

Can SaO₂ measurements during recovery be used to detect lung barotrauma in freedivers?

Erika Schagatay, professor. Environmental Physiology Group, Dept. of Health Sci., Swedish Winter Sports Research Centre, Mid Sweden University, Östersund, Sweden.

This is a hopefully “user friendly” **preliminary report** of studies in the ongoing project “**Lung squeeze and pulmonary edema in freedivers**” supported by AIDA-international, with the purpose of evaluating SaO₂ recovery for detecting lung barotrauma in competition freediving. Note that this is not a review of the field of pulmonary barotrauma, or a complete scientific report. A final, fully referenced scientific report including additional data will be published once the project is finished.



ABSTRACT

Aims: Repeated lung barotraumas is an identified risk factor in deep apnea diving, and a delayed SaO₂ recovery most likely reflects pulmonary edema. The aims were to determine: 1) the normal rate of SaO₂ recovery after deep dives; 2) if delayed SaO₂ recovery is associated with chest symptoms.

Methods: SaO₂ recovery was studied after CWT, CNF and FIM competition dives, which are more likely to involve lung strain than training dives. Both genders were included; some participated several times with dives from different disciplines or events. SaO₂ was recorded via finger pulse oximetry directly after judgment and until at least 3 min of full recovery, or for at least 10 min. Timing after surfacing was noted and divers were observed and asked for subjective symptoms from the lungs. Measurements of approximately 100 competition apnea dives for depth (25-100m) were included in the study, after exclusion of incomplete recordings. Analysis was done in two steps: **1)** To determine the normal rate of SaO₂ recovery, dives with symptoms (chest discomfort, coughing, heavy breathing, blood from upper or lower airways) and/or syncope were excluded, and average recovery rates calculated. **2)** With normal SaO₂ recovery established, the dives with symptoms were included (n=12), and the dives with SaO₂ > and < 97%, and > and < 95% after 10min were analyzed for symptoms. In addition, pulmonary ultrasound was used to detect “ultrasonic lung comets” (UCL) indicating lung fluid, in parallel with SaO₂ measurements after deep dives, to determine whether these were correlated, i.e. if SaO₂ reflects pulmonary fluid.

Results: Part 1) In all disciplines, normal recovery of SaO₂ to 97% occurred within 4 min after dives <50m, and within 8-9min after dives >50m, with differences between depth categories until 4 min of recovery (P<0.05). **Part 2)** At 10 min after surfacing, there were 14 cases of SaO₂ <97%, including 8 cases with symptoms. There were 6 cases of SaO₂ below 95%, of which 5 cases reported symptoms. Among dives with >97% SaO₂, there was 1 case with chest symptoms and 3 cases with BO. The number of UCL detected by pulmonary ultrasound correlated with SaO₂ values after diving, which verified that SaO₂ is a good indicator of pulmonary fluid associated with barotrauma.

Conclusions: SaO₂ recovery occurs within 10min after surfacing, in almost all dives without symptoms. SaO₂ <97% at 10min is usually associated with lung symptoms, with a few false negative cases, and SaO₂ <95% at 10 min is almost exclusively associated with symptoms. There were a few false positive cases, but these can most likely be detected with continued monitoring. We concluded that recording SaO₂ recovery after deep dives is useful to detect lung barotrauma.

BACKGROUND

After limited training, most healthy humans can learn to make voluntary dives to 20-30 m depth thereby likely not diving past a depth where compression has reduced the lung volume below residual volume (RV), which is normally about 25% of the total lung capacity (TLC) at young age (Paoletti et al 1992). Since the introduction of organized competition freediving in the 1990-ties, "Apnea" depth records have increased significantly without apparent signs to level off. Initial estimates proposed that pulmonary barotrauma would occur in humans when lungs are compressed below residual volume (RV) at depths around 30 m, but the current record without pulmonary complications is 214 m. This large discrepancy may possibly be explained by training methods that alter lung function and chest compliance to help prevent negative pressures from developing inside the chest. However, certain individuals appear to be particularly prone and suffer from lung squeeze even at moderate depths (Cialoni et al 2012). Consequently, the current understanding of how pressure affects respiratory physiology is rudimentary at best, and there appears to be considerable plasticity and potential to alter human lung function and mechanics by directed training methods and exposure to extreme environments rather than evolutionary traits that limit deep diving.

While lung squeeze seems not to occur in most divers and dives despite exceeding depths where compression reduces lung volume well below the expected RV, the incidence of pulmonary barotrauma after competition dives has increased with progressively deeper diving. In 2013 there was a lethal accident, the first in competition, the likely cause being lung barotrauma. The diver surfaced unassisted after a constant weight no fins (CNF) dive to 72m, but failed to recover from the diving induced hypoxia due to problematic breathing, and later lost his life.

In an investigation of this accident, two contributing factors were identified; 1) repeated barotrauma close in time before the lethal event, and 2) strain due to unusual movements by the diver at depth during repeated turns. An aim of AIDA since the accident has been to find means of limiting such risks, if possible by rule changes in deep freediving. The primary aim of this research project is to establish a system for detecting mild and severe edema/ lung barotrauma via a simple measurement via e.g. pulse oximetry in order to determine the period a diver should be restricted from further diving after such an event.

What is lung barotrauma (lung squeeze/pulmonary edema)?

Pulmonary edema involves fluid in the lungs, and it can occur in a number of situations, e.g. as a consequence of heart failure, high altitude exposure, immersion or deep breath-hold diving. Fluid in the lungs may limit gas exchange and in situations with high demands on efficient gas exchange, e.g. at high altitude or after a straining breath hold dive, the situation may become critical. With pulmonary edema the fluid comes from the blood, when plasma filters in through the capillary walls, or it may occur as a result of a rupture of vessels in the lungs or airways due to lung compression. Both more widespread pulmonary edema and local lung squeeze related ruptures may be categorized as "lung barotrauma" or "pulmonary barotrauma" when caused by pressure differences. The cause of lung squeeze in deep freediving is mainly related to the development of negative pressure in the lungs at depth where the increased hydrostatic pressure compresses the air inside. However, the great individual differences in susceptibility for squeeze show there are other factors present, which may not be directly related to the hydrostatic pressure/depth of diving. However, the problem seems to become relevant with diving to depths of over 40 m (Linér and Andersson 2008), although sensitive individuals can develop symptoms also at shallower depths where their pulmonary mechanics suggest they should not be at risk. It is not known to what extent genetic factors or training allow some people to dive to more than 100 m depth without apparent adverse effects, while others suffer from pulmonary hemorrhage and signs of pulmonary edema at much shallower depths.

