For many modern people, as well as many Buteyko students, large CP fluctuations can favor spread of infections and intensify inflammatory processes.

Consider a typical person who has about 12-18 s CP in the morning and correspondingly heavy breathing during early morning hours (4-7 am). This physiological state is characterized, due to reversal of the citric acid cycle, by tissue hypoxia and production of incompletely oxidized chemicals (free radicals), due anaerobic energy production mechanism in mitochondria, causing chemical cellular damage. The immune system, instead of body repair and fighting pathogens, spends its resources on elimination of the damage done by tissue hypoxia. This favours susceptibility to getting cold and infections, sensitization of the immune system to relatively harmless chemicals and substances (dust, dust mites, mold, etc.) or development of new allergies, and advance of various pathogens in the respiratory, GI, and other systems and organs of the body. Feeling tired, inability to get out of the bed, blocked nose, production of extra-mucus, all are known effects of less than 20 s CP.

Later during the day, due to variety of positive factors, the CP can get up to 25-35 s. At this state, the immune system is able to fight many pathogens (e.g., in airways, sinuses, GI tract, etc.) due to greatly increased cellular oxygenation, improved perfusion of all vital organs and tissues, and normalization of the citric acid cycle. Even higher CPs (above 30 s) produce a powerful mobilizing effect on the immune system to deal with common bacteria, fungi, and viruses that accompany colds, flue, blocked nose, lung infections, GI complaints and many other infectious diseases and processes. Temporary inflammatory response is a very useful and even necessary reaction of the organism in order to recover. This response directs immune system components to the site of injury or infection and is manifested by increased blood supply and vascular permeability which, in technical terms, allows chemotactic peptides, neutrophils, and mononuclear cells to leave the intravascular compartment and to do the job of elimination and dis-activation of unwanted intruders. Once the inflammatory response and other defense mechanisms, due to high CPs, are triggered it takes more than 24 hours to normalize physical appearance and functions of the involved cells.

However, if the CP drops, especially below 20-25 s, as it is often the case with morning hyperventilation, all mentioned positive effects are substituted by the degenerative processes described above. Moreover, increased regions of inflammation favors advance of pathogens since inflamed cells have less resistance, in conditions of reduced blood supply and when the immune system is suppressed, in comparison with cells that are free from inflammation.

Hence, in conditions of large CP fluctuations, especially from over 30 s to below 20 s, inflammation, instead of being a sign of healing and cellular regeneration, becomes the ground that promotes advance of infectious processes even in tissues that have blood supply.

Does it take a long time for these negative effects of hyperventilation to appear? Even a temporary CP drop, just for 30-60 min can be sufficient, for these negative effects to take place. Practically, this usually happens during early morning hours. Moreover, when morning hyperventilation is a typical phenomenon, cyclic and large CP fluctuations, present for days, months, or years, result in chronic inflammation, possible adrenal exhaustion, lack of energy, and low cortisol reserves.

A similar effect takes place in cases of focal infections. Consider how the immune system deals with dead tonsils. At low CPs (less than 10 s), the immune system does not bother with dead tissues and pathogens harboured there. When the CP gets higher (up to about 25-28 s), the area around dead tonsils gets inflamed since the immune system starts to recognize and attack pathogens hidden in tonsils. However, in this case, the immune system is unable to reach pathogens who are hiding in tissues with no blood supply. Hence, the inflammatory response creates ground for spread of the infection. The person experiences severe tonsillar infection, fever, soar throat and other symptoms. The higher the CP achieved, due to reduced breathing, the stronger the anti-response that makes, after some hours, breathing heavy and drives the CP down to 12-15 s. Fungal infections on feet (athlete's feet) has the same general mechanism when higher CPs makes the condition much worse.

Large CP variations can practically take place in other zones of health as well. For example, the CP can drop, from about 15-20 s to below 10 s, or in the severely sick people below 5 s during early morning hours. While there are many negative and even life-threatening episodes in this zone (below 10 s), the "inflammatory resonance" effect is present only for the ranges described above and this is the scenario, when attempts to get higher daily CP numbers should be accompanied by activities related to normalization of factors that increase morning CP (mouth taping, prevention of sleeping on one's back, poor air quality, too warm sleeping conditions, deficiencies in EFAs or cortisol, and other factors). Otherwise, the effects of very high CPs can play the same negative role as in cases of focal infections.

The CP can also fluctuate for higher values, e.g. between 35 and 50, 60 or even 70 s for the advanced students who are trying to break through the 40 s threshold. Such persistent efforts may sometimes result in a breakthrough and getting consistently high CPs (above 50 s), and there are many specific details of this process that present a different topic.

