Dive Briefing

- Decompression Sickness

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Slide #1 - Introduction

Did you ever wonder why Decompression Sickness (DCS) occurs? What is Decompression Illness (DCI) and is it the same as The Bends. Actually, bubble disease in divers goes by many names including Caissons Disease, and Taravana. As Diving Medical Officer (DMO) for the University of Washington, I find myself explaining these issues often. Now is a good time to review current knowledge on the subject. I invite comments and welcome feedback. How well you understand this topic may make a difference someday. The first concept I like to share with you is that decompression sickness is a ***probabilistic phenomenon***. That means it can happen anytime and anywhere. The causes of decompression sickness are so complex and varied, and the variables so nuanced, that it literally is impossible to predict when it will occur. It is possible to reduce your risk of the disease, but you cannot reduce your risk of getting bent to "zero". Decompression sickness is not one disease. When bubbles form in the skin, blood vessels, lymphatic system, or nerve tissue, the symptoms produced are vastly different. Skin Bends looks quite different from Lymphatic DCS, and a spinal cord hit has vastly different implications than elbow pain. In this Dive Briefing, I hope to acquaint you with some of the newest concepts in bubble disease of divers.

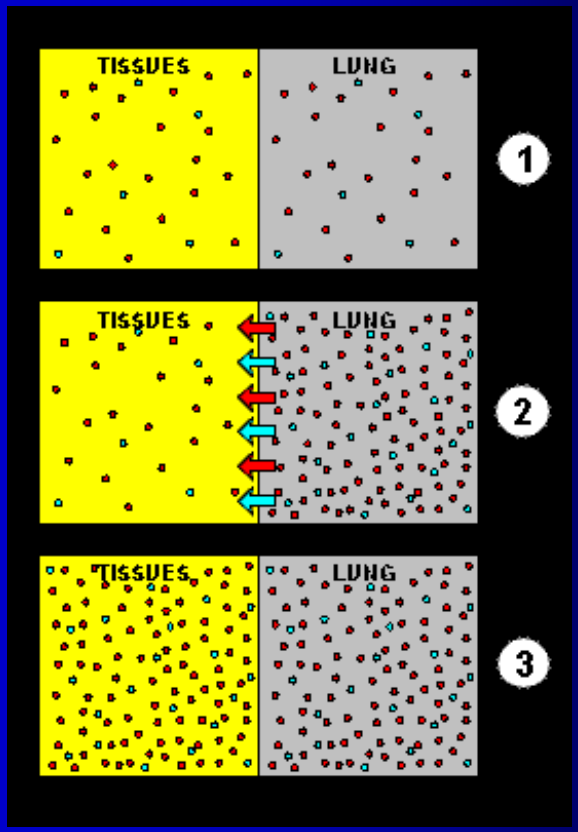
Henry's Law

Amount of gas dissolved in a liquid directly proportional to the partial pressure of that gas

On surface

On decent

At pressure



Slide #2 – Henry's Law

First, it is helpful to understand Henry's law as it is the reason the body accumulates nitrogen under pressure. Henry's Law states "the amount of gas dissolved in a liquid is directly proportional to the partial pressure of that gas". As seen in the second slide, the images depict areas of the body under different pressures (gas filled lung and gas dissolved tissue). The upper part of the image represents diver before he descends in the water. The molecules of nitrogen gas dispersed throughout body fluids and tissues are at equilibrium with the gas in the alveoli. You could say that the diver is saturated at 1 atm, meaning that there is no net movement of nitrogen gas across the alveolar /capillary membrane and for the purposes of diving I will be speaking only of nitrogen.

In the center frame, the diver has started to descend in the water and the pressure is increasing. The gas pressure in the alveoli exceeds the gas pressure in the bloodstream. According to Henry's law, molecules of gas now have a partial pressure gradient driving them into solution in the blood. From there, these molecules are carried to the tissues where diffusion continues down a gradient.

