

What you need to know about diving medicine but won't find in a textbook

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Abstract

The old adage that 'if a patient in your emergency department (ED) is wearing a wetsuit, fins and a mask, then he/she probably has a diving related illness' is one that should be remembered. This is an obvious statement that should not need stating; however, simple clues can be missed or disregarded. This article will address issues that may confront emergency physicians and for which there are few resources to find the answers. It aims to explain the reasons behind some of the advice given during consultation with a hyperbaric physician. The second aim is to bring emergency physicians up to date with new diving practices and how these may impact upon traditional diving injuries. To achieve these aims, this article is a compilation of answers to frequently asked or pertinent questions related to diving medicine.

Key words: *decompression illness, diving, oxygen, recompression.*

Can a diver get decompression illness despite diving within dive table limits?

There is a widely held belief that a diver cannot get decompression illness (DCI) while diving within dive table limits. Consequently, divers may not seek aid for symptoms and doctors may misinterpret them. Dive tables give no-decompression limits. These are the maximal times the diver can spend on the bottom without having to do a decompression stop (a short delay in ascent from depth to 'breathe off' accumulated nitrogen). More recently, dive computers have become popular and they adjust the no-decompression limits with every change in depth.

They also enable the diver or diving physician to download the dive profile onto a computer. In a similar way to the paracetamol toxicity continuum, where the nomogram treatment line has been drawn arbitrarily at a level where the risk of liver toxicity is thought to be 'acceptable', no-decompression limits have been designed to give guidelines where the risk of DCI is 'acceptable.' With the enormous amount of variability in every dive and diver, it is impossible to test the tables to evaluate the exact risk. So, the possibility of DCI should not be discounted from a differential diagnosis, just because a diver dived within their table or computer guidelines. Rapid or multiple ascents during the dive also invalidate the tables because

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they have been designed for a diver with a single, controlled ascent.

What is the difference between a decompression stop and a safety stop?

A decompression stop is a planned stop for a specific time, at a specific depth, as indicated by dive tables. This allows extra 'off gassing' of nitrogen when the limits of the no-decompression stop tables are exceeded. Alternatively, safety stops are recommended (but not compulsory) stops at 3 m or 5 m to add extra time for off gassing in divers who are within the limits of their no-decompression dive tables. There is good evidence that venous bubble numbers are dramatically reduced by safety stops.

Serious problems may occur if the stops are forgotten or missed, e.g. insufficient residual air supply for the duration of the stop. All commercial dive boats should have diving instructors familiar with missed decompression stop procedures. However, emergency departments could conceivably be contacted by local divers requesting advice. If these divers have missed a safety stop, they should be advised to watch themselves closely for symptoms of DCI and proceed immediately to hospital if any develop. If they have missed a decompression stop, they should be started on 100% oxygen as soon as possible, treated for 2 h and then observed. A diving physician should be consulted because, if the missed decompression was significant, the risks of developing DCI may be high enough to recommend evacuation to a hospital with a recompression chamber.

Are there any risks in retrieving a diver with decompression illness in a helicopter, if the helicopter stays under 300 m?

The 300 m guideline is not evidenced based and divers have had DCI precipitated by driving over hills of less than 300 m altitude on the same day as diving. This results from the drop in ambient pressure allowing intravascular bubbles to expand. However, the risk of this should be mitigated somewhat by the diver breathing 100% oxygen in the helicopter. This provides an increased gradient for gas within the bubbles to diffuse out. Similar to many treatment decisions in medicine, it may be necessary to weigh the risks of deterioration from altitude exposure with the benefits of earlier recompression if the air retrieval saves time.

Is it appropriate to use the administration of 100% oxygen as a diagnostic test for decompression illness?

I am aware of several incidences where 100% oxygen has been administered to divers with equivocal symptoms. The rationale is that if the 'sore shoulder' improves with oxygen then the diagnosis is DCI. If it does not improve, then the diagnosis is not DCI. Unfortunately, there is no support for this practice in the literature. If the diver improves, they still need recompression. If the diver does not improve, it does not exclude DCI and the diver may still need recompression. Most cases of equivocal musculoskeletal DCI present several days after the dive and are very unlikely to respond to 100% oxygen.

