Bubble Stories

By JP Imbert

"You are wrong" said Philip James. "Me wrong!" I was at the time the Safety & Diving Manager of Comex and had designed all kinds of decompression tables including deep heliox saturation and fancy excursion procedures. Non-linear, second-degree differential equations were my cup of tea and I was running programs which would take a night to simulate an hour of decompression. This does not allow for modesty. I would accept polite remarks on my decompression modeling but nothing like a confrontation. Philip, I should say Dr. Philip James, had recently been appointed as the Company Medical Advisor and his task was not easy.

At the turn of the 80s, the diving industry had to drastically change its way of operating in the North Sea. There were platforms, pipes, flares, and loading buoys all over the place. The time of large juicy construction contracts were gone and the only jobs left were to maintain the existing structures. This does not mean that there was no work because all structures at sea tend to rust, fatigue, and crack. Not to mention trawlers that drop anchor on expensive subsea structures or a German submarine that ran into bracings of the Oseberg platform. However, wave action, oxygen corrosion, marine life and supply boats tend to act shallow and the whole character of commercial diving changed from sea floor work to near the surface activities.

In terms of operations, this meant that the industry had to do less saturation bell runs and more air-supplied dives. Saturation diving had been improved over the previous decade and had become a vary safe and efficient way of intervention. As a consequence air diving had been considered as a secondary way of intervention and left aside without any further consideration. For this reason, when the industry started using the available air tables intensively, it began treating divers for decompression sickness (DCS) as never before.

How safe is a safe decompression

Let me recall that the safety of decompression tables is defined in term of the risk of DCS occurrence per dive exposure. DCS symptoms cover a wide span of problems ranging from skin rash to articular pain and neurological symptoms which for operational reasons have been classified into two categories, type I and type II DCS.

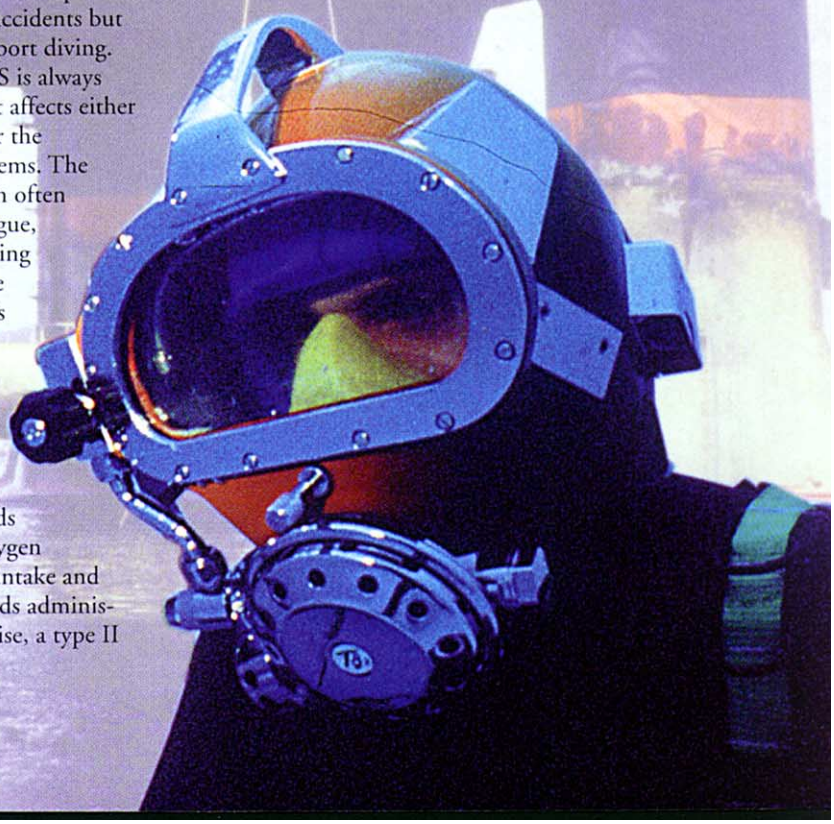
Type I includes simple symptoms like skin rash or articular pain. Because the symptoms are obvious, they are reported early and the treatment is initiated without hesitation. In most cases, administration of hyperbaric oxygen at 40 feet (12m) will rapidly clear the symptoms. Safety wise, a type I DCS is a "good DCS" because the diagnosis is easy, the treatment is applied rapidly, and the symptoms are treated efficiently. Technical divers should not be afraid of type I symptoms. It hurts at the time but it has little consequence for their diving career once it has been treated. In fact type I is typically a problem in commercial diving (long bottom time, heavy work) where it builds up to 2/3 of reported diving accidents but remains rare in sport diving.

