SPUMS ANNUAL SCIENTIFIC MEETING 1995

MEDICAL ASPECTS OF SPORTS DIVING

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Key Words

Air embolism, asthma, decompression illness, ENT, fitness to dive, flying and diving, pulmonary barotrauma, recreational diving.

Although the title is Medical Aspects of Sports Diving, cardiovascular problems,[1] diabetes and asthma will be passed over rapidly because they have been dealt with in other papers.

Pressure

Why do we discuss pressure in diving medicine? Because divers immerse themselves in water and change the ambient pressure by going up and down. There are a number of different ways to express pressure. There are pounds to the square inch (psi), millimetres of mercury (mm Hg or torr), feet and metres of sea water (fsw and msw), pascals (Pa), kilopascals (kPa) and megapascals (MPa), atmospheres (atm) and atmospheres absolute (ATA) and the other absolute measure, bar. There is plenty of scope for confusion. Table 1 (page 248) is the pressure conversion table published in every issue of Undersea and Hyperbaric Medicine. Descent increases ambient pressure and coming to the surface reduces it. These two changes generate most of the problems in diving medicine. The other major problem in diving medicine is the concern for physical activity.

Pressure drops as one goes to altitude. Whenever one moves from a higher to a lower pressure, some tissues are going to supersaturate. Things get worse going above sea level in an aeroplane after diving, as the tissue overpressures at altitude can be large enough to cause bubble formation when bubbles would not be formed if one had stayed at sea level. Most commercial aircraft are pressurised to the equivalent of 8,000 feet, about three quarters of an atmosphere. Eighteen thousand feet is exactly half an atmosphere. Sixteen thousand feet is 0.45 atm. Small unpressurised aircraft normally fly at around ten to twelve thousand feet. Some of them can go to 16,000 or 17,000 feet. Pilots in some of the better twin engine unpressurised aircraft, which can fly at 18,000 to 20,000 feet, can develop decompression sickness (DCS), without diving before flying, because they have reached a pressure which induces bubble formation in someone saturated at sea level.

Boyle's law is the cause of most diving problems. Ears are the most common problem. A good ear squeeze (aural barotrauma) early in the week will ruin the week's diving. Table 2 (page 249) shows the change of volume as a percentage for each 3 m (10 ft) as one descends in sea water from the surface to 30 m (100 ft). Going from the surface to 3 m (10 ft) there is a 32-33% reduction in volume. From 3 to 6 m (10 to 20 ft) there is a 20% reduction, from 6-9 m (20 to 30 ft) there is about a 13% reduction. Volume continues to shrink and a 3 m (10 ft) excursion from 27 to 30 m (90 to 100 ft) only produces a 2% change in volume. Why is this important? It is important because every now and then someone comes to me and says "My family doctor told me it is OK to dive as long as I don't go below thirty feet". When a physician says "Do not dive below 9 m (30 ft)" it is obvious that the doctor does not know about diving medicine, because this is the most dangerous area for barotrauma. The volume changes are largest and occur most rapidly between the surface and 9 m. It is very easy to get barotrauma effects in this zone. Below 10 m (33 ft) the volume changes are smaller per metre of depth change. Then it is easier to clear one's ears and much easier to control your buoyancy.

Ears

The tympanic membrane likes to have equal pressure on both sides. It requires effort to get ambient pressure into the middle ear. If the middle ear is not at ambient pressure the tympanic membrane will deflect and may ultimately tear. If one has difficulty equalising the middle ear, this means that there is a negative pressure in the middle ear with a differential not only between the outside water across the tympanic membrane but also between the middle ear and the perilymph (around the cochlea), the endolymph (in the cochlea and vestibular apparatus), and the spinal fluid, all of which are at ambient pressure, across the inner ear windows. The Eustachian tube closes at around 0.6 m (2 ft) of pressure differential and will not open. A forceful Valsalva manoeuvre trying to clear the ears can blow out the round window, causing an inner ear fistula damaging both vestibular and auditory apparatus. So, if one is having trouble equalising on the way down, it is not appropriate to keep going down doing increasingly forceful Valsalva manoeuvres.

