PULMONARY FITNESS TO DIVE

James Francis

Key Words

Asthma, diving medicals, fitness to dive, medical conditions, pulmonary barotrauma.

Abstract

Determining pulmonary fitness to dive is not a scientific process. While it may be possible to make an informed judgment on the likely effect of diving on pre-existing disease, predicting the risk of diving diseases caused by pre-existing disease is not yet possible with confidence. This is because our understanding of the pathogenesis of the major diving diseases, decompression illness (DCI) and pulmonary barotrauma (PBT), is incomplete. The situation is made more difficult because even measurement of the principle function of the lung and circulation, aerobic capacity, is poorly standardised outside physiology laboratories and there is no agreed aerobic standard for diving. The determination of pulmonary fitness to dive therefore relies upon the physician being able to make a reasonable assessment of the physical fitness of the candidate and be aware of what is known about the causes of PBT and DCI. From this baseline a judgment can be made. The conditions that should preclude diving include: those with intrinsic or poorly controlled asthma or asthmatics with a reduced peak flow; recent spontaneous pneumothorax; bullous disease detected on chest X-ray and significant lung parenchymal or pleural scarring.

Introduction

Determining fitness to dive, like much of medicine, is not a precise science. While we can make a fairly educated judgment on the likely effect of diving on some pre-existing conditions, we are a long way from being able to do the reverse with any degree of confidence. Not only is our knowledge of the pathophysiology of the diving disorders incomplete, but our understanding of the risk factors for important diving-related disease, such as pulmonary barotrauma and decompression illness, is still at a primitive stage.

The are a number of reasons for this. The most important, in my view, is inadequate epidemiology. It is only in the last ten years or so that serious attempts have been made to collect diving accident data systematically. This is not a criticism of past generations of diving physicians. The reason for our lack of epidemiological data is that, until about ten years ago, there was a lack of the widely available, user-friendly software and hardware that are necessary to design and run sophisticated databases. Dissecting diving accidents into fields for databases is a time-consuming process. A good database will include fields for: personal characteristics of the diver, past medical and diving history, the dive profile(s) prior to the causative dive, unusual features of the dive such as cold water, lost weight belt or out of air, the onset and progression of each of the manifestations, any delay to treatment, the first aid and subsequent management the casualty received and the outcome. This is a staggering amount of information to try to process. The Royal Navy's Institute of Naval Medicine (INM) database, for example, has over 100 fields for each case and now contains many thousands of cases. Despite this effort, we are still some way from identifying the natural syndromes of disease, let alone their risk factors.

A substantial problem with collecting data in this way is that there are no denominators with which to compare the numerators – how many uneventful dives to 30 m are there? How many uninjured divers have lung parenchymal scarring? It will be for the likes of Dick Vann and his colleagues at the Diver Alert Network (DAN) with their prospective studies, using down-loads from recreational divers' computers, to answer some of our basic questions.

Another fundamental problem is that inadequate resources have been devoted to basic physiological research in the field of diving medicine. This is a problem that has been getting more severe since the ending of the Cold War and the advances that have been made in the capabilities of remotely operated vehicles underwater. No longer are the navies of the world or the oil companies willing to sponsor such studies. Having said that, I am delighted to say that the US Navy will shortly be making available substantial amounts of money for precisely such work in a program that will last for multiple years. This money will not be ring-fenced for US Navy scientists or even American universities, but the Office of Naval Research will consider funding proposals from other countries. So, in a few years, I hope we will be less able to use the fig leaf of lack of funds to excuse our lack of knowledge.

I have chosen to introduce my talk in this way to make the point that what follows is not science. What I intend to do is review the conditions that we are trying to prevent and discuss some of the strategies we employ to try to achieve this.

Lung function testing in diving

We can look at lung function testing with the view to checking that a diver can achieve adequate ventilation during exercise. We wish to avoid pulmonary barotrauma and testing for this risk is mostly based on theoretical grounds of obstruction as there is little hard data about why divers develop pulmonary barotrauma. Then there are physiological changes combined with behavioural patterns which can cause PBT.

Exercise Testing

The most important function of the heart and lungs in a diver, just as with anybody else, is to provide body tissues with an adequate supply of oxygen and remove their metabolic waste, mainly carbon dioxide. Failure to achieve this will result in the diver becoming incapacitated in the water and may lead on to the ultimate failure of the heart and lungs, drowning.

