THE PHYSIOLOGY OF DECOMPRESSION SICKNESS

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Decompression is reduction of ambient pressure, and decompression sickness is any ailment resulting thereby, except for some. We don't count reverse squeeze, lung overpressure, or hypoxia of altitude. DS (decompression sickness or bends) is essentially bubble trouble. Innumerable other factors enter in, but bubble formation is the primary event, and bubble behaviour is the prime source of grief. Therefore this talk is concerned mostly with how and where bubbles form, how they make mischief, and how we get rid of them.

The answers seemed reasonably clear after the excellent work of Bert and Haldane many years ago. Bubbles formed because of a 2:1 supersaturation ratio was exceeded. They distorted tissues and blocked blood vessels. They were banished by recompression. Experience has shown those answers overly simplistic. There are too many things they don't explain. Divers get hit while obeying Navy rules: others don't in spite of flagrant violations. Many divers with proven bubbles have no symptoms, while others are bent in the absence of detectable bubbles. Some serious DS cases recover without treatment; others do not even if recompressed. Bends is more likely after two 40 minute dives three hours apart than after one 80 minute dive, in spite of outgassing during the surface interval. Yet frequent diving confers partial immunity. It's plain that we're talking about a very complex disorder. Today, after thousands of studies by hundreds of investigators, we're more confused than ever, but we have learned a few things.

How Bubbles Form

First, you've got to have micronuclei, or so it seems. Put a glass of water into a chamber and run it to 20 ATM's. If you then decompress it rapidly, it bubbles. But if while still saturated at 20 ATM the water is poured into a cylinder that has no air space, and is further compressed by a piston to 2,000 ATM, when decompressed to the surface it won't bubble at all. We infer that water contains micro-bubbles that are normally stable, but can be dissolved by immense pressure. We call them gas micronuclei. Without them, water tolerates huge supersaturation stress without bubbling.

How do people get micronuclei? The stuff we drink is one source. Scientists say that cosmic rays from space, and radioactive decay of trace elements in our diets cause micro-explosions that make more. And statistical analysis of the random movement of dissolved gas molecules shows that frequently some of them come close enough together to crowd out other molecules and so find themselves out of solution. What stabilizes micronuclei is not known. Surface tension forces, huge at small diameters, would inhibit their growth, but would also tend to squeeze them back into solution. Maybe they escape surface tension by hiding in tiny tissue crevices. Or maybe they're really something other than microbubbles.

Anyway, real bubbles can form in the body in a number of ways even without reduction of ambient pressure. Pull a finger till the knuckle pops. The pop announces sudden gas in the joint, caused by the vacuum you made in pulling. Let go, and the gas goes away in 20 minutes.

Exercise generates cavitation forces along muscles and tendons that produce bubbles. And bubbles can form in the skin by a process known as counter-diffusion. If you breathe nitrogen while surrounded by helium (as commercial divers often do) the two gases diffusing through the skin in opposite directions cause local super-saturation and bubbling - a common form of skin bends. A similar mechanism might help explain the high incidence of vestibular hits in deep diving. Theoretically bubbles could form in tissue saturated with gas while cold, and then quickly warmed, because gas solubility falls as temperature rises. Strong sound waves can also cause bubbles.

Any or all of the above may contribute to DS, but there's no doubt that the most effective way to get a lot of bubbles in a hurry, short of boiling a diver, is to rapidly decompress him. How much supersaturation a diver will tolerate before bubbles form is not known. Haldane thought the total tension of dissolved gases would have to be more than double the ambient pressure before bubbles would form - hence his exhortation not to exceed a 2:1 supersaturation ratio during ascent, and his stipulation of 33 feet as the no-decompression limit.

He was wrong. How do we know? Weighing a diver underwater before and after decompression shows that he gains buoyancy, implying a gas phase separation. Also, dives to 33 feet or even less have on occasion produced DS. The connection is confirmed by Doppler monitors which, placed over the vena cava, sound off upon decompression.

Since Haldane, others have held that supersaturation tolerance is properly expressed not as a ratio, but as a pressure differential - so many feet sea water less than tissue gas tension. Both methods for building tables share a common fault - they really aim at defining how much supersaturation stress can be tolerated without producing symptoms, not bubbles. More recently, Brian Hills came up with the astounding notion that divers can count on little or no supersaturation tolerance. A glass of 7-Up sitting still tolerates considerable super-saturation, but stir it and bubbles burst forth. A diver is more like 7-Up being stirred than sitting still. What he tolerates is not so much a specific supersaturation stress as a certain silent bubble load.

