-7

Branger, Annette B., and David M. Eckmann. Theoretical and experimental intravascular gas embolism absorption dynamics. J. Appl. Physiol. 87(4): 1287–1295, 1999

Bubble Models

VPM in a nutshell (1):

Bubble-Counts at Knox-Gelatin
- **Bubble seeds, ca. 1 µm**
- **Bubbe Growth through Δp and Diffusion**
- **Bubble Distribution: exponential (many small, less big)**
- **Criteria for „safe“ Decompression:**
- **Initial hypothesis:**
  - **constant # of Bubbles (= const. „deco stress“):**
  - **good for saturation dives, failed for short dives!**

- **Later on, modified hypothesis:**
- **(dynamical) critical net volume of free gas $V < V_{crit}$**
  - **How can we keep V small?**
  - **High Pressure! Ergo: deeper stops than USN!**

Bubble Distribution (1)

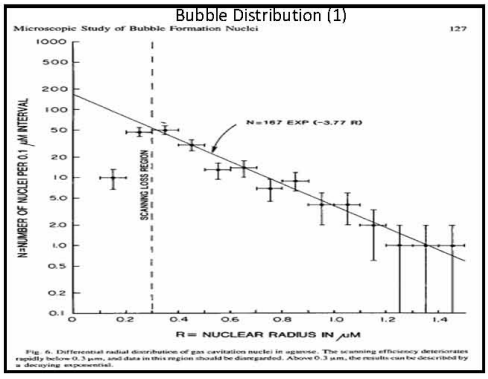
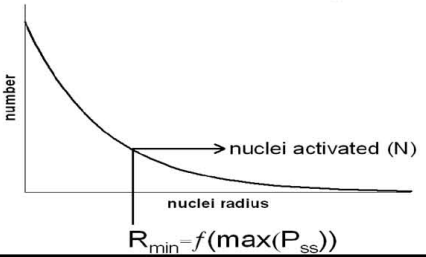


Fig. 6. Differential radial distribution of gas cavitation nuclei in agarose. The scanning efficiency deteriorates rapidly below 0.3 µm, and data in this region should be disregarded. Above 0.3 µm, the results can be described by a decaying exponential.

Bubble Distribution (2)

Exponential distribution of gas nuclei radii. The number of gas nuclei activated into growth as bubbles (N) is all nuclei of R_{min} and larger. R_{min} is a function of the maximum supersaturation (P_{ss}).



$R_{min}=f(\max(P_{ss}))$

Bubble Models

VPM in a nutshell (1a, history)

PROCEEDINGS OF THE SIXTH SYMPOSIUM ON UNDERWATER PHYSIOLOGY
Sponsored by
Institute for Environmental Medicine, University of Pennsylvania Medical Center
Undersea Medical Society
U.S. Office of Naval Research
U.S. National Oceanic and Atmospheric Administration
Edited by C. W. Shilling and M. W. Beckett

T. D. KUNKLE AND D. E. YOUNT

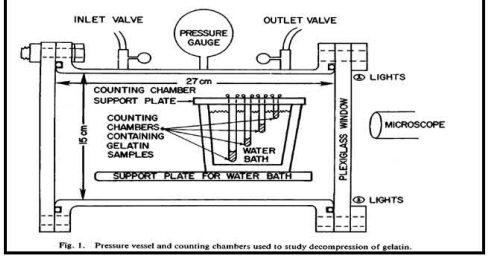


Fig. 1. Pressure vessel and counting chambers used to study decompression of gelatin.

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Bubble Models

VPM in a nutshell (1b, history)

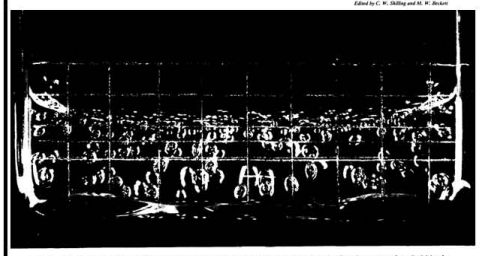
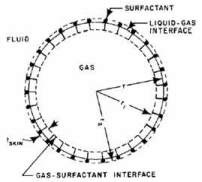
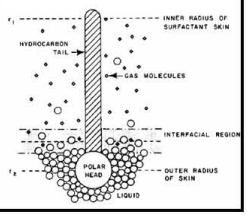


Fig. 2. Photograph of a counting chamber and gelatin sample taken a few minutes after decompression; bubbles in the upper 1 mm were noticeably smaller and more numerous than elsewhere; only those in the lower 3 mm were counted (this corresponds to a fiducial volume of about 3 mm × 6 mm × 27 mm).

Bubble Models

VPM in a nutshell (2)





Note of Editor: See Addemdum A (Bubbles are probably not covered by a fatty acids monolayer or other lipids but by protein).

Bubble Models

VPM in a nutshell (3):

- **5 Parameters, via best fit with established tables:**

→ **USN (United States Navy)**
→ **RNPL (Royal Navy Physiological Laboratory)**
→ **Tektite, saturation dives at 110 feet, Carribean, 1971, with ca. 21 h decompression**

- **Test-Bubble, min. initial radius $r_{0\ min}$ ca. 0,8 µm**
- **Surface Tension: Γ = 17,9 mN/m***
- **Skin Compression: Γ_c = 257 mN/m**
- **Regeneration Time constant: 20160 min**
- **Composite Parameter λ = 7500 fsw (=228 m) x min**
- **(Onset of impermeability: p* = ca. 9.2 ata)**
- * Note of Editor: See Addemdum B (A realistic Γ = 58 mN/m)

VPM in a nutshell (4):

Comparison with „OLD USN":

(new ones from 2008 and 12/2016 look different: no more 3 m stops! longer stops At 6 m and deeper)

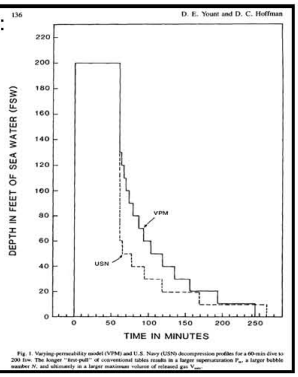
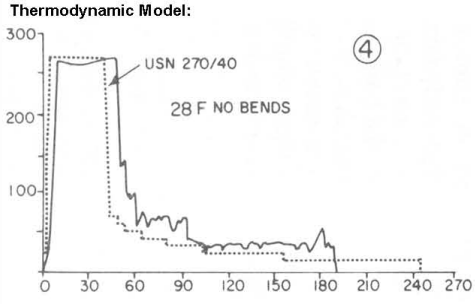


Fig. 1. Varying-permeability model (VPM) and U.S. Navy (USN) decompression profiles for a 60-min dive to 200 fsw. The longer "first pull" of conventional tables results in a larger supersaturation P_{ss}, a larger bubble number N, and ultimately in a larger maximum volume of released gas V_{max}.

Thermodynamic Model:

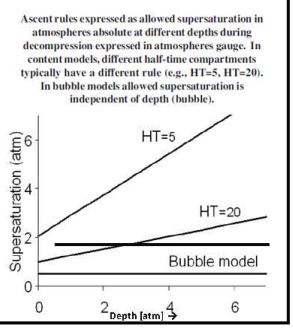


Source: Hills, Brian Andrew (1977), Decompression Sickness, Volume 1, The Biophysical Basis of Prevention and Treatment, John Wiley & Sons, Ltd.. ISBN 0 471 99457 X, [102], p. 260

Shortcomings (1) etc.:

→ **Saturation &**
→ **De-Sat. Calculation**
→ **same as Perfusion-**
→ **models**
→ **no tissue compliance**
→ **Bubble Distribution?**
→ **i.e. r_{Bubble} & #**
→ **Parameter Fit …**

→ **thus:**
→ **many**
→ **„Colours" of the VPM:**



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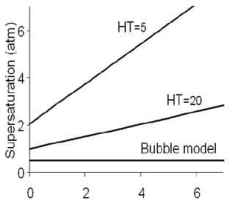
Shortcomings (2) etc.:

Ascent rules expressed as allowed supersaturation in atmospheres absolute at different depths during decompression expressed in atmospheres gauge. In content models, different half-time compartments typically have a different rule (e.g., HT=5, HT=20). In bubble models allowed supersaturation is independent of depth (bubble).



→ as per nearly ALL perfusion models:
only one pressure step;
→ i.e.: NO multi-level tests
→ up to now (2018):
→ not seriously challenged with
→ Trimix / Heliox

Shortcomings (3) etc.:

..., thus the many „Colours" of the VPM (1):

→VPM-B: B = Boyle; includes the expansion of the bubbles at ascent

→ VPM-B/E: additional manual input of conservativism for Extreme dives

→VPM-CVA: Critical Volume Algorithm

→ VPM-B/FBO: Fast Bail Out option

Colours of the VPM (2):

→VPM-B/GFS: regularly the deep stops are too long and the shallow to short; thus a parallel ZH-L with Gradient Factor Surfacing (GF Hi = GF Lo = 0,9) is calculated and the more conservative profile is displayed for the shallow stops

→VPM-BS: there is only one critical radius for each gas: ca. 0,6 µm for N_2 and ca. 0,5 µm for He;

now each compartment gets its own value:
N_2: 0,35 µm fastest compartment → 2,0 µm slowest compartment
He: 0,25 µm fastest compartment → 1,9 µm slowest compartment

Shortcomings (4)

In the own words of Yount & Hoffman;
(Source: l.c., p. 144):

Another criticism is that we have said very little about the physiological processes that presumably underlie our mathematical equations. We take oxygen and carbon-dioxide into account and assume a reasonable range of tissue half times, but many other details are overlooked. We make no distinction, for example, between "fatty, loose tissue" and "watery, tight tissue" (14), nor do we state explicitly where the bubbles form or how they grow, multiply, or are transported. Finally, we say nothing about such factors as solubility, diffusion versus perfusion, tissue-deformation pressure, or tissue-specific differences in surface tension. Our response to criticisms of this type is that most of the omitted processes are poorly understood, and their inclusion at this stage would serve only to complicate the model and increase the number of undetermined parameters.

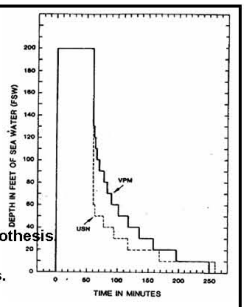
VPM in a nutshell (5):

more sources:

36th. UHMS workshop p. 48

and, as well:

Yount DE, Hoffman DC.
Decompression theory:
a dynamic critical-volume hypothesis.
In:
Bachrach A J, Matzen MM, eds.
Underwater physiology VIII:
proceedings of the eighth symposium on underwater physiology.
Bethesda: Undersea Medical Society, 1984:131-46.



VPM in a nutshell (7):

The REAL Source!

ON THE USE OF A GAS-CAVITATION MODEL TO GENERATE PROTOTYPAL AIR AND HELIUM DECOMPRESSION SCHEDULES FOR DIVERS

A DISSERTATION SUBMITTED TO THE GRADUATE DIVISION OF THE UNIVERSITY OF HAWAII IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

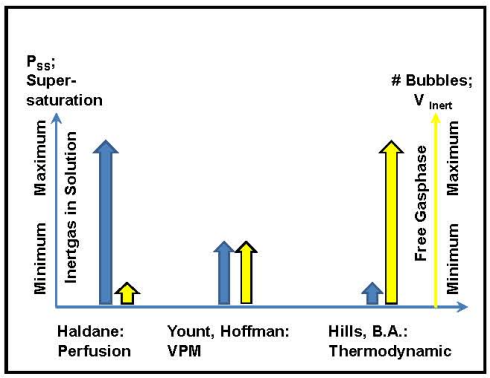
IN PHYSICS

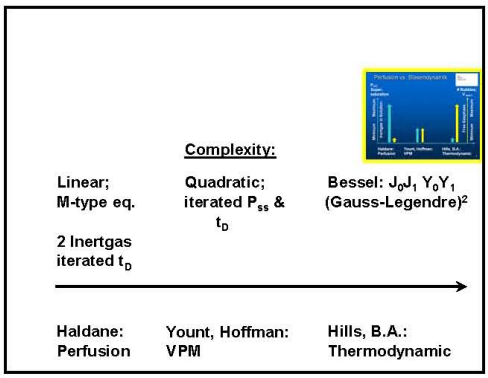
AUGUST 1985

By

Donald Clinton Hoffman

26-2-2018





Copernicus: Original Charts from Alf Brubakk @ ntnu.no

Copernicus (1)

- Copernicus consists of:
  - *The model itself.* A descriptive model of the mechanisms behind the occurence of serious (CNS) DCS (evolution of vascular bubbles)
  - *A control algorithm.* It calculates a procedure that control the model according to our request (Dynamic optimization algorithm)

Copernicus (2)

- Copernicus consists of:
- Validation strategy through bubble measurement rather than DCS/NO-DCS endpoint
- The model will have two distinct applications:
  - Use the model as a simulator to compare and evaluate procedures and logged dives
  - Use accepted risk/bubble scores as threshold for calculating optimal decompression schedules

Physiological model

- Individually adaptive
- Input:
  - Weight
  - Height
  - Fitness (fat percentage)
  - Gender
  - Cardio-pulmonary performance
  - Workload

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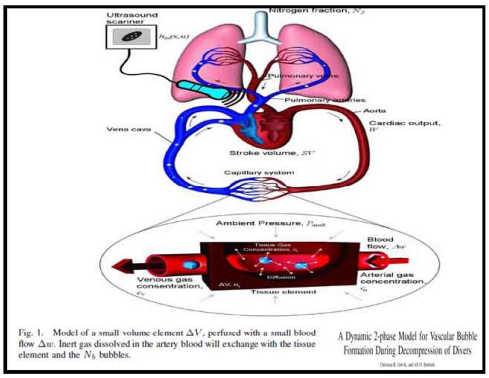
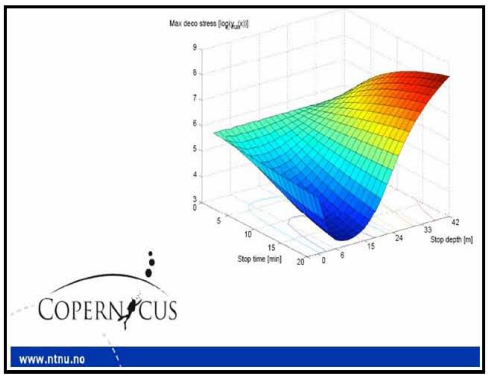
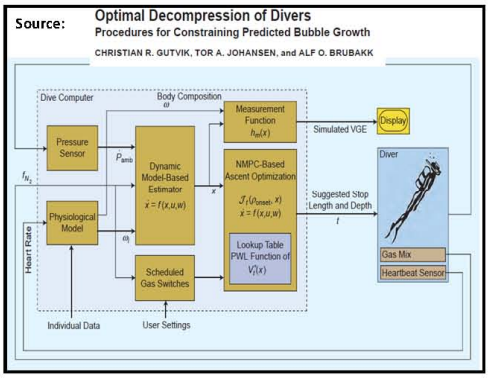
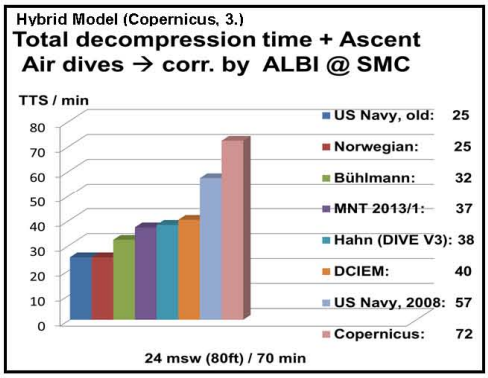








Possible, tentative and surprising conclusion

- Dives with long, > 30 minutes, bottom time may benefit from deep stops
- Dives with short, < 30 minutes, bottom time will not benefit from deep stops.

Take home (1):

VPM is a MODEL

→ not better or worse than any other (→ next slide …)
→ many simplifications
→ 5 free parameters, with:
→ "best fit" through traditional diving tables
→ many different implementations

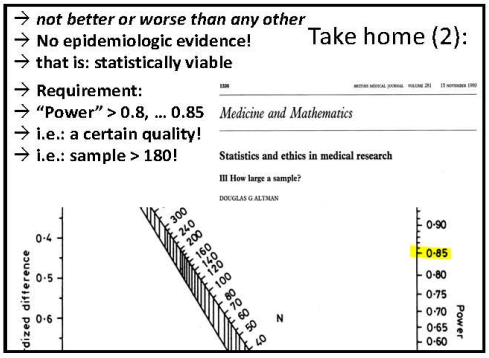
→ Which one is "true"?
→ ALL AD HOC
→ NONE Tested
→ Don't use a single source!
→ Compare always with established tables!

26-2-2018

→ *not better or worse than any other*
→ No epidemiologic evidence!
→ that is: statistically viable

Take home (2):

→ Requirement:
→ "Power" > 0.8, ... 0.85
→ i.e.: a certain quality!
→ i.e.: sample > 180!



21st century decompression?

"Note:
The senior (elder) author believes that
the only explanation for most cases of DCS lies in the random application of Chaos Theory,
which he also does not understand,
or String Theory which no-one understands."

[Diving Medicine for Scuba Divers, Edmonds et al.,
ISBN: 978-0-646-52726-0, p. 138]

Addendum 2A

Aviat Space Environ Med. 2014 Nov;85(11):1086-91. doi: 10.3357/ASEM.3985.2014.

Free fatty acids do not influence venous gas embolism in divers.

Schellart NA[1].

Author information

1 Biomedical Engineering and Physics, Academic Medical Center, University of Amsterdam, Amsterdam, The Netherlands.

Abstract

BACKGROUND: Decompression sickness is caused by bubbles of inert gas predominantly found in the venous circulation. Bubbles may exist longer when covered by a surfactant layer reducing surface tension. Surfactant candidates, based on 3D-structure and availability, are long-chain fatty acids (FFAs). It is hypothesized that sufficient molecular dissolved FFA (dFFA) result in higher bubble grades (BGs).

METHODS: Participating divers (52) either had a fat-rich or a fat-poor breakfast. After a dry dive simulation (21 msw/40 min), BGs were determined at 40, 80, 120, and 160 min after surfacing by the precordial Doppler method. The four individual scores were transformed to the Kisman Integrated Severity Score (KISS).

RESULTS: Kiss was not affected by meal fat content, and KISS and dFFA (calculated) were not associated, even though the fat-rich group had 3.5 times more dFFA. A paired approach (11 subjects exposed to fat-rich and fat-poor meals) yielded the same results. The measured FFA (albumin bound) was present in abundance, yet the long-chain dFFA concentration was probably too low (nM range) to form a surfactant monolayer, as follows from micelle theory.

CONCLUSION: Bubble scores are not associated with dFFAs. Theoretically it is questionable whether long-chain dFFAs could form post-dive monolayers. It remains unclear which substance forms the surfactant layer around bubbles.

PMID: 25329940 DOI: 10.3357/ASEM.3985.2014

Addendum 2B

Undersea Hyperb Med. 2015 Mar-Apr;42(2):133-41.

Relationships between plasma lipids, proteins, surface tension and post-dive bubbles.

Schellart NA, Rozložník M, Balestra C.

Abstract

Decompression sickness (DCS) in divers is caused by bubbles of inert gas. When DCS occurs, most bubbles can be found in the venous circulation: venous gas emboli (VGE). Bubbles are thought to be stabilized by low molecular weight surfactant reducing the plasma-air surface tension (γ). Proteins may play a role as well. We studied the interrelations between these substances, γ and VGE, measured before and after a dry dive simulation. VGE of 63 dive simulations (21-msw/40-minute profile) of 52 divers was examined 40, 80, 120 and 160 minutes after surfacing (precordial Doppler method) and albumin, total protein, triglycerides, total cholesterol and free fatty acids were determined pre- and post-exposure. To manipulate blood plasma composition, half of the subjects obtained a fat-rich breakfast, while the other half got a fat-poor breakfast pre-dive. Eleven subjects obtained both. VGE scores measured with the precordial Doppler method were transformed to the logarithm of Kisman Integrated Severity Scores. With statistical analysis, including (partial) correlations, it could not be established whether γ as well as VGE scores are related to albumin, total protein or total cholesterol. With triglycerides and fatty acids correlations were also lacking, despite the fact that these compounds varied substantially. The same holds true for the paired differences between the two exposures of the 11 subjects. Moreover, no correlation between surface tension and VGE could be shown. From these findings and some theoretical considerations it seems likely that proteins lower surface tension rather than lipids. Since the findings are not in concordance with the classical surfactant hypothesis, reconsideration seems necessary.

PMID: 26094288

Addendum 2C

Aviat Space Environ Med. 2008 May;79(5):488-94.

Bubble formation after a 20-m dive: deep-stop vs. shallow-stop decompression profiles.

Schellart NA[1], Corstius JJ, Germonpré P, Sterk W.

Author information

Abstract

OBJECTIVES: It is claimed that performing a "deep stop," a stop at about half of maximal diving depth (MDD), can reduce the amount of detectable precordial bubbles after the dive and may thus diminish the risk of decompression sickness. In order to ascertain whether this reduction is caused by the deep stop or by a prolonged decompression time, we wanted to test the "deep stop" theory without increasing the total decompression time. From a modeling point of view, Haldanian theory states that this situation would increase the probability of observable bubbles, because of a longer stay at depth. Under these conditions, we examined whether a "deep-stop dive" (DSD) produces more bubbles or less than a "shallow-stop dive" (SSD).

METHODS: Recreational divers performed either a DSD or a SSD. Both groups were matched biometrically. MDD was 20 msw, bottom time 40 min and total diving time 47 min. In DSD, the "deep" stop (10 msw) replaced 3 min of the 7 min stop at 4 msw of SSD.

RESULTS: DSD produced significantly more precordial bubbles than SSD after knee bends (P-values ranging from 0.00007 to 0.038).

DISCUSSION: Our results indicate that at least for the tested dive profile, the higher supersaturations after surfacing overruled any possible beneficial effects of the deep stop on bubble formation. The usefulness of substituting a shallow stop with a deep stop in dives up to 20 msw can be questioned; at the least, more research is needed.

PMID: 18500045

Chapter 3 Differential Diagnosis for Decompression Illness

Adel Taher

It is of paramount importance that we understand the importance and value of establishing a differential diagnosis (DD) in dive accidents. The ramifications that follow will affect the patient, the prognosis, the rescue personnel involved with the medivac of the accident and the costs that will need to be covered by the insurance company.

One should consider that recently, dive destinations are becoming more exotic, further and harder to reach and the medical infrastructure in that remoteness varies from scarce to nonexistent. The physicians on site and in the field are faced with the burden of taking the decision, whether a diver should be moved to a recompression chamber or not. Actually, the first question they have to answer before that is whether the presenting accident is a dive accident or not?

Divers reached recompression chambers following very risky, costly and difficult evacuations to discover that the symptoms they had were attributed by a whiplash or cervical spondylosis and not in the least diving related or in other instances, they arrived free of symptoms, as the normobaric oxygen they breathed during the transport was enough to cure them!

