CHOCOLATE, SEX AND SCUBA DIVING

WHY THEY'RE GOOD FOR YOU AND HOW TO DO THEM SAFELY...

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An overview of decompression theory and how to help make your SCUBA diving safer

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SSI + NAUI Sports & Technical Diving Instructor Trainer
IANTD Technical Diving Instructor
HSE Offshore Commercial Diver Medic
IMCA Saturation Life Support Technician
SAUU Recompression Chamber Operator
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INTRODUCTION

As I sit and pencil this riveting edge-of-your-seat narrative about current day decompression theory, chocolate, sex and how they all fit together beautifully & poetically, it's literally 30-years to the day since I first learned to SCUBA dive in January 1988 at Sodwana Bay in South Africa and I've been a SCUBA diving Instructor Trainer myself now for over 23-years.

That time has blessed me well and I've travelled, adventured and dived extensively along the KwaZulu Natal east coast from Protea Banks, Aliwal Shoal, up through Sodwana Bay and towards mid-Mozambique; I've extensively dived the Red Sea from Aqaba in Jordan in the very north all the way down to the Ras Banas peninsular in the deep Egyptian south near the Sudan border; and I've also dived both the northern and southern atolls of the Maldives.

A lot has changed in that time and some things not at all…! We all have cell phones and now take those completely for granted, the space shuttles no longer fly, the Concorde's have all been grounded, Angelina Jolie divorced Brad Pitt, I've fallen completely in love with Rey in Star Wars, Mugabe has finally been unseated from power, SCUBA diving itself has changed almost not at all from the simple and crude activity of getting wet and blowing bubbles that it's always been (how hard can it be) and I've seen the diving industry go from boom to almost bust and back to the current day where it is fortunately starting to build back up in popularity with some zest.

Diving medicine, thanks to the extensive use of dive computers used now-a-days by individuals worldwide and the data that has provided to hyperbaric facilities in the face of real decompression illness events, has exponentially helped medicine get a better look at the factors surrounding those outcomes and thus helped hyperbaric & decompression specialists develop better theories around what might really cause **Acute Decompression Illness** and how that can help the rest of us dive safer.

I've always had a fascination with subsea and hyperbaric medicine... I studied paramedics in my early diving days and served as a non-commissioned officer with the training wing of an elite special forces military unit that trained combat medics and also ran the training cycle for special forces operators; it was my first experience of professional instruction at the very highest level and inspired me into SCUBA leadership.

I qualified as a recompression chamber operator lending my hand driving chambers as well as acting as an in-chamber medic treating patients with Acute Decompression Illness so got to see and experience the realities of that first hand. As a SCUBA Instructor Trainer, I largely taught anatomy & physiology as well as decompression theory (as we understood it then) to advanced level SCUBA divers as well as Divemaster and Instructor candidates.

I topped out my diving medical training in an active A&E ward at a university hospital in Glasgow, Scotland, where I qualified as a Commercial Offshore Diver Medic (Critical Care Assistant Remote Medic). I also qualified as a Saturation Life Support Technician in Fort William in the Scottish Highlands hoping to get onto the North Sea oil platforms to work with the deep-water saturation diving teams running the habitats and working as a trauma medic going to depth if there was a medical emergency with the saturation teams, although regrettably the onslaught of the recession largely shut down the North Sea oil fields and I had to fall back on my architecture to forge an income and build a career.

I've none-the-less managed to keep at it driving recompression / decompression / hyperbaric chambers wherever I could in South Africa and the UK where I lived and worked most of my adult life and kept active teaching high-level diving science & medicine wherever I could.

What all of this has fortunately permitted for me, as a SCUBA Instructor Trainer with a passion for knowledge and understanding, is give me access to in-depth medical knowledge and data around subsea and hyperbaric medicine and many of the most current hypothesis around Acute Decompression Illness, what it quite actually is, what might be the precursors for it occurring in divers, and how we can therefore help make our diving safer and more enjoyable.

Through my own ever-ongoing studies around all of this, what follows is a dissemination of the latest data as medicine understands it, which I hope you'll find

interesting, **eye opening** and **educational**. It's essentially not much more than a précis of all the books I currently have my head buried in studying the topic related specifically to decompression theory and Acute Decompression Illness.

Much of what we now currently know about **Acute Decompression Illness** turns much of what we previously thought entirely on its head…! It's also a truth that many/most/ all of the teaching material and theory used by the sports diving associations is decades out of date and urgently in need of modernisation. This text will thus hopefully help many of the active SCUBA Instructor's and Divemaster's to more clearly understand Acute Decompression Illness and how they can help themselves, their students and clients dive safer.

SO WHY PUT TOGETHER ALL THIS THEORY?

Sports SCUBA diving for the most part remains a **very safe sport**. The incidence of Acute Decompression Illness is only suggested to be at 2-5% of all dives done although the total dives done worldwide to assume this is really an unknown!?

Finding actual diving incident reports is difficult as in most countries, like South Africa, the data isn't collected or disseminated for public access. Reading the latest 2016 DAN Annual Diving Report we have available out of the USA however, there were some **484 cases** of ADI reported to DAN call centres and hyperbaric facilities and almost half of those were the very serious neurological type which have become more prevalent now-a-days due to extended bottom times enjoyed from diving with dive computers. The remaining half of incidents (skin or tissue bends) were more likely caused by rapid ascents which is a common-place short-coming in all scuba diving that I see. There additionally 448 barotrauma cases reported (also caused by rapid ascents and injury due to pressure change).

It has been found that in many cases where neurological decompression illness occurs (i.e. damage to the central nervous system, brain or spinal cord) that **full recovery does not occur without long-term residual damage**.

The diving manuals of almost all of the sports diving associations are perhaps decades out of date given what medicine now knows about decompression theory and how to help make diving safer, so the emphasis falls back onto SCUBA Instructors, Instructor Trainers and Course Directors to be the qualitative difference in that shortfall and to disseminate all the information they can between one another and down to their students.