Where Bubbles Form

In practice, bubbles are commonly detected in veins and in fat. The electron microscope reveals bubbles in fat breaking through capillary walls to reach the blood, and some think that may be a main source of venous bubbles. Another source is lymph, which drains tissue fluid into veins. With increasing provocation, we'd expect bubbling in other tissues. Last to bubble would be arterial blood. It's fresh from the lungs, which swept out excess nitrogen, and it's at higher hydrostatic pressure than obtains elsewhere in the body. This is fortunate, since bubbles in arteries are particularly dangerous.

How Bubbles Make Mischief

Bubbles do indeed distort and disrupt tissues and block blood vessels. But this doesn't necessarily hurt. If you place your hand into a little chamber that seals around the wrist, and pump the air out, your hand swells with the bubbles, yet you feel no pain. To feel pain, you need pain nerves, and most tissues don't have them.

Painful DS is mostly limb bends. The site of the bubble in a limb bend has long been debated. Recently, bubbles have been seen in tendons. Maybe they arise from small fat inclusions, which tendons have. Other factors fit.

Tendons have poor circulation, so they lose gas slowly. They have a structural pattern which a growing bubble would deform, and they have pain nerves to complain about it. Exercise, known to favour bends, creates a relative vacuum in a tendon by stretching it, and generates shear forces by sliding it; both favour bubbling. And finally, injecting saline into a tendon produces bends-like pain. Of course other tissues such as ligament, cartilage, and periosteum, have not been ruled out as sites of limb bends pain.

Pain can also be a feature of serious DS. When bubbles bother pain fibres in the spinal cord, pain is felt wherever the fibres originate - usually the lower limbs, and often the back and abdomen.

Bubbles can cause pain with or without doing much damage, and they can cause damage with or without pain. Bubbles in the spinal cord or a coronary artery cause both pain and damage. Bubbles in the inner ear can cause ringing noise, hearing loss, dizziness, staggering, and nausea, all without pain. Bubbles in the brain can cause stroke symptoms - no pain. Bubbles blocking vessels in various large organs cause no pain. They don't even cause damage if collateral circulation is good enough that blood flow bypasses the block. If it doesn't, damage must result. Evidence for it is a feeling of great fatigue several hours after diving. A blood sample taken then will show elevation of enzymes released from damaged cells. In this latter case, healing seems to be prompt and complete else a lot of us here today wouldn't be functional.

Finally, bubbles (or other emboli) in bone cortex may cause silent damage that erupts in painful disability months or years later. We're talking about aseptic bone necrosis, or as it is now termed, dysbaric osteonecrosis. When such areas of bone death occur near a major joint, and eventually the joint surface collapses into them, it's sudden crippling. Dysbaric osteonecrosis correlates poorly with limb bends, and not at all with serious DS. Nobody knows why. An intriguing hypothesis is that uranium^{238}, which tends to concentrate near the ends of long bones, insures a plentiful supply of gas micronuclei to initiate bubble formation in those areas.

The Pulmonary Bubble Trap

Since ordinary dives produce bubbles capable of blocking blood vessels, why isn't DS far more common? Recall that most bubbles either arise in veins or are delivered to venous blood. Venous blood goes to the right heart, which pumps it through the lungs. A lung happens to be a superb bubble trap. Bubbles big enough to block vessels are filtered out, and remain stuck till their gas diffuses out to the alveoli.

Isn't this hard on the lungs? It appears that a diver won't notice symptoms until 25-60% of his pulmonary circulation is blocked, depending on how active he is, and on whose figures you read. Near the limit, there are warnings. Greek sponge divers learned that when a drag on a cigarette sets off a fit of coughing, it's time to pack in the day's diving and take an underwater decompression stop. Even a deep breath may provoke the coughing.