"A proper treatment is based on a correct diagnosis." *Carl Edmonds.*

A multitude of factors need to be considered when examining a presumed ‘diving accident’: Pre-existing conditions, medication and their side effects, medical history, elements during the dive that could have provoked pathophysiological changes resulting in symptoms, other ailments that could have been dive related, but do not classify as decompression illness, equipment considerations, environmental conditions and many other factors.

The current population of divers is partly an aging one, as many have learned in the 70ies boom. Many of them have cardio-vascular and other chronic diseases and may not be fit to dive, but continue to engage in the sport.

We should consider any symptom following breathing a gas under pressure or even following deep, repeated breath hold diving, a possible decompression illness (DCI) until proven otherwise.

Some guidelines for the train of thought may help in reaching a DD. Any symptoms developing at depth, during the isopression phase, are usually safe to exclude from DCI, though some could be misleading, like immersion pulmonary edema, as the symptoms will continue through the recompression phase and at the surface. Some symptoms of arterial gas embolism and cerebral arterial gas embolism (AGE/CAGE) can start in the last few meters of ascent and continue at the surface. Pneumoencephalus is another rare and confusing incident that will worsen with the reduction of the ambient pressure.

Pre-existing conditions could be affected by the increase in ambient pressure and the barotraumata of ascent can cause seventh cranial nerve and trigeminal neuralgia symptoms. Previous trauma could result in nerve entrapment that could easily be attributed to DCI.

The author mentions a multitude of conditions that could easily be confused with DCI and directs the examining physician to use all the tools at hand to identify the causes of the symptoms and decide then whether these are DCI related or not and whether the patient needs recompression and evacuation or not. Examples are: pre-existing disease, like migraine or multiple sclerosis (MS), cardio-vascular conditions, metabolic derailment, like diabetes mellitus (DM), pulmonary disease, allergic dermatitis, drug and alcohol abuse, anxiety and psychiatric disorders, seizure disorders, vertebral disc prolapse, effects caused by medication and many other conditions.

An example of a difficult DD with inner ear symptoms and the possible consequences of a wrong diagnosis is discussed. A short account of Facial baroparesis is also mentioned.

Consultation with more experienced specialists and consultants should be secured 24/7 and are the safeguard against premature, and often wrong, decisions.

At the end, a reminder that divers receiving recompression therapy and not showing signs of improvement are most likely not suffering a DCI.

Note that in the slide show the slides are ordered vertically.

Differential Diagnosis for Decompression Illness

Amsterdam, AMC Capita Selecta Duikgeneeskunde, 2018

- ***Establishing a differential diagnosis (DD) using modern investigative and diagnostic tools is possible, though still non-conclusive if not directly related to the medical history, study of the dive profile and the results of the clinical examination.***
- ***We will talk about the DD from the perspective of the field physician in a remote diving destination faced with a case and needing to take a decision!***

- This presentation is based on information from 'Diving Medicine' by Alfred A. Bove,
- 'Diving and Subaquatic Medicine', by Carl Edmonds and others,
- DAN-Europe and DAN-USA Annual Reports,
- the authors own observations in the field.

- ***Before*** sending an air ambulance or a Search & Rescue (SAR) boat and team, one needs to be certain that it is indeed ***a DCI in need of recompression therapy and … requiring an evacuation!***

Differential Diagnosis

- ***the process of differentiating between two or more conditions which share similar signs or symptoms***
- ***"A proper treatment is based on a correct diagnosis."*** *Carl Edmonds*

- Divers reached recompression chambers after ***long, risky, rescue medical evacuations*** suffering from:
- ***a disease that was NOT a DCI, or***
- ***fully RECOVERED on normobaric O_2 and rehydration, without the need for recompression***

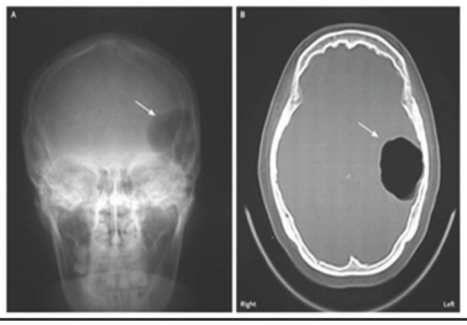
What should be considered when thinking about DD?

- Factors that need to be considered when examining a *"probable"* diving accident and deciding whether or not it should be evacuated and recompressed:
  - ❑ **Pre-existing conditions, medication and their side effects & medical history**
  - ❑ **Diving physiology/pathophysiology (other ailments that are not a DCI)**
  - ❑ **The accident & the diver**
  - ❑ **Equipment consideration**
  - ❑ **Environmental conditions**
  - ❑ **others**

- Symptoms occurring **under the water** at the ***isopression phase*** are usually non DCI related
- Symptoms of ***Immersion Pulmonary Edema*** will occur mostly at depth and can be confusing
- Some cases of ***AGE/CAGE, Round Window Rupture & Pneumoencephalus*** can manifest symptoms in the last few meters of the ***ascent*** (decompression phase) and at the surface

- About **23 to 25 millions actively dive around the globe**
- In the **70's** the diving boom attracted thousands of thrill seekers
- They are **now older**, mostly suffer from a variety of **diseases** and are on **medication**

What's your diagnosis?



- Many of them are ***neither fit, nor diving eligible, and they do not feel comfortable in the water…***

but, they still dive!!

Symptoms following a scuba or rebreather dive are considered DCI until proven otherwise…

(skin rash, pain, difficulty in breathing, neurological deficits, other abnormalities)

- ***Pre-existing conditions*** could be affected by the ***increase in ambient pressure*** and its ramifications and contribute to the observed symptoms (e.g. CV, epilepsy & medication!)
- ***Barotrauma of ascent*** can cause an increase in the ***middle ear*** pressure and affect the ***VIIth*** cranial nerve (Facial). The ***maxillary sinus squeeze*** will affect the ***Vth*** cranial nerve causing ***Trigeminal neuralgia***
- ***Nerve entrapment*** and swelling could be the result of ***previous trauma*** and not DCI linked

- The ***first 6 to 8 hours following the ascent are crucial in diagnosis.*** Symptoms manifesting after 12 hours should be scrutinized and symptoms after 24 hours are most probably not DCI
- ***CAVE!*** Driving to ***altitude or flying*** can produce symptoms in some cases ***after 24 hours.*** Careful history taking is mandatory!

Countless conditions mimic symptoms of DCI…

- ***Ingested sea food poisons*** (ciguatera, tetrado-toxin, paralytic shell fish poisoning {PSP: is a serious illness caused by eating **shellfish** contaminated with dinoflagellate algae that produce harmful toxins. Some of these toxins are 1,000 times more potent than cyanide, and toxin levels contained in a single **shellfish** can be fatal to humans}.,…)
- ***Immersion Pulmonary Edema***
- ***Coincidental neurological disorders, e.g.: stroke*** (Vann et al 2011)

Countless conditions mimic symptoms of DCI:…

- ***Contamination of breathing gas*** (CO_2, CO, Oil mist, solvents,…)
- ***Salt water aspiration, near drowning and the resultant pulmonary edema and brain hypoxia***
- ***Guillian-Barre Syndrome***
- ***Multiple sclerosis***
- ***Migraine***

- ***Transverse Myelitis*** Transverse myelitis is an inflammation of both sides of one section of the spinal cord. This neurological disorder often damages the insulating material covering nerve cell fibers (myelin).
- Transverse myelitis interrupts the messages that the spinal cord nerves send throughout the body. This can cause pain, muscle weakness, paralysis, sensory problems, or bladder and bowel dysfunction. (Mayo Clinic)
  - ***Spinal cord compression***
  - ***Post-ictal state after unrelated seizure***
  - ***Ischaemic or haemorrhagic stroke***
  - ***Subarachnoid hemorrhage***
  - ***Cold water immersion edema***
  - ***Unrelated seizure*** (hypoglycaemic, epileptic,…)

Countless conditions mimic symptoms of DCI:…

- ***Porphyria*** (hereditary disease in which there is abnormal metabolism of the blood pigment haemoglobin. Porphyrins are excreted in the urine, which becomes dark; other symptoms include mental disturbances and extreme sensitivity of the skin to light)
- ***Inner ear barotrauma***
- ***Trauma before, during and after the dive***
- ***Marine life envenomation*** (scorpionidae family, cone shells, jelly fish, sea snakes…)

Typical signs and symptoms of Transverse Myelitis include:

- ***Pain.*** Transverse myelitis pain may begin suddenly in your lower back. Sharp pain may shoot down your legs or arms or around your chest or abdomen. Pain symptoms vary based on the part of your spinal cord that's affected.
- ***Abnormal sensations.*** Some people with transverse myelitis report sensations of numbness, tingling, coldness or burning. Some are especially sensitive to the light touch of clothing or to extreme heat or cold. You may feel as if something is tightly wrapping the skin of your chest, abdomen or legs.

- ***Weakness in your arms or legs.*** Some people notice that they're stumbling or dragging one foot, or heaviness in the legs. Others may develop severe weakness or even total paralysis.
- ***Bladder and bowel problems.*** This may include needing to urinate more frequently, urinary incontinence, difficulty urinating and constipation.

Rebreather Problems *Semi-Closed and Closed Circuit Rebreathers (SCCR & CCR)*

- **Hypercapnia:** scrubber problems
- **Hypoxia:** problems with O2 sensors and solenoids, inefficient initial flushing of loop, lack of mixed gas additive or breathing gas

- ***Environmental thermal stress:*** heat exhaustion, heat stroke, hypothermia

Diving Physiology/Pathophysiology

- ***Apnoea diving after scuba diving*** could result in:
  -dislodging of bubbles in the lung filter and their arterialization (AGE/CAGE)

Failure of any of the dive gear components can produce confusing symptoms, e.g.: leaking membrane of 2nd stage causing ***salt water aspiration***

- ***Hypercapnia:*** Insufficient CO_2 elimination in commercial helmet divers, i.e. the ventilation is not adapted to the depth and work load

Diving Physiology/Pathophysiology

- **DCI: (decompression sickness + AGE/CAGE)**
  -DCS: exceeding the dive tables limits, out of air situations, emergency ascents, "Divemaster syndrome"

  AGE/CAGE: buoyant ascents, sealed lips syndrome, panic, air trapping! (asthma, pulm. cysts and bullae), arterialization of bubbles

Differential Diagnosis

Other diagnoses have to be excluded, e.g.:

- Effects and side effects of ***medication***
- Newly incurred ***psycho-neurotic problems***
  (panic attacks with hyperventilation, psychosomatic and somatoform disorders)

- **Metabolic derailment:**
  - e.g.: diabetes mellitus
- **Pulmonary disease**
  - Asthma, COPD, cysts and bullae
- **Peripheral neuropathy**
- **Intracranial aneurysms**
- **Cerebro-vascular events**
- **Inner ear diseases:** e.g. viral labyrinthitis
- **Allergic contact dermatitis and other allergic reactions**

Differential Diagnosis

- **Pre-Existing Disease**
  (e.g.: Migraine headaches, multiple sclerosis, joint- and musculo-skeletal complaints,...etc)
- **Cardio-vascular Conditions**
  Sick-Sinus-Syndrome → Syncopy due to pressure exerted through narrow hood or narrow neck collar with dry suits

- **Gastric ulcer**
  -Mesenteric artery embolism
- **Acute & chronic ischemia**
- **Drug, alcohol and nicotine abuse**
- **Anxiety and other psychiatric disorders**
- **'Photographer's arm', bursitis and Tennis elbow**
  - due to holding heavy equipment or camera in one hand during the dive

- **Cardiac dysfunction:**
  Vasovagal reflexes, stress, vasoconstriction, Cold water immersion, hyperventilation
- **Angina / acute ischemia / MI**
- **Pneumopericardium and supraventricular tachycardia**

- **Whiplash!!**
  - small road accident on the way to the boat or few days earlier… symptoms appear after diving due to over-extension of neck
- **Seizure disorders**
- **Guillain-Barre syndrome**
  - acute, progressive form of neuropathy, accompanied by muscle weakness and mild sensory loss. Begins usually after a trivial infection, OP or immunization

- **Brachial plexus Pull:**
  - getting on dive boat ladder in high waves, hanging to line and being pulled by zodiac,…
  - pain in the elbow joint, tingling, hyper-aesthesia
- **Trigeminal neuralgia**
- **Vertebral disc prolapse and lower back pain**
  -acute or chronic, nerve roots irritated by mechanical stress caused by unequal weight distribution on uneven surfaces (e.g. a moving boat)

- ***New, remote and exotic diving destinations*** make it ***increasingly difficult*** to organize ***medical evacuations***. So, terms such as:

FIRST AID MEASURES

DIFFERENTIAL DIAGNOSIS,

EVACUATION (MEDEVAC)

bear a ***new meaning*** and have ***costly*** consequences

Medication

Very often the diver does not confess to taking medication…! These could cause:

- **Unexpected side effects**
  - Under conditions of elevated ambient pressure the function of the blood-brain barrier (BBB) can be greatly impaired
- **Antiemetics**:
  - tiredness, fatigue, loss of consciousness
- **Antihypertensives**:
  - reflex time reduction up to somnolence

- ***A very thorough medical history taking***
- ***dive pattern and dive profile study***
- ***general, neurological and clinical examinations*** are needed to rule out and/or to confirm a diagnosis
- ***Information should be carefully gathered*** from diver, log book, medical statements, dive buddies and family members and friends

- **Antihistamines:**
  - vasoconstriction, dizziness up to unconsciousness
- **Psychotropic medications:**
  - antidepressants and mood stabilizers crossing the Blood Brain Barrier (BBB)… effects produced by high ambient pressures are not studied yet!

- ***SAR missions can be very hazardous to the rescue crew and extremely costly***
- ***Giving the wrong treatment, based on the wrong diagnosis can have catastrophic results***

Exemple Grazia:

- DD is based on ***detailed history taking, analysis of the information, profile study*** and ***clinical examination***
- **Inner ear symptoms** can be very tricky
- *following a dive, a patient started vomiting repeatedly, had bouts of vertigo, was disoriented and could not walk unsupported*
- *SAR transported the patient to the chamber in two hours*

Clinical Examination:

- *Severe nystagmus >>>RWR*
- *Otoscopy: ear wax impaction, imbibed with salt water and explaining the decreased hearing due to conductive deafness*
- *If the picture was that of otitic barotrauma with or without haemotympanum >>> in favor of middle and inner ear barotraumata*

- *He also suffered from a moderate degree of hearing loss*
- *When asked about the dive profile, he said it was his first dive of the day, 32 msw for 25 minutes and he had some problems equalizing on his descent*

⇨ *pt. could be suffering from* ***vestibulo-cochlear DCS, Round Window Rupture or barotrauma of inner ear without RWR***

What if the wrong diagnosis is made?

- *If RWR is mistaken for vestib.-cochlear DCS and the patient is placed in the recompression chamber for treatment, the equalization would force the endolymph through the fistula from the inner ear into the middle ear >>> worsening of the case and permanent damage to the balance could occur*
- *RWR is one of the contraindications for recompression therapy!!*

⇨ *analyzing dive profile and gases used showed dive was within safety parameters and the gas used was EAN32 {Heliox,Trimix}*

⇨ *Time of Onset of 1st Symptom:*

- *vertigo under the water in the last 3 meters then vomiting immediately upon surfacing >>> RWR*
- *If 30 minutes after surfacing >>>vestib.- cochlear DCS*
- *RWR is usually not accompanied by decreased hearing!!i*
- *Difficulty in equalization on the descent >>> barotrauma of inner ear*

- *If it was a vestib.-cochlear DCS and was diagnosed as RWR, the pt. would then be denied the hyperbaric Oxygen treatment needed and the case would worsen!*

Other points to consider:

- *If it was the 3rd dive of the day?*
- *Nitrogen load?*
- *If difficulties were encountered in equalization in all 3 dives?*

Do not rush in taking decisions, buy time with normobaric Oxygen and consult!

- Sometimes, the ***elder divers*** taking psychotropic medication and / or sleeping pills, ***forget*** that they have taken the prescribed dose and repeat it several times...***overdosing!***

The differential diagnosis of facial neurological deficit after diving includes ***decompression sickness, cerebral air embolism due to pulmonary barotrauma, facial nerve barotrauma and common conditions such as stroke and Bell's palsy.***

Undersea and Hyperb Med 2014 Sep-Oct;41(5):407-9.
Facial baroparesis: a critical differential diagnosis for scuba diving accidents—case report.
Iakovlev EV, Iakovlev VV

Other DD...

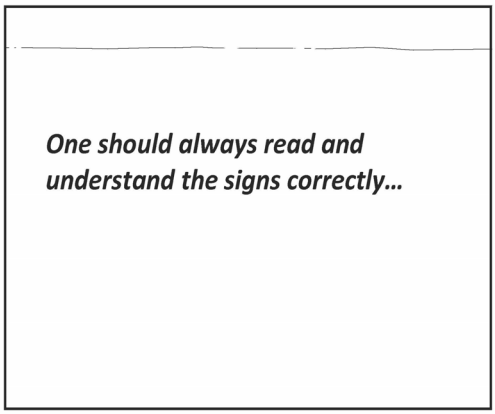
- **Retinal artery, retinal vein occlusions**
- **Retinal detachment**
- **diving induced conditions/maladies:**
  - Sinus barotraumas (easily confused with pneumo-encephalus or CAGE)
  - Pulmonary edema following saltwater aspiration
  - Vertigo (inner ear)

Physicians in charge in ***remote diving destinations*** are very often ***not trained diving medical specialists*** and either end up taking premature decisions and committing any diving related injury to a recompression therapy and possible evacuation, or sometimes panic

Consultation with more experienced specialists and consultants should be secured 24/7
(e.g. Hotline of DAN-Europe)

Facial Baroparesis:
A diver, after surfacing from a shallow dive, developed isolated left-sided facial palsy accompanied by pain and decreased hearing in the left ear. Forty minutes later, he heard a "pop" in the affected ear, after which all symptoms quickly resolved. Repeat neurological and ear examinations were normal. He showed no residual or new symptoms 24 hours later.

Divers committed to a recompression therapy that are not showing any improvement, are a good reason to reconsider the diagnosis!


One should always read and
understand the signs correctly…

Chapter 4 Selection of breathing gases for diving, deep tunnelling and decompression

Jean-Claude Le Péchon

Why not use compressed air in diving?

Compressed is the simplest and easiest way to dive, however there are also several reasons for not using compressed air in diving. The limitation of air breathing while under water pertains to various physical and chemical properties of pressurized gases and breathing condition:

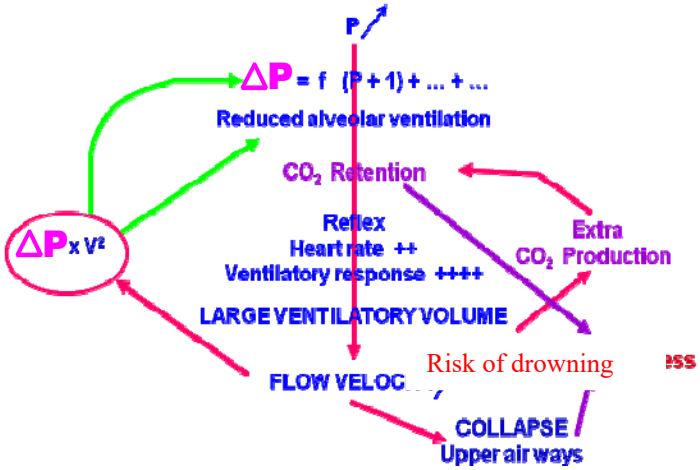
- Changes in volume due to Boyle-Mariotte's law,
- Effects of gases partial pressure on their toxicity
- The consequences of inert gas loading of tissues and decompression issues…,

Physics

Boyle-Mariotte and gas density

Gas compression reduces the volume corresponding to a given mass of gas. This may cause barotrauma. However is not related to the chemical composition of the gas breathed. We are not concerned here.

Compression of gases increases the mass of gas in a given volume which results in an increased gas density, proportional to absolute pressure. The gas density is a significant factor in breathing resistance in the airways as well as in the equipment supplying the breathing gas (regulator or rebreather …).



In the airways the alveolar ventilation is impaired by gas density according to the square root of gas density for the same flow rate. With shallow depths this does not affect the physiology, but for dives deeper than 40 m this finally results in hypercapnia. This can be coped with when there is a low level of exercise. The situation is different when diving with open circuit (no contaminated inspired gas) and with rebreather diving when inspired gas may become contaminated. With a high level of exercise ventilation is strongly increased to prevent the blood carbon dioxide content to exceed a critical level. Now, the extra effort of breathing produces more carbon dioxide; this becomes a vicious circle aggravated by the fact that with high flows also the middle sized airways become turbulent, increasing the breathing resistance even more. Breathlessness may result. *The only way to cope with that evolving condition is to abort the dive and start a prudent ascent trying to breathe deeply and slowly.*

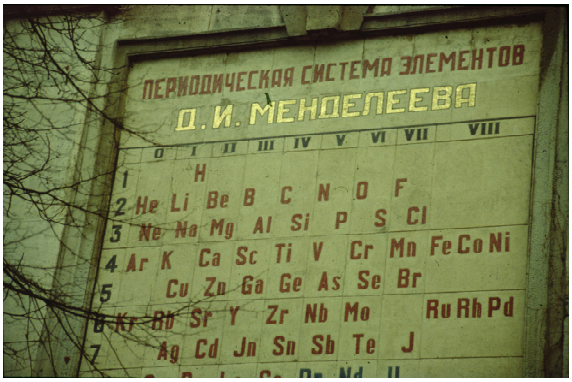
Diving on air beyond 50 m is not recommended. For deeper excursion, a mix gas containing a proper amount of helium is required.
Helium is 7 times less dense than nitrogen and this solves the problem of gas density at least up to about 400 meters.

Dalton and partial pressures

In a gas mixture at a pressure P a component partial pressure is the product of the centesimal concentration of the gas multiplied by the absolute pressure.
All physiological effects of a gas in the body are directed by alveolar partial pressure which is close from atmosphere partial pressure of the gas.
Selecting a suitable gas for a dive requires that all potentially beneficial or toxic effects of each component are optimised according mainly to the depth and duration of the dive.

$$\frac{PO_2}{P} = [O_2]$$

Chemistry Mendeleiv



There are many candidate gases for inclusions in breathing mixtures. Of course there must be oxygen; however the inert gas can be selected among gases from the Mendeleiv classification. Several of them have been tested and proved to be unacceptable for various reasons (narcosis, density, safety, availability and cost …). This is the case of neon, argon, krypton and in part hydrogen. The only efficient ones are nitrogen and helium!

