DCI Demystified
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Everyone who takes a formal diver training course receives at least a basic education in the signs, symptoms, etiology, and prevention of pressure-related diving injuries. You might even know someone who's experienced one firsthand and been treated in a hyperbaric chamber. But what does it all mean? What's the difference between decompression sickness (DCS) and decompression illness (DCI), and where does arterial gas embolism (AGE) fit in? Let's take a look at some of the physics and physiology behind these sometimes misunderstood and often complex injuries.

Pressure? What pressure?

Our diving gear, whether open-circuit SCUBA, rebreather, or surface-supplied, is designed to deliver breathing gas at a pressure that allows us to inflate our lungs against the ambient water pressure. This means that the gas pressure in our lungs at depth is equivalent, within a few millimeters of mercury (mmHg), to the pressure of the water around us. Water pressure increases linearly with depth, so the greater the depth, the greater the gas pressure in our lungs.

That's where our good friends John Dalton and William Henry come into play. Dalton's Law is the law of partial pressure. It states that the pressure of any gas mixture (e.g. air, nitrox, heliox, trimix) is the sum of the *partial pressures* of the gases in that mixture. For example, on the surface where the only pressure we experience is from the atmosphere, we can say that we're under one atmosphere of pressure (1 ATA). Let's say we're breathing Enriched Air Nitrox with 32% oxygen (EAN 32) on the surface at 1 ATA. Dalton's Law tells us that 32% of that 1 ATA is caused by oxygen. So, the partial pressure of oxygen (pO_2) is 32% of 1, or 0.32 ATA. The partial pressure of nitrogen (pN_2) is 68% of 1, or 0.68 ATA. If you add up the pN_2 (0.68 ATA) and the pO_2 (0.32 ATA), you get 1 ATA, which is the total pressure of the gas being delivered to us on the surface.

$$\begin{array}{rl} & 0.68 \text{ ATA } pN_2 \\ + & \underline{0.32 \text{ ATA } pO_2} \\ & 1.00 \text{ ATA} \end{array}$$

Now let's dive that EAN 32 to 99 feet of sea water (FSW). We learned in dive class that for every 33 FSW, the pressure increases by one atmosphere, which gives us 3 atmospheres at 99 FSW. To keep the physicists happy, we'll also count the 1 atmosphere of pressure caused by the air above us. So, we find that we're under a total of 4 ATA of pressure at 99 FSW. The partial pressures of the gases in our mixture, then, are:

$$\begin{array}{rl} 0.68 \text{ x } 4 \text{ ATA} = & 2.72 \text{ ATA } pN_2 \\ 0.32 \text{ x } 4 \text{ ATA} = & \underline{1.28 \text{ ATA } pO_2} \\ & 4.00 \text{ ATA} \end{array}$$

Since we're not doing anything to alter the gas mixture, the percentages of N_2 and O_2 don't change with depth. However, the partial pressures of these gases DO change, and that's where the rubber meets the road in diving physiology.

Enter Henry's Law, which says that at a given temperature, the amount of gas that will dissolve in a liquid is directly proportional to the *partial pressure* of that gas on the liquid. By "on the liquid", we mean literally the pressure of the gas being exerted on the surface of the liquid. In our bodies, the gas/liquid interface occurs in the tiny air sacs, or alveoli, of the lungs. The alveolar membrane is so thin that effectively, the gas that we breathe is in direct contact with our blood. So, the higher the partial pressure of a gas, the more of that gas will dissolve in our blood.

In normal diving, we don't usually consider the effect of oxygen dissolving in the blood. Oxygen is a metabolic gas and is rapidly consumed. Nitrogen gas, on the other hand, is not used by the body. At depth, the nitrogen in our breathing gas starts to dissolve in our blood plasma via the alveoli. The plasma carries it to the rest of the body, where it dissolves in the tissues at various speeds depending on the composition of the tissue. Nitrogen is highly fat soluble, so it dissolves well in lipid-containing tissue like myelin (the sheaths around our nerve cells) and adipose tissue. It dissolves slowly in tissues like bone, tendons and ligaments. The deeper we go and the longer we stay at a given depth, the more nitrogen is dissolved.