Although this sounds like a fairly specific requirement it is not. The demands made on the cardiopulmonary system will depend on what the diver does during the dive. There is a world of difference in the cardiopulmonary function required by a US Navy SEAL or RN Special Boat Service diver conducting a five mile covert swim in 4°C water compared with a timid tourist hovering just under the surface, taking in the delights of the Great Barrier Reef. Even within sports diving there is a considerable spectrum of activity from the hairy-chested tech diver exploring the RMS Lusitania at 90 m to the novice diver learning buoyancy control in a swimming pool. Furthermore, there is a wide age range in recreational divers. A standard that might be reasonable for a twenty year old to achieve is likely to be unreasonable for a fifty or sixty year old. Given this great disparity of demand on the cardiopulmonary system is it reasonable or even possible to determine a "fitness standard" for divers?

One way of looking at the problem is to assess the VO_2 required to swim at certain speeds.[1] Table 1 shows swimming speed and the VO_2 required to achieve these speeds.

TABLE 1

SWIMMING SPEED AND VO_2

Speed (knots)	VO_2 (l/min)
0.6	1.0
1.0	1.7
1.2	2.4

It can be seen that swimming at 1.2 knots requires a considerable VO_2 which equates to a V_E of about 80 l/minute at the surface. However, with increasing depth the air breathed will become more dense with a consequent increase in the work of breathing and the VO_2 required. Thus, even stipulating a swimming speed standard is imprecise because the demand it will make on the cardiopulmonary system will depend on the diver's depth and other factors such as what the diver is wearing. However, a more basic question is how fast does a diver need to be able to swim? This gets us back to the issue of what kind of diving does the individual intend to do. Setting a standard based on an arbitrary swimming speed, therefore, has severe limitations in terms of practicality with respect to where the standard is set. A bigger problem, however, is how should an index of that standard, the VO_2, be measured?

The UK Health and Safety Executive, which regulates the medical standards for UK commercial divers recognised this problem and decided to specify no formal fitness standard in the regulations issued in 1998.[2] Instead, these state:

"A commercial diver must be able to meet the physical requirements of the task to be performed. That includes the ability to rescue a stricken diver and effect a rapid recovery. An assessment of exercise capacity must be carried out at both the preliminary examination and each subsequent annual assessment."

The guidance goes on to suggest that a step, swimming or cycle ergometer test may be appropriate means of determining adequate fitness. The guidance gets close to specifying Bove's suggested standard of 13 Mets,[3] but backs off and notes in the appendix that:

"without being too prescriptive, it is important that some form of standardised exercise test is performed."

The UK Sport Diving Medical Committee, which determines fitness standards for most UK sports divers, states:

"The value of screening exercise tests in apparently normal populations has now been largely discredited because of the appreciable false positive and false negative results in such groups. Furthermore we have no control over the quality of equipment or type of standardisation on which the Exercise ECGs on our members would be performed. This only compounds the possibility of false reporting of the test."

Our own society, SPUMS, also takes a pragmatic view:

"Consideration must be given to the candidate having adequate reserves of physical fitness to cope with unexpected demands inflicted by adverse weather or sea conditions, surfacing away from a boat, having to aid a distressed buddy, or other emergencies."

In conclusion, while responsible bodies recognise that an adequate level of exercise tolerance is necessary for all divers, the practical difficulties in setting a standard and reproducibly measuring the fitness of diver candidates makes this a qualitative rather than a quantitative assessment.

Causes of pulmonary barotrauma

We all know it is important that there is free communication between the pulmonary air spaces and the

mouth, particularly during ascent. An airway with an obstruction that prevents air distal to it escaping from the lung has the potential to cause barotrauma. As the volume of trapped gas expands, it eventually causes the parenchyma to expand beyond its elastic limit and break. Let us look at what can cause such an obstruction.

PERMANENT AIRWAY OBSTRUCTION

If an airway is permanently obstructed the tissues distal to it are unlikely to suffer barotrauma. This is because these tissues, if they contain gas, will behave like the bowel during a dive, with its volume decreasing during descent and returning more-or-less to their original volume during ascent. More often than not, the air in lung tissue distal to a permanent obstruction will be absorbed and so again, barotrauma will not be a problem.