In the bottom frame, the diver is now at his maximum depth and has stayed there long enough for the gases to equilibrate across the alveolar capillary membrane again. This is a relatively bad situation as a diver can no longer ascend directly to the surface. There is too much gas dissolved in the blood and tissues, and if a direct ascent were attempted, bubbles would form and grow. If this diver ascends slowly, obeying all the safety stops and proper ascent rate for his depth and time, the gas gradually is eliminated in a manner that does not allow macro-bubbles to form. Notice I make the distinction here between macro (disease causing) and micro (silent) bubbles. Some 40 years ago Dr. Merrill Spencer identified bubbles in divers with Doppler technology that did not appear to cause disease, but was related to decompression Sickness. We now know that micro-bubbles are not totally silent. They actually trigger some events that can be perceived by the diver (for instance: migraine headaches and fatigue). The newest information on bubbles is quite surprising. It appears that nano-bubbles may be one of the reasons decompression sickness is so hard to predict.

Gas Phase ***ALWAYS*** Present

- Nano-particles (cellular dust)
- Hydrophobic niches or crevices
- Mechanical forces
  - cavitation at valves
  - shear forces at joints
- Heterogeneous sources

Slide #3 – Gas phase is ***ALWAYS*** present.

If you take a detailed look at how gas is dissolved in the body, you find that it does not always stay in solution. Gas seems to accumulate and form bubbles on hydrophobic surfaces. Lipids are hydrophobic and there are many lipids in the circulatory system. Nanoparticles are the debris remnants of cells that have been previously removed from the circulatory system. This "cellular dust" is made up of a heterogeneous mixture of bits of membrane as well as nuclear material. We actually do not know all the constituents of nanopartcicles. It is clear to researcher Dr. Steve Thom that some of it is hydrophobic and readily forms gas phase "bubble nuclei". These nano-bubbles can grow under the influence of nitrogen loading which is exactly what happens in diving. Other sources of bubble formation are hydrophobic niches or crevices in the walls of blood vessels. Various mechanical forces are at work such as cavitation around heart valves and shear forces at joints. As the joints are pulled tightly together by gravity and muscle load, the forces between ligaments as they slide together create tremendous opportunity for bubble formation. This fact is thought to be the main reason compressed air workers are more likely to get decompression sickness than in-water divers who are buoyant if using equivalent decompression tables. The least likely to form bubbles by "shear stress" are astronauts working in zero gravity. Workers under pressure are not equally likely to experience bubble formation. Cavitation around heart valves happens more readily at high heart rates and does not happen at all at slow heart rates. As you can see from the variables that I have presented above, no two divers and no two dives are alike in the production of potentially dangerous bubble nuclei. Even more interesting is the fact that the bubbles interact with the body in different ways, triggering different pathological processes.