When we ascend, the pressure of the gas mixture in our lungs starts to decrease. Henry's Law again comes into play here – as we decrease the pN_2 *on the liquid* (our blood plasma), we decrease the amount of nitrogen that can stay dissolved. The key here is to keep the pressure gradient (the difference between the pN_2 in our body and the pN_2 in our lungs) shallow enough that the nitrogen exits the body the same way it entered – through the lungs. If the difference is too large, the pressure gradient becomes steep and the dissolved nitrogen doesn't have time to exit the blood through the lungs.

Pop goes the top!

The next time you reach for your favorite carbonated beverage, take a look at it before you open it. Unless you've shaken it, you'll notice that you don't see any bubbles in it. Only after the cap is removed, and the carbon dioxide that's trapped under the cap is released, do the bubbles form. An overly large pressure gradient between the pN_2 (body) and the pN_2 (lungs) sets up a very similar situation in the body. Instead of forming in our soda or beer, though, the bubbles form in our body tissues. Sometimes this is because the diver makes an obvious error and either ascends too quickly or misses a required decompression stop. This is known as a "provoked hit" (or, in more judgmental parlance, a "deserved hit"). Other times, the diver is stricken despite completing his or her decompression properly or remaining within no-decompression limits. This is known as an "unprovoked" or "undeserved" hit. In either case, the bubbles that come out of solution in the tissues start causing problems. This is known as decompression sickness, or DCS.

Bubble trouble.

The location of the bubbles will determine what the diver's symptoms are. Classically, DCS symptoms are divided into two categories, Type I and Type II, based on the severity of the symptoms and the areas of the body affected. Type I symptoms are less severe and include joint pain, itching skin, reddened, hive-like skin rash and swelling related to bubbles in the lymphatic system. Type II symptoms are more severe and include brain and spinal cord symptoms like paralysis and numbness; pulmonary DCS, or "chokes"; inner ear DCS, or "staggers"; pain in the trunk or head; and *cutis marmorata*. Cutis marmorata means "marbling of the skin" and appears as reddish or bluish streaks against pale skin. It isn't particularly severe in itself but it can be accompanied by more severe symptoms so it's historically been categorized as a Type II symptom.

The Type I/Type II designation has been used by the U.S. Navy for many years to help determine the appropriate treatment for their injured divers. According to the U.S. Navy Diving Manual, the hyperbaric treatment for uncomplicated, quickly-resolving Type I symptoms is about 2 ½ hours shorter than the treatment for Type II symptoms. This works very well for Navy divers, who frequently dive with recompression chambers just steps away from the dive station. In clinical practice in civilian hospitals, however, it's often impractical to differentiate between Type I and Type II symptoms. For example, here at Duke, most of the divers we see come in from the coast of North Carolina two or more hours' drive from our location in Durham. There's also often a delay before the diver decides to seek treatment, so we frequently see divers 8 or more hours after they initially notice their symptoms. By this time, the bubble is probably resolved but the inflammatory effects of the bubble remain and may even make the symptoms worse. The longer treatment is delayed, the more difficult it is to treat the injury. For that reason, we'll normally use the longer treatment protocol no matter what symptoms the diver feels.

Symptom onset for DCS can vary widely. Some divers report DCS symptoms soon after surfacing; others may not feel anything for a few hours. Either way, if a diver feels any symptoms that he/she believes might be DCS, it's crucial that he/she be evaluated as soon as possible. Making a repetitive dive while feeling DCS symptoms is especially risky since it adds to the diver's nitrogen load. This makes that pN_2 (body)/pN_2 (lung) pressure gradient we spoke of earlier much, much steeper and increases the chance of more severe symptoms appearing after the repetitive dive.

My lungs do WHAT?

Another thing we all learned in diving class is that pressure and volume are inversely related; that is, if one goes up, the other must go down. Let's use a free diver as an example. If our diver takes a breath on the surface and then descends, her lung volume will decrease proportionally to the amount of pressure that's on her body. At 33 FSW, she's at twice atmospheric pressure so her lungs are one-half their original volume. At 66 FSW, she's at three times atmospheric pressure so her lung volume has decreased to one-third its original amount. If she's still going at 99 FSW, she's at four times atmospheric

pressure so her lungs have decreased to one quarter their original size. This inverse relationship between pressure and volume is known as Boyle's Law.

