



## WHY HAS CENTRAL SLEEP APNEA SUDDENLY BECOME SO COMPLEX?

by Steven Grenard RRT, RPSGT

There are patients who cannot breathe due to a variety of reasons, most of which are related to neuromuscular function that we call “apneic” or “centrally apneic.” This condition can occur in people who are awake, as well as asleep. If these patients were in the right place at the right time (e.g. in the hospital or in the presence of someone willing and trained to give them mouth to mouth or rescue breathing) they might be lucky enough to end up on a mechanical ventilator until the underlying problem was treated and resolved. With the advent of sleep medicine technology and treatment it wasn’t long before a “new” kind of central apnea was discovered in some sleeping patients. And as most sleep techs know, it is often seen after some people are placed on PAP therapy to prevent obstructive sleep apnea.

An important property of central sleep apnea is that it spontaneously starts and ends within 10 to 30 seconds, whereas non-sleep central apnea can persist until brain and cardiac death intervene. In sleep apnea of either the obstructive or central type, a cortical arousal seems to jolt the patient into taking a breath, or maybe even a few breaths, enough to keep them alive before it occurs again. Although disconcerting, it is often ignored as a problem. It doesn’t happen often, or in all patients at all times, but it does happen. Usually, these central apneas are not present during the diagnostic portion of a sleep study but start to occur after either CPAP or BPAP is initiated. And if the sleep technologist raises either expiratory or inspiratory pressures in response to continuing obstructive apneas, hypopneas or snoring, both the frequency and length of the central events seem to increase. If there was ever a dilemma or catch-22 in titrating patients, it is in those who respond to the titration with central events. The sleep tech is charged by protocol to eliminate obstructive events, apneas, hypopneas and snoring, but by so doing, they sometimes actually increase the severity of this “side-effect”.

**If there was ever a catch-22 in titrating patients, it is seen in those who respond to the titration with central events**

Three years ago, Gilmartin et al in *Current Opinion in Pulmonary Medicine* (11:485-93) used the term “complex” sleep apnea to lump central sleep apneas and other CPAP and non-CPAP related problems into one broad category. I don’t remember when I first saw this term, but I began to notice it thanks to a few PAP equipment manufacturers who started to use the term in conjunction with modified PAP machines that were intended to deal with the problem. In one recent grid of U.S. CPAP/BPAP machines, there are 23 different machines ranging from the most basic to those with special features of one kind or another, many of which target “complex” sleep apnea.

In the *October* issue of the *AASM Journal of Clinical Sleep Medicine* (4(5): 403-408), Dr. Peter C. Gay of the Mayo Clinic and Drs. Malhotra, Bertisch and Wellman of Brigham and Woman’s Hospital and the Harvard Medical School argue the definition of “complex sleep” apnea in a pro-con debate. Dr. Gay takes the position that complex sleep apnea is a disease, whereas Dr. Malhotra et al feel it is not a disease, but rather “a vaguely defined group of enti-

ONE CALL.  
(SAVE TIME)

ONE BOX.  
(SAVE THE EARTH)

One shipment.  
(Save money)

**FAITH MEDICAL, INC**  
FOR ALL YOUR  
NEUROLOGY AND SLEEP SUPPLIES  
CALL (800) 600-1390

CIRCLE READER ACTION CARD # 22

ties with varying etiologies." This debate is definitely worth reading. It will help to put the controversy of complex sleep apnea into perspective. For central events that show up in non-PAP patients while asleep, and for which there are no identifiable extrinsic causes, CSA may be a symptom of a disease. Numerous neuromuscular or related "disorders" come to mind.

However, if these types of events do not occur except *after* a patient is placed on PAP, it seems more than merely intuitive that there is something about the CPAP or BPAP therapy that is responsible. And, if you notice the complex or central sleep apneas becoming more frequent and prolonged as PAP pressure is increased, then this further confirms the relationship.