Type II DCS is always serious because it affects either the respiratory or the neurological systems. The symptoms, which often only include fatigue, headache, or feeling unwell, are vague and the diagnosis may be difficult at an early stage. The treatment is complex and requires deep recompression, significant periods of hyperbaric oxygen breathing, fluid intake and sometimes steroids administration. Safety wise, a type II is a "bad decompression accident" because the diagnosis may not be easy, the treatment is often delayed, and its efficiency depends on the circumstances. Technical divers should consider type II DCS as their main risk because its consequences can be dramatic.

Deeper into modeling

All the classical models for decompression have adopted a general approach to DCS. The problem is considered as a whole and type II symptoms are regarded by some as merely an aggravation of type I symptoms (fig 1).

These assumptions are the basis of present decompression table calculations although a large variation exists in the gas exchange models and in the criteria used to control bubble formation. These models cannot be denied a certain efficiency since the present commercial diving tables have an overall safety record at around 0.5% bends incidence. However, it is easy to show their limits by quoting numerous



and Theories

accounts of "undeserved accidents" where people did the right thing and got bent.

To be creative you have to remember that models are just a simplified way of representing the reality. Models are like human beings. They are born from unexplained data that initiate a fresher view of the problem. They reach maturity after being checked against new data. Models also bear their own cause of death as one day, one of their predictions will fail to match experience. This shortcoming will define their domain of validity and new models will have to be proposed beyond the frontier. However, models can survive their contradictions if they are kept as a local explanation, as for instance linear and quantum optics. It is thus reasonable to attempt a multi-model approach when considering different aspects of the problem. It is also a granny's rule to cut the piece into smaller chunks when it is difficult to swallow.

The first idea of Phillip James was that we should return to the old classification and study DCS through its various manifestations. A nice structuring idea that required a different scenario for each of the symptoms. Let us start with the easy one.

When bubble volume is critical

Type I can be explained without being too mean to the classic theoreticians because the onset of articular pain fits perfectly the predictions of Critical Volume Assumption. This hypothesis was published by Hennessy and Hempleman who found a linear relation between the initial saturation depth and the depth to which divers could safely ascend. They linked the symptoms to the critical volume of the gas phase generated during the ascent. It is a sort of Haldane's critical ratio concept revisited for the benefit of type I symptoms only.

However, the important discovery is that only one tissue is critical. It corresponds to the tendons and the ligaments wrapping the articulations. Such tissues are very dense and strong, as you have already experienced when carving a roasted chicken. They are also very innervated and if you were to knock your elbow on a wall, you know it would hurt painfully.

Philip James has told me that during WWII, the Flying Fortress crews were suffering from articular pain because the plane had to fly at a very high altitude to avoid the German fighters and their cabins were not pressurized. In experiments in an altitude chamber at Farnborough, the immersion of an arm in mercury bath would relieve the pain, certainly by recompressing the bubbles which had appeared around the articulations.

The critical volume model can be handled mathematically without difficulties and I used it at Comex for designing air diving procedures which later became the French official air tables in 1992. However, because of the assumptions introduced, I must admit that these tables are limited to the prevention of type I DCS. It might seem provocative as they are still good tables but let me explain the reason. If you accept the idea that gas dissolved in tissues is only relevant when it comes to articular pain, then decompression tables which only track the amount of gas in the various compartments can only prevent type I DCS. Who cares of type I? To be definitively blunt, I should add that classical tables only work at preventing a problem which is not a major concern to technical divers.

Compartment models are just "has been"

If you are not ready to give away your confidence in decompression models based on multiple compartments, think in term of tissue gas load and try to imagine what could be the worst tissue during a decompression. The answer is the fat on the belly. It grows with age and drinking and is our largest reservoir of dissolved nitrogen. Well, have you ever heard of a bend in the belly? Not me. Despite the fact that there is a large amount of gas available and that bubbles certainly grow during decompressions, this tissue is irrelevant because it is soft and poorly innervated and fat belly bubbles have no consequences.