LABILE AIRWAY OBSTRUCTION

Here we do have a potential problem, particularly if the obstruction occurs at depth. What could cause such an obstruction?

Bronchospasm may be provoked by exercise, cold air, dry air or by the stress of an emergency, such as being out of air. Another potential cause is nebulised saline which may be injected into the airways by a faulty regulator.

Tumours. The remarkable case described by Liebow et al. has alerted us to the possibility of a ball-valve action by a calcified mass.[4] However, careful reading of this case, a submariner undergoing escape training, leaves some questions unanswered. For example, the calcified mass was in the left lung and yet there was evidence of barotrauma affecting both lungs. Thus the relevance of the calcified mass is open to question. Unsworth reported a very rare case of pulmonary barotrauma occurring in a compression chamber following a session of hyperbaric oxygen therapy.[5] The patient was found to have a neoplastic mass in the right middle lobe and, given the rarity of PBT in compression chambers, the possible role of the mass in generating the injury cannot be ignored.

Mucus. An argument against asthmatics diving is that tenacious mucus could obstruct an airway. Here we enter a Catch 22 situation because, to my knowledge, no case has been reported in which mucus plugging was shown to be the cause of pulmonary barotrauma. There is a reason for this, of course, and that is that for many years asthmatics were supposed to be banned from diving. However, things are changing and in the UK some asthmatics are allowed to dive, as I shall discuss later. Patrick Farrell is in the process of completing a study of diving asthmatics and this should be complete in about six months. It will be interesting to see if they are at increased risk of PBT. One point that should be borne in mind is that asthmatics have made many millions of flights in commercial aircraft. These commonly run at a cabin altitude of 8,000 ft or 196 mmHg below 1 ATA, a rather greater excursion than the 70 mmHg or so that is needed to rupture a normal lung. If mucus plugging was a serious risk factor, I would expect there to have been many more examples of PBT in airline passengers than have been reported.

PARTIAL AIRWAY OBSTRUCTION

Air can escape from normal lung at a rate that even the fastest of ascents can be conducted safely. In submarine escape training, for example, ascent rates of over 3 m per second can be reached wearing the Beaufort Submarine Escape and Immersion Equipment. This requires that the airways communicate with the mouth such that over the last 10 m the volume of the lung can be exhaled in about three seconds. For comparison, a healthy person can exhale about 75% of vital capacity in a second. In a forced expiratory manoeuvre the rate at which air can be exhaled decreases with time as the small airways close. During the last 10 metres of a submarine escape, air will be expanding rapidly within the lung and thus the airways will not close and so this high rate of exhalation can be maintained. A problem may occur if even a small part of the lung, such as a bullus, can not empty at the required rate. In a rapid ascent, particularly near the surface, it may reach its elastic limit and rupture. The problem is that small bullae are common, impossible to detect clinically and may be invisible or easily overlooked on a p-a chest X-ray. There must be many thousands of people with bullae who dive without difficulty. Thus, the extent to which this is a genuine hazard is in some question.

SMALL OR STIFF LUNGS

Lungs break or tear when they reach their limit of elasticity. When breathing in, the elasticity of the lungs decreases as total lung capacity (TLC) is approached. The lungs are vulnerable to over inflation because they become stiff and eventually break. In a study of barotrauma in submarine escape trainees Benton et al. found that it is not trainees with evidence of obstruction on spirometry, lower than predicted forced expiratory volume in 1 second ($FEV_{1)}$ or FEV_1/FVC (forced vital capacity) ratio, who were over represented amongst those with barotrauma, but those with evidence of restriction (smaller than predicted FVC).[6] This is supported by the original work of Colebatch et al. who showed that reduced compliance is a risk factor for PBT.[7,8]

LUNG PARENCHYMAL SCARRING

In the only study of its kind that I am aware of, Calder looked for scarring in the lungs of deceased divers with

pulmonary barotrauma.[9] He found that victims of PBT frequently had lung parenchymal scarring but, interestingly, the site of lung rupture was usually remote from the scarring. David Denison and I concluded that parenchymal scarring may provoke lung rupture by effectively shortening the elastic fibres that radiate from the hilum to the pleura. If fibrosis renders a section non-elastic, when stretched the remaining elastin will reach its elastic limit sooner and may rupture. This would render the elastic fibres in series with an area of fibrosis vulnerable to pulmonary over-inflation and thus it is they, rather than the scar that will break.[10]