The goal is always to try and help ensure that the divers we train and lead into the water are always diving **as safely**

as possible where they can to avoid injury and maximise enjoyment at every opportunity. I don't expect every Instructor to read these notes (although they arguably should) or have as much interest in advanced decompression theory as I do but sometimes it's the simplest things that can make the biggest difference. If you perhaps find that you lack a propensity to focus then by all means just skip to the last page and read the 13 tips provided as summary for enhancing dive safety.

At advanced diver training level we can easily spend two 3-hour lectures just taking about decompression history and development around the various theories but I've included a very précised version here as an overview as to how we have arrived at the advanced hypothesis currently suggested for enhancing dive safety.

It has essentially been found that **individual physiological factors** seem to contribute more pertinently to the risk of Acute Decompression Illness that just the simplistic previously believed and widely taught concept of bubbles forming from a reduction in pressure after a dive with insufficient decompression. Modern strategies are thus now rather aimed at how we might each improve the way we prepare ourselves for SCUBA diving in order to minimise the formation of micro bubbles.

THE DEVELOPMENT OF DECOMPRESSION THEORY *(in a VERY simplified & précised overview)*

The problem of bubbles and Acute Decompression Illness has been with us since as far back as **1841** when **Trigger**, a French engineer, designed the first pressurised underwater caissons to assist in the construction of foundations for a bridge across the Loire River near Chalonnes in France.

A succession of smart people are credited with research into 'bubble trouble' for caisson workers in the late 1800's looking for ways to understand the problem and make decompression safer but it was not until **1908** that the Scottish physiologist J**ohn Scott Haldane** proposed his famous '2:1 critical supersaturation ratio hypothesis', based on a simplistic 5-tissue compartment model (with half-times ranging from 5 -75 minutes), and developed the first ever set of tables giving working divers specific

time and depth guidelines for exposure to pressure. Haldane's decompression theories and tables formed the basis of the original **1957 US Navy dive tables**. (A half-time is the length of time it takes a particular tissue group to 50% saturate or desaturate with gas).

Haldane basically believed that each of the 5 tissue groups in the body absorbed and eliminated gas independently and exponentially and could tolerate a supersaturation pressure gradient double the ambient pressure before gas came out of solution and bubbles formed.

Haldane's hypothesis had limitations and was overly simplistic and limited, but he fuelled the progress of many contra-Haldanian theories of decompression and also deeper refinement by others of his supersaturation theory. Haldane is non-the-less regarded as the father of modern decompression theory. Decompression theory was always centred on 'fixing an end problem' (i.e. a bent caisson worker) but no one had any real idea of what actually caused a bend in the first place other than that they believed it was related somehow to 'time at elevated pressure' and 'rate of decompression' (tissue supersaturation).

In **1951 Harvey** postulated the importance of bubble **seed nuclei** in the formation and development of bubbles. Haldane's perfusion-limited model was used until the 1960's without any real fundamental

changes. The US Navy however found that Haldane's 2:1 ratio was shown to be too conservative for the faster tissues and not conservative enough for the slower ones, and the idea that each compartment should have its own allowable supersaturation ratio (rather than the uniform 2:1 ratio) was introduced.

In **1961** the Swiss decompression expert **Professor Albert Buhlmann** develops advanced tables comprising 16 tissue compartments with half-times ranging from 4 – 635 minutes. They are adapted to suit altitude diving, have reduced ascent rates, include safety stops, and are designed to allow shorter initial dives and longer repetitive dive times.

Until 1965, decompression status was calculated based largely on Haldane's initial method of comparing various individual and independent surfacing ratios – a complex and mathematically tedious process. In work around the ongoing development of the US Navy dive tables, **Robert Workman** of the US Navy Experimental Diving Unit revised Haldane's theory and defined the maximum amount of nitrogen allowed in any compartment not as a ratio, but as an expression of pressure.

With the development of his **M-Value** system Workman was able to simplify Haldane's computational process although he also added three more slower tissue type compartments into his decompression model *(An **M-Value** is a theoretical maximum super-saturation limit permitted in any specific tissue type)*.

In **1983 Professor Buhlmann** published more a more advanced concept around M-values which soon became the basis for most of the algorithms used in today's modern dive computers. He developed his own M-Value limits which are more conservative than those by Workman.

With the explosion in popularity of recreational sports diving in the 1980's, various sports diving associations produced their own versions of dive time and depth limits. With a competitive challenge to win market share and dominate the industry, some adapted their deemed-to-be-acceptable dive time limits to give divers increased repetitive dive times for resort type diving whilst some shortened their allowable dive times to reflect an attitude of enhanced safety. It became a battle around what was deemed to be 'acceptable risk'… (centred around commercialism and profitability).

CONTRA HALDANIAN THEORIES OF DECOMPRESSION

Decompression theories have been no stranger to debate and contest and some serious challenges have been made to the Haldanian view of decompression.

Also recently called into question have been many of the assumptions used in developing the US Navy dive tables (which form the basis of almost every modern dive table we know).

One of the first challenges to Haldane's theory centered around the way gas bubbles form. Haldane believed bubble formation was essentially spontaneous; occurring soon after a tissue's saturation limit was exceeded – i.e. you either had bubbles or you didn't. This concept was challenged as early as **1942** by one of the great names in decompression research, **Albert Behnke**. He deduced that small harmless bubbles probably formed gradually, developing from tiny gas micronuclei, before reaching a size that caused Acute Decompression Illness, to become known as **asymptomatic** or **silent bubbles**. As there was no technology at the time to confirm this hypothesis, Behnke's theory remained just that until Doppler ultrasonic technology first became available in 1969.

Despite volumes of research and development on the Haldanian hypothesis, some researchers believe that he was fundamentally wrong. They contend that his models have no foundation in physiological reality. Other researchers say Haldane's theory works only under certain circumstances, or only explains part of the picture.

