Unfortunately her Emergency Department notes are not available, nor are any details as to her pre-dive course medical examination, if she had one.

I think it likely that she, for whatever reason, experienced a change in consciousness during her final dive and aspirated water even though she apparently did not lose her second stage regulator. I think it likely that air embolisation occurred during ascent while unconscious. Air in the basilar artery would have involved her respiratory centre in the brain stem producing cessation of respiratory muscle activity.

Assuming that the history of blackout one year earlier was provided to her training agency or to her examining doctor, should she have been passed as fit to dive? I think not!

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This is the second paper in the Why Divers Die series by Dr Rees Jones. The first will be found in SPUMS J 1998; 28 (2): 113-117.

PEAK EXPIRATORY FLOW AT INCREASED PRESSURE

Paul S Thomas, Clifford Ng and Michael Bennett

Key Words

Equipment, hyperbaric research, medical conditions and problems.

Abstract

It has been estimated that asthma is as common amongst divers as in the general population. Thus, asthmatic individuals may require hyperbaric therapy and monitoring in this treatment. Asthma is usefully monitored by the Wright's standard and mini-peak flow meters. These devices are versatile and can be used under conditions where electrical supply is difficult or inadvisable. In a hyperbaric chamber electrical sources are restricted due to the risk of fire. We therefore compared the performance of Wright's mini- and standard peak flow meters with a rolling seal spirometer, especially adapted for use in the chamber. The hypothesis tested was whether the peak flow meters, which are density dependent, would over-read compared with the spirometer which is unaffected by changes in density because it is volume dependent.

Seven normal subjects performed volume-dependent spirometry to derive peak expiratory flow (PEF) and PEF manoeuvres using standard and mini-PEF meters at sea level and at 3, 2.5, 2, 1.5 and again at 1 atmosphere (ATA). There was a progressive and significant decline in PEF with increasing pressure as measured by the spirometer (69.5+/-0.8% baseline at 3 ATA), while the PEF meters showed a progressive increase in their readings (107.9+/-1.7% at 3 ATA). Using these data points we were able to derive a correction factor which allows the appropriate PEF values to be calculated from the Wright's PEF meters, if the pressure is known. Thus, the Wright's PEF meters can be used under conditions of hyperbaria, if a suitable correction factor is used.

Introduction

It is well recognised that flow is dependent upon density and the flow of gas from the lungs is no exception. In situations of increased gas density, flow is reduced and becomes increasingly turbulent, which can result in an increased time to exhale and less efficient gas exchange. If there is already a critical narrowing of the airway, then this reduced flow can impede exhalation, so that it is incomplete before there is a need for the next inspiratory cycle to begin. This can lead to gas trapping in the airspaces or alveoli. Asthma is the most common reversible disorder with airway narrowing. Airway obstruction in the older population is usually secondary to smoking and only partially reversible.

In situations where gas density is reduced airway obstruction can be measured under both field and experimental conditions, e.g. mountaineering expeditions and hypobaric chambers.[1] In hyperbaric situations where gas density is increased, e.g. diving, studies must be confined to hyperbaric chambers.

Previous work has indicated that the peak expiratory flow (PEF) and forced expiratory volume in the first second (FEV_1) are increased under conditions of reduced gas density. Therefore, as gas density increases the PEF and FEV_1 will fall. This has been demonstrated previously by breathing mixtures of gases of different density at sea-level and in hyperbaric chambers.[2,3]

The number of indications for hyperbaric oxygen therapy is increasing,[4] and therefore some individuals with airway obstruction may have indications for such treatment. In addition, since an unknown number of asthmatic subjects dive, it is evident that some may well develop problems relating to decompression and may require treatment in a hyperbaric unit. A subject with asthma or airway obstruction may require airway monitoring while in the chamber. To reduce the risk of fire, equipment within a hyperbaric chamber is preferably non-electrical or low voltage, particularly where supplemental oxygen is being used. A number of incidents, many fatal, have been described where these precautions were not observed.[5]

Most respiratory monitoring equipment requires an electrical supply, with the exception of the peak flow meter. We hypothesised that it would be easy to use a handheld peak expiratory flow meter in the hyperbaric chamber, which could be used to monitor those who require hyperbaric therapy and who also have airway obstruction.