PLEURAL ADHESIONS

It was Malhotra and Wright who first observed that pulmonary adhesions are a risk factor for pulmonary barotrauma.[11] In their classic study of fresh cadavers they showed that lungs ruptured where the visceral and parietal pleura were connected by adhesions. Interestingly, in the world of aviation, air crew who have had spontaneous pneumothoraces can be made fit to fly by undergoing pleurectomy. This will render the lung more-or-less completely adherent to the chest wall. This substantially reduces the risk of a recurrent pneumothorax. The hazard of pleural adhesions may be that they focus the stress of pulmonary over-inflation at the site of the adhesion. If this has a small surface area, the force acting on the lung may be sufficient to cause it to tear.

Behavioural and physical factors

For completeness, it is perhaps worth just mentioning some behavioural and physical factors that may predispose a diver to pulmonary barotrauma. The most obvious is voluntary breath-holding during ascent. This is often associated with panic after a mishap and is most common in inexperienced divers. It is usually addressed by training. It is perhaps worth mentioning the possible role of involuntary breath-holding. It is striking that in a substantial proportion of cases of pulmonary barotrauma in submarine escape trainees, no cause is found. When close to the surface, the rate of change of pressure is about 250 mmHg per second. A person with their lung volume close to TLC would only have to hold their breath for about a third of a second to generate an overpressure that may be sufficient to cause lung rupture. This could happen if they coughed, hiccuped or sneezed.

Head-out immersion in water results in a shift of about a litre of blood from the periphery into the chest. A diver, who is submerged vertically in the water, and has a regulator in his or her mouth is in a similar situation. One effect of this shift in blood volume is that full capacitance vessels splint the lung and reduce its compliance. It may therefore be that an inescapable consequence of diving is to make the lungs more vulnerable to pulmonary barotrauma than if similar pressure changes were experienced out of water. Other factors are involved, but this may be one reason why pulmonary barotrauma is rare in aviators and those who undergo pressure excursions in compression chambers.

As has already been discussed, the compliance of the lung is at its lowest at TLC. Thus the lung is more vulnerable to a reduction in ambient pressure at TLC than when it contains less gas. It is perhaps surprising that divers ever skip breathe. Unlike normal breathing in which the sequence is breathe in, breathe out, hold; skip breathers breathe in, hold, and then breathe out. It is a practice in which the lung is deliberately held fully inflated, and at its most vulnerable to barotrauma, for as long as possible. There may be some advantage with respect to gas usage or a psychological comfort of having lungs filled with air, but from a physiological perspective it makes no sense. Even modest loss of buoyancy control while skip breathing renders the diver vulnerable to pulmonary barotrauma.

Pulmonary fitness to dive

Having explored what the lung is for and why it may fail underwater, can we come up with some pulmonary standards for divers? We have already concluded that this is not possible in a quantifiable way with respect to assessing physical fitness. What about other aspects of lung structure and function?

ASTHMA

In the UK, a more relaxed attitude is developing towards asthma and diving. Rather than a blanket prohibition, which was always porous to dedicated divers, the following position has been adopted. Those with allergic asthma who are well maintained on inhaled steroids or chromoglycate, and only need to use a bronchodilator occasionally, may dive. They are advised to take a peak flow measurement twice a day in the diving season and if the result is within 10% of their usual best, they may dive. If not, they should wait until the result has been within 10% of their usual best for 48 hours. A $Beta_2$ agonist can be taken prior to diving as a preventative measure but not to relieve symptoms. Those whose asthma is provoked by exercise, stress or cold air are not considered fit to dive. This strikes me as a balanced approach.

LUNG FUNCTION TESTS

What is the role of lung function tests in determining fitness to dive? Speaking as somebody who has put literally hundreds of prospective and practicing divers through the lung function laboratory, and making only a tiny percentage of them unfit to dive on the basis of their lung function tests, I am forced to concede that the role is limited. There are

two reasons for this. If a standard is to be imposed:

a It has to be measured in a standard way.

b It has to be relevant.

As we have seen with respect to exercise testing, standardising tests is difficult. It relies on each testing station having similar equipment and using it in a similar way. The same is true of lung function tests. As soon as anything more complicated than a peak flow meter is required standardisation will become an issue. The introduction of provocation testing would add another layer of difficulty. Testing bronchoreactivity to exercise, cold, saline or a general stimulant such as methacholine could only be standardised if it were done at a very limited number of centres and this would introduce serious transport and other cost implications for diving candidates.

