Winter 2008

Everest and Oxygen—Ruminations by a Climber Anesthesiologist

By David Larson, M.D.

Gaining Altitude and Losing Partial Pressure with Dave and Samantha Larson

Dr. Dave Larson is an obstetric anesthesiologist who practices at Long Beach Memorial Medical Center, and his daughter Samantha is a freshman at Stanford University. Together they have successfully ascended the Seven Summits, the tallest peaks on each of the seven continents, a feat of mountaineering postulated in the 1980s by Richard Bass, owner of the Snowbird Ski Resort in Utah. Bass accomplished it first in 1985. Samantha Larson, who scaled Everest in May 2007 (the youngest non-Sherpa to do so) and the Carstensz Pyramid in August 2007, is at age 18 the youngest ever to have achieved this feat.

Because of varying definitions of continental borders based upon geography, geology, and geopolitics, there are nine potential summits, but the Seven Summits is based upon the American and Western European model. Reinhold Messner, an Italian mountaineer known for ascending without supplemental oxygen, postulated a list of Seven Summits that replaced a mountain on the Australian mainland (Mount Kosciuszko—2,228 m) with a higher peak in Oceania on New Guinea (the Carstensz Pyramid—4,884 m). The other variation in defining summits is whether you define Mount Blanc (4,808 m) as the highest European peak, or use Mount Elbrus (5,642 m) in the Caucasus. Other summits include Mount Kilimanjaro in Kenya, Africa (5,895 m), Vinson Massif in Antarctica (4,892 m), Mount Everest in Asia (8,848 m), Mount McKinley in Alaska, North America (6,194 m), and Mount Aconcagua in Argentina, South America (6,962 m).

The Larsons have ascended Kilimanjaro, Elbrus, McKinley, Aconcagua, Kosciuszko, Everest, Vinson, and Carstensz—eight peaks to qualify unequivocally for the Seven Summits list of climbers. As of March 2007, 198 climbers have climbed all seven in either the Bass or Messner lists, and a mere 30 percent climbed the eight peaks required to complete both lists, as have the Larsons. What follows is a personal account from Dr. Larson, a CSA member.

—By Kenneth Y. Pauker, M.D., Associate Editor

Anesthesiologists, unique amongst all physicians, have a special relationship to oxygen. We measure it obsessively in our patients, and know from the tone of the pulse oximeter with every heartbeat just how “full” of oxygen our patient’s blood is. If it drops, we react quickly to diagnose and treat the change. We are keen on 100 percent oxygen saturation.

As an anesthesiologist and climber, my own personal relationship with oxygen changed when I started climbing 8,000-meter peaks, culminating with my summiting Mount Everest on May 17, 2007. At Everest’s 29,032 foot summit—the highest point on earth—there is merely a third of the partial pressure of oxygen that exists at sea level, and therefore just a third of the amount of
oxygen available for respiration. Alveolar \( pO_2 \) is about 35 mm Hg and arterial \( pO_2 \) is about 30 mmHg! While tackling Everest requires a concatenation of incredible physical and mental stamina, good weather, two months in the Himalayas, and luck, it is the body’s process of acclimatization to hypoxia that is fundamentally critical for success. Anyone magically transported from sea level to 29,000 feet without acclimatization would die from hypoxia within minutes. Time is required to allow the body to adapt with physical processes that enable humans to survive above 25,000 feet, the so-called “death zone.” Even when fully acclimatized, life above 25,000 feet is tenuous—you lose your appetite, your weight plummets, you become lethargic, and muscle wasting ensues.

A rule of thumb is to ascend no more than 1,000 feet per day, and then to take a rest day every other day to allow time for acclimatization. The trek to Everest starts at about 9,000 feet, and hence requires at least 30 days to reach 29,000 feet.

The first and most important compensatory change is an increase in respiratory rate, which, by presenting an increased opportunity for oxygen uptake in the pulmonary capillaries, increases oxygen delivery. Minute ventilation increases in response to (a) stimulation of the peripheral chemoreceptors (carotid bodies) by hypoxia and (b) a change in the central chemical control of breathing, wherein hypoxia causes a reduction in CSF bicarbonate, which of itself stimulates ventilation. Full respiratory acclimatization requires about 45 days.
The second compensatory change is an increase in red cell mass. Since oxygen is carried by red blood cells, the more the merrier. Having more red blood cells increases oxygen-carrying capacity. Hypoxia induces release of erythropoietin, which stimulates bone marrow to increase red blood cell production. A significant increase in red cell mass takes weeks and hemoglobin levels can increase from 14.5 g/dl to 20 g/dl.

A third physiologic compensation is the marked leftward shift of the oxyhemoglobin dissociation curve. This increases the affinity of hemoglobin for oxygen, which in turn enhances diffusion across the blood-gas barrier and enhances oxygen loading in the pulmonary capillaries. The curve is also shifted to the left by a marked respiratory alkalosis. On the summit of Everest, minute alveolar ventilation is at least 40 liters per minutes, arterial pH is about 7.7, and alveolar PCO2 is around 14.