Oxygen

High partial pressure of oxygen is both beneficial and toxic in diving.
In a breathing mixture, the more oxygen, the less inert gas; inert gas being responsible for the decompression requirements, it is beneficial to increase oxygen partial pressure as much as permitted both during exposure and decompression.
On the over hand high partial pressure oxygen is producing adverse effects of cells; pulmonary for long term exposures at moderate values and on the brain for acute exposures to values above 1.6 bar, specifically when immersed. All breathing mixtures are hyperoxic. Hypoxic Trimix does not exist in diving.

Nitrogen narcosis

Nitrogen at partial pressure above 4 bars impairs synapse communication which induces progressive narcosis, enhanced by hypercapnia, stress and poor environmental condition.
Diving deeper than 50 meters breathing air involves a serious risk of miss behaviour eventually leading to complete loss of control… The maximum accepted partial pressure of nitrogen in most of diving regulation is 4.8 bars (air at 50 m).
A reasonable value for Trimix is in the range of 3 to 4 bars of nitrogen.

Helium

- Helium is not narcotic; it is rather an excitant of central nervous system. This effect appears in the range of PHe > 20 bars, depending of compression rate to reach that pressure; it is called High Pressure Nervous Syndrome (HPNS).
- Helium is a very efficient heat transfer agent; consequently helium atmosphere must be maintained at relatively high temperature (30 to 32 °C) depending of total pressure, and breathing gases must be heated for long duration dives, in particular deeper than about 80 m.

Decompression

Oxygen

Safe decompression requires as much oxygen as permitted by oxygen toxicity.
This advantage of oxygen is due to the so called “Oxygen Window” or inherent under-saturation, caused by oxygen destruction by tissue metabolism, replaced by the equivalent quantity of carbon dioxide, which is extremely soluble and chemically bond in plasma which results in very low tension of dissolved carbon dioxide.
When choosing a mix the more oxygen the less inert gas, the less decompression time in shallow water diving this the principle of using enriched air called Nitrox.

When decompression stops become long, they should be performed on oxygen breathing which reduces stops duration by about 1/3, and improves decompression quality in all cases.

Helium versus nitrogen

Elimination of nitrogen loaded up during the air or Nitrox dives requires decompression time, reducing the nitrogen content to the advantage of oxygen, thanks to the so called Oxygen Window, reduces the decompression penalty.
High diffusion rate of helium fasten the helium exchanges into the body. Uptake and elimination are faster than for nitrogen. The classical value based on diffusion properties is a ratio of 2.6 (He/N_2) used for half time of equivalent compartments in calculation of gas uptake. Consequence short dives require more decompression time than with nitrogen, and saturation exposures require about half as long as similar exposures in Nitrox saturation at the same pressure with same PO_2 values.
In Trimix for excursion exposures adding helium may reduce total decompression time when compared to air exposures at the same pressure for the same duration (Sterk procedures).

Practical choices of gases

Moderate depths

- Air is of course the most used breathing gas for commercial and recreational dives down to 50 m.
- Nitrox has several applications :
  - ✓ Improve safety of decompression breathing Nitrox and decompressing on air tables,
  - ✓ Increase working time using air equivalent depth to enter tables or computers,
  - ✓ For altitude diving it allows longer working time and help compensating altitude penalty,
  - ✓ In rebreathers supplied with air and pure oxygen, Nitrox is common practice.
  - ✓ When long daily Nitrox dives are performed, oxygen toxicity requires to be monitored using OTU and keeping daily dose below 400 OTU.

Rebreathers which gases?

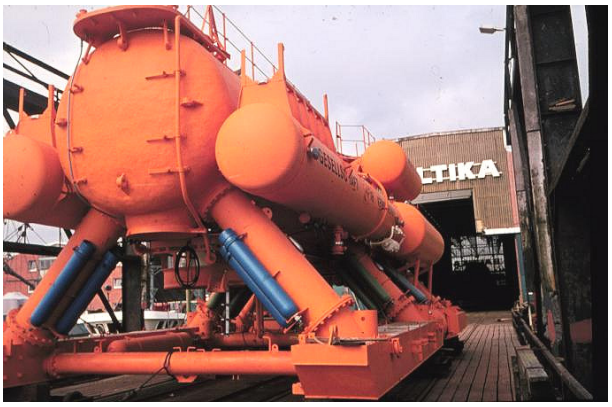
- Closed circuit units

Oxygen supplied from pure oxygen, volume compensation gas can be either air, Nitrox, Heliox or Trimix according the type of dive. The most important is that this gas be breathable at maximum depth of the dive and should not produce PO_2 higher than 2 bars at that depth.

- Semi-closed circuits units

Oxygen is supplied with an over oxygenated mix oxygen concentration being selected according to the type of injection system and the depth of he dive.
The inert gas can be Air, Nitrox, Heliox or Trimix. In some systems there are even 2 mixes when the depth range is large.

Underwater habitats



There are two types of sub-sea habitats: Shallow water (less than 15 m) and deeper ones more than 15m.

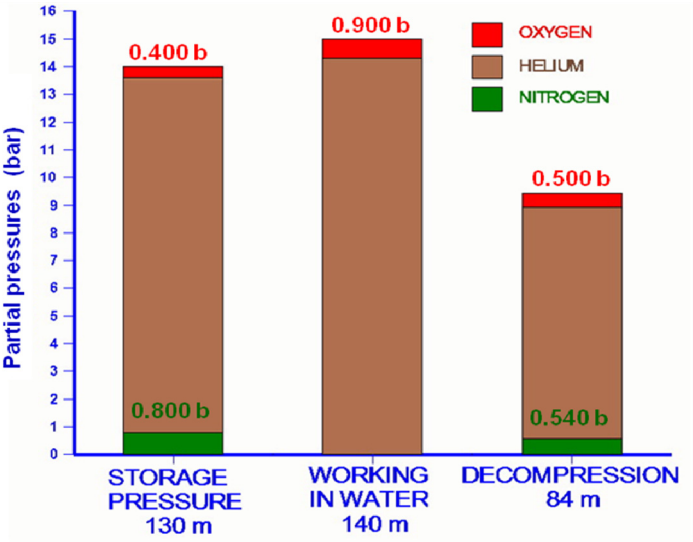
Shallow water can use compressed air to ventilate and keep the atmosphere clean because PO_2 is at most close to 0.5 bar and can be breathed continuously (Diogène 1962, Conshelf 2 (1963), Tectite, Aquarius (2018).
Decompression can be performed in water after some denitrogenation on pure oxygen or high PO_2 mix breathed on masks in the habitat and pure oxygen in-water stops.
Deep habitats need an internal regeneration system like a large rebreather. Atmosphere may be Nitrox, Trimix Conshelf 2 – 25 m (1963) or Heliox depending of the depth. Decompression must be made with a TUP system or ascent of the pressurized habitat and decompression in surface (Conshelf III experiment 100 m 1965!)
The German habitat Helgoland had a Nitrox atmosphere, with PO_2 at 0.21 bar and and immersion depth from 9 m to a maximum of 33 m according to the missions between 1971 and 1980.

Deep dives and deep compressed air work

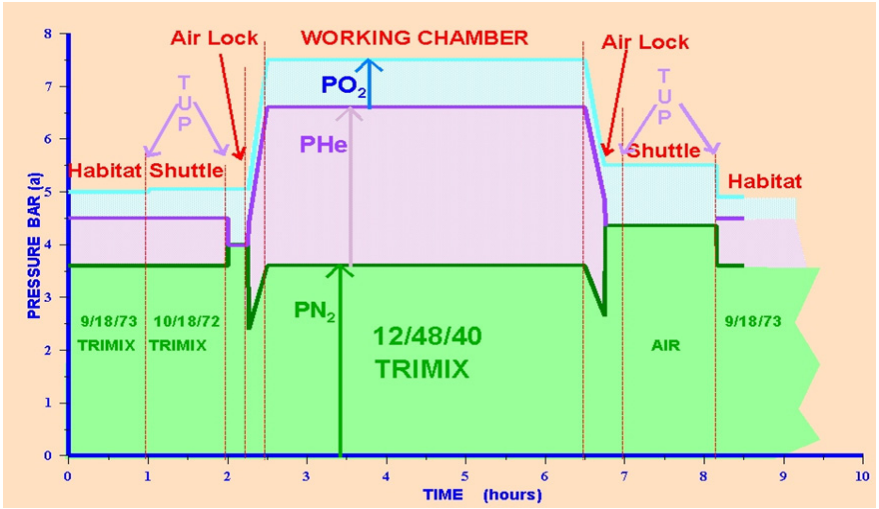
- Bounce dives

✓ For very long time only Heliox mixtures have been used, following the US Navy diving manual
✓ Some diving companies (CG Doris, IUC and in the Netherland) and the French Navy, successfully dove with Trimix since the early 70's.
✓ More recently "technical divers" discovered the advantages of Trimix and nowadays most of deep recreational dives, open circuit or rebreathers, are performed with Trimix. Extreme bounce dives on rebreathers are performed with minimum nitrogen in the mix. The actual limit of such exercise is in the range of 300 m, with many casualties… HPNS, cold, decompression sickness in particular inner ear cases…

- Saturation dives Heliox, Trimix and tunnelling



✓ Commercial dives in the offshore industry are always performed with Heliox mixes even when in the chambers there is 0.8 bar of nitrogen left! Mix delivered to the working diver does not contain nitrogen.
✓ Tunnelling at pressure in the range of 3.5 to 6 bars may require bounce exposures, most of times they are carried out on Trimix : Westerschelde – Sterk-Seattle JCLP.
✓ In some tunnelling operations at moderate pressure (3.5 to 7 bars) and when saturation is needed, it may be practical to keep a constant partial pressure of nitrogen during transfers and breathe air in the shuttle. Then the saturation mix should be Trimix. At higher pressures (ex: Bosphorus - 2016- 10 bars) Heliox has been used all the way.



Very deep experiments

- The early experiments (1970's) have evaluated Heliox and discovered HPNS (400-600 m). Serious improvements have been gained by controlling compression procedures.
- The idea of compensation of HPNS in adding significant level of the "narcotic" Nitrogen, had been successful (Janus IV – 1977 – 501 m)… Although this compensation effect has not been fully confirmed.
- Deeper than 400 m even Heliox has a density equivalent to air at 50m… To reduce the density and eliminate HPNS using hydrogen was a tempting possibility. Many experiments have been carried out in particular at Comex. It demonstrated the huge advantage on breathing comfort; however hydrogen is psychotropic above about 25 to 30 bars PH_2. A very deep trial in Comex hyperbaric centre had one diver compressed to 70 bars on Trimix (P_2 0.5 bars, PHe 35.5 bars, PH_2 35 bars). The Hydra X open sea dive on board DSV Aurelia, demonstrated a very efficient underwater working capacity of the six divers as deep as 534 m with a similar Trimix. Practical application of hydrogen diving is not needed any more by the industry and the risks associated with explosive properties of hydrogen ruled out this gas.

Aurelia - Hydra VIII support vessel

Conclusion

Gas management in diving is easy with air diving or compressed air work.
When it becomes necessary to change the breathing media, it is only a matter of selecting the partial pressures of the various gases that must be adjusted to optimise the procedure taking into account:

- ✓ the various phases of the operation,
- ✓ the maximum pressure,
- ✓ the duration of exposure,
- ✓ the condition of decompression,
- ✓ the overall environment and the available equipment,
- ✓ Incidentally, for recreational divers, the cost of the gases!

19-2-2018

SELECTING BREATHING GASES

Jean-Claude LE PÉCHON

http://jclp-hyperbarie.fr

Selecting breathing gases (continued…)

AGENDA

1 – WHY NOT USE COMPRESSED AIR

2 – PHYSICS

3 – CHEMISTRY

4 – PRACTICAL CHOICES

5 – CONCLUSIONS

Selecting breathing gases (cont'ed…)

1 – Why not use compressed air ?

Compressed air is the easiest breathing gas available for diving or hyperbaric breathing.

However due to increased pressure Air may not be the best adapted gas in many diving or hyperbaric configurations

Those possible limitations may come form

- volume changes (Boyle-Mariotte's law) and gases density
- chemical properties of gases Partial Pressures and Dalton's law
- consequences of tissue gas loading (Henry's law)

Selecting breathing gases (cont'ed…)

1 – Why not use compressed air ?

Absolute pressure = Relative pressure + Local atmospheric pressure

Notation :

Bar is gauge pressure = 1000 hPa relative pressure

$Bar_{(a)}$ is absolute pressure = 1000 hPa + Local $pressure_{(a)}$

Depth of Immersion is measured in meters – 10 msw is = 1 bar (fresh water nearly 1 bar)

Selecting breathing gases (cont'ed…)

2 – Physics

BOYLE-MARIOTTE

VOLUME CHANGES → GAS DENSITY

BREATHING AT PRESSURE → VOLUME CHANGES

2 – Physics (cont'ed..)

2.1 – Boyle-Mariotte and gas density

Density (or specific gravity) is the mass of fluid per unit volume

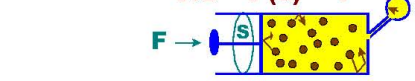
Example : Air density (STPD) is : 1.29 g/dm^3

For "density" we shall use the Greek letter «ρ»

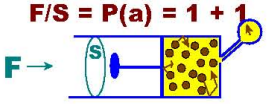
19-2-2018

2.1.1 – Gas density and pressure

F/S = P(a) = 1 + 0



F/S = P(a) = 1 + 1



ρ(P) = ρ(STPD) x P

When absolute pressure is multiplied by 2,
Volume is devided by 2 and
Density is multiplied by 2.

2.1.2 – Viscosity and pressure

VISCOSITY is a measure of flowing capability of a fluid

VISCOSITY is not a direct function of Pressure

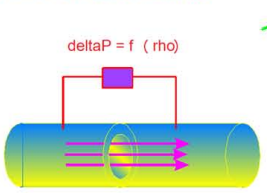
VISCOSITY is function of :

the type of gas (Higher for He than for O_2 or N_2),

and temperature.

2.1.3 – Flow resistance and pressure

Pressure drop (ΔP) alongside a tube for a constant velocity depends on fluid density (ρ)



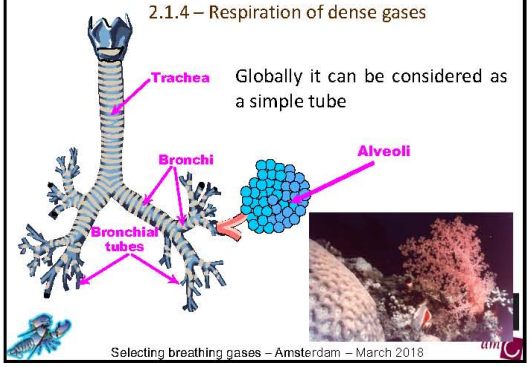
Pressure drop (**ΔP**) depends on the flow velocity (V)

Laminar flow : (slow) : $\Delta P = k_1 \times V$

Turbulent flow (fast) : $\Delta P = k_1 \times V + k_2 \times V^2$

Squared flow rate ! ! ! ! !

2.1.4 – Respiration of dense gases



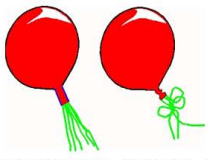
Globally it can be considered as a simple tube

However !!!

Maximum expiratory flow is a function of gas density :

Maximum expiratory flow = A x 1 / $\sqrt{\rho}$

The tube modelling the respiratory track should be very flexible to accept collapse at higher flows



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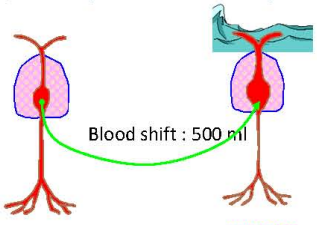
Breathing in immersion

Available pulmonary volume is reduced by blood shift



Pulmonary compliance is increased

Breathing movements require more energy in water than in air.

Depending on posture, intra pulmonary pressure is not balanced with external pressure:

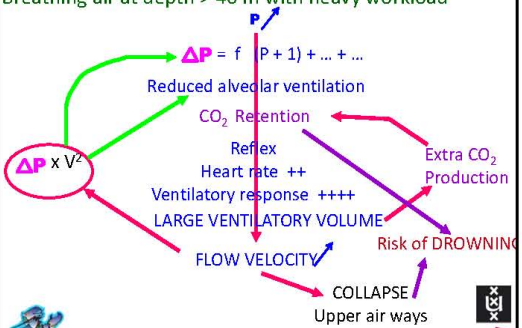
20cm of water when standing up in immersion to the neck

These small physiological factors, they increase the work of breathing

In addition, one needs SCUBA. The regulator breathing resistance increases with depth.
Also the diving suit increase work of breathing, be it independent on depth.

Those technical resistance adds to the overall work of breathing and to the effects of high density gases.
Under specific conditions this may result in a fatality.

Breathing air at depth > 40 m with heavy workload



What about oxygen ?

At depth, alveolar PO_2 is always hyperoxic

Increased ventilation tries only to keep $PACO_2$ normal

There is never hypoxia in diving breathlessness !!!

It is very different from asthma

PO_2 being controlled for other reasons,
Oxygen density is never a significant problem

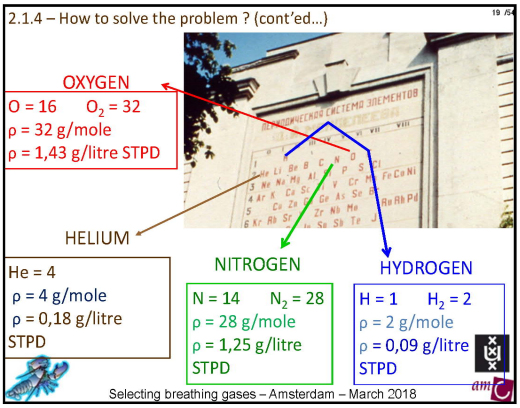
2.1.4 – How to solve the problem ?

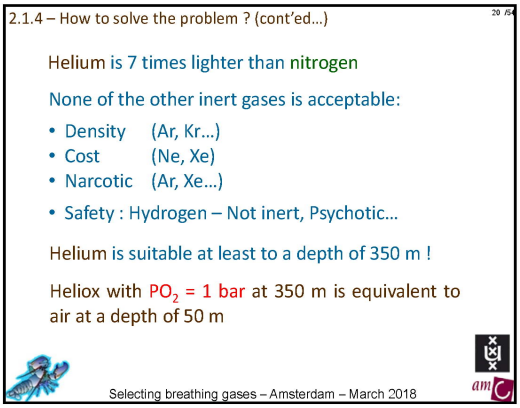
Nitrogen is responsible for most of the density effect

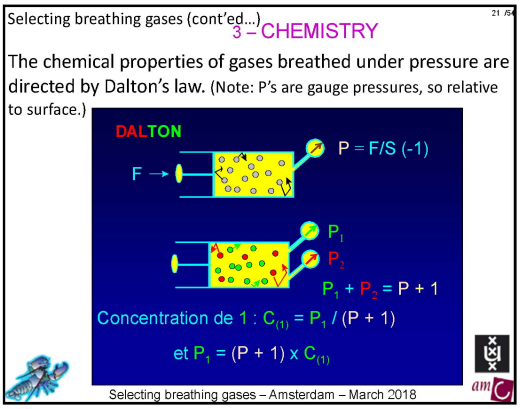
Nitrogen is necessary as diluent of oxygen

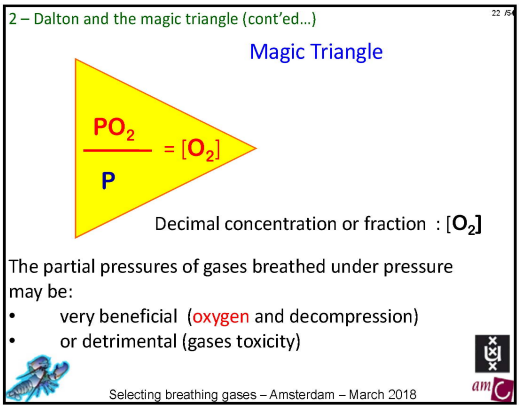
Nitrogen can be replaced by an other lighter inert gas

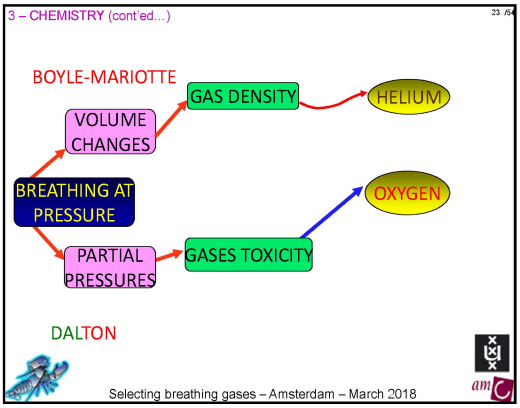
19-2-2018

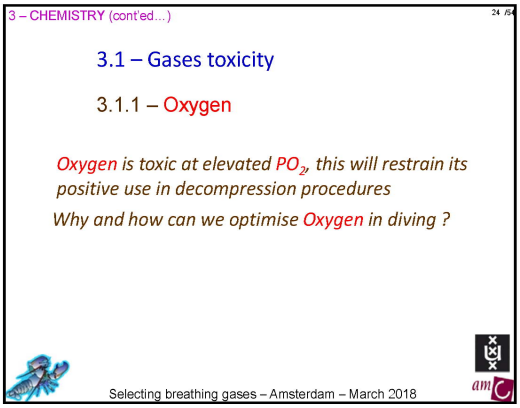

2.1.4 – How to solve the problem ? (cont'ed...)
19 /54
OXYGEN
O = 16 O_2 = 32
ρ = 32 g/mole
ρ = 1,43 g/litre STPD
HELIUM
He = 4
ρ = 4 g/mole
ρ = 0,18 g/litre
STPD
NITROGEN
N = 14 N_2 = 28
ρ = 28 g/mole
ρ = 1,25 g/litre
STPD
HYDROGEN
H = 1 H_2 = 2
ρ = 2 g/mole
ρ = 0,09 g/litre
STPD
Selecting breathing gases – Amsterdam – March 2018


2.1.4 – How to solve the problem ? (cont'ed...)
20 /54
Helium is 7 times lighter than nitrogen
None of the other inert gases is acceptable:
• Density (Ar, Kr...)
• Cost (Ne, Xe)
• Narcotic (Ar, Xe...)
• Safety : Hydrogen – Not inert, Psychotic...
Helium is suitable at least to a depth of 350 m !
Heliox with PO_2 = 1 bar at 350 m is equivalent to air at a depth of 50 m
Selecting breathing gases – Amsterdam – March 2018


Selecting breathing gases (cont'ed...)
21 /54
3 – CHEMISTRY
The chemical properties of gases breathed under pressure are directed by Dalton's law. (Note: P's are gauge pressures, so relative to surface.)
DALTON
F
P = F/S (-1)
P_1
P_2
$P_1 + P_2 = P + 1$
Concentration de 1 : $C_{(1)} = P_1 / (P + 1)$
et $P_1 = (P + 1) \times C_{(1)}$
Selecting breathing gases – Amsterdam – March 2018


2 – Dalton and the magic triangle (cont'ed...)
22 /54
Magic Triangle
$\frac{PO_2}{P} = [O_2]$
Decimal concentration or fraction : $[O_2]$
The partial pressures of gases breathed under pressure may be:
• very beneficial (oxygen and decompression)
• or detrimental (gases toxicity)
Selecting breathing gases – Amsterdam – March 2018


3 – CHEMISTRY (cont'ed...)
23 /54
BOYLE-MARIOTTE
VOLUME CHANGES
GAS DENSITY
HELIUM
BREATHING AT PRESSURE
PARTIAL PRESSURES
GASES TOXICITY
OXYGEN
DALTON
Selecting breathing gases – Amsterdam – March 2018


3 – CHEMISTRY (cont'ed...)
24 /54
3.1 – Gases toxicity
3.1.1 – Oxygen
Oxygen is toxic at elevated PO_2, this will restrain its positive use in decompression procedures
Why and how can we optimise Oxygen in diving ?
Selecting breathing gases – Amsterdam – March 2018

19-2-2018

Lorrain-Smith Effect (1899)

Oxygen Pneumonia

SYMPTOMS

Progressive pulmonary pain
Dry coughing
Dyspnoea
Labour respiration
Pulmonary oedema.....