Can decompression illness be treated with oxygen alone?

There is no evidence to suggest that oxygen alone is as good a treatment as oxygen with recompression. Nor will there ever be a randomized, double-blind study comparing the two options. Recompression therapy is the gold standard treatment and other options should only be entertained when all attempts at obtaining a means to recompression have failed. With the almost universal availability of oxygen on dive boats presently, there is usually the opportunity to observe the effects of oxygen alone while transport to a hyperbaric facility occurs. A significant number of divers with severe DCI arrive at the chamber with minimal improvement on 100% oxygen. There is also a significant number who deteriorate when they are given a break from oxygen.

Does a diver still need to be recompressed if he/she improves on 100% oxygen?

Some physicians have argued that, if 100% oxygen is instituted immediately, the diver becomes asymptomatic and there is no recurrence after stopping oxygen, then there is no need for further treatment. However, several reports have shown that neurological injury may coexist with what initially appears to be only musculoskeletal DCI.^{1,2} Frequently, the attending neurological signs are subtle or may be neuropsychiatric in nature. Few emergency physicians have the time or skill to perform, or access to complicated neuropsychiatric testing. Failure to identify these subtle changes may result in the diver returning to

work with an inability to concentrate or analyse data that he/she was previously able to.

Treatment with 100% oxygen alone is an option which should be confined to divers in remote locations, who do not have access to in-water recompression, a recompression chamber or timely retrieval to one.

After what delay in presentation is it no longer worth recompressing a diver with decompression illness?

Divers often present several days after the onset of their symptoms. Most bubbles will have resolved spontaneously by then and the symptoms will be secondary to persisting inflammation. Recompression is beneficial even when the bubbles have resolved. Several years ago, multiple divers on a Singaporean fishing boat developed DCI and remained on the boat for up to a fortnight before reaching treatment. The mean time to treatment was 90 h. Despite this, about 80% had a significant neurological improvement.³ It is therefore reasonable to offer recompression to any diver who presents within weeks of their injury.

Can I discharge a diver, who developed sudden onset of neurological symptoms on surfacing, if they have completely recovered by the time they arrive in ED?

It is likely that the diver's symptoms were caused by intravascular gas secondary to pulmonary barotrauma. It is also likely that the dive master and later the paramedics will have kept the diver supine and administered 100% oxygen. The natural history of an arterial gas embolism (AGE) is one of sudden deterioration as the gas embolizes to the cerebral circulation. However, the air may subsequently redistribute to the venous system, resulting in the diving equivalent of the transient ischaemic attack. Relapse is common and can be more severe than the original presentation. Any diver with sudden onset of neurological symptoms on surfacing should be recompressed and observed for 24 h regardless of their apparent condition on arrival at the ED.

Why are divers with decompression illness coming back from the hyperbaric unit on lignocaine infusions?

Despite recompression, divers with DCI often do not make a full recovery, particularly in terms of higher-centre brain function, i.e. memory, mood, personality and concentration. Research is ongoing into adjuncts

to recompression that may reduce the incidence of residual symptoms. Recent work on the use of lignocaine as a cerebral protectant during cardiac surgery has shown fewer neuropsychiatric defects in the lignocaine group compared to placebo.⁴ The injuries of gas emboli from cardiac surgery and neurological DCI are similar enough to warrant the use of lignocaine infusions in severe neurological DCI until further research is available. Lignocaine infusions have no role in other forms of DCI.

Can decompression illness present as an isolated headache?

Headaches while diving are best classified according to whether they occur during or after a dive.

Headaches beginning during descent or ascent are usually due to ear or sinus barotrauma. Headaches starting while on the bottom are most commonly due to hypercarbia. Hypercarbia may result when excessive work underwater is combined with increasing depth (denser air) and a poorly maintained regulator that increases the effort of breathing. Such headaches can be associated with breathlessness and dizziness and can persist for hours after the dive. Migraines can be precipitated by diving and are another common cause of headaches coming on during the dive.