Chokes

If the lung's capacity to trap and scrub bubbles is exceeded, a lot of bad things can happen - like the chokes, spinal hits, and arterial gas emboli. The chokes is a syndrome of pain, shortness of breath, and coughing. The lungs get water-logged and still - it's harder to breathe. Reflex or chemically induced bronchospasm further reduces air delivery. Alveoli without blood flow can't exchange gas, so oxygen uptake goes down. Yet these alveoli are still ventilated, so part of the work of breathing is wasted. All this blockage causes high resistance to flow of blood through the lungs. Back pressure builds up in the pulmonary artery, and the right ventricle must work harder to pump the blood it gets from the systemic circulation. When it can't maintain the pace, circulation slows and back pressure builds up in the systemic veins. Nitrogen delivery from tissues to lungs falls off, and this slows outgassing and favours more bubbling. When the capillary beds feel the back pressure, there is a shift of water from blood to tissue, reducing the blood volume - a prelude to shock.

Spinal Hits

Serious DS can occur well before the bubble load in the lungs is large enough to cause chokes. Spinal bends is a special case. It is often due to blockage of the veins that drain the spinal cord - the vertebral venous plexus. It is most apt to occur when circulation is slow, and it's aided by an anatomic peculiarity. Most veins have one way valves to prevent back flow. The veins of the azygous system, which drains the vertebral plexus, do not. If anything increases the pressure in the chest, like say a cough, or exhaling against airway obstruction, there could be a temporary back flow of blood through the azygous system pushing bubbles just emerged from the vertebral plexus back into it, where they hang up. Indeed, venous blockage isn't unique to the spinal cord. Studies of other tissues have shown bubbles first apparent in venules to grow back into capillaries. And spinal hits can also result from arterial emboli.

Arterial Embolism

Bubbles gain access to the arteries when allowed to by-pass the pulmonary trap, or when it is somehow released. An unborn baby has a hole in the wall between the upper heart chambers, so that blood can flow directly from the right to the left side, by-passing the non-functioning lungs. This opening (the foramen ovale) is supposed to close after birth, but doesn't always. Up to 50% of adults retain at least a pin-hole defect, and the vast majority don't know it. Normally it doesn't matter. But when lung circulation is blocked and back pressure in the right atrium

builds up, a significant amount of blood and bubbles will take the short cut to the arterial side of the circulation.

Lungs themselves have shunts that bypass their own capillary beds. These seem to be closed most of the time, but they open as the bubble load increases. In one experiment, tiny beads of various sizes were injected intravenously. A few beads with diameters larger than lung capillaries always got through. As the rate of injection was increased, more beads of larger sizes got through. What opens the shunts is uncertain - maybe increased pressure, hypoxia, elevated CO_2, or all three. Or conceivably, bubbles could rupture pulmonary arterioles and break directly into venules, to by-pass the trap.

Finally, a repetitive dive could spring the bubble-trap by squeezing some of the bubbles down small enough to pass through. More nitrogen is taken aboard during the dive, and upon ascent it diffuses into those pre-existing bubbles and enlarges them to more than original diameter. Any that happen to be in the arteries at the moment they become too big to transit the micro-circulation become arterial emboli. As mentioned before, the harm they cause depends upon where they happen to lodge. This helps explain the random incidence of DS for a given provocation - the fact that if you plot the incidence on a graph, you get the bellshaped curve that reflects pure chance.

Since some bubbles do reach the arteries, the question arises why brain hits are so rare in DS, while so common in the air embolism of lung over-pressure accidents. There is disturbing news. Maybe brain hits are not so rare. After lung tear, a lot of air enters the carotid artery all at once. DS bubbles are much smaller, but they keep coming. They would tend to block much tinier vessels in more scattered areas. It has been reported that very careful neurological examination of DS patients often does show evidence of subtle brain damage. It has also been casually observed that some commercial divers of long experience seem to undergo personality changes. Finally, autopsies of goats exposed to frequent decompression stress showed unequivocal brain damage. The pathologist opined that one of the brains couldn't have been more than fifty per cent functional. Yet none of the goats had demonstrated abnormal behaviour.

Biochemical Disorders

So far, we've been looking at DS as a mechanical disorder - the gas laws in action. There's another side - biochemistry in action and it's a whole new ball game. It's tempting to say that blood recognizes bubbles as foreign invaders and counter-attacks. That's probably nonsense, since the counter attack does more harm than the bubbles would if left alone. More likely, bubbles trigger the damage control system for plugging up leaks in vessels, but at the wrong time and in the wrong places.