Clark & Lambertsen (1972)

Measure : Vital capacity reduction

They created a Pulmonary Toxic Dose (UPTD) to quantify pulmonary damages

1 UPTD corresponds to the potential pulmonary damage due to the breathing of pure O_2 with PO_2 = 1 bar, for 1 minute

Number of UPTD = Kp x Duration of exposure at constant PO_2

Kp can be calculated, however there is a table.

Table : Kp values as a function of PO_2

PO_2	0,5	0,6	0,7	0,8	0,9	1	1,1	1,2	1,3	1,4	1,5	1,6
K_p	0	0,26	0,47	0,65	0,83	1	1,16	1,32	1,48	1,63	1,78	1,93

PO_2	1,7	1,8	1,9	2	2,1	2,2	2,3	2,4	2,5	2,6	2,7	2,8
K_p	2,07	2,22	2,36	2,5	2,64	2,77	2,91	3,04	3,17	3,31	3,44	3,57

Generally accepted limits :
reduction of CV : about 2 %
for 50% of the population exposed

Original (1971) : 615 UPTD for each exposure
600 UPTD per day
1425 UPTD for a medical treatment (-10 %)

Presently (STERK 1986): 400 OTU per day during 1 week

OTU are the same as UPTD, but address other effects of oxygen than pulmonary only

These units, originally created only for pulmonary effects, may be used as an index for long oxygen exposures at moderate PO_2

« LORRAIN-SMITH : No risk in immersion diving »

Signed

Kenneth DONALD (1992)

Paul Bert effect

Since 1878, then Kenneth Donald (1942 & 1992)

SYMPTOMS ARE DIVERSE

10 % Hallucinations, cognitive problems
40 % Lips tingling,
40 % Vertigo, nausea, tunnel vision then
10 % Convulsions : while immersed : fatal danger

PREDISPOSING FACTORS

Individual Susceptibility
Hypercapnia Physical work, CO_2 ++++
Immersion Cold and warm
Immersion Lack of perception of signs

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Paul Bert effect (cont'ed...) 31 /54

The SCN clock !

NOAA has produced rules for the maximum duration of exposures to high PO_2

Example :

For 1.4 bar max 150 min
For 1.6 bar max 45 min

Many dive computers have used those limits to elaborate a SNC toxicity counting system

30 min at 1.6 bar is accounted for 66 %
75 min at 1.4 bar is accounted for 50 %

To evaluate the level of intoxication you add up the % calculated for each PO_2 exposure and find an index...

The SCN counter (cont'ed...) 32 /54

This the most stupid thing I ever saw in diving physiology :

1 – The selected limit values are only a decision of NOAA.

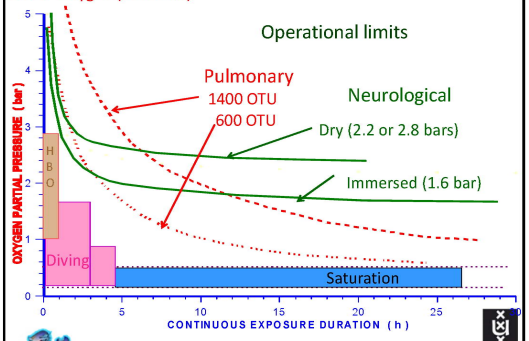
This is totally arbitrary, French regulation limits 1.6 bar to 3 h !!!!

2 – SNC intoxication is NOT linear neither proportional to the duration of exposure

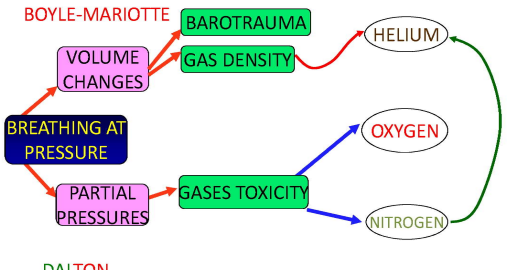
3 – The effects of a given PO_2 level on the brain are extremely different for 1.4 or 1.6 b, and those cannot be added.

This SNC clock is of NO value and cannot predict any risk of convulsion. When it is incorporated in a dive computer you should question the manufacturer's understanding in diving physiology ...

3.1.1 – Oxygen (cont'ed...) 33 /54



3 – CHEMISTRY (cont'ed...) 34 /54



3.1 – Gases toxicity (cont'ed...) 35 /54

3.1.2 – Nitrogen Narcosis

Nitrogen narcosis directly due to PN_2 (> 4 bars)

Hypercapnia is an aggravating factor

Signs are increasing with PN_2 :
- Reduced cognitive performance,
- Reduced immediate memory,
- Double task very hard to achieve
- However habituation is possible by training

3.1 – Gases toxicity (cont'ed...) 36 /54

3.1.3 – High Pressure Nervous Syndrome - HPNS

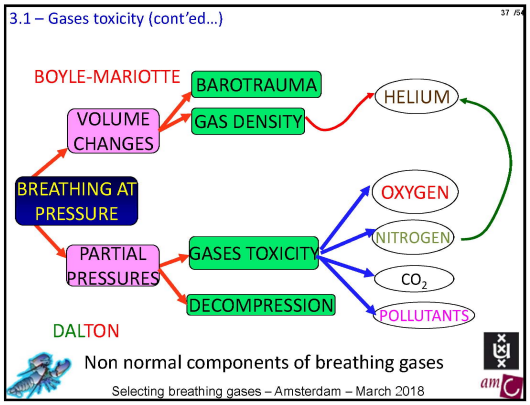
HPNS is due to very high PHe and rate of compression

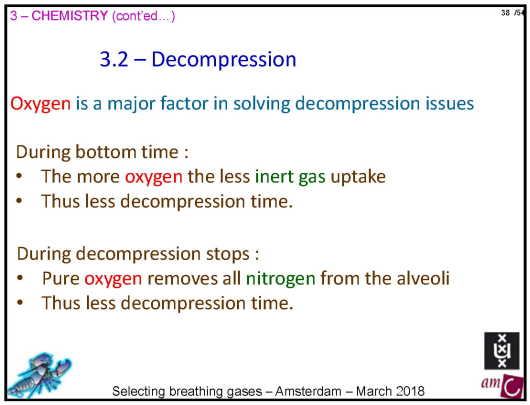
After very fast compression it starts in the range of 180 m

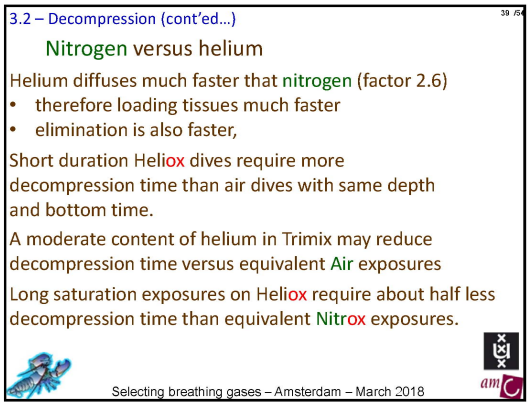
The main signs are
- tremor,
- fever impression
- sound hallucination
- EEG Theta waves

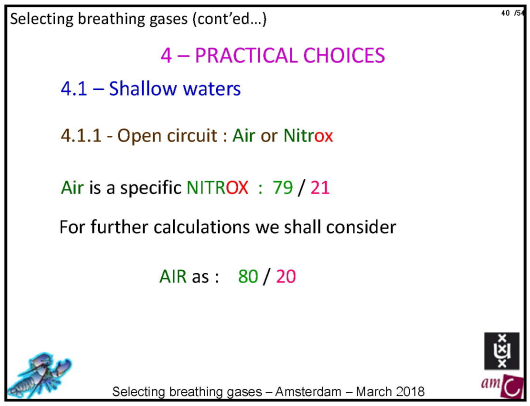
It is fully reversible upon decompression

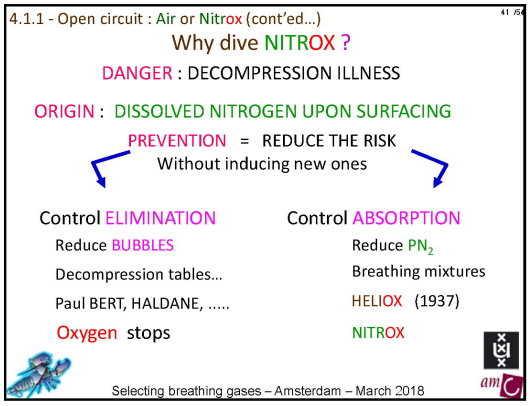
19-2-2018

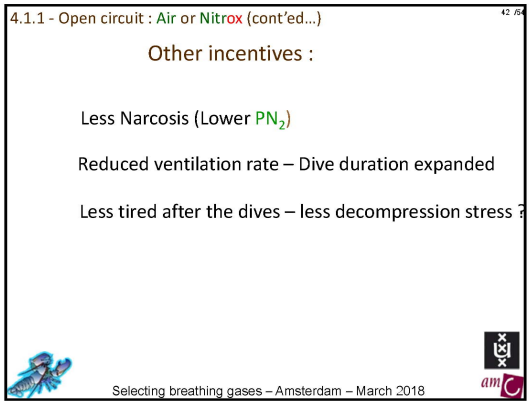

3.1 – Gases toxicity (cont'ed...)
37 /54
BOYLE-MARIOTTE
BAROTRAUMA
VOLUME CHANGES
GAS DENSITY
HELIUM
BREATHING AT PRESSURE
OXYGEN
NITROGEN
PARTIAL PRESSURES
GASES TOXICITY
CO_2
DECOMPRESSION
POLLUTANTS
DALTON
Non normal components of breathing gases
Selecting breathing gases – Amsterdam – March 2018


3 – CHEMISTRY (cont'ed...)
38 /54
3.2 – Decompression
Oxygen is a major factor in solving decompression issues
During bottom time :
• The more oxygen the less inert gas uptake
• Thus less decompression time.
During decompression stops :
• Pure oxygen removes all nitrogen from the alveoli
• Thus less decompression time.
Selecting breathing gases – Amsterdam – March 2018


3.2 – Decompression (cont'ed...)
39 /54
Nitrogen versus helium
Helium diffuses much faster that nitrogen (factor 2.6)
• therefore loading tissues much faster
• elimination is also faster,
Short duration Heliox dives require more decompression time than air dives with same depth and bottom time.
A moderate content of helium in Trimix may reduce decompression time versus equivalent Air exposures
Long saturation exposures on Heliox require about half less decompression time than equivalent Nitrox exposures.
Selecting breathing gases – Amsterdam – March 2018


Selecting breathing gases (cont'ed...)
40 /54
4 – PRACTICAL CHOICES
4.1 – Shallow waters
4.1.1 - Open circuit : Air or Nitrox
Air is a specific NITROX : 79 / 21
For further calculations we shall consider
AIR as : 80 / 20
Selecting breathing gases – Amsterdam – March 2018


4.1.1 - Open circuit : Air or Nitrox (cont'ed...)
41 /54
Why dive NITROX ?
DANGER : DECOMPRESSION ILLNESS
ORIGIN : DISSOLVED NITROGEN UPON SURFACING
PREVENTION = REDUCE THE RISK
Without inducing new ones
Control ELIMINATION
Reduce BUBBLES
Decompression tables...
Paul BERT, HALDANE, ...
Oxygen stops
Control ABSORPTION
Reduce PN_2
Breathing mixtures
HELIOX (1937)
NITROX
Selecting breathing gases – Amsterdam – March 2018


4.1.1 - Open circuit : Air or Nitrox (cont'ed...)
42 /54
Other incentives :
Less Narcosis (Lower PN_2)
Reduced ventilation rate – Dive duration expanded
Less tired after the dives – less decompression stress ?
Selecting breathing gases – Amsterdam – March 2018

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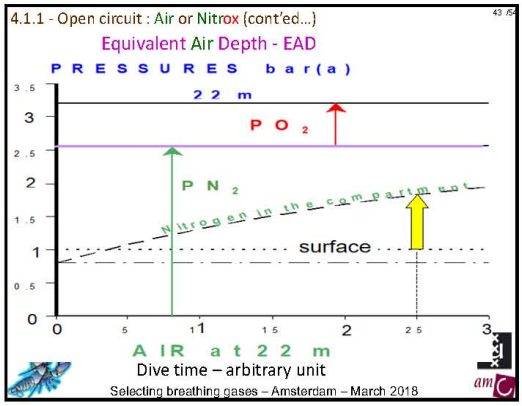
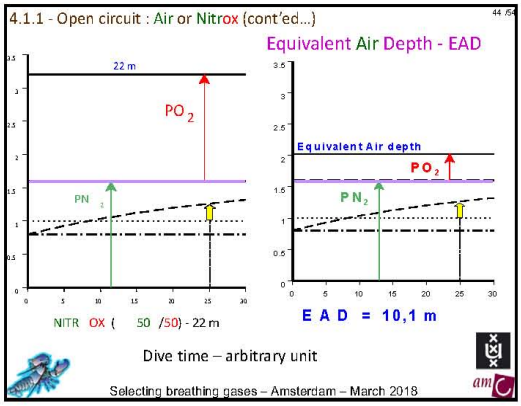
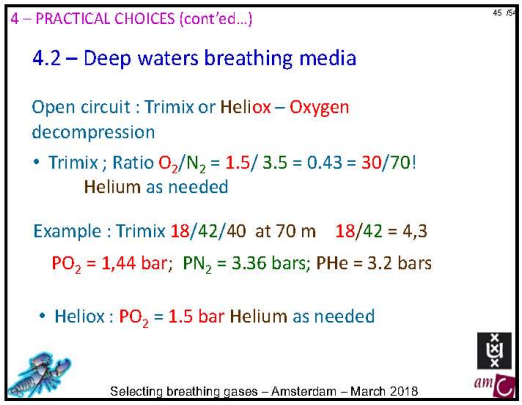
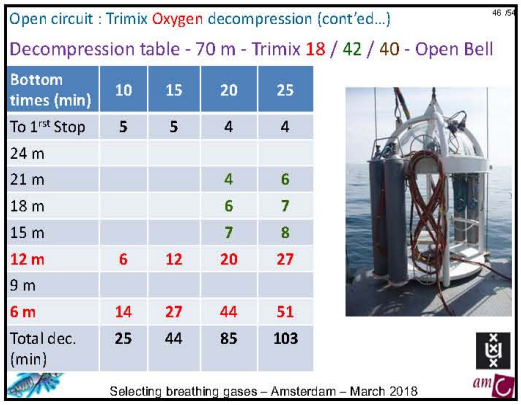
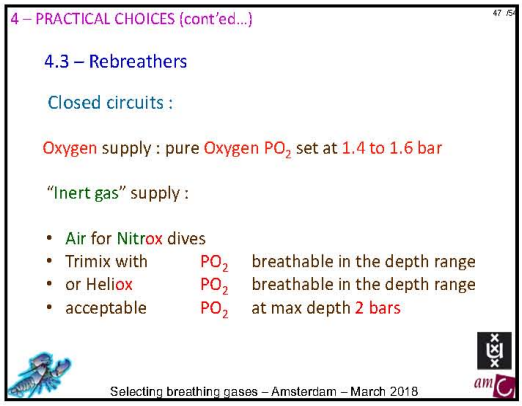
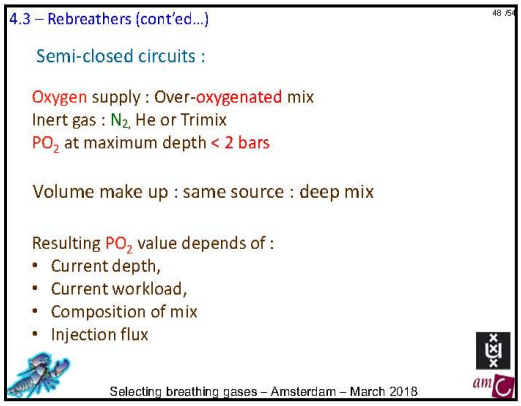

4.1.1 - Open circuit : Air or Nitrox (cont'ed...)
43 /54
Equivalent Air Depth - EAD
P R E S S U R E S b a r (a)
2 2 m
P O 2
P N 2
Nitrogen in the compartment
surface
A I R a t 2 2 m
Dive time – arbitrary unit
Selecting breathing gases – Amsterdam – March 2018


4.1.1 - Open circuit : Air or Nitrox (cont'ed...)
44 /54
Equivalent Air Depth - EAD
22 m
PO 2
PN 2
NITR OX (50 /50) - 22 m
Equivalent Air depth
PO2
PN2
E A D = 10,1 m
Dive time – arbitrary unit
Selecting breathing gases – Amsterdam – March 2018


4 – PRACTICAL CHOICES (cont'ed...)
45 /54
4.2 – Deep waters breathing media
Open circuit : Trimix or Heliox – Oxygen decompression
• Trimix ; Ratio O2/N2 = 1.5/ 3.5 = 0.43 = 30/70!
Helium as needed
Example : Trimix 18/42/40 at 70 m 18/42 = 4,3
PO2 = 1,44 bar; PN2 = 3.36 bars; PHe = 3.2 bars
• Heliox : PO2 = 1.5 bar Helium as needed
Selecting breathing gases – Amsterdam – March 2018


Open circuit : Trimix Oxygen decompression (cont'ed...)
46 /54
Decompression table - 70 m - Trimix 18 / 42 / 40 - Open Bell
Bottom times (min) 10 15 20 25
To 1rst Stop 5 5 4 4
24 m
21 m 4 6
18 m 6 7
15 m 7 8
12 m 6 12 20 27
9 m
6 m 14 27 44 51
Total dec. (min) 25 44 85 103
Selecting breathing gases – Amsterdam – March 2018


4 – PRACTICAL CHOICES (cont'ed...)
47 /54
4.3 – Rebreathers
Closed circuits :
Oxygen supply : pure Oxygen PO2 set at 1.4 to 1.6 bar
"Inert gas" supply :
• Air for Nitrox dives
• Trimix with PO2 breathable in the depth range
• or Heliox PO2 breathable in the depth range
• acceptable PO2 at max depth 2 bars
Selecting breathing gases – Amsterdam – March 2018


4.3 – Rebreathers (cont'ed...)
48 /54
Semi-closed circuits :
Oxygen supply : Over-oxygenated mix
Inert gas : N2, He or Trimix
PO2 at maximum depth < 2 bars
Volume make up : same source : deep mix
Resulting PO2 value depends of :
• Current depth,
• Current workload,
• Composition of mix
• Injection flux
Selecting breathing gases – Amsterdam – March 2018

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4 – PRACTICAL CHOICES (cont'ed...)

4.4 – Subsea habitats

Shallow water habitats:

Shallower than 15 m air can used in the habitat

PO_2 is 2.5 x 0.21 = 0.525 bar

Flushing air in the habitat is an easy way clear carbon dioxide

Diving outside the habitat is done breathing air

It has been the case for Diogène (Conshelf I), and Conshelf II

Also for Tectite

Selecting breathing gases –

4.4 – Subsea habitats (cont'ed...) 50 /54

Nitrox habitats

Reduced oxygen Nitrox is used down to ca. 30 m (PO_2 < 0.4 b)

It has been the case for Helgoland (PO_2 = 0.21 b), and it is the case for Aquarius (2018)

Selecting breathing gases – Amsterdam – March 2018

4.4 – Subsea habitats (cont'ed...) 51 /54

Deep Heliox habitat

The habitat has a closed circuit atmosphere regeneration system and works on constant PO_2

It requires to be pressure resistant to decompress in surface, or to be fitted with a diving bell system.....