Can lung barotrauma be prevented?

Mammalian divers may dive to great depths, despite breathing air. In deep divers among seals and whales, different modes of counteracting negative effects of pressure on the lungs have been developed, suggesting a number of factors may be involved – and could possibly be explored to avoid squeeze in humans. The pressure related compression possibly causing injuries also relates to the mammalian deep diver's need to avoid narcosis and excess nitrogen build up in tissues – two sides of the same coin; for avoiding problems with high pressure gases including decompression sickness (DCS) a compression of air spaces where gas exchange takes place may be desirable. While most mammalian deep divers seem to have solved this problem, human divers may or may not succeed in avoiding injury.

In order to develop routines for determining the degrees of lung damage or impaired lung function in human freedivers, we need to better understand what is the normal recovery after deep dives and how to identify pathological development of such conditions, and how they heal. To distinguish between mild cases which heal rapidly and spontaneously – and more severe cases which may be improved by on-site treatment, or require long term rest or possibly hospital medical treatment, we need to know what identifies these different categories. A first step towards this is to establish baseline measurements of what is the “normal” recovery pattern from depth exposure during apnea, to use this as a background for identifying mild and severe cases of pulmonary injury.

The overall aim of this research is to develop a better understanding of the occurrence of pulmonary edema and barotrauma (squeeze) in freedivers, and if possible means for detecting and preventing injury. The specific aims of the present study are **1)** to determine the normal rate of recovery of arterial oxygen saturation (SaO₂) in freedivers after deep competition dives in the respective disciplines CWT, CNF and FIM, and **2)** to determine if delayed recovery is associated with symptoms of barotrauma. The knowledge could be used for identifying cases of delayed recovery, which may suggest pulmonary edema or other types of lung barotrauma, an identified risk factor with repeated deep diving. When normal recovery is known, abnormal values can be identified and used to make divers rest before deep-diving again, aiming to prevent accumulated lung injury.

BRIEF METHODS

Subjects

All registrations were done in competition dives. The reasons for studying factors indicating lung barotrauma in competition, not during training, is that divers strain more in competition dives and more often produce their personal best, which may lead to increased lung strain. The main reasons why this cannot be studied in experimental dives is ethical, as maximal dives may involve risk of injury. Both men and women volunteers were included. Some divers participated repeatedly with dives from different disciplines or events.

Measurements

Recordings were done during several international AIDA competitions before (when possible) and after dives on or near the platform after the diver had been judged. SaO₂ was recorded via finger pulse oximetry directly after judgment and until at least 3 min of full recovery with values of at least 97% saturation, or for at least 10 min if not recovered. Data about the dives were collected, timing after surfacing was noted and divers were observed and asked for subjective symptoms from the chest. Measurements of approximately 100 competition dives to 25-100m depth were included in the study, when dives with incomplete information or recordings had been excluded (small gaps in recordings were extrapolated). In recorded divers, SaO₂ was normal (>97%) before the dives.

In a separate study, SaO₂ values were collected and pulmonary ultrasound used to detect Ultrasonic Lung Comets (UCL) indicating lung fluid.

Analysis

Recovery of SaO₂ after diving was analyzed in two steps:

- 1)** For establishing normal SaO₂ recovery rates, dives were first analyzed with the exclusion of dives with observed or reported symptoms. The excluded dives showed symptoms of chest discomfort, coughing, heavy breathing, blood from upper or lower airways and/or syncope.
- 2)** When normal SaO₂ recovery had been established, the dives with symptoms were included (n=12), and the frequencies of symptom dives among dives with SaO₂ >97% versus <97% and <95% after 10min was counted.

Data is presented as mean values for normal recovery for separated disciplines and for all disciplines pooled, but always in separate depth categories; < 50 m and >50 m. A plot of SaO₂ at 10 min and depth of all dives including symptomatic dives is provided, with cut off SaO₂ of 97% marked, and symptomatic dives identified. Some examples of long term SaO₂ development of dives with delayed recovery are presented separately, and a graph of SaO₂ versus lung comets is presented.

Statistical analysis was done using unpaired Students t-test, with significant differences accepted at P<0.05. Pearson's correlation analysis was done between SaO₂ values and UCL after deep dives.

PRELIMINARY RESULTS

Summary normal recovery

In this part, normal recovery is presented for all disciplines, first with disciplines separated (**Fig.1**) and then pooled (**Fig. 2**), after exclusion of dives resulting in symptoms of squeeze/edema or blackout (BO). In these "normal" dives, in average, full recovery to SaO₂ of 97% occurred within 10 min in all disciplines (**Fig. 1**). In all disciplines there was a slight effect of dive depth (and accordingly possibly duration) on SaO₂ recovery; dives deeper than 50 m required slightly longer time for full SaO₂ recovery. This depth difference was significant only for FIM dives (P<0.05) where deeper dives showed a lower mean SaO₂ until 5 min recovery (**Fig. 1c**). In the deeper dives (mean depths shown in figures) a period of 8-9 minutes in average was needed to reach 97% SaO₂. Normal SaO₂ of 97% is reached within 5 minutes in shallower dives in all disciplines. When comparing disciplines, the slowest recovery thus seems to occur in deeper FIM dives. Average depths were similar between disciplines. Limitation: Deep CNF includes few dives, and all disciplines lack dives of >100 m.