It all starts with blood globulins - large, complex, biologically potent molecules. These have one pole that attracts to water, and another that attracts to fat, and typically arrange themselves in groups with the fat loving poles inward. Bubbles change this. For reasons best known to biophysicists, the blood-bubble interface generates an electro-kinetic force that makes the molecules flip. They re-align with their fat loving poles at the interface. This somehow activates them to do strange things. Nearby blood platelets become sticky. They adhere to bubbles and to each other, whereupon they too become activated. The result is the release or the induction of a witch's brew of highly bioactive substances with odd names like serotonin, bradykinin, kallikrein, histamin, SMAF, prostaglandins, etc. Then things really liven up.

The blood clotting mechanism is cranked up. Lipids split off lipoproteins and coalesce into globules of fat. Capillary permeability is increased, and fluid leaks from blood into tissue spaces, decreasing blood volume. Arterioles constrict intensely, reducing and sometimes shutting off circulation to capillary beds. By the time waste accumulation and hypoxia make the arterioles relax, the capillaries have been damaged. Some of their lining cells peel off and are swept into the blood stream. Plasma leaks through the damaged capillaries into tissue spaces, so blood loses more volume, and becomes further concentrated. This increases its viscosity, which slows circulation, favours more clotting, and makes red cells sludge together in clumps.

How far all this goes depends upon the bubble load. When the body's compensatory mechanisms are overwhelmed, the victim slides into shock. This is one reason why it's so important to get potentially serious DS into a chamber fast. Wait till the victim's in bad shape, and the results of recompression will be disappointing. You can't squeeze out the platelet masses, fibrin clots, fat globules, red cell clumps, and other cellular debris clogging up his circulation. He needs a lot of medical intervention - intravenous hydration, anticoagulants, etc.

In practice of course the mechanical and biochemical effects of bubbles interact. Bubbles might hang up where tiny arteries branch into two smaller ones because surface tension opposes deforming a sphere into two wieners. But why should bubbles hang up in small veins? Doubtless because they attract platelets, which become sticky.

Predisposing Factors

Some things predispose to DS, and we may now ask how. The most important seem to be obesity, age, exertion, and illness. After a long dive, fat holds a lot of nitrogen which, because of poor circulation, it can't unload. So it Bubbles. Also, overweight people tend to have elevated blood lipid levels, which favours the biochemical derangement in DS. Incidentally, fatty meals temporarily raise blood lipids and so favour DS.

Estimates of the importance of age vary, up to an 11% annual increase in bends liability for young men. All agree that the risk increases sharply at middle age and beyond. The simplest explanation is increase in body fat and, more important, in arteriosclerosis, which reduces circulatory efficiency.

Exercise during a dive speeds up circulation and, therefore, nitrogen uptake. It also generates lots of CO_2 which, because ventilation is impaired at depth, results in elevation of the CO_2 tension. We don't know why, but CO_2 seems quicker than nitrogen to promote early bubble growth. Once bubbles form, any excess CO_2 enlarges them faster. Upon ascent, exercise

promotes bubbles by cavitation and shearing forces, and by generally shaking up the body.

A few years ago, aspirin got an undeserved reputation for causing bends. The real culprit turned out to be the condition for which the diver had taken the aspirin. Though the mechanism is not understood, illness seems to predispose especially to serious DS. In this context, illness includes alcohol abuse and other conditions that lower metabolic efficiency. Dehydration predisposes at least in part because it's a head start to haemoconcentration. Divers shouldn't be thirsty.

De-disposing Factors

Caisson workers and people who dive a lot acquire partial immunity to bends. This is said to peak in two weeks at a level of 75% protection. We think it results from subclinical bubbling, since intravenous injections of air in animals also confers immunity. A favoured explanation is that silent bubble showers from repeated decompressions consume micronuclei faster than they can be replaced. The curious fact that immunity for one depth does not extend to deeper depths is consistent, if we assume that some micronuclei are more stable than others and require more supersaturation stress before they will commence expansion. Consumption of blood clotting factors faster then replacement might further add to the protection. To avoid unpleasant surprises, it is most important for a diver to remember that immunity is lost in 2-3 weeks of not diving.

Out-gassing

We'll now address our final question. How do all these divers running around with silent bubbles ever get rid of them? Say for example you've just completed a nice legal dive - 50 minutes at 60 feet. You have silent bubbles. When all of your excess dissolved nitrogen has diffused either into the bubbles or into the alveoli, and equilibrium is reached, what's to keep those bubbles from becoming permanent guests? They're at ambient pressure, so why should any gas leave them? One reason is that a bubble is never really at ambient pressure. Surface tension and tissue elastic recoil exert at least a little compressive force upon it. Another and far more important reason is the inherent unsaturation of living tissue.