Assuming, then, that there is a highly variable cause and effect relationship between PAP and the occurrence of complex sleep apneas, Malhotra et al point out a number of hypotheses as to why. There are clear and predictable changes in the CO2 dynamic which can occur with the relief of chronic upper airway obstruction. Increased CO2 elimination yields a lower PACO2 and PaCO2. This level may fall below the so-called "CO2 apnea threshold" which can then result in apnea. Marked declines in the PaCO2 occur by increased tidal volumes as well as dilution or washout of the CO2 in the anatomical dead space. CPAP can produce both of these effects.

Malhotra et al also refer to the activation of the lung stretch receptors. This is also a likely cause of the complex or central apneas seen in sleeping patients on PAP therapy. In 1868 Josef Breuer and Ewald Hering discovered that distention of the lungs of anesthetized (sleeping) animals decreased the frequency of the inspiratory effort, in effect causing a transient apneic event. This was subsequently named the Hering-Breuer (H-B) reflex. Pioneering physiologists thought the H-B reflex played an important role in establishing the rate and depth of breathing in humans. But soon researchers discovered that while this was true for animals, it did not seem to be the case in adult humans at rest.

Observers did note that the H-B reflex was activated in infants with very small tidal volumes who were suddenly ventilated with larger volumes and in adults receiving tidal volumes over 1 Liter. Of course none of these early human subjects were on CPAP or BPAP at the time. So are the complex or central events merely extended post expiratory pauses in people who get larger than normal tidal volumes? This is an effect seen when one sighs or takes a deep breath. The length of the pause serves to define and distinguish it from a central apnea. By definition it is apnea if it lasts 10 seconds or more.


What's more, an increase in pulmonary stretch receptor activity leads to inhibition of cardiac vagal neurons resulting in an elevation of heart rate or sinus tachycardia. This is a normal occurrence in healthy individuals and is known as a sinus arrhythmia. It is interesting to note that application of PAP therapy in a previously bradycardic patient causes their heart rate to reach non-bradycardic or normal levels...in other words, to speed up. Recently in our lab, a patient's bradycardia progressively resolved and his heart rate increased, in fact normalized, almost in direct proportion to his upward PAP titration pressures. Could resolution of bradycardia during sleep be all about pressure? PAP increases intrathoracic pressure and slows venous return, decreasing cardiac stroke volume and therefore lowering cardiac output. To maintain the cardiac output with smaller stroke volumes the heart will speed up. This cause and effect can be found embodied in Starling's Law.

**Ambulatory Monitoring's**  
**Highly Validated**  
**Micro Mini-Motionlogger**  
(Documented to Have the Highest Sleep Specificity of Any Actigraph)  
**Now Contains a**  
**Time-of-Day Feature**



**MICRO MOTIONLOGGER**  
**SLEEP WATCH®**

- Real Time Display
- 192K Memory
- Off-Wrist Detection
- Multi-Channel Collection
- Event Marking
- Water Resistant
- Status Feedback
- 1-4 Month Runtime



**800.341.0066**  
[info@ambulatory-monitoring.com](http://info@ambulatory-monitoring.com)  
[www.ambulatory-monitoring.com](http://www.ambulatory-monitoring.com)  
 731 Saw Mill River Rd. Wadley, NY 10982-0006

**CIRCLE READER ACTION CARD # 24**

**Equipment Review...** *Continued from previous page*

situations, when talking about ET tube stabilization. Using tape is often messy and bothersome. The Stabilock™ provides a built-in bite block/tube channel which this evaluator believes to be a distinct advantage, especially in patients who are not well controlled and who are biting down on the just-inserted ET tube. Even when the device does absorb some moisture, retains its shape and integrity, a major advantage over tape. In the trial period, the stability of the Stabilock™ was very apparent and the main concerns, accidental extubation or tube misplacement (slipping into the right mainstem, for example), are much less likely. Additionally, the biocompatibility of the device with the patient's skin, the ease of moving the tube for oral hygiene, the low cost, patient comfort make this an ideal device for securing the patient's airway.