Spinal cord

The spinal cord is the common site for injury due to decompression sickness in sport diving and is the common site for injury for bounce diving with air, to below 45 m (150 ft) for short times. James Francis[2] showed that there is a certain amount of supersaturation in cord lipids, and

TABLE 1

PRESSURE CONVERSION TABLE

The units of pressure preferred for manuscripts submitted to the *Undersea & Hyperbaric Medicine* are the pascal (Pa = Newton x m-2), kilopascal (kPa), or megapascal (MPa), defined by the International System of Units (SI). If the nature of the subject matter makes it appropriate to use non-SI units, such as fsw, msw, atm or bar, a parenthetical conversion to pascals, kilopascals, or megapascals should accompany the first mention of a pressure value in the abstract and in the text.

Atmospheres absolute is a modified unit of pressure due to the appendage "absolute"; the symbol "atm abs" is preferred over "ATA" for the modified unit.

1 atm	=	1.013250 bar	1 atm	=	33.08 fsw	1 atm	=	10.13 msw
1 atm	=	101.3250 kPa	1 bar	=	32.646 fsw[b,d]	1 bar	=	10.00 msw
1 atm	=	101.3250 kPa	1 bar	=	32.646 fsw[b,d]	1 bar	=	10.00 msw
1 atm	=	14.6959 psi	1 fsw	=	3.063 kPa	1 msw	=	10.000 kPa[c,d]
1 atm	=	760.00 torr[a]	1 fsw	=	22.98 torr	1 msw	=	1.450 psi
1 bar	=	100.000 kPa	1 psi	=	2.251 fsw	1 msw	=	75.01 torr
1 bar	=	100,000 Pa[a]						
1 bar	=	14.50377 psi						
1 bar	=	750,064 torr						
1 MPa	=	10.000 bar						
1 psi	=	6,894.76 Pa[a]						
1 psi	=	51.7151 torr						
1 torr	=	133.322 Pa[a]						

[a] Signifies a primary definition[1] from which the other equalities were derived.

[b] Primary definition for fsw; assumes a density for sea water of 1.02480 at 4°C (the value often used for depth gauge calibration).

[c] Primary definition for msw; assumes a density for seawater of 1.01972 at 4°C.

[d] These primary definitions for fsw and msw are arbitrary since the pressure below a column of seawater depends on the density of the water, which varies from point to point in the ocean. These two definitions are consistent with each other if a density correction is applied. Units of fsw and msw should not be used to express partial pressures and should not be used when the nature of the subject matter requires precise evaluation of pressure; in these cases investigators should carefully ascertain how their pressure-measuring devices are calibrated in terms of a reliable standard, and pressures should be reported in pascals, kilopascals. or megapascals.

Reference

1 *Standard Practice for Use of the International System of Units (SI). Document E380-89a*. American Society for Testing and Materials. Philadelphia, Pennsylvania, 1989.

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probably brain lipids as well, which is associated with nitrogen uptake in that kind of a profile. He dived dogs, in a chamber, for four hours at different depths, 6 m (20 ft), 12 m (40 ft), 18 m (60 ft), 24 m (80 ft), 30 m (100 ft) and so on, then decompressed and sacrificed them to study the spinal cord. He found is that no bubbles formed inside the cord until a saturation depth of about 24 m (80 ft). Shallower than that there were no bubbles. This data suggests that there was a critical depth, which provided a critical gas content in the spinal cord myelin, which would produce bubbles. This would explain why sport divers who go deeper and longer are more likely to suffer spinal cord injury.

Many people think the brain is involved in DCS, even though there are no clear cut neurological findings. Few people do behavioural testing. Careful psychological testing sometimes shows that there are brain injuries or mental changes with DCS. Some patients have bizarre behaviour patterns with their spinal cord injuries. When they are recompressed they get better, including the

TABLE 2

VOLUME CHANGES WITH DEPTH IN 3 m INCREMENTS SHOWING PERCENTAGE CHANGE FROM SURFACE AND FROM PREVIOUS DEPTH.

All figures rounded to nearest whole number.

