Physiology of Altitude Illness

Its Role in Alpine Style Rush Tactics

CHARLES S. HOUSTON, M.D.

LAST YEAR a world-class climbing physician, an expert in altitude illness, died of high-altitude pulmonary edema while descending from an attempt to rush a major Himalayan peak. It shocked but did not surprise me because alpine style climbing (rushing) at great altitude is a very dangerous game—a bit like Russian Roulette with half the chambers loaded. I'd like to explain, on the basis of what we know today, why this is so.

We all know that lack of oxygen stimulates breathing and increases the output of the heart. Increased breathing increases the oxygen deep in the lungs by better mixing of fresh and stale air, but at the same time it washes out carbon dioxide. This increases the alkalinity of the blood (raises the pH), causing respiratory alkalosis.

We know that lack of oxygen dilates the small arteries serving the brain, while lack of carbon dioxide constricts them. As a result, whether these crucial blood vessels are dilated or constricted (and thus carry more or less blood to the brain) depends on how much the increased breathing raises oxygen and lowers carbon dioxide. At the same time the increased output of the heart increases blood flow, probably everywhere in the body. The net result at moderate altitude is likely to be increased blood flow to brain and lungs, unless alkalosis has constricted arteries excessively.

One of the immediate effects of lack of oxygen is an increase in the blood pressure in the pulmonary arteries which carry oxygen-poor blood to the lungs. We don't know why this happens, but it happens fast and some people show a greater increase than others. By contrast, altitude causes little change in blood pressure to the rest of the body.

Recent studies have shown that increased blood flow together with increased pressure in the pulmonary arteries causes several reactions. Certain white blood cells called "mast cells" (particularly numerous in the lungs) respond by releasing a substance called arachidonic acid which quickly breaks down to several compounds, among them thromboxane, a substance that constricts blood vessels and causes blood platelets (important in blood clotting) to clump together, and prostaglandins which are rapidly converted to several different prostacyclins with exactly the opposite effect, that is, they cause vessels to

dilate, and they prevent platelet clumping. Usually these reactions cancel each other out.

There's another effect of increased blood flow in the small vessels of the lung, called "shearing." Our concept is that the endothelial cells which line small blood vessels are "ruffled," and when so disturbed they release a family of substances called leukotrienes. One of these increases the permeability of capillaries, making their thin walls more easily penetrated by fluids. Other leukotrienes are also released, but we know less about them. Whether vessels dilate or constrict, and whether platelets clump or separate depends on which substances dominate, and what controls this is unknown today.

Mast cells occur in all parts of the body, and there's no reason to doubt that they react in the same way as in the lung. So we suspect that in the brain the same balances may occur. Capillaries in the brain are lined with endothelial cells just as in the lung; they too may be "ruffled" and release leukotrienes, making brain blood vessels more permeable, and allowing fluid to leak into the brain tissue.

To some degree this may also happen elsewhere, but blood pressure does not increase throughout the body, and the increased blood flow is more widely dispersed than in brain and lungs. Consequently less of the arachidonic acid products and leukotrienes might be released, and their impact would be smaller.

If these speculations are correct, we come closer to understanding high-altitude pulmonary edema (HAPE) and high-altitude cerebral edema (HACE). Tiny clumps of platelets may obstruct blood flow to some parts of the lung, causing greater flow to other parts and, in combination with increased capillary permeability, producing the patchy edema (accumulations of fluid) so characteristic of HAPE. In the brain, leakage of fluid and scattered obstruction in small vessels might cause similar patches of swelling.

This concept is strengthened by the recent observation that fluid obtained from lungs of persons with HAPE is very similar to blood plasma—the liquid part of blood. This implies that plasma and red blood cells have gotten through the vessel walls and the linings of the lung air sacs (alveoli), most likely through "holes" which develop between the cells when the walls are stretched. These observations confirm suggestions made twenty years ago and begin to make a plausible explanation for many things not well understood.

We can combine these recent findings and speculations with an already well-accepted theory to make a unified concept. For many years we have been confident that lack of oxygen causes a reversible breakdown of the "sodium pump," a bio-electric activity of all cell membranes. This "pump" constantly pushes sodium ions out of cells while holding potassium inside. It uses a good deal of oxygen, and at altitude the pump may falter, allowing sodium to build up in the cell and water to enter, causing the cell to swell. This is likely to be more pronounced in some places than others, resulting in patchy evidence of "pump failure." We have used this neat theory to explain the variety of signs and symptoms of AMS, but it has been harder to apply to HAPE and HACE. The studies on arachidonic acid and leukotrienes round out a plausible concept though it still needs more information about several points.

One of these is the complicated hormone response to oxygen lack. We know a great deal about several hormones with complicated names and functions: anti-diuretic hormone, somatotrophin (human growth hormone), the renin-angiotensin family, and others. But fitting the large body of information into a unified theory is very difficult.

If and when we find safe medicines that cancel out the prostacyclins the leukotrienes and thromboxane, we may be well on the way to a safe preventive and treatment for all forms of altitude illness.

What has all of this to do with alpine style climbing or rushing? If—and it's a moderately large if—the above train of events accurately indicates what happens, then we can better understand why rushing may have greater danger for some individuals and on some occasions than for others or at other times. First, the changes take time to develop and while they develop, opposing changes are developing. Secondly, hormonal responses are conditioned by a great many other factors which we don't understand very well. Thirdly, other influences such as amount of water, salt, and type of diet taken, the intensity of work, extent of fatigue and many others change the responses. Finally, speed of ascent is important: if one goes up slowly, the body compensates and slowly acclimatizes (another fascinating story). If one goes up *and down* very fast, the damage may not have time to develop. But we cannot be sure how all of the influences are lined up in any individual under conditions prevailing at a specific time: perhaps they may be favorable, or perhaps, as in the case of my friend who died, they may not.

On the other hand there are certain real advantages to alpine style climbing. First: it is well recognized that one deteriorates rather than acclimatizes above 20-21,000 feet. The longer the climber lives very high on a mountain, the feebler he becomes. Siege tactics wear a party down. Second: sleeping low and packing (climbing) high is a spendid maxim, well proven over decades. It makes good sense to live as low as feasible when attacking a very high summit. Third: the risk of being caught by prolonged storm, making advance or retreat impossible increases the higher one goes. Fourth: dehydration, hard to combat on a big mountain, carries the ever present danger of blood clots (thrombo-embolic disease) which occur dismayingly often on major climbs. Fifth: the small fast-moving party is less expensive and places smaller demands on the strained local economy. Finally: climbers who spend weeks acclimatizing to altitudes up to 18,000 feet are sometimes better able to keep moving toward a high summit than are those who inch their way in siege style.

These arguments are attractive and they might prevail if rushing were the only alternative to siege tactics. But an ideal expedition can be small and inexpensive, the party can pack high and sleep low, can spend weeks acclimatizing and choose optimal weather, and can climb at a rate well within the limits of safety. These are set by how the weakest member feels and acts. If the pace is well-tolerated by every one, the risks of serious altitude illness are small. It is possible to minimize dehydration if the party is conscientious.

When one considers how the beauty and pleasure of climbing are diminished by haste, and calculates the risk (however spicy danger may make a venture), rushing seems neither wise nor attractive. The intensity of effort obscures the beauties, hypoxia blunts the senses and hurts the head. If only the individual were at risk, it might be left to individual choice. But far too many others take great risks trying to rescue the foolhardy. Rushing a high mountain may appeal to those whose motives are to set records but not to the true mountain lover.