The foregoing assumes that we know which tests to perform. For many years the Royal Navy has imposed a spirometric standard on its divers and submarine escape trainees based upon indices of obstruction. This was introduced after a cluster of deaths in the submarine escape training tower in the early 1970s. At that time it was believed that airways obstruction was the most important cause of pulmonary barotrauma. However, as we have seen, this may not be the case and the standard may be irrelevant to preventing barotrauma. My opinion is that until we have a better understanding of how and why the lung ruptures in barotrauma, it is premature to introduce lung function standards that are intended to prevent this.

THORACIC SURGERY

It is inevitable that people with lung parenchymal scarring will have reduced the compliance of the lung at the site of injury and this will make the normal lung proximally and distally to the lesion(s) vulnerable to over stretching. The problem we have is in quantifying how much more vulnerable the lung is. Where there has been mediastinal chest surgery and the pleura have remained intact, there is no theoretical reason for preventing diving; although, the reason for the surgery should be determined and a full recovery confirmed. If a pleural cavity has been opened and there is sufficient parenchymal or pleural scarring to be detected on X-ray or CT, it is my opinion that the individual is unfit to dive. Similar arguments apply to accidental traumatic chest injury.

BULLOUS DISEASE

Small bullae are common and asymptomatic and are likely to remain undetected at a routine medical examination. It is therefore irrational to ban all people with bullae from diving. A chest X-ray will only be requested where there is an indication based on family, social or past medical history or if an abnormality is detected on examination. It is my opinion that if bullae are found on such an X-ray, this is sufficient to render the individual unfit to dive.

SPONTANEOUS PNEUMOTHORAX

A past history of spontaneous pneumothorax is important. This occurs most commonly in young adults (up to age 40), is much more common in males than females (6:1) and is associated with smoking.[12] Almost all are unilateral (only 2% are bilateral) and affect both sides with equal frequency. About 50% recur, most commonly on the same side. However, after two years recurrence is very uncommon. After age 40 pneumothorax is increasingly associated with underlying disease. Given this, it is reasonable to ensure that there is no underlying disease and that there has been at least two years since the pneumothorax. Under such circumstances, I feel that individuals are fit to dive.

Making a decision on fitness to dive

In making a decision on fitness to dive the physician has a responsibility to the diver and the diver's buddy. The process involves striking a balance between risk and benefit. In sports diving, the benefits are exercise and pleasure and diving is but one of many ways in which the individual concerned can gain these benefits. The benefit, therefore, is not great and so nor should be the risk associated with diving. I have found that where there is an above average risk associated with an individual diving, if the risks are explained, the individual will make an appropriate decision as to whether or not they are fit to dive. In professional diving, the benefits associated with diving are great and physician is constrained by a regulatory framework and a duty to the employer. Determining fitness to dive is therefore considerably more demanding. Nonetheless, I have found it useful to involve the diver in the decision making process and it has been mercifully rare that I have resorted to imposing a decision on a recalcitrant diver.

Conclusions

In an ideal world, determining pulmonary fitness to dive would involve a candidate undergoing a series of laboratory tests to determine aerobic capacity and the ventilatory function of the lungs. The results would then be compared with an absolute standard and a determination made. However, this is not an ideal world. There is no agreed series of tests and no standard against which to judge the results. Thus the determination of pulmonary fitness to dive is not a scientific process, it is a judgment that is reached by weighing up an incomplete knowledge of the hazard posed by existing pathology against a subjective assessment of the benefits of diving. Despite the inadequacy of this approach, some generalisations can be reached. Those with

intrinsic or poorly controlled asthma or asthmatics with a reduced peak flow should not dive. Other conditions that should preclude diving include: recent spontaneous pneumothorax; bullous disease detected on chest x-ray; significant lung parenchymal or pleural scarring.

