

health surveillance should be understood by those who intend to engage in such screening.

Key words

Pre-placement, discretionary assessment, prescribed assessment, asthma.

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OBSERVATIONS ON ASTHMA IN THE RECREATIONAL DIVING POPULATION

A Bove

I will review a little bit about the pathophysiology of asthma. Some of the data that we collected for DAN in an attempt to make a statement about what to do with asthmatics in diving. Asthma is a common disease. Some people estimate about 10% of the US population are asthmatics. I was in the desert for several months during the Gulf War and a large number of the young American marines, who went into the desert, came to our hospital wheezing with significant asthma because of the organic dust that is in the air there. Our surveys in the US indicate that the incidence of asthmatic divers is the same as the incidence of asthma in the general population which means there is no effective screening. Asthmatics are getting into diving, probably by not revealing their past history. So, generally, asthma comes under the list of pulmonary disorders in diving, and I do not want to dwell on the other ones, although they are there. The history of pneumothorax, the history of any other chronic lung disease, pneumoconiosis, all would eliminate somebody from diving. The question about what we do with previous barotrauma is also unanswered.

If one looks up the text book definition of asthma it is usually stated as "generalised airway obstruction due to the contraction of bronchial smooth muscle". It has a series of clinical characteristics. Often it is associated with a cough, dyspnoea (shortness of breath) with mild exertion, wheezing, the over inflation syndrome, that is the lungs are over inflated, and often the auscultatory finding of wheezing and crackles throughout the lungs because of secretions retained in the airways. I do want to make light of the cough because many people who have very mild asthma, develop a cough and do not understand what it is about. People are sent to me with a cough thought to be

heart failure, it often turns out they are asthmatics and a bronchodilator gets rid of the cough. The cough was related to airway reactivity, so there are a number of different presentations. The severe obstructive airway disease which causes wheezing and dyspnoea is only one end of the spectrum.

Allergy and infection are the two most common trigger mechanisms. Most asthmatics have a family history, in parents, siblings or children, of other allergies. Infection of the upper airways is often a trigger in the person with hyper-reactive airways. Adult bronchiolitis, when a viral infection of the airways causes wheezing, is a truly transient phenomenon and it is not related to reactive airways. There is a small number of patients who wheeze with bronchitis, but if that is the case one should not classify that patient as an asthmatic. In these cases one needs to wait several months to allow the airways to settle down before doing any testing. Acute anxiety will do this and I think it is probably because of the change in hormone that stimulates the airways. Parasympathetic stimulation will cause reactive airway disease, and of course the catecholamines usually cause relaxation of the airways so we use epinephrine (adrenalin) to relax the airways. Exercise will induce wheezing, and cold will induce wheezing. Cold and exercise are somehow irritants to the airways which can cause bronchospasm under those conditions. So there is a number of trigger mechanisms.

In a chronic asthmatic the process goes beyond just pure smooth muscle activation and bronchial constriction. There ultimately becomes hypertrophy or overgrowth of the bronchial smooth muscle so there is thickening of the bronchial walls. There is mucosal oedema and secretions in addition to bronchial hyperaemia. All these things will cause airway obstruction. In particular the retention of secretions in the small airways is a common complication in asthmatics because with bronchial relaxation, the airways do not always completely clear. Often one must use inhalation therapy with mucolytic agents to clear the bronchial secretions. This is an important part of the chronicity of asthma.

Fishman¹ is a well respected pulmonary physiologist who studied asthma for a long time and classified severity in a range of one (most) to five (least). I think the single most useful measure of an asthmatic is the alteration in airway conductance. The normal person, or the minimal asthmatic, has essentially normal airway conductance. As one goes through the spectrum of severity to what essentially is chronic obstructive lung disease, there is a progressive decline in the airway conductance, that is there is more and more resistance to the motion of air through the airways.