Inherent Unsaturation

Full equilibration between tissue and alveolar air cannot be reached, but rather a steady state in which tissue gas tension is less than ambient, and therefore less than bubble gas tension. This is tricky to grasp, but it's very important. Here's how it goes. We say that, by the laws of Henry and Dalton, dissolved gas tensions in the body are in equilibrium with gas tensions in alveolar air, which must add up to ambient pressure. But it's not quite true. Actually, only arterial blood equilibrates with alveolar air. When arterial blood gets into capillaries, oxygen is lost and CO_2 is picked up. Oxygen is poorly soluble in blood plasma, and the small amount dissolved is rapidly soaked up by the tissues, so oxygen tension drops way down. True, haemoglobin unloads more to meet tissue needs, but only at this low tension. Conversely, the CO picked up is very soluble, and most of it is found in chemical combinations anyway, so while oxygen tension drops a lot, CO_2 tension rises very little. Therefore, the sum of all dissolved gas tensions is lower in venous blood than in arterial blood. Since tissues are in equilibrium with venous blood, their total gas tension is also less than ambient pressure, and therefore less than gas tension in bubbles. Thus, a bubble is always super-saturated relative to the tissue it occupies, and the tissue is unsaturated relative to the bubble. So Bubble gas must slowly dissolve into tissue fluid until there is no more bubble.

The magnitude of inherent saturation has been measured in rabbits at the surface as 80-94 mm of mercury. At depth, breathing compressed air, arterial oxygen tension is very much greater, but venous oxygen tension is not. Inherent unsaturation, determined by the difference between them, therefore becomes very much greater. This has surprising implications for decompression practice. Take a commercial diver who has put in four hours at 150 feet and wants up. Haldane would bring him up half way to achieve maximal tolerable super-saturation, and thus maximal out-gassing gradient. Hills says that's wrong. People don't tolerate much super-saturation, so you'll just produce a lot of bubbles. Once bubbles appear, excess dissolved nitrogen will diffuse into them. Gas tension in tissues and blood will therefore drop toward ambient, and the out-gassing gradient in the lungs will become low. The diver must then wait till inherent unsaturation and slow alveolar diffusion remove a lot of gas from the bubbles before he can safely ascend to the next stop. The more shallow he gets, the smaller is the inherent unsaturation, so the stops have to be longer. What a drag!

The right way, by Hills, is the opposite. Start the ascent slowly, with much deeper stops, so any bubbles starting to form will be promptly banished by the high inherent unsaturation at those depths. This is greatly facilitated if the bubbles are never permitted to grow, for with tiny bubbles, you get much more help from surface tension, and the surface to volume ratio is very great, permitting rapid re-solution of gas.

This approach has good experimental support, and is now being used in some commercial operations. The Navy tables are based on the Haldane model, but extensively modified through experience. Let us emphasize that, as of now, they are the only ones thoroughly tested and currently recommended for sport diving.

Oxygen in DS Treatment

Since inherent unsaturation results from oxygen metabolism, it is sometimes called the oxygen window for out-gassing. To open the window wider for faster safe ascents (in commercial diving) or for treatment of DS, you need simply replace all or part of the diver's air supply with oxygen. This trick further improves the out-gassing gradient for nitrogen because it lowers inspired nitrogen. Another advantage of oxygen for treatment is that at three atmospheres absolute, enough dissolves in plasma to supply basal tissue needs without help from red blood cells. At such high tensions, oxygen will diffuse through tissue fluid around blocked vessels enough to keep some of the deprived cells alive.

Other Aspects

We're done with bubbles, but before we have done with DS, we must at least consider the

possibility that other factors associated with decompression may play a role. Notable is osmotic pressure - the tendency of water to flow (along its own concentration gradient) from areas where the concentration of dissolved materials is low to where it is high. During ascent, fast tissues lose gas more rapidly than slow tissues, and the resulting osmotic gradients would tend to pull water from the fast into the slow tissues. A special case is blood, which is thought to equilibrate with tissues by the time it traverses capillaries. In the arterial portion of the capillary, up to where equilibrium is reached, blood would lose water to the tissue. As we've seen, haemoconcentration favours DS.