Following experiments and reviewing of the established US Navy/Haldanian decompression tables between 1960 and 2000, a whole series of innovations and modifications were introduced. **Dr Bruce Bassett** (a USAF physiologist) concluded that the US Navy Tables resulted in an excessive incidence of about 6% Acute Decompression Illness, when pushed to the no-decompression limits. **Merrill Spencer** verified this observation and supported it with extensive Doppler monitoring, showing that bubbles developed in many routine dives – implying inadequate decompression.

Alterations to improve safety included:

- Reducing the previously acceptable no-decompression times by 10-20%
- Reducing ascent rates from 18 m/min to 9-10 m/min
- Adding a "safety stop" of 3-5 minutes at 3-5m depth

In essence all that this entire situation showed us is that modern science actually knew very little for certain about the

decompression phenomenon and what actually caused it. **Researchers in the field today still remain far from unified in either their theories or methods of explaining it.**

In an excerpt from the 2013 5th edition book **Diving Medicine for SCUBA Divers** by Drs Edmonds, McKenzie, Thomas and Pennefather:

"It is not possible to make a perfect mathematical model of the decompressing human body. Even under normal conditions, the blood flow to some capillary beds shuts off from time to time, while flow to other areas increases. During decompression, nitrogen elimination will virtually cease from an area of shutdown capillaries. As decompression proceeds, this area will have a much higher N_2 concentration than predicted. No mathematical model can predict biological phenomena such as this.

When one considers the ill-defined complexities of gas transport through the range of different tissues, all with different solubility's, different supersaturation tolerances, different nuclei quantity and location - all at different depths, durations and ascent rates - the accurate representation of bubble development with mathematical equations, models or algorithms, is impossible at this time. Even if one could so define the evolution of bubbles, there are many other problematic biochemical and haematological sequelae, as well as individual susceptibilities, in the development of ADI

manifestations, that reliable modelling is not yet likely.

Any decompression table or computer algorithm that offers longer durations underwater, deeper or more dives, will result in a greater incidence of decompression illness, unless compensated for with slower ascent rates and longer decompression.

The imminent release of brilliant "new" tables is a permanent rumour. The difficulty in producing mathematical models which truly reflect human decompression physiology upon which to base the tables or computers, as well as the difficulty and expense of testing them, makes the development and validation of tables based on truly new models unlikely.

Towards the end of last century recreational diving involved more repetitive dives and multilevel diving. It reflected the interests of recreational divers, taking diving holidays, on liveaboard boats, exploring reefs and drop-offs. This was considerably different to the pre-planned single-depth/designated-duration dives of navies and commercial divers as with much recreational diving relatively little time is spent at the maximum depth.

For any given nitrogen loading, multi-level diving from deep to shallow should produce less ADI than the single-depth dives documented in the formal dive tables. However, decompression from the infinitely variable multi-level diving is almost totally based on theory. There have been no adequate and comprehensive trials performed to show the tables reliability.

For both theoretical and practical reasons, most dive authorities advised divers to dive to progressively shallower depths, when repetitive or multi-level diving. This is referred to as a "Forward Dive Profile".

About a decade ago (early 2000's) there was a misplaced academic campaign to accept "Reverse Dive Profiles" as having equivalent decompression obligations, as an acceptable diving practice, without incurring decompression penalties. Both experimental evidence and diving experience subsequently showed that the traditional "deep dive first" concept is correct, in both multi-level and repetitive diving. *If reverse dive profiles are to be used, they will require new decompression schedules, often with longer decompression requirements."*

Traditional decompression models are sometimes referred to as **'dissolved gas models'** as they are based on the assumption that inert gas is held in solution in the tissues until they exceed their maximum supersaturation M-Values and then form bubbles. We now know that these models were faulted and that **silent bubbles do develop after every dive**.

Decompression theories called **'free phase dynamics'** were thus developed although **'dual phase bubble model'** theories, a mix of the two, also still exist. The **Varying Permeability Model** (VPM-B/E being the most current version of this) is a dual phase model with 32 tissue compartments is popular with advanced technical divers using the **Z-Planner/V-Planner decompression software** to calculate decompression stops.

There are currently around **11 known varying decompression models** each with their own conflicting theories around how bubbles form and how to control the formation and avoidance thereof. **Consensus is far from unified!**

FREE PHASE DYNAMICS

Haldanian models of decompression essentially believe that in a symptom-free diver the nitrogen within each compartment is assumed always to be

in a dissolved state. Recent biomedical research however disputes this assumption, and has given rise to a whole new approach to decompression theory sometimes called 'free phase dynamics'.

In the forefront of this research are the **RGBM** (Reduced Gradient Bubble Model) decompression algorithms developed by **Bruce Weinke**. The RGBM is a dual phase model following the logic of the VPM model.

The modern RGBM algorithms encompass up to 32 tissue compartments ranging from 1 to 720 minutes for any gas mixtures composed of helium, nitrogen & oxygen, and are particularly ground-breaking with regards to repetitive dives, reverse profile diving and multiple day diving profiles carried out by vacationing divers – a weakness in most other decompression algorithms. They incorporate theories of both dissolved and free phase gas bubbles.

In technical diving, the model calls for much deeper initial stops and slower ascents to limit micro-bubble formation and evolution. An effect of this is reductions in shallow stop times as well as total decompression times compared to schedules with traditional Haldanian decompression models.

A version of the algorithms are available in table format through the NAUI technical diving program for nitrox, heliox, & trimix diving, are included in the latest model **Suunto** & **Mares** dive computers, and the **Abyss decompression software**. They probably represent the most advanced decompression thinking available today, especially with 'air-integrated' computers that have the ability to monitor breathing rates and thus more accurately calculate for tissue in-gassing and out-gassing levels.

Despite many claims, the RGBM-based algorithms are no better at preventing decompression sickness than the Buhlmann-based algorithms, so they still don't solve the decompression problem in preventing divers from bending.

The popular **Ratio** range of dive computers use the modern (and very good) **Buhlmann ZHL-16B** decompression algorithms.