Headaches coming on after diving are usually unrelated to the dive and alternative diagnoses should be sought. Isolated headache is a very rare presentation of DCI. Recompression is usually not offered in the absence of other symptoms.

Can a diver get decompression illness from breath-hold diving or snorkelling?

Decompression illness can result from breath-hold diving but it is rare and would need depths only accessible to professional breath-hold divers. Calculations using dive tables suggest that repeated breath-hold dives can safely be made to 20 m, as long as the surface interval is greater or equal to the bottom time of each dive. Decompression illness from nitrogen bubbles can occur after only 2 h of repetitive breath-hold diving if the surface intervals are half the duration of the bottom time.⁷

Arterial gas embolism can also occur despite the fact that the diver should arrive back on the surface with the same volume of air as when he/she left. Possible explanations include local air trapping, reduced compliance of the lungs with immersion and

embolization of venous bubbles across a right to left shunt. So, remembering the rule I presented in the introduction, you will note that no mention of compressed air cylinders being present has been made. Just because the patient in a wetsuit in your resuscitation area doesn't have a cylinder attached to his back, doesn't mean he can't have DCI!

Can vertigo, commencing after a dive, be related to the dive?

Vertigo related to diving is also best interpreted by its temporal relationship to the dive. Vertigo commencing during descent suggests tympanic membrane rupture, inner ear barotrauma, alternobaric vertigo or benign positional vertigo (although it's hardly benign if swimming down an anchor line 20 m under the water). Alternobaric vertigo usually lasts only a few seconds and occurs in divers with difficulty equalizing who develop unequal middle ear pressures. It is more common during ascent than descent. Vertigo occurring on the bottom suggests hypercarbia or benign positional vertigo while vertigo occurring during ascent suggests alternobaric vertigo or DCI.

Vertigo presenting soon after diving suggests either inner ear barotrauma or DCI. Inner ear barotrauma may result in round or oval window rupture. It most commonly results from overly vigorous attempts at the Valsalva manoeuvre. Presentation may be with any combination of vertigo, deafness and tinnitus and may be delayed until the diver does a Valsalva soon after the dive such as would occur with heavy lifting.

Decompression illness affecting the inner ear alone is very uncommon in sports divers breathing air but does occur in oxygen-helium diving. Vertigo which persists until presentation at the ED is therefore most likely from inner ear barotrauma, DCI in heliox divers or tympanic membrane rupture.

Are there good data to justify our recommendations on diving again after an episode of decompression illness?

Current recommendations suggest that a diver can dive again after a month if he/she has made a full recovery after treatment, the dive was consistent with the occurrence of DCI, and there was no pulmonary barotrauma. Unfortunately, many divers with DCI present late or not at all for fear that they will be advised by the doctor never to dive again. There is

little evidence to suggest that a previous episode of DCI predisposes to subsequent episodes. The only evidence comes from data showing that more divers with DCI have had it previously than would be expected. However, there are multiple other explanations for this. For example, these divers were shown to dive more frequently and more aggressively than the average diver.

The recommendation that divers should not dive again if they have residual symptoms is based on anecdotal case reports where divers with residual symptoms have presented with more severe symptoms with a subsequent episode of DCI. There is also a theory that the central nervous system has an intrinsic functional reserve that allows for a degree of injury without showing functional problems. Once this neurological reserve has been exceeded, however, subsequent injury may result in more severe defects.

A predisposition to DCI is suggested if the occurrence of DCI was unlikely, given the dive profile. Accordingly, the decision to recommend no further diving is a difficult one and should only be made by an experienced diving physician. It may involve the search for a patent foramen ovale in a diver who suffered severe neurological DCI.

Alternatively, pulmonary barotrauma is an absolute contraindication to diving because the resultant lung scarring theoretically predisposes to further lung injury with pressure changes. I am not aware of any reports that have specifically investigated DCI incidence in divers with previous barotrauma.

What is the difference between a middle ear squeeze and a reverse squeeze?