KP: What about increased 2,3-DPG? Doesn’t this shift the curve to the right to facilitate off-loading of oxygen in the tissues? Does this contribute to conditioning at altitude or only at sea level? Is the curve left shifted in the lungs to pick up oxygen and right shifted in the tissue to facilitate delivery?

DL: Animals that live in oxygen-deprived environments have hemoglobins with high oxygen affinities. For example, fetal hemoglobin has a p50 of 19 mm Hg, compared with a p50 of 27 in normal adult hemoglobin.

2,3-DPG is a product of red cell metabolism. Increased 2,3-DPG in the red cell reduces oxygen affinity of hemoglobin by increasing the chemical binding of the subunits and converting more hemoglobin to the low affinity T form. The effect of the profound respiratory alkalosis at extreme altitude overwhelms the small decrease in oxygen affinity caused by the increased concentration of 2,3-DPG in the red cells.

An increased oxygen affinity is advantageous at high altitude because it assists in the loading of oxygen at the level of the pulmonary capillaries. Moreover, at extreme altitude, metabolic compensation for the respiratory alkalosis is slow, possibly because of chronic volume depletion caused by dehydration.

The reality is that, at extreme altitudes, the blood oxygen dissociation curve shifts progressively leftward (increasing oxygen affinity of hemoglobin) primarily because of respiratory alkalosis. This effect completely overwhelms the relatively small tendency for the curve to shift to the right because of the increase in red cell 2,3-DPG. The oxygen gradient between the blood and tissues must be so great that a small right shift in the curve
caused by increased 2,3-DPG is not particularly helpful in supplying oxygen. The key is to facilitate oxygen loading in the pulmonary capillaries, and this is dramatically enhanced by the marked left shift of the oxygen dissociation curve caused by the extreme respiratory alkalosis.

Twenty-nine thousand feet is at the cusp of human physiological ability to survive. I wanted all the help I could get. I breathed oxygen through a recently developed high altitude climbers' mask called “Top Out.” This mask is designed with a 500 cc reservoir so that although the flow rate is just two liters per minute, the first 500 cc of intake is enriched with oxygen and is delivered to the most distal alveoli. These new masks have been used for about four years and are a great improvement over the old reservoir-less Russian climbing masks.

High altitude climbers have long used acetazolamide (Diamox) to enhance and speed up acclimatization. Diamox is a carbonic anhydrase inhibitor. Interference with CO₂ transport is thought to result in intracellular acidosis of cells of the central medullary chemoreceptor. In this way, it acts as a respiratory stimulant. It also changes CSF pH and causes a left shift of the O₂ dissociation curve. It also increases cerebral blood flow and cerebral pO₂. Studies have shown that it can reduce altitude deterioration. Acetazolamide also effectively eliminates the disturbing Cheyne-Stokes or periodic respiration that frequently affects climbers at high altitude and makes it easier to get a good night's sleep. Side effects include paresthesias in the hands and feet, and mild diuresis. These side effects diminish with continued use and are reduced by using a dose of 125 mg p.o. BID. Unfortunately, inhibition of carbonic anhydrase in the tongue prevents the conversion of carbon dioxide to carbonic acid (in fizzy drinks like beer), and the acid-sensing taste buds are not activated. This makes beer taste awful.

Although dexamethasone has been shown to improve acclimatization in combination with acetazolamide, most climbers view Decadron® or “DEX” as a rescue drug, to be used only after one develops high altitude pulmonary edema (HAPE) or high altitude cerebral edema (HACE). Assiduous compliance with a thorough acclimatization strategy gives the highest chance of success in reaching Everest's summit, as well as making the two-month Himalayan sojourn considerably more pleasant.

I acclimatized well and suffered very few of the symptoms of acute mountain sickness (AMS), which include headache, nausea, insomnia, lethargy, loss of appetite, and dizziness. AMS portends the potentially life-threatening conditions HAPE and HACE, and should be treated with rest and analgesics. If symptoms do not resolve or progress, descent, supplemental oxygen, and dexamethasone are necessary.
In spite of thorough acclimatization, I was still hypoxic—for many weeks. I carried a portable aviation-type pulse oximeter with me and measured my oxygen saturation until it became psychologically uncomfortable—I did not want to know just how low it could go. At base camp, 17,500 feet, with one-half the partial pressure of oxygen compared to sea level, my maximum oxygen saturation was 91 to 92 percent; and that was achieved by hyperventilation in a standing position. At rest in a supine position (sleeping), it was 84 to 88 percent. At camp 3 (24,000 feet) it was in the low 80 percent range at best. We breathed oxygen by facemask above 24,000 feet, but the oxygen saturation was not greatly improved.