This is manifest by a number of different measurements. The forced expiratory flow, between twenty five percent and seventy five percent (FEV₂₅₋₇₅), or any of

the measures that record the rate at which air leaves the lungs, will show abnormalities which are characteristic of asthma. That is the nature of the pathophysiologic limits on an asthmatic. They cannot move air rapidly out of the lung, therefore they cannot ventilate adequately and develop hypoventilation syndromes, relative hypoventilation syndromes, and sometimes even significant CO₂ retention. The ability to move air rapidly through the airways declines as the severity of the asthma increases.

The total lung capacity also increases as an asthmatic becomes worse. The chronic asthmatic who progresses ultimately to chronic obstructive lung disease has an expanded chest, an increase in total lung volume. With the mildest form of asthma the volume pressure curve, the lung compliance, is basically normal. As asthma becomes more severe the lung volume increases and as it increases there are decreases in lung compliance. Increasing lung volume is characteristic of the long standing asthmatic. The residual volume goes up. That is a problem in diving because long standing asthmatics are working at higher lung volumes and have trouble with buoyancy because they cannot get the air out of their lungs appropriately.

There is a gradual increase in total lung capacity as the severity of obstructive lung disease or of asthma increases. This is because of the destruction of the alveolar structure of the lung. In the end stages of obstructive lung disease, the total lung capacity is markedly increased. So two things, the lack of adequate airway conductance, and ultimately an increase in lung volume, cause the asthmatic to breathe at higher and higher lung volumes. These are two characteristics that can get the asthmatic into trouble with any sort of physical activity including diving.

The severe end stage lung disease patient has a very high residual volume. This is obvious when you look at the patient. The diaphragms are flattened, and low, the chest is expanded outward, the clavicles are elevated. The whole lung volume is increased including residual volume and there is a lot of intrinsic lung (alveolar) damage by the time you get to this level of obstructive lung disease. Only the mildest forms of asthma have residual volume unchanged. The more severe have a continuous increase in residual volume. Increasing resting lung volume can cause problems with buoyancy.

An interesting thing is the change in lung compliance as individuals develop hyperventilation. In the normal individual lung compliance is much the same when breathing at ten breaths a minute (resting breathing) and when breathing rapidly. With increasing severity of asthma there is very little change in lung compliance when resting. But with significant obstruction, lung compliance decreases. With rapid breathing rates, because of exercise, the asthmatic's lung compliance goes down. In other words the lung gets stiffer and the work of breathing goes up

progressively as the respiratory rate goes up. So the exercising asthmatic has dyspnoea for a couple of reasons. First, they can not ventilate adequately, and two, they are really working the respiratory muscles much harder than a non-asthmatic because of the change in lung compliance. The lung volume is larger. The lung is stiffer, it takes much more energy to move the chest back and forth. So, during the hyperventilation of exercise, the asthmatic is consuming significant amounts of energy in the respiratory muscles and getting more and more severe sensations of dyspnoea because of this and because of the inability to ventilate the alveoli properly. So an asthmatic who develops significant airway obstruction can develop problems with exercise and particularly a problem when diving because of the expanding lung volume and alterations in buoyancy.

During ascent, the expanding gas in the lung can be trapped. Overdistension can occur causing pulmonary barotrauma with mediastinal emphysema, pneumothorax and air embolism. This is of concern because of the difficulty asthmatics have in exhaling properly and getting air out of the alveolar spaces. This is one major concern. The other is that when one looks at diving accidents in asthmatics, some of them are actually failures to be able to exercise appropriately on the surface. They just can not exercise, get severe dyspnoea, panic and drown. This is the key to many deaths.

There are about three million sport divers in the United States doing somewhere between twenty and fifty million dives a year. It is very hard to guess the right number but somebody said if one estimated between eight and ten dives per person per year one would end up with about thirty million dives, so twenty million dives a year is a conservative estimate.

Normally about a hundred diving deaths are recorded every year, sometimes fewer and sometimes more. Of those diving deaths about 30% are due to air embolism. There many reasons for these deaths including such things as acute myocardial infarction and getting run over by boats. The University of Rhode Island kept statistics, which were basically collections of newspaper clippings, for a long time. It is a fairly accurate way to look at reported deaths and they found one death due to asthma in ten years of collecting data. One diving death due to asthma in ten years of collecting data, so it is apparent, from the data bases that are available, that asthma is not showing up as a major player in the causes of death in the diving population.