Depth in m	Volume in ml	% of original volume	% reduction from surface volume	*Often quoted % reduction in volume for each depth change	Actual % reduction from previous depth volume
Surface	1,000	100	0	0	0
3	770	77	23	23	23
6	625	63	37	14	19
9	530	53	47	10	17
12	455	45	54	7	14
15	400	40	60	6	12
18	357	36	64	4	11
21	322	32	68	4	10
24	294	29	71	3	9
27	270	27	73	2	8
30	250	25	75	2	7

* These figures are derived from the difference between the % reduction in surface volume between two adjacent depths. The true reduction in volume, as a percentage of the volume at the shallower depth, between two adjacent depths is given in the right hand column.

behaviour patterns. Sometimes if one examines a diver carefully, including psychological tests, the results are not normal. People have complained, after a diving accident, that they could not work their computer or they could not write their signature. These are central nervous brain injuries associated with DCS. The reason the brain and the spinal cord are susceptible to DCS is because of myelin. Neurones have myelin sheaths. Myelin is a lipid which has a high affinity for nitrogen and can be easily supersaturated. The brain is also obviously involved in barotrauma and air embolism.

In the past we identified neurological deficits by clinical examination. Now there are some very sophisticated scanning techniques. One can use various radio isotopes or contrast enhancement. One can use CT or MRI. These investigations have been used in many other diseases to study brain and cord lesions. We are beginning to use them to study the brain and spinal cord in decompression sickness. The indications for these kinds of scans are not clear. I think if a diver has a cord injury and there are questions about behavioural patterns or psychological changes it is probably worthwhile doing an enhanced MRI.

Air embolism

The classical description of air embolism was a diver who comes to the surface, cries out, perhaps coughs up frothy, bloody sputum, falls back in the water unconscious, has a seizure and evidence of neurological damage. This description came from submarine escape training done in a hundred foot tower of water. The trainees were quickly pressurised in a chamber, then passed through a lock at the bottom of the tower and made a buoyant ascent, exhaling continuously all the way. Sometimes they wear a hood over the head, sometimes they wear a survival suit but always they must exhale continuously to prevent lung over-inflation. The incidence of pulmonary barotrauma as severe air embolism, at least in the experience of the United States Navy (USN), was about one in seven thousand escapes and about 10% of those would die. The USN has now abandoned this training.

It is rare for that presentation to occur in sport diving. It happens occasionally. What usually happens is a subtle air embolism event. Such as when a diver, who is hanging at 3 m (10 ft), forgets to deflate his BC (buoyancy compensator) as he comes to the surface. He gets a minor pulmonary barotrauma and some air enters the brain, but the diver is not unconscious. He may have minor visual

changes, personality change, fatigue, but nothing obvious. This is the new type of air embolism that one must be aware of because some divers who sustain air embolism have non-specific and vague symptoms. The diver who is unconscious after an air embolism can wake up and appear normal, but relapse is common. Diagnostic mistakes have been made in the lucid interval when a physician examined the diver, found nothing and sent the diver home. Four or five hours later the diver was back unconscious with severe neurological abnormalities. A diver with a history of unconsciousness on surfacing, whatever the conscious state, whether they look good or not, should be recompressed and observed for twenty four hours.

Look for irritability and for changes in concentration. Both divers with DCS and with air embolism have complained of such things as being unable to write their signature correctly. There was the accountant who could not add a column of numbers the week after a dive in which he had a brief rapid ascent. No other symptoms were evident, he could not concentrate or add. There are people who went to work on Monday and could not open their computer files because they could not remember the password. If one looks one can find subtle changes. Most sports divers do not panic and ascend uncontrollably to the surface. Air embolisms result from minor mistakes near the surface; trying to clear the mask on the way up, doing a Valsalva manoeuvre as a wave changes the diving depth. There are many things that can occur between 6-7 m (20-25 ft) and the surface which will cause minor barotrauma and minor amounts of air embolism with not so obvious signs or symptoms. Treatment for subtle air embolism is in a hyperbaric chamber using an 18 m (60 ft) table rather than a 50 m (165 ft) table.

Epilepsy

At present the rule is that anyone who has an active seizure disorder or who is taking medication to suppress the seizure disorder should not be diving. This applies to all sport diving and all commercial and military diving. The reason is that if an individual has a seizure in the water, and becomes unconscious, that person will drown. This has happened more than once. In a few instances the buddy, trying to rescue the fitting diver underwater, has also drowned. In the United States, epilepsy advocacy groups support a policy without restriction, suggesting that people with active epilepsy should be allowed to dive.

At the moment it is impossible to tell one person from another in terms of their risk for seizures. Somebody with severe, easily provoked seizures is going to get an easily provoked seizure by exposure to cold water, by hyperventilating, by having anxiety or excess adrenalin. However, it is not clear whether somebody, who has not had a seizure for fifteen years, will be prone to a seizure while in the water. Because we cannot make that differential gradation of risk, we try to give everyone the same risk and say no to diving.