Now let's outfit our free diver with a tank and a regulator. She no longer needs to hold her breath and instead can inflate her lungs normally against the ambient water pressure. If, however, she experiences an emergency at depth and holds her breath on ascent, Boyle's Law comes into play again. The fixed amount of gas in her lungs, caused by her closed glottis as she holds her breath, will increase in volume as the pressure around her decreases with her ascent. If she fails to vent this expanding gas via her mouth or nose, it will eventually force air through her alveolar membrane and/or cause her lungs to rupture. The gas, some of which is now outside her lungs, can go several different places. It can remain trapped in her chest between her lungs in what's known as mediastinal emphysema; it can migrate up her trachea and lodge under the skin of her neck and shoulders in a condition called subcutaneous emphysema; it can lodge between the lung and chest wall and cause a pneumothorax; or it can enter the pulmonary circulation, where it's carried back to the heart and may be pumped out to the carotid arteries, causing stroke-like symptoms, or the coronary arteries, causing heart-attack-like symptoms. This is known as arterial gas embolism, and of the pressure-related diving injuries, it's the most serious. A diver with arterial gas embolism usually experiences a sudden, dramatic onset of severe neurological symptoms like unconsciousness or altered level of consciousness, paralysis, weakness, and numbness.

But it's not quite that simple…

Even after a dive with only moderate decompression stress, bubbles can form in the venous blood. So-called "silent" bubbles have been well documented in the diving literature. Normally these bubbles are trapped by the circulation in the lungs (pulmonary circulation), where they are slowly eliminated during normal respiration. Sometimes, though, the bubbles can pass into the arterial circulation through a direct connection, or *shunt*, between the venous and arterial sides of the bloodstream. Shunts may be found in many areas of the body. If bubbles are shunted into an artery, they can move downstream and cause local tissue hypoxia (lack of oxygen) by physically blocking the circulation. A common area for shunts to occur is the heart, where a patent foramen ovale (a hole between the right and left atria) or other defect may allow blood to cross from the right (pulmonary) side to the left (systemic) side. Frequently, the brain and spinal cord are affected when this happens. In these cases, it can be difficult to tell whether a diver's symptoms are from decompression sickness or arterial gas embolism. A diagnosis of decompression illness (DCI) is often assigned when the etiology of a diving injury is unclear. DCI is also used as an umbrella term to describe arterial gas embolism and decompression sickness in general terms.

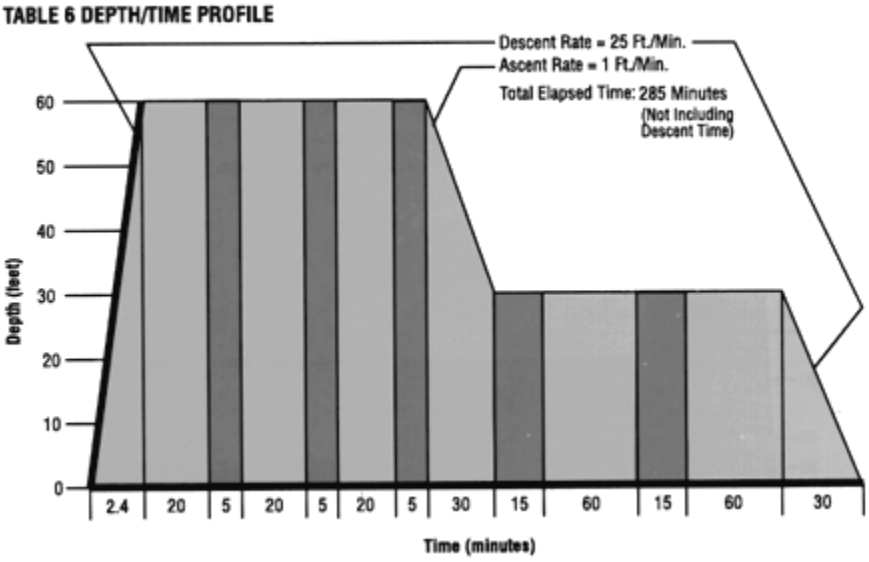
I'm bent, now what?

