A CLUSTER HEADACHE SUFFERER’S SUPPLEMENTAL GUIDE

TO

ABORTING CLUSTER HEADACHE ATTACKS WITH 100% O2

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Abstract

The following Supplemental User’s Guide and Information Paper on the use of Medical O2 as a Cluster Headache Abortive were developed for cluster headache sufferers by fellow cluster headache sufferers experienced in its use. Too often, a prescription for medical O2 is the only guidance a cluster headache sufferer receives. Most sufferers found a prescription by itself to be inadequate as it lacked the necessary “how to” procedures and information needed in inspecting, assembling, and using medical O2 equipment. Accordingly, we have developed the Supplemental User’s Guide and accompanying Information Paper to meet these needs. They are provided for information purposes only. As such, cluster headache sufferers should use these two documents in concert with, and supplemental to, guidance provided by the sufferer’s physician who has examined the sufferer and is familiar with his or her medical history.

In gathering and analyzing the background information used to develop these two documents, it became clear from the overwhelming body of evidence including clinical studies, that if administered properly, 100% medical O2 is the most efficient and cost effective cluster headache abortive available to sufferers today. We also found the frequently prescribed flow rate of 7 liters/min was insufficient to achieve the desired abortive effect for a significant number of cluster headache sufferers and that flow rates of 12 to 15 liters/min proved far more effective. Our analysis of available data concluded that there are two mechanisms in play when using 100% medical O2 to abort cluster headache attacks. The first mechanism was obvious in that a higher partial pressure of O2 in the alveoli and bloodstream serves as a vasoconstrictor, shrinking the vascular structures surrounding the trigeminal nerve and aborting the cluster headache attack. The second mechanism, respiratory alkalosis, achieved by hyperventilating on medical O2 at flow rates between 12 and 15 liters/min, also serves as a vasoconstrictor.
Finally, our analysis of available open source data indicates that if the cluster headache sufferer has reasonably healthy cardio-pulmonary functions, there are no significant health risks or lasting side effects from breathing 100% O2 with a properly functioning non-rebreather mask at flow rates of 12 to 15 liters/min for multiple sessions up to 20 minutes each in duration provided the sessions are separated with five minute breaks breathing ambient air.
Background and Purpose

**DISCLAIMER:** The following Supplemental User’s Guide and supporting information paper on the use and risks of using 100% Oxygen (O2) to abort Cluster Headache attacks are provided for informational purposes only. Use these documents as supplements to professional medical treatment. See your doctor for a checkup and medical O2 prescription. Once the decision is made and you have the prescription for medical O2, the following User’s Guide and information paper will serve as a handy guide and reference in its use.

**Background**

Like many of our fellow Cluster Headache (CH) sufferers (CHers) and their supporters, we have found the use of 100% O2 works wonders in aborting CH attacks. It works best and is most effective if used early at the first signs, or onset of an impending attack. Although the abortive effect of 100% O2 is not as rapid when you wake up with the attack in full progress, it also works here too. It just takes longer. Unfortunately, like many of you, each of us spent several months or years and countless miserable nights dancing in the grips of “the beast” or watching a loved one suffer before convincing the family doctor and neurologists that a prescription for medical O2 may be beneficial. And, when we finally received a prescription for medical O2, all too often there was little more than the recommended dosage in the prescription on how to use it properly. Ultimately, a growing number of CHers have found that 100% O2 really works in aborting CH attacks when they finally learned how to use it correctly.

**Purpose**

The purpose of the Supplemental User’s Guide is to provide CHers with essential procedures and information developed by fellow CHers with a successful track record in using 100% O2 to abort CH attacks. The purpose of the accompanying information paper is to arm users with additional relevant information to dispel any rumors or myths associated with breathing 100% O2 and to place any risks associated with its use in a proper perspective. To
Background and Purpose

meet these goals, the authors have gathered the material used in the Supplemental User’s Guide and Information Paper from a number of credible sources and studies. Annex A includes excerpts from some of the more pointed source documents for readers who would like more detailed information.

The biggest challenge we faced in developing the supplemental user’s guide was trying to tailor the available information in an optimized format acceptable to the majority of the international community of CHers. In order to accomplish this, we have structured this document in two sections. The first section provides the basic User’s Guide with safety and user procedures in simple numbered formats. The Information Paper in Section 2 and Annex A, go into considerable detail explaining how and why we think 100% O2 works as an abortive including the rationale behind certain procedures. These sections also discuss the risks associated with breathing 100% O2. For readers in need of a quick start in the use of 100% O2, read Section 1. For all readers, the bottom line of this User’s Guide and associated information paper is summarized in the following:

You need a prescription from your doctor in order to obtain medical grade O2.

See your doctor for a checkup to be sure you are free of any medical conditions that would preclude the use of 100% O2 when you ask for a prescription.

The use of 100% O2 is effective in aborting Cluster Headache attacks for most sufferers, most of the time, if used properly.

100% O2 is more cost effective than most prescribed cluster headache abortive medications.

Repeated use of 100% O2 for 20-minute periods does not appear to pose a health risk if you have normal cardio-pulmonary (heart-lung) functions, as long as there are short breaks between O2 sessions, breathing normal air.
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Section 1 - A CDers Guide To Using Medical O2

1-1 General Procedures For Using Medical O2

The following general procedures are provided as a guide in the use of medical O2. Any directions for use or specific procedures provided with your medical O2 system and components should be followed carefully and the contents of this user’s guide used as a supplement.

1-2 Oxygen Safety

This is a “no brainer”, but it needs to be said. NO SMOKING or OPEN FLAMES in any room containing 100% O2 equipment. Smokers should also be very cautious about lighting up immediately after a session on 100% O2. Contrary to what you may have heard, oxygen is not explosive but it is an oxidizing element that supports combustion. This means that any combustible materials or flammable liquids will burn faster and at much higher temperatures in the presence of an oxygen-enriched atmosphere. That's why warning placards for oxygen mention flames, and a strict "NO SMOKING" policy. Hair and clothing that have become saturated with high concentrations of O2 during a session can be set aflame by a spark and burn explosively. Stepping out of the room where the O2 is stored or used and waiting a few minutes for high concentrations of O2 to dissipate is a good practice prior to lighting up.

Most military aircrews and aircraft maintenance technicians are familiar with the Man from LOX safety film. For the uninitiated, this aviation safety film came from a surveillance camera on the flight line. During the film, an aircraft technician was observed filling aircraft Liquid Oxygen (LOX) bottles on the flight line. While filling the bottles, he stood in front of the relief valve that was venting a white cloud of 100% O2 to stay cool in the mid day sun. After filling the last bottle, he walked to what he thought was a safe distance from the LOX farm, took out a cigarette, and lit up. His cotton coveralls were still saturated with 100% O2. The instant he lit his cigarette, a spark caused his clothing to explode in a bright flash and
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burn furiously for several seconds. He died within minutes.

O2 cylinders are filled to over 2200 psi (155 bar) and should be handled with care. A dropped bottle can turn into a missile if the valve is broken off. You will need to keep E size or portable cylinders secured in the caddy that’s delivered with the rest of the O2 supplies. CHers and their supporters should be familiar with and practice the following compressed O2 safety procedures:
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1-3 Safety Procedures For Handling Compressed O2 Cylinders

- Always check that the cylinder is clean.
- Ensure that cylinders are correctly marked for medical O2.
- Always use the correct yoke with pressure gauges.
- Ensure that “O” ring or yoke seal and valve seat are clean and undamaged. New seals should come with refilled O2 cylinders.
- Store cylinders upright and secure in a safe area.
- Adhere to applicable regulations regarding the storage and use of high-pressure oxygen cylinders if you take them out of your home.
- Store full and empty cylinders separately. Mark empty cylinders clearly.
- Ensure that you have read instructions and user’s guides supplied with your oxygen equipment prior to use.
- If you use O2 in your office at work you should place a warning label on the door to your office like the label in Figure 1.

![Figure 1 Oxygen Warning Label](image)

- DO NOT drop or roll cylinders.

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- DO NOT completely empty a cylinder – leave a little pressure in the cylinder to prevent moisture from entering.

- DO NOT expose cylinders to extreme heat or flame (oxygen is a strong oxidizing agent and when mixed with combustibles the mixture will burn rapidly and is potentially explosive).

- DO NOT smoke near oxygen equipment.

- DO NOT use petroleum-based oil or grease products on or near oxygen equipment.
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1-4 How Do I Get Medical O2?

See your doctor for a checkup and ask for a medical O2 prescription. Although statutes governing the use of medical O2 vary between countries, US State and Federal laws prohibit the sale or dispensing of medical O2 without a prescription signed by a licensed medical practitioner. Some medical supply companies will accept these prescriptions by fax. Call prior to faxing the prescription to make sure this is acceptable. Be sure to ask your doctor to indicate “8 to 15 liters/minute as needed” and refills to last for at least a month or longer when the prescription is written. The initial prescription should be for a minimum of three to four 700-liter O2 cylinders, an O2 regulator capable of flow rates up to 15 liters/minute, and a non-rebreather (NRB) mask. If your CH attacks are coming fast and furious, you may want to order a larger cylinder. Make sure to ask for a non-rebreather mask. How much O2 you will need depends on several factors that include, if you are a chronic or episodic CH sufferer, and where you are in the present cycle. Once the prescription is on file with the medical O2 supplier, they will generally accept a phone call order directly from you for refills. Discuss this with your doctor when the prescription is written and with the medical O2 supplier when you place the order to make sure they offer this service.

1-5 O2 System Component Descriptions

1-5.1 How is Medical O2 Equipment Delivered?

Most medical O2 suppliers in the US offer the 25 cu ft (708 liter) “E” and “M” size cylinders but other sizes are available. They deliver them directly to your home usually with a cart or caddy, and sealed bags containing the O2 regulator, O2 mask and tubing. It’s wise to keep a log containing the flow rate and number of sessions you are able to get from the first cylinder then calculate out how long your supply should last. As a good rule of thumb, call in for a refill when you load the last, or next to the...
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last bottle. It usually takes 24 hours from the time you make the call to get a refill delivered. Most medical O2 suppliers don’t make weekend or holiday deliveries without special arrangements or additional delivery charges, so plan accordingly.