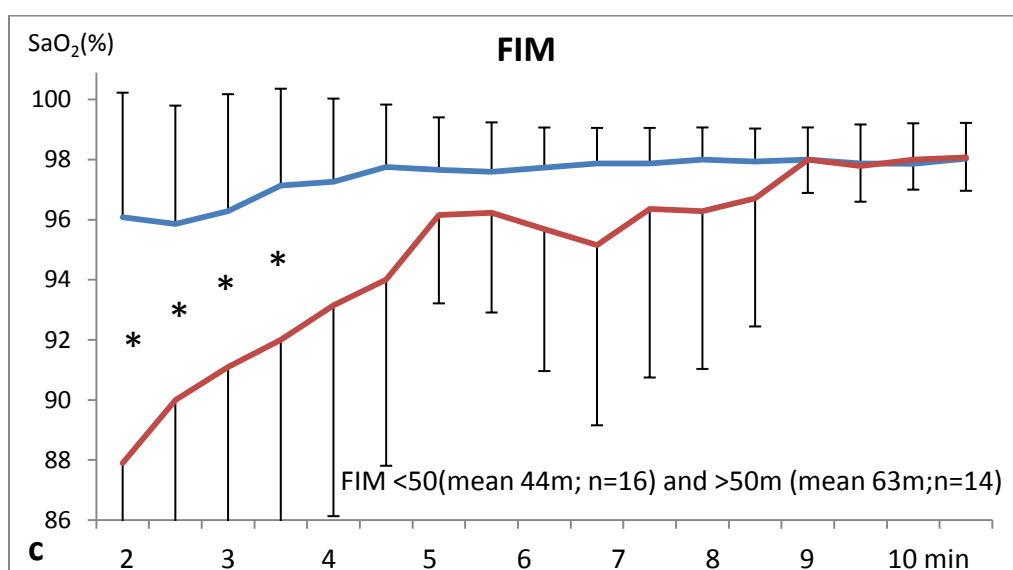
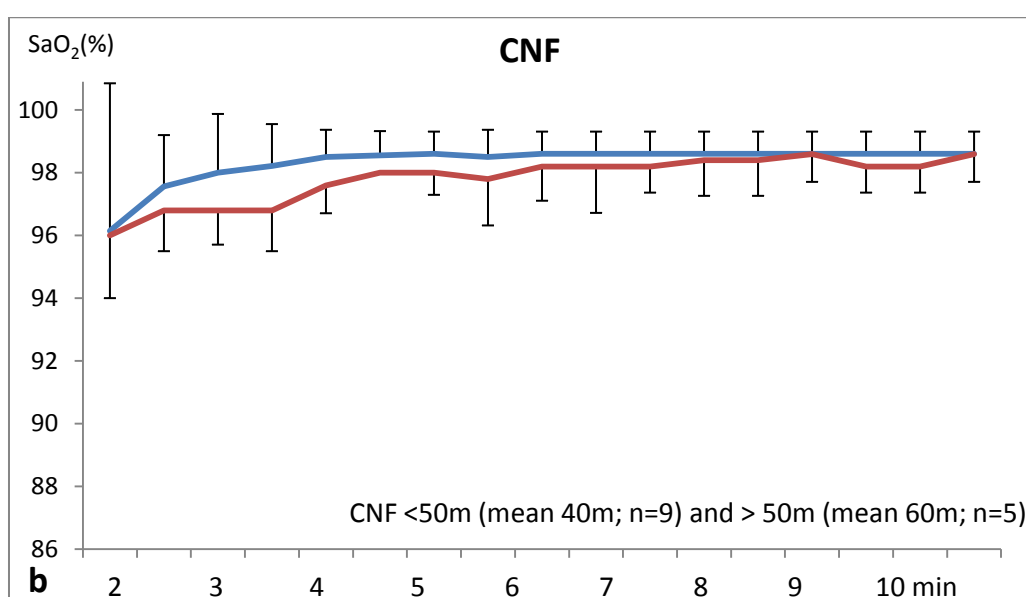
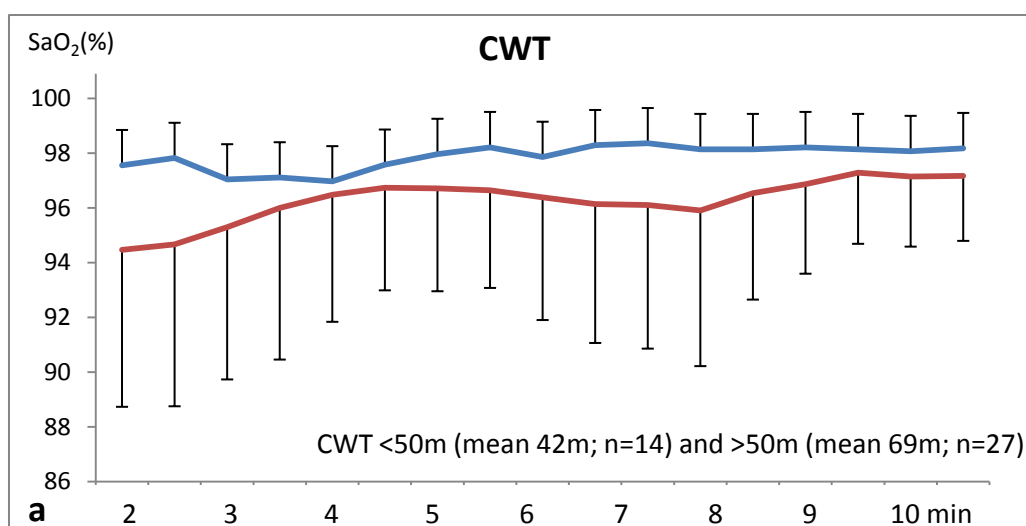


Figure 1a-c. Summary of mean (SD) SaO₂ recovery for dives without symptoms from all disciplines separated, pooled for average depths <50 m (blue; upper line) and >50 m (red, lower line), respectively. Mean depths and number of divers are indicated in the figures. Dives with symptoms of bleeding, heavy breathing, subjective “bad feeling from lungs” or BO were excluded. Significant difference at P<0.05 is indicated by *.