Peak flow meters, however, only measure one point of the expiratory phase, and also are dependent on gas density.[6,7] True peak flow increases as gas density falls, because gas flow under these conditions exhibits less turbulence and becomes more laminar. Previous work has demonstrated that under hypobaric conditions (either in a hypobaric chamber or at altitude), or breathing gas which is less dense than air, then the "true" peak flow as measured by a spirometer will increase.[1,2] The mini-PEF meter, however, demonstrates a fall in PEF under these conditions, as it is dependent upon the density of the ambient gas. Previously we have demonstrated that these values can be corrected to yield the true values by the use of an appropriate equation.[1]

We therefore hypothesised that, under hyperbaric conditions, the mini-PEF meter would over-read, while true PEF would fall. The data derived from the mini-PEF meter should, however, enable a correction factor to be employed to derive true PEF. This presentation is based upon data which has already been published elsewhere.[8]

Methods

We compared the performance of Wright's mini- and standard peak flow meters with a rolling seal spirometer, especially adapted for use in the chamber. The hypothesis tested was whether the peak flow meters, which are density dependent, would over-read compared with the spirometer which is unaffected by changes in density because it is volume dependent, and whether a correction factor could be derived to yield "true" PEF.

Seven normal subjects performed volume-dependent spirometry to derive peak expiratory flow (PEF), and also PEF manoeuvres using standard and mini-PEF meters at sea level (1 atmosphere absolute, 1 ATA) and at 3, 2.5, 2, 1.5 and again at 1 ATA. The chamber was pressurised from sea-level to 3 ATA and then held at 15 minutes at each stage thereafter.

Results

There was a progressive and significant decline in PEF with increasing pressure as measured by the spirometer (69.5+/-0.8% baseline at 3 ATA), while the PEF meters showed a progressive increase in their readings (107.9+/-1.7% at 3 ATA) (Figure 1). Figure 2 indicates the difference between the spirometer flow-volume loops for normal subjects at 1 and 3 ATA. It can be seen that flow is significantly reduced at 3 ATA under these conditions (p<0.001 ANOVA).

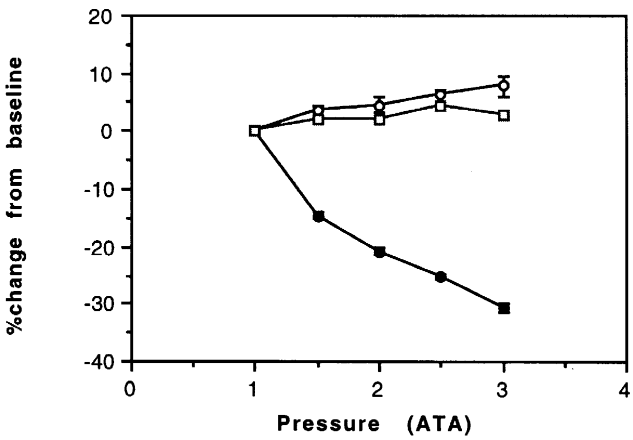

Figure 1. Change in PEF with increasing pressure, as measured with a Wright's standard peak flow meter (open squares), a mini-peak flow meter (open circles) or a spirometer (closed circles). Change in mean PEF is expressed as percentage of sea-level value. (*Reproduced with permission from reference 8, copyright The Biochemical Society and the Medical Research Society*).