1-5.2 What are the different types of O2 Masks?
The preferred type of O2 mask for use in aborting CH attacks is the non-rebreather (NRB) as it delivers 100% O2 without diluting it with ambient air. Non-rebreather O2 masks come in two types: constant flow and demand flow. The constant flow O2 non-rebreather masks generally cost less than $2.00 USD, and are readily available from several sources. A variation of the constant flow O2 non-rebreather mask called Clustermasx™ was designed and developed by a CH sufferer. Although this paper is not an endorsement for any particular brand, the Clustermasx™ has been in use by CHers worldwide with excellent results and positive reviews. A Clustermasx™ kit costs $25 USD. Demand flow NRB mask systems cost $250 to $350 USD or higher and are also available from several sources. Figure 2 illustrates the three types of NRB O2 masks.

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Figure 2 Constant Flow, Clustermask™, and Demand Flow NRB Masks

1-5.3 Constant Flow NRB O2 Mask

There are several NRB O2 mask manufacturers, and in the age of globalization, these inexpensive constant flow masks are manufactured and assembled all over the world. Regardless of where it was made, inspect your mask as soon as it’s delivered before you sign the receipt for the O2. Make sure that there are silicone flapper valves installed on both exhaust ports and the inhalation port. These are one-way check valves made out of silicone rubber and all must be functioning properly for you to get the full benefits of 100% O2. If one or both of the flappers are missing from the exhaust ports on either side of the constant flow non-rebreather mask, outside air will enter the mask, and you will not be receiving O2 in the highest concentration. This can easily weaken the full abortive effect of 100% O2, and you could be wasting a lot of good O2. If one or both of these flappers are missing, ask for another mask immediately before you sign the delivery receipt. Once the deliveryman has gone, it could take 24 hours or longer to get a replacement.

These valves work in sequence. When the regulator has been turned on, O2 flows through the clear tubing to the supply manifold and into the soft vinyl reservoir bag attached to the lower part of the mask. During inhalation, the exhaust valves close to prevent ambient air from entering the mask, and the inhalation valve opens to allow 100% O2 to flow from the reservoir bag into the mask. During exhalation, the inhalation valve closes to prevent exhaled air from entering the reservoir bag, and the exhaust flapper valves lift to exhaust to the exhaled flow out of the mask. The photo’s in Figure 3 illustrates the exhaust flapper valves installed on both sides of a constant flow NRB mask and the inhalation flapper valve on the supply manifold inside the mask.
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Figure 3 Constant Flow O2 NRB Mask Exhaust and Inhalation Valves

As the exhaust flapper valves are located on the outside of the NRB mask shown in Figure 3, they can be easily dislodged during use. Reattach them by stretching them over the center post on the valve seat. If one flapper valve is missing, and you have a spare mask, use one of the valves from the spare mask. If you don’t have a spare mask, cover that exhaust port with vinyl or masking tape to create an airtight seal. This type of mask will still function reasonably well with only one operable exhaust flapper valve. The NRB O2 mask shown in Figure-3 is inexpensive and should be provided along with the O2 cylinders and regulator. Be sure to ask for two NRB O2 masks when you get your prescription and when you place the order with the medical O2 supplier.

1-5.4 Clustermasx™ NRB Constant Flow O2 Delivery System

The Clustermasx™ System is still undergoing product validation for safety and efficacy, so its status as a respiratory medical appliance is still pending. Evaluation kits consist of a vinyl air cushion mask, a separate mouthpiece, a flexible mouthpiece extension, three sizes of...
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reservoir bags, and 4 meters of flexible tubing. A Clustermasx™ can be attached to any constant flow regulator capable of delivering 100% O2 at 7 to 15 liters/minute. Figure 4 illustrates the construction of a Clustermasx™ with the air cushion facemask and mouthpiece attachment shown below.

Figure 4 Clustermasx™ System

The Clustermasx™ O2 delivery system can be configured with a facemask or mouthpiece depending on the method that works best for you. The Clustermasx™ kit also comes with three different sizes of reservoir bags. Figure 5 illustrates the Clustermasx™ O2 inlet and breathing manifolds. It also illustrates the system of internal one-way check valves.
Figure 5 Clustermasx™ O2 Inlet and Breathing Manifolds

Figure 5 on the left, illustrates the wide seal area of the vinyl air cushion mask used in the Clustermasx™ system. The mask’s wide seal area combined with the low volume of the breathing manifold and internal system of one-way check valves combine to provide the user with a high concentration of O2. Although the Clustermasx™ system is still in the validation process, CHers that have had the opportunity to evaluate this system continue to give it high marks. Controlled studies of the Clustermasx™ system are planned, but the cost of conducting clinical trials like this can be very expensive process.
1-5.5 Demand Flow Non-Rebreather O2 Delivery Systems

Demand flow non-rebreather O2 systems use two stages of pressure regulation and a non-rebreather mask. These systems are almost identical in operation to the Open Circuit Downstream Demand "SCUBA" Regulators commonly used today by SCUBA divers. Fire & Rescue personnel and EMTs use Demand Flow O2 delivery systems to treat patients with impaired breathing problems or as part of emergency CPR. An integrated demand and constant flow dual mode O2 system is shown in Figure 7.

Figure 7 Dual Mode Demand and Constant Flow O2 Delivery System

The first stage pressure regulator of a demand flow O2 delivery system is mounted on the O2 cylinder valve stem similar to a constant flow regulator. The purpose of this stage is to reduce the line pressure coming from the O2 cylinder down to ambient pressure plus an additional pressure differential. This differential is adjustable on
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most systems and calibrated to flow rates from 0 to 26 liters/minute. Regulated O2 coming from the first stage regulator is delivered through a high-pressure hose to the second stage regulator attached mask. The second stage regulator is designed to provide O2 on demand by creating a small negative pressure inside the mask as the user starts to inhale. The negative pressure inside the mask shifts a diaphragm in the second stage that unseats the demand flow valve and O2 from the first stage starts flowing into the mask. O2 flow continues at the selected flow rate until the user stops inhaling and starts to exhale. At this point a slight increase in pressure above ambient seats the flow valve stopping the flow of O2 into the mask. Exhaled breath exhausts through a one-way check valve similar to that used in the constant flow O2 non-rebreather mask.

1-5.6 Proper Mask Fit

Your NRB O2 mask should fit properly regardless of the type of mask used to ensure maximum benefit of the O2 consumed. All masks should have their straps removed and be held in place with your hand during operation as a safety consideration. The Clustermasx™ and Demand Flow O2 masks use an air cushion seal or wider seal area on the face so require less pressure to achieve a gas tight seal than the less expensive constant flow O2 mask shown in Figure on the left. This mask requires slightly more attention to achieve a gas tight seal but works adequately with a good “cost/performance” ratio. The adjustable metal noseband needs to be pinched together so that the mask seals
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properly on both sides of the nose and cheeks.

1-5.7 Why is a proper fitting O2 mask so important?

The effectiveness of administering 100% O2 as a CH abortive is based on three primary factors: The flow rate of administered O2, the duration of use, and the concentration of O2 reaching the lungs and alveoli. Flow rate and duration are easy to control. Ensuring the highest concentration of O2 reaching the alveoli in the lungs requires the NRB O2 mask to have a gas tight seal and properly operating inlet and exhaust valves. The bar graphs shown in Figure 9 below, illustrate a comparison between breathing air and 100% O2 at sea level pressure (1 ATA) and at normal respiration rate. These graphs also illustrate how breathing 100% O2 increases the exchange of O2 into hemoglobin in arterial blood by displacing nitrogen that makes up 80% of air we normally breathe.

Figure 8 NRB O2 Mask

If the mask does not have a gas tight face seal or the valves are not functioning properly, the abortive effect of 100% O2 can be greatly reduced. The graphs in Figure 9 also illustrate the increase in water vapor that occurs when either air or 100% O2 reaches the lung’s alveoli. This increase in water vapor actually starts in mouth and nasal

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passages as you inhale. As 100% O2 has no water vapor, it produces a greater evaporative cooling effect than air as it passes through the upper respiratory tract. If O2 users are able to inhale at least part of the dry O2 through the nose, particularly on the same side as the CH pain, the evaporative cooling effect will lower the temperature of structures lining the nasal passages and surrounding tissues. Many CHers have reported this appears to improve the effectiveness of the O2 session by shortening the time to abort the CH attack. As most CHers also exhibit Horner’s Syndrome with watery eyes and running nose on the hit side, breathing through the nose may not be possible. Using an over-the-counter saline nasal spray at the onset of an attack may help keep the nasal passages clear on the hit side to improve the evaporative cooling effect.
1-5.8 O2 Regulators

The basic function of an O2 regulator is to reduce the pressure of the compressed O2 in the cylinder so that it can be delivered to the mask. There are two basic types of O2 regulators used to regulate the cylinder pressure down to lower pressures used by O2 masks. These are Constant Flow, and Demand Flow. Constant Flow O2 Regulators like the attached US regulator shown in Figure 10 below, are equipped with a cylinder pressure gauge, a flow rate selector valve, and flow gauge calibrated in liters/minute. Once the regulator has been properly attached to the O2 cylinder, and the O2 mask tubing attached to the low-pressure port, the cylinder supply, or D-Stem cylinder valve at the top of the tank can be opened to allow high-pressure O2 into the regulator. A knob on the back of the regulator opposite the high-pressure gauge is used to select the desired flow rate. The regulator gauges in Figure 1 indicate a cylinder pressure of 1000 psi and flow rate of 12 liters/minute.
Figure 10 US Constant Flow O2 Regulator

O2 regulators found in the UK, and Europe have a similar construction, but are usually configured with only a high-pressure gauge calibrated in BARs and a flow rate selector valve calibrated in liters/minute like the O2 regulator shown in Figure 11.
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The key point to remember when you talk to your doctor about getting medical O2 is to be sure you get an O2 regulator that is capable of delivering flow rates up to 15 liters/minute.

