



# Adult Acute Care Bulletin

Sept./Oct. '01

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## Notes from the Editor: *The Death of the Hypoxic Drive Theory*

by Jeff Whitnack, RRT

Last March, I gave a talk at the 22nd Annual Tahoe "Odyssey" Conference, a meeting for respiratory therapists, nurses, and physicians. The topic was, "The Death of the Hypoxic Drive Theory." By "hypoxic drive theory" I am referring to either the default assumption that any chronically compensated respiratory acidosis implies reliance on the hypoxic drive to maintain adequate gas exchange, or that chronically compensated respiratory acidosis means the central chemoreceptors are defunct or deficient.

This is more than just a theory. It has become a clinical mindset; almost a medical urban legend. "He's a retainer," "that's where he lives," "he's in the 50/50 club" — we all know the clinical buzzwords.

A hypoxic drive does exist. It normally accounts for about 10-15% of the total drive to breathe. We all have it, unless perhaps we've had bilateral carotid surgery. It becomes obliterated at a PaO<sub>2</sub> above about 170 and becomes a greater stimulus as the PaO<sub>2</sub> drops below 70, and especially below 50. There is a hyperoxia associated hypercarbia, which can develop in certain patients while they are in crisis. But it has little, if anything, to do with respiratory drive.

When COPD patients are in acute respiratory failure, they are usually breathing somewhere near their maximum limit. When 100% O<sub>2</sub> is applied, the CO<sub>2</sub> can be driven up by three factors: the Haldane Effect, the release of hypoxic pulmonary vasoconstriction, and the removal of life-threatening hypoxemia in the setting of acute respiratory failure.

### The Haldane Effect

Unsaturated hemoglobin carries CO<sub>2</sub>. A patient in crisis may arrive in the ER with an SpO<sub>2</sub> on room air of 75%. The unmeasured mixed venous saturation may in turn be much lower than the 75% norm as well. All this unsaturated hemoglobin is then carrying an extra CO<sub>2</sub> load. This is in the setting where the patient has an already elevated PaCO<sub>2</sub>, perhaps an elevated Hgb after years of hypoxemia, and is "topped off" on his ability to ventilate. So, for every rise in his SpO<sub>2</sub>, we are driving more CO<sub>2</sub> into the plasma. If this were you or I, we would simply ventilate this extra CO<sub>2</sub> out via the lungs. But his lungs can't and don't; therefore, the CO<sub>2</sub> shows up in the "downstream" ABG.

### The release of hypoxic pulmonary vasoconstriction

Imagine the worst-ventilated alveoli. The local CO<sub>2</sub> pressure may be 100 or more. On room air the local O<sub>2</sub> pressure will surely be less than 60 torr. At this level of local hypoxemia, the adjacent pulmonary vasculature will constrict. Blood will then be sent to the alveoli, which is ventilating more effectively. Ventilation/perfusion matching is enhanced. But if 100% O<sub>2</sub> is given, the O<sub>2</sub> pressure will not drop below 60, the pulmonary vasculature will not constrict, and V/Q matching will not be optimized. Just as giving Nipride may drop the PaO<sub>2</sub> as hypoxic pulmonary vasoconstriction is released, so too giving 100% O<sub>2</sub> may raise the PaCO<sub>2</sub>. This can also happen to patients in an asthmatic crisis who are given 100% O<sub>2</sub>. It's not that we knock out a hypoxic drive so much as we drive in a hypercarbic potential, then further compromise ventilation through increased V/Q mismatching.

### Central Deficit or Central Wisdom?

A small number of the CO<sub>2</sub> retainers who are in acute failure, and who have their PaCO<sub>2</sub> increased further from the two mechanisms listed above, will further reduce their minute ventilation by about 15-20%. Usually, the PaO<sub>2</sub> will have been about 40 on room air, and the PaCO<sub>2</sub> 70. Given 100% O<sub>2</sub> the PaO<sub>2</sub> rises well above the 170 range whereby all hypoxic drive is obliterated, and the PaCO<sub>2</sub> rises to 90 or more.