Models are just models and there is no point in defining the heresy but there are people who believed so hard in tissue compartments that they can give them a mind of their own. I once attended a

Comex divers conducting standard repairs on offshore oil rig.

conference at Aberdeen where Dr. Bulhman came and declared in front of the commercial diving community, which at the time had become very worried about air diving safety, that his tables never produced accidents. As the audience was recovering from this questionable statement, the chairman viciously recalled that he himself had published cases of recompression treatments. Dr. Buhlman then went on explaining that these cases were related to the 10-minute compartment but that he had changed its coefficients and that his tables were now perfect. If you can buy this dogmatic approach, it might relieve your anguish about DCS. The point is that present compartment models only cover part of the problem. They are not bad, they're just limited. Another piece had to be added to the puzzle.

More about commercial diving and theory

That summer, Comex had a series of work sites in the southern part of the North Sea, that is relatively shallow, and intensively dived on air using surface supply decompression techniques. A diver got a hit, and then a second, and then a third and I did not know what to do. I had used up all my tricks like recalculating tables for more conservative coefficients or lengthen oxygen breathing periods. On site, the diving supervisors went into heavy "Jesus factoring" by selecting longer bottom times or deeper stops. Nothing worked and the client started to put pressure upon us.

Philip, who treated the cases, had been working on microembolism. Although it was recognized many years ago that the lung not only exchanged gases, they also filtered the blood, this concept had been forgotten. Lungs as bubble filters! It came back to him in a strange way. Phillip had moved his office and he found himself one morning in his new place, waiting for his crates to come. While looking at the bare walls, dust on the floors and a rusty file cabinet, Phillip spotted a brain specimen in a plastic pot from a patient that his pathologist predecessor had left on the top of the cabinet. It appealed to Phillip that the brain was from a DCS case. It turned out that the man had died from a car accident. The emboli that had affected his brain were not bubbles but fat from the fractures and soft tissue injuries. The droplets of fat had entered the circulatory system, traveled through the lungs and carried in the arterial blood the brain. So here was the parallel to arterial bubbles in DCS (see fig 2).

When bubble diameter is critical

The contribution of the arterial bubble model is to provide a separate explanation for the onset of type II DCS. It is nice to know that the very idea of arterial bubbles can be tracked to Haldane himself. Philip showed me the following text on page 352 of Haldanes 1908 publication:

"If small bubbles are carried through the lung capillaries and pass, for instance, to slowly desaturating part of the spinal cord, they will increase in size and may produce serious blockage of the circulation or direct mechanical damage."

Then after, the Haldanes idea became perverted and only the tissue and critical ratio concept was remembered, but the basic idea was already there. During the 80s, the arterial bubbles were detected and their possible role discussed by the scientists running Doppler detection studies. Arterial bubbles were used to discuss the cerebral perfusion deficit in divers who had symptoms mainly referable to the spinal cord and the possible role of heart shunt in the divers susceptibility to type II DCS. Finally, Tom Hennessy, the designer of the BSAC 88 tables, published all the physical aspects of the arterial bubbles in a remarkable paper which unfortunately has remained confidential as it was presented at Trindheim and nobody reads proceedings of Norwegian conferences.

The beauty of the arterial bubble model

The first merit of the Arterial Bubble Model is to introduce variability through the lung function. It is reasonable to accept that the filtering capacity of the lung may vary from person to person. Some filters are better than others. Thinking in term of filters, we understand why some factors lake age, physical fitness, smoking, fatigue, and hang over, may have an influence over DCS susceptibility.

It also permits us to speculate on the possible role of CO_2 in the onset of neurological symptoms. You only have to figure out that CO_2 decreases the performance of the lung filter. Then think of all the situations that create CO_2 retention like stress, anxiety, hyperventilation, hard work, cold, and poor regulator performance. All these factors have been known to increase the risk of DCS for many years.

Finally, the most important contribution of this model is to explain how pressure variations during dives may affect the decompression safety. Assume