Figure 11 European Constant Flow O2 Regulator

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1-5.9 O2 Bubble Humidifiers

O2 bubble humidifiers come in several categories. The preferred type is the “dry” or “high flow” bubble humidifier capable of handling flow rates of 6 to 15 liters/minute. Medical O2 is dry and may cause you to cough for the first few breaths of each session. If you feel you need a humidifier, a good rule of thumb is to look for a 3 to 6 psi rating with a “Pop Off” pressure relief valve, and a diffuser that generates smaller bubbles like the model shown on the right in Figure 12. Some of the pre-filled disposable bubble humidifiers like the model shown on the left in Figure 12 may work, but it’s a good idea to ask the medical supply company, if the models they carry are capable of flow rates of 12 to 15 liters/minute. Low flow rate humidifiers are not designed for flow rates of 7 to 15 liters/minute, and their use at these flow rates could force water up the tubing into the mask.

If you fill your own bubble humidifier, be sure to use sterile distilled water. Tap water carries dissolved impurities like calcium and iron that will eventually coat

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the inside of the O2 tubing and mask. Most drug stores and chemists carry sterile distilled water. If you want to add ice cubes for “O2 on the Rocks,” use sterile distilled water to make the ice cubes.
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1-5.10 Compressed O2 Cylinders

Compressed O2 cylinders or tanks come in a variety of sizes, capacities and colours. Most are manufactured from aluminum and are rated by volume in liters with maximum pressures varying from 2015 psi /134 BAR to 2215 psi/152 BAR. O2 Cylinder paint colours and markings vary between countries as well. For example, in the US, compressed O2 cylinders are painted green or have silver bodies with green shoulders as shown in Figure 13. In the UK compressed O2 cylinders are painted pure white with “OXYGEN” stenciled in black and gray letters over the length of the cylinder. In Australia O2 cylinders are painted black with “OXYGEN” stenciled in Orange.

Figure 13 Compressed O2 Cylinders (US Paint Scheme)

Regardless of the colour code, all compressed O2 cylinders have the word “Oxygen” stenciled on them. They also carry a diamond shaped safety placard with “OXYGEN” printed across the center, the

Figure 14 O2 Placard

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compressed O2 symbol directly above, and hazardous material code number below as shown in Figure 14 on the right.

1-5.11 Integrated Backpack and Portable O2 Delivery Systems

For the more adventuresome among us that are determined not to let the CH beast rule our lives, there are backpack assemblies you can use at home and take to work. Integrated backpack O2 delivery systems, like the unit shown in Figure 15 below, provide nearly 3 hours continual use at a flow rate of 10 liters/minute. It is also configured with a “bubbler” humidifier illustrated in the center photo.
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Figure 15 Integrated O2 Backpack Carry Systems

As indicated earlier in the section on humidifiers, a bubbler adds enough moisture to the normally dry O2 to prevent dryness in the mouth and upper breathing passages. The bubbler and attached tubing should be inspected frequently and the bubbler refilled with sterile distilled water.

There are also smaller portable O2 kits with backpacks that CHers can use to gain even more mobility. The portable O2 kit shown in Figure comes equipped with three 1-liter bottles of compressed O2 charged to 3000 psi (200 BAR), a constant flow regulator, NRB mask, tubing, and the backpack. Depending on flow rate, each bottle holds 200 liters of O2 that can be used to abort from one to three CH attacks.

Figure 16 Portable O2 Kit with Backpack

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These portable O2 systems make travel by private vehicle possible for many CHers. Laws that govern carrying or using portable O2 cylinders while traveling by public ground transportation or airlines vary greatly. For example, unless you provide your portable O2 system to the air carrier 48 hours in advance of the flight for inspection and approval, travel with private O2 cylinders is prohibited even as checked baggage. Although not required by law, some air carriers may provide O2 systems for passengers at a cost, and it must be arranged days in advance of the flight. It’s always best to plan ahead by checking with carriers well in advance of travel. If you must travel by air, take your regulator, mask, and prescription for medical O2 with you and order O2 cylinders at your destination.

1-5.12 Home O2 Delivery Systems

Home O2 systems are usually associated with higher capacity cylinders like the M60 or MM sized O2 cylinders found in the US. An M60 or 60 cubic foot cylinder contains 1,699 liters of O2 and the MM cylinder contains 3,400 liters. Many of the larger home O2 cylinders are fitted with high-impact plastic or nylon protective collars. These collars provide protection for the high-pressure valve stem assembly and also a handle for easy carry. Figure illustrates two different designs used on home O2 cylinders.
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Figure 17 Home O2 Cylinders

Although they are not portable, home O2 cylinders carry the obvious advantage of higher capacities for more frequent usage. For example, an M60 cylinder with a capacity of 1,699 liters would provide 141 minutes of use at an average flow rate of 12 liters/minute. Assuming 5 minutes are required to abort a CH attack, a single M60 cylinder could abort 28 attacks. As each of us responds a little differently to O2, these times will vary with each user and severity of the CH attack.

1–6 Preparing Your Medical O2 System for Use

Carefully read and follow all attached instructions or manuals supplied with your medical O2 system. Once you’ve done this, assemble your system following these instructions. It’s best to do this between attacks as skipping a step may cause the system to malfunction or...
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create a safety hazard. We have provided the following instructions as a supplement to any instructions given to you by your health care provider.

1. Check the external condition, attached fill tag, placards, and color of the cylinder. (Colors will vary by country but green, white, and black are three of the colours most commonly used). Most medical O2 cylinders will have a placard or label indicating compressed oxygen as illustrated on the right.

2. Remove the plastic stopper or tape, and check for moisture in the outlet port and that a yoke gasket or O-ring seal is present.

3. Check the valve stem and outlet port, and wipe them with a clean dry cloth. Never use oil.

4. Slide the O2 cylinder into the caddy or cart and secure with setscrew knob or straps as appropriate.

5. Attach the valve handle.

6. Point the cylinder away from you and 'crack' the valve open for one second. This clears the outlet port of any foreign material or moisture that could foul the regulator causing it to malfunction. If the yoke gasket blows off, reinsert it in the gas port.

7. Ensure the cylinder supply valve is closed tightly.

1-6.1 Attaching the O2 Regulator

As discussed earlier, there are several different types of O2 regulators and each may attach to the O2 cylinder differently. Some regulators may even come attached to the cylinder. In all cases, follow the directions provided with the regulator and use the following as a supplemental guide as appropriate:

1. Attach the regulator yoke device or union connector to the cylinder stem. Ensure that the regulator fittings correspond to those on the O2 cylinder valve stem. If
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a gasket is used, make sure it is in place to ensure a proper seal.

2. Tighten the yoke locking screw or union ring gently with a cylinder spanner on larger cylinders, or “finger tight” on smaller cylinders.

3. Attach appropriate O2 mask tubing/hose. Non-rebreather constant flow O2 masks use clear plastic tubing with a push on nipple. Demand flow NRB O2 masks use a high-pressure hose with a threaded nipple.

1-6.2 Setting up the O2 Mask and Regulator

With the O2 cylinder installed in the cart or caddy and the regulator properly hand tightened on the on the D-stem valve:

1. Close the flow control knob by turning it clockwise.

2. Open the D-stem or supply valve on the top of the cylinder by turning the valve handle counter clockwise. The cylinder pressure gauge should read 2015 to 2215 psi (137 to 152 BAR) if full. Smaller O2 bottles may be filled to 3000 psi (200 BAR).

3. Place the mask on your face and check for proper fit by inhaling with the flow valve closed. If the mask fits properly, and the exhalation flapper valves are seated closed, the mask should remain sealed to your face without holding in place with your hand.

4. Open the flow control valve by turning the knob counter clockwise until you see the desired flow rate on the gauge or selector, and start breathing 100% O2.

5. Once you are satisfied with the flow rate and the mask is working properly, close the D-stem or supply valve by turning it clockwise.

6. Place the mask inside a zip-lock bag and hang on the caddy hook. Your system is now ready for operation. All you need do is remove the mask from the ziploc bag, place the mask on your face, and open the D-stem or supply valve.
1-6.3 What Flow Rate Should I Use?

There’s no single answer here as 100% O2 works differently on each of us. A good rule of thumb is to start at flow rate of 10 to 12 liters/minute and adjust lower or higher accordingly. Studies have shown that some CHers do not achieve an abortive effect from 100% O2 until flow rates are 12 to 15 liters/minute. Going above 15 liters/minute may not be beneficial as you may not be able to use all the O2 completely at that flow rate and could be wasting good O2 you may need later. Higher O2 flow rates will require higher respiration rates to be effective. Given the average tidal volume of air flowing in and out of the lungs with each breath is one-half liter, at a flow rate of 12 liters/minute; your respiration rate should be 24 cycles/minute.

The majority of the testimonials and studies of O2 users indicate successful O2 users achieve the greatest success in aborting a CH attack by starting the O2 treatment as soon as they notice the onset of an attack. A lower flow rate of 7 to 8 liters/minute may be sufficient at this point. If the pain continues to build or you awake in the middle of a CH attack, use the highest flow rate that has been most effective for you during previous sessions.

1-7 What Tips and Techniques Work Best When Using O2?

There are as many tips and techniques in using 100% O2 to abort CH attacks as there are successful users. Ultimately, once provided with 100% O2 along with the proper basic procedures to ensure they breathe O2 at the proper rates and in the maximum concentration, CHers who achieve success in aborting CH attacks tend to develop techniques that work best on their own. Common themes among CHers with successful O2 experiences generally include a combination of prescribed medication regimens and homeopathic aids. Effective tips and techniques include but are not limited to the following:

- Start on O2 at the first indication of an approaching CH attack.
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• Set the O2 flow rate at 10 to 12 liters/minute.

• Relax. Tension, or even worse, anxiety during a CH attack, appears to lessen the effectiveness of 100% O2. In addition, too low a flow rate or buildup of CO2 can easily trigger a sensation of claustrophobia. If this happens, increase the O2 flow rate and check the reservoir bag as you exhale. If the reservoir bag fills rapidly as you exhale, the inhalation flapper valve may not be seating properly. This allows exhaled breath to enter the reservoir bag causing a buildup of CO2. Exhaling a little faster should help seat the inhalation valve. Giving the reservoir bag a slight squeeze during inhalation makes breathing a little easier. If the reservoir bag continues to fill rapidly as you exhale, check both the inhalation and exhaust valves for proper operation.