But is this a result of a central drive deficiency? Or of central wisdom? When the PaO<sub>2</sub> is 40 the patient can't let his PaCO<sub>2</sub> go up to 90. If he did, the PaO<sub>2</sub> would plummet to about 20 and rapid death would ensue (per the alveolar air equation). But when the hypoxic drive "gun to the head" is removed, the patient then titrates his respiratory effort such that the ventilatory effort and work is proportioned out for the long haul. It is not a drive deficiency. We may view this as patient permissive hypercapnea, may apply non-invasive ventilation, may simply realize that hypoxemia kills and hypercapnea does not, or may intubate. Or hypoxemia may be used as a respiratory stimulant. But if this is the tactic cho-

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sen, it should be viewed as akin to giving epinephrine to an already compromised myocardium in order to maintain adequate perfusion pressure. Just as if we were to see this same patient arrive in stable condition later on for a clinic visit we wouldn't insist he needed an epi drip to maintain a sufficient cardiac output, so too should we not insist that a CO<sub>2</sub> retainer not in crisis needs hypoxemia in order to stimulate adequate respiratory drive.

### Debunking the myths

The May 1998 issue of *Clinical Pulmonary Medicine* featured an article titled "Acute Respiratory Failure in Chronic Obstructive Pulmonary Disease" by Schiavi. In it the author concludes that, "The traditional idea that oxygen induces hypoventilation by suppressing hypoxic ventilatory drive at the level of peripheral chemoreceptors is no longer tenable."

During my talk I read almost all of an editorial that appeared in the September 1997 issue of *Critical Care Medicine*, "Debunking Myths of Chronic Obstructive Lung Disease," by Dr. John Hoyt. I will repeat the pertinent excerpts here:

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*There are examples of mythology that float about in the atmosphere of medical information that desperately need to be debunked because they influence the care of patients. One sample of medical mythology is the commonly told story that the administration of oxygen to a patient with chronic obstructive lung disease will shut down the patient's hypoxic respiratory drive and lead to apnea, cardiorespiratory arrest, and the subsequent death of the patient.*

*It is not clear where this fallacious information comes from, but it seems to enter the medical information database at an early age, almost like a computer virus corrupting the appropriate function of the equipment. In addition, this myth becomes very difficult to extinguish during the career of the physician, even with clear factual information of long standing. The danger here is that this medical mythology will inappropriately influence treatment decisions in patients.*

*The basic issue in this story is oxygen. The human body, particularly key organs such as the heart and brain, are not all that forgiving of insufficient supplies of oxygen. Thus, medical decisionmaking — based on the mythology that oxygen causes apnea and cardiorespiratory arrest in patients with chronic obstructive pulmonary disease by turning off the oxygen respiratory drive — might take the path of withholding or delivering inadequate doses of oxygen to meet the metabolic needs of the patient in respiratory failure. This mistake is generally fatal for the patient, and a treatment tragedy for the misinformed physician . . .*

*Most mythological stories are based on some observation, which may be a correct observation, but an incorrect interpretation of the facts. It is true that the administration of oxygen to a patient with an exacerbated chronic obstructive lung disease and acute respiratory failure may lead to an increased CO<sub>2</sub>. It is true that the hypercarbia may become severe and be associated with cardiorespiratory arrest. The problem is with interpreting the cause of the events . . .*

*One should not fear apnea and cardiorespiratory arrest when giving oxygen to a patient with an exacerbated chronic obstructive lung disease and respiratory failure. Instead, one should be prepared to help the patient eliminate CO<sub>2</sub> when deadspace increases. Providing assistance with the elimination of CO<sub>2</sub> has been around since the beginning of critical care medicine. It is called mechanical ventilation.*

Focusing on one of the real causes of oxygen-induced hypercarbia, enhanced V/Q mismatch, may also allow us to recognize that a rising CO<sub>2</sub> level in a patient with status asthmaticus (on 100% O<sub>2</sub>) may not be so much an indication of advancing respiratory failure but, rather, of a worsening V/Q mismatch arising from the release of regional hypoxic pulmonary vasoconstriction.

Furthermore, it may not be so benign to have a COPD patient — even a real CO<sub>2</sub> retainer — chronically hovering the boundary of an acceptable PaO<sub>2</sub> or SpO<sub>2</sub> value. There is growing evidence that the pathogenesis of Cor Pulmonale, nutritional status (lack of weight gain despite adequate nutritional consumption), cardiac modulation, post-operative wound healing, and recovery from acute respiratory distress are all adversely affected by the default acceptance or goal of an SpO<sub>2</sub> of 88-90% in these patients.