Divers commonly refer to the pain of middle ear barotrauma as 'squeeze'. A middle ear squeeze occurs on descent if the diver is unable to equalize pressures on both sides of the tympanic membrane. The increasing pressure, as the diver descends, reduces the volume in the middle ear cavity and the eardrum initially retracts inwards. This may be followed by mucosal swelling, bleeding and possibly inward rupture of the tympanic membrane. Deafness and vertigo can occasionally occur but, if present in the absence of a ruptured tympanic membrane, mandates the exclusion of inner ear barotrauma.

In contrast, ear pain on ascent is called a 'reverse squeeze' and most commonly occurs in divers who have used decongestants to clear their eustachian tubes before they dive. If the decongestants wear off

during the dive, the expanding air cannot escape on ascent. If bad enough, this can eventually lead to outward rupture of the tympanic membrane. Attempts at the Valsalva manoeuvre, to correct the symptoms, should not be performed.

How long after ear barotrauma can diving be resumed?

Diving can be resumed when there are no ongoing symptoms, inner ear barotrauma and a perforated membrane have been excluded, counselling about equalization techniques has occurred and both ears equalize during a Valsalva manoeuvre.

Do you need to recompress a diver after an episode of near-drowning?

In theory, near-drowning should increase the risk of DCI by decreasing inert gas elimination from tissues due to the reduced cardiac output and increased intrapulmonary shunting. However, there is no *in vivo* research to support this. The best evidence is recent research using probabilistic decompression modelling. This showed that divers who have been resuscitated from a cardiac arrest, or are severely shocked at presentation, have a risk of DCI between 25 and 52%.⁵ Calculations using a normal cardiac output with no shunting showed a maximal risk of 1.6%. Recompression is therefore indicated for divers with near-drowning if they have had a cardiac arrest or are shocked at presentation. All others should be admitted for a minimum of 6 h and observed closely for signs of developing DCI or pulmonary oedema.

Can nitrogen narcosis cause loss of consciousness underwater or cause symptoms after the dive?

The increased partial pressure of inspired nitrogen at depth may precipitate a narcotic-like effect. Most air divers notice this 'nitrogen narcosis' at depths of about 30 m and by 70 m it is usually incapacitating. A rapid descent usually causes a greater narcotic effect than a slower one. However, the effects are rapidly reversible and do not cause symptoms upon surfacing. If a diver surfaces and is behaving strangely, an alternative diagnosis should be sought. Similarly, it is very rare for nitrogen narcosis to cause loss of consciousness at depth. There are multiple other causes of this disastrous event and if it has occurred at depths greater than 70 m, oxygen toxicity is much more likely

to be the cause. The danger of nitrogen narcosis is that it predisposes to other diving injuries because of its associated loss of judgement.

Does diving-induced pulmonary oedema only occur in very cold water?

It was traditionally thought that diving-induced pulmonary oedema was due to the intense vasoconstriction associated with immersion in very cold water. Recent evidence suggests that this is not so and other factors are involved. In fact, there is a recent report of pulmonary oedema occurring in a diver in a warm swimming pool.⁶ A combination of factors are probably involved including vasoconstriction from cold and/or elevated partial pressures of oxygen, increased cardiac output, increase in pulmonary vascular pressures and increased preload from immersion. Cardiac enzymes are normal. Diving-induced pulmonary oedema should only be diagnosed once other causes of shortness of breath have been excluded. These include all the non-diving related causes as well as pulmonary DCI ('the chokes'), AGE, pneumothorax, pneumomediastinum and salt water aspiration syndrome.

Can a diver get oxygen toxicity when diving on air?

Oxygen toxicity in mixed gas diving is discussed below. Mixed gas divers are usually well trained in the hazards of oxygen toxicity and its avoidance. However, this is not the case for recreational air divers as this toxicity should not occur at depths recommended for safe air diving. Unfortunately, these depth limits are frequently exceeded.