• Hold the NRB mask to your face using one hand and place your other hand on the vinyl reservoir bag. If you’re using the standard NRB mask with external exhaust valves, you can test their operation by placing your thumb and forefinger lightly over these valves as you inhale. You should feel them seat as you inhale and lift when you exhale. Be careful not to block the operation of these valves during use. If the valves are working properly and the reservoir bag fills tightly before you take the next breath, either the flow rate is too high, or you are not breathing fast enough.

• Your respiration rate when breathing 100% O2 is just as important in aborting a CH attack as the O2 itself. At 12-15 liters/minute flow rate, your respiration rate should be 24-30 cycles/minute. This elevated respiration rate is essential in reducing the level of CO2 in the bloodstream and forcing your body into respiratory alkalosis. Section 2 provides a more detailed explanation of respiratory alkalosis and how this temporary condition helps to abort CH attacks.

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• If you’re not too congested with Horner’s, try to inhale through your nose, and relax as you exhale through your mouth. Inhaling through the nose provides the added benefit of evaporative cooling inside the nasal passages that may increase the effectiveness of O2 as an abortive.

• Some of the more adventurously CHers add Ice cubes to their bubbler for an “O2 on the Rocks.” Cooling the O2 may help make it work a little faster. Cold O2 flowing through the nose and the evaporative cooling effect of inhaling dry O2 through the nasal passages have similar effects. Both cause the capillaries lining the nasal passages to give off heat and cool. The thinking here is this cooling of the nasal passages triggers the body’s thermostat to start constricting the capillaries lining the nasal passages and possibly the capillaries in the surrounding structures near the Trigeminal nerve.

• It’s okay to use 100% O2 as an augmentation to preventative and abortive medications prescribed by your doctor. There do not appear to be any harmful interactions with commonly prescribed CH drugs as long as you’ve had a checkup and your cardio pulmonary functions are normal.

• Another effective technique is to start at 12 to 15 liters/minute and maintain this flow rate until the pain subsides. At this point, reduce the flow rate down to 7 liters/minute and stay on the O2 for a few minutes longer to guard against a rebound.

• If you are unable to abort a CH attack after 20 minutes of administering O2 at high flow rates, turn off the O2 and take a five minute break breathing normal air, then start the procedure again at 12 to 15 liters/minute.

• Heat or Ice Packs. Some users find placing an ice pack or frozen gel pack to the temple area on the affected
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side, top of the head, or to the back of the neck while using O2, takes the edge off CH pain.

1-8 How Often Should I Clean My O2 Mask?

O2 masks will start accumulating oil and other contaminants each time it’s used. The build up of dust, facial oil, or makeup can serve as a breeding ground for bacteria. This can taint the mask with bacteria and odors you might find offensive in the middle of an attack and after. It is equally important to keep the flapper valves clean and free of any foreign matter to ensure they operate properly. A good rule of thumb is to clean and test your O2 mask and system at least once a week. If you are using it several times a day, clean and check it more frequently. A good method of cleaning your O2 mask is to use a Benzalkonium Chloride antiseptic towelette. Benzalkonium chloride is a mixture of alkylbenzyl dimethylammonium chlorides. It is a rapid acting surface disinfectant and detergent that is active against both gram-negative and gram-positive bacteria, certain viruses, fungi, yeasts, and protozoa. It can be applied topically to the skin and mucous membranes and is tolerated well by most people. Benzalkonium chloride towelettes come in packets available over the counter at any drug store or chemist. If you are hypersensitive to cleaning agents, dipping a clean paper towel or tissue in a mild solution of a hypoallergenic soap will work equally well in keeping your mask clean. Once you’ve cleaned your O2 mask, place it inside a ziploc bag to keep it free of dust and lint when not in use.
Section 2 - Breathing 100% O2 as a CH Abortive

The following information paper addresses a number of topics as they pertain to the use of medical O2 as a CH attack abortive. The purpose of this paper is to provide CHers with additional information on how 100% O2 works in aborting CH attacks and the risks associated with its use.

2-1 A Chemistry Lesson

No discussion about the benefits, risks, pharmacology, or physiological effects of breathing 100% Oxygen (O2) would be complete without a short refresher in the acid-base body chemistry. Aviators and SCUBA divers need to learn about the following chemistry in order to better understand what is happening to them when they fly or dive. Accordingly, it’s not unreasonable to expect CHers using 100% O2 to understand it as well.

Absent any external chemical sources, a system of sensors in the body controls the bloodstream’s pH by adjusting the rate of respiration. Under normal conditions, these sensors work to maintain a neutral blood pH. pH is a measure of the acidity of a solution. It is equal to the negative logarithm of the concentration of hydrogen ions in a solution. A pH of 7 is neutral. Values less than 7 are acidic, and values greater than 7 are basic or alkaline. The body adjusts the pH of the bloodstream through an increase or decrease in the content of Carbon Dioxide (CO2) by changing the rate of respiration to maintain the pH between 7.35 and 7.45. The chemical equation involved here is:

\[(\text{Acid}) \ H_2CO_3 \rightarrow HCO_3^- \ (\text{Base})\]

Adding CO2 to this equation by allowing the buildup of the partial pressure of CO2 (PCO2) in the lung’s alveoli and bloodstream pushes the reaction above to the left making the solution more acidic. Reducing the PCO2 in the lung’s alveoli by breathing faster or breathing 100% O2, casts off more CO2 than the body generates. This causes a resultant decrease in the CO2 dissolved in the bloodstream and drives the equation above towards the right decreasing the acidity of the solution increasing the basivity. Any changes in the
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level of acidity of the bloodstream above or below the neutral range, are detected by the body’s respiratory control centers. In response, these respiratory control centers automatically signal an increase or decrease in respiration rate to adjust the pH back towards neutral.

You can demonstrate this principle to yourself by taking in a single breath of air and holding it. This will force a buildup of CO2 in the bloodstream. Unless you’re a trained free diver, taking in one breath and trying to hold it for 30 to 40 seconds results in an overpowering urge to exhale and inhale fresh air. Now try this demonstration again, only this time hyperventilate by breathing in and out rapidly for 10 seconds and then take in a deep breath and hold it. This should add another 10 to 20 seconds or more until that overpowering urge to exhale and inhale fresh air takes effect. Why the difference? By hyperventilating, you’ve reduced the total amount of CO2 dissolved in the bloodstream and it takes longer to build up so the body’s respiration sensors detect the trigger level and signal the dummy holding it’s breath to start breathing. The important thing to remember here is, it’s not the lack of O2 that causes the respiratory control centers to signal an increase in the respiration rate, it’s the build up of CO2 in the bloodstream.

There are two more respiration related conditions worth discussing at this point; they are Respiratory Acidosis, and Respiratory Alkalosis. The eMed web site defines respiratory acidosis as a clinical disturbance that is due to alveolar hypoventilation (not enough respiration). Production of carbon dioxide occurs rapidly as a product of normal metabolism, and failure of normal lung ventilation promptly increases the partial arterial pressure of carbon dioxide (PaCO2). Alveolar hypoventilation leads to an increased PaCO2 (i.e., hypercapnia). The increase in PaCO2 in turn decreases the HCO3⁻/PaCO2 and decreases pH. Hypercapnia and respiratory acidosis occur when impairment in ventilation occurs and the removal of CO2 by the lungs is less than the production of CO2 in the tissues.
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Similarly, eMed defines respiratory alkalosis as a clinical disturbance due to alveolar hyperventilation (too much respiration). Alveolar hyperventilation leads to a decreased PaCO2 level (hypocapnia), which results in cerebral vasoconstriction. In turn, the decrease in PaCO2 level increases the ratio of bicarbonate concentration (HCO3-) to PaCO2 and increases the pH level. Hypocapnia develops when the lungs remove more carbon dioxide than is produced in the tissues. Respiratory alkalosis can be acute or chronic. In acute respiratory alkalosis, the PaCO2 level is below the lower limit of normal and the serum level is alkalemic. In chronic respiratory alkalosis, the PaCO2 level is below the lower limit of normal, but the pH level is normal or near normal because of renal compensation (kidney functions).

You’re probably wondering what all this chemistry has to do with Cluster Headaches and breathing 100% O2 to abort them. Well, guess what? When you breath 100% O2 at a normal respiration rate for more than a few seconds, you start to deplete CO2 from the bloodstream faster than it is produced and you begin to induce respiratory alkalosis. You also elevate the level of O2 in the bloodstream and tissues. And, if you’re breathing 100% O2 at higher respiration rates between 10 to 15 liters per minute, you are driving yourself farther into respiratory alkalosis. This is not necessarily bad, because both elevated O2 in the bloodstream and respiratory alkalosis serve as vasoconstrictors.

2-2 Clinical Pharmacology of 100% O2 in Aborting CH Attacks

The exact mechanism by which 100% O2 exerts its abortive actions on a CH attack are not clear, nor is there a lot of detailed authoritative information available on this topic. The two main factors responsible appear to be a combination of an elevated level of O2 in the lungs and bloodstream, a drop in the partial pressure of Carbon Dioxide (PCO2), and a resulting condition previously discussed, respiratory alkalosis. At higher concentrations above that found in

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normal air, O2 is a vasoconstrictor. Studies have shown that respiratory alkalosis is also a cerebral vasoconstrictor. What is very clear, however, is the overwhelming body of open source information from testimonials and surveys that confirm the benefits of using 100% O2 to abort CH attacks for most sufferers most of the time.

2-3 How 100% O2 Works as a CH Abortive, Connecting the Dots

One theory on how 100% O2 works is based on hyperoxygenation or hyperoxia, the process of driving the PO2 up above normal and respiratory alkalosis, the process of driving PCO2 down through hyperventilation. Maintaining these two conditions for periods ranging from 3 to 5 minutes on the low end, and up to 20 minutes or longer on the high end maximizes the abortive effect on CH attacks. In simple terms, breathing 100% O2 results in an increased level of O2 dissolved in the bloodstream, vascular walls, and tissues around the Trigeminal nerve and pain centers elsewhere in the brain. Elevating PO2 while depressing the PCO2 by breathing 100% O2 at higher than normal respiration rates also alters the acid-base balance of the bloodstream inducing respiratory alkalosis. As it is widely held in most circles that vasodilation is one of the leading mechanisms associated with headache pain, and that elevated concentrations of O2 in the lungs and ultimately the bloodstream along with respiratory alkalosis serve as vasoconstrictors, one can start connecting the dots. In addition, if nasal congestion is not too pronounced due to Horner’s Syndrome, breathing dry O2 may also play a minor role in aborting CH attacks due to an increase in the evaporative cooling effect as the O2 passes through the nasal passages.