Carl Edmonds' data would suggest that there is a fairly significant contribution of asthma to diving deaths.² He said that with a 1% incidence of asthmatic divers 9% of diving deaths were associated with asthma. The original paper describing the series where 9% of deaths were in asthmatics had no mention of the incidence of asthmatics

in the population studied,³ I think there are questions about the accuracy of Edmonds' report as it is the only report that suggests that asthmatics are so under represented in the diving community.

The DAN data on diving accidents, collected by voluntary reporting, has established a good reputation in the United States and other countries. The reporting is not compulsory, but reasonably reliable. Up to 1987 they had recorded 95 arterial gas embolism cases and estimated the risk of an arterial gas embolism at about one in two hundred thousand dives. Of the 95, thirty eight cases had enough data to find a history of asthma. Unfortunately there was inadequate data in the other 57. There were five asthmatics in the 38 cases where there was adequate data. So you could argue that the incidence of asthma causing arterial gas embolism was either 13% (5 of 38), or 5% (5 in 95). Remember that the estimated incidence of asthma in the general US population is around 10%. Based on this information one could come up with an estimate that asthma increases risk for arterial gas embolism by about two or three times. If the risk is one in two hundred thousand without asthma, then a factor of two, one in one hundred thousand, is still a very low risk of arterial gas embolism in an asthmatic diver. So, here, unlike the data that Carl Edmonds published,² the contribution of asthma as a risk is really quite low in the population of reported injury data from the DAN database.

Corson et al. did some more sophisticated statistical analyses of the DAN database.⁴ In the 1991 data there were twelve hundred cases of decompression related illnesses. One hundred and ninety six of them were gas embolism. Sixteen of the 196 (8%) had a history of asthma. There were 755 type two decompression sickness, 54 (7%) with a history of asthma, and 25 of the 54 (3% of the 755) were active wheezing asthmatics when they got their decompression sickness. So, there was some interest in the fact that an asthmatic would not only have an increased risk of arterial gas embolism, but also, for some reason, the risk of serious decompression sickness would also increase.

Using logistic regression analysis, Corson came up with the ratio for an asthmatic versus a non-asthmatic diver of about 1.58 to 1 increase in risk. He came up with a ratio for an asthmatic with clinical symptoms at the time they were diving, versus a non-asthmatic of about two to one. The 1.58 to 1 was not significant and 2 to 1 just barely made significance, so it is hard to say that the asymptomatic historic asthmatic has any higher risk than a non-asthmatic in a large database.

The active asthmatic seems to have roughly 2 to 1 increase in risk for having any kind of a diving accident. That includes arterial gas embolism and serious decompression sickness. But again, 2 to 1 in a population where the risk is 1 in 200,00 leaves the active asthmatic with an average risk of about 1 in 100,000 dives for an

accident. That is a very low risk for having a diving accident relating to the active asthma patient. The non-active historic asthma patient essentially has no increase in risk, or if you want you can use this 1.58 to 1 but even that was not statistically significant. That is the data that comes from a large database reported to DAN.

The DAN investigators have reanalysed the data each year subsequently, now that they have got it all modelled in their computer, and it continues to support this idea that the active asthmatic has about a 2 to 1 risk in a pool risk of about 1 in 200,000 to start with.

There was a survey done where people were asked if they dived with asthma and if they had had any accidents.⁵ Obviously all the dead asthmatics could not respond to the survey, so it is really hard to tell what the denominator was. I thought it was interesting that there were nine recreational divers in England, who wheezed every day, who had logged about twelve hundred dives over several years and had no adverse effects. All these folk felt that they could dive within one hour of an asthma attack. A couple of them commented that if they had an acute attack of asthma they would take their adrenalin, wait about an hour and then go diving again. Yes, one can dive with asthma. People have done it before. The question is what is their risk. The fact is there are acute serious asthmatics who dive and somehow do it safely. This was not a statistically valid survey because we do not know the denominator. We do not know the number of people with asthma who did not respond.

