Dive safety is all about one of two scenarios: Reacting appropriately when something goes wrong, or taking the appropriate action needed to prevent something from going wrong in the first place. In both cases the response typically involves having certain skills, and practicing them often enough to ensure that those skills don’t decay. Whether it’s a simple task like finding your way back to the boat or a more urgent matter like dealing with an out-of-air emergency, the operative word is “action” — doing the right thing.

By Alex Brylske
Photos by Joseph C. Dovala
Hedging Your Bets: How to Reduce Decompression Stress

Get plenty of rest prior to diving. Sleep has a vital restorative function, so only a well-rested body can be at peak performance.

Remain well hydrated. Drink enough water so that your urine is copious and clear. And don’t forget to continue drinking water between and after diving. There’s no need to create any more gas seeds than necessary. So, avoid heavy exercise immediately prior, during, and for at least six hours after diving.

Dive conservatively. Avoid reaching any no-decompression limit by giving yourself at least a 5- to 10-minute safety margin. And those who have celebrated their 40th birthday should be even more cautious.

Pay close attention to your ascent. Don’t exceed a rate in excess of 30 feet per minute (even if your computer says 60 is OK).

Make safety stops. On deeper dives, take a one-minute safety stop at half your maximum depth, and a three- to five-minute stop at a depth of between 15 and 20 feet (4.5 and 7 m).

Yet, some dive safety issues involve knowledge more than skill. Take, for example, decompression safety. Unlike most other problems that divers face, the effects of decompression sickness (DCS) are not immediate; they take time to evolve. Bends occurs only after your dive is over, and often takes an hour or more to manifest. Of course, proper reaction — in the form of knowing how to care for a diver experiencing DCS — is important, but it’s much better and much easier to take proactive measures so that you might prevent it.

Another way of looking at how decompression sickness differs from other diving emergencies, like lung expansion or other forms of traumatic injury, is that the latter are “all or none” events while DCS is gradual. In fact, you can view the risk of DCS on a continuum based on how close a diver is to the no-decompression limits. But it’s important to understand that these so-called limits are not, and may never be, firmly established. People get the bends even when they don’t exceed no-decompression limits. The reason is that no theoretical limit can ever fully take into account the nuances of an individual’s physiology and diving conditions.

It’s also inaccurate to view the decompression continuum entirely as one based on time because an individual’s susceptibility to the bends involves many factors unrelated to how long they’ve spent at depth. For this reason, experts in the field now use a different approach to describe this continuum of risk from a predive state to a point where DCS occurs. That concept is termed “decompression stress.”

The dictionary defines stress as “pressure or tension exerted on a material object,” which isn’t a half-bad definition when we consider that material object the human body. Take, for example, two short 10-minute dives, one to a mere 10 feet (3 m) and another to 100 feet (30 m). While both dives involve the same duration, they differ in pressure exposure. Still, as both result in some excess nitrogen absorption, they each subject the diver to some de-
compression stress. Of course, the deeper dive results in more stress because more nitrogen was absorbed. But time and pressure aren’t the only factors contributing to decompression stress exerted on any dive; and to comprehend this requires a bit more insight into the mechanisms at work.

**Gas Absorption and Decompression**

Let’s start with the physics of gas absorption. One challenging concept for many is the fact that gas can be absorbed into a liquid. However, that’s what happens when drinks are “carbonated” — the liquid absorbs pressurized carbon dioxide. The difference between DCS and a carbonated beverage is that the former involves nitrogen and the latter CO₂. Still, the concept is pretty much the same: High pressure drives gas into the spaces between the molecules of liquid. Then, when the pressure in contact with the liquid is reduced by ascending (or the bottle top is removed from the soda bottle), gas begins escaping from the liquid. In the case of a bottle of soda, this pressure deduction is often enough for the carbon dioxide to come out of the liquid so fast that the gas molecules coalesce into bubbles.

Because of the molecular arrangement of oxygen and hydrogen atoms in the water molecule, water has some very special bonding characteristics. In essence, each molecule of water is electrostatically attracted to another in what are termed “polar bonds.” This bonding holds water molecules together so tightly that it makes spontaneous bubble formation in water very difficult. For example, believe it or not, you could compress pure water to over 200 atmospheres — that’s almost 3,000 psi — and, provided you didn’t shake it, immediately decompress it without forming a single bubble.

But if that’s true, why does a bottle of soda, which is under a pressure of only a few psi, fizz when it’s opened? And, more importantly, why can’t a diver ascend from as little as 2 atmospheres (33 feet [10 m]) without risking a trip to the recompression chamber? The answer is that there’s more to the bubble formation phenomenon than just pressure reduction.