If we place these dots of information in a more familiar setting, albeit more painful, the basic theory starts to emerge and we’re able to connect more of the dots. Most of us are all too familiar with waking up in the middle of a CH attack. For the majority of CHers, these attacks
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unfortunately occur more frequently while sleeping or resting in a reclined position. The thinking here is the reduced respiration (breathing rate) we routinely encounter while sleeping, depresses the PO2, elevates the PCO2 and this drives the acid-base balance or pH of the blood and tissues towards the acid side of the equation. The resulting condition is called respiratory acidosis. As respiratory acidosis leads to cerebral vasodilation, increased cerebral blood flow, and intracranial pressure, this condition appears to support an increased susceptibility to CH attacks.

Another related dot some CHers may want to consider connecting involves Sleep Apnea. During sleep apnea, an individual may stop breathing for several seconds due to a collapse of the upper airway structures obstructing the flow of air to the lungs. With no respiration, the PO2 level drops precipitously as the body consumes O2 and the PCO2 level elevates even more dramatically. This causes sleep apnea sufferers to awake with a sudden convulsive gasp for air. Sleep apnea sufferers encounter this condition repeatedly throughout the night at rates of 15 episodes per hour (one every four minutes). Aside from a number of very serious health risks including heart attack, as sleep apnea pushes the sufferer into respiratory acidosis, it further increases the sufferer’s susceptibility to a CH attack. A surprising number of CHers also experience some form of sleep apnea. If you think you have sleep apnea, get to a doctor or sleep disorder specialist ASAP.

2-4 What Are the Side Effects and Risks of Using 100% O2?

There are a number of side effects and potential risks associated with breathing 100% O2. Nearly all the side effects are short in duration and reversible if users are in reasonable health and have been to a doctor for a checkup prior to starting on 100% O2. There are also a
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number of risks associated with using 100% O2 and like side effects, these risks can be minimized by being aware of them and taking prudent precautions. Some of these side effects and potential risks are interrelated. The following sections summarize these side effects and potential risks.

2-4.1 Hyperventilation and Respiratory Alkalosis

As discussed previously, the primary side effects of breathing 100% O2 at flow rates of 10 to 15 liters/minute are hyperventilation and respiratory alkalosis.

Placing this in perspective, the normal respiration rate or tidal flow of air in and out of the lungs is between 12 and 20 cycles per minute with the normal average being 16 cycles per minute. Given the total lung capacity of most adults is 5 liters, and the average tidal flow volume during normal respiration is 0.5 liters, normal respiration rates do not pose a problem of hyperventilation breathing air. However, you begin to hyperventilate on 100% O2 at this rate, and by increasing the respiration cycle rate above 16 to 20 cycles per minute you are clearly hyperventilating. The thing to remember here is air contains roughly 21% oxygen. The actual composition of dry air is 20.9% oxygen, 79.02% nitrogen, and 0.03% carbon dioxide by volume. By breathing 100% O2 even at normal respiration cycle rates, CO2 is depleted from the lungs and bloodstream far more rapidly than hyperventilating on air, so it’s easy to hyperventilate and achieve respiratory alkalosis. The attendant risk here is associated with a loss of consciousness.

The following scenario will help to illustrate how this can happen and explain the nature of this risk. You are racked with a head-splitting, high Kip Scale (levels of CH pain defined by Mr. Bob Kipple), Cluster Headache attack. You’re tired and haven’t slept well in nights. You plug into your O2 and crank the flow rate up to 12 liters/minute and start sucking it down. Luck is with you and after a while, the attack subsides. You feel so good, the next thing you
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Know, the mask is in your lap and you realize you just took a short nap... or did you?

Let's look more closely. Less than a minute into your O2 session you began to enter respiratory alkalosis. After all, upping the O2 in your system and a mild case of respiratory alkalosis is the condition we are trying to induce in order to abort an attack. Rapid breathing has caused the CO2 in your system to drop well below normal and your respiration control sensors are now frantically trying to get you to slow down the breathing. The cluster beast is still hammering away so you force yourself to keep on sucking. Now, when the pain has subsided, you’ve entered into a psychological and physiological state of exhausted euphoria so could care less about anything. At that point the respiratory control centers win the battle, and you stop breathing or breathe at a very low rate and tidal volume. Now the race is on. The level of O2 is going down and the level of CO2 is rising. If the level of O2 reaches the minimum to maintain consciousness prior to the level of CO2 rising to the trigger point to start breathing, you slip from consciousness and pass out. If you’ve used your non-rebreather (NRB) O2 mask correctly with it held in place by hand and not the straps, and loss of consciousness occurs, the mask falls away. Once the O2 mask falls away, you start breathing ambient air again and regain consciousness in a few seconds.

This is the same scenario as shallow water blackout that divers face, only you are comfortably seated in a soft armchair, a bed, or on the floor, and not under water.

2-4.2 Pulmonary Oxygen Toxicity

Under certain conditions, oxygen has a toxic effect. Navy Underwater Demolition Team divers were the first to discover oxygen toxicity during dives below 30 ft (10 meters) in depth during the development of the Emerson-Lambertsen closed-circuit oxygen rebreather in the early 1950s. The rebreather rig recycled the exhaled oxygen through a CO2 scrubber into a bladder where it is mixed with more oxygen for inhalation. This rebreather system
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was designed to eliminate bubbles rising to the surface that could give away the divers location. Oxygen toxicity was also discovered during treatment of decompression sickness in Navy divers at a depth of 165 feet or 6 atmospheres absolute (ATA) using air. It can occur at as low as 0.7-0.8 ATA (532 mmHg or 9,500 feet) breathing 100 percent O2 for prolonged periods. The type of oxygen toxicity associated with breathing oxygen at sea-level pressures and above is called the Lorrain-Smith Effect (Pulmonary Oxygen Toxicity).

Prolonged use of 100% O2 at pressures above 1 atmosphere while diving can lead to oxygen toxicity with lung damage that consists of fluid accumulation and hemorrhage into the alveoli with a resulting pneumonia-like condition. Some people are much more likely to suffer damage than others, but it has been noted that most people develop symptoms and signs in about 20 hours breathing 100 percent oxygen at sea level. The significant point to note here is the lengthy exposure time to 100% O2. You don’t need to use it for 20 hours and clearly not underwater to abort a CH attack.

2-4.3 Pulmonary Edema

Pulmonary edema is a condition medical teams guard against when treating intensive-care patients on breathing machines with 30 hours or more exposure to high concentrations of 100% O2. This condition is characterized by the following exposure times to 100% O2:

- Decreases in the rate of gas exchange across the alveoli (intensive-care patients on breathing machines at 30 hours of exposure)
- Chest pains that were worse during deep breathing (volunteers with 24 hours of exposure)
- Decrease in the total volume of exchangeable air in the lung (vital capacity) by 17 percent (volunteers with 24 hours of exposure)

Again, the significant point to note here is the exposure time to 100% O2. Sustaining exposure to these times would
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require several bottles of 100% O2 making this an unlikely problem for CHers.

2-4.4 Atelectasis

Atelectasis is defined as local areas of collapsed alveoli that have been plugged by mucus. This condition arises when breathing 100% O2 and the oxygen entrapped in the plugged alveoli is absorbed into the blood. With no nitrogen left to keep the plugged alveoli inflated, it collapses. Mucus plugs happen normally, but are easily cleared by coughing. Atelectasis does not happen when breathing air, as 80% of the air we breathe is nitrogen. As nitrogen is not readily absorbed at sea level pressure, it remains trapped in the alveoli as the O2 is absorbed, keeping the alveoli inflated. It is also interesting to note that military pilots flying high performance aircraft and breathing 100% O2, routinely encounter atelectasis during high-G force maneuvers. They clear atelectasis with a simple cough once they release the G force. Atelectasis should not pose a problem for CHers if they have normal lung functions.

2-4.5 COPD and Asthma

Although the use of medical grade O2 does not cause Chronic Obstructive Pulmonary Disease (COPD) or Asthma, these two conditions do complicate its use in aborting CH attacks. If you have COPD or asthma, be sure to talk with your doctor about using 100% O2 to abort CH attacks. The bottom line with CHers who also suffer from asthma, deals with knowing when and how to treat the asthma or the CH attack if both happen at the same time. Again, talk with your doctor to work out a protocol for O2 use that minimizes risks to COPD or asthma and maximizes the abortive effect on CH attacks.

The common side effects of breathing 100% O2 that should concern CHers who also suffer from COPD or asthma run exactly opposite the desired goal of aborting a CH attack. Ideally, the goal of O2 therapy in aborting a CH attack is to elevate the partial pressure of O2 in the lungs and bloodstream while at the same time, achieving a level of
Section 2 - Breathing 100% O2 as a CH Abortive

respiratory alkalosis. The two problems that COPD and asthma sufferers need to be aware of when breathing 100% O2 at normal respiration rates are atelectasis and a lowered respiration rate. These two temporary conditions can lead to below normal oxygenation levels. The important note here is what is your normal respiration rate? The normal or average adult respiration rate is around 16 cycles/minute. If you are using 100% O2 to abort a CH attack, you should be breathing at 20 to 24 cycles/minute, a higher respiration rate than normal.

Remember, it's not the lack of oxygen that creates the urge to breathe; it's the amount of CO2 in the blood stream. If the partial pressure of CO2 in the blood stream is above normal, the respiration rate rises above normal to expel excess CO2 and bring in more O2. If the partial pressure of CO2 in the blood stream is lower than normal, the respiration rate drops blow normal to allow the CO2 to come back to normal levels. Accordingly, atelectasis and a depressed respiration rate are the two conditions that concern doctors and respiratory therapists if you are a COPD or asthma sufferer.