Oxygen toxicity can either effect the brain (Paul Bert effect) or the lung (Lorrain-Smith effect). Pulmonary toxicity is not seen in divers because it is related to the duration rather than the magnitude of exposure. Neurological oxygen toxicity, however, can occur while diving and is devastating because the initial manifestation is often a grand mal seizure. The risk of neurological oxygen toxicity increases with the pressure of oxygen breathed and the duration of exposure. Early research that examined military volunteers swimming deeper and deeper until they seized, suggested that the oxygen partial pressure threshold for a seizure was 1.6 bar (1.0 bar = 1 atmosphere (ATA) = 0.01 kPa). Many mixed gas divers now limit their oxygen exposure to 1.4 bar. An air diver at 30 m depth is breathing 20% oxygen and is supplied by a regulator at 4.0 bar (sea level is 1.0 bar

and each 10 m underwater is an additional 1.0 bar). This equates to an inspired oxygen pressure of 0.8 bar. However, this increases to 1.6 bar if the diver descends to 70 m (8.0 bar). A seizure at 70 m is often, if not usually, fatal yet some air divers have certainly pushed these limits. They not only put themselves at risk but also their dive 'buddy' who must attempt to retrieve them to the surface quickly. I have personally been involved with divers where one required treatment for near-drowning and AGE secondary to a seizure, while his buddy required treatment for severe DCI after, not surprisingly, ascending too rapidly during the rescue.

What is mixed gas diving?

In attempts to go deeper for longer, mixed gas or 'technical' diving is becoming increasingly popular. The most common form of mixed gas diving is *nitrox diving* (otherwise known as enriched-air diving). Air is 'enriched' with oxygen, resulting in a lower concentration of nitrogen. The advantages of this are longer bottom times because of the reduced risk of DCI. The disadvantages are the higher risk of oxygen toxicity. Nitrox is therefore good for more prolonged but shallow dives. The medical implications involve the risks of oxygen toxicity if safe depths are exceeded.

The way around the oxygen toxicity problem is to add a third gas, helium. Helium has several benefits. It is less dense and therefore reduces the work of breathing and is less narcotic than nitrogen (it's fun to be mildly euphoric from nitrogen narcosis at 30 m but devastating to be severely intoxicated at 70 m). It also reduces the concentration of oxygen therefore lessening the risk of oxygen toxicity. At some depths, the diver will be breathing a gas mix which, if breathed at the surface, would have less oxygen than air. For example, if a diver is planning to go to 90 m (10.0 bar), the maximum oxygen levels in the gas mix would be 14%. Obviously, this gas mixture cannot be breathed in shallow waters because it would result in hypoxia. This problem is solved by having two or more different cylinders containing different premixed gas mixes. However, diving accidents can occur if the diver accidentally breathes from the wrong cylinder.

Alternatively, the diver can use a rebreather system where a computer calculates ambient gas pressures and provides the diver with the appropriate oxygen concentration for that depth. Recently, during a record-breaking wreck dive to 175 m off Brisbane, the divers (one of whom was the medical director of the Wesley

Centre for Hyperbaric Medicine.) were breathing a gas mix on the bottom containing 7% oxygen.

Rebreathers also allow the continued breathing of the original gas. They require the incorporation of a CO₂ scrubber system and a gas addition system to add more oxygen, as required. The advantage for the diver is that the system produces no bubbles (allowing them to get much closer to marine life), dive duration is increased, gas consumption is much less (allowing the carriage of smaller gas cylinders) and greater depths can be achieved. From a medical perspective, the dangers of hypoxia or hyperoxia predispose to loss of consciousness under water. There is also a risk of hypercarbia if the CO₂ reabsorbant system malfunctions. If DCI does occur, it is often neurological with early onset of symptoms and a rapid deterioration.

Summary

Scuba diving has changed dramatically over the last few years with the advent of technical diving. Technical divers are exposed to different dysbaric risks than air divers. The differential diagnosis for a patient with symptoms developing within 48 h of diving should always include DCI. Normobaric oxygen is a mandatory part of first aid for DCI but is not a diagnostic test or a definitive treatment. Headache and vertigo related to diving are best diagnosed by reviewing their temporal relationship to the dive.

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