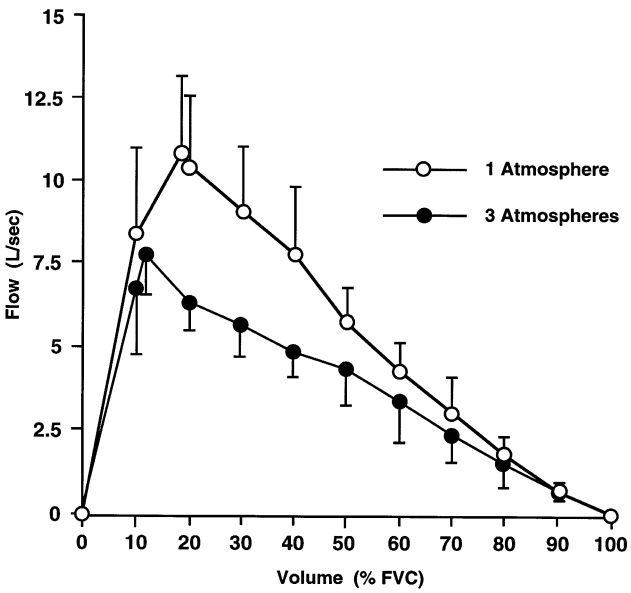

Figure 2. Mean flow volume loops for seven subjects at sea level (open circles) and at 3 ATA (closed circles). (*Reproduced with permission from reference 8, copyright The Biochemical Society and the Medical Research Society*).

Using the these spirometric PEF data points, and the spirometric PEF data from the previous study under hypobaric conditions,[1] plus data from the experiments of Schilder et al.,[2] we were able to demonstrate a relationship which indicates that PEF readings depend mainly on gas density (Figure 3).

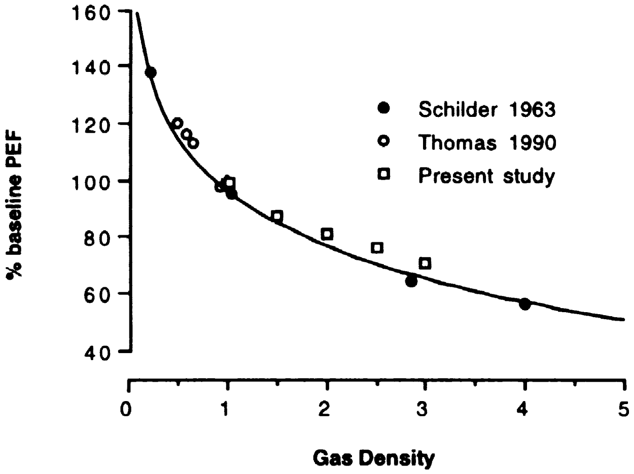

Figure 3. Effect of gas density on peak expiratory flow. The plot was obtained by using data from the present study, (open squares), from Thomas et al.[1] (open circles) and from Schilder at al.[2] (closed circles). (*Reproduced with permission from reference 8, copyright The Biochemical Society and the Medical Research Society*).

From these data, it was possible to derive a correction factor which allows the appropriate PEF values to be calculated from the Wright's PEF meters, if the pressure is known (Figure 4).

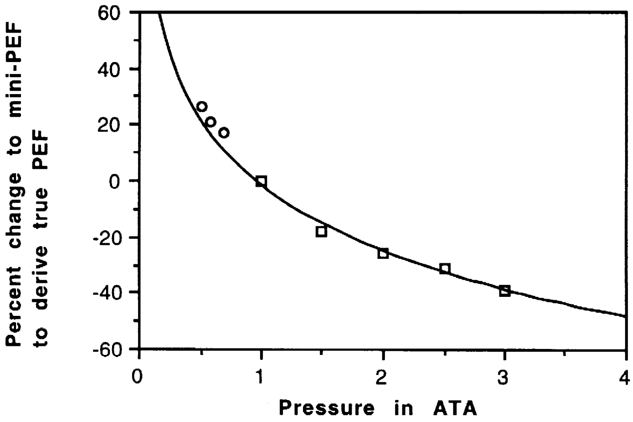

Figure 4. Plot of pressure against percentage change required to derive "true" peak expiratory flow, as measured by the spirometer, from the Wright's mini-PEF meter values. Data were derived from the present study (open squares) and from Thomas et al.[1] (open circles), using identical devices. (*Reproduced with permission from reference 8, copyright The Biochemical Society and the Medical Research Society*).