When dives from all disciplines were pooled based on depth alone, the SaO₂ was lower after dives deeper than 50 m (P=0.05) across the first 4 min of recovery (Fig 2). Recovery until 97% SaO₂ was completed within 4 min in shallower dives, while it reached 97% in 8 min 30 s in the deeper dives.

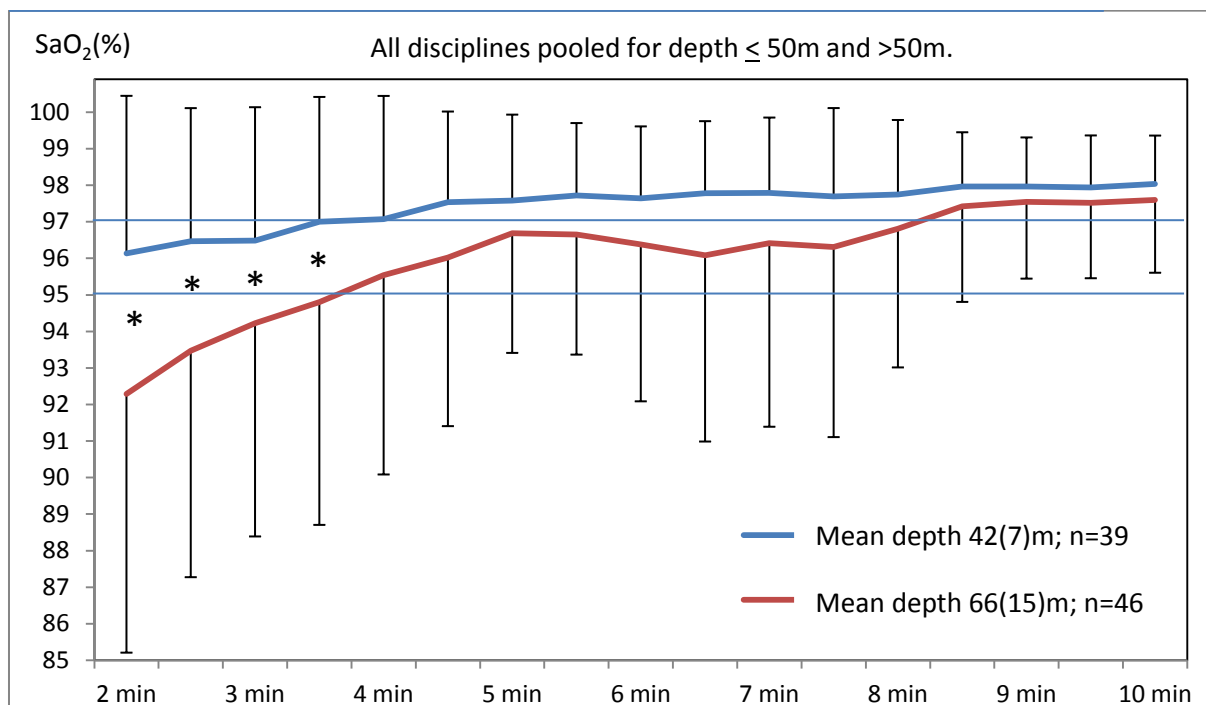


Figure 2. All dives without symptoms pooled for depths < 50m and >50 respectively. Mean depths and number of divers indicated in the figure. Return of SaO₂ to 97% occurred in average within 8-9 min after deeper dives, and within 4 min after dives < 50 m. SaO₂ levels during recovery were different between deep and shallow dives up to 4 min of recovery (P<0.05 indicated by *). The large standard deviation (SD) during the initial 7-8 min of recovery indicates that some divers may present with lower SaO₂, while small SD during the last two minutes shows that most divers have fully recovered.

Summary delayed recovery

Delayed SaO₂ recovery was often but not always associated with adverse symptoms; subjective discomfort, shortness of breath, bleeding or sometimes BO (**Fig. 3**). The dives involving subjective symptoms of mild squeeze may or may not be associated with delayed SaO₂ recovery, and BO is often present without a delay in SaO₂ recovery. Combinations of symptoms of barotrauma and BO may occur. **Figure 3** shows SaO₂ at 10 minutes against depth for all dives with registration at 10 min, including cases with symptoms. In 6 cases SaO₂ is below 95%, five of which have obvious symptoms. In 14 cases SaO₂ is below 97%, with 8 cases of symptoms observed in or reported by divers. It appears that a depth of > 45 m is associated with an increased risk of squeeze, which accords with previous observations (Linér and Andersson 2008). Some of the deeper dives were followed by O₂ breathing (indicated in figure 3), although it stopped at least 2 min before recordings were done.

It seems clear that SaO₂ indicates several different types of lung problems, most likely barotraumas from deep diving, however, there are also both "false positive" and "false negative" cases present. With a cut-off level at 97%, some "false positive" are accepted; in this material 8/14 with lower SaO₂ had lung symptoms, thus 6/14 were possibly falsely positive (**Fig. 3**). Using 97% SaO₂, nearly all detectable symptom cases are included. When the 95% SaO₂ level was used, 5/6 cases with < 95% had symptoms. For an approach accepting that some positive cases may be undetected, a level of 95% could thus be used (**Fig. 3**).