DIVE COMPUTERS & ALGORITHMS

Modern dive computers today are essentially dominated by the 50/50 use of one of two decompression algorithm theories: **Bulhmann's ZHL** 16-tissue theory or **Bruce Weinke's RGBM theory** with about the same level of ADI incidence with them both. The no-stop dive times differ widely between different brands of computer and the truth is that we still don't really know which is 'correct'. Not many divers realise that at the moment there is **no uniform procedure for testing and validating dive computers**. CE certification usually listed is only concerned with the accuracy of the depth sensor and the timer.

Dive computers have however been used extensively in sports diving for the last 25-years with a low incidence of ADI so it could be argued that their design and use has been successful. However there have been extensive reports of cases of Acute Decompression Illness on dives where divers have been **well within their dive limits** so clearly something else is going on beyond all the tissue saturation limit theories proposed.

Indeed, DAN Europe statistics suggest that around **80% of divers who got Acute Decompression Illness did not violate the recommended maximum dive times on their computers!**

The price to be paid for more repetitive dives and longer dive durations, without decompression, is increased risk of Acute Decompression Illness and by all accounts of evidence increased risk of the more

dangerous neurological ADI.

Dive computers are popular and deservedly so, but they should not be perceived as an alternative to assuming personal responsibility for understanding decompression theory and the risks involved in pushing dive limits to the maximum.

It is estimated that some 95% of sports divers now dive with dive computers, which is of course an excellent thing. Most people trust and follow their dive computers completely, however it is a known truth that the algorithms they all use are far from perfect and even the most reliable computers still accept a probability of Acute Decompression Illness of up to 5%.

One of the positive outcomes of divers using dive computers, is that when they do bend their precise dive profiles can be downloaded by hyperbaric facilities and that data can be kept for further analysis and understand of the situations that indeed commonly lead to ADI.

Having complete dive profiles permits the calculation of inert gas super-saturation levels for different tissues at different points in time (i.e. tissue M-values). Interestingly, DAN Europe Diving Safety Laboratory studies revealed that only 10% of ADI cases involved a level of 90% or more of the M-value of the fastest tissue compartment. Only another 10% recorded a super-saturation level of between 80-90% of the M-value. The remaining 80% of ADI cases involved fast tissue super-saturation levels lower than 80%...!

This suggests that there is more to decompression algorithm validation than just compartment super-saturation estimates based on depth/time profiles alone. Some divers seem to be able to tolerate 'bubbling' well in excess of what was previously considered safe limits, whilst others have developed ADI at levels well within maximum dive limits.

VASCULAR GAS EMBOLI (VGE)

As we all know, when we ascend at the end of a dive super-saturated gas starts to get eliminated from our tissue cells (different tissue types absorb and release gas at a rate dependant on the decompression theory algorithm your dive computer is assuming) forming a fine mist of asymptomatic silent bubbles in your blood plasma. The primary driver for the amount and size of microbubbles released is influenced by your **rate of ascent**.

These microbubbles form mainly at the beginning of your venous system at the point where the gas comes out of the tissue cells. As they become mobile these **Vascular Gas Emboli** (VGE) are carried in the venous bloodstream towards the heart and into the lungs to hopefully be expelled without any trouble. We can detect and measure these VGE, in size and extent, using Doppler ultrasound.

When nitrogen bubbles form too quickly, become too large and/or too numerous, they become trapped in the venous capillary system before they can be flushed away by the blood plasma and you develop what is commonly know as Acute Decompression Illness. Statistically, the presence of a large quantity of bubbles means there is higher risk of developing ADI but many divers have been observed to have sometimes very high bubble loads without any ADI symptoms at all.

It used to be commonly thought, and taught, that bubbles simply coming out of solution was the primary problem but we now know that this is not strictly true. One diver can have an excessive amount of bubbles in their system and not present with ADI symptoms whilst another can get ADI with a relatively low bubbles count. The use of decompression algorithms

and hypothesis to limit the incident of ADI now **seems limited** and current thought patterns rather seem to be moving towards dive management that minimises the production of silent bubbles (VGE) although the exact relationship between VGE and ADI remains unknown.

The production of silent bubbles has also been shown to be significantly higher after a wet dive compared to the same dive profile in a dry chamber. This is particularly important with respect to the testing of decompression models, which are increasing relying on VGE counts, and their reliability towards real in-water diving activities.

The complex physiology of decompression involves not only gas pressures and virtual compartments or other models, but also **highly variable physiological parameters** such as body temperature, hydration state and vascular vs. extracellular fluid shifts. In view of the very low incidence of ADI, DAN believes that **any major effort to improve decompression algorithms in recreational dive computers may be a costly and ultimately rather futile endeavour**, unless they are directed towards a personalized decompression algorithm that takes into account individual physiological parameters.

It may be much more useful to collect more data and run more controlled experiments to find out what influence these physiological variables have and how important they are. As a primary outcome measure, the objective and quantitative measurement of VGE seems to be a much more logical choice than tracking the incidence of ADI because it allows for a calibration and validation outcome below clinical symptoms. The aim would be to reduce decompression stress, instead of just prevent clinical ADI. As the relationship between bubbles and ADI is not straightforward, additional factors such as oxidative stress also need to be measured.

It seems the reliability limit of existing dive computer validation protocols has been reached and that the **new frontier will be to further improve our ability to customize the conservatism levels in dive computers according to physiological variables**.

BUBBLE FORMATION

What IS known at this time is that bubbles form in the body after scuba diving and that those bubbles can sometimes cause Acute Decompression Illness (although no one strictly still seems to know how or why). Dive theories and algorithms thus all similarly try to limit bubble formation and growth during decompression and after any dive. The current centre of attention is thus focussed on rather trying to understand how bubbles **initially form** and what contributing and connecting factors seem to initiate ADI.

Bubbles don't just vaporise out of nothing! For a bubble to form spontaneously in supersaturated pure water, a level of saturation greater than 100 bar is required – this is the equivalent of a sudden reduction in ambient pressure from a depth of 1,000m to the surface! This means that bubble formation by **homogeneous nucleation** is impossible within the range of human decompression.

