Reports of Scientific Meetings

Bryan E. Marshall, M.D., Editor

Arctic Institute's First Symposium on Hypoxia

It was a curious gathering. There were physiologists, pathologists, and biochemists, along with university professors from a variety of medical specialties; in short, the usual make-up of a scientific meeting. However, at the Arctic Institute's First Symposium on Hypoxia, held in Banff, Canada, February 21–24, 1979, there were also mountain climbers, park rangers from the world over, and skiers. This unlikely mix of researchers, clinicians, and laymen was deliberate. The symposium director, Dr. Charles Houston, of the University of Vermont, a mountain climber himself and a pioneer in the field of high-altitude research, brought together a wide range of individuals experienced with hypoxia, both in the laboratory and in the field.

The program was divided into two parts. During the first three days, keynote speakers reviewed basic topics concerning hypoxia, and 50 original papers were presented. On the final day, a "state of the art" review was held, especially designed for the lay public, affording climbers, skiers, flyers, trekkers, and others who might be exposed to hypoxic conditions an opportunity to hear where the scientific work has led.

The scientific sessions opened with Dr. D. J. M. Campbell, of McMaster University, reviewing the history of oxygen on earth over the past four billion years. In this provocative talk Dr. Campbell pointed out that for two billennia oxygen was a toxic byproduct of life, and raised the question, "Could it be that the mitochondria primarily evolved as mechanisms for scavenging oxygen to protect some cellular mechanisms from P_{O_2} which they found intolerable, and that the mitochondria only secondarily adapted to other cellular purposes?"

After posing this question, Dr. Campbell went on to open an issue that was to be much discussed, both seriously and playfully, throughout the conference. This issue was the appropriate definition of the terms hypoxia, anoxia, dysoxia, etc. Although much lively debate took place, no official concensus was reached, and several schools of thought about appropriate definitions remained.

Dr. Campbell was followed by Dr. David Denison, of the Brompton Hospital, London, who has worked extensively on the aviation aspects of hypoxia. In his excellent talk, he presented evidence of cerebral dysfunction, particularly of learning and judgment, at rather mild hypoxic levels, for instance, that which occurs in a pressurized cabin of a commercial jet aircraft. Dr. Denison pointed out that a wide variety of metabolic processes in the brain require oxygen, and that some of these enzyme systems require much higher partial pressures of oxygen to operate than the oxidative phosphorylation pathway.

This point was also made by Dr. Bo K. Siesjö, from Lund, Sweden, during his address, "The Brain during Hypoxia." In this presentation, he made the point, surprising to those present who were brought up on the dictum that oxygen lack not only "stops the machine but wrecks the machinery" that hypoxia alone doesn't seem to cause cellular damage in the brain. The damaging situation occurs when cerebral hypoxia is coupled with a decrease in cerebral blood flow.

Other keynote speakers included Dr. Eugene Robin, of Stanford University, who spoke about metabolism during hypoxia, Dr. John T. Reeves, of the University of Colorado, who traced the evolution of the pulmonary vasoconstrictive response to hypoxia in man, and Dr. Norman Staub, of the University of California, who presented the concept that this uneven pulmonary arterial constriction is the mechanism for the pulmonary edema of hypoxia (high-altitude pulmonary edema, or HAPE).

The scientific papers addressed a wide range of subjects, from retinal anoxia in the rainbow trout to oxygen transport in malnourished children. Perhaps the most controversial subject was presented by Dr. B. Burns, of Johns Hopkins University, under the title, "Maximal DL_{O_2} revisited: Oxygen diffusion in the lungs and body tissues." Dr. Burns and his colleagues used a sophisticated isolated animal-lung model, perfused with sodium dithionite (which combines rapidly and irreversibly with oxygen) to measure DL_{O_2}. They found that by eliminating all oxygen back-pressure with the dithionite the membrane diffusion capacity of the dog lung was enormous, and that it would be most unlikely ever to limit gas exchange. Much animated discussion followed this paper, and it was unfortunate that Dr. John West, who had been scheduled as a keynote speaker, was ill and unable to attend and take part in this discussion concerning diffusion capacity.

During the "layman's session" on Saturday, clinical experts from around the world presented their experience of treating patients with acute, altitude-induced disease. As more travelers go to high altitudes during vacations, these entities are becoming more common. Several physicians, notably Peter Hackett, of Pheriche in Nepal, and Rodman Wilson, in Anchorage, have a large part of their practice devoted to treating acute mountain illness, and both shared their experiences during this session.

Two basic points emerged from the discussions among the doctors, park rangers, and scientists involved in treating high-altitude illness. The first is that the best way to avoid illness is to ascend slowly, and the second is that the best way to treat it is to descend rapidly. The use of acetazolamide, diuretics, steroids, and rest was both endorsed and questioned. There simply are inadequate data to decide definitely on the places of these therapies.

The symposium organizer, Dr. Houston, conducted a session on retinal hemorrhage, a common occurrence at high altitude. He feels that asymptomatic retinal hemorrhages in a well individual do not constitute a reason to abandon a high-altitude venture. As with most of the clinical points, this position was controversial.

Dr. Roman Zink, of Munich, West Germany, again discussed the work he presented at the ASA meeting in Chicago. Dr. Zink has hemodiluted acclimatized mountain climbers by bleeding them and replacing the blood with albumin solution, thus lowering their very elevated hematocrits into the high-normal range. The work was done in an uncontrolled fashion, so it is impossible to draw conclusions from it, but all the climbers manage to climb strongly after the hemodilution, and the approach represents a significant departure from the usual thinking about oxygen transport.

The surrounding snow-covered mountains of Banff National Park provided recreational diversion for the participants, and an exciting slide show of an ascent, without the aid of supplemented oxygen, of the world's second highest peak, K-2 (28,000 feet, with a partial pressure of oxygen of 55 torr ambient) afforded an example of human capacity for acclimatization to hypoxia.

I suspect from the enthusiasm at the end of the symposium that another such meeting will be planned, and if it is up to the quality of this meeting in Banff, it will have much to offer to the anesthetist interested in hypoxia, whether it occurs in the operating room, in the intensive care unit, or on a mountain top.

FRANK H. SARNQUIST, M.D.
Department of Anesthesia
Stanford University
Medical Center
Stanford, California 94305