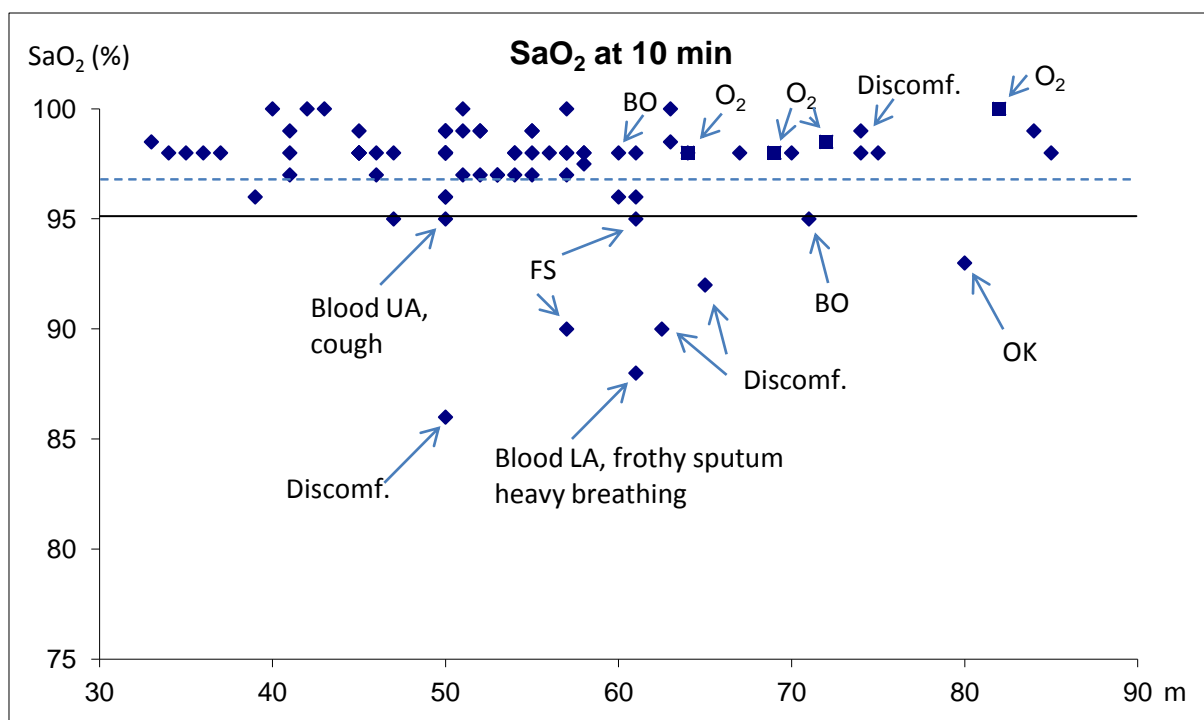


Figure 3. SaO₂ at 10 minutes is shown against depth for all dives with recordings at 10 min, including cases with symptoms. In 6 cases SaO₂ is below 95% (5 with symptoms) and in 14 cases it is below 97% (8 with symptoms). Symptoms observed or reported are: Discomfort from the chest, Blood from upper airways (UA) or lower airways (LA), “FS” indicates frequent squeezers experiencing mild (“usual”) symptoms. One diver with normal SaO₂ reports chest Discomfort, and another with low SaO₂ reports being OK (details presented in Fig 6). Four of the deeper divers were breathing oxygen > 2 min before measurements as indicated by “O₂” (big square dots). The 97% SaO₂ level is indicated by - - - and the 95% level by – in the figure.

Cases with delayed recovery will need further analysis for detailed conclusions; some examples are presented here. A closer look at selected cases shows that responses may vary between individuals and dives. In **figure 4** two CWT cases involving BO are presented with different outcome; the deeper dive with a delayed SaO₂ recovery. In **figure 5** two cases are presented involving barotraumas and subjective symptoms with delayed recovery after CNF dives. Both divers with delayed recovery received oxygen for several minutes, elevating SaO₂, after which it fell again (**Fig 5**). While case 5a seems to be improving, case 5b could be worsening. A follow up at at 20 min would likely determine the severity of their barotraumas. **Figure 6** shows a case with delayed recovery without symptoms.

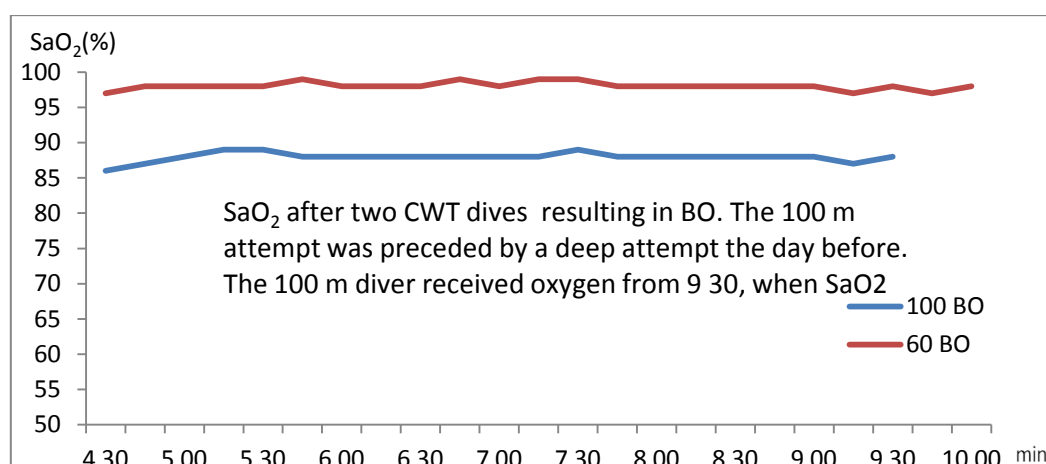


Figure 4. Two CWR dives resulting in BO, one with normal recovery, the other deeper dive showing delayed recovery, with SaO₂ still below 90% at 10 min. The 100 m dive situation may include a lung squeeze/edema. Details are presented in the figure.