Notice that I said pure water, meaning that it contains no other substances or impurities. Why is this important? It can be best explained by yet another phenomenon: rain formation. We all know that rain comes from clouds, which are made up of water vapor. But it’s just how a raindrop develops from the water vapor that’s at issue here. At the core of every raindrop is a particle of dust. This dust particle acts as a focal point or “seed” around which the water vapor collects and grows into a drop. That’s also very much like the way bubbles form in liquids.
If we take a liquid containing other substances or particles along with water molecules, we'll see a big difference from the pure water experiment. Just like raindrops, foreign particles in the water seed the production of gas bubbles. Exactly how many bubbles form depends on the number of particles dissolved in the liquid, the pressure differential and other factors. The point is, the seeds make all the difference. You can confirm this prediction by sprinkling a pinch of salt into soda left standing in a glass for days. Even though it has gone "flat" (seemingly lost all its carbonation), this will produce more bubbles because something — the salt crystals — act as seeds for bubble formation.

**From Physics to Physiology**

So what exactly does the evolution of a raindrop, or how bubbles form in soda, have to do with decompression sickness in humans? It all comes back to the seed idea. Like the flat soda, our bodies can be "seeded" to form bubbles. These seeds are actually called gas micronuclei, which are microscopic pockets of gas caused by factors such as movement. Note that in the pure water experiment I qualified the conditions by saying that the water couldn't be shaken. The reason is that the turbulence caused by shaking the water generates micronuclei that would, in turn, seed the formation of bubbles.

Of course, our bodies move all the time, as does the blood in our veins. This continual motion, combined with the normal turbulence of blood flow, is one theory behind how gas seeds form in tissues. During an ascent, nitrogen diffuses into these areas of low pressure forming tiny microbubbles — the precursor of decompression sickness.

Researchers speculate that this phenomenon occurs primarily in the capillaries — the smallest structures of the circulatory system. From there, many of the bubbles enter the venous circulation and flow back to the heart. Since they're very tiny bubbles, they normally don't cause any blockage in the vessels during their transit. From the heart, blood travels to the lungs. When the bubbles reach the extremely fine capillary bed of the alveoli, they're trapped. The bubbles diffuse back into the alveoli and nitrogen gas is expired in the normal respiratory process. Since these bubbles have no obvious effect, they're called subclinical or asymptomatic. More commonly they're known as "silent bubbles."

Bubble formation is also facilitated in humans — and other animals — for yet another reason. Unlike the soda experiment, in which the fluid was contained in a glass, blood is contained within vessels. A large portion of blood...
vessel lining is made up of lipid (fat) tissues. This is significant because bubble formation can occur more easily on oily or what are termed “hydrophobic” surfaces, like the interior walls of blood vessels. Bubble formation is further enhanced because the vessel walls aren’t smooth but highly irregular, providing places in which gas seeds can form. (For more insight into why the surfaces of tissues are so irregular, see “As Young as You Feel? Diving and the Effects of Aging,” Dive Training, September 2008.)

When a diver ascends, pressure is reduced and the nitrogen absorbed by the tissues begins to release. The term used to describe this is “offgassing.” (Remember, only the nitrogen is at issue because oxygen is used up in the metabolic process.) Most of the excess nitrogen escapes while still in solution, but some can coalesce around the gas seeds, forming microbubbles. The more nitrogen that’s released, the more the bubbles tend to grow. But here’s something interesting: These bubbles tend not to form the familiar spherical shape we see in our soda experiments. Instead, they become elongated — a shape that increases their surface area and resistance to movement. Of course, if they’re big enough, bubbles in blood vessels can stop or interfere with normal blood flow. This further compounds the decompression process because the decreased circulation efficiency makes it more difficult for dissolved nitrogen to escape from the tissues.

Some researchers have described this phenomenon as the “bottleneck effect.” The dissolved nitrogen tries to escape, but the localized bubble formation slows the flow of blood that would otherwise carry away the dissolved nitrogen. The nitrogen has to go somewhere, so it diffuses into the newly formed bubbles, causing them to grow even larger. This is the mechanism now believed to be a primary cause in Type II or neurological DCS, which is also the most serious form of the disorder.

To further complicate matters, bubbles don’t just form within the blood vessels. Nitrogen can diffuse into seeds between tissues. In this case, as the bubbles grow, they can damage the tissue and compress nerves. This type of bubble formation is called extravascular, meaning “outside the vessel.” Aqueous (watery) tissues — the type that make up ligaments and joints — are especially prone to developing these types of bubbles; and it’s one reason why researchers believe that extravascular bubbling may be the primary cause of the classic symptoms of DCS, joint pain, in the less serious form of decompression sickness collectively termed Type I.

