

## **The language of dyspnea or shortness of breath (SOB) With an explanation of how dynamic inflation causes SOB**

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It would almost seem a given that with a few exceptions we all describe symptoms of shortness of breath using many of the same words. Cardiac patients commonly describe their chest pain as “pressure,” “tightness,” “dull” or perhaps crushing weight. Cardiologists are very good at using these descriptions to help them with a specific diagnosis. Unfortunately, when it comes to describing dyspnea things become a bit more nebulous. Over the past 10 years or so physicians from around the world have been studying something they call “the language of dyspnea.”

There are literally hundreds of causes of dyspnea. As just one example, over **200** different causes of Interstitial Lung Disease (ILD) have been identified. Patients with ILD suffer from shortness of breath as a daily part of their lives. There are cardiac causes, blood disordered causes, traumatic causes, neuromuscular causes, and even obesity can cause an increase in dyspnea. The list is very, very long, and for the patient it can be an extremely uncomfortable and even downright frightening experience. It is for this reason that the physician taking a medical history from a pulmonary patient should begin to *speak* the same language as they’re patients are *speaking*.

Having said all that, most non-acute cases of shortness of breath can be treated fairly quickly once the cause has been determined. This leads directly into the discussion of the *language* of dyspnea. Patients (it turns out) describe their shortness of breath in a variety of ways.

### **Descriptors of Dyspnea**

I feel that my breathing is rapid.  
My breath does not go out all the way.  
My breath does not go in all the way.  
My breathing is shallow.  
My breathing requires effort.  
My breathing requires more work.  
I feel that I am smothering.  
I feel a hunger for more air.  
I feel out of breath.  
I cannot get enough air.  
My chest feels tight.  
My chest feels constricted.  
My breathing is heavy.  
I feel that I am breathing more.

A patient of mine suggested that it might be very useful to be even more specific with the list of descriptors and tell your doctor under which conditions you might feel *your breathing is shallow*, or when you feel *your breathing requires more work* etc. I’m guessing that if you are reading this article you have used one or more of the above phrases when talking to your pulmonologist or

family doctor. Going back a bit in history, it was the brilliant Julius Comroe MD who first began the study of dyspnea back in the mid-1960's. He described 6 possible causes that might explain the sensation of dyspnea that he was hearing from pulmonary patients. In the early 80's the list was shortened to 4 symptoms representing the basic sensations of breathing. They were tightness, excessive ventilation, excessive frequency, and breathing difficulty. By the year 2000, studies were being done to see if different ethnic groups used different words or phrases to describe their dyspnea. Comparing well matched groups of white and African American patients, Metachloine was given to induce bronchoconstriction to the same degree in all patients. African Americans more commonly used the term "tight throat", compared to whites who most commonly used the descriptors related to "deep breath." It shouldn't really surprise us that there are ethnic and racial differences that affect the language of dyspnea. Further studies proved that the major pulmonary disease groups do use different descriptors to describe their dyspnea. Asthmatics report differently than patients with COPD, as well as ILD patients. Add in the racial and ethnic variations and you can see that what used to be a rather simple couple of questions asked during a routine medical history, has become even more important as the "language" develops.

Adding to the confusion over the language of dyspnea is the fact that the pulmonary community has done a stellar job in making our patients aware of their oxygen saturation levels due to the ubiquitous use of digital pulse oximeters. In fact, this may (IMHO) have turned into a bit of a double edged sword. The reason I say this is we have concentrated now for so long on the *all important* saturation reading that many, many patients believe that whenever they get SOB, it is most likely due to a low oxygen blood level. When they check their oximeter, they find that their saturations are either near normal or not so low it can explain their degree of dyspnea.

Certainly low oxygen saturations can make any of us SOB. I take our respiratory therapy students up to the top of Mt. Evans (14,000 foot peak about an hour outside of Denver. Paved road all the way to the top.) every year to induce SOB in young healthy students. It's the only way I can make them short of breath at rest. At least they stand in a patient's shoes for an hour or so. If it isn't a low saturation that is making you SOB, maybe it's the other gas involved in ventilation. I call it the "Insidious Gas," and that of course is carbon dioxide or CO<sub>2</sub>. Let's pass the baton to my friend and colleague Mark Mangus, for his beautifully written description of the physiologic effects of carbon dioxide and how it can lead to a phenomenon called "Dynamic Hyperinflation."

**Shortness of breath (SOB) from high arterial blood carbon dioxide (CO<sub>2</sub>) levels:  
The role of 'Dynamic Hyperinflation'**

So, you're exercising, or doing chores around the house that require quite a bit of exertion. And here it comes! You're breathing hard and then, harder and harder. Your respiratory rate is increasing and yet you feel like it's harder and harder to get enough air into your lungs. So, you work yet harder. You check your oxygen saturation and see that it is falling, despite your increased efforts to get more air into your lungs. You have your oxygen flow up higher and think it 'should' be enough to help you breathe better. But, it's just not helping.

The thought that comes to mind for most folks in this situation is: “My oxygen level in my blood is dropping.” Yet, even turning the oxygen up more doesn’t seem to be helping. You wonder what is going on! You resort to using pursed-lips breathing (PLB) which seems to help some. But, you’re already so SOB, it is only of just so much help – AND is NOT enough. You think back to other times when, knowing that you will fall into that cycle of more breathing work and worsening SOB, you start out your activity using PLB. It seems to stave off the SOB, but that SOB STILL takes hold at some point and literally brings you to your knees. And you’re left to wonder what the heck is this all about?!