It is commonly thus rather now believed that silent bubbles 'seed' from **microscopic micronuclei** (impurities) that we are all believed to carry in our bodies at all times (a process known as **heterogeneous nucleation**).

There are two other possible bubble producing mechanisms in our bodies: **tribonucleation** (which might occur at the edge of our rapidly fluttering heart valves) and **cavitation** (as a result of pressure reduction in body fluids).

Nanobubbles have also recently been shown to form on smooth surfaces within our tissues (even on blood cells themselves) although heterogeneous nucleation and tribonucleation are still considered prime candidates for actual bubble formation.

In any case, incorporating bubble formation and growth mechanisms into decompression models is important and research is proceeding in this general direction in an effort to make models more biophysical and allow better extrapolation. A consistent quantitative, unambiguous and reliable post-dive, venous bubble monitoring system needs to be developed to calibrate and verify experimental results.

We still do not know exactly where and how bubbles form. Neither the physics of scuba diving nor the physiological changes associated with it are fully understood. Technical diving and other dives that push the limits of the algorithms carry an even higher risk of ADI. Since the risk of ADI has been shown to be dependent on numerous **physiological variables**, research needs to focus on how individual physiological factors affect bubble number and bubble growth, so this information can be incorporated into personalised decompression algorithms.

DEHYDRATION AND HYDRATION

One of the recurring abnormalities that hyperbaric facilities have picked up in ADI patients is dehydration (hemo-concentration). It is something that is on the table for further study and hypothesising and there are questions around how immersion diuresis might affect in-gassing and off-gassing. Variances in hydrostatic pressure between the body core and the arms & legs, as well as wearing a tight wetsuit, pushes fluid towards the vascular compartment and creates a fluid shift (which is what drives us to need a wee on dives). Vascular hydration is a good thing during off-gassing at the end of the dive as it may aid with decompression and flushing silent bubbles however there is a reversal of the fluid shift once we surface which leads to a hemo-concentration and a reduced ability to flush saturated nitrogen unless a diver takes on extra fluids straight after a dive.

Asymptomatic silent bubble production (off-gassing) peaks at about **30-minutes after surfacing** so drinking that bottle of water straight after getting back onto the dive boat really is a great idea (essential). The guidance for maximum dive safety would be to drink **500ml of water before your dive** (slowly) and **another 500ml straight afterwards** to help with maximum hydration of your body and a reduced risk of ADI. The best way to stay hydrated before and after a dive is to drink a little at a time, say a cup of water every 15-20 minutes. Drinking a large amount of water too fast will increase diuresis (your need

to wee) and will not hydrate your tissues. Keep a bottle of water in our car as you drive to your dive site and sip on it slowly as you're driving.

ASYMPTOMATIC SILENT BUBBLES

Studies from as early as the late 1960's using Doppler ultrasound testing showed that divers who did not have ADI still had bubbles in their blood vessels after diving. This challenged the Haldanean models which were based on the assumption that bubble formation only occurred after the tissue M-value was exceeded. Because the divers tested had all dived within their allowable dive limits and showed no signs of ADI, these bubbles were referred to as 'silent' or 'asymptomatic' bubbles.

Typically, it was thought that silent bubbles did not interfere with normal body functioning and were simply breathed out over time unless any of them grew together

or stuck anywhere to create a larger problematic bubble and symptomatic ADI.

The presence of silent bubbles in the blood, which can take well in excess of 24-30 hours to completely eliminate post dive, however influence repetitive diving and are needed to be taken into account with multi-dive or multi-day diving events.

The presence of silent bubbles are also linked to **post dive fatigue**. The lymphatic system in our bodies attacks these microscopic bubbles after a dive, seeing them as foreign objects in our blood stream, and in the same manner as how we feel drained when we have a cold *(men of course get a more severe version of this known as man-flu which can be completely debilitating)* we can feel drained after a dive or after a series of dives.

This is one of the most valuable benefits of diving **Nitrox** because the resultant reduced total nitrogen in-gassing on any dive thus equates to less silent bubbles being in our blood streams after any dive, and thus permits us to feel more energetic.

Everyone should be diving on Nitrox by default. Dive resorts should perhaps fill cylinders with Nitrox by default even for non-nitrox qualified divers and rather perhaps permit them to dive air-profiles on their dive tables or dive computers for the added safety that provides (as long as depth limits are controlled by the Divemaster for the Nitrox mixes being used).

The presence of silent bubbles is also a reason why, contrary to popular belief, that **reverse profile diving is ill-advised**. If silent bubbles are present in the blood following a previous shallower dive, then on any subsequent deeper dive those bubbles can be compressed and may be forced through the lung filter into the arterial circulation, which could cause arterial gas embolism (AGE). This is potentially **more dangerous than a bend**, because bubbles, or free gas, can be fed directly in to the central nervous system.

Symptoms of AGE usually present earlier than with a bend, within minutes of surfacing and may even show during ascent. It is crucial therefore to plan your diving so that the first dive of a series is the deepest and all others are progressively shallower.

Research debated at the "Reverse Dive Profiles Workshop", conducted at the Smithsonian Institution in Washington D.C., in October 1999, resulted in the conclusion that in fact reverse profile diving was perfectly acceptable as long as the generally accepted 40m depth limit was not exceeded and that any repetitive dive was not more than 12m deeper than the preceding dive. **More recent findings, as recent as 2013, have however disputed that.**

All dives cause the generation of silent bubbles. Even an ascent from as little as 7 meters can result in silent bubbles. Relatively fast ascents, saw-tooth and yo-yo dive profiles may present sufficient silent bubbles to clog the lungs. This reduces surface area and limits gas exchange. In these circumstances nitrogen-laden venous blood can be returned into the arterial circulation, in what is known as an **intrapulmonary right-to-left shunt**.

The number and size of silent bubbles produced are affected predominantly by the rate of ascent, and safety stops. Your MAXIMUM rate of ascent should **never exceed 10m/minute**, and you should always hang a **safety stop for 3-5 minutes at a steady 5 or 6m depth**.