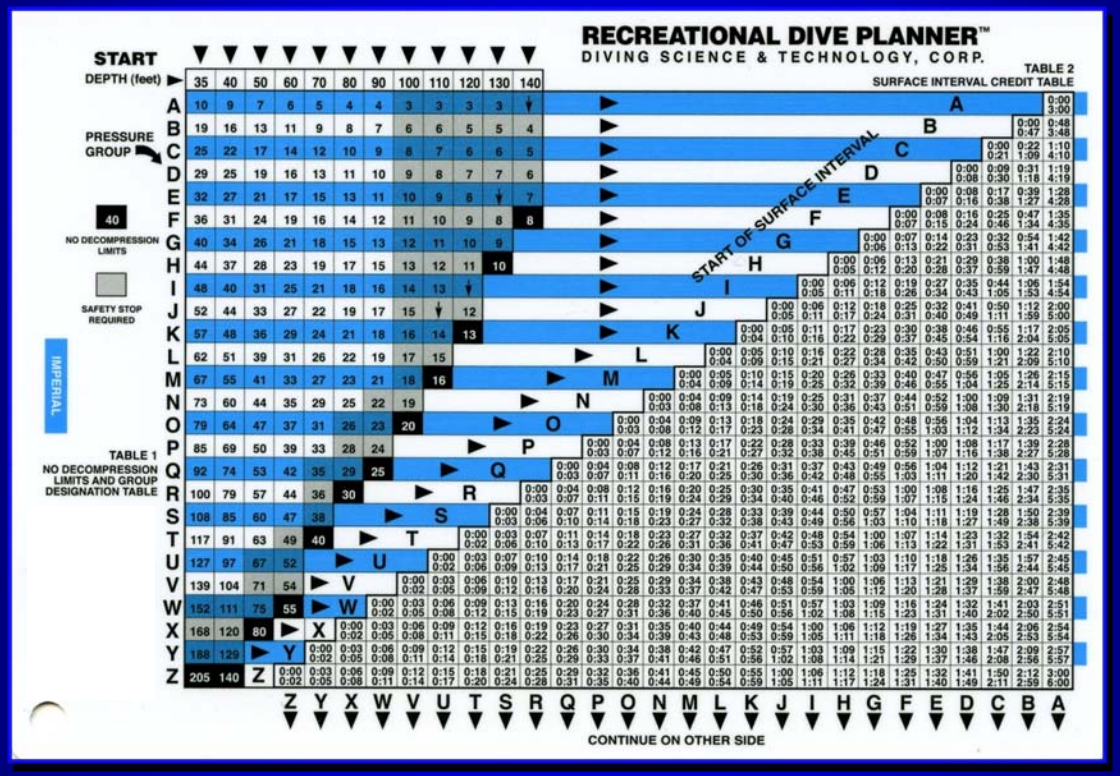
Mechanisms of Injury

- Bubble vascular occlusion
  - mechanical disruption + coagulation
- Endothelial injury
  - leukocyte adhesion, plasma loss
- Tissue anoxia
- Inflammatory Cascade
  - cytokines
  - eicosanoids
- Reperfusion injury
  - oxygen free radical damage
  - lipid peroxidation
- Programmed Cell Death
  - Apoptosis

Slide #4 – Mechanisms of Injury

Thirty years ago when I first learned about decompression sickness, it was thought that blocking the vascular system was the primary cause of decompression sickness. It was thought that the bubbles interacting with the bloodstream created a coagulation cascade. This simple explanation proved to be quite wrong, or at least inadequate. It is true that bubbles do block the vascular system and can cause complete occlusion of a vascular bed, and they do initiate a coagulation cascade. The main injury however is caused by the bubbles scuffing along the vascular endothelium, triggering a cascade of eicosanoid mediated inflammatory events. Bubbles activate adhesion molecules that cause white blood cells to stick to the vascular endothelium. This leukocyte adhesion in blood vessels liberates inflammatory cytokines. The leukocytes loosen the tight junctions of the vascular endothelial lining, causing an increase in permeability and plasma loss. This results in sludging of an already compromised "bubble congested" circulatory system. When freshly oxygenated blood is in short supply, tissue hypoxia occurs. Reperfusion injury begins to damage cells irreparably. Oxidative damage from reactive oxygen species (oxygen free radicals) and lipid peroxidation further damage hypoxic tissue. Excitatory neurotransmitters are liberated, and eventually this process leads to an acceleration of programmed cell death (Apoptosis). This is the cause for the drop out of cells long after the original insult of DCS has occurred.

Dive Plan to Prevent DCI
(mathematical precision?)



Slide #5 – Can a conservative dive plan prevent DCS?

Years ago a physiologist at NASA, Dr. Michael Powell observed that while dive tables and computers display data with what appears to be “mathematical precision”, actually nothing could be farther from the truth. The data displayed is an approximation of tissue tensions and gas distribution in the body as it diffuses from one hypothetical tissue compartment to another. Each algorithm and each set of tables is based on a series of assumptions that may be statistically true for large groups of people, but it is certainly not true for any one individual. From the 10 + variables besides depth and time I have discussed so far, it becomes clear that at any one given time, a single person could not possibly know how susceptible they are to development of decompression sickness. Dr Richard Moon reminds us that a number of other factors may play a role in fostering a more DCS susceptible individual. Factors like a diver’s age, female gender or menses, obesity, dehydration, or the presence of an atrial septal defect, or a PFO affects how a body handles bubbles. The number of independent variables is more likely above 15!