If a diver feels any unusual symptoms within twelve hours after surfacing from a dive, it's important to be evaluated by a diving physician to rule out DCI. Attention-grabbing symptoms like severe weakness or paralysis are normally strong motivators to seek help;

it's the more minor symptoms that are often ignored or denied until they either become serious or don't go away after a number of hours. We often see divers who have made repetitive dives despite feeling symptoms of decompression illness. This practice increases the diver's inert gas load and can lead to more severe symptoms. If you think you may have DCI, do NOT make another dive. Immediately call the emergency number on the back of your DiveAssure card, and Travel Guard will connect you with a Duke Dive Medicine physician who will assist you and answer any of your questions. Delay in recompression can reduce the effectiveness of hyperbaric therapy, so do not wait to call. Our service is available to you 24 hours a day, 7 days a week and is provided as part of your DiveAssure membership.

If our physician believes you may have decompression illness, he or she will recommend that you be evacuated to a hyperbaric facility for further evaluation. The Duke Dive Medicine physician will continue to be available for consultation throughout your treatment.

Divers with decompression illness are treated in a hyperbaric chamber. The treating physician will select the appropriate treatment protocol based on the diver's symptoms. The protocol most often used at Duke is the U.S. Navy Treatment Table 6, shown below excerpted from the U.S. Navy Diving Manual.



The diver will be carefully monitored for improvement throughout the treatment. More than one treatment may be required if symptoms are particularly severe or slow to resolve.

Many divers treated for DCS at Duke ask us, "Do I still have bubbles in me?" It isn't possible to rule out the presence of bubbles without ultrasound or other studies, but due to the extended transport times involved, in most cases the bubbles actually resolve either before the diver arrives at our facility or soon after treatment is begun. DCI symptoms are often not caused by the bubbles themselves, but by the after-effects of the bubbles.

Bubbles can activate the coagulation system and cause localized clotting. Foreign bodies in the circulatory system cause a cascade of events that may result in adhesion of white blood cells to the lining of the blood vessels and subsequent blood vessel damage. These effects impair circulation, which in turn may cause a diver with DCI to continue to feel symptoms even after the offending bubbles are gone. Hyperbaric oxygen therapy helps mitigate the inflammatory cascade, but again, the longer treatment is delayed, the more damage occurs and the less effective the treatment is.

Return to diving

A diver who has been treated for DCI should be evaluated by a board-certified diving physician before returning to diving. The diver should ensure that all treatment information is available to the physician so that he or she can make an informed recommendation. Individual ability to return to diving will vary greatly based on severity of the DCI incident, residual symptoms, and the results of any other studies that are performed.

Can I avoid DCI?

There are a few things that you can do to reduce your risk of DCI:

1. Dive within the scope of your training and ability, and practice your skills regularly.

2. Be familiar with your equipment.

3. Stay hydrated.

4. If you're on an extended dive vacation, take a "diving holiday" in the middle of the week. Do some sightseeing on land, or just snorkel.

5. Avoid diving with a recent soft tissue injury, as the impaired circulation to a joint can theoretically increase the risk of decompression sickness.

6. Know your diving environment, and prepare yourself to deal with poor visibility, current, waves, or cold water. Ensure that you have proper thermal protection – a cold diver holds onto dissolved gas better than a warm one, just like a soda goes flat more slowly when it's cold.

7. Stay in shape. Strenuous exercise during a dive (e.g. kicking against a strong current) can increase the risk of DCI.

8. Don't let your dive computer do all of your thinking for you. The vast majority of divers treated at Duke tell us that they stayed within the safe parameters specified by their dive computers. Be aware of any external influences that the computer can't account for like hydration and level of work, and adjust your diving accordingly.

9. Be mindful of your dive profile. The two biggest, and most controllable, risk factors for decompression sickness are depth and bottom time.

A wise man once said that the only way to avoid getting bent is to avoid diving. That's not a workable solution for most divers, so it's up to us to manage our own risks appropriately. With proper education, prudent diving practice and common sense, you can minimize your risk of DCI and enjoy your diving the way it's meant to be enjoyed.

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