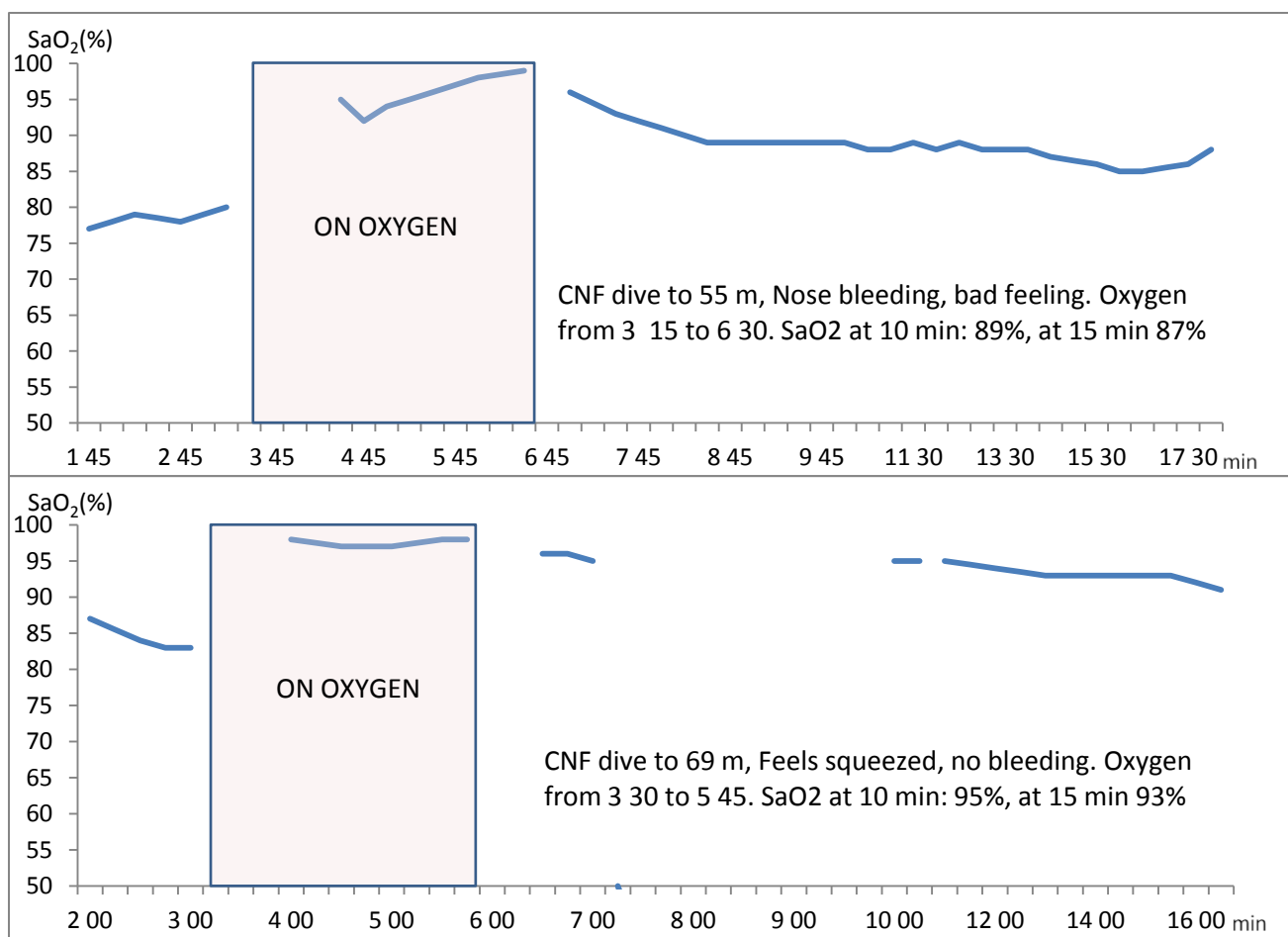


Figure 5 a,b. Two cases of delayed recovery with symptoms after CNF dives. Both received oxygen for several minutes, elevating SaO₂, after which it fell again. While case a. may be improving from a lower level, case b. could possibly be worsening, and both need follow up to determine severity. Details are presented in the figures.

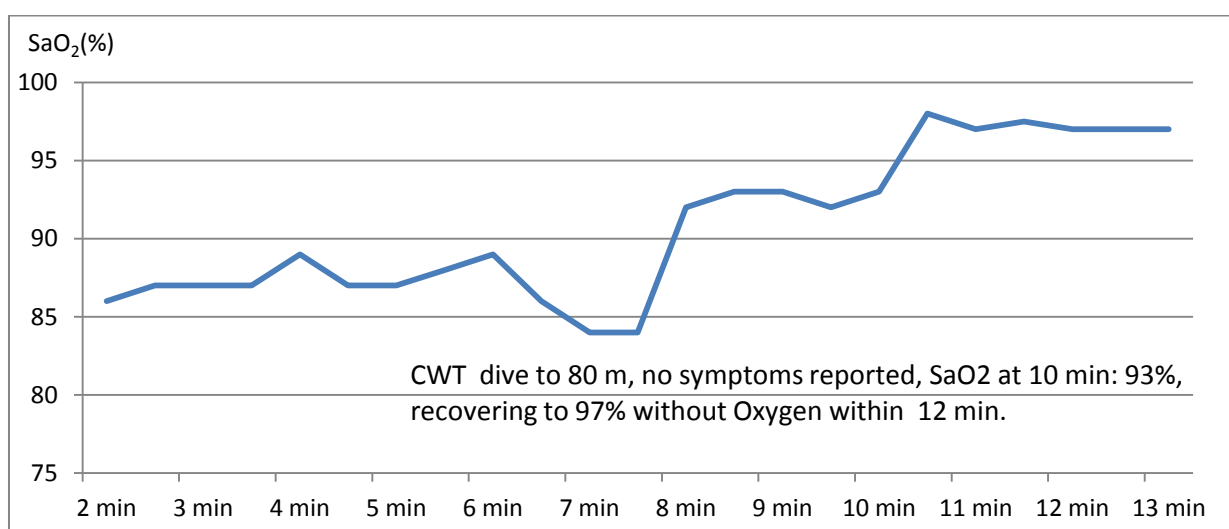


Figure 6. Some deep dives without symptoms may need over 10 min for full recovery. This diver needed just over 10 min to fully recover to 97% SaO₂ after an 80 m dive with no adverse symptoms.