Conshelf III and SeaLab 2 (1965)

Selecting breathing gases – Amsterdam – March 2018

4.4 – Subsea habitats (cont'ed...) 52 /54

4.5 – Commercial saturation dives

Control panels

Living chamber

Diving bell Pétrel Janus IV – 501 m

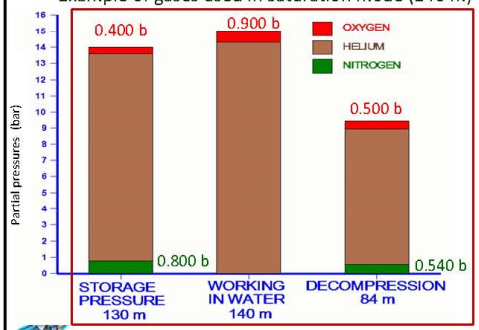
Diving support vessel

Inside the diving b

Selecting breathing gases – Amsterdam – March 2018

4.4 – Commercial saturation dives (cont'ed...) 53 /54

Example of gases used in saturation mode (140 m)



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4 – PRACTICAL CHOICES (cont'ed...) 54 /54

4.6 – Tunnelling

Up to 3.5 to 4 bars : Air

oxygen decompression for exposures above 1.5 bar

Above : Bounce intervention : Trimix as for diving

oxygen decompression

Saturation mode up to 6 to 6.5 bars Trimixes

Saturation mode above 6.5 to 7 bars, Heliox

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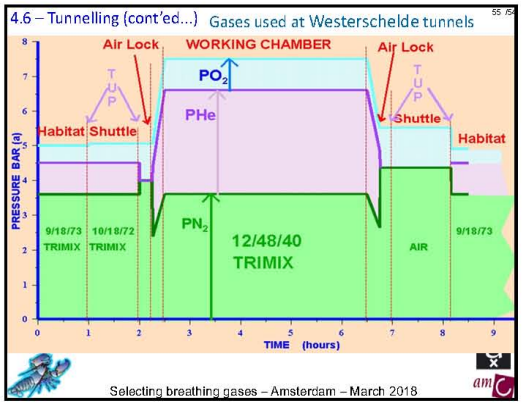
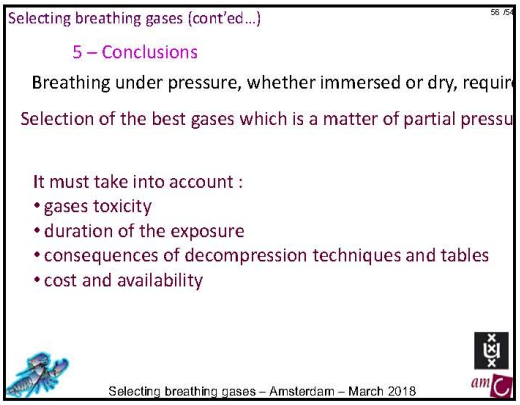
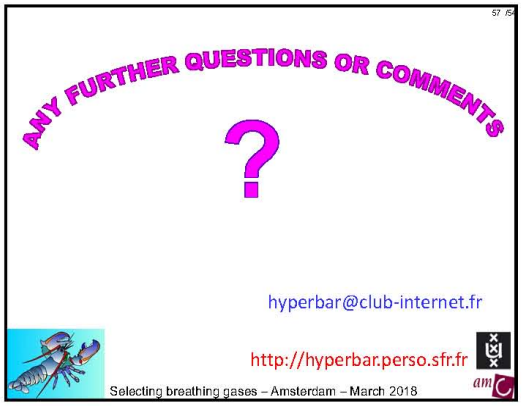

4.6 – Tunnelling (cont'ed...) Gases used at Westerschelde tunnels
55 /54
Air Lock
WORKING CHAMBER
Air Lock
PO2
PHe
PN2
Habitat
Shuttle
Shuttle
Habitat
9/18/73 TRIMIX
10/18/72 TRIMIX
12/48/40 TRIMIX
AIR
9/18/73
PRESSURE BAR (a)
TIME (hours)
Selecting breathing gases – Amsterdam – March 2018


Selecting breathing gases (cont'ed...)
56 /54
5 – Conclusions
Breathing under pressure, whether immersed or dry, requir
Selection of the best gases which is a matter of partial pressu
It must take into account :
• gases toxicity
• duration of the exposure
• consequences of decompression techniques and tables
• cost and availability
Selecting breathing gases – Amsterdam – March 2018


57 /54
ANY FURTHER QUESTIONS OR COMMENTS
?
hyperbar@club-internet.fr
http://hyperbar.perso.sfr.fr
Selecting breathing gases – Amsterdam – March 2018

Chapter 5 The endothelial response related to DCI with specific attention to the role of antioxidants

Sigrid Theunissen

Abstract

Diving involves constraints related to immersion, hyperbaria, hyperoxia, decompression, cold and physical exercise. These stressors can cause the appearance of circulating bubbles, platelet activation and / or the presence of microparticles leading to endothelial dysfunction and oxidative stress. All of these factors are potential risks for the development of decompression sickness (DCS). Indeed, higher amounts of detected bubbles post-dive has been correlated with a higher probability of DCS. Nevertheless, there are so-called "silent" bubbles that do not cause decompression sickness and many divers may have circulating bubbles after diving without developing DCS. The bullae phenomenon does not constitute all the pathology and it is important to turn to other factors that may be at the origin of these DCS. After saturation dives, it was observed that the number of platelets was decreased, and this was inversely correlated with the number of bubbles. Contact of bubbles with the endothelial wall also releases microparticles (MP) and a greater number of microparticles are observed in blood after the dive. These MP result in platelet aggregation, inflammation, and neutrophil activation. Bubble-induced MP can mediate endothelial dysfunction and it was even proposed that DCS would not be the consequence of the bubbles themselves but of endothelial dysfunction and microparticles associated with diving. Endothelial dysfunction is characterized by a decrease in the availability of nitric oxide (NO), a powerful vasodilator. It reacts with oxygen-free radicals, augmented by diving, to form peroxynitrites (ONOO-), an oxidative stress marker. The antioxidant status of the diver is an important mechanism in protecting against diseases. It is influenced by genetic factors but also by food. Among them, dark chocolate is a powerful antioxidant, as it includes a large amount of polyphenols. For this reason, 30g of dark chocolate was given to divers before diving to assess its effect on endothelial function and oxidative stress post-dive.

A preliminary study compared the effects of dark and white chocolate on the endothelial function of healthy subjects and assessed the dynamics of any changes. Dark chocolate was shown to increase flow-mediated dilation (% change in brachial artery diameter before and after 5 minutes of occlusion) 120 minutes after ingestion. White chocolate had no effect on endothelial function. We therefore administered 30g of dark chocolate 1h30 before a SCUBA dive (30min - 33m) to 21 experienced divers and compared them to a control group (n = 21) that did not take chocolate. The results of this study showed that chocolate is able to increase FMD and NO production after a dive while no variation of NO, combined with a reduced FMD, was observed in the control group. In previous studies, it was hypothesized that NO transforms into ONOO-, which is why it did not change. However, the rate of ONOO- decreased in the control group. Because oxidative stress was already shown after SCUBA diving, we concluded that ONOO- is not a good marker of oxidative stress in diving.
Conclusion: The reduction of FMD observed post SCUBA dive is shown to be linked to oxidative stress since it is prevented by dark chocolate ingestion prior to diving (antioxidant administration). Dark chocolate is therefore a good method to prevent post-dive endothelial dysfunction. The absence of NO variation in a control SCUBA dive suggests that FMD is the consequence of the autonomic nervous system and / or an alteration of vascular smooth muscle.

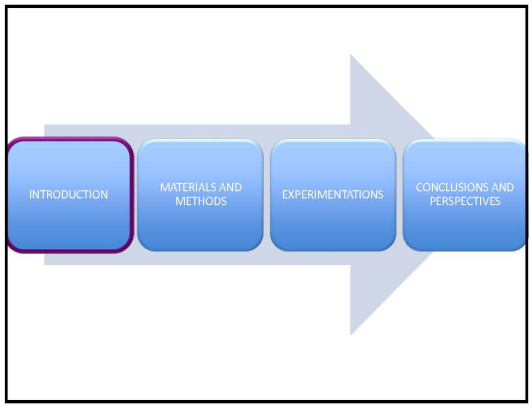
1-3-2018

23th Capita Selecta Duikgeneeskunde

THE ENDOTHELIAL RESPONSE RELATED TO DIVING WITH SPECIFIC ATTENTION TO THE ROLE OF ANTIOXIDANTS

Sigrid Theunissen

Amsterdam, March the 17th, 2018



Introduction

Diving

Contraints

Immersion, Hyperbaria, hyperoxia, hyperoxic gaz mixture, work of breathing, decompression, cold, physical exercise)

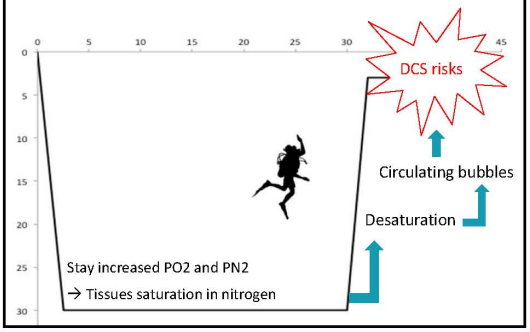
↓

Potential risk factors for DCS

(bubbles, platelets activation, microparticles, endothelial dysfonction, oxidative stress)

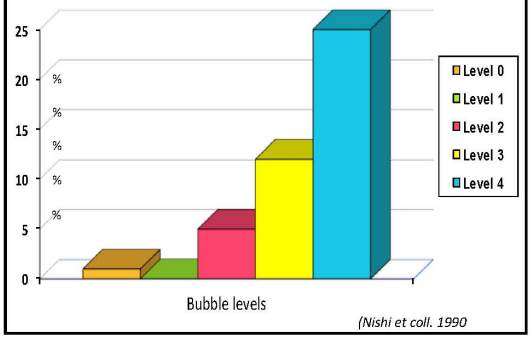
Introduction

Bubbles



Introduction

Bubbles levels and DCS Risks

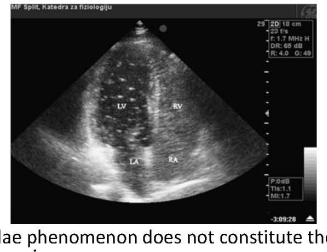


(Nishi et coll. 1990

Introduction

Bubbles

- Asymptomatic bubbles – « silent bubbles » *(Behnke,1951)*
- High bubble level without DCS *(Dujic, 2008)*



- The bullae phenomenon does not constitute the whole pathogenesis

1-3-2018

Introduction

Bubbles

The content
(Plasma + Blood Cells)

Platelets
→ Platelets Activation

Introduction

Platelets activation

- Interaction bubbles – platelets (Philip, 1974)
  - Microscopy : activated platelets on bubbles surface in pulmonary microcirculation
  - Leukocyte aggregation → leuko-platelets aggregates (inflammation)
- ↘ platelets in man (asymptomatic) after saturation dives *(Domoto 1990; Olsanski 1997)*
- ↘ platelets in correlation with ↗ bubbles *(Pontier 2008)*

Introduction

Endothelial Microparticles

- Bubble contact with endothelial cells releases endothelial micro-particles (MP) (Yu et al., 2017)
- These MP are tiny vesicles (0.1–1.0 µm) derived from debris of cell membrane from leukocytes, erythrocytes, platelets, and endothelial cells.
- After diving, there is an increase in circulating microparticles.

Introduction

Endothelial Microparticles

- Bubble-induced MP
  - ↘ cell viabiliy
  - ↗ cell apoptosis
  - ↗ cell permeability
  - ↗ over-expression of pro-inflammatory cytokines
- MP cause platelet aggregation, inflammation, and neutrophil activation (Thom, 2012)

Introduction

Endothelial Microparticles

- Some of the enlarged MP contain gas
  → inverse correlation between bubbles and MP
- Correlation between MP and decompression sickness (Thom et al., 2015)
- Bubble-induced MP can promote endothelial dysfunction

→ Endothelial dysfunction and MP, not gas bubbles, may be the underlying cause of DCI
(Madden and Laden, 2009)

Introduction

Endothelial dysfunction

The container
(Vascular wall)

The content
(Plasma + Blood cells)

Endothelium
→ Endothelial Dysfonction

Platelets
→ Platelets Activation

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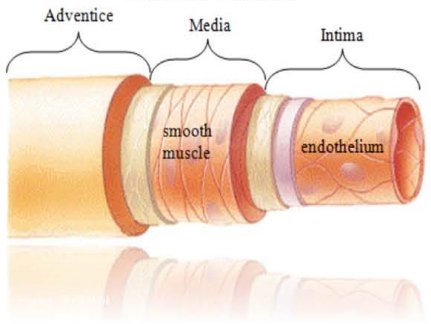
Introduction
Endothelial dysfunction



Introduction
Endothelial dysfunction

- Functions : Balance between

Pro-atherogenic Actions	Anti-atherogenic Actions
• Vasoconstriction	• Vasodilation
• Inflammation	• Anti-inflammation
• Agregation	• Anti-aggregation
• Oxydation	• Anti-oxidation

- Impairement of endothelial function = endothelial dysfunction

Introduction
Endothelial dysfunction

- Endothelial dysfunction = risk of cardio-vasc. disease
- Shown in a large number of cardio-vascular diseases
- Associated to
  - Age
  - Oxidative stress
- Characterized by a ↘ in NO availability

Nitric Oxide

- L-arginine $\xrightarrow{eNOS}$ L-citrulline + NO
- Co-factors : HSP90, NADPH, **BH4**, FAD, FMN
- VASODILATOR
- Dose-dependant effects :
  - Less NO ↘ ROS → antioxidant
    $NO^{\circ} + O_2^{-\circ} \longrightarrow ONOO^-$ ($^{\circ}$ means 'exited')
  - A lot of NO ↗ $ONOO^-$ → oxidative stress

Introduction
Oxidative stress

- The antioxidant status of the diver is :
  - an important mechanism in the protection against injury
  - influenced both by genetic factors and diet.

Antioxidants

- Substances able to delay or prevent oxydation of substrates
- They trap free radicals such as $O_2^{-\circ}$(superoxide anion), H_2O_2, OH^-,...)
- Protective effects against
  - Cancers
  - Cardio-vascular disease
  - Metabolic disease
  - Neuro-degenerative disease
- Polyphenols = class of antioxidants

1-3-2018

Antioxidants

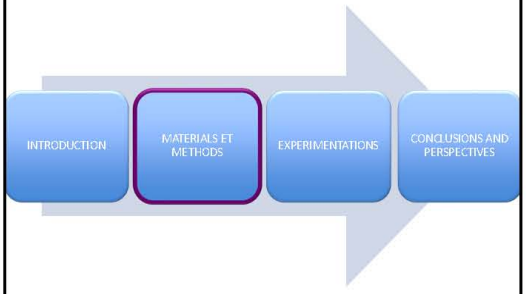
- Polyphenols
  - Prevent degradation of NO by ROS
  - ↘ vasoconstriction
  - ↘ pro-inflammatory responses
- Are found in :
  - Some wines and fruit juices
  - Black and green tea
  - Cacoa -> DARK CHOCOLATE

Dark chocolate

- Large quantity of polyphenols (flavonoïds)
- It is able to :
  - Inhibit production of O_2^{-0}
  - ↗ production of BH4 → ↗ activity eNOS → ↗ NO
  - ↘ arterial stiffness → benefit for cardio-vascular system (hypertension)
  - Trap O_2^{-0} and $ONOO^-$ → ↘ oxidative stress
  - → improve function of vascular smooth muscle
  - → Would prevent vasoconstriction

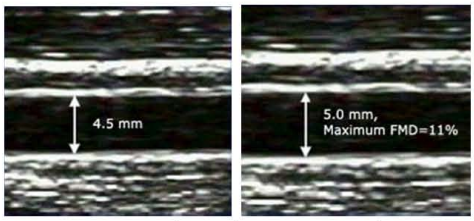
Objectives of the study

- Compare the effects of scuba diving on
  - Endothelial vasodilatation
  - Oxidative stress
- Try to prevent post-dive endothelial dysfunction via dark chocolate ingestion



Exploration of endothelial function

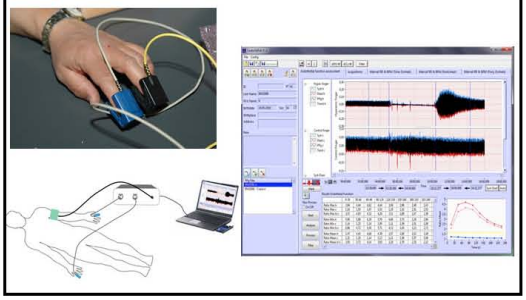
- Flow Mediated Dilation (FMD)



FMD = Post occlusion diameter / pre occlusion diameter (%)

Exploration of endothelial function

- Post Occlusion Reactive Hyperhemia (PORH)



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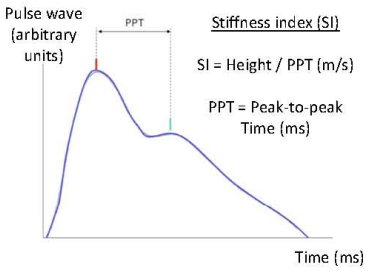
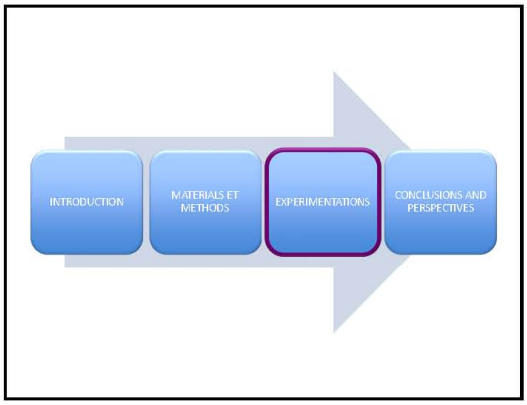
Exploration of endothelial function

- Photoplethysmography



Hematological parameters

- NO and $ONOO^-$



Experiments

ENDOTHELIAL DYSFONCTION AFTER A SCUBA OR BREATH-HOLD DIVING	THE EFFECTS OF DARK AND WHITE CHOCOLATE ON ENDOTHELIAL FUNCTION OF HEALTHY SUBJECTS	DARK CHOCOLATE PREVENTS ENDOTHELIAL DYSFUNCTION AFTER A SCUBA DIVE
Objective	Objective	Objective
Methods	Methods	Methods
Results	Results	Results
Discussion	Discussion	Discussion

UHM 2013, VOL. 40, NO. 2 – ENDOTHELIAL FUNCTION IN SCUBA AND BREATH-HOLD DIVING

Nitric oxide-related endothelial changes in breath-hold and scuba divers

S. Theunissen[1,2], F. Guerrero[2], N. Sponsiello[1,3,4], D. Cialoni[1,3,4], M. Pieri[3], P. Germonpré[1,5], G. Obeid[5], F. Tillmans[1], V. Papadopoulou[1,6], W. Hemelryck[1], A. Marroni[3], D. De Bels[1,7], C. Balestra[1,3,4]

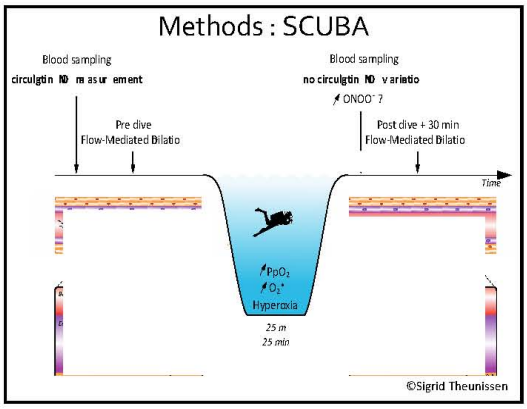
[1] Haute Ecole Paul Henri Spaak, Environmental, Occupational & Aging Physiology Lab., Brussels, Belgium;
[2] Université de Bretagne Occidentale, UFR Sciences et Techniques, Brest, France;
[3] DAN Europe Research, Brussels, Belgium;
[4] DAN Europe, Apnea Task Force, Roseto, Italy;
[5] Center for Hyperbaric Oxygen Therapy, Military Hospital Queen Astrid, Brussels, Belgium;
[6] Department of Bioengineering, Imperial College London, London, UK;
[7] Intensive Care Department, Brugmann University Hospital, Brussels, Belgium

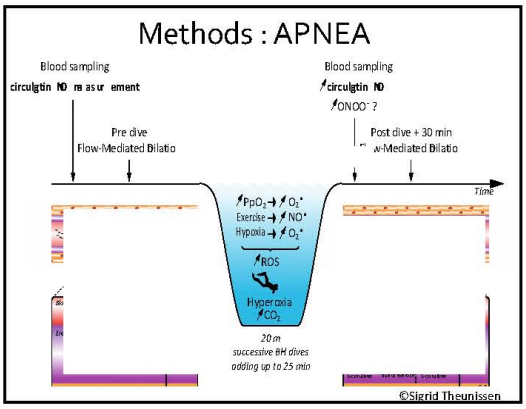
CORRESPONDING AUTHOR: Sigrid Theunissen M.Sc. – *sigtheunissen@gmail.com*

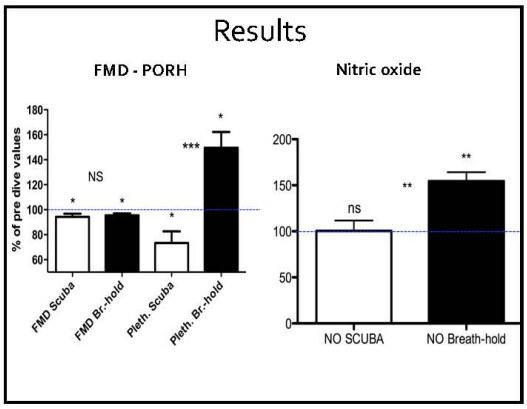
Objective

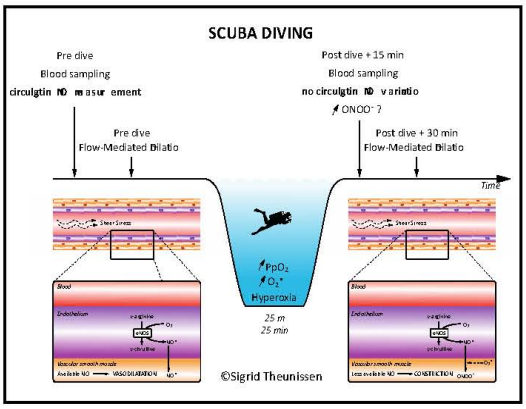
SCUBA	APNEA
Continuous Hyperoxia	Intermittent Hyperoxia
-> oxidative stress	-> oxidative stress ?
• Endothelial dysfunction :	• Endothelial dysfunction ?
– Due to oxidative stress ?	
– Due to bubbles ?	

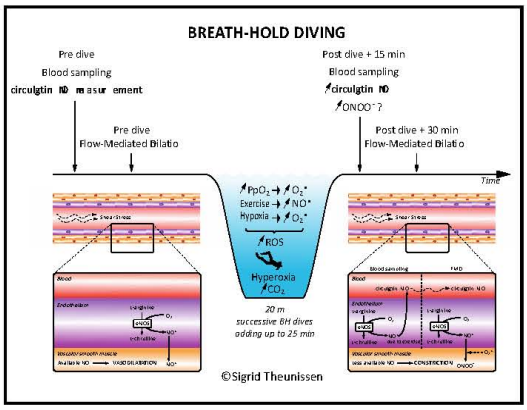
1-3-2018











Conclusion

- In BH diving: No bubbles but FMD ↘
- ➔ Bubbles are not the cause of the ↘ in FMD
- The 2 types of diving ↘ FMD
- SCUBA : NO doesn't change
- Breath-hold : NO increases (Exercise)
- Hypothesis : NO inactivated (transformed in ONOO-)
- Oxidative stress ? To test with ONOO-

1-3-2018

Experiments

ENDOTHELIAL DYSFONCTION AFTER A SCUBA OR BREATH-HOLD DIVING	THE EFFECTS OF DARK AND WHITE CHOCOLATE ON ENDOTHELIAL FUNCTION OF HEALTHY SUBJECTS	DARK CHOCOLATE PREVENTS ENDOTHELIAL DYSFUNCTION AFTER A SCUBA DIVE
Objective	Objective	Objective
Methods	Methods	Methods
Results	Results	Results
Discussion	Discussion	Discussion

Objective

- Before giving chocolate to the divers, we had to know :
  - What chocolate ?
  - When giving chocolate before the dive ?

➔ Compare the vascular effects of dark and white chocolate on healty subjects

Methods

- Timing :

Pre-tests T= 0 +30 min, 60 min.....180 min

- Choice of chocolate :

Pre-tests Dark or white chocolate +120 min

Results

Timing :

FMD n=9	Avant chocolat	Post 30 min	Post 60 min	Post 90 min	Post 120 min	Post 150 min	Post 180 min
Mean (%)	109,6	108,3	107,6	107,6	**115,7**	109,9	109,7
Standard Deviation (%)	7,7	5,1	6,1	6,8	**7,4**	6,7	7,9
T test btw before and after chocolate		ns	ns	ns	*	ns	ns

Choice of chocolate :

White Chocolate n=20	Before chocolate		After chocolate		t test
	Mean	St. Dev.	Mean	St. Dev.	
Pre occlusion brachial diameter (mm)	3,7	0,5	3,7	0,5	ns
Flow-mediated dilation (%)	115,0	5,3	111,1	7,9	ns
Arterial stiffness (m/s)	7,0	1,6	7,2	**1,4**	ns

Dark Chocolate n=20	Before chocolate		After chocolate		t test
	Mean	St. Dev.	Mean	St. Dev.	
Pre occlusion brachial diameter (mm)	3,6	0,7	3,8	0,7	*
Flow-mediated dilation (%)	109,5	7,2	112,1	7,6	*
Arterial stiffness (m/s)	7,2	1,1	7,0	0,6	ns

Conclusion

- Which one ?