Below is a list of references on this topic. I would most enthusiastically recommend in particular reading the studies and discussion occurring in references 11 and 17, both from the *European Journal of Respiratory Disease*.

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## Notes from the Chair

by Nick Widder, RRT

The AARC International Congress is quickly approaching (December 1-4), and we are still looking for nominations for our section’s Specialty Practitioner of the Year. This important award, which will be presented during the Awards Ceremony at the Congress, must go to a

member of the section. Please note my new contact information, which appears on page two, and be sure to send any faxes or letters nominating deserving individuals to my attention as soon as possible. You can also visit the AARC web site at [http://www.aarc.org/sections/adult-acute\\_sec-](http://www.aarc.org/sections/adult-acute_sec-)

[tion/mpoty/poll\\_form.html](http://www.aarc.org/sections/adult-acute_sec-tion/mpoty/poll_form.html) to fill out the form online. Anyone interested in serving on a committee to review the nominations should contact me as well. ■

## Considerations for Involving Respiratory Therapists in Conscious Sedation

by George Gaebler, MS Ed, RRT, director of respiratory care and cardiovascular service line administrator, University Hospital, Syracuse, NY

Respiratory care departments at several hospitals across the country have become involved in providing conscious sedation and conscious sedation monitoring for patients under their care. It is important for anyone who may be considering providing these services to distinguish between the monitoring of conscious sedation and the provision of conscious sedation. In states with licensure or certification laws on the books, you will want to check with the Licensure Board to determine the extent to which respiratory care personnel can provide conscious sedation. In states without such laws, you need to consult with the state health department, which probably regulates the provision of drug therapies such as conscious sedation.

In New York State, we have a licensure law that provides fairly clear delineation as to the role that can be performed by the respiratory therapist. There is clear indication that respiratory therapists are able to provide conscious sedation drugs to patients under their care for respiratory-related diseases. An example would be a patient undergoing bronchoscopy when the respiratory therapist is involved in the procedure. It is probable that most states would allow RTs to be involved in protecting the airway of conscious sedation patients, as protecting the airway is fair-

ly common practice for RTs, without considering the fact that it may be for conscious sedation purposes.

One reason why respiratory therapists are being asked in many hospitals to consider provision and monitoring activities for conscious sedation is the shortage of registered nurses to perform these activities. However, at least in Central New York, there is currently a shortage of respiratory therapists as well, which makes the substitution of RTs for RNs not as easy as it was even a year ago. Any department considering the monitoring or provision of conscious sedation would want to consult with members of the anesthesia team, perioperative team, and probably the pain management team. These groups would be available to provide input to protocols for conscious sedation which probably are already in place at your institution.

### Basic considerations for conscious sedation monitoring

The respiratory therapist needs to be free to monitor the patient on a regular basis, preferably every 15 minutes or so to assure that the patient’s airway is intact and protected during sedation. Obviously, patients who are on mechanical ven-

tilation do not fit the definition for conscious sedation because, in reality, their airway is already protected.

The person providing conscious sedation monitoring should be in addition to the individual — usually an RN or physician — who is actually providing the sedation medication.

### Basic considerations for provision of conscious sedation

Develop a list of approved medications for conscious sedation through a task force made up of anesthesia, perioperative, nursing, and pain management folks, along with the respiratory therapy department from your hospital.

Respiratory therapy staff will probably need training, education, and credentialing in the insertion, maintenance, and management of IVs, and the provision of analgesics. As indicated earlier, they will also need to be available to the patient for provision of this therapy.

It must be noted that any protocol that is developed should include distinguishing characteristics that describe the levels of sedation

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beyond what is considered conscious sedation. The American Society for Anesthesia has solid definitions in this area and will provide most of the guidance on what constitutes conscious sedation and what constitutes deep sedation, which really requires anesthesia in attendance.

Some of the specific considerations that fall into this area are services where patients are

being sedated fairly deeply for cardioversions, electrophysiology procedures, and other cardiology procedures. You may find that this is a gray area for individuals within your hospital and that people outside the anesthesia department are providing this deep sedation without proper credentialing. This can create problems with regulating agencies, such as the Joint Commission on the Accreditation of Healthcare Organizations.

One of the best outcomes from the provision of conscious sedation and conscious sedation

monitoring by the respiratory therapy department is the firm establishment of respiratory therapists as team providers in other high tech areas. RTs are recognized for their skills in the management of the patient beyond the typical duties associated with the field.