The best way to ensure a steady out-gassing depth on your safety stops is to buy & have a deploy buoy, deploy that on ascent, and clip that onto your buoyancy compensator at the end of a dive so that you can hang off it at a steady depth (but keep relatively active to ensure optimal blood circulation).

ADAPTION & REPETITIVE DIVES

Repetitive and recent exposure to increased pressure has been shown to reduce the likelihood of ADI *(what more motivation do you need to get diving every week!?)*. It is currently believed that frequent diving removes or crushes the naturally occurring gas nuclei that are precursors to bubble formation. Regeneration times for these classes of micronuclei are estimated to be around a week or two, so a lay-off of this period puts you back at square one.

Bubbles are also 'crushed' by increasing pressure because of Boyle's law, and then additionally shrink if gas diffuses out of them. Divers can significantly increase tolerance against bubble formation, and therefore ADI, by following three simple practices:

- Make the first dive of a repetitive series a deep, short (crush) dive, thereby constricting these micronuclei down to smaller, safer size.
- Make succeeding dives progressively shallower, thus diving within crush limits of the first dive and minimising excitation of smaller micronuclei.
- Make **frequent dives** (i.e. every other day), thus depleting the number of micronuclei available to form troublesome bubbles.

The mechanics of bubble behaviour are fairly complex and many factors have only recently been quantified. **It is important to accept that quite how bubbles exactly form and what instigates these is still not clearly understood.**

It's also food for thought aimed at dive centres and their Divemaster's that just because they can dive aggressive singular and repetitive dive profiles, get away with reverse profile diving, and swim around the reef at excessive speed trying to see everything there is in the ocean on a single dive, this doesn't mean that their customers (who aren't diving as often/daily/weekly) can do so as safely and they may be putting their customers at risk by doing so themselves on guided dives!

VASCULAR ENDOTHELIUM AND DARK CHOCOLATE

Most people probably don't even know what their vascular endothelium is despite that it's the bodies second largest organ by surface area. It is a single layer of cells that completely covers the inner surface of all the blood vessels and organ system in your body. It forms the barrier that is strategically located between the blood circulating in your body and your various body tissues and governs the well-being of your entire circulatory system. Amongst much it vitally regulates the permeability of the vessel walls and how in-gassing and out-gassing is though to occur.

One of the constituents of our blood that keeps the functionality of the vascular endothelium in balance (amongst many of course) is **nitric oxide**. Endothelial dysfunction is associated with a number of pathological conditions such as diabetes, atherosclerosis and heart disease.

In studies of ADI there is a common association of fluid leaking from blood vessels in a condition known as increased vascular permeability. Severe ADI has also been shown to cause severe damage to the endothelial cells themselves. Studies of healthy experienced divers in both controlled hyperbaric chamber environments and open sea dives revealed a decrease in the dilation capacity of the brachial artery in all subject divers, despite that no clinical signs of ADI were present, suggesting that even standard safe diving might produce acute sub-clinical levels of endothelial dysfunction. Constriction and dilation of blood vessels are regulated by small muscles in the cell walls which react to nitric oxide produced by the endothelial cells.

Decompression stress seems to impair vascular function and although silent bubbles are frequently detected in divers and do not necessarily cause ADI symptoms, the bubbles still act like foreign objects and somehow **exert stress on tissues**. In one laboratory study it was indeed shown that endothelial cells were damaged by mechanical contact with air microbubbles. Hydrostatic pressure and oxygen both seem to also play a role in this, but not nitrogen, so it is postulated that it is in part this action that may be contributing to the initiation of ADI rather than the actual bubbles themselves. There is currently however no definitive conclusion

about the way in which bubble formation impacts on vessel function or vice versa.

Anything that interferes with the bodies ability to freely circulate blood and flush saturated nitrogen from tissues is going to be a bad thing - whether that is due to a reduced ability of the blood vessel walls to diffuse out nitrogen or a constriction of peripheral circulation!

Studies on skin microcirculation, related to vascular stress by silent bubbles, were also performed on divers and again impairment was noted with a peak immediately after diving. A variety of theories have been raised to explain the decrease in blood vessel dilation ability and one of these is that oxidative stress is involved, which is a **state of imbalance between oxygen free radicals and antioxidant activity**.

It has been postulated that this can partially prevented by **consuming anti-oxidants two hours before diving** and there is scientific evidence that, because of its anti-oxidant properties, that eating **30 grams of dark chocolate** before a dive partially prevents this decrease in endothelial dysfunction. The **flavonoids** in the dark chocolate seem to be the key ingredient, generating nitric oxide secretion and decreasing platelet adhesion two hours after ingestion which seems to help bubble stability.

The information we now have available suggests that when ADI strikes the

impairment of vascular function might just be another link in a chain that also includes oxidative stress, circulating microparticles and white blood cell activation.

Microscopic silent bubbles are also prevented/minimised by the administration of nitric oxide. Both bubble formation and ADI seem to be moderated by the presence of nitric oxide from the vascular endothelium. Theoretically then, strategies aimed at increasing nitric oxide production should decrease bubble formation and help prevent Acute Decompression Illness but the jury still seems to be out in the medical fraternity about how it is all interconnected at this time.

EXERCISE AND DIVING

An aerobically fit diver has a lower risk of developing Acute Decompression Illness than an unfit diver as they seem to be at lower risk for vascular bubbling although

the case for why is not yet clear.

An accidental occurrence with experimental studies in rats have shown that a single bout of high-intensity exercise 20-24 hours before a simulated dive **significantly decreases bubble formation** and a subsequent human study has supported this data. In two further studies, it was also shown that a single running session of 45-minutes at 60-80% of the subject's maximum heart rate performed 2-hours before a dive also led to decreased bubble production.

No one however seems to know yet what the optimal timescale is for doing pre-dive exercise and it is not clear why it should have this affect. It is thought that (again) **nitric oxide production**, which increases after exercise, might play a role and be connected to vascular endothelium function. Nitric oxide causes the blood vessels to dilate, possibly helping the body eliminate gas nuclei before they turn into bubbles, but no one quite seems to have precise answers.