Osmotic gradients created by the descent phase of the dive could also be significant. Dissolved gas in working muscle and in bone marrow would rise much faster than in the adjacent bone cortex, tending to dehydrate it. Haemoconcentration in the cortex capillaries would favour red cell sludging and blood clotting, and thus osteonecrosis. If this is significant, as animal studies suggest it may be, dysbaric osteonecrosis becomes, to that extent, compression sickness.

UNDERWATER RECOMPRESSION OF BENDS

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People dive everywhere. Charles Howell dived in Thailand, which is fine except that the nearest recompression facility is far away in the Philippine Islands. When he got bent, he opted for recompression underwater instead of the 15-plus hour journey to Subic Bay. Here's how it went.

The dive was the first of the weekend. It was mostly at 60 to 70 feet, with a maximum depth of 85 feet and a bottom time of 23 minutes. Soon after surfacing he noticed loss of sensation in his right hand, and then complete paralysis of the right arm and leg. What to do? He could go to hospital and hope for the best, or set out for Subic Bay, or put on a tank and go back down. None of the options was attractive, so it became a matter of deciding which was the least bad.

A quick inventory of resources showed that: 1. Seven full tanks of air were available; 2. The victim was mentally clear and felt confident that he could manage his own air supply; 3. The water temperature would permit several hours of exposure without hypothermia becoming a problem; and 4. There was present a very experienced divemaster who could accompany and observe the victim. All things considered, underwater recompression seemed the best bet.

The profile elected was 20 minutes at 60 feet, 10 minutes at 50 feet, 10 minutes at 40 feet, 20 minutes at 30 feet, 30 minutes at 20 feet, and 60 minutes at 10 feet. It worked - Mr Howell regained the use of his limbs at 40 feet on the way down, and felt quite normal after decompression. The following day, however, the right limbs felt a little weaker than the left, and ever since they tire more easily with exercise.

We learnt of this case from Arthur Rhodes, NAUI, in Thailand. He is disturbed. He takes the US Navy view that underwater treatment of bends is dangerous and should be condemned. He fears that its success in the present case will entice others to try it, with disastrous results, and he asks for our comments.

First, we congratulate Mr Howell and the people who advised and assisted him. He had a very serious problem that needed a quick solution, and it was provided. It's hard to argue against success. Yet looking through our retrospectroscope (the medical equivalent of Monday morning quarterbacking) we can see possibilities for improving the management. First, use of the reverse slant position at the first sign of trouble might or might not have been beneficial. Second, newer theories of bubble resolution suggest that some of the time spent at 10 feet might have been better spent at the 30 and 20 foot stops. Still, the schedule used was more in keeping with conventional practice and cannot be faulted. Third, the 2 and a half hour treatment that was possible falls far short of any approved schedule. If oxygen could have been obtained, its use for some hours after surfacing might have improved the outcome. Fourth, a neurological exam after the apparent cure might have revealed subtle residual defects and resulted in advice to get to a chamber. Finally, since central nervous system symptoms can occur after apparently successful treatment, and since a recurrence is best treated with hyperbaric oxygen, it would have been well to get near a chamber for a day or two of observation - assuming a flight with cabin pressure near sea level and oxygen available.

And now that we've gone on record as agreeing with Howell's underwater recompression - a controversial opinion - we hasten to add that we also agree fully with Arthur Rhodes. A decision to treat bends underwater is almost always wrong. Rarely is anyone present with the expertise to analyse the situation and invent the best possible treatment schedule. For many reasons, safe treatment of adequate duration would usually be impossible anyway. The victim might be or become unable to help himself, or the air supply inadequate, or the water too cold, or darkness would set in, or the weather deteriorate. The victim's nitrogen burden would usually be large enough that any feasible underwater schedule would hardly dent it. The depth required to relieve symptoms would usually cause further ingassing, adding to the nitrogen burden. Unless the ascent could be stretched over a great many hours, it would produce larger bubbles and worse symptoms than before treatment. Finally, valuable time would be lost. The victim needs hyperbaric oxygen, and the longer he is deprived of it, the less will be his chances of recovery. There are horror stories of victims and rescuers dying during prolonged underwater treatment attempts. In most cases, the outcome will be better if all efforts are directed towards reaching a chamber