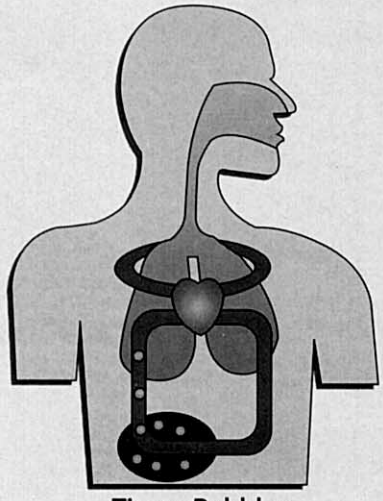


Fig 1 The classical approach

- **Diving requires compressed air breathing and causes nitrogen to dissolve in the divers tissues. The critical issue is the amount of nitrogen stored in the tissues (dose) prior to the ascent.**
- **The primary insult is bubble formation during the ascent. DCS is considered as a whole. Limb bends and neurological symptoms are seen as different levels of severity of a general problem.**
- **The sites for bubble formation are the tissues or on the venous side of the blood circulation but none is specifically identified and a series of "compartments" is considered.**
- **The decompression strategy consists of managing the amount of gas dissolved in the tissue to control bubble formation and to avoid DCS during ascent.**

you were decompressing with some nice little bubbles stuck in your lung filter (so far so good) but suddenly re-immersed. Boyle just did not have a good idea, he found a law. The recompression will inevitably produce smaller diameter bubbles which might pass through the lung filter and become responsible for the occurrence of type II symptoms.

Lessons learned from the Arterial Bubble Model

Normally, a diver is committed to keep a constant depth and follow a square profile which corresponds to the assumption used to calculate and validate the decompression schedule. In practice, we know that the diver may perform repetitive ascents and descents between levels, or when in shallow waters, even repetitive ascents to the surface. These depth variations are termed "yo-yo diving." Yo-yo diving may kill you, bubbles with a smaller diameter may pass through the lung capillaries and dump into the arterial bed. Once arterial bubbles are formed, the rest of the scenario is known, but what is critical in yo-yo diving is that the process does not require much gas stored in the tissues. This explains why severe type II DCS cases have been reported in shallow diving operations, even in the no-stop decompression range.

Open sea divers should also avoid the 10 foot (3m) stops and accumulate their stop times at 20 feet (6m). I found that the 10-foot stop is difficult to perform and I don't like the slight pressure changes at a depth where Boyles law is quite effective and the lung likely to be involved in the process of filtering bubbles. Moreover, the 10-foot stop becomes quite uncomfortable when the sea is rough and divers may be tempted to perform a Valsalva. A Valsalva in decompression is a nasty thing. It induces a significant lung over pressure and may produce arterial bubbles as "water is squeezed out of a sponge."

However, the best demonstration of the Arterial Bubble Model came from commercial diving and surface decompression. Surface decompression is a standard technique in the North Sea where divers directly ascend to the surface at the end of their bottom time and are immediately recompressed to 40 feet (12m) into a deck chamber. The advantages are obvious in cold and difficult seas, because the diver is safer and much more comfortable in the chamber than in the water. However, accenting to the Arterial Bubble Model, the consequences are dramatic. The scenario is that the ascent to the surface generates bubbles and the recompression in the chamber facilitates their transfer through the lungs. Working on the idea, I gathered 300,000 commercial diving exposures from data bases and proved that surface decompression tends to produce 10 times more type II cases than in-water decompression methods.

This is why Phillip James kept treating divers that summer before we understood the Arterial Bubble Model and switched to another method of intervention. Since then Philip has constantly crusaded against surface decompression in the North Sea, and eventually lost his appointment with one of the main diving contractors in the process (not Comex)! He has also battled for the use of deeper recompression tables than the traditional U.S. Navy Table 6 which has been found to induce worsening of symptoms on certain occasions.

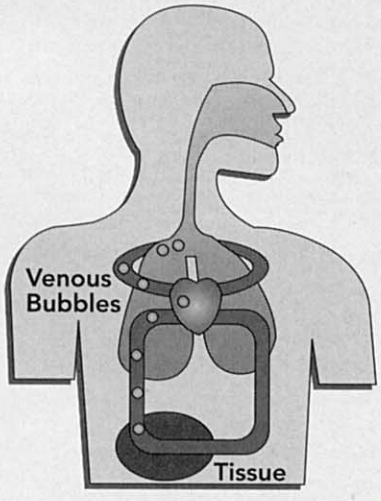


Fig 2 The Arterial Model

- **Bubbles are normally produced during a decompression in the vascular bed, transported by the venous system and filtered out in the lung. The critical issue is the filtering capacity of the lungs.**
- **In case a bubble crosses the lung and is injected in the arterial system, the distribution of blood at the level of aortic cross is such that a bubble is likely to reach a neurological tissue.**
- **The decompression strategy consists in preventing the occurrences of arterial bubbles to avoid type II DCS during the ascent.**