The change required in mini-PEF reading to give "true" PEF(%) is
= [77.92 x log(pressure in ATA)]-1.61

Using this correction factor, PEF meters can be relied upon under conditions of hyperbaria.

Conclusion

As predicated, Wright's mini-, and standard PEF meters over-read under conditions of increased pressure. These values can be corrected to the "true" PEF values by the use of a correction factor. It is therefore feasible to use the Wright mini-PEF meter in the hyperbaric chamber, should PEF monitoring be appropriate.

The study has some limitations in that it assumes that dynamic resistance remains unchanged, which may not be the case in smokers who have small airway obstruction. These latter subjects also have lung function abnormalities which are particularly seen in the latter part of expiration, after the point of peak expiratory flow. Therefore, it should be appreciated that PEF does not reflect the entire flow volume assessment of expiratory flow.

Asthma is a condition of reversible airway obstruction, most manifest in the expiratory phase of respiration. It affects up to 10% of the population and, in some studies, this prevalence is the same in the recreational diving community.[9] Most recommendations suggest that those who have poorly-controlled asthma should not dive,[10] and some more stringent guidelines suggest that those with any history or evidence of asthma should not dive. If there is doubt about the history of asthma, the subject may be referred for a bronchial provocation test. These use one of a variety of different substances (e.g. methacholine, histamine, hypertonic saline) in increasing concentration to induce constriction of the airway. Criteria for these tests have been established and define those who are likely to have asthma bronchoconstrict (usually a 20% fall in the forced expiratory volume in the first second (FEV_1)) at or below a certain concentration or dose of the substance inhaled. Those who do not react at this concentration, are considered non-asthmatic. On this basis, potential divers can be advised not to dive. As many asthmatic subjects nonetheless dive, it is likely that some will require monitoring in the hyperbaric unit either for decompression illness or for hyperbaric therapy.

Hyperbaric treatment is indicated for a variety of conditions and, inevitably, the benefits of the treatment will have to be considered in the light of any relative contra-indications, such as airflow obstruction. A simple PEF monitor may help to monitor such patients should such therapy be strongly indicated.

Acknowledgments

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VISION, DARWIN AND THE DEEP BLUE SEA
The visual sense and adaptations for terrestrial and aquatic sight.

Malcolm Le May

Key Words

Physiology.

Introduction

When we learn to dive we are told that the ocean is a "hostile environment" and that we are wholly adapted for life on the land. This is only partly true. Man has shown a liking and a fascination for immersing himself in water which is unlike that of many of our fellow apes. Our closest relative, the chimpanzee, has such an aversion for water that a narrow moat is sufficient to confine captive apes. With the exception of a Japanese relative with a penchant for volcanic springs, other monkeys tend to avoid immersion.

In spite of our seeming adaptation to the land, we continue to carry with us reminders of our life in the sea. Our internal environment is isolated from the hostile dry outside by a space suit of waterproof skin, and when that is damaged, we leak. If we damage enough of our skin, for example by burning, we die. Our internal osmolarity, equal to 0.9% saline, is a reminder of the salinity of the primordial sea. The process of reproduction remains a function conducted in a moist environment and we spend the first nine months of our life immersed. During the process of birth we mimic the change from an ocean dweller to a land mammal, repeating the invasion of the land in microcosm over and over again as each child is born.

In spite of a long history of living on the land, our eyes remain an aquatic based sense. In changing from vertebrate life underwater to life above, gills and swim bladders have become lungs, fins have become limbs, but eyes still remain essentially the organ that evolved in the sea. The continued function of our vision requires an adequate supply of tears and our eyelids ensure that our eyes are kept moist, returning briefly to the aquatic environment twenty times every minute as we blink to maintain the pre-corneal tear film.

We do not see well underwater without the aid of a mask.

Land animals rely on the interface between the air and the cornea for most of their refractive correction and in water man is hyperopic by some +43 Dioptres. Vision evolved in the sea and an interest in how the eye was adapted to different environments led me to a search for the origins of the eye as we know it.