

Research News

Lake Nyos Was Rigged for Disaster

Studies suggest that magmatic gas seepage had turned Lake Nyos in Cameroon into a time bomb; evidence mounts against a volcanic trigger for last August's disaster

THE report of the 11-member U.S. team of experts that investigated the Cameroon lake disaster of last August confirms that ground-hugging rivers of carbon dioxide asphyxiated the 1700 victims. The report argues that the lethal gas had slowly charged the deep waters of Lake Nyos until some disturbance, in all likelihood not a volcanic eruption, precipitated its release much the way popping the top of a warm bottle of soda creates a fountain of foam. The report warns that disastrous degassing could happen again at Lake Nyos or other Cameroon lakes nestled in deep volcanic craters.

According to the report submitted to the Office of U.S. Foreign Disaster Assistance, even after the catastrophic gas release a liter of water from below the lake's stable surface layer was charged with an estimated one to five liters of dissolved gas. Ninety-eight to 99% of the gas was carbon dioxide. But the carbon dioxide does not seem to have had a volcanic source. Typical volcanic gases—hydrogen, hydrogen sulfide, carbon monoxide, hydrogen fluoride, and sulfur dioxide—were below detection limits of a few parts per million 12 days after the disaster. Carbon dioxide in the lake water was at least 100 times more abundant relative to sulfur than in volcanic gases.

But there is still strong evidence that the carbon dioxide came from within the earth. Its carbon-14 age is greater than 35,000 years, so organic decomposition in sediments of the lake, which was probably created only a few hundred years ago, could not have produced it. Its ratio of helium-3 to helium-4 is 6; any ratio greater than 1 means that a significant fraction of the helium escaped from magma. And the carbon dioxide's ratio of carbon-13 to carbon-12 is consistent with a magmatic source.

A possible subterranean but, strictly speaking, non-volcanic source of carbon dioxide for Lake Nyos is apparent. Nearby springs are exceptionally rich in carbon dioxide and magnesium, as is the lake. And the stable isotope compositions of both the oxygen and hydrogen of Lake Nyos water are heavier than those of nearby springs but distinctly lighter than those of four other Cameroon lakes whose water compositions

resemble slightly concentrated rainwater. That suggests that the deep waters of Nyos are fed by carbon dioxide-laden spring water as well as by rainfall. Intriguingly, the waters of Lake Monoun, the only other Cameroon lake known to have a similar catastrophic gas release, have isotopic compositions and mineral contents that bear a similar relationship to the springs and the Cameroon lakes.

The American experts point out that Lake Nyos would be a natural receptacle for gas released by magma. It sits within a maar, a roughly circular crater formed by an explosive eruption driven by gases from magma pushing toward the surface or by ground water turned to steam by the magma. A vertical, rubble and ash-filled pipe undoubtedly extends downward several kilometers beneath the lake to the remains of the intruding magma. Gas originally dissolved in magma could easily have lost its noxious components through chemical reactions as it cooled on its way from the deep-seated magma to the lake, the report notes.

Contrary to the impression formed by early press reports, the American team could find no evidence of a volcanic eruption and

its attendant heat, toxic gases, and violent disruption. According to the report, the evidence does "not support a hypothesis of recent, direct injection of lava or volcanic gas." In contrast, S. J. Freeth of the University College of Swansea and Linden Kay of the British Geological Survey in Wallingford conclude in a News and Views story in *Nature* that although the lethal carbon dioxide may have quietly accumulated in the lake, a minor volcanic eruption beneath the lake did trigger its release. George Kling, a U.S. team limnologist from Duke University, sees such a trigger as difficult to rule out but less likely.

In addition to the absence of volcanic gases, the American team cites several arguments against an eruption or even a sudden breakthrough of magmatic gas stored just beneath the lake. The American pathologists attribute the victims's "burns" or lesions to more mundane causes: preexisting disease, the skin pressure of lying unconscious up to 36 hours that created bed-sore-like lesions, and the collapsing of victims on or near cooking fires. Plants appeared unaffected. Reports of streams flowing with hot water could not be substantiated.

In addition, the team could find no sign of disruption of the lake bottom. Below the surface layer, lake water was clear and free of suspended sediment. Bottom sediment contained no fresh volcanic material. A depth-finder survey showed a broad, flat bottom at a depth of 208 meters and smoothly sloping bottom almost to the shoreline. Freeth and Kay backtracked from the areas of wave runup around the lake shore to locate their eruption in the northeast corner of the lake, within a pocket formed by a wall of volcanic rock that they call a volcanic cone. The American bottom survey found nothing unusual there; nor did a sounding-line survey in 1912. Water temperatures measured from the surface to the bottom after the event were typical of other lakes in the region.