My “summit day” was 20 hours long and started at 11 p.m. after a fitful four-hour rest at the South Col (camp 4) at 26,000 feet. I had only been able to drink about a liter of fluid and brought 1.5 liters of liquid with me. We had a good weather window, which means high barometric pressure and hence more oxygen availability. I headed out in darkness for the summit. Despite using supplemental oxygen at two liters per minute via “Top Out” mask, I occasionally experienced hypoxic panic after finishing an especially vigorous move such as climbing the Hillary step just before the final summit ridge. After struggling up the Hillary step, I laid down in the snow, breathing 60 times per minute and with a horrible sensation of absolute suffocation. I reached deep into my mind and was able to hear my voice saying, “Slow down. Take it easy,” and I fully regained my equilibrium.
I recovered and made my way to the summit, avoiding the precipitous 6,000-foot drop into Tibet. It is here where you take 10 deep breaths for every step, and rest after every step. I do not know what my oxygen saturation was, but I do know that I was on the edge of viability and critically dependent on my oxygen canister. A sudden failure of an oxygen delivery system can cause hypoxic panic, extreme hyperventilation, limb paresthesias, and urinary incontinence. Supplemental oxygen clearly increases the chance of success and survival by increasing endurance and climbing speed. When I finally reached the summit, I sat down and rested, and I felt great—figuratively and literally on top of the world. However, with any exertion my body would immediately react with hyperventilation. In spite of the high altitude hypoxia, and save for my few episodes of utter hypoxic panic, I felt fantastic—truly high as a kite. I knew where I was at each moment and can remember each step, although many were indistinguishable from their predecessors. It was a thrill and an honor to be on the top of Everest.

Now back in the OR, I still obsessively monitor my patients’ oxygen saturation. On Everest, my body screamed for each molecule of oxygen—now I admit I often take the rich atmosphere at sea level for granted. However, having lived and thrived in a hypoxic world for weeks, I have a better appreciation for the reserve that most patients have, and do not panic if their oxygen saturation drops during a difficult intubation (I still do everything I can to prevent it). I can also empathize with patients who have end-stage pulmonary disease. They have to live as though they were at the top of Everest while at sea level, and moreover they have nowhere to descend to, to get more oxygen.

**KP:** Dave, considering your mature age, and with so many opportunities for travel, exercise, and vacations to “exotic” locales, why do you choose to subject yourself to the rigors of this “extreme sport”? Will you continue? Is there more to do? How much of your doing this is about your relationship with Samantha?
DL: A somewhat obtuse and overused answer to the “Why Climb” question is: “If you have to ask the question, you won’t understand the answer.” Suffice it to say, there is some exhilarating, compelling, and mysteriously rewarding aspect to alpine climbing that needs to be experienced firsthand if one is to understand its alluring appeal.

While the Seven Summits quest may look like a list, Samantha and I chose the seven because we needed a structure to continue our climbing activities after Kilimanjaro in 2001 when Samantha was 11. Kilimanjaro set the stage; we both loved the experience and wanted to continue climbing. We had limited time, but could get away with one major “family” climbing trip a year. The Seven Summits fit the bill. It gave us the opportunity to travel the globe with climbing challenges of increasing complexity, culminating with Everest and Carstensz Pyramid in 2007.

Our climbing became a joint passion, and I don’t think either of us would have persisted if the other chose not to continue. It was a unique vantage point to share such an “extreme” activity with my daughter and watch her grow to young adulthood in the mountains of the world. We are both very busy and have lots to do in the lowlands, but would leave tomorrow for the hills when the chance arises. Samantha, what are your thoughts?

Samantha Larson: I’d like to add that a huge part of climbing the Seven Summits was the shared goal with my dad. He will always be my favorite climbing partner. Although right now I have no definite goals, I will definitely continue climbing. Having just completed the Seven Summits, I am back to daydreaming about the possibilities of peaks to come.

KP: What do you think about and what do you feel upon reaching an “extreme” summit?

DL: In one way, the summit is just one more step on our journey. After completing this quest, we will always carry the summits with us and can revisit them in times of quiet introspection. In another way, reaching an extreme summit is so extreme that all you can think about is the next step and the next breath. The world is distilled to NOW—there is no past or future. Finally, it is also the culmination of years of preparation and weeks of hard work, and so you do shout to the sky, “We made it!” and high five your climbing partners, then take the customary summit photo to make the summit real.

SL: Reaching a summit is the cherry on top of a great expedition, but for me the summit is not the ultimate point of a climb. It’s about those
Everest and Oxygen (cont’d)

moments, wherever they may strike you, where you look around and realize that even though you putting yourself through hell, there is nowhere else you would rather be.

References

The Educational Programs Division of the California Society of Anesthesiologists gratefully acknowledges

SUSTAINING PATRON
Abbott Laboratories

for its support of the 2007 CSA/UCSD Annual Meeting & Clinical Anesthesia Update

Abbott has a broad range of pharmaceutical products for Anesthesia and Pain Management Care