For more information about the provision of conscious sedation and conscious sedation monitoring by RTs, contact George via e-mail at [gabelberg@upstate.edu](mailto:gabelberg@upstate.edu) or phone at (315) 464-4490. ■

## CCM-L Discussion Part Four: A Day in the Life . . .

*Editor's Note: In our last edition of this discussion, physicians from around the globe weighed in on the use of respiratory therapists on the health care team. This installment continues that discussion, then segues into a day in the life of an RT.*

**French MD #1:** Is it possible to have some precision on the role of the respiratory therapist in ventilator regulation, physical respiratory activity, drugs, or any other things about the treatment?

**USA Surgeon #2:** Let it be fully understood that my attack on cookbooks and the QM process had NOTHING to do with respiratory therapists. It had to do with the process of practice guidelines. I am totally supportive of all of the members of the health team, the names of which are different in many different locations. You have heard NO respiratory therapy bashing from this quarter.

**USA Clinician (unknown specialty):** These strange beasts give neb treatments to all comers. They set up the O<sub>2</sub> when needed, deliver loving CPT, intubate in a pinch, monitor the vent and adjust the settings, do ABGs and CBGs. We are privileged to have a blood gas lab in the unit. We get stat gasses and lytes within 3 minutes. They can snog and declug and suction and assist with bronchs. In codes you want three of them. In the ER they are an integral player 24x7x52. For transport they are half of the team with a trained PICU nurse. We are trying to get 4 or 5 of them in our cost center so that we never have to share them again. Like the man said, don't knock it till you try it.

**Netherlands MD:** We do not have RTs here and there is no intention to introduce them. I, myself, think that mechanical ventilation is a doctor's issue and is in need of dedicated doctors who want to put effort into their patients and not leave all the parts to consultants, if you know what I mean . . . where is the prospective evidence of the extra benefit from consultants in the ICU who take over part of the treatment? Just because the system is the way it is in the US doesn't mean it is the right system. I do not need a respiratory therapist because I am 100% available to the ICU if on duty (not on call) because I am at the ward all the time. I just do not see what the benefit will be in my situation.

**USA Surgeon #1:** This is not a matter of consultants; and I would echo earlier statements that difference does not necessarily imply that something is better or worse. I would further submit that I have had the privilege and pleasure

of working with respiratory therapists and, contrary to your sentiments, I believe that their presence ENHANCES my ability to provide care and provides a very meaningful division of labor in regard to clinical practice.

Just as there are many modes of ventilatory support, antibiotic therapy, nutritional supplementation, and antiarrhythmic therapy, we need to accept this diversity in the process of care and organizational issues and reckon that there are many ways that services can be staffed and executed. Perhaps if I trained or worked in another environment, I'd share your opinion and view; my corollary to your sentiment is that I initially find it difficult to fathom how a unit could survive without respiratory therapy. But then again, your system works, your results are stellar, we all read the same literature, and hence, the obvious conclusion is that it survives because it just does, just as my system “just does.”

I would hope, however, that variability “liberates us” to become intellectually expansive and forces us to think about potentially beneficial and equally efficacious alternatives rather than becoming insular and perhaps even oblivious to other means to similar ends.

Isn't this part of the fun of it all?

**New Zealand MD #2:** To all potentially offended respiratory therapists (and supporters): There have been a number of comments in this discussion from people in countries that do not have respiratory therapists as part of their health care system. In these places, the jobs done by respiratory therapists are part of the normal work of nurses, physiotherapists, and doctors. These health systems have developed to provide excellent care in a different way – i.e., without respiratory therapists. No one (I think) has criticized the value of respiratory therapists to the health care systems that do use them.

Please do not take offense in a discussion which (appropriately) considers whether the overall systems are better or worse than one another; this is not intended to denigrate respiratory therapy where it is used.