Clearly there are people with minimal seizure risk and yet they get lumped in with any adult seizure disorder which is treated with drugs. Someone who had a fever-associated seizure at six years old, has never had a fit since does not have epilepsy and can dive. Someone who is free of seizures and does not take anti-epileptic medication does not have epilepsy. If somebody had a seizure disorder and has been off drugs for some years, never had a seizure since starting the drugs and has led an active life, can that person dive? In the United States such people have been prohibited from diving for a certain number of years, usually five years seizure free and drug free, then allowed to dive. In commercial diving it would be out of the question. Some neurologists would say that if there is no seizure focus on electroencephalogram (EEG) and there have been no seizures for five years, then the person can sports dive. The approach that I would recommend is that if someone is on drugs for a seizure disorder and the treating physician considers that the drugs must be taken to prevent fits, that person should not dive. The reason is that these people have a seizure focus which is being suppressed by drugs and it is possible that the seizure focus could be activated by diving to break through the drug and cause a seizure. If someone is off drugs and has not had a seizure for four or five years, I generally get an EEG done. If the EEG is normal I allow diving, if the EEG still shows a focus, I say no. None of this is written in a rule. Physicians do not have legal authority to prevent people from doing anything. All one can do is provide advice.

Coronary disease

Coronary disease is a fairly common problem. Sport diving started in the mid to late fifties, so a 22 year old sports diver then is now in his sixties. As the risk of atherosclerosis increases with each year of life, age becomes the most common risk factor that is present. Of course there are cholesterol, hypertension and other factors, but age is the most common risk factor. Unfortunately it is inevitable. There are pills that will lower cholesterol or blood pressure, but no pill will lower age. Sport divers, as time moves on, are at increasing risk for coronary disease. Some of them ultimately get an narrowed artery. The narrowed artery limits blood flow to the heart muscle, the heart muscle does not work well under increasing loads and becomes ischaemic, when it is ischaemic it can either infarct or cause serious or even lethal arrhythmias.

The problem with cardiac atherosclerosis is that it is silent for a very long time. It progresses over time going from a normal vascular endothelium to small plaque formation. These plaques ultimately increase in size until the lumen is occluded by a thrombus. We have learned over the last ten years that plaque growth is not just deposition

of cholesterol over time in a steady fashion, related to having high blood cholesterol. What happens is something goes wrong with the endothelium. It is not clear what, we think cigarette smoking has some direct effect on the endothelium. We think hypertension has direct effects on the endothelium, LDL cholesterol has some direct effects on the endothelium, all these seem to make the endothelium less resistant to lipid deposition and other types of injury. The normal function of the endothelium is to resist platelet stickiness and to secrete vaso-relaxing agents which keep the blood vessels dilated. When an artery is injured the endothelium malfunctions, loses the protection against platelet adhesion and stops vasodilatation, in order to protect one from bleeding. This mechanism is excellent for trauma, but there are other triggers for this reaction. With endothelial damage there is lipid deposition. The lipid deposition causes an inflammatory reaction, the inflammatory reaction becomes organised, with calcium, fibrous tissue, cholesterol crystals and smooth muscle proliferation at which stage it becomes a plaque. Later a plaque may rupture. The same process occurs producing another lamination on top of the original plaque. Histologically plaques are laminated like the rings of a tree. Each event produces more occlusion until the plaque rupture causes thrombus formation which occludes the artery instead of just narrowing it. Sometimes thrombolytic agents wash out the thrombus and one finds that the patient only had a fifty percent narrowing underneath the thrombus. Usually the end point is thrombus formation, total occlusion and an infarct. Sometimes an infarct does not occur if the narrowing occurred slowly enough to allow development of collaterals which can carry enough blood to allow occlusion to pass unnoticed.

When one gets into the sixties with no cardiac disease one can be sure that there will be plaques here and there and hopefully some collaterals to protect one from a major catastrophe when one of the arteries occludes. There is no way to know and no one should volunteer for a coronary angiogram without good reason.

Cardiac blood flow and occlusion.