Summary SaO₂ and Ultrasonic Lung Comets

Data from a pilot study including measurements of SaO₂ in combination with lung ultrasound for detecting pulmonary edema via “ultrasonic lung comets” (ULC) is presented in **Figure 7**. In **figure 7**, a preliminary analysis in a limited number of subjects suggests there is a correlation between low SaO₂ and the number of ULC indicating fluid in the lungs. At the time of writing this report, we have just confirmed the finding of a significant correlation between UCL and SaO₂ in a larger material. In this study we also observed that SaO₂ may return to normal levels within 1 h also in barotraumas cases.

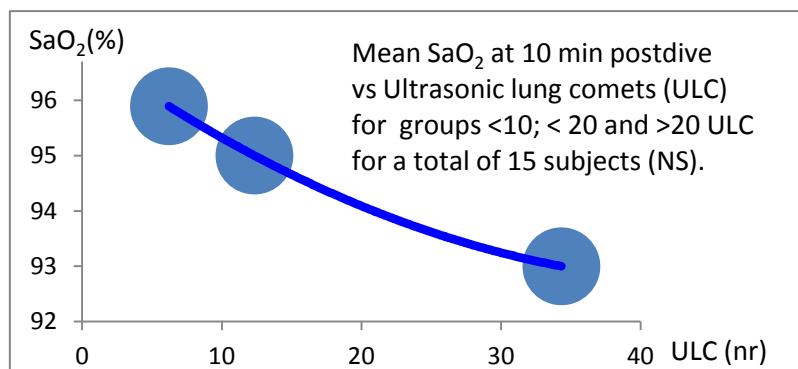


Figure 7. Preliminary data from ongoing study showing mean SaO₂ at 10 min postdive versus ultrasonic lung comets (ULC) measured by lung ultrasound. Subjects have been divided in 3 groups based on ULC<10; < 20 or >20. The data suggest that UCL count correlates with SaO₂ levels. This has been verified by continued studies.

BRIEF DISCUSSION

Using SaO₂ to detect lung barotrauma

The main conclusion from this study is that recovery to normal SaO₂ levels occur in all disciplines within 10 minutes after surfacing in dives of up to 100 m depth. SaO₂ levels lower than 97% at 10 min after diving may suggest lung problems, and levels below 95% SaO₂ very likely involve some problem related to pulmonary barotrauma. Varying approaches can be chosen based on this information concerning which levels of SaO₂ should be used to advice divers to dive or not the following day.

Results thus suggest that a SaO₂ value below 97%, taken in the period 10 – 15 minutes after surfacing, may indicate some kind of lung problem in all disciplines. If this value is used, very few false negative (unhealthy neglected) cases will result. There will be a few false positive cases using this strict approach, but this can be handled with continued monitoring. Suggested approach: A value below 97% at 10-15 min after surfacing could be monitored again and if not resolved at 20-30 min, with SaO₂ back at 97%, a squeeze is likely present, and the diver should probably rest the next day.

A less strict approach, using a level of 95% SaO₂ for detecting problems at 10-15 min, would likely identify the more severe cases, but may allow some false negative milder cases. From a safety perspective, it would be preferable to have some false positive, which can be followed up for determining development. It should be noted, however, that deeper dives (>100 m) may need longer time than 10 min for normal recovery. This approach is less time consuming, and will probably be better accepted by the divers, as it involves limited numbers of false positive cases.

For practical purposes, monitoring SaO₂ for at least a 2 min period within the interval 10–20 min after the dive would likely be manageable. Testing should be done within a maximum of 30 min after surfacing, as after this period, SaO₂ may increase also in cases with pulmonary barotrauma. Measurements of SaO₂ for determining lung injury should be done after at least 2 min without oxygen breathing, as this will temporarily increase SaO₂ levels despite underlying problems.

Approach tested at Cyprus World Championship 2015

During the individual Depth World Championship in Cyprus, the following approach was used for detecting lung barotrauma. Divers reported to the testers within 20 min after their dives, and SaO₂ was monitored for 2 min. Divers with < 95% SaO₂ reported back to the testers no later than 30 min after the dive. If SaO₂ was still below 95%, the diver had to be cleared by the competition medic before diving the next day. Depending on the presence or not of other symptoms, the diver was cleared to dive or advised to rest the following day. This regime was easily managed and seemed agreeable from the diver's perspective. Many divers said this advice was useful for deciding how to plan ahead.

Different types of barotrauma

There are different types of symptoms and conditions of lung squeeze, and also likely different contributions from various risk factors. It is speculated based on limited data (few cases) that squeeze presenting as bleeding from airways is a less severe condition than a more global edema, experienced by the dives as heavy breathing and "feeling squeezed". Irrespective of the exact cause, a low SaO₂ shows that lung function is compromised, and in the critical stage of recovery from maximal apneic diving efforts there is a short window where oxygen uptake has to be maximized. This is why it is such a good indication of lung conditions to monitor SaO₂ recovery during this phase.

It appears from these preliminary data (few cases) that BO is not in itself a condition causing delayed recovery, as even severe BO cases (deep and/or long during) may be followed by rapid SaO₂ recovery once breathing is resumed. In cases where BO is associated with delayed recovery, an underlying problem may be pulmonary edema or other functional disturbances, not the BO in itself. With a normal lung function, SaO₂ seems to be restored to normal (97%) within a few minutes also after a maximal dive ending with BO. This has been confirmed by measurements from STA and other pool disciplines, where no barotraumas are present (data not included here).

Reliability and validity of method

Using SaO₂ as an indicator of lung function after freediving in field conditions is a clear advantage before other potentially useful methods, e.g. spirometry or pulmonary ultrasound. It requires no effort from the diver during monitoring, and is a method that is easily learned and controlled by the tester, and the monitor is not very expensive. SaO₂ % reflects the total outcome of the body's strive to restore the normal oxygen saturation to 97-98% after the desaturation resulting from a competition apnea dive. The recovery thus reflects lung function as well as cardiovascular functions. However in this situation the lung function is expected to be most crucial to the outcome.