Interestingly, **30-minutes of whole-body vibration** before a dive has also been shown to medically reduce the quantity of bubbles produced after a dive. It is thought that the vibration of a dive boat travelling at speed could induce changes in blood flow, as well in the endothelium, resulting in the elimination of **gas nuclei** that are the seeds that bubbles are though to grow from. It is also thought that vibration could increase lymphatic circulation, thus removing some gas nuclei in intracellular tissues. In a South African context (where I currently do most of my diving) that might actually mean that a long bouncy boat ride out to the Aliwal Shoal reef system could in fact rather contribute more towards a safer dive than a short ride out to the coral reefs at Sodwana Bay?

Running on a treadmill for 30-45 minutes seems to yield better results for dive safety than say riding a bicycle and they suspect it's to do with the whole-body vibration caused by the impact of footfall. It is hypothesised that the **vibration** may reduce the micronuclei that are thought to be the seeds for bubble birth.

In light of it all, the take home message for adult divers might indeed then seem to be that vigorous high-intensity sex intermixed with eating dark chocolate may indeed be the solution to avoiding Acute Decompression Illness after all…!? Chocolate, sex and scuba diving… who's going to argue against that…!? (It's not strictly what the books say but it seems like a great deduction?)

Swimming about moderately on your safety stop at the end of a dive, rather than hanging immobile, has also been shown to be beneficial in elevating circulation and helping with off-gassing.

DEEP STOPS

Some (argumentative) theories were developed by **Richard Pyle** around the use of deep stops to aid decompression and reduce silent bubble formation which seem to correspond with the pattern of ascent used in the respected VPM decompression dive profiles.

Deep stops are nothing other than an additional safety stop of between 1-3 minutes at the half way point on your ascent between your bottom depth and your safety stop. So if you'd been diving at say 35m you would stop at 15m depth for a minute or so before continuing your ascent up to 5m to perform your 3-5 minute safety stop.

Most dive computers on the market now have deep stops included in them (certainly any based on the **RGBM algorithm** models of decompression) although they can mostly be switched on or off dependant on diver choice; they are included in some of the advanced **Suunto**, **Cressi**, **Dacor**, **Zeagle** and **Mares** computers that I know of.

The computers will sound an alarm at the half way point of ascent to suggest the deep stop to the diver although it seems the **Mares** and **Dacor** computers will only suggest deep stops if you got to within 3 minutes of your no-decompression time dive for the depth you were diving at.

Opinions on the effectiveness of deep stops remain varied and heavily arguable in the diving community. Some suggest that they rather add to your decompression stress rather than reducing it but many divers who do the deep stops do report feeling better post dive (less fatigue, which can only mean less silent bubbles).

Deep stops are **not recommended** as necessary for shallow diving above 20m and are rather generally/historically incorporated on deeper dives beyond that and in technical diving. Some of the dive computers will only activate the deep stop function automatically when the 20m threshold has been exceeded.

The argument against still suggests that a SLOW ascent rate and hanging a safety stop is **sufficient** for recreational sports diving, and that the extra few minutes you might have added at depth would be better served by increasing your safety stop time from 3 to 5 minutes at 5m.

NAUI and **PADI** have both adopted the recommended use of deep stops on dives. So, although the jury still seems to be out on them it is valuable to note that the most recent DAN injury reports suggests that around 50% of all ADI cases are the more serious neurological type affecting the central nervous system and often leaving permanent injury.

Deep stops are generally now widely accepted to **be bad for deep and/or technical diving** as they rather fuel on-gassing of the slow tissue types whilst we're delaying at depth. It is believed to be better for decompression to get shallow as soon as possible (within acceptable ascent rates) to drive the pressure gradients to enhance off-gassing.

With a typically overly rapid ascent rate (that I frequently see divers do), a shortened safety stop and a thereafter explosive

ascent to the surface, divers can be on the surface from a 30m odd dive well within 5 minutes… well short of the time required to safety eliminate the gas that might cause a serious bend. Blood has a 5-minute half-time so it would just clear safely but the slightly slower tissues (ie spinal chord) would still be at risk. If you ascend at 10m/minute, hang a 5 minute safety stop, and still take a full minute to ascend from the 5m stop to the surface, you'd spend 9 minutes outgassing all the tissue groups (from a 30m dive) which would be substantially better erred in your favour.

There is suggestion in decompression circles that the faster tissues groups can rather tolerate higher supersaturation ratios than previously thought, as high as 4:1, but nothing is certain in diving medicine and everything we know is rather based on theory and postulation.

The very nature of our diving habits now-a-days with dive computers is to push our dive depths and times to the maximum often at 2-3 times the bottom times previously recommended as safe by dive tables! So if you are going to get a bend, it is more likely than not going to be a bad one, so what better motivation than to err on the side of safety and include all the small things that rather play the odds in your favour!?

TREATMENT FOR SUSPECTED ADI

Experience, and scientific reports show that some symptoms of decompression sickness arise more often after certain dive depth-time profiles than others. Articular pain symptoms in limbs, for instance, are more frequently observed after long dives to relatively shallow depths of 15 to 20m, often after exercise involving the limb in question.

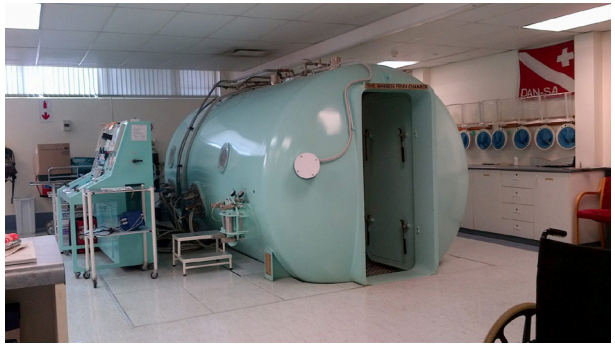
On the other hand, lower spinal cord symptoms, such as paralysis and bladder dysfunction, seem to arise more often after deeper (40m) bounce dives. However, while these observations can be attributed to the saturation-desaturation behaviour of the affected tissues, they cannot be relied upon blindly.