Fortunately, these two conditions can be easily avoided by consciously keeping the respiration rate elevated above normal while breathing 100% O2 until the CH attack and pain starts to abate. An O2 flow rate of 10 to 12 liters/minute is a good starting point for most CHers experienced with its use. It’s easy to tell if you’re using this flow rate effectively by watching the O2 Reservoir Bag on the non-rebreather mask. If you’re breathing fast enough to keep the reservoir bag from filling completely, you’re using the O2 flow rate effectively. If the reservoir bag remains tightly inflated, you're not breathing fast enough. If the CH pain hasn't started to subside after 5 minutes at 12 liters/min, gradually increase the flow rate to 15 liters/minute. This should equate to a respiration rate of 30 cycles/minute or one respiration cycle every two seconds. Again, make sure you discuss the use of 100% O2 with your doctors if you suffer from COPD or asthma.
Section 2 - Breathing 100% O2 as a CH Abortive

2-5 Conclusions

This information paper arrives at the following conclusions regarding the use of 100% medical O2 as a CH abortive:

- The preponderance of evidence supports the practice of breathing 100% O2 at flow rates between 8 to 15 liters/minute as beneficial in aborting CH attacks.

- Breathing 100% O2 is effective in aborting CH attacks for a majority of CHers most of the time.

- The exact mechanisms by which 100% O2 exerts its abortive action on a CH is not clear, but increasing the PO2 above normal to maximize the oxygen’s vasoconstrictor effect, and achieving respiratory alkalosis through hyperventilation are key factors as they go hand in hand.

- A well fitting non-rebreather O2 mask with properly functioning inhalation and exhaust valves is essential in delivering sufficient quantities of 100% O2 at the highest concentration possible to abort CH attacks.

- A non-rebreather O2 mask that fits poorly or is missing the silicone flapper on one or both of the exhaust valves will allow ambient air to enter the mask and not deliver a sufficient concentration of O2 to abort CH attacks.

- Breathing 100% O2 continuously for one or two hours does not appear to pose a significant health risk to individuals with normal cardio-pulmonary functions.

- Cooling down the affected side with ice and chilling the O2 appear to increase the effectiveness of the O2 in aborting CH attacks. This may be due to the vasoconstriction response to chilling the skin and nasal passages.

- There are still many CHers that have not tried 100% O2 as an abortive, or who have tried it once or twice and not achieved success in aborting CH attacks due to a lack of information on how to use it effectively.
Section 2 - Breathing 100% O2 as a CH Abortive

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About the Authors

Ben Khan

Ben is a long-term chronic cluster headache sufferer. He was the first to undergo Professor Goadsby’s pioneering research using Positron Emission Tomography (PET Imaging) to determine there was cluster headache related activity in the SCN region of the hypothalamus responsible for circadian rhythms. Ben’s background includes training with DAN (the Divers Alert Network) and he’s certified in advanced oxygen use by PADI (the Professional Association of Diving Instructors). Ben’s own research into oxygen therapy and related delivery systems led him to design and develop the Clustermasx O2 delivery system. The Clustermasx is the first O2 delivery system specifically designed for cluster headache treatment. Ben and his wife of 22 years, Jane, have 2 children, Hannah 14, who unfortunately also suffers from cluster headaches, and Matthew 13, who mercifully doesn’t.

Dr. Annette Do, MD

Annette is a practicing physician living in Sydney, Australia. Married with two young boys, Annette is one physician who’s developed an up close and personal understanding of the debilitating effects of a CH attack. Her husband Daniel is a cluster headache sufferer. Daniel was going through his first CH cycle, and was having a very rough time of it until he started using 100% O2 properly. Like many CHers who tried 100% O2 once or twice without achieving relief, Daniel became frustrated and was sure O2 would not help. It wasn’t until Annette and Daniel had researched the topic extensively, talked to people experienced with the use of oxygen, and obtained a proper non-rebreather mask that Daniel finally achieved success in aborting a CH attack with O2. In a matter of a few days, Daniel became very proficient in the use of 100% O2 and he’s now confident that he can use 100% O2 to abort most of his CH attacks. Daniel and Annette's experience illustrates another important point. Being confident in using 100% O2 as the first line of defense in aborting CH attacks also reduces the anxiety many people feel about
About the Authors

Michael Berger

Michael is a Test Engineer specializing in the design and testing of high tech construction equipment. He lives in Wildhaus, near St. Gallen, Switzerland with his wife Marta and their two children, JJ and Ben. Michael is a former Israeli Naval Officer with over three years at sea, specializing in Electronic Warfare. He is a mixed gas diver with hundreds of open ocean dives so is no stranger to the use of breathing equipment and the use of O2. Michael has been a chronic cluster headache sufferer for 2 years and uses 100% O2 as a part of his regular regiment of CH pain management.

Svenn Thørn

Svenn has been a chronic cluster headache sufferer since 1986. He lives in Oslo, Norway and worked in the logistics department at Norway’s largest brewery until the attacks made work impossible. A confident user of 100% O2, Svenn is a vocal proponent of using O2 as the first line of defense in aborting CH attacks followed by other regiments of prescribed medications. Married for 27 years with two children, Svenn travels extensively all over Norway speaking to groups to raise CH awareness. He routinely makes presentations to fellow CHers, doctors and researchers working on Cluster headaches.

Pete Batcheller

Pete is a retired Naval Aviator with 24 years Naval Service and another 14 years in the defense industry as a program manager for combat information systems. He has been an episodic cluster headache sufferer since 1995 and chronic since 2005. He routinely uses 100% O2 to abort CH attacks with a great success rate. Pete and Joyce live in Lake Ridge, Virginia. As a Navy pilot, Pete accumulated over 3,000 flight hours in fighter aircraft, all of it breathing 100% O2, so he’s no stranger to the use of O2. Pete is
About the Authors

also a certified SCUBA diver with over 300 open ocean dives.
Annex A - Source Material

Annex A contains excerpts and links to relevant source material used in developing the User’s Guide and accompanying information paper.

The following section was lifted directly from Chapter 2: HIGH ALTITUDE RESPIRATORY PHYSIOLOGY, of the US Air Force School of Aerospace Medicine FLIGHT SURGEON'S GUIDE. An on-line copy of this book is available at:


This section provides for some heavy reading associated with the use of 100% O2. The U.S. Navy has a similar book on Aviation Physiology that Navy Flight Surgeons and Aviation Physiologist use as their Bible in teaching the principles of Aerospace Medicine and Aviation Physiology to Navy and Marine Corps aircrews. Although this selection appears to be pointed only at military pilots and astronauts, rest assured, they are human too. We also selected this section as it speaks very clearly to the side effects and potential risks of breathing 100 % O2.

Excerpt from Chapter 2 - High Altitude Respiratory Physiology

Acidosis. Acidosis can be produced by a number of factors. For example, acidosis will result from any disturbance causing an excessive production of acids or preventing their elimination. Acidosis resulting from excessive production of acid will be seen in uncontrolled diabetes or indulgence in diets high in organic acids. Acidosis from failure to eliminate CO2 can be caused by obstruction of air passages, pulmonary diseases (asthma, pneumonia), or cardiac disease where there is insufficient blood pumped to the lungs.

Alkalosis. The usual way in which alkalosis develops is by excessive elimination of acid since, in most cases, ingestion of high alkali or excessive alkali in the
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Annex A - Source Material

Bloodstream will be neutralized immediately and quickly eliminated. The most common cause of alkalosis is excessive CO2 elimination from the lungs. The development of this alkalosis is seen relatively frequently in naive aircrew personnel during aircraft and chamber flights. It appears that one effect of alkalosis is on the neuromuscular system. Alkalosis also seems to interfere with normal oxygen utilization by the brain cells. The symptoms of these effects on cerebral tissue are euphoria (a feeling of well-being) and eventually unconsciousness. When alkalosis occurs, the low CO2 content of the blood induces the respiratory center of the brain to inhibit breathing to retain CO2 and the cerebral blood vessels to constrict, thus inhibiting blood flow and O2 transport to the brain.

The events leading to unconsciousness from hyperventilation are as follows:

a. Increase in minute volume (inappropriate for metabolic needs) leading to:
   1. Decrease in partial pressure of alveolar CO2.
   2. Decrease in partial pressure of arterial CO2.
   3. Increased blood pH.
   4. Respiratory alkalosis.

b. Vasoconstriction of blood vessels supplying brain (opposite to the normal vasodilating effects elsewhere).

c. Pooling of the blood present in the brain at the moment.

d. Brain utilizes O2 available in the pooled blood.
   1. O2 concentration here drops.

e. Unconsciousness (due to hypoxia of cerebral tissue).

The symptoms manifested by alkalosis are neuromuscular irritability, muscular spasms, tingling and numbness of the extremities and around the mouth, and a sense of euphoria. Most people also feel short of breath. It is evident that these symptoms are somewhat similar to those of hypoxia; consequently, confusion about hypoxia in the untrained person is understandable. Cyanosis is seen in many cases of hyperventilation at altitude. This cyanosis is not a result
of hyperventilation itself, but is often associated with it because of concurrent hypoxia.

**Mechanism of Hyperventilation:** The symptoms of hyperventilation are the same as those encountered in alkalosis since hyperventilation will always result to some degree in alkalosis.

a. General. Normally, the amount of O2 and CO2 diffusing through the capillary alveolar membrane in the lungs is regulated to promote a proper balance between these gases. When there is a normal increase in respiratory rate, as during exercise, there is an increase in the O2 demand of the body and in the CO2 production by the body; therefore, proper CO2 balance is maintained. If the respiratory rate should increase without need for additional O2, then excessive CO2 is eliminated inasmuch as additional CO2 is not being produced. If this imbalance continues, alkalosis results. Examples of the causes of this alkalosis are voluntary overbreathing or, as in certain instances of anxiety and apprehension, involuntary overbreathing. Involuntary overbreathing is the most common cause of alkalosis in aircrew personnel and may result in a vicious cycle — the effects of hyperventilation will produce anxiety and anxiety in turn aggravates hyperventilation.

b. At altitude the picture is slightly different. At altitudes above 10,000 feet O2 tension in the lungs is reduced below minimum acceptable levels. This reduced O2 tension sets up a reflex impulse to the respiratory center which in turn increases respiration to increase the amount of O2 presented to the blood. With the increase O2, more CO2 is eliminated in proportion to the O2 received and the blood becomes slightly alkaline. This is normal (a slight increase in blood pH, in fact, is beneficial to O2 transport and delivery to the tissues). It becomes abnormal when continued for a long period of time, causing severe alkalosis. In untrained or apprehensive individuals, hyperventilation may occur without the presence of hypoxia. The knowledge of the dangers of altitudes, i.e., reduced O2
pressures, may incite the individual to breathe faster. The effects of hyperventilation are soon mistaken for hypoxia and the individual aggravates the condition by breathing even faster. All observers on a chamber flight must be constantly alert for any sign of abnormal respiration, and be prepared to issue corrective instructions. It should be noted that in hyperventilation, hypoxia occurs only in the brain and nowhere else since there is a reflex vasodilation of all blood vessels except those in the brain; there is no oxygen deficiency. Hence, cyanosis will only be seen when hypoxia occurs concomitantly with hyperventilation. After unconsciousness, respiration is reduced sufficiently to increase the CO2 tension and correct the alkalosis, but sufficient O2 must be present to maintain life.