Dark chocolate increases FMD, white not

➔ DARK CHOCOLATE

- When ?

The maximum effect is 2h after ingestion

➔ 1h30 before the SCUBA dive

Experimentations

ENDOTHELIAL DYSFONCTION AFTER A SCUBA OR BREATH-HOLD DIVING	THE EFFECTS OF DARK AND WHITE CHOCOLATE ON ENDOTHELIAL FUNCTION OF HEALTHY SUBJECTS	DARK CHOCOLATE PREVENTS ENDOTHELIAL DYSFUNCTION AFTER A SCUBA DIVE
Objective	Objective	Objective
Methods	Methods	Methods
Results	Results	Results
Discussion	Discussion	Discussion

1-3-2018

4 Diving and Hyperbaric Medicine Volume 45 No. 1 March 2015

Original articles

The effect of pre-dive ingestion of dark chocolate on endothelial function after a scuba dive

Sigrid Theunissen, Costantino Balestra, Antoine Boutros, David De Bels, François Guerrero and Peter Germonpré

Abstract

(Theunissen S, Balestra C, Boutros A, De Bels D, Guerrero F, Germonpré P. The effect of pre-dive ingestion of dark chocolate on endothelial function after a scuba dive. *Diving and Hyperbaric Medicine*. 2015 March;45(1):4-9.)

Objective: The aim of the study was to observe the effects of dark chocolate on endothelial function after scuba diving.

Methods: Forty-two male scuba divers were divided into two groups: a control ($n = 21$) and a chocolate group ($n = 21$). They performed a 33-metres deep scuba-air dive for 20 minutes in a diving pool (Nemo 33, Brussels). Water temperature was 33°C. The chocolate group ingested 30 g of dark chocolate (86% cocoa) 90 minutes before the dive. Flow-mediated dilatation (FMD), digital photoplethysmography and nitric oxide (NO) and peroxynitrites ($ONOO^-$) levels were measured before and after the scuba dive in both groups.

Results: A significant decrease in FMD was observed in the control group after the dive (91 ± 7% (mean ± 95% confidence interval) of pre-dive values; $P < 0.001$) while it was increased in the chocolate group (105 ± 5% of pre-dive values; $P < 0.001$). No difference in digital photoplethysmography was observed between before and after the dives. No variation of circulating NO level was observed in the control group whereas an increase was shown in the chocolate group (154 ± 73% of pre-dive values; $P = 0.04$). A significant reduction in $ONOO^-$ was observed in the control group (84 ± 12% of pre-dive values; $P = 0.003$) whereas no variation was shown after the dive with chocolate intake (100 ± 28% of pre-dive values; *ns*).

Conclusion: Ingestion of 30 g of dark chocolate 90 minutes before scuba diving prevented post-dive endothelial dysfunction, as the antioxidants contained in dark chocolate probably scavenge free radicals.

Key words

Antioxidants, cardiovascular, hyperoxia, nitric oxide, diving research, scuba, circulation

Objective

To observe the effects of 30 g dark chocolate before a SCUBA dive

Population

- 2 groups (control and chocolate) of 21 divers (35,3 ± 5,9 years vs 36,8 ± 6,2 years)
- Diver 3* CMAS, AOW Padi or licence allowing to dive to 30 msw
- Experience of more than 50 dives
- Never had DCI
- Non – smoker
- Good health, good physical condition

Protocol

1h30' before The dive

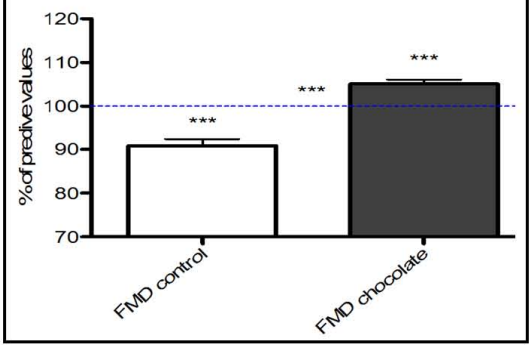
Pre-tests

Nemo 33
SCUBA dive
33 m
20 minutes

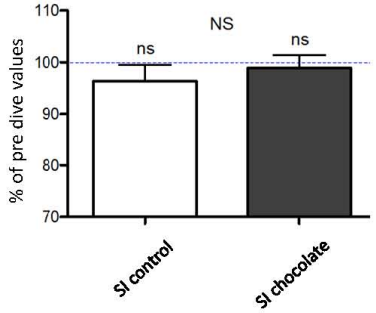
Post-tests

Chocolate group only

Results : FMD

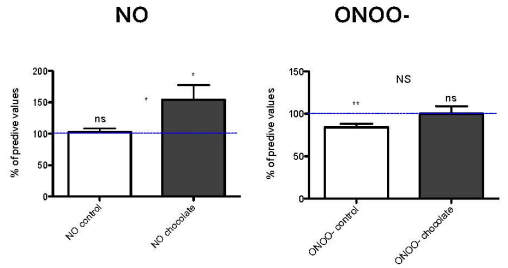


Results : Photoplethysmographie



1-3-2018

Results : Blood sampling

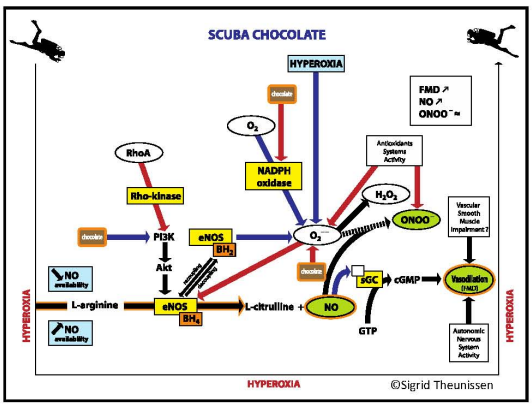


Conclusion

- Chocolate is able to increase FMD
- No variation in NO is observed
- Hypothesis of previous studies : NO is transformed in ONOO-
- BUT, $ONOO^-$ ↘ after a SCUBA dive !

➔ NO is not transformed in ONOO ??

➔ no oxidative stress ??

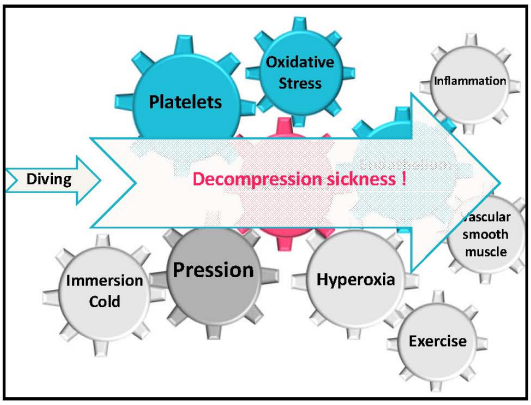


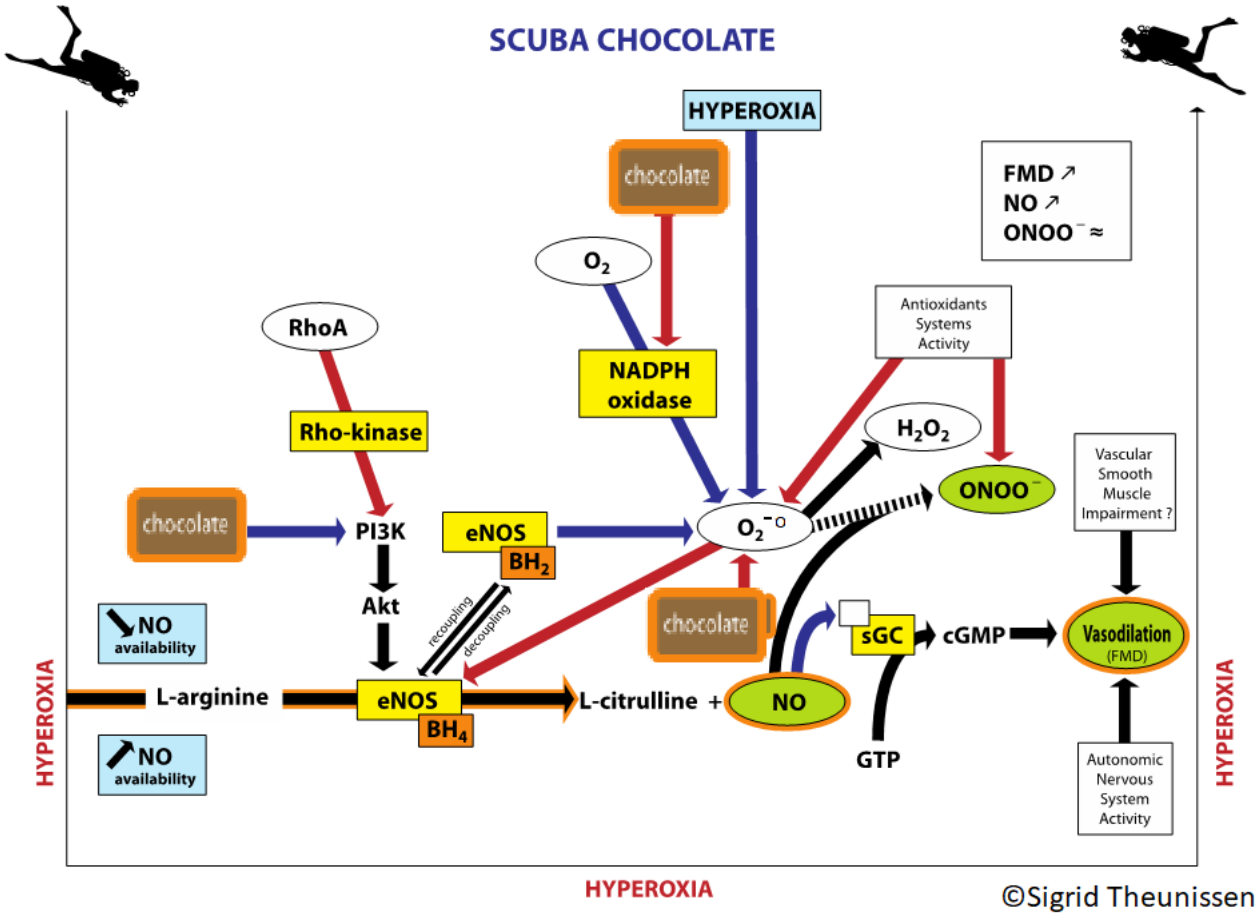
Conclusions and perspectives

- ↘ FMD after breath-hold diving ➔ not linked to bubbles
- In SCUBA diving, the ↘ of FMD is linked to oxidative stress because it is prevented by dark chocolate
- ONOO- ➔ not a good marker of oxidative stress
- Dark chocolate ➔ good method to prevent post-dive endothelial dysfunction

Conclusions and perspectives

- The absence of NO variation suggests that the ↘ of FMD is the consequence of :
  - Autonomic nervous system
    and/or
  - Alteration of vascular smooth muscle
- PORH is a better tool than SI to observe endothelial function in small arteries
- Mecanisms = a good model for the elderly person




SCUBA CHOCOLATE
HYPEROXIA
chocolate
FMD ↗
NO ↗
ONOO⁻ ≈
O2
RhoA
NADPH oxidase
Antioxidants Systems Activity
Rho-kinase
H2O2
ONOO⁻
Vascular Smooth Muscle Impairment ?
chocolate
PI3K
eNOS
BH2
O2⁻
Akt
recoupling
decoupling
chocolate
sGC
cGMP
Vasodilation (FMD)
NO availability
L-arginine
eNOS
BH4
L-citrulline +
NO
GTP
NO availability
Autonomic Nervous System Activity
HYPEROXIA
HYPEROXIA
HYPEROXIA
©Sigrid Theunissen

Chapter 6 Dual phase decompression theory and bubble dynamics

Albrecht Salm

28-2-2018

Implementation of
Dual Phase Decompression Models
in Tables & Meters for technical diving
Albrecht Salm

THE

SUB
MARINE
CONSULTING

GROUP

TEL AVIV – SAN FRANCISCO – STUTTGART

WWW.SMC-DE.COM

Agenda:

- Dive tables versus dive computers in technical diving
- Shortcomings of dive computers
- The controversy around "Deep Stops":
  "My model is better than yours …"
- Solution to this dilemma:
  statistically based decompression tables / P(DCS)
- In-sights from real world diving:
  → DAN DSG database (35.000 dives)
- DJRS, the Dive Jump Reporting System;
  → USN (0,8 million dives)

Test „Dive": 42 m, 25 min, Air
comparison with
desktop deco software:

→ Subsurface
→ DecoPlanner
→ (1st.) Excel-Version

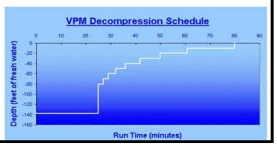
and dive computers:

→ Ratio iX3m
→ Scubapro / Uwatec G2 (Galileo 2)

VPM Implementations

… with:
Subsurface 4.7.4
(11/2017)
https://subsurface-divelog.org

DecoPlanner 3.1.4



Tauchplandetails

Subsurface (4.7.4.0) Plan erstellt am 28.11.17
Runtime: 73min

	Tiefe	Dauer	Runtime	Gas
↘	42m	2min	2min	Luft
→	42m	25min	27min	
↗	21m	2min	29min	
-	21m	1min	30min	
↗	18m	0min	30min	
-	18m	2min	32min	
↗	15m	0min	32min	
-	15m	3min	35min	
↗	12m	0min	35min	
-	12m	5min	40min	
↗	9m	0min	40min	
-	9m	6min	46min	
↗	6m	0min	46min	
-	6m	10min	56min	
↗	3m	0min	56min	
-	3m	17min	73min	
↗	0m	0min	73min	

CNS: 12%
OTU: 37

42 m,
25 min,
Air:

Conservatism:
0 → decotime: 46'
1 → 50'
2 → 56'
3 → 63'
4 → 72'

Test „Dive": 42 m, 25 min, Air:

Subsurface (4.7.4.0) Plan erstellt am 28.11.17
Runtime: 55min

	Tiefe	Dauer	Runtime	Gas
↘	42m	2min	2min	Luft
→	42m	25min	27min	
↗	9m	4min	31min	
-	9m	1min	32min	
↗	6m	0min	32min	
-	6m	7min	39min	
↗	3m	0min	39min	
-	3m	16min	55min	
↗	0m	0min	55min	

GF: gradient factor. See for explanation last slide.

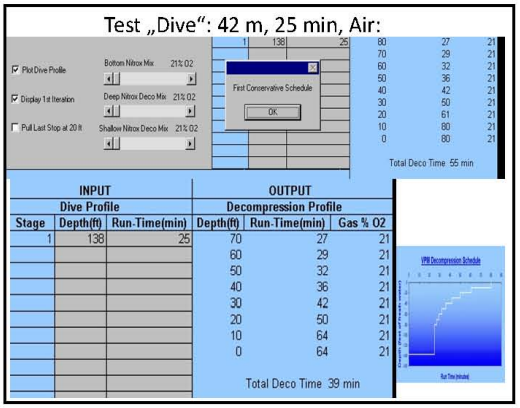
CNS: 11%
OTU: 35
Dekomodell: Bühlmann ZHL-16C mit GF niedrig = 100% und GF hoch = 100%
Oberflächendruck: 1013mbar (0m)

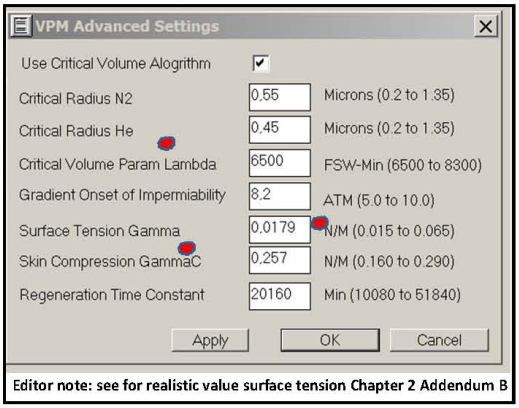
28-2-2018

Stop depth [m]	VPM Cons. = 0	ZH-L16 GF Hi/Lo = 1.0
21	1	-
18	2	-
15	3	-
12	5	-
9	6	1
6	10	7
3	17	16

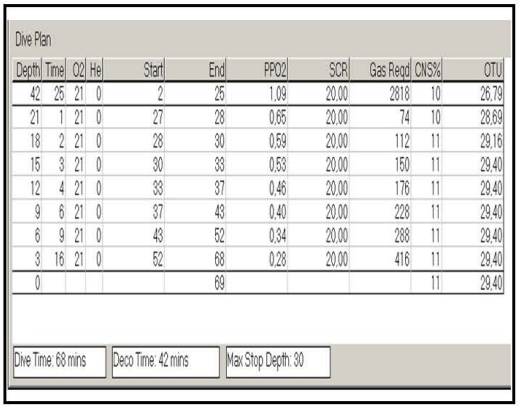
Test „Dive“: 42 m, 25 min, Air:

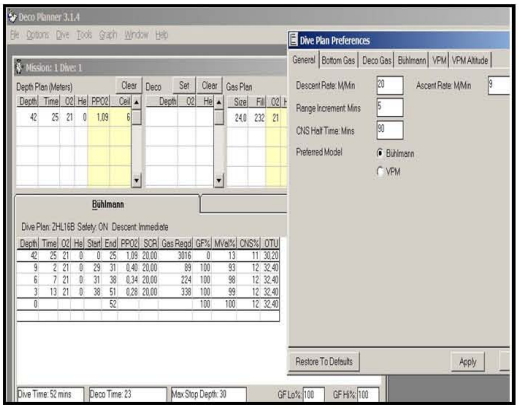
Test „Dive“: 42 m, 25 min, Air:

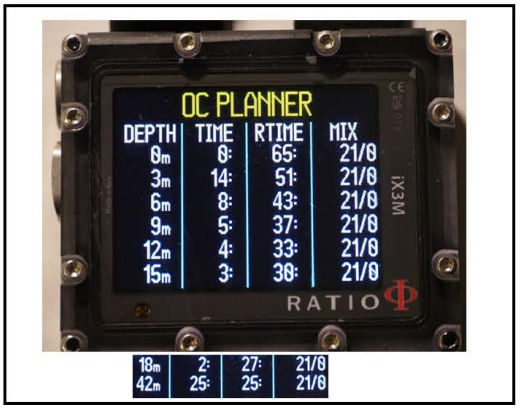




Editor note: see for realistic value surface tension Chapter 2 Addendum B







28-2-2018

Test „Dive“: 42 m, 25 min, Air:

Conservatism:	decotime:	deepest stop:
0 →	40’	18/2
1 →	69’	18/2
2 →	73’	18/3
3 →	81’	21/1
4 →	91’	21/2
5 →	108’	21/3

Info: RGBM Table from rgbmdiving.com
Bruce Wienke; 2003

depth = 140.00 fsw ppO2 = 1.10 atm
bottom time = 25. min OTU = 29. min CNS = 0.10 gas = 115. cft

stop	depth	wait	tissue	tension	grad	ppO2
1	60.00	0.00	1.67	94.91	46.79	0.59
2	50.00	1.00	1.67	85.29	46.79	0.53
3	40.00	2.00	3.33	73.17	43.83	0.46
4	30.00	2.50	3.33	62.52	43.83	0.40
5	20.00	3.00	3.33	53.82	43.83	0.34
6	10.00	7.00	6.67	40.04	40.44	0.27

total time = 45. min OTU = 29. min CNS = 0.11 gas = 134. cft

Test „Dive“: 42 m, 25 min, Air:

Info: USN
von Rev. 6: 2008→ Rev. 7: 12/2016

Table 9-9. Air Decompression Table (Continued).
(DESCENT RATE 75 FPM—ASCENT RATE 30 FPM)

Bottom Time (min)	Time to First Stop (M:S)	Gas Mix	DECOMPRESSION STOPS (FSW) Stop times (min) include travel time, except first air and first O_2 stop 100	90	80	70	60	50	40	30	20	Total Ascent Time (M:S)	Chamber O_2 Periods	Repet Group
140 FSW														
10	4:40	AIR									0	4:40	0	E
		AIR/O_2									0	4:40		
15	4:00	AIR									5	9:40	0.5	H
		AIR/O_2									3	7:40		
20	4:00	AIR									13	17:40	0.5	J
		AIR/O_2									7	11:40		
In-Water Air/O_2 Decompression or SurDO_2 Recommended														
25	3:40	AIR								3	24	31:20	1	L
		AIR/O_2								2	12	18:20		
30	3:40	AIR								7	37	48:20	1	N
		AIR/O_2								4	19	27:20		

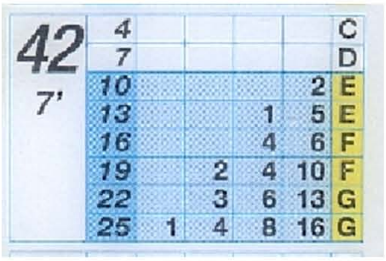
Info: DCIEM
Kidd-Stubbs 1983

42	20	-	-	-	-	-	4	7	10	21	G
	25	-	-	-	-	-	7	8	17	32	I
	30	-	-	-	-	4	6	8	28	46	K
	35	-	-	-	-	5	7	9	37	58	L
	40	-	-	-	-	7	7	10	46	70	N
	45	-	-	-	3	5	8	16	53	85	O
	50	-	-	-	4	6	8	21	62	101	

The Department of National Defence (Canada), Defence and Civil Institute of Environmental Medicine (DCIEM), and Universal Dive Techtronics, Inc. (UDT) disclaim any and all responsibilities for the use of these tables and procedures.