Any symptom that presents itself within hours after surfacing from a dive with a certain degree of inert gas saturation should be considered as decompression-related unless another, more obvious, cause can be pinpointed. In some ADI cases, symptoms have not appeared until 24 hours or more after a dive.

Diving accident first aid is taught by DAN and many other agencies and organisations - it is not complicated.

- The first priority always is to check and control the ABCs: airway, breathing and circulation.
- Phone the DAN call centre if Decompression Sickness is suspected.
- Then, administer **100% oxygen** with the patient in a resting position and give them fluids to drink if they have no abdominal pain, nausea or vomiting and are able to freely urinate (water or electrolyte fluids can be given but **not acidic drinks** i.e. orange juice or diuretics).
- If a suitably medically trained person is present then they may administer fluids via an IV drip.
- Finally, evacuate the patient to a hyperbaric facility.

Contrary to popular previous belief, **Aspirin** as a first aid measure has **not been demonstrated to be of any value in treating ADI**. It may rather interfere with blood clotting and cause haemorrhaging which is already a major risk problem if the diver has spinal cord or inner ear decompression illness. Joint pains can rather be significantly eased without risk of serious side effects by the administration of



1,000mg of paracetamol.

The actual recompression treatment administered to a diver with ADI may vary considerably depending on local circumstances, technical limitations and the experience of medical staff. It is very difficult to compare the outcomes of different treatment tables, as ADI is relatively rare and each case is different.

Therefore, there is no point discussing what constitutes the best hyperbaric treatment. All you can say for sure is that recompression treatment with oxygen is better than no recompression treatment at all or recompression treatment using air only. And any recompression treatment should be carried out in a safe way that does not involve further risk to the patient.

If you know what you need to provide first aid in cases of suspected ADI, you can make the necessary preparations. You need basic life support equipment as well as sufficient oxygen and fluids. You also need to have a means of emergency communication and know how to get an injured diver to the nearest hyperbaric chamber as quickly as possible.

Really, all this should be part of the preparation for any dive. Nevertheless, many divers choose to dive in places and with operations that have none of these preparations in place. Statistically, on most dives there are no problems, but, once in a while, something will go wrong and someone will suffer.

If a patient has mild ADI and their condition does not deteriorate after 24-hours then the risk of further deterioration is considered virtually non-existent. This means that in cases with no neurological impairment or symptoms such as shock, the effort, risk, and expense of evacuation from remote dive sites is probably not justified.

Even in urban areas, statistics compiled by DAN and other teams show that fewer than 50% of patients with ADI are recompressed **within 6 hours** of symptoms appearing. Studies in Europe, the UK and France have all found that there was no difference in outcome if a diver was recompressed within 6 hours or later.

It is suggested that once ADI is present, it rapidly triggers biochemical and biophysical reactions and becomes a systemic disease (of the circulatory system) rather than just a bubble disease that is

treatable with hyperbaric therapy.

Generally speaking it seems that about 30% of all divers who get ADI suffer some sort of residual outcome even after full and timely hyperbaric treatment.

Some forms of ADI produce more long-term effects than others… inner ear ADI appears to cause permanent damage and neurological ADI often does not heal completely.

Acute Decompression Illness, although relatively rare, is not a great thing to get so the advice is rather to do everything you can to tip the odds in your favour of not ever getting it at all.

ONSET OF SYMPTOMS AND SEVERITY OF ADI

The clinical features of ADI are seen during or soon after ascent. In the majority of cases, symptoms will be evident **within six hours**, and 50% within the first hour of the dive. Less commonly, a delay in onset of 24 hours or greater has been described. Divers often deny the reality of their symptoms, or will rationalise them. In one large series, only half the divers requested assistance within 12 hours of the first symptom developing.

Dive professionals may have financial reasons not to seek treatment, as someone who is diagnosed with ADI is usually advised not to dive for some time afterwards and

no work means no pay. In 2000, data from questionnaires returned by 740 recreational divers, 365 professional divers and 112 saturation divers in Norway found that 19% of the recreational divers, 50% of professional divers and 63% of saturation divers had experienced symptoms of ADI that they never reported nor sought treatment for. This showed that the true incidence of ADI was almost 10 times higher than the official figure assumed. This was due to a wait-and-see attitude or deliberate non-reporting.

The time of onset of symptoms depends to some degree on the type of dive. Deep dives (greater than 30 metres), especially those that require decompression or are close to the no decompression limits or in which decompression has been omitted, are likely to present early. In extreme cases, symptoms may present during ascent or at the decompression stops. In general, the earlier the symptoms, the more potentially serious the form of ADI.

feel alive, learn to dive !

HOW TO ENHANCE DIVE SAFETY:

1. Pre-dive aerobic endurance exercise
2. Pre-dive **hydration**
3. Eat **30 grams** of dark chocolate 2 hours before a dive
4. Pre-dive vibration (long boat ride)
5. Everyone should be diving on **Nitrox** (even air profile divers)
6. Always dive a **deep-to-shallow** dive profile (same applies to repetitive dives)
7. Stay well within your dive computers **maximum dive time** limits
8. Dive with integrated transmitter dive computers that monitor actual breathing rate to aid with calculating more accurate in-gassing and decompression requirements
9. **Children** should dive to more conservative maximum dive time limits than adults! They should certainly ascend slower and be more mindful of doing that (essential) safety stop at 5m
10. Slow ascent rate: ideally 10m/minute **(the most critical component of dive safety)**
11. **Safety stop**: 5 minutes at 5m (shown to be an essential component for pulling back shortcomings in pushing dive limits and overly fast ascent rates)
12. **Post dive hydration** on dive boat (drink the water!)
13. Take it easy & rest for at least **2 hours** post dive (more depending on how aggressively you pushed your dive limits and ascent rates)

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