Summary of Hyperventilation Signs and Symptoms.

a. Neuromuscular. The increased sensitivity and irritability of neuromuscular tissue, due to an elevation in blood pH, gives rise to a superficial tingling of the extremities (this tingling is not limited to the extremities, but is usually encountered there). The tingling usually precedes muscular spasm (i.e., a fixation of the hand wherein the fingers are drawn back toward the wrist). In severe cases facial muscles will be tetanically contracted, and the face will give an appearance of being pulled downward. The most dire and dramatic reaction is the "stiffening" of the entire body due to generalized muscular tetany. It is believed that a reduction in the partial pressure of alveolar CO2 to 24-30 mmHg is the critical level for the onset of these symptoms.

b. Psychomotor. Deterioration of muscular control and coordinated activity is invariably seen during severe hyperventilation. Performance deterioration is encountered whenever the partial pressure of alveolar CO2 is reduced below 25 mmHg. As the value falls below this level, performance deterioration becomes more marked.

Treatment of Hyperventilation
Annex A – Source Material

Voluntary reduction in the rate or depth or both of respiration of the individual affected is the most effective method of treatment, when applicable. It is conceivable, however, that an extremely apprehensive person would not respond to directions to slow respiration.

It should be noted that the symptoms of hypoxia and hyperventilation are virtually indistinguishable. The individual must treat for both simultaneously. If either occurs, a decrease in the respiratory rate and breathing 100 percent O2 will correct the condition. In the presence of hypoxia, if other disturbances coexist, or in more severe cases, it is imperative to return to ground level before more serious developments occur.

OXYGEN TOXICITY

Oxygen is vitally necessary for the flier to operate an aircraft safely and efficiently. This chapter has emphasized its importance. The indispensability of O2 to maintain life at altitude is undisputed. However, excessive amounts of O2, or excessively high O2 partial pressures can prove fatal. Death will result from too much O2, paradoxically enough, because of tissue hypoxia. The O2 partial pressures utilized by USAF aircrew are never great enough to cause harm to the body. On the other hand, prolonged 100% oxygen breathing at sea level as in denitrogenation could lead to pulmonary oxygen toxicity. The harmful manifestations of elevated partial pressures of oxygen are directly related to two factors: a. level of elevation of partial pressure, and b. duration of exposure. At altitude, because of decreasing ambient pressure, breathing even 100 percent oxygen produces alveolar O2 partial pressures, which generally do not produce damage. The type of oxygen toxicity, significant in sea-level oxygen breathing, is the Lorrain-Smith Effect (Pulmonary Oxygen Toxicity). This phenomenon, first recognized during treatment of decompression sickness in deep sea divers at a depth of 165 feet or 6 atmospheres absolute (ATA) using air, can occur at as low as 0.7–0.8 ATA (532 mmHg or 9,500 feet) breathing 100 percent O2 for long periods.
Annex A – Source Material

The lung damage which can result consists of fluid accumulation and hemorrhage into the alveoli with a resulting pneumonia-like condition which can be fatal. Some people are much more likely to suffer damage than others, but it has been noted that most people develop symptoms and signs in about 20 hours breathing 100 percent oxygen at sea level.

Chapter 2. HIGH ALTITUDE RESPIRATORY PHYSIOLOGY
Revised by Paul W. Fisher, Ph.D.
Annex A - Source Material

Pulmonary Oxygen Toxicity - Early reversible changes in human alveolar structures induced by hyperoxia

WB Davis, SI Rennard, PB Bitterman, and RG Crystal

Abstract

To study the early changes in the lower respiratory tract in persons exposed to periods of hyperoxia usually considered safe, we evaluated 14 normal subjects by bronchoalveolar lavage before and immediately after 16.7 +/- 1.1 hours of breathing more than 95 per cent oxygen. Hyperoxia caused a significant alveolar-capillary "leak" as detected by the presence of increased plasma albumin and transferrin in lavage fluid. These changes were reversible, as shown at repeat lavage in four subjects two weeks after oxygen administration. Hyperoxia for an average of 17 hours did not change the total number or type of lung inflammatory and immune effector cells recovered by lavage (P greater than 0.05, all comparisons). However, alveolar macrophages from subjects exposed to oxygen released increased amounts of fibronectin (P less than 0.05) and alveolar-macrophage--derived growth factor for fibroblasts (P less than 0.01)--mediators thought to modulate fibroblast recruitment and proliferation in the alveolar wall. Thus, although some of the effects of exposure to 17 hours of more than 95 per cent oxygen are reversible, hyperoxia for even this short period lowers the structural or functional barriers that normally prevent alveolar-capillary "leak" and induces processes that can culminate in fibrosis of the alveolar wall.

Pulmonary Oxygen Toxicity
Compiled by Ernest S Campbell, MD

Why does oxygen cause damage to the lung?
Animal studies have shown that when the lungs are exposed to high levels of oxygen that deterioration occurs progressively by steps that overlap. The first step is an acute outpouring of fluid into the tissues of the lung, filling the spaces usually full of air. Following this there is bleeding between the air sacs that changes into a gummy layer and then into tough membranes and destruction of capillary and type I alveolar epithelial cells. The fluid phase merges into a subacute phase that is characterized by production of tissues usually seen in attempts at healing, result in thickening and scarring. There are type II alveolar epithelial cells produced and partial clearing of earlier acute changes. These changes are influenced by the concentration of inspired O2, duration of exposure, and other factors such as species differences.

The lungs of human patients who die after prolonged oxygen therapy have the same damage as is seen in pulmonary oxygen toxicity in experimental animals. The clinical course of these patients, in conjunction with the known susceptibility of humans to oxygen toxicity, leaves no doubt that the observed pathologic changes are caused by pulmonary oxygen toxicity. In monkeys and presumably also in humans, recovery from pulmonary oxygen intoxication is accompanied by complete resolution of changes typical of the early fluid phase. However, when exposure to hyperoxia is sufficiently prolonged for the development of prominent scarring, recovery from these pathologic effects is greatly delayed, and chronic changes may be left in the lungs.

Symptoms of pulmonary oxygen poisoning begin slowly as a substernal irritation that becomes progressively more intense and widespread along with increased coughing. Uncontrollable coughing occurs in severe cases, symptoms originating in the trachea and major bronchi associated
with a constant burning sensation, which is worsened by inspiration. The most severe symptoms are associated with shortness of breath on exertion or even at rest. The onset of symptoms is variable among individuals but usually occurs after about 12 to 16 hours of exposure at 1.0 ata (sea level pressure), 8 to 14 hours at 1.5 ata, and 3 to 6 hours at 2.0 ata. (1.0 ata = 33 ft sea water, i.e., at a depth of 33 feet, (10 meters) the actual pressure is 2.0 ata.)

Pulmonary function changes to hyperoxic O2 exposures include:
1. decreases in inspiratory and expiratory lung volumes
2. decreases in flow rates
3. decreases in carbon monoxide diffusing capacity
4. decreases in lung compliance.

Arterial oxygenation was maintained at rest during early reversible stages of pulmonary intoxication but was detectably impaired during exercise after hyperoxic exposure. The ability to move air in and out is impaired earlier and more severely than is gas exchange function in normal humans exposed continuously to elevated oxygen pressures.

Should I be worried about oxygen treatments in a chamber?
Humans can live normally for seven days with elevated oxygen levels at about half ata, although the level of hyperoxia that can be tolerated indefinitely with no pulmonary effects cannot be identified with certainty. However, exposure for 24 hours at 0.75 ata causes pulmonary symptoms in association with a significant decrease in vital capacity, and the rate of pulmonary intoxication increases progressively at higher oxygen pressures.

Nevertheless, the majority of current applications of hyperoxia in hyperbaric oxygen therapy and diving do not cause pulmonary symptoms or functional deficits.
Hyperbaric oxygenation causes pulmonary symptoms in patients only when used very aggressively for serious conditions, such as severe decompression sickness or arterial gas embolism. Commercial divers who use intermittent hyperoxia to hasten inert gas elimination after unusually long or deep dives also frequently experience some degree of midchest discomfort. When hyperbaric oxygenation is combined with saturation exposure in the treatment of refractory decompression sickness, it is not uncommon for diving chamber attendants and the patient to experience pulmonary symptoms. In all of these situations, careful monitoring of symptoms and appropriate alternation of hyperoxic and normoxic exposure periods can avoid irreversible pulmonary intoxication.

Biochemistry of Oxygen Toxicity

Gerschman and Gilbert were the first to propose that oxygen toxicity is caused by the production of free radical intermediates in excessive concentrations during exposure to increased oxygen pressures. The initial involvement of these agents is now well established, and several excellent reviews have summarized the literature on the biochemistry of oxygen free radicals. Although exact mechanisms are not yet known, free radical intermediates including superoxide anions, hydrogen peroxide, hydroperoxy and hydroxyl radicals, and singlet oxygen are potentially toxic to cell membranes, enzymes, nucleic acids, and other cellular constituents. Along with better understanding of oxygen free radicals has come a greater awareness of the dependence of vital biologic processes on cellular antioxidant defenses such as superoxide dismutase, catalase, and the glutathione system. It is now thought that in the absence of these defenses, the same oxygen pressures required to sustain life would cause lethal oxygen poisoning.