Info: DECO 2000
Max Hahn

Test „Dive“: 42 m, 25 min, Air:



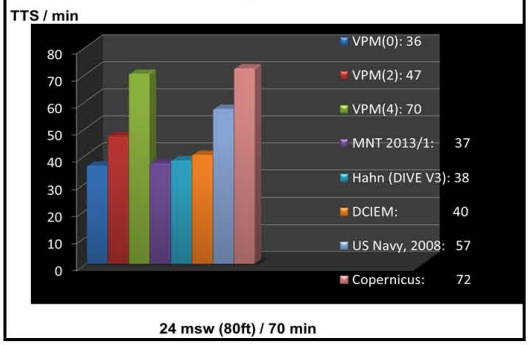
28-2-2018

42 m, 25 min, Air; Synopsis:

VPM	Name	Deepest stop [m]	TTS [min]	Variation
-	RGBM	15	25	n.a.
X	Ratio	18	40	40 – 108
X	Excel	21	39	n.a.
X	SubS.	21	46	46 - 72
X	DP	21	47	47 - 74

Per-fusion	Name	Deepest stop [m]	TTS [min]	
X	Hahn	12	34	
X	DP	9	28	
X / other	USN/ DCIEM	9	32	
X	G2	9	39	

Total decompression time + Ascent
Air dives → compared with VPM DP 3.1.4



24 msw (80ft) / 70 min


PROCEEDINGS

DECOMPRESSION AND THE DEEP STOP

Salt Palace Convention Center
Salt Lake City, Utah, USA

JUNE 24-25, 2008

SPONSORED BY:

Undersea and Hyperbaric Medical Society
US Office of Naval Research
Divers Alert Network
National Association Underwater Instructors
Professional Association of Diving Instructors

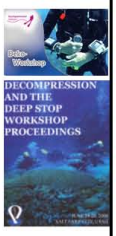
DAN

NAUI TECHNICAL

PADI

Editors: Peter B. Bennett, Ph.D, D.Sc. Bruce Wienke, Ph.D, Simon Mitchell, MB, Ch.B., Ph.D

„Consensus Statement" [100], p. 324:



Statement regarding the efficacy of „deep stops" appropriate for release to the diving community:

„In respect of decompression diving there is conflicting evidence regarding the relative efficacy of decompression regimens that include empirical or model-derived deep stops (as defined) and decompression regimens prescribed by gas content models"

deco workshop @ Tauchsportcenter Esslingen

Results from the USN / NEDU „deep stop" study:

→ NEDU Report

→ Source: NEDU TR 11-06 July 2011

→ REDISTRIBUTION OF DECOMPRESSION STOP TIME FROM SHALLOW TO DEEP STOPS INCREASES INCIDENCE OF DECOMPRESSION SICKNESS IN AIR DECOMPRESSION DIVES. Navy Experimental Diving Unit, Authors: DAVID J. DOOLETTE, WAYNE A. GERTH, KEITH A. GAULT.

deco workshop @ Tauchsportcenter Esslingen

→ 81 Navy divers
→ 192 dives / # DCS = 3 with the shallow stops
→ according to USN- VVAL18 (A1, „shallow")

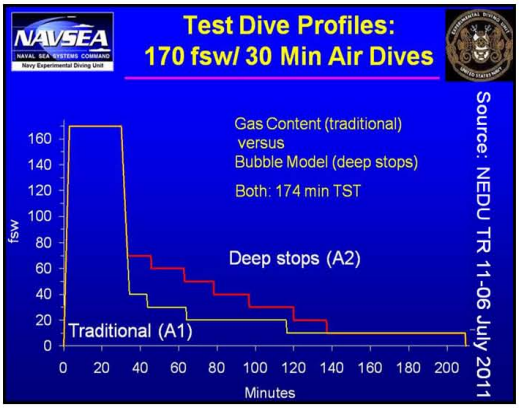
→ 198 dives / # DCS = 10 with "deep stops"
→ according BubbleVolumeModel(3) (A2, „deep")

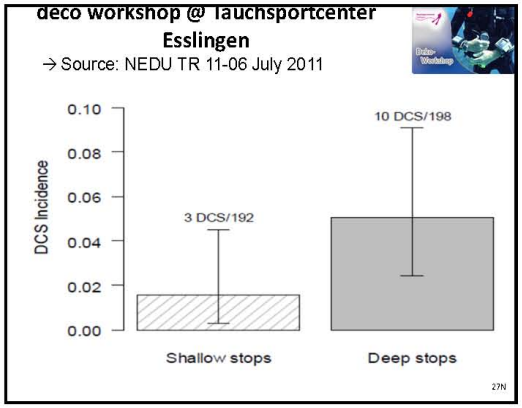
→ 170 feet (ca. 52 m) / 30 min bottom time
→ workload ca. 130 W, O_2-consumption ca. 2,3 L/min
→ ascent- & descent rates and water temperature meticously controlled!
→ TST (time-to-surface) = constant for both profiles = 174 min

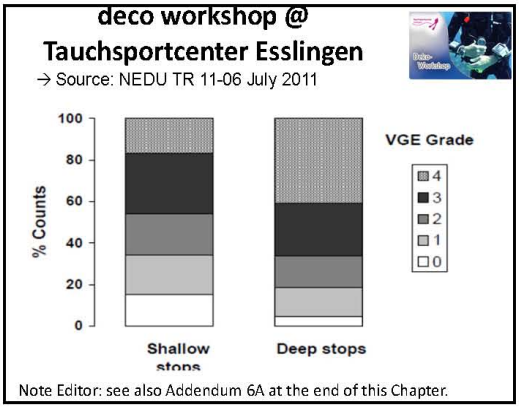
28-2-2018

deco workshop @ Tauchsportcenter Esslingen

depth / time	[feet]	[min.]					P (DCS)	[%]
stops:	70	60	50	40	30	20	10	BVM(3)	NMRI98
shallow (A1)				9	20	52	93	6,158	4,429
deep (A2)	12	17	15	18	23	17	72	3,664	5,880







28-2-2018

YouTube decompression controversies

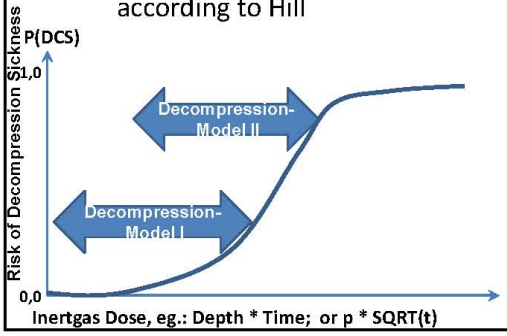
Conclusions

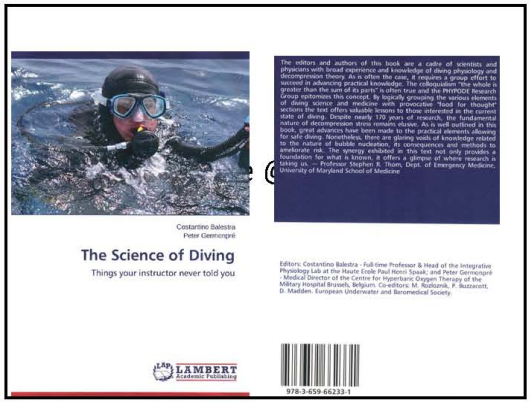
- "Optimal" decompression from deep bounce dives is unknown
- Approaches that emphasise deep stops are not supported by available data and have probably been 'oversold' to the technical diving community
- Best evidence suggests we should de-emphasise deep stops, but it remains uncertain by how much!

Probabilistic DCS moddeling
P(DCS):
statistical probability (P) of contracting a decompression sickness (DCS)

P(DCS) = 1.0 – P(no DCS)

P(DCS): dose- / response curve according to Hill





DAN DSG data bank

The Science of Diving – Things your instructor never told you ...

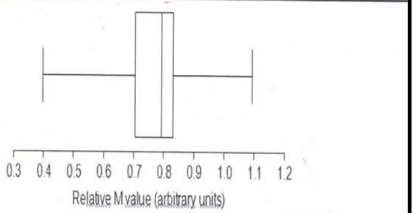
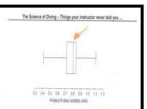


Figure 7: Box-plot of calculated tissue supersaturation levels on 165 cases of DCS recorded over 10738 DSL dives.

DAN DSG data bank



DSG (Diver Safety Guardian):
until 03/2015: 10,738 uploaded dive profiles with 165 DCS cases, unspecified, rate of 1.58 %;

→ DL 7 level 3 dive computer logs @ DAN DSG Portal.

→ Distribution of DCS cases:
ZH-L computers : RGBM
ca. 50 : 50

Hardly ever any compartment supersaturation!
The calculated saturations avg. < 80 % of allowed M-Values (source: lc. p. 29):

28-2-2018

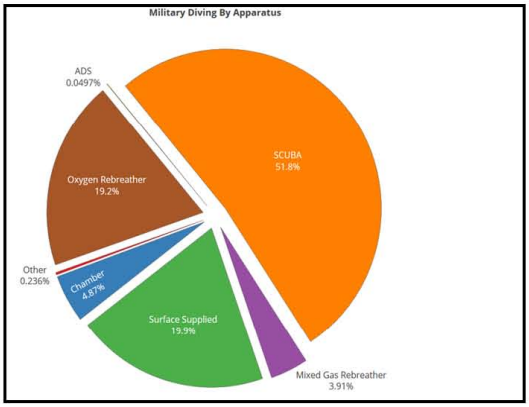
„BIG DIVE DATA“
Source: http://
divingresearch.scripts.mit.edu/militarydivingdata/

DIVE INTO DATA.

In 2008 the US military transitioned to a central, electronic dive logging system called the Dive Jump Reporting System, DJRS. Meant to increase visibility on diving trends and maintain an accessible system to view dive currency, the system has been a great asset to the dive community and its leadership.

In the summer of 2015, I requested the largest data draw in DJRS history: every electronic dive log and dive related mishap since DJRS implementation. Following a Freedom of Information Act request, the Naval Safety Center, owner and maintainer of the system, provided 768,851 dive log entries and 39 mishap reports (since 2008). In addition to the DJRS data, Atmospheric Dive Suit (ADS) dive logs were collected from the Portsmouth Naval Shipyard to further strengthen the dataset. Saturation Dive logs, however, were not obtained. With six years' worth of military dive history, we can quantitatively learn a lot about our community.

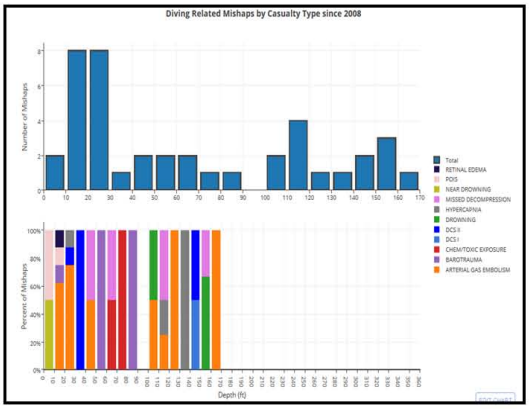
Figure 1 is a traditional histogram showing all military dives since 2008. All services, types of diving rigs, and tasks are represented. Some technical notes: (1) dives are broken into incremented 10 foot "bins," starting from the left limit; (2) The blue columns represent the total number of dives on a logarithmic scale; and (3) The orange line represents the cumulative percentage (Empirical Cumulative Distribution Function, ECDF) of dives achieved to a given depth.



→ Deeper than ca. 44 m (145 feet)

→ mainly „mixed gas rebreathers“ & „surface supplied“

→ Deeper than 65 m (215 feet):
no more SCUBA!

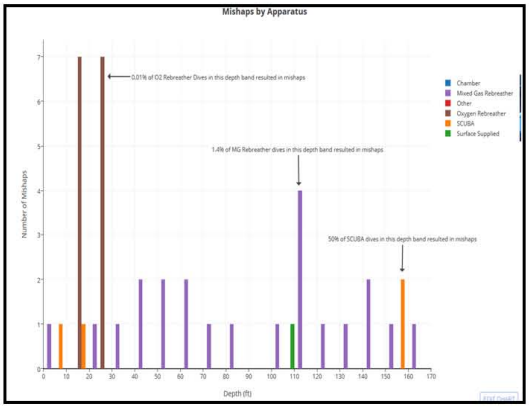


„BIG DIVE DATA“:

→ only 41 „mishaps“,
with:

➔ DCS Type I + II: 3
➔ AGE: 18
➔ missed deco: 5

➔ all 3 categories
➔ DCI: 26 / 768.851 = < 0,04 % !!!



28-2-2018

deco workshop im Tauchsportcenter Esslingen

„**BIG DIVE DATA**": Summary / safety @ diving:

→ dive logs + Cochran® log-files USN
→ analysis of ca. 0.8 million dives
→ from 2008 until 2014
→ Average ca. 115,241 dives p.a.
→ only (ca.) **41 „mishaps"**

➔ 90 % of all dives < 18 m (60 feet)
➔ 60 % of all dives < 6 m (20 feet)
➔ Average depth 8.5 m (27.8 feet)

Take home (2nd. lecture):

→ **Dive Computers & Desktop Deco Software**
→ **may have bugs!**

("may" means: they HAVE bugs!!!)

→ **There are no consensus standards on:**

→ **Implementation & Parameters**
→ **as well no documentation or public quality control!**

→ **Do not rely on a single source!**
→ **Use multiple products!**
→ **Compare!**
→ **Do not fall prey to the (not-) RTFM-Error !!!**
→ **(Go & buy & use a portable doppler unit!)**

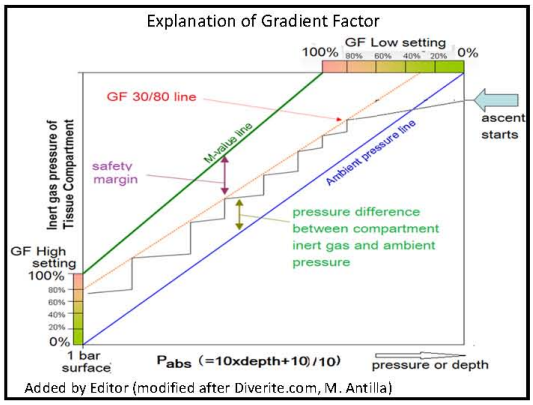
Downloads:

→ „all" VPM (& RGBM ...) papers:
https://www.divetable.info/area.htm#Kap%207

→ 6 Copernicus papers:
https://www.divetable.info/TEMP/CSD_2018/Copernicus.zip

DAN TEC Conference 2008:
https://www.divetable.info/TEMP/CSD_2018/Proceedings.pdf

→ USN, only tables 2008 / 2016:
https://www.divetable.info/workshop/USN_Rev7_Tables.pdf



Addendum 6A

Aviat Space Environ Med. 2012 Oct;83(10):951-7.

Doppler bubble grades after diving and relevance of body fat.

Schellart NA[1], Vellinga TP, van Dijk FJ, Sterk W.

Author information

Abstract

BACKGROUND: From the literature on venous gas embolism (VGE) and decompression sickness (DCS), it remains unclear whether body fat is a predisposing factor for VGE and DCS. Therefore, this study analyses body fat (range 16-44%) in relation to precordial VGE measured by Doppler bubble grades. Also examined is the effect of age (range 34-68 yr), body mass index (BMI; range 17-34 kg x m(-2)), and a model estimate of VO2(max) (maximal oxygen uptake; range 24-54 ml x kg(-1) x min (-1)).

METHODS: Bubble grades were determined in 43 recreational divers after an open sea air dive of 40 min to 20 m. Doppler bubble grade scores were transformed to the logarithm of the number of bubbles/cm2, logB, and the logarithm of the Kissman Integrated Severity Score (KISS) to allow numerical analysis. Statistical analyses were performed with Pearson's regular and partial correlations, and uni- and multivariate linear regressions.

RESULTS: For divers in their midlife (and older), the analyses indicate that neither body fat nor BMI stimulate bubble formation, since correlations were nonsignificant. In contrast, age and especially VO2(max) appeared to determine VGE. For these types of dives it was found that logB = -1.1 + 0.02 age - 0.04Vo2(max).

CONCLUSION: Based on these data we conclude that body fat and BMI seem less relevant for diving. We recommend that medical examinations pay more attention to VO2(max) and age, and that international dive institutions come to a consensus regarding VO2(max) criteria.

PMID: 23066616

Addendum 6B (free access paper)

J Appl Physiol (1985). 2013 Mar 1;114(5):602-10. doi: 10.1152/japplphysiol.00949.2012. Epub 2013 Jan 10.

Body fat does not affect venous bubble formation after air dives of moderate severity: theory and experiment.

Open/close author information list

Schellart NA[1], van Rees Vellinga TP, van Hulst RA.

Author information

Abstract

For over a century, studies on body fat (BF) in decompression sickness and venous gas embolism of divers have been inconsistent. A major problem is that age, BF, and maximal oxygen consumption (Vo2max) show high multicollinearity. Using the Bühlmann model with eight parallel compartments, preceded by a blood compartment in series, nitrogen tensions and loads were calculated with a 40 min/3.1 bar (absolute) profile. Compared with Haldanian models, the new model showed a substantial delay in N2 uptake and (especially) release. One hour after surfacing, an increase of 14-28% in BF resulted in a whole body increase of the N2 load of 51%, but in only 15% in the blood compartment. This would result in an increase in the bubble grade of only 0.01 Kisman-Masurel (KM) units at the scale near KM = I-. This outcome was tested indirectly by a dry dive simulation (air breathing) with 53 male divers with a small range in age and Vo2max to suppress multicollinearity. BF was determined with the four-skinfold method. Precordial Doppler bubble grades determined at 40, 80, 120, and 160 min after surfacing were used to calculate the Kisman Integrated Severity Score and were also transformed to the logarithm of the number of bubbles/cm(2) (logB). The highest of the four scores yielded logB = -1.78, equivalent to KM = I-. All statistical outcomes of partial correlations with BF were nonsignificant. These results support the model outcomes. Although this and our previous study suggest that BF does not influence venous gas embolism (Schellart NAM, van Rees Vellinga TP, van Dijk FH, Sterk W. Aviat Space Environ Med 83: 951-957, 2012), more studies with different profiles under various conditions are needed to establish whether BF remains (together with age and Vo2max) a basic physical characteristic or will become less important for the medical examination and for risk assessment.

PMID: 23305985 DOI: 10.1152/japplphysiol.00949.2012

[Indexed for MEDLINE] **Free full text**

Chapter 7 Treatment of Decompression Illness (DCI)

Adel Taher

Abstract

The treatment of Decompression Illness (DCI) went through various stages and experimental guess work before reaching the state it has now. Decompression was observed first in Caisson workers spending long shifts working hard under pressure to build foundations for bridges and to dig tunnels. Those suffering the strange pains and sensory losses and even paralysis had nothing to help them, except good wishes and hopes for a spontaneous recovery.

1854, Pol & Watelle made the first attempts at alleviating these symptoms by returning the workers to pressure. Foley followed by constructing a small portable chamber, and many other pioneers came after that. In 1937, Behnke and Shaw performed the first 'modern' treatment of decompression sickness in a recompression chamber. Two years later, Behnke & Yarnbrough introduced hyperbaric oxygen into the standard treatment modalities of the US Navy. The rest is history that we know!

Other treatment gases were added, like HELIOX and EANx and new tables were developed. Saturation diving developed and created new challenges and saturation treatment tables were designed to cope with the new problems.

The concept of "treatment" is actually narrow and usually refers to the treatment in the recompression chamber. We prefer to term that, the "definitive treatment". The management of a diving accident should actually begin with educating the new divers and teaching them how to recognize the possible symptoms that could indicate a diving accident 'early'. Early recognition means early intervention, early activation of the rescue chain and starting 1st aid measures, which will have a direct impact on the prognosis of the anticipated 'definitive treatment'.

The 1st aid measures and the steps that need to be taken by the examining physician on site are mentioned and stressed. They represent the backbone for any subsequent treatment. After the patient reaches the recompression chamber, the Initial Assessment begins. This serves reaching a differential diagnosis and assessing the general condition and establishing a neurological base line and deciding whether the case needs recompression or not. The methods used to gather the information, get a detailed medical and diving history and the pitfalls are all discussed in detail. The time of arrival at the recompression chamber in relation to ending the dive and to the appearance of the first symptom are very important to notice and will eventually influence the choice of the suitable treatment table. The rationale behind using hyperbaric oxygen is also mentioned with a hint at the value of the 'oxygen window' in treatment.

Some figures comparing diving accidents among sport and recreational divers and commercial and military divers, are discussed. All the steps that need to be taken to start and conclude a chamber treatment are reviewed and some are discussed in detail. Also the question of starting physiotherapy under pressure is mentioned.

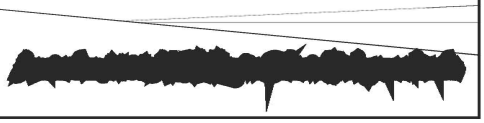
The decision making process and choice of treatment table is discussed and some tables are presented. The difficult questions that the treating physician might face are asked. Then HELIOX is presented as a possible modality for treating specific cases and the author's experience with such treatments as well as parts of his lecture in the HELIOX workshop in 2015 in Amsterdam is included.

At the end we look at various ways of treating the persistent symptoms following the initial recompression therapy.

Note that in the slide show the slides are ordered vertically.

2/22/2018

Treatment of Decompression Illness (DCI)



- He ***recompressed symptomatic workers*** to ***two thirds (2/3)*** of their working pressure ***on air*** for 25 to 30 minutes.
- ***The mortality rate dropped from 25% to 2.5%!!***
- ***1872, Nathan Zuntz***, suggested the use of ***hyperbaric Oxygen to treat cases of caisson's disease.***
- ***1908, John Scott Haldane*** proposed his decompression model for diving at sea level. He was famous for self experimentation.
- ***1924, the US Navy*** announced the ***first recompression treatment tables in its "Diving Manual".***

Short History...

- Historically, the ***only*** line of treatment available when decompression sickness (DCS) hit tunnel and Caisson workers was: ***"…hope for spontaneous recovery!"***
- ***1854, France Pol & Watelle,*** put workers back under pressure and noticed that they improved >>> they suggested systemic treatment in that manner
- ***Foley,*** constructed the first ***'portable' recompression chamber*** when supervising the building of a bridge on the Seine river. It sustained ***2.5 atm*** of pressure

- Over the years that followed several changes were made and ***the treatment depth depended on: depth of dive, fractions or multiples of that depth or depth of relief.***
- In ***1870, Paul Bert*** had announced in Paris, that he believed that ***100% Oxygen could have a beneficial effect delivered at 1 ata (2 atm), but no one implemented that for years.***
- ***1937, Behnke and Shaw*** first used a recomp. chamber in the ***treatment of decompression sickness.***

- ***1872, Dr. Andrew Smith***, in charge of the ***New York Bridge Company***, reported ***110 DCS cases*** among his Caisson workers. He treated them with ***Atropine, Mercury Chloride, Ergot, Ginger and Whisky.*** The toll of permanent disability and death due to neurologic DCS kept rising.
- ***1874***, during the building of the ***Eads bridge*** over the Mississippi River at St. Louis, ***91 DCS cases*** were observed and ***13 workers died***
- ***1889, Sir Ernest William Moir,*** was a British civil engineer and is credited with inventing the ***first medical airlock*** while working on the ***Hudson River Tunnel*** in New York in

- **1939, Yarnbrough & Behnke,** medical officers in the US Navy, treated DCS cases with ***Oxygen under pressure*** and scored ***good results***. In spite of that, it took a ***further 20 years*** for the USN to initiate standard treatment with ***hyperbaric O_2***
- **1960** the USN experimented with ***low pressure Oxygen tables at 33'/10msw.*** The results were not satisfactory. Consequently they doubled the pressure of O_2, ***60/18msw*** and the outcome was impressive.
- Treatment Tables (TT) were configured with ***O_2 delivered at 60'/18msw followed by 30'/9msw to safely decompress the tenders that accompany the patient***.

