time. Use of alternate air source breathing such as alternate second stage, Air II, pony bottle, etc., also involves the learning of a series of skills. These procedures are as complex as buddy breathing up to the point of sharing. The basic difference is that the recipient receiving an alternate air source need not alternate breathing with the donor. This is a substantial advantage in many cases. It is folly, however, to assume that these alternatives to buddy breathing do not require substantial learning and reinforcement.

It is possible to conceive of "what ifs" that could create additional variables and interfere with a smooth procedure. Adequate training, education and dive planning will still be required in order to minimize the "what ifs" and their effects.

This is an edited text derived from a lecture with slides and from the text of a previous publication provided by Dr Egstrom

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RESPIRATORY FUNCTION IN INTENDING DIVERS

Andy Veale

Introduction

The history of diving medicine has moved through a number of different phases. Firstly, divers simply went diving to accomplish a particular aim, there was no consideration at all about the physical or physiological attributes necessary to perform this work safely. Occasional deaths and illnesses then occurred, and attempts were made to explain these deaths using physiological and pathological knowledge obtained in other situations and in other disorders. Rules have then been derived from these extrapolations. Of necessity these rules or standards, are conservative due to the lack of basic knowledge, the desire to be exhaustive and to avoid any perceived medico-legal risks. Finally, the "natural" data accumulates and research data is collected, suggesting that theoretical concerns have been overstated and standards are ultimately relaxed. One very good example of this is the relaxation of standards for aircrew following spontaneous pneumothorax in all Air Forces.

I believe diving medicine needs to become more scientifically rational in terms of risk assessment in order to be perceived by the diving community as acting in the interests of divers, to avoid the "them and us" situation.

Lung anatomy and physiology

I shall briefly cover some aspects of the normal lung anatomy and physiology before pointing out some of the changes in normal physiology which occur during diving. I will then discuss some of the possible mechanisms of barotrauma and how these have been used to justify some of the theoretical risks, and hence contraindications, in current diving standards. I will then discuss the actual risk data, and the potential pitfalls in interpretation of this data, before proceeding to a brief philosophical discussion of what the doctor's role should be.

The lung is a very elastic structure which tends to collapse towards functional residual capacity (FRC). FRC represents a balance between the tendency of the lung to collapse and the tendency of the chest wall to spring out. Most of the lung elasticity is in the bronchovascular bundles which contain most of the elastic and non-elastic connective tissue. The bronchi and vessels tend to run together within bronchovascular bundles and during inspiration or over-inflation there tends to be a tractional force along these bronchovascular bundles. Within the walls of the bronchi smooth muscle is oriented in a circular or spiral fashion, becoming increasingly discontinuous toward the terminal bronchioles, leading to areas of potential weakness.

During a normal forced expiratory curve flow rate rapidly reaches a maximum and then falls as the airways become narrower, acting as a flow limiting step. Flow at low lung volumes is thought to reflect flow within the small airways but even in these terminal portions of expiration, flow is still significant at around 800 ml per second.

The compliance of the chest wall and lungs varies considerably with the phases of respiration. Starting from expiration increases in lung volume cause little change in intrathoracic pressure. However at the extreme of inspiration a very small increase in volume is associated with a marked increase in intrathoracic pressure. So any reduction in depth (pressure) while a diver is at total lung capacity (TLC) will very rapidly increase the intrathoracic pressures and as a result the tractional forces along the bronchovascular bundle.

During head out immersion there are significant changes in pulmonary physiology. The lung becomes much less compliant due to the central redistribution of blood volume, closing volume is increased and specific airways resistance and the work of breathing are increased dramatically.

Increasing gas density leads to progressive, and quite marked, declines in flow at all lung volumes.

The net effect of these changes, together with the viscosity of water through which the diver must swim, leads to significant work, not only in respiratory terms but also for the swimming muscles. Swimming at 0.85 knots raises oxygen consumption to six times normal which is close to the maximum aerobic threshold for normal people. The physical work of sport diving is an important, and under-recognised, factor in assessment of diving fitness and contributes significantly to drownings.

Pulmonary barotrauma pathology

The pathophysiological mechanisms of pulmonary barotrauma are entirely unknown. Two possible mechanisms which may occur are alveolar rupture, where alveolar over-distension rather than over-pressurisation leads to shearing stresses on the alveolar bases where they abut the bronchovascular bundle or adjacent segments. The second possible mechanism relates to the tractional forces which may develop along the longitudinally oriented bronchovascular bundle. The circular smooth muscle becomes increasingly discontinuous and spiral towards the terminal bronchioles. In asthmatics who die in during an acute attack it has been shown that bronchiolar glands and ducts can rupture through the bronchiole wall, leading to interstitial emphysema. It is not known whether this mechanism also occurs in divers.