**USA RT #3:** In answer to the question by (United Kingdom MD #1), “. . . but what do these people do all day?”

What do I do all day? Gee, since you asked — sleep. I work the night shift in a community hospital. I ride my bike ten miles to work each way. While I climb the last hill to get there I feel a certain sense of triumph, as a few years back some consultant-prodded fools actually thought they could develop a model without us. As elsewhere, the consultants came, people were laid off, others hurriedly retrained, and in short order we were back in force as outcomes quickly plummeted. I shower, hang my bike duds out to dry, and get report from the outgoing RT. After

11 p.m. I am the sole respiratory therapist in the hospital. I start at 6:30 p.m. and work 12-hour shifts. I start usually by joining the ICU RN report to get a better idea of what's going on with the patients. What follows is a collage of some of the many and varied clinical activities I am engaged in. (Everything is true and has happened over the last few years.) In a way, I'm like a macrophage — I've got my routine and mundane things, then I get activated. I go about my “routine duties” — scheduled things like Q4 HHN tx's, ventilator checks, equipment calibration. But often, when the night is over, it's those unexpected things that go bump in the night which allow me to add the most value . . .

I get a call to ER. A severe asthmatic is arriving code 3. I administer nebulizer treatments, check peak flows, do some asthma teaching as indicated during neb tx's, and do various things to aid in sputum clearance as needed (positioning, segmental breathing, auto-genic drainage). Should an asthmatic need to be intubated I know how best to ventilate that patient without further causing hyperinflation (and have had to administer some heated tongue lashings to other clinicians whom sought to normalize ABGs). In the ER I also go to all codes, maintain the airway (I have intubated in other institutions), and do some dynamite chest compressions. (The RNs always have me do them when things are to be optimized.)

Once a patient came in who had aspirated a huge vitamin E pill. The MD wanted a neb tx and perhaps some CPT. In just ten seconds of segmental breathing (I could feel the pill in the RML) he coughed out the pill. They were planning to do a bronch.

I get a call to the psyche unit. A patient has been ordered on MDI albuterol. I check out his technique. “It's like smoking a joint,” he says. I agree, and turn over the MDI to the patient for self-administration from then on (with the RN to oversee).

I get a call to do a sputum induction from a patient who “has a dry cough” and has RLL pneumonia. I just turn the patient on the left side or prone, do some segmental breathing to the RLL, and get a good spontaneous sample.

I get a call to see a patient. He is in CHF. His SpO<sub>2</sub> is 88% on 2l/m and he is SOB. The RN is hesitant to turn up the O<sub>2</sub> as the patient also has COPD and she is worried that too much O<sub>2</sub> will knock out the drive to breathe. With froth coming out of my mouth I explain, gesticulate, and draw little pictures of Hgb and the Haldane effect, plus alveoli/circulation examples of the release of hypoxic pulmonary vasoconstriction. I

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assure the RN that it is indicated to increase the O<sub>2</sub>. I wonder if I need to have my rabies vaccine boosted. I recommend that we give the patient HHN Atrovent to relieve CHF SOB as he is being tuned up.

I get an urgent call to give a scheduled Q4/Q2 p.m. tx early. The patient sounds wheezy and a little wet. I look back and see that the patient has been getting a lot of tx's and the pattern doesn't fit anything pulmonary in nature. I go out and inquire as to EKGs, fluid status, etc. The flow-sheets are pulled out, I&Os looked at. Gee, the patient is 7 liters up post-op. The MD is called and an order is gotten for Lasix.

I get a call back to ICU. The other RT (it's not 11 p.m. yet) was in ER with a young patient with severe LLL pneumonia. He is on a NRB, RR 40, SpO<sub>2</sub> 79%. Upon arrival to ICU he is intubated and placed on a ventilator. Oxygenation is still in the toilet with 7 Peep and 100% O<sub>2</sub>. We only have one ventilator with Pressure Control; it was put on by accident by the company. But I learned it on my own. The MD, unfamiliar with PCV, lets me run the show. We change out the vent and I use the Open Lung Approach. By morning he is on 50% with Inspiratory Pressures of 25 and VTs 900. At midnight he looked like he wasn't going to make it. By morning his ICU monitor signs all look like ones I'd be happy to have, save the temp of 38 degrees.

I get a call to the Level 2 Nursery. A baby is 32 weeks delivered and on a ventilator. I run the ventilator per MD order (in attendance) and help manage the airway. I have passed my Perinatal/Pediatric Specialist Exam and am ready for these episodes.

I get called to the ICU. A patient in the late stages of ARDS and on a ventilator has suddenly developed very high pressures. The SpO<sub>2</sub> and BP are dropping. I start to bag him. A CXR was earlier taken and an obvious pneumo is present. The BP and SpO<sub>2</sub> drop to truly dangerous levels as I'm bagging the patient, and I call out for someone to bring me an 18 gauge needle quick. Luckily, just then the ER MD arrives and a real

chest tube is quickly inserted. I have a long talk with him later about my responsibility and obligation should he have been delayed any longer.