References

1 Neuman TS. Pulmonary fitness for diving. In *The Lung at Depth*. Lundgren CEG and Miller JN. Eds. New York: Marcel Dekker Inc. 1999; 73-90

2 UK Health and Safety Executive. *MA1, the Medical Examination and Assessment of Divers*. Glasgow: Health and Safety Executive, 1998

3 Bove AA. In *Fitness to Dive. 34th Undersea and Hyperbaric Medical Society Workshop.* Linaweaver PG and Vorosmarti J. Eds. Bethesda, Maryland: UHMS, 1987; 26-41

4 Liebow AA, Stark JE, Vogel J and Schaeffer KE. Intrapulmonary air trapping in submarine escape training casualties. *US Armed Forces Med J* 1959; 10: 265-289

5 Unsworth IP. Pulmonary barotrauma in a hyperbaric chamber. *Anaesthesia* 1973; 28: 675-678

6 Benton PJ, Pethybridge RJ and Francis TJR. A review of spirometry and UK submarine escape training tank incidents (1975 -1997) using objective diagnostic criteria. *Undersea Hyper Med* 1999; 26 (4): 213-217

7 Colebatch HJH, Smith MM and Ng CKY. Increased elastic recoil as a determinant of pulmonary barotrauma in divers. *Respir Physiol* 1976; 26: 55-64

8 Colebatch HJH and Ng CKY. Decreased pulmonary distensibility and pulmonary barotrauma in divers. *Respir Physiol* 1991; 86: 293-303

9 Calder IM. Autopsy and experimental observations on factors leading to barotrauma in man. *Undersea Biomed Res* 1985; 12 (2):165-182

10 Francis TJR and Denison DM. Pulmonary barotrauma. In *The Lung at Depth*. Lundgren CEG and Miller JN. Eds. New York: Marcel Dekker Inc. 1999; 295-374

11 Malhotra MS and Wright HC. The effects of a raised intrapulmonary pressure on the lungs of fresh unchilled cadavers. *J Pathol Bacteriol* 1961; 82: 198-202

12 Light RW. Pneumothorax. In *Textbook of respiratory medicine. 2nd Edition*. Murray JF and Nadel JA. Eds. Philadelphia: Saunders 1994; 2193-2210

Dr T J R Francis, MFOM, PhD, is Consultant in Diving Medicine to the Diving Diseases Research Centre, Derriford, Plymouth, Devon, UK. He has been guest speaker at the 1997 and 2001 SPUMS Annual Scientific Meetings. His address for correspondence is 2 Merton Cottages, Tregatta, Tintagel, Cornwall PL34 0DY, UK. E-mail <tjrf@btinternet.com>.

ASTHMA AND DIVING SCREENING PROTOCOLS

Cathy Meehan

Key Words

Asthma, diving medicals, fitness to dive.

Background

Ten to fifteen percent of children have some history of recurrent wheezing. It is estimated that 5 to 8% of adults are diagnosed as asthmatics. Asthma is an air trapping disease and the diving environment contains several potent triggers to asthma, such as exercise, inhalation of cold, dry air and also the possible inhalation of non-physiologically isotonic water which can be hypotonic fresh water or hypotonic salt water. There is no hard evidence that asthmatics are at greater risk of pulmonary barotrauma or death during diving. We know that some recreational divers who have asthma dive. They are often failed by the first diving doctor they consult but passed by the second because they suppress their asthma history.

Should asthmatics dive?

There is a divergence of opinion in the guidelines issued by authorities in different countries regarding fitness to dive. The UK recommendations for recreational divers can be briefly summarised as allergic or well controlled asthmatics may dive.[1] In America, as far as I am aware, there is no current agreed standard though active asthma is regarded as a contra-indication and provocation testing is regarded as a useful tool. In Australia there are various opinions. Carl Edmonds believes that asthmatics should not dive.[2] The Thoracic Society of Australia and New Zealand have published guidelines and there are Australian Standards for Recreational and Occupational Diving.[3-5]

Both Australian Standards state that any evidence of obstructive airways disease, such as current asthma, chronic bronchitis, allergic bronchospasm, shall automatically disqualify. In case of doubt, specialist medical opinion should be sought.

The Thoracic Society of ANZ states that the student should fail if there is a history of asthma or use of bronchodilators within the last 5 years.[3] If there have been no symptoms for 5 years and there is evidence of bronchial hyper-responsiveness after provocation testing they fail. A 20% fall in FEV_1 is usually needed to fail. Edmonds et al. consider that a greater than 10% reduction in FEV_1 after both histamine and hypertonic saline is a fail.[2] I recently surveyed some diving specialists in Australia through the ANZ HMG chat line. I want to thank everyone who replied