During inspiration lung volume increases and the alveolus enlarges. At the same time there is an increase in intrathoracic blood volume which causes a lengthening of blood vessels and an increase in their transverse diameter During expiration the reverse of this occurs and hence the relationship between the alveolus and the vessel remains constant. If there is alveolar distension without a corresponding increase in intrathoracic blood volume, a shearing stress between the alveolar base and the vessel may cause rupture of the alveolar lining epithelium and basement bundle. This mechanism of causing interstitial emphysema of the lung was clearly explained in the 1940s.[1] In divers hypovolaemia is very common and could exacerbate these shearing forces.

Pulmonary barotrauma

Air within the bronchovascular connective tissues may lead to interstitial emphysema and if this tracks toward the mediastinum, to pneumomediastinum and subsequently to surgical emphysema. If air ruptures through from the mediastinal pleura or from subpleural blebs into the pleural space, a pneumothorax will occur. If entry is into a vessel then arterial gas embolism occurs.

I have an X-ray of an asthmatic woman, taken shortly before she died, showing marked pulmonary over-inflation with surgical and mediastinal emphysema. Her heart was squashed flat by the high intrathoracic pressure. The over-inflation was not air-trapping behind obstructed bronchi. It is due to air in the pulmonary interstitium which tracked along the bronchovascular bundles to the mediastinum. The steadily increasing amount of this interstitial air raised the intrathoracic pressure. Small bronchi and vessels were compressed by this air.

Pneumothorax may be subtle or it may be obvious. A tension pneumothorax can be rapidly fatal.

CAGE pathophysiology

Arterial gas embolus may be fatal at onset, or more usually, bubble transit through the cerebral circulation may result in subsequent reduction in cerebral flow, causing neuronal dysfunction.

These physiological and physical factors, and the serious disorders which may occur in divers, have led to the suggestion that disorders associated with air flow limitation, with gas trapping, with intrapulmonary gas collections, or with patchy lung scarring, should all be contra-indications to diving with compressed air. This has led to the application of the "What if this were to occur at depth" test, without the consideration of the probability of "it" occurring. Many of us have said that whilst there is no definitive proof, there are sound physiological and physical reasons why people are likely to be at an increased risk and we have consequently been restricting large numbers of potential divers.

Discussion

Colebatch looked at pulmonary compliance in six divers with pulmonary barotrauma, comparing them with sixteen divers of similar dive experience but without barotrauma, and demonstrated that the barotrauma divers had a significant reduction in pulmonary compliance: their lungs were stiffer.[2] In this population there were 500 divers at risk of barotrauma and only six developed barotrauma. Of 26 normal divers, three had reduced compliance of similar degree to those with barotrauma, and if this is extrapolated to the total diving population at risk in this study, namely 500 divers, 58 would have been expected to have similar reduced compliance as those diver who suffered pulmonary barotrauma.

In a number of studies of submarine escape and in the military, pulmonary barotrauma has been shown to be a very uncommon event although it must be appreciated that considerable pre-screening has occurred. Professor David Dennison at the Brompton Heart and Lung Institute has examined many survivors from pulmonary barotrauma with specialised pulmonary function tests and high resolution thin section computerised tomography, and has been unable to demonstrate significant abnormalities of lung function

which would predict the occurrence of pulmonary barotrauma. Brooks, Pethybridge and Pearson showed that in 34 cases of barotrauma in 3,788 escape tower ascents, forced vital capacity (FVC) < 2SD predicted 8/34 while the ratio of forced expiratory volume at one second (FEV_1) to forced vital capacity (FEV_1/FVC) < 75% would only predict 1/34.[3]

In the Australasian series of 100 deaths reported by Edmonds and Walker[4] nine deaths occurred in divers who were asthmatic. Only two of these showed evidence of PBT. In reported cases of arterial gas embolism there has been only one asthmatic reported in forty episodes of arterial gas embolus treated at Catalina Island, there were none in the 42 cases reported from Hawaii and in the Auckland series of 125 civilian divers[5], of whom one-third had arterial gas embolus-type syndrome, there were also no asthmatics. This data by itself is not entirely reassuring as the denominator, the number of asthmatics who have dived without getting arterial gas embolism, is unknown.

In a paper in the British Medical Journal, Farrell and Glanville[6] reported 104 asthmatics who had dived for a number of years, producing a combined total of 12,000-plus dives without incident. These divers were a self-selected population obtained through a magazine questionnaire however, and again the denominator is unknown.

Spontaneous pneumothorax and thoracic surgery are also conditions which conventional wisdom says are contraindications to diving. Tom Neuman, in recent book on diving medicine edited by Bove and Davis[7], comments that there has not been one incident in the USA Accident Data Base of a diver with past spontaneous pneumothorax or thoracic surgery developing barotrauma or arterial gas embolism while diving. One reason for the lack of incidents may be that these people are screened out by doctors already, and hence the denominator is extremely small. However, this does raise the question of whether we should apply the principles of risk assessment to each individual case, rather than using black and white "standards".