That is why the tables have been written to be very conservative in their estimate of DCS risk. When one considers probability, it is not possible to achieve zero incidence of DCS by diving very conservatively, but it does help lower the odds. There is ALWAYS a risk of DCS. If a diver understands this, then it follows that the diver should be prepared to act appropriately and seek medical attention should symptoms of DCS occur. Unfortunately that does not always seem to be the case as there is a general perception that *the diver could not be bent as he or she did nothing wrong*. Also, a huge problem is the financial impact DCS has on a diver. Recompression in a medical facility costs in the tens of thousands of dollars and therein lies a major disincentive to consider decompression sickness as the cause of a given symptom. This false assumption that decompression sickness can be avoided, leads to more significant injury when it occurs because there is often a delay in treatment. Remember in the previous slide, under “mechanisms of injury”, oxidative damage and apoptosis are irreversible processes, and if central nervous system tissue is destroyed, it will not grow back.

Symptom Onset

Time after dive	Manifestations of DCI
1 hr	42%
3 hrs	60%
8 hrs	83%
24 hrs	98%
36 hrs	100%

Slide #6 - Symptom Onset

One of the most useful tools in diving medicine is this simple chart. It provides data that is not only useful for the clinician, but for diver as well. It shows that after an insult has occurred and bubbles have formed because of nitrogen loading, the actual manifestations of disease apparent to the diver are not immediately recognized. This delay in the perception of symptoms is well known among diving medicine specialists and among researchers in the field, but it is less well known in recreational and scientific dive circles. If one considers all of the individuals who will eventually experience decompression sickness after a dive, 42% will know it within one hour. 82% will know it after eight hours. But it will take a full day and a half (thirty-six hours) for all of the decompression sickness symptoms to be fully manifested. Some divers will not know they are bent until the next day after a dive. With fatigue being one primary symptom, a bent diver will go to bed thinking “I’ll feel better in the morning”, only to wake up with severe neurological deficits. This delay in the onset of symptoms stymies one’s ability to deliver treatment early in the course of the disease. If a diver does not realize that some symptoms will not appear until a day and a half after a dive, they may falsely assume that the symptom could not be due to decompression sickness.

Symptom Clusters
1929 CASES OF DCI

Total	Manifestation	Cluster 1	Cluster 2	Cluster 3	Cluster 4
1003	Numbness	0	690	201	112
935	Pain	300	337	137	161
740	Paresthesia	0	555	161	24
306	Malaise	0	0	301	5
258	Muscular Weakness	2	0	256	0
248	Skin Sensitivity	0	140	79	29
195	Fatigue	0	40	37	118
168	Dizziness	0	35	35	98
157	Headache	0	28	25	104
142	Confusion	0	22	42	78
123	Paralysis	0	7	113	3
122	SkinRash/Mottling	0	18	21	83
96	Nausea	0	22	13	61
92	Dyspnea/Chokes	0	10	32	50
60	Incoordination	0	15	23	22
53	Vision	0	10	16	27
52	Muscular Problems	0	12	13	27
47	Vertigo	0	7	10	30
43	Unconsciousness	0	2	12	29
37	Abnormal Sensations	0	7	11	19
36	Bladder - Bowel	0	1	35	0
22	Local Swelling	0	4	1	17
16	Hearing Loss	0	0	2	14
4	Cardiovascular	0	1	2	1
4	Tinnitus	0	1	0	3