The answer is that a complicated set of mechanisms is at play that together, present as several conflicting considerations when you look at what is “physically” happening. The signals sent to your brain are: “I can’t get enough air ‘in’.” Yet the reality is that you already have TOO MUCH air in your lungs. You see that your oxygen saturation is low and think: “My blood oxygen level must be too low.” Yet, if you were to have a sample of your blood tested, you’d see that, while your oxygen level is not in the ‘text-book’ normal range, it is plenty high enough that your saturation ‘shouldn’t’ be as low as you see that it is. THAT would surely make you scratch your head wondering why? How can that be?

What is going on is the phenomenon we call “dynamic hyperinflation”. Looking at those words to try to understand what they mean – how they apply – we see that “dynamic” means moving, in motion, the opposite of ‘static’, or ‘motionless’. What is ‘dynamic’ here is our breathing ‘mechanics’ AND their effect on how air gets into our lungs AND how “much” air gets into our lungs.

“Hyperinflation” suggests that our lungs are ‘over-inflated’. Well, that is no surprise! Having advanced COPD, we understand that our lungs have too much air in them. So, what is so different about ‘this’ kind of over-inflation? The answer is that it results from breathing faster and faster AND taking in more air with each breath than we can breathe ‘out’ from breath to breath. So, our lungs go from our usual state of hyperinflation to an even higher state of hyperinflation as our breathing becomes more and more disturbed.

Think of a balloon that has been blown up too many times. It has lost its elasticity to the point that it can’t spring back to its smaller size – as it was when it came out of the package. It represents your lungs with emphysema. Now, think of blowing up that balloon and with each breath you blow into it, you let out only part of the air you blew into it. Soon, it becomes inflated to as much air as it can hold. Now imagine that balloon is your lungs during “dynamic hyperinflation”. They reach a point that trying to breathe in more air becomes impossible because they are already so full – to the maximum! Yet, your brain is telling you that you NEED to get more air in! Now you see the “physical” part of dynamic hyperinflation. But, what is causing the drop in your oxygen saturation is NOT the lack of oxygen. It is the fact that with so much air in your lungs, CO<sub>2</sub> increases because not enough ‘dilution’ of that excess air can occur to allow CO<sub>2</sub> to remain near your ‘usual’ or normal levels.

Now, understanding that oxygen saturation is not based ‘only’ upon what the oxygen level is in the blood, but a combination of the oxygen level AND the blood pH (acidity or alkalinity) AND CO<sub>2</sub>, if we look at all three of those levels in your blood we would see that oxygen is still

relatively high, BUT, the pH is severely decreased (acidic) AND the CO<sub>2</sub> is severely INCREASED! THAT is why the saturation has dropped! That is what the effect of dynamic hyperinflation does to your oxygenation. Even though your oxygen level itself may be still fairly high, you are still 'hypoxic' because of the extreme derangement of your pH and CO<sub>2</sub> – the result of your disturbed “ventilation”. Dynamic hyperinflation is the product of abnormal ‘ventilation’, not so much because of abnormal ‘oxygen’ availability or levels. There are other factors at play that contribute to the resulting disturbance in your blood gases. But, they are beyond the scope of this explanation.

So, what are you to do about this problem? Well, Pursed Lip Breathing (PLB) is a good start. At least, it will slow down the onset and severity of dynamic hyperinflation. Depending upon how well you can pace BOTH your breathing AND your activity will determine how effectively you can control and combat dynamic hyperinflation. Even then, you may find that you reach a point that you simply can't overcome the discomfort of the process, in which case you will simply have to stop and allow yourself to recover. As you rest, your demand for more breathing decreases and dynamic hyperinflation subsides. Your lungs ‘deflate’ back towards your ‘resting volume’ – that is, the amount of hyperinflation you have that is totally due to the physical changes in your lungs from COPD and in relation to the mechanics of your breathing at your resting, or ‘baseline’ level. So, remember, the important benefit of PLB in combating dynamic hyperinflation is that it slows down your overall breathing. The slower you breathe, the less you tend to over-inflate your lungs. PLB also facilitates splinting your airways open and especially, slowing your exhalation so you can empty more air out of your lungs from breath to breath.

Hopefully, you can now appreciate the utter complexity of the simple phrase “short of breath.” Maybe we should turn the telescope around and have the physicians ask their questions based on the known descriptors used by pulmonary patients. They might read like this:

1. Is your breathing shallow?
2. Do you feel the urge to breathe more?
3. Is your chest constricted?
4. Does your breathing require effort?
5. Do you hunger for more air?
6. Do you feel out of breath?
7. Are you getting enough air?
8. Does it feel like your breath is not getting all the way in?
9. Does your chest feel tight?
10. Are you working hard to breathe?
11. Do you feel that you are smothering or suffocating?
12. Do you feel you cannot take a deep breath in?
13. Are you breathing more than you normally do?
14. Do you feel that your breath isn't getting all the way out?
15. Do you feel that your breathing is heavy?

In reality, if our list of 15 questions takes even one minute each to ask and answer, it would take longer than the current U.S. average office visit. Recent studies have shown that during a typical 15 minute visit, 6 topics are normally discussed with just 5 minutes spent on the longest topic.

But I am sure Mark would agree with me on this observation, if we don't begin to change the paradigm, the paradigm just isn't going to change.

N.B. Mark Mangus is one of the very best known respiratory therapists in the United States. He regularly contributes to a wide variety of respiratory publications. His areas of expertise encompass many different aspects of respiratory care. He is probably best known for his knowledge of "all things oxygen" covering everything from the lung disease itself to and through reimbursement issues. It is my honor to have him co-write this chapter with me. JRG 4/28/16.