The evaluation of the Dale® 240 Blue™ was similar to the Stabilock™ but was used on two adult patients due to availability of adult tracheotomized patients. The 240 Blue™ performed as expected and the same remarks and performance were noted and expressed by nurses and respiratory therapists. The trial period remained at 72 hours although we did leave the holder on the patients since neither the patients nor the therapists & nurses wanted them removed! This time, however, the patients were able to let their voices be heard, literally, on the comfort of the 240 Blue™. Their preference for this device was very high mainly because of the softness and comfort provided by the neckband. The therapists and nurses said that they preferred working with the 240 Blue™ mainly because of its simple design. There were no problems with disconnection from the tracheostomy tube, skin irritation or excoriation.

The evaluation of the 241 PediStars™ and 242 Pediducks™ Tracheostomy Tube Holders was similar to that done with the adult patients. We did, however, use all three of these devices on the three patients we found in the pediatric unit. The only difference between these two models is the print on them: ducks vs. stars. I'm partial to ducks myself but stars were also popular with therapists, nurses and parents. These pattern designs are a smart idea since it is, as the Dale literature states, "warm and comforting to patient and caregiver." These models come with a similar "Clinical Advantages" table talking about the features, benefits and clinical advantages of these tracheostomy tube holders. The same 72 hour evaluation period was used and, yes, we again left these holders on the patients at the request of the nurses, therapists and parents. The same question was asked here as in the adult unit: "Can we please have more of these Dale PediStars™ and 242 Pediducks™ Tracheostomy Tube Holders?". The same recommendation was given: talk to your department supervisors and administrators! I again urge the same of the reader. Beyond objective testing, the highest recommendation a medical device can receive, is the positive accolades of the users. When the patient also appreciates the device, it becomes a "must-have" in my opinion. Our evaluation showed both.


Those interested in Dale Medical Products can learn more by contacting the company at 800-343-3980 or by visiting them online at [www.dalemed.com](http://www.dalemed.com).

*Joseph Sorbello is an Associate Professor and Chairman, Department of Respiratory Therapy Education, at SUNY Upstate Medical University.*



# WINTERIZE



your pulmonary lab with the




## Turboaire Challenger

Portable arctic air for bronchial provocation and Exercise Induced Asthma studies: all year 'round

- Economical operation
- Adult and pediatric adaptors
- Uses standard PFT equipment
- Exercise or isocapnic hyperventilation
- Instant generation of cold dry air at -20°C
- No electricity, chemicals or liquids required



**Equilibrated Bio Systems, Inc.**1-800-327-9490

22 Lawrence Avenue - Suite LL2 • Smithtown • NY • 11787  
Phone 631-863-3500 • Fax 501-421-6575

CIRCLE READER ACTION CARD # 33

**Central Sleep Apnea..** *Continued from page 36*

Malhotra et al also present a brief trouble shooting table listing a number of PAP and non-PAP related issues or problems and recommended solutions. Under "treatment-emergent central apnea" the solution is listed as "wait until it goes away." And even if it doesn't go away by the end of the titration period. Patients who are re-tested on PAP after several weeks to months frequently do show resolution of the problem. For now this seems to be the most often recommended solution. Another would be to make sure the patient is not over-titrated, solved by reducing CPAP levels (but not so low that obstructive events return). And since snoring is the last thing one titrates upward for, one can dial back the pressure if complex sleep apnea occurs. Malhotra et al also conclude: "If we were to limit the definition of complex apnea to treatment-emergent central apneas, the bulk of the evidence would suggest that this 'disease' is transient and inconsequential. The use of expensive new generation devices is currently unproven in such cases." Dr. Gay presents a slightly different set of concluding remarks. On the subject of newer modifications of PAP machines, he rhetorically asks "Does every CompSA patient need a more expensive and complicated PAP device?" Clearly the answer is no, but failure to understand and explore further questions in this regard does a disservice to our patients and can create obstacles to a better understanding of CompSA as a unique disease process.

*Steve Grenard RRT, RPSGT is a research coordinator at the Sleep Apnea Center at Staten Island Univ Hospital in NY; [Steve\\_Grenard@SIUH.edu](mailto:Steve_Grenard@SIUH.edu)*