



## OHIO POLIO NETWORK NEWSLETTER WINTER ISSUE 2005

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### OPINION ABOUT OXYGEN USE by Lee Seitz (Polio Reporter, San Fernando PPSG, CA 3/2003)

We have printed articles regarding this subject in the past, but we feel it is important to renew it every once in awhile. Many people have gotten in trouble with respiratory problems related to having had polio. Many physicians deal with non-paralytic respiratory problems and think that the same fix works in both settings. If your doctor is proposing oxygen as a solution, please make sure she/he knows about the special needs of someone with a paralytic condition.

Breathing is a two-part process. 1) Your lungs must provide Oxygen to the body, and 2) they must remove carbon dioxide from the blood. There are normally two ways for a person to "adapt"; you can take larger breaths and/or you can take more frequent breaths. Unfortunately, both of these options can be difficult for someone with a neuromuscular disease. Increasing breath size may be difficult due to scoliosis and/or weakened diaphragm or intercostal muscles. Any increasing breathing rate leads to fatigue of the muscles (and a larger oxygen demand - all muscle work will increase oxygen demand.)

If you address this problem with respiratory support (BiPap, positive or negative pressure, respirator, etc.) you get both increased oxygen uptake and increased carbon dioxide clearance. If you choose to do it by oxygen enrichment alone, you may solve the oxygen problem but not the carbon dioxide clearance problem, leading to a buildup of carbon dioxide in the blood. Normally, your body has a feed back loop which helps you adjust to changes in exercise, and hence oxygen demand. More exercise=more demand=more carbon dioxide. This can lead to respiratory acidosis. The body detects the change in pH and says "breathe faster" until the pH goes back up.

There is a second feedback loop that looks at oxygen level. If you have lived a fairly long time with chronic underventilation, your body can ignore the pH detector and go on oxygen alone. This seems to be a learned behavior to suppress flair craving. If you learn to ignore the pH side of the equation and you add supplemental oxygen, the body detects "enough" oxygen and actually slows down breathing demand. This leads to even further carbon dioxide problems. You feel better because you feel like your need to fight for air has decreased. (and you are already "ignoring" the Ph sensor.)

An additional complication to using supplemental oxygen is that it's an "aggressive" chemical. At high concentrations, it actually attacks the lung tissue and can lead to a pneumonia-like condition. Oxygen is also very dry, compared to normal air, so you have problems with drying of tissues. (this is why they use the "bubblers" on hospital oxygen lines.) The dried out tissues are much more susceptible to infection and problems with clearing mucus.

When IS oxygen ever appropriate? 1) If your lung area is diminished (pneumonia, emphysema, etc.) 2) If you have problems moving oxygen to the tissues, such as a lack of hemoglobin (from anemia or carbon dioxide poisoning, for example). The chronic use of oxygen is a slippery slope. Once you start down it, you can often get in REAL trouble. Most commonly, ours is not a problem with oxygen uptake, it's underventilation, solved by the use of ventilatory support. You would be wise to be careful about just opting for the quick fix.

Oxygen looks like it presents, consult a doctor who really knows how to deal with respiratory insufficiency due to neuromuscular disease [and specifically Polio].



**Clue to why some die during sleep**  
Courtesy of BBC UK Online

Sleep apnoea usually jolts people awake. Scientists believe they may have solved the mystery of why some people stop breathing fatally in their sleep. They say a cumulative loss of cells in the area of the brain that controls breathing is to blame - triggering a condition called central sleep apnoea. However, they believe many such deaths in elderly people are misdiagnosed as heart failure. The study, by the University of California, Los Angeles, is published in Nature Neuroscience.



**SLEEP APNOEA**

**Central sleep apnoea:** Triggered by problems with the brain's breathing centre

**Obstructive sleep apnoea:** Breathing stops when the airway collapses

The researchers had previously pinpointed a region of the brainstem they dubbed the preBöttinger complex (preBötC) as the command post for generating breathing in mammals. They had also identified a small group of cells within this area as being responsible for issuing the commands.

**Cells killed**

In the latest study, they injected rats with a compound to kill more than half of these cells - and then monitored the animals' breathing patterns.

When the animals entered the rapid eye movement phase of sleep - when dreaming occurs - they stopped breathing completely, and were jolted into consciousness in order to start again.

Over time, the breathing lapses increased in severity, spreading to other phases of sleep, and eventually occurring when the animals were awake as well.

Rats possess 600 of the specialised cells. The researchers believe humans have a few thousand, which are slowly lost over a lifetime. Lead researcher Professor Jack Feldman said: "We speculate that our brains can compensate for up to a 60% loss of preBötC cells, but the cumulative deficit of these brain cells eventually disrupts our breathing during sleep.

"There's no biological reason for the body to maintain these cells beyond the average lifespan, and so they do not replenish as we age. "As we lose them, we grow more prone to central sleep apnoea." The UCLA team believes that central sleep apnoea may pose a particular risk to elderly people, whose heart and lungs are already weaker due to age. They also suspect the condition strikes people suffering the late stages of neurodegenerative disorders, such as Parkinson's disease.

And Post-Polio

These people often have breathing difficulties during sleep, and the researchers believe their bodies eventually reach a point where they are unable to rouse themselves from sleep when they stop breathing.

The UCLA team plans to analyse the brains of people who die from neurodegenerative diseases to determine whether these patients show damage in their preBöttinger complexes.

Frank Govan, of the UK Sleep Apnoea Trust, told the BBC News website that previous work had linked cot death to obstructive sleep apnoea - caused by collapse of the airways.

However, he said science had failed to prove the link. He said: "These chaps may well be right, that the link is between central sleep apnoea - rather than obstructive sleep apnoea - and cot death, and unexplained adult death."

I'M ON THE  
PACIFIER PATCH.