Resting flow in the heart is about one ml/minute/gram. The maximum flow is about 5, so the myocardium has a five to one flow reserve. If one really pushes oneself to extremes (i.e. in competitive athletics) one might get up to four times resting blood flow. Getting five times resting blood flow can only be achieved by reactive hyperaemia after occluding the artery for thirty seconds. One finds that as the cross sectional area is reduced to 80% of normal this ratio does not change very much. Changes are in the range 3-4 ml/minute/gram where very few people ever get. Ordinary walking takes about 1.2 times resting blood flow. A very arduous dive, swimming on the surface with all one's gear on will reach two. Running a mile in 8 minutes one might be up to two and a half times resting blood flow. One has to be into competitive athletics to get up into the threes and fours. We lose flow reserve as we move through 20%, 40% and 60% narrowing with imperceptible reductions in the capacity for blood to go through the coronary arteries. At about 70% reduction in cross sectional area there are large losses in flow reserve. From 80% to 85% there are significant reductions in the peak/resting flow ratio. When the peak/resting ratio is two to one symptoms can be easily provoked. One of the reasons why we worry about divers is that, when people do not exercise very much, they not know that they have limitations. The first time they may sense coronary ischaemia is when they are diving. In the United States sudden death in diving is mostly from acute myocardial infarction. It is usually in people who did not exercise enough to provoke symptoms. The first symptom may be sudden death, which is not an efficient method to determine whether one has coronary disease.

Deciding who can dive and who cannot dive with cardiac problems, is related to physical capacity.

Pulmonary oedema in cold water

Every now and then a healthy middle aged man is exposed to cold water and develops pulmonary oedema. He is helped into the boat, treated and recovers. When investigated nothing abnormal can be found. They were men in their fifties or older, with a history of hypertension, otherwise healthy but not in good physical condition, who went into pulmonary oedema diving in cold water.[3] These were obvious cases of pulmonary oedema which quickly cleared. One suggested mechanism is that vasoconstriction, on entering cold water, in the presence of a raised blood pressure leads to uncontrolled hypertension and ventricular failure.

Asthma

This subject has been well ventilated in the December 1995 issue of the Journal[4-12] so this part of the paper has been very much shortened.

We know there are many asthmatics who dive. There was a survey in a diving magazine, published in the USA, which showed that about 6% of the respondents were asthmatics who dived In the United States about 6% of the population has asthma. Obviously the diving population was not being properly screened. The problem with that survey was that those who dived and died did not answer the survey. So watch out for the controls. But the fact is there are lots of people with asthma who dive. Even asthmatic divers who develop symptoms in the water still dive. I heard this story from a diver. He was on the bottom of St Lawrence River, wearing a dry suit, at 24 m (80 ft), when he had an acute asthma attack. He swam quietly and

comfortably to the surface, got on the boat, took his mask off and used his inhaler a couple of times and was fine. There is probably a small increase in risk, perhaps a factor of two in terms of risk for air embolism or decompression sickness. The danger is that the diver's status may change while diving. Cold water, exercise and other things can change a stable non-reactive airway to a reactive airway while in the water. The message in that is if one is a good diver and keeps one's head, one can get to the surface without a problem.

Diabetes

Diabetes is covered in the March 1996 issue of the Journal.[13-15]

Dysbaric osteonecrosis

There appears to be no risk of dysbaric osteonecrosis (DON) in sport divers. I know of a few cases among people who claimed to be sport divers but when one studied them they were making a living as dive guides. They were not sport divers, but they had bone necrosis. Dysbaric osteonecrosis is definitely a disease caused by diving but it is not a disease of the usual sport diver. There are many causes of osteonecrosis. Certain fractures will cause osteonecrosis by damaging the blood supply to the distal part of the bone. Other causes include chronic uraemia, alcohol intake, steroids and decompression sickness. Transplant patients take large amounts of steroids to suppress rejection and osteonecrosis is relatively common.

The Japanese have shown a high incidence of osteonecrosis among divers who followed improper decompression procedures. Not only did they have many cases of DCS, accepted as an essential part of the diver's occupation, but a very high incidence of DON. Total diving experience was the main determinant. Early papers on DON were published about 20 years ago. Dysbaric osteonecrosis can be in the shafts of the long bones, where it produces no symptoms, or close to the articular surface, juxta-articular lesions. These usually lead to damage to the joint surface with joint collapse leading to disablement. Nowadays an artificial joint can restore mobility.