References:
Bove, Diving Medicine, 1997
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Annex A – Source Material

Cerebral Hyperemia During Spontaneous Cluster Headaches With Excessive Cerebral Vasoconstriction to Hyperoxia

The Journal of Head and Face Pain
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Abstract:

Values for local cerebral blood flow (LCBF) were measured in three dimensions utilizing xenon enhanced computerized tomography among patients during spontaneously occurring cluster headaches, during headache-free intervals and immediately after terminating attacks by inhalation of 100% oxygen. Results were compared with values measured among age-matched normal volunteers. LCBF values measured in five cluster patients while headache-free did not differ from similar measures among age-matched normal volunteers. In three patients during attacks of spontaneously occurring cluster headache, LCBF values for temporal cortex, basal ganglia and subcortical white matter were increased. Immediately after terminating attacks of cluster by 100% oxygen inhalation for five minutes, LCBF values for temporal cortex and basal ganglia became significantly decreased below normal values in five patients with spontaneously occurring cluster headache. Prompt relief of head pain by inhalation of 100% oxygen is associated with
Abolition of the hyperperfusion of both cortical and subcortical brain structures that occurs during spontaneously occurring cluster headaches and is followed by excessive cerebrovascular constriction. It remains to be determined whether the cerebral hyperemia occurring during cluster headaches is causally related to the head pain or is secondary to the pain itself. Rapid termination of head pain by hyperoxia associated with excessive cerebral vasoconstriction suggests that this vascular phenomenon is unique to cluster headache and offers clues to its pathogenesis.
Annex A – Source Material

Placing the Contents of these References in Perspective

Before you do something silly like ignore or read more into the information presented in the Supplemental User’s Guide, Information Paper or the supporting references, and as a result, avoid or stop using 100% O2 as a CH abortive, read them again. Pay close attention to the sections on Hyperventilation and Alkalosis as well as the paragraphs on Pulmonary Oxygen Toxicity with a critical eye. Understand that the sections on alkalosis, hyperventilation, and pulmonary oxygen toxicity covered in Chapter 2 of the Flight Surgeon’s Guide were written with military aviators flying high performance tactical aircraft at high altitudes in mind, not from the perspective of a CH sufferer using 100% O2 to abort an attack at altitudes much closer to sea level. Although the risk is remote, passing out due to respiratory alkalosis brought on by hyperventilating on 100% O2 for a military aviator is significant if he or she is flying the aircraft. This risk is significantly lower for CHers sitting at home.

Using 100% O2 while comfortably seated in a soft armchair or couch at home to abort CH attacks is different. Yes, a CH sufferer is just as likely to induce respiratory alkalosis by hyperventilating on 100% O2. The first difference is simple. A mild case of respiratory alkalosis is the condition the CHer is trying to induce in order to abort an attack. The second difference is equally simple. If the CH sufferer is using a non-rebreather O2 mask correctly held in place by hand without straps, and a loss of consciousness occurs, the mask falls away. Once the O2 mask falls away, the CH sufferer starts breathing ambient air and regains consciousness in a few seconds.

The last paragraph in Chapter 2 add important numbers that should stick in your mind: “20 hours breathing 100% oxygen at sea level to “note” the symptoms of Pulmonary Oxygen Toxicity”. If you take it at face value, it would appear the risk of encountering Pulmonary Oxygen Toxicity while using 100% O2 as a CH abortive is very low for people with normal cardio-pulmonary (heart-lung) functions. And, if you have normal cardio-pulmonary functions, breathing 100% O2...
at sea level for as long as it takes to achieve relief from a CH hit would appear to be far less dangerous that smoking cigarettes, or driving to work. Moreover, you can’t order enough 25 cubic foot (708 Liter) O2 “E size” cylinders to sustain 20 hours of continuous 100% O2 at the flow rates needed. Depending on the flow rate, that equates to 35 to 40 “E size” cylinders of O2. Finally, what is also very remarkable about this chapter, is the absence of any other lasting maladies associated with prolonged use of 100% O2.

Again, in case you missed it, please read the following disclaimer.

**DISCLAIMER:** The following Supplemental User’s Guide and supporting information paper on the use and risks of using 100% Oxygen (O2) to abort Cluster Headache attacks are provided for informational purposes only. Use these documents as supplements to professional medical treatment. See your doctor for a checkup and medical O2 prescription. Once the decision is made and you have the prescription for medical O2, the following User’s Guide and information paper will serve as a handy guide and reference in its use.
Annex B - Glossary of Acronyms and Terms

The following partial list of acronyms and terms are provided to help the reader to better understand the contents of the User’s Guide and Information Paper.

Acid – In chemistry, an acid is most commonly thought of as a substance that can donate protons. Vinegar is an acid.

Alveoli – Anatomical structures that have the form of hollow cavities. In the lung, the pulmonary alveoli are spherical outcroppings of the respiratory bronchioles and are the primary sites of gas exchange with the blood. Alveoli are peculiar to mammalian lungs.

Atelectasis – The medical term defined as a state in which the lung, in whole or in part, is collapsed or without air. It is a condition where the alveoli are deflated. Atelectasis can be induced through the elimination of Nitrogen from the lungs by breathing 100% O2.

ATA – Shorthand for 1 atmosphere of pressure.

ATM – Shorthand for 1 atmosphere of pressure expressed in pounds per square inch.

BAR – A commonly used shorthand notation for a unit of pressure that is about the same as one atmosphere pressure. One BAR equals 14.5 pounds/square inch of pressure.

Base – In chemistry, a base is most commonly thought of as a substance that can accept protons. Ammonium Hydroxide is a base.

Cerebral Vasodilation – The dilation of blood vessels in the brain.

CH – The acronym for Cluster Headache.

CHer – The acronym for Cluster Headache Sufferer.

CO2 – The chemical shorthand for the Carbon Dioxide molecule.

Horner’s Syndrome – A central nervous system condition often associated with cluster headaches. Symptoms include: sinking of the eyeball into the face, small (constricted) pupil, drooping eyelid (ptosis), and lack of facial sweating. Additional symptoms of a cluster headache that...
Annex B - Glossary of Acronyms and Terms

may accompany Horner’s Syndrome include tearing and running nose on the affected side

**Hypercapnia** - A medical condition where there is too much carbon dioxide (CO2) in the blood. Carbon dioxide is a gaseous product of the body's metabolism and is normally expelled through the lungs. Hypercapnia is generally caused by hypoventilation (reduced respiration), or diminished consciousness. It may also be caused by exposure to environments containing abnormally high concentrations of carbon dioxide, or by rebreathing exhaled carbon dioxide.

**Hyperoxia** – In medicine, hyperoxia is a condition where excess oxygen exist in the bloodstream and body tissues, Hyperoxia is caused by breathing gas at pressures greater than normal atmospheric pressure or by breathing oxygen-rich gases at normal atmospheric pressure for a prolonged period of time.

**Hyperventilation** – In medicine, hyperventilation (or overbreathing) is the state of breathing faster and/or deeper than necessary, thereby reducing the carbon dioxide concentration of the blood below normal. Prolonged hyperventilation causes various symptoms such as numbness or tingling in the hands, feet and lips, lightheadedness, dizziness, headache, chest pain, slurred speech and sometimes fainting.

**Hypoventilation** – In medicine, hypoventilation (also known as "respiratory depression") occurs when ventilation is inadequate (hypo means "below") to perform needed gas exchange. It generally causes an increased concentration of carbon dioxide (hypercapnia) and respiratory acidosis. It can be caused by medical conditions, by holding one's breath, or by drugs. Hypoventilation may be dangerous for those with sleep apnea.

**Ion** – In chemistry and physics, an ion is an atom or group of atoms that normally are electrically neutral and achieve their status as an ion by loss or addition of one or more electrons. The simplest ions are the proton (a hydrogen ion, H+, positive charge), and alpha particle (helium ion, He2+, consisting of two protons and two neutrons).
Annex B - Glossary of Acronyms and Terms

N2 - The chemical shorthand for the Nitrogen molecule
NRB Mask - The Acronym for NonReBreather Mask
O2 - The chemical shorthand for the Oxygen molecule
PaO2 - The chemical shorthand for the partial pressure of alveolar O2
PaCO2 - The chemical shorthand for the partial pressure of alveolar CO2
PCO2 - The chemical shorthand for the partial pressure of CO2
pH - The chemical shorthand for the negative logarithm of the hydrogen ion concentration of in a solution
PO2 - The chemical shorthand for the partial pressure of O2

Respiratory Acidosis - A temporary condition that results from an increase in acidity or acidosis (abnormal acidity of the blood) due to decreased ventilation of the pulmonary alveoli, leading to elevated arterial carbon dioxide concentration (PaCO2).

Respiratory Alkalosis - A temporary condition that results from a decrease in acidity due to increased alveolar respiration (hyperventilation) leading to decreased plasma carbon dioxide concentration. This leads to decreased hydrogen ion and bicarbonate concentrations. This can occur when a person moves from sea level to high altitudes or hyperventilates.

Sleep Apnea - A sleep disorder characterized by pauses in breathing during sleep. These episodes, called apneas (literally, "without breath"), each last long enough so one or more breaths are missed, and occur repeatedly throughout sleep. There are two distinct forms of sleep apnea: Central and Obstructive. Breathing is interrupted by the lack of effort in Central Sleep Apnea, but from a physical block to airflow despite effort in Obstructive Sleep Apnea. In Mixed Sleep Apnea, both types of events occur. Regardless of type, the individual affected with sleep apnea is rarely (if ever) aware of having difficulty breathing, even upon awakening. Sleep apnea is recognized as a problem by others...
witnessing the individual during episodes, or is suspected because of its effects on the body (sequelae).

**Vasoconstrictor** - A vasoconstrictor, also vasopressor or simply pressor, is any substance that acts to cause vasoconstriction (narrowing of the lining of blood vessels) and usually results in an increase of the blood pressure. Oxygen and Imitrex (sumatriptan succinate) are vasoconstrictors. Vasoconstriction also occurs in superficial blood vessels of warm-blooded animals when their ambient environment is cold; this process diverts the flow of heated blood to the center core organs and brain of the animal, preventing the loss of heat.

**Vasodilator** - A vasodilator is a substance that causes vasodilation (widening of an artery or vein caused by a relaxation of the smooth muscle in the vessel wall). Several vasodilators are used as drugs to allow blood to flow more easily and reduce blood pressure. Flushing may be a physiological response to vasodilators.