I think the way we should approach asthmatics is, first of all tell them that if they are an active asthmatic they probably have an increased risk of arterial gas embolism or decompression sickness which is about twice the average pooled risk, which in numbers is about a 1 in 100,000 dives risk of having an accident. This is the Desert Storm approach, young people come with a history of asthma, but no symptoms, enter a new environment, and all of a sudden they have asthma. One concern is that the status of an asthmatic can change while diving and that is an unpredictable process. Generally, we would advise an asthmatic not to dive, particularly the active asthmatic, but not necessarily the asymptomatic historic asthmatic, but the percentage of divers, at least in the United States, who were diving with asthma is the same as the population percentage. So these people are getting into diving and obviously, for the most part, doing it safely because the statistics are not singling them out as a high risk population.

I wanted to finish with the facts that the pathophysiology of asthma is characteristically defined by airway obstruction and by over inflation of the lungs and that there are a lot of asthmatics diving. There are lots of symptomatic asthmatics diving and they do not seem to incur the kind of risk that we hypothesise from the

theoretical aspects. Tom Neumann and I and a few others did a survey of the literature on asthma, published in the *Annals of Allergy*,⁶ from which much of this paper has been taken. The issue for this meeting is to try to define a class of asthmatics who can dive safely and to screen out those who should not dive.

Veale

The pathophysiology of the changes, in lung volumes and in compliance, with worsening asthma, suggest that full spirometry, which would include the measurement of FRC and residual volume and total lung volume, is necessary in the assessment of all asthmatics. Therefore doing simple spirometry in this group is quite inadequate.

Bove

In my institution the pulmonary department is on the same floor and within sight of the cardiology department. So, whenever I get an asthmatic diver referred to me, after taking a history and doing the examination, I walk them down the hall to the asthma team, and have them take care of the patient. If you give a person with asthma to a pulmonologist, you certainly do not get just spirometry. You get a very thorough pulmonary function testing, volumes and all. However I personally think that ordinary spirometry is probably enough to screen out the worst of the asthmatics and the subtleties that one gets by going further may not really give one much more useful information. In other words the asthmatics that show up during spirometry may be the ones that should be screened out and everybody who has normal spirometry probably can dive. I throw that up as an issue because I do not know the answer.

Veale

The other slight problem is that asthma is totally dynamic in that one may have perfectly normal lung function one day and be in a critical care unit two days later.

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A RESPIRATORY PHYSICIAN'S APPROACH TO ASTHMA AND DIVING

A Veale

Prescriptive standards are designed to overcome an area of ignorance. They are designed for people without knowledge to lead them to the right answers. If they do not fulfil that then they are bad standards. Prescriptive standards have been designed for the ignorant and uneducated by those who do not trust us and who refuse to accept responsibility and therefore assume that you and I do not accept responsibility, you and I being the doctor and the patient.

Prescriptive standards by their nature are an easy way out. For example how does the standard handle, a twenty three year old woman who has hypoparathyroidism, who is, I submit, at much greater risk of death than perhaps some of the people with past asthma. But it is not in the standard so one is able, with a clear conscience, to certify this person as "fit to dive". A fourteen year old with a slipped femoral epiphysis may have some risks from diving. I think prescriptive standards are a cop out for those that are not prepared to think.

I must put the medical risks into perspective. Many, many, many, many more people are killed through poor training or absent training or poor practice or absent practice than by medical factors. As we get older medical risks become much more important in the genesis of morbidity and mortality. Training has been long forgotten. Equipment failure from ones buoyancy compensator which has not been serviced for twelve years and lack of practice after an interval becomes a more important. There is a little blip of medical factors in diving deaths that occur at the beginning, but it is pretty small. I think we have to remind ourselves constantly at this sort of meeting that what we are concentrating on here is nothing more than a pimple.

I think every diver should have a medical examination before diving. I think it should be a proper medical examination, not a Mickey Mouse medical.¹ I think it should be done for you in Australia according to AS4005.1. I think that this standard should outline the