Caisson workers and deep commercial divers also figure in the statistics. The decompression tables used for caisson and pressurised tunnel work prevented DCS but they were ultimately modified to prevent bone necrosis. It is not a sport divers' disease.

Fitness

A brief word about fitness. The major problem for sports divers is being able to handle the physical activity required in diving. If one is in good shape, one's maximum oxygen uptake is higher. One can sustain work at 50% of maximum oxygen uptake on a continuous basis. Working at 70% or 80% is stressful, producing lactic acidosis. If one is in good condition, meaning one's maximum oxygen uptake is increased, a workload that requires 50% of maximum oxygen uptake is comfortable and can be dealt with. If one is in poor condition that same workload would require 70% of maximum oxygen uptake and would be impossible to maintain. A fit diver might handle the workload of swimming 200 m back to the boat against the current after a dive and be short of breath. An unfit diver would get severe lactic acidosis, hyperventilate, panic and risk drowning.

Fitness, measured objectively as oxygen consumption, declines with age, as the maximum heart rate declines with age. It is important to match the exercise capabilities of buddies and groups to prevent older divers from working beyond their capacity.

Calculating decompression

A number of surveys in the United States of trained divers have shown that two thirds could not use a decompression table properly. Certainly there are computers now, but it is still important for people to understand the whole process and to be able to calculate a repetitive dive. Interestingly about one third of the instructors tested could not calculate a repetitive dive using tables. Education is important for diving safety and probably the most important factor in safe diving after making sure that the individual has a reasonable capacity for physical activity.

Audience participation

Greg Leslie

How much disease does a normal thallium scan really exclude?

Bove

Post mortem data shows evidence of plaques even in twenty and thirty year olds. Years ago Clarence Demar, who had run marathons up until the age of sixty five, and won the Boston marathon nine times, died of colon cancer. At post mortem he had dilated coronaries, lots of plaques but nothing that was anywhere near obstructive. Neither a thallium nor a regular stress test would have shown ischaemia. Most people are likely to have some plaques as they get older. Without severe narrowing one is not at risk. A normal stress test shows that there is no ischaemia. There are some data suggesting that if a regular stress test is normal it means the person can handle the exercise, which is the key issue. If a 55 or 60 year old wants to start diving and is not an exercise person, I definitely want an exercise

tolerance test to be certain that he or she can handle the workload of diving, which can reach around 13 mets. Thirteen mets is the equivalent of about a ten minute mile jog. It is not a really heavy workload but it is significant, equivalent to swimming against a one knot current with full gear on. A sensible diver does not try to swim against a stronger current.

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OPHTHALMOLOGICAL ASPECTS OF FITNESS TO DIVE

Malcolm Le May

Abstract

The diving medical examiner may be faced with potential or current divers who exhibit either problems with vision or ophthalmic pathology. A review is presented in which the required visual standards are discussed, together with strategies for correction of common vision problems in the diver. Some of the more commonly encountered ophthalmic conditions are mentioned and their possible impact on fitness to dive is discussed. An attempt is made to consider how eye conditions might be adversely affected by the hyperbaric or aquatic environment. Among conditions reviewed are visual acuity, refractive errors, contact lenses, corneal disease, cataract, glaucoma, retinal detachment, ischaemic conditions, lacrimal duct and orbital abnormalities.

Key words

Eyes, fitness to dive.

Introduction

The diving medical examiner may be faced with questions of a potential diver's fitness in the presence of a previous ophthalmological condition or previous surgery. There have been considerable advances in ophthalmology in the last decade and many examiners may feel that there are gaps in their knowledge of eye conditions, particularly if this knowledge is based mainly on recollection of undergraduate tuition. This review attempts to cover aspects of vision and the eye that may concern the examiner regarding fitness to dive.

The purpose of being fit for a recreational pursuit is to increase both enjoyment and safety. In diving, fitness is important to ensure that the diver is not a danger to himself* or to others.

It is impossible to reduce this risk to zero. The diver may have a right to endanger his own life, but the possibility of endangering others is the more important consideration. It may not be possible or even desirable to reduce either risk to zero, but the diving medical examiner should regard the minimising of danger to others as the primary purpose of the diving medical. The secondary purpose of the diving medical is to minimise the risk to the diver.

* *I have written throughout in my own gender to indicate both sexes.*