SaO₂ monitors are sometimes criticized for being difficult to handle, and for not showing accurate results. However, knowing how to use it helps a lot. Using a good brand of monitor is essential; there are monitors that cannot measure low values correctly. We have used Nonin monitors (also sold as "Medair") and found them reliable, and values have been very close to those measured with advanced and expensive hospital monitors. There are likely other good brands on the market.

It should be realized that there is a time lag of 10 – 20 sec in the recording of SaO₂ in the finger, compared to values from for example the head. Thus the value shown in the finger corresponds to the value that was present in the head 10 s earlier. This is due to the blood circulation time between lungs, heart and the spot for measurement, and it does not affect the results in any way relevant here. Monitoring for a period of at least two minutes is suggested, and monitoring for detection of delayed recovery should be done after a period of at least 2 minutes off oxygen.

If the subject is very cold, vasoconstriction in the fingers may make it difficult to get good readings. However, after a straining dive, once breathing is established, the capillary blood flow is most often

very high even at slightly cold conditions, to compensate for the period it was restricted. In very cold environments, the hand can be warmed before measurements (e.g. in warm water). Monitors must however, be kept dry. The finger should thus be wiped dry before measurements, and the arm and hand not held in a position where blood flow is obstructed, and the finger clip should not be pressed on, just stay on the finger by itself. A few seconds will be needed after application for the monitor to show good readings, thus the very first value can often be disregarded. The monitor should indicate when blood flow is sufficient for reliable values. The Nonin monitor indicates with a green light when reliable values are obtained, if there is no green light, values should not be trusted. There are monitors with probes to be placed on ears or face, but from a practical standpoint it is probably more difficult to use these on a diver just having surfaced and with wet face and a hood.

What does SaO₂ indicate?

SaO₂ % is the percentage of hemoglobin molecules in the arterial blood that carry oxygen, which is 97-98% in a breathing person at sea level. Directly after an apneic dive, SaO₂ is normally lower, as stored oxygen has been used and not yet replaced by resumed breathing. If SaO₂ recovery is delayed from normal after diving, there is a problem somewhere along the train of oxygen transport from the surrounding air to the arterial blood, where SaO₂ is measured. We know that divers often surface with very low SaO₂ levels, sometimes near the point for BO, but as soon as breathing is resumed, SaO₂ rises and with normal lung and circulatory (heart) function normal levels are restored within minutes. Heart rate is restored from low diving levels (diving response) to high recovery levels within the first 30 s – 1 minute after a dive, and it is from a functional point very likely the lungs that are the limiting factor. Why then would there be limitations in lung function? The main problem is if the respiratory exchange in the lungs is blocked by fluid.

What does Heart-Rate indicate?

Heart rate (HR) values were collected together with SaO₂ for a number of dives, but data is not included here. HR, while often high after dives with symptoms, is in itself not likely a good indicator of barotraumas. A high HR indicates stress – but is it good or bad with a high HR? HR increases to compensate for a low oxygen uptake due to poor lung function, so a high HR with a low SaO₂ just shows the system is coping with the hypoxic stress by increasing circulation. Conditions are likely worse should this not happen. However, best is a high SaO₂ together with a low HR, showing everything is back to normal resting conditions, and with normal respiratory function. Another problem with using HR as an indicator of strain or damage is that normal HR varies between individuals to an extent that makes it difficult to determine what is elevated HR. Normal SaO₂, however, varies very little between individuals.

Conclusions

Using SaO₂ for detection of squeeze seems promising (reliability and validity are high for field methods) and it can be easily handled from a practical perspective in a competition situation. The likelihood of detecting pulmonary barotrauma, with possible negative consequences for diving the next day, seem to be good by measuring the SaO₂ on the finger at some point during the time period 10 – 30 min after surfacing. The level for continued monitoring should not be set to low, as some divers may develop edema that worsens after the dive. Whether mild versus severe cases can be identified using this method only is less clear, but using the suggested approach at least severe cases would likely be identified and divers prohibited from diving during the necessary recovery process.

It seems that BO is not a major cause of delayed SaO₂ recovery in itself. It is suspected that cases of BO with a prolonged recovery time beyond the normal pattern related to diving depth and duration of the apnea are related to underlying lung squeeze. There could be other reasons to rest after BO.

Together with other rule changes e.g. limited announcements and improved medical preparedness and procedures at diving competitions, monitoring SaO₂ can most likely help make freediving safer. However, the most important factor is to raise the awareness among the divers themselves, of how apparently small adverse symptoms from their lungs may be a warning to take seriously, not to compromise lung function – and life - after deep dives. Monitoring of SaO₂ during competitions can likely be one aid in increasing this awareness.

Further studies

Further, ongoing studies include measurements of SaO₂ in additional and deeper dives in all depth disciplines, and SaO₂ in combination with lung ultrasound for detecting pulmonary edema. We plan to present a final scientific report concerning these results once recordings are completed.

ACKNOWLEDGEMENTS

I wish to thank all divers voluntarily participating in these measurements and the competition organizers and coaches for their help and a workplace near the diving lines during deep diving competitions. I also want to acknowledge the many coworkers in Environmental Physiology Group at Mid Sweden University, and other people helping us with these measurements. This study was funded by a grant from AIDA-international during 2014. The continued study of pulmonary edema in freedivers was granted by Swedish Centre for Research in Sports for 2015, and additional funding for the continued work is being applied for in various research funding agencies.

References

- Cialoni D, Sponsiello N, Marabotti C, Frammartino B. Prevalence of acute respiratory symptoms in breath-hold divers. *Undersea & hyperbaric med.* 39(4): 837-44, 2012.
- Liner MH, Andersson JP. Pulmonary edema after competitive breath-hold diving. *J Appl Physiol* 104: 986–990, 2008.
- Paoletti P; Viegi G; Carrozzi L; Di Pede F; Modena P; Ballerin L; Baldacci S; Pedreschi M; Pistelli G; Giuntini C. Residual volume in a general population. Effects of body size, age, cigarette smoking, and respiratory symptoms. *Chest.* 102(4):1209-1215. doi: 10.1378/chest.102.4.1209, 1992.