The final part of the puzzle is to look at whether the incidence is great enough to justify mass screening and restrictive rules. In New Zealand PADI train 95% of all trained divers. There are 7,000 new divers trained each year. The numbers of untrained divers who take up the sport each year is unknown but the untrained proportion is likely to be reducing each year. These 7,000 new PADI divers will perform 70,000 dives in their first year and it is quite conceivable that an excess of 200,000 or 250,000 dives are performed each year in New Zealand. Ten of these end in death, the majority from drowning, and 50 end in a recompression chamber. As I mentioned, none of those survivors in the recompression chamber when examined, have shown evidence of abnormalities of lung function or have been asthmatic.

We now come to what a physician's role should be. Are we to act as advisers for divers, in which case it would be perfectly appropriate to advise an asthmatic not to dive on theoretical grounds stated but to teach them ways of overcoming these theoretical risks, i.e. reinforce sound diving practice. Should we administer black and white rules which brook no discussion and exclude any grey area, or could we, with the intending diver, weigh the evidence.

Would it not be best for knowledgeable and experienced doctors to perform a good history and examination, to use such investigational tools as seem appropriate for risk assessment and education; taking account of all the available information, including the mental and general physical state of the person before them; to discuss the risks of diving and give appropriate advice?

Recommendations

I believe the following recommendations are justifiable.

I think that the medical examination for intending divers should be compulsory as it is in New Zealand. I think that this examination should be performed by a doctor knowledgeable about the hyperbaric and diving environment and with a good understanding of the certainties and uncertainties of the advice to be offered. I think that every diver should undergo an FEV_1 and FVC at entry and if there is a history of asthma or of past pulmonary disorder then tests of pulmonary physiology may be useful. These should be performed not for a pass-fail response but as an aid for explanation and rational discussion. I think doctors should advise and support.

In my experience the majority of intending divers who see a doctor they perceive as knowledgeable, caring and acting in their interest, will take the advice offered and be very grateful for it. The same is not the case for an existing diver who has dived for ten years and seeks higher certification, who is then advised that he can't dive because his FEV_1/FVC ratio is less than 70%.

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This is an edited transcript of a paper, illustrated with slides, delivered for Dr Veale, at the 1991 SPUMS Annual Scientific meeting held in the Maldives.

Some references have been provided by editorial staff where they could be easily worked out from the Editor's library.

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DIVING SAFETY, WHERE ARE WE GOING ?

John Knight

Summary

Diving safety, as for all safety, requires an attitude of mind as well as technical competence. It requires the proper equipment which must be well maintained. Inexperienced and out of practice divers dominate the diving deaths. Diving is always a serious business, but should be enjoyable. Diving for fun may mean that the diver does not bother to dive safely. Current training turns out divers who need extra training to dive safely anywhere other than where they have been trained. Experienced divers seem to be able to avoid problems if they take care.

Introduction

Scuba diving is an intrinsically dangerous sport as it is performed in an unbreathable medium with a limited air supply. It is equipment dependent. The scuba diver must have a reliable breathing system to survive. Raised nitrogen partial pressures change a diver's thinking. Immersion alters physiology and being in water increases heat loss. A diver can kill himself (or herself) by holding his breath and rising in the water. Sea conditions can change rapidly and become dangerously hostile.

Decompression sickness is an unavoidable hazard of scuba diving or any sort of diving. It is very, very, difficult to come up slowly enough to form no bubbles at all. It is a statistical accident whether one forms enough bubbles in the wrong places to get decompression sickness symptoms. There is quite a lot of evidence that coming up faster than 18 m per minute is associated with cases of decompression illness. There is also evidence that multiple ascents during a dive are associated with decompression sickness.

Breath-hold divers continue to die unnecessarily every year. Post-hyperventilation blackout has been known for about 30 years, but its dangers are regularly forgotten. One of our past guest speakers bore the scars of two chest drains and a tracheostomy, the results of a post-hyperventilation blackout in the university swimming pool. He was lucky to be rescued and revived with CPR. Thechest drains were needed for the pneumothoraces CPR gave him ! He went on to become a Diving Medical Officer in the USN.

Safe diving

What is a safe dive ? Is it one where the diver never makes a mistake or is it one when he or she survives to get back to land alive, or more importantly alive and well ? That is a huge range from which to take your pick. Diving is always a serious business, but it should be enjoyable to avoid stresses which can cause disaster. Diving for "fun" may mean that the diver does not bother to dive safely.

Diving safety requires an attitude of mind as well as technical competence. It requires the proper, well maintained equipment. It requires knowledge of the physiological effects of immersion, of hypothermia as keeping warm underwater is difficult, of the effects of partial pressure changes to name but a few requirements. It requires thought on the part of the diver. It requires judgement and courage to stick to doing what is safe, to refuse to dive because one is not happy with some aspect of the dive, often the weather or sea conditions.