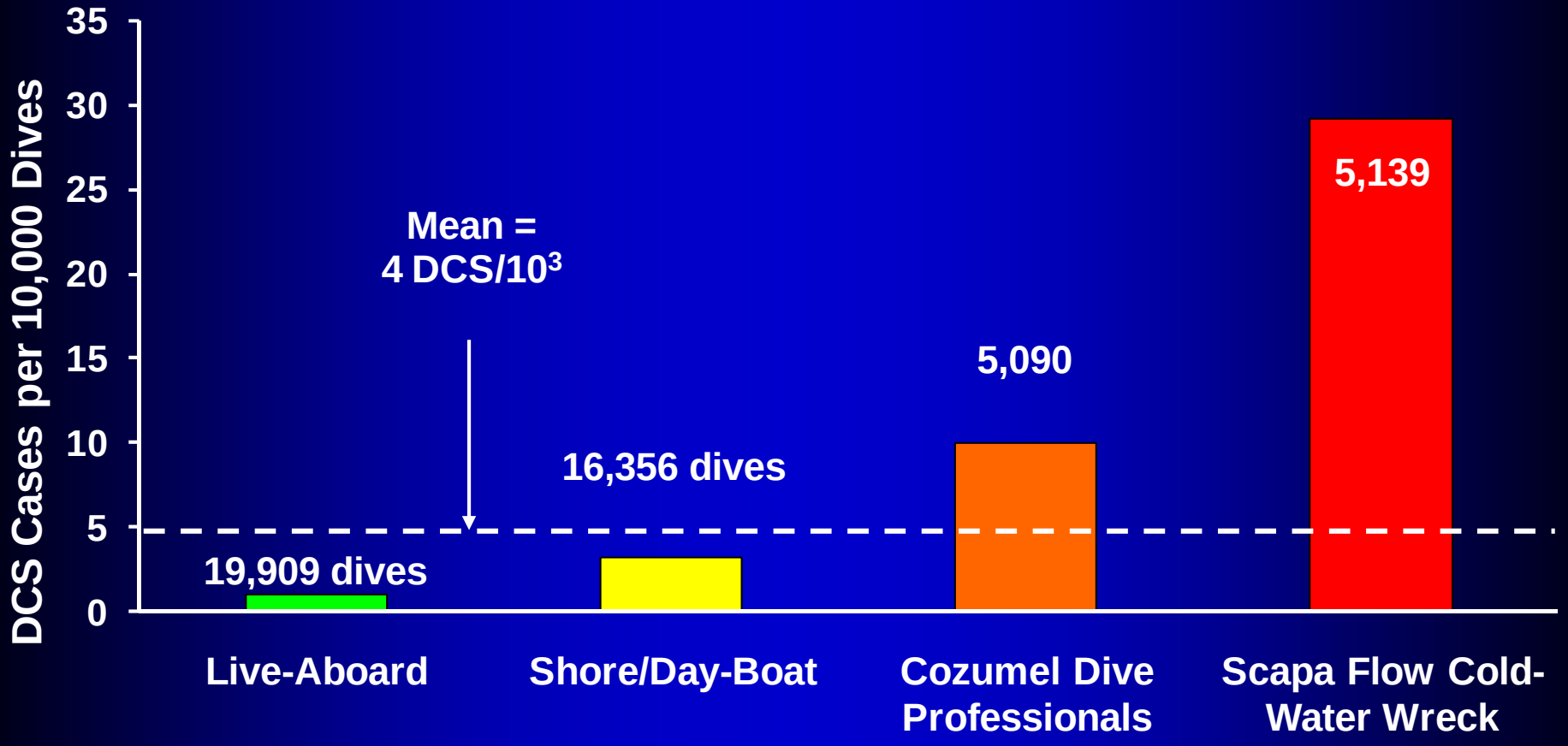
Ozyigit T, Egi S, Denoble P, Balestra C, AydinS, Vann R, Marroni A. Galatasaray University, Istanbul. Divers Alert Network. Haute EcolePaul Henri Spaak, Brussels, Istanbul University, Hyperbaric Center, Duke University, Durham NC

Slide #7 - Symptom Clusters

Statistical evidence confirms that DCS symptoms occur in clusters. Some of these clusters are now recognized as requiring different forms of treatment. In this slide from a research topic presented at a recent UHMS conference, 1929 cases of decompression sickness are analyzed by symptoms. The frequency of symptoms has been tabulated and analyzed for patterns. While skin bends is generally thought to be an insignificant complication of diving, lymphatic bends is a bit more problematic. Bubbles in the lymphatic system obstruct the usual flow of lymph fluid and this causes an abnormal, quite perceptible fluid buildup (or backup) in fatty tissue. It is most noticeable in the trunkal region, but women can notice it in breast tissue. The fatty tissues of the body feel heavy and definitely ache with the slightest movement. The skin is hypersensitive and tissue swelling occurs. Treatment with recompression is ineffective and the symptoms regress within one to two weeks. This has been known to cause abdominal pain as well. Of the twenty-five symptoms listed, fatigue, malaise and headaches may not be due to decompression sickness at all. These symptoms may be due to the presence of micro-bubbles tickling the inflammatory cascade, and the symptoms overlap with the well known “viral prodrome”. This usually is why divers ignore the first signs of DCS as they have seen it before, the last time they had the flu.