From the standpoint of our discussion of decompression stress, the take-home message is simple: The more gas seeds we produce, the more bubbles we might form. So, if we do all we can to reduce the seeds, we also reduce the stress. Unfortunately, a major cause of gas seed development appears to be movement. And it’s movement of all sorts: movement of joint surfaces, movement of one tissue over another and even movement of the blood itself. While it’s impossible to avoid all movement, one thing we can do to reduce the likelihood of gas seed formation is to avoid excessive or even unnecessary exercise before, during and after a dive.

Beyond the Bubbles

Ask any diver you encounter to explain what causes DCS and the likely answer you’ll get is simply, “bubbles.” That is, of course, correct, but it’s only part of the answer. The more complete story is that bubble formation gets the ball rolling, but what actually happens inside our body is a highly complex interplay of both bubbles and biochemistry. During the treatment of DCS, in fact, dealing with the biochemical complications is as important as recompression therapy.

A good way to conceive of the biochemical effects of bubbles in our blood is by looking at how our immune system deals with any foreign invader. Gas bubble formation brings on changes in blood chemistry such as ac-
tivating the clotting process. Platelets — blood components responsible for clotting — become sticky, attaching themselves to each other and the newly formed bubbles. The bubbles also cause inflammation of the capillary walls, causing leakage of fluid into the tissues, which contributes to dehydration. Eventually, some blood vessels themselves begin losing their integrity and start breaking down. This causes the vessels to narrow and dislodges fat particles into the blood, becoming another source of gas seeds.

Studies have also shown that bubble formation activates an immune response called the complement system. This biochemical response helps clear pathogens from an organism using a group of molecules termed complement proteins. These proteins cause the release of histamines and other chemicals that also cause fluid to leak from the capillaries into the surrounding tissues. This process, incidentally, is similar to the way our body reacts when we go into shock. These factors are all reasons why medical support, in the form of IV drug and fluid administration, is a vital part of the emergency response to victims of decompression sickness.

The overall effect of this biochemical activity is like a snowball rolling downhill. The blood thickens, becomes sticky and cannot move as efficiently through the vessels. Red blood cells now clump together. This, in turn, decreases circulatory efficiency and the efficient release of nitrogen. More bubbles form, and bubbles that already exist grow. The blood flow slows even further, and in a downwardly cascading cycle, the condition gets worse.

The point of this discussion is not to advocate IV fluid administration as a way of reducing decompression stress; but there is something we can all do in its place — stay well hydrated. One of the best ways to hedge your bet on the side of decompression safety is by keeping your fluid volume as high as possible. That means drinking copious amounts of water before, between and after diving.
Stretching the Balloon

A pioneering advocate of the decompression stress continuum concept is Dr. David Sawatzky. To explain his perspective, he uses the analogy of how stretching a balloon is akin to tissue damage done by bubble formation. For example, you can stress an inflated balloon by squeezing it, yet it will return to normal once you release your grip. But if you squeeze harder — or inflate it repeatedly — the balloon will begin to weaken. Eventually, the weakened state could result in a failure, and the balloon pops. In a sense, the same thing happens with the cells of our body. If a mild stress is placed on a cell, it will undergo both physical and — unlike the balloon — biochemical changes, but will return to normal when the stress is removed. However, if the stress is increased or is continual, pop goes the cell; it’s permanently damaged or dies.

Continual or significant decompression stress, over time, could at some level cause cell damage; and this may occur even though no obvious signs or symptoms are present. Of course, as the decompression stress increases, so does the level of cell damage, as well as the severity of any DCS symptoms. And even if the signs and symptoms are resolved through treatment, there’s still a strong possibility that permanent cell damage will occur.

In Sawatzky’s model, the decompression stress continuum is divided into four categories, as illustrated. The first category is in the range in which the diver emerges from a dive and has no DCS signs or symptoms. Moving up to the second category range, a diver may notice some subtle symptoms such as skin itching or unwarranted fatigue, but no cell damage occurs nor is any treatment required. Moving into the next category range, the diver demonstrates clear signs and symptoms and cell damage occurs. This warrants treatment to minimize the amount of damage. Finally, the fourth category range is where the diver has severe signs and symptoms, and without treatment the amount of damage could become severe and permanent. Probably the only good side of DCS is that it often takes several hours for permanent cell damage to occur, and treatment even after days of...