While it may seem purely obvious that over breathing will cause a lowering of CO2 available, it is a curiosity that at times, someone who is asthmatic will have a high CO2 blood level. What are some of the various causes of High as well as low CO2 in Blood is a question that has been raised recently by a group of physicians very interested in the Buteyko approach. I want to answer them as thoroughly as possible.

Ans from Jennifer Stark

When people die from asthma they usually have high CO2, and it is due to the plugging of the airways, which prevents adequate gas exchange. As far as I am aware, the only way that you can have too much CO2 is if you don't have sufficient gas exchange for the level of CO2 you that producing. Because the alveoli in COPD are damaged, gas exchange is impaired and it leads to some people with COPD to have more CO2 than they are supposed to, while others still have low CO2.

and another from Rosalba in terms of a specific person shifting between high and low CO2 blood levels:
I dont know why this person had high CO2 and later low CO2. (refering ton a chart that I sent her from one of the Doctor's patients) It would be interesting to know bicarbonate levels and anion gap. Might make more sense then.

Ans from Peter Kolb

You raise a really important issue that we all need to be able to respond to when it is raised.

Firstly, McFadden found in 1968, that when the physical obstruction caused by the asthma is mild, during an asthma attack the CO2 level is very low, down at around 23mmHg (35-45 is considered normal). When the obstruction is severe, CO2 can be very much higher than normal. So it is necessary to consider the development of asthma from mild to severe.

In mild obstruction there is little damage to the lungs, little mucus plugging and air can access pretty much all of the lungs. However, once there is serious obstruction, parts of the lungs are significantly blocked while other parts have to do all the work to try to maintain normal CO2. This results in what is called a ventilation/perfusion mismatch, a condition in which blood is supplied to some of the alveoli that are not being ventilated. The bits of the lungs that are still working become heavily over-ventilated, and they then need to be kept open with bronchodilators to stop them closing down too. By working really hard, these parts of the lungs can usually get the CO2 level down unless the lungs are very badly blocked. However, the story for Oxygen is different. Because the blood in these parts of the lungs can't get more than 100% saturated (actually around 98 - 99% because of the bronchial circulation), the extra ventilation in the good parts of the lungs cannot compensate for the Oxygen that has not been delivered in the bad part of the lungs. So in the case of steady deterioration we see a progressive deterioration in Oxygen, while the CO2 level remains low initially and then appears to improve. There comes a critical point when low Oxygen starts to drive respiration instead of CO2 driving respiration.

So one reason some asthmatics have high CO2 is because the lungs have

become damaged and the asthmatic is suffocating. And why are the lungs damaged? Because of all the over breathing done in the early stages of asthma, when CO2 was low.

But there is also another possibility.

I'm not sure to what extend the CO2 level fluctuates. But you would expect it to drop during stressful times, such as during allergic reactions. Whenever it drops below the trigger level for bronchospasm, that's when you have and asthma attack. During the good times the CO2 may, indeed, rise into the normal region, and if you measure it during this time it would appear quite normal. People don't have asthma attacks continually, so that seems like a plausible summation. By raising baseline CO2 level with Buteyko, Yoga, meditation etc, you would keep the troughs from dropping below the trigger level. Bear in mind that the goal of Buteyko therapy is to re-set the capnostat to higher levels, which in practical terms means getting the bicarbonate concentration in the cerebrospinal fluid back up to normal.

The other very important point to remember about chronic hyperventilation is that it affects everyone differently. The trigger level for one person is not necessarily the trigger level for another. We should not be surprized when people trigger at different levels.

Ans from Artour

Practically, however, it is good to explain to our students why they will suffer more from hv and why they will benefit from the RB.

First, during their hv, their bronchi and brocnhioles further constrict causing even greater number of alveoli become dysfunctional due to reduced air access. This causes even higher arterial CO2, while alveolar CO2 decreases.

During the RB, as Patrick explained, large, medium and small airways dilate causing air access to a greater number of alveoli. Hence, oxygenation of the blood improves and CO2 in blood drops.

Both effects are easy to measure with oxymeters (which getting much cheaper in recent years and soon can be some tens dollars only). During the RB, people with ventilation-perfusion mismatch will find, if they use oxymeters, that their O2 in capillaries and tissues (this is what oxymeters measure) is improved.

As about the mechanism, Clinical Science published in 1968 an article, The mechanism of bronchoconstriction due to hypocapnia in man. In this paper, Sterling explained that CO2 deficiency causes an excited state of the cholinergic nerve. Since this nerve is responsible for the state of the smooth muscles in bronchi, its excited state leads to the constriction of air passages.

Sterling GM, *The mechanism of bronchoconstriction due to hypocapnia in man,* Clin Sci 1968 Apr; 34(2): 277-285.

These things should be explained to these students so that they have 100% confidence in the method