Later that night at 2 a.m. I am doing a scheduled Q4 nebulizer tx to a post op open heart surgery patient. I remember her from the ICU. She had been sluggish post op and had been congested. But now she is on room air with SpO<sub>2</sub> of 96%, has clear breath sounds, has a strong effective dry cough, and is ambulating. I leave a note in the Progress Notes section of the chart to recommend making the tx's p.m.

Now bear in mind that the only MD in house after hours is the ER physician. He/she is pretty much bound to the ER and will only see floor patients with either a call from the patient's MD or a special plea from the RN (usually the ICU or RN supervisor). So I get a frantic call to come see a floor patient. When the patient was last checked all was fine. But now, on the same 2l/m that used to give him an SpO<sub>2</sub> of 95%, he is 84%, RR 38 and labored, diaphoretic, no pee, temp is up, and he is confused and removing his O<sub>2</sub>. We need to call the MD after gathering some quick data. But the suits got rid of the charge RN and it's like little tribes of RN teams. So I do an ABG, we do an EKG, and I stay with the patient to keep his O<sub>2</sub> on. The ABG is okay on “100%” non rebreather mask, the EKG looks okay, and after some sedation the patient looks better (but still not back to where he was). The MD is called with the information. In the morning the MD comes in and ships the patient out to ICU.

The next night when I arrive to work the patient is intubated and in the cardiac cath lab. The MD remarks, “Only when I put the swan in could I see he had had an MI.” I discuss how it would have been nice if I could have been given the clinical responsibility to do after hours echocardiography with modem hookup. He agrees, but things return to “normal.” Later I get a very thankful note from the RN. When discussing it with her she remarks, “I don't know what I would have done without you.” (It would be nice now to hear that with an Aussie or Dutch accent.) I inquire as to why she didn't call the RN supervisor for help. She thinks a bit, then says,

“We get told to just ‘handle it yourselves’ so much we've stopped trying.”

I finish up the shift, tx's are done, ventilators are checked, patients weaned and extubated per post-op protocols. During break I talk with an ICU RN from Australia. She knew Dr. Tuxen and we talk about how she used to do ventilator management. I get the impression that the training there was indeed far more than just cookbook ABGs. But I also get the impression that there was a cost for such training by a deficit in other areas of clinical knowledge and expertise needed in USA ICUs.

I gather information for the oncoming shift. I ride home. The hills I raced to work on are now a focus for a slow meditative cadence. I check my email and see some rude comments from foreign shores in regards to my profession. Some people think they get along fine without us. Perhaps they do. Or perhaps it's more parallel to how my nine year old son grimaces and thinks he gets along fine without sex. He does. But not for long.

Everything I do — in ER, ICU, the Nursery, the floor wards, etc. — could all be done by other health care professionals PROVIDED they were trained on a consistent and national basis, AND provided that their respective professions took responsibility for not allowing things to stagnate.

But we're already here, a real bargain for what we do, and still ticking.

The respiratory therapy profession sort of culled out parts of MD (intubation, ventilator management, inserting arterial lines, treatment and weaning protocols), part of RN (medication delivery and 24 hour patient care with a similar focus, ventilator management), physical therapy (CPT and bronchial drainage techniques), and the lab (ABGs).

Any more questions?

*The preceding discussion was reprinted with permission from the Critical Care Medicine Listserv. In order to protect the privacy of those involved, all names have been replaced with more generalized identifications. ■*

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## Positively 4th Street

by Jeff Whitnack RRT

Did you read the article in this issue on the death of the hypoxic drive theory? If so, take a minute to enjoy the following parody, sung to the tune of Bob Dylan's “Positively 4th Street.”

You had a lot of nerve  
To turn up his O<sub>2</sub>  
Just because he was dying

And really turning blue.

I don't know the reason  
why you won't accept the show  
When it comes to the oxygen drug  
You just have to “Say No.”

You had a lot of nerve, to turn up his O<sub>2</sub>

Just because he was distressed and turning blue

I don't know the reason you worry about his hypoxic distress  
If he were hiking Everest, it would be even less. ■

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