Absolute DCI Risk

1995-2002 PDE Data



Slide #8 - Absolute DCI Risk

Huge amounts of data have been gathered by Dr. Dick Vann during Project Dive Exploration. In this slide more than nineteen hundred dives have been analyzed with the location being the primary variable. The object was to determine what the absolute risk of decompression sickness was among series of divers participating in different styles of diving. Dr. Vann looked at divers on tropical vacations staying on luxury "live-aboard" dive boats, day-boats from shore based operations, dive instructors in Cozumel and wreck divers. The divers on tropical vacations tended to be a very conservative lot. There was much peer pressure from the group to dive carefully, cautiously and conservatively as anyone who got bent affected the entire boat. The stickered diver required travel back to port for emergency transport to medical care, thus effectively terminating everyone's vacation. Day-Boats have no such peer pressure but the overall diving is fairly conservative and the dive locations are relatively close and easy. Dive professionals in Cozumel work every day in the water. Their income depends on their ability to shepherd around large numbers of divers and their repetitive dive numbers are quite high. It is easy to understand how financial pressures could influence dive patterns of these instructors.

The highest incidence of decompression sickness occurred in an area of the world where all of the dives are deep and cold. Scapa Flow is an area where many wrecks from the worlds navies lie at the bottom of the ocean, and none of these wrecks are shallower than 120 feet. Due to the rigors of the area these dives are generally done by more advanced "tech" divers, often using rebreathers. The technical nature of the dives and the high degree of difficulty makes Scapa Flow quite challenging. Some of the live-aboard boats that take divers to these areas often maintain a full bar on board for bit of libation after the dive. It is not hard to understand that a higher incidence of decompression sickness might occur if beer was substituted for water as the main beverage of hydration.

While the mean incidence of decompression sickness was found to be 4 per 10,000 in this study, there was a great deal of difference between the actual incidence among live-aboard vacationers and aggressive rebreather divers on deep wrecks in cold water. It appears that human behavior is yet another variable one needs to factor in when determining risk of decompression sickness. Who knew!

Role of Oxygen

- Accelerates off-gassing of Nitrogen
  - reduces decompression time
  - excellent First-Aid gas in DCS
- Does not guarantee full resolution of DCS
  - neurological symptoms may persist
- Should not be used as a diagnostic tool
  - improvement (or not) on oxygen can be misinterpreted

Slide #9 – Role of Oxygen

Oxygen serves a valuable role in diving. When breathing 100% Oxygen, off-gassing of nitrogen is accelerated. This reduces the time it takes to decompress after a dive. The cause is enhanced washout of nitrogen, and this makes Oxygen an excellent First-Aid gas for treating decompression sickness. However, Oxygen does not guarantee that all symptoms will resolve. It is a First-Aid gas only and is not considered a treatment gas without the addition of recompression. It should not be used as a "diagnostic tool" as the outcome from its use cannot be predicted. Some cases of decompression sickness will not respond to Oxygen, and that could be incorrectly interpreted as a sign that decompression sickness was not present after all. On the other hand, symptoms could regress and leave the false impression that decompression sickness has been fully and effectively treated. If the diver wakes up after a long sleep, there may be dense paralysis due to the delay in definitive treatment of this disease. The apparent improvement in symptoms could cause one to delay recompression. The use of Oxygen does not guarantee full resolution of symptoms and should never be used for treatment only.

If the diver thinks his bent, the appropriate course of action is to breathe 100% Oxygen on the way to recompression. As decompression sickness is not preventable with certainty, it would be prudent for a well-informed diver to carry dive accident insurance. The cost is minimal, amounting to only a few air fills of the scuba tank, yet the benefit can be measured not only in the tens of thousands of dollars saved when decompression sickness strikes, but the presence of insurance allows the diver the luxury of seeking help whenever needed without the concern of financial ruin.

I would like to conclude with this wish: dive safely, dive conservatively and dive with DAN Dive Accident Insurance.