- ***USN TT 6 and 5*** saw the light and later they developed for ***AGE/CAGE*** the ***USN TT6A*** starting at a depth of ***165'/50 msw***, although the same results were achieved at 100'/30msw!!
- Many other treatment tables followed for ***saturation therapy (eg: Catalina Regime, Prof Joe Pilmanis),*** and ones for ***"blow ups"*** in deep commercial diving, when more than 60 min decompression time is omitted and then ***proprietary TT*** like the ***HELIOX TT*** and many others.

- The other option is using a ***constant flow, set at 15 L/min*** (can be initially raised to 25 L/min) with a ***non-rebreather mask.***
- ***Normobaric Oxygen*** should be administered ***without interruption*** for as long as the supply lasts or until rescue/medical personnel arrive and take over the case.
- ***Oxygen rebreathers*** could also be used for longer transport times.

  Note the time when Oxygen breathing is started for calculation of unit pulmonary toxic dose ***(UPTD/OTU)!***

The concept of "TREATMENT"

- The proper ***"treatment"*** covers more than the actual dealing with the symptoms and alleviating them.
- We should be regarding the larger concept of ***"management"*** and I would even expand the circle to include the ***"education of divers".***
- Treatment and management depend on ***"early recognition of symptoms"*** which in turn should be taught to divers and dive professionals.
- ***Early recognition*** also means ***early "On Site 1st Aid Measures"*** and early activation of the ***"rescue chain"*** which reflect on the ***prognosis*** and direct outcome of the treatment.

Normobaric O_2 will treat ***hypoxaemia*** generated by the ***salt water aspiration, inhaled vomitus, pneumothorax, hypoventilation or venous gas emboli.***

It will improve the ***oxygenation of all the hypoperfused tissues.***

It also facilitates the ***washout of inert gas*** from tissues and bubbles and ***helps reduce the bubble volume/diameter.***

Once symptoms are observed...start "on site First Aid"

- Symptoms are conveyed to the Divemaster or dive buddy and the ***time should be noted*** in relation to surfacing or ending the dive.
- ***Check Airway, Breathing and Pulse***

  > CPR + defibrillation (if needed and available!)
- ***Inform Recompression Chamber (RCC) and SAR***
- ***Normobaric Oxygen 100%*** should be administered at the highest possible concentration. This is facilitated by using a ***multifunction regulator*** and a ***demand 2nd stage*** with a well fitting ***TRU-Fit mask*** whenever the patient is conscious and can use it.

- Patient should be preferably ***lying down (supine)*** as long as this position is not causing discomfort. ***Left lateral position if unconscious.***
- ***Body temperature*** should be adjusted to the patient's ***'comfort zone'.***
- ***Pneumothorax?*** Dive history, dyspnoea, thoracic pain/expansion pain, tracheal deviation >>> Auscultate and check for 'rice crispies' indicating subcutaneous emphysema
- ***Medication*** are best left to the experience of the treating physician... Usually none are given! (Aspirin, 2 X 500 mg? Lidocaine, Diazepam, Thiopental, others...?) Do not mask symptoms!

2/22/2018

- ***Fluid therapy*** to conscious patients is ***essential*** and ***all diving accidents*** are assumed to be ***hypovolaemic and dehydrated*** . 1000 ml to 1500 ml/first hour is targeted. ORS or water are used.
- ***Normobaric Oxygen availability?*** Volume/pressure, delivery system, DAN versus Wenoll and other systems, trained personnel, how far to the recomp. chamber? ***Oxygen CCR?***
- ***Other injuries?*** Trauma, propeller injuries, poisonous marine life mimicking confusing symptoms...etc.
- ***Communication:*** mobiles, VHF, Sat-Tel

[redacted]

- The earlier the better... ***after 4 to 6 hours we are faced with "bubble organization"*** (platelet and leucocyte deposition, fibrin clot formation), i.e. reducing the size of the bubbles mechanically by elevating the pressure will not be effective.
- ***Neuronal ischaemia*** and damage can lead to **irreversible deficits.**
- This will directly reflect on the ***choice of treatment tables.***

Initial Assessment

- ***The initial assessment serves reaching a differential diagnosis and assessing the general condition and establishing a neurological base line and deciding whether the case needs recompression or not.***
- ***History:*** - breathing ***any compressed gas*** (scuba open circuit or rebreather) or ***repetitive, deep, breath hold diving***
- ***Dive profile:*** - dive profile, rate of ascent, gas breathed, repetitive dive, days of repetitive diving, activities in between dives, time of onset of 1st symptom, changes in character and/or intensity of symptoms, or new symptoms, response to 1st aid measures

- ***Early arrival*** to chamber gives the treating physician the chance to ***reverse the direct effects of the bubble formation: vascular occlusions, tissue mechanical distraction, inflammatory reactions, endothelial damage and leakage causing edema.***
- ***Delay in recompression therapy will have less favorable initial results and more sessions of recompression as well as longer physiotherapy and rehabilitation periods.***

- ***Previous medical history, medication, allergies***
- ***Time of onset of 1st symptom ...AGE/CAGE symptoms*** can occur in the ***last few meters of ascent and up to 10 minutes after ending the dive***
- ***DCS symptoms require not less than 15 min after surfacing*** to materialize
- Patient's account, dive buddy, and DM – esp. if patient unconscious!!
- ***Dive computers*** and number of previous repetitive dives and dive days to assess the Nitrogen load in tissues (DM Syndrome!).
- ***Beware of dive computers!!***

The Rationale behind Recompression Therapy...

- If bubbles are ***not*** 'organized', a ***mechanical reduction in volume & diameter of bubbles*** in blood vessels & tissue is possible
- ***Physically dissolved O_2*** in plasma and interstitial fluids will oxygenate ischaemic tissues through ***diffusion***
- ***Hyperbaric O_2 is a strong vaso-constrictor*** and reduces the ***edema*** in the central nervous system (CNS)
- It also ***inhibits the inflammatory reactions*** and curtails the endothelial damage
- The oxidative reactions in the tissues surrounding the bubbles reduces the total gas pressure, thus furthering gas diffusion of inert gas from bubble to tissue: ***OXYGEN WINDOW***

- In treatment depths, where 100% hyperbaric O2 cannot be used, we use **EAN**x 40/60 and 50/50 as well as **HELIOX** 50/50%.

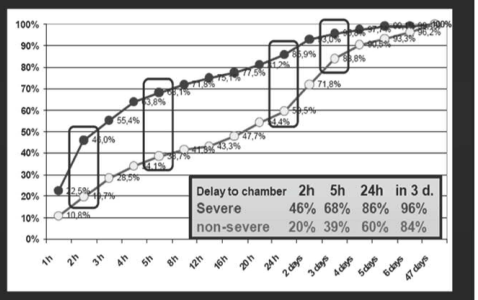
- The **proximity of a recompression chamber (RCC)** at the working place translates into a fast treatment after the first appearance of symptoms.
- **Recreational divers DCI accidents in the USA**:
  - only **24%** were treated within **6 hrs** of onset of symptoms
  - **40%** within **12 hrs**, which allows for worsening of symptoms and prognosis

In **Sharm el-Sheikh**, we managed to ***improve*** these figures for the ***severe*** accidents.

- When dive table or computer limits are exceeded, DCS cases have a plausible explanation, ***BUT 57% of all treated cases were dives made within the safety parameters of dive tables and dive computers and with a conservative gradient!*** They were ***"undeserved".***
- ***DAN-USA*** reported that the **median time** (time at which 50% of symptoms occurred) for all DCI reported cases is **30 min** and **90%** within **24 hrs**.
- ***DCI*** can be a **localized** or a **multi-system** disease
- We make a clear differentiation between **DCI cases** among the **recreational** and the **military- and commercial divers** and **compressed air workers.**

Time delay to chamber treatment

depending on severity of DCI (647 cases)



Delay to chamber	2h	5h	24h	in 3 d.
Severe	46%	68%	86%	96%
non-severe	20%	39%	60%	84%

- Cases of **DCI** among **recreational divers** show **75% neurological** signs and symptoms
- **Professional, military, commercial divers and compressed air workers** on the other hand show only **30% neurological** signs and symptoms
- *Their **training** is more disciplined, they are **medically screened** and checked by **diving medical specialists,** an **infrastructure** is present to deal efficiently with emergencies, **no extremes of age, no procedural mistakes,** well maintained **equipment,** good **purposeful training** and well established **working procedures**.*

Once the patient arrives in the RCC for the definitive treatment...

1. **Physical Examination:** General & Neurological, preferably without the effect of analgesics
2. **Review** of all the gathered **information** regarding the dive profile, the diver, medical history & medication....
3. Securing his **dive gear**
4. Establishing a **DD**
5. Matching your findings with the **available capabilities of the RCC:** personnel (available tenders), technical capabilities (treatment gas mixes, size and pressure of air bank, life-support systems, back up of O_2,...)

Once the patient arrives in the RCC for the definitive treatment...

6. Deciding **which TT** to use
7. **Preparing the patient**, physically and psychologically
8. **Carrying out the treatment** and deciding on changes in the course of treatment
9. **Documentation**
10. Contact **insurance** company
11. **Re-evaluate patient after end of treatment, repeat neuro exam and decide on further steps:** further recompression therapy, physiotherapy, rehabilitation, psychological support,...

Is it a case for Recompression?

- Medical History
- Neurological Examination
- Information gathered from dive profile study, buddy and dive computer (3 days back, if possible!)
- Examination of dive gear and braething gas analysis when needed (especially with gas mixtures in technical diving)
- Diagnosis/DD? (are further diagnostic investigations needed?)
  Cutis Marmorata... DCS I... DCS II... AGE... CAGE...
  Combined cases... other: mesenteric infarct, coronary infarct, pneumoencaphalus... etc

Physical Examination:

- ***General Examination:***
  - all ***vital signs*** are examined and noted
  - check for evidence of ***pulmonary barotrauma*** *(pneumothorax, pneumomediastinum, subcutaneous emphysema)*
  - check for ***otitic barotraumata***
  - check the whole body for ***cutis marmorata*** and/or ***lymphoedema***
  - other signs and symptoms

[redacted]

- Preperation
- I.V. Lines and substitution of volume
- Intubation ? Ventilation?
- Pneumothorax? (X-ray ⇒ chest tube)
- Haemodynamics and cardio-vascular: stable
- Adjuvant therapy: when needed!
- Inside tender and substiute tender
- Accompanying Physiotherapy...

- ***Neurological Examination:***
  - Orientation
  - Coordination & Balance & Gait
  - Cranial Nerves
  - Motor Power
  - Sensation
  - Muscle Tone & Deep Tendon Reflexes
- All ***neurological deficits*** are noted and ***documented*** together with the ***time they started*** and how they ***responded to 1st aid measures***. This is the ***neurological 'base line'*** for the case.
- *Stroke has different patterns of neurological deficits when compared to DCI.*

Preperation

„worst case scenario!"

Check the following:

1. **Emergency medication**... what goes in... what is sent through the medical lock
2. **Briefing**: breathing through the BIBS, Intercom, emergency procedures... etc.
3. **If medical equipment inside the RCC** (Siare ventilator, suction, vital signs monitor...), follow the local regulations and manufacturers recommendations!
4. **Diving eligibility examination** to all the personnel that might enter the RCC during the treatment

I.V. Lines and Volume Substitution

- **Insert I.V. Canula prior to entering the RCC**
- **Use the largest possible caliber**
- **Do not use glass containers for IV fluids or suction**
- ***Bubble-free infusion line!*** *With pressure reduction, small bubbles grow into large bubbles!*
- Infusions not using an infusion pump should be ***stopped during compression and decompression*** **phases**

Pneumothorax

- **Relieve before the compression with a chest tube** *(In an emergency, use a wide caliber canula)*
- **Heimlich-Valve (one way valve) is a must, if none avilable, then use an under-water seal!**
- ***Occlude the tube during the compression phase***
- ***Before you commence with the decompression phase: check for patency!***

Intubation

- If ventilation judged to be insufficient, then ***intubate before recompressing***
- **Remember: careful auscultation** of all lung fields is a must!
- **Caution:** ***the cuff of the endo-tracheal tube should ONLY be filled with fluid. If you receive an intubated patient, always double check the cuff filling!***
- Intubating patients in smaller RCC can be quite challanging. Auscultation would also be quite difficult due to the noise!

Cardio-vascular/Haemodynamics

- **B.P. measurements are possible with a normal cuff under pressure**
- **Palpatory method for systolic values is accurate because of the noise levels**
- 4 RCC in Egypt have **„vital signs monitors“** approved for work under pressure
- ECG-monitoring and print outs are available in 3 RCC in Egypt (SSH, HRG, ALEX)
- ***Beware of arrhythmias and signs of M.I. (coronary embolisation)!***

Ventilated Patients

- ***Always a problem for RCC‘s treating recreational diving accidents!***
- In earlier years we used the „Dräger Oxylog“ (pressure driven) – today the „Siare“
- In most remote areas, the AMBU-bag is still used
- AMBU-bag could be attached to the BIBS-Oxygen-line, preferably one with a reservoir bag
- **Caution:** ***exhaled Oxygen will raise the O_2 concentration inside the chamber environment... augment the ventilation and track the Oxygen analyzer reading!***

Cardio-vascular

- **CPR in the chamber** represents a hightened risk also to the **tender** performing it...add decompression stops or exchange tenders!
- **Defibrillation** inside a RCC? Extremely questionable and very risky. *(Special preperation and isolation needed. None of the chambers in Egypt is equipped for such a procedure)*
- **Precordial fist thump and medication (Amiodaron) are still first choice!**

Medicated Therapy

- Generally speaking, **all emergency medications are allowed**
- **Vaso-constrictors** could affect the gas elimination
- Most diving accidents arrive in a **hypovolaemic state...Volume substitution should not be delayed**
- Often used medication: Benzodiazipine with acute CNS O2-toxicity, Narcotics (Trapanal) with convulsions (CAGE), Diazepam with severe claustrophobia
- **CAVE: if convulsions ⇒ arrest ascent**, check for etiology and make sure the patient is breathing before resuming the ascent! **In acute Oxygen CNS- toxicity convulsions, the patient is not breathing!!**

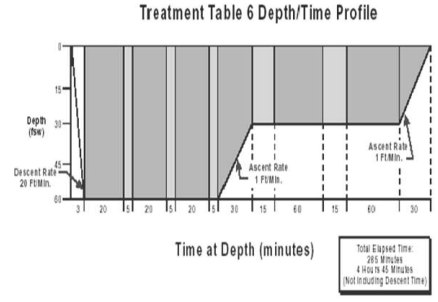
USN Air TT
COMEX Proprietary TT
Other...Russian, Italian and Australian Navy TT,...

Difficult Questions:

- **Extend** the TT or not?
- **Switch** to other, more aggressive TT or give a second traetment session?
- How many long tables can I **repeat** (USN TT6 or COMEX CX30)?
- **Go back to depth** or resume decompression and add an extension?
- What if I need to go into **saturation**?

Accompanying Physiotherapy

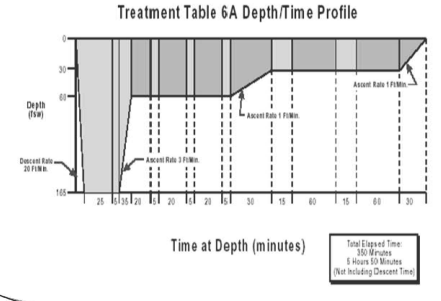
- **In cases of paraplegia and other types of paralysis we recommend that physiotherapy should start early *during* the recompression therapy.**
- **The advantages are not „evidence based", but we have scored excellent results!**
- ***Diving eligibility examination* to the physiotherapist!**



DECISION?

Recompress — **No Recomp. needed**

USN TT 5
USN TT 6
USN TT 6A modified ⇒ **according to gas**
⇒ to depth
⇒ to depth and gas
COMEX CX 30: 50/50 Heliox
COMEX CX 30, 50/50 Nitrox
HBO / **mod. HBO** (Problem wound table at 2.8 ATA)
RN TT 72, 71, ...
Catalina Regime



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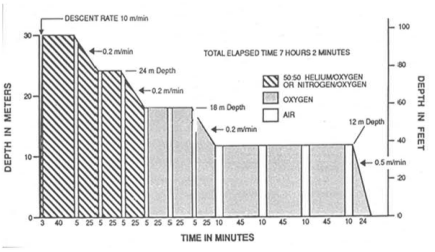

Is HELIOX 50/50% a better treatment gas when compared to Oxygen?

Our experiences with HELIOX as a treatment gas started in ***1994***.

It was the first COMEX treatment to be performed for a sport diving accident in the Middle East.

It followed the initial treatments with two long tables:

- a USN TT6A with extensions, followed by
- a USN TT6 with extensions

The case was an AGE + CAGE affecting the brain and the spinal cord.

The results were phenomenal, although the treatment started after more than 78 hours (30+48).

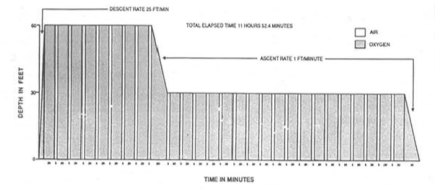

Figure 4. Catalina Treatment Table. This is a modified version of USN Treatment Table 6. All oxygen breathing cycles are of 20 minutes duration, followed by 5 minutes breathing air. In its current implementation, shorter versions of this table may be used as follows. After 3 oxygen cycles at 60 ft, a minimum of 6 cycles are required at 30 ft (equivalent to USN Table 6); after 4 cycles at 60 ft, 9 cycles are required at 30 ft; after 5 to 8 cycles at 60 ft, a minimum of 12 cycles are required at 30 ft. Up to 18 cycles at 30 ft (as shown above) can be used. Tenders must breathe oxygen for 60 minutes at 30 ft and during the decompression to the surface (total 90 minutes). If there have been fewer than 4 oxygen cycles at 60 ft and fewer than 9 cycles at 30 ft, then only 30 minutes of oxygen breathing is required for the tender at 30 ft in addition to the decompression time (total 60 minutes). Further treatments can only be started after 12 hours of air breathing at the surface. Further details of this treatment table have been published by Pilmanis. (Drawing reproduced from Moon[55]).

- Several studies were made comparing the outcome of USN TT6 and COMEX CX30 showed that ***there was no special advantage using either table.***
- We believe that these studies were not conducted in an unbiased manner and we believe that more questions should have been dealt with when constructing such studies, eg:
  - was the COMEX CX30 the ***first choice*** in the treatment or was it referred to after using other long tables?
  - what was the ***time delay*** to the beginning of the recompression?
  - how did the use of HELIOX affect the ***physiotherapy results and the prognosis*** in general?

Principles of Adjuvant Therapy

- Volume replenishment
- Support haemodynamics
- Sufficient ventilation
- Sedation, if needed
- Anti-convulsives, if needed
- Forced cerebral post-ischaemic reflow
- Lidocaine (sometimes in severe CAGE associated with severe neurological deficits, infusion pump!)
- NSAID, after the initial evaluation and start of therapy

From my lecture in the HELIOX Workshop in Amsterdam 2015:

COMEX CX30 is hardly mentioned as an option in diving medical curricula.

- Most treatments performed to manage sport diving accidents are an individual experiment by the treating physician.
- HELIOX TT are still considered only after failure of USN TT, but not as a first choice TT for specific, clearly identified cases (e.g.: progressive spinal DCI).
- A registry for HELIOX treatments in sport/technical diving accident treatment should be started and the results compared with treatments for commercial diving accidents.

- Could the beneficiary effect be more depth (pressure) related rather than gas?
- changing of inside tenders during a CX 30 is highly recommended. Holger Schoeppentau and Armin Kemmer published a poster on the subject.
- It seems to me after analyzing the clinical path and results of the accidents treated with COMEX CX 30, that **the actual value of the HELIOX is in preparing the body and tissues for further HBO sessions and physiotherapy. Often single treatments were sufficient for the cure.** The response is very different compared with cases treated with pure Oxygen treatment tables

Treatment of Persistant Symptoms following Initial Recompression Therapy

Different schools of thought:

A) According to severity of symptoms

Severe symptoms:
2x **HBO**/day at 12-18m
PWS (modified problem wound treatment table)

Mild symptoms

then 1x **HBO**/day untill a clinical plateau is reached

- "The work of Hyldegaard and co-workers in animal models indicates that at 1 ata Heliox breathing can promote faster air-bubble resolution than air or oxygen breathing" *Bove and Davis, Diving Medicine*

Treatment of Persistant Symptoms following Initial Recompression Therapy

B) Repeat Initial long Therapy

e.g.:**USN TT6** could Be repeated after 6 to 12 hrs. up to two times.

another long TT e.g.: **COMEX CX 30** (HELIOX 50/50%)

In severe diving accidents, up to *three* trials are acceptable!

If no RCC available or reachable within 12 hours:

One could consider under certain circumstances and in selected cases and provided all the necessary equipment and trained personnel and diving medical specialist are present to venture into *O_2 In-Water Recompression (O_2-IWR)*

Treatment of Persistant Symptoms following Initial Recompression Therapy

C) provided the Infrastruktur and Personnel are available:

High Pressure Treatment Tables:

- over 50 msw, mainly used in „Offshore Commercial Dive Operations"
- Suitable for dive accidents where 1st symptoms appear under pressure (in-water during decompression)

Treatment of Persistant Symptoms following Initial Recompression Therapy

- **R Navy TT 71**: max. depth 70 msw, Total duration almost 48 hrs.
- ***Lambertsen/Solus* Ocean Systems TT 7A**: max. depth is calculated as 10 msw over *depth of symptom relief*
  - symptoms that develop at pressure.
  - recompression deeper than 165 fsw (50msw)' or
  - Where extended decompression is necessary.
  - Depth limit 200 fsw for air.
- **USN TT8**: 69 msw, duration 57 hrs., used for „**Blow Ups**" (fast ascent with omittance of over 60 min decompression stops) in commercial diving

Treatment of Persistant Symptoms following Initial Recompression Therapy

D) Treating with Saturation Treatment Tables

- Used in life-threatening conditions or worsening of symptoms during ascent
- Patient remains at depth of relief until there are no symptoms (many hours up to days!)
- This is followed by very slow desaturation...
- Lookout for pulmonary O_2-toxicity! Fractionation of Oxygen breathing periods, prolongation of air breathing periods!

Treatment of Persistant Symptoms following Initial Recompression Therapy

- **USN TT7**: 12 hrs. minimum at 18msw, no set maximum, decompress over 36 hrs.
- **CATALINA-Regime**: by *Joe Pilmanis*. The alternative allowing the transition of USN TT6 to a saturation treatment table. A viable choice for RCC in remote areas treating difficult recreational diving accidents.