Freeth and Kay cite reports of the smell of rotten eggs or gunpowder as evidence of volcanic activity, but the American pathologists disagree. "The description of the odor of 'rotten eggs' or 'gunpowder', by many survivors," the report says, "although usually associated with sulfur gases, is also com-



George Kling

Another bomb waiting to go off?
Lake Mfou, like Lake Nyos, is a Cameroon crater lake. Whether it is being charged with a deadly load of carbon dioxide is unknown.

monly described by individuals exposed to high concentrations of carbon dioxide. This phenomenon is termed an 'olfactory hallucination'."

An undetectable volcanic eruption could conceivably have triggered the gas release, says Kling, but the American team only specifically discusses several other possibilities. All that was needed, assuming the deep water was saturated or nearly saturated with carbon dioxide, was a disturbance that raised the water to the point that the resulting pressure reduction allowed the gas to come out of solution and form bubbles. That would have further reduced the weight of overlying water that had kept the gas in solution and begun a runaway release. In the case of Nyos, the team estimates, as much as 1.0 cubic kilometer escaped. An earthquake (none was reported), a landslide (a fresh scar on the lake's western cliffs was found but no subsurface disturbance was apparent), or a volcanic eruption could have triggered the release, but something as ephemeral as a wind that started the water sloshing within the lake basin would have sufficed.

Kling still finds quite suggestive the coincidence of both Cameroon lake disasters in the month of August (Lake Monoun on 16 August 1984 and Lake Nyos on 21 August 1986). August through September is the time of minimum stability of some Cameroon lakes, when they are closest to mixing their anoxic deep water with their surface water. The occurrence of two otherwise undetectable volcanic eruptions beneath lakes 95 kilometers apart at the time of minimum stability seems less likely to Kling than normally inconsequential disturbances striking lakes on the verge of turning over, as some Cameroon lakes do from time to time and temperate lakes do every fall.

Whatever the immediate trigger, the team recommends that the 40 crater lakes in Cameroon be better understood in order to predict and prevent future disasters. Nine of the lakes have not been sampled to determine if their bottom waters are highly charged with carbon dioxide. Repeated sampling would detect any buildup. Dangerous lakes, including Nyos, could be defused in a few years' time by starting fountains of water supplied by pipes from the bottom. Once started, the fountains would be driven solely by the lifting force of the gas release.

Understanding these lakes would come none too soon. The American team has been told that according to legends of the Lake Monoun area, there may have been at least three earlier cases of exploding lakes or mass deaths. And there is a new report of three small explosions on Lake Nyos within a 5-minute period on 30 December. ■

RICHARD A. KERR

Oxygen Free Radicals Linked to Many Diseases

The oxygen free radicals, although made as by-products of normal oxygen-using reactions, nevertheless have a wide potential for causing cell injury

BREATHING oxygen is, it seems, hazardous to your health. Although the element is indisputably necessary for life, many of the biochemical reactions in which it participates generate oxygen-containing free radicals as by-products. These highly reactive chemical entities can injure and even kill cells. "Oxidative damage is common, and DNA, proteins, and lipids are all at risk," says William Pryor of Louisiana State University. According to presentations at a recent symposium sponsored by the National Heart, Lung, and Blood Institute,* oxygen free radicals may contribute to the development or exacerbation of many of mankind's most common ills, including cancer, heart attacks, stroke, and emphysema.

The work may have immediate clinical application in the treatment of heart attacks. Approximately 700,000 heart attack victims are admitted to hospitals every year in the United States. Many of these individuals now receive therapy aimed at restoring the blood flow to the damaged heart muscle. Usually this involves treatment with the enzymes streptokinase or urokinase to dissolve the clots that block the coronary arteries. However, these enzymes may soon be replaced by another, tissue plasminogen activator, which may be more specific in its action. The arteries may also be opened mechanically by a technique called coronary angioplasty, in which a small balloon is inserted in a blocked artery and inflated.

Although these treatments are beneficial, there are indications that restoration of the blood flow to heart tissue that has been deprived of oxygen may contribute to the heart muscle damage, at least partly because of the production of oxygen free radicals. If that is the case, then treatment to destroy the free radicals may help to minimize the extent of the permanent damage to the heart muscle, and thereby improve the outlook for the patient. Evidence presented at the symposium suggests that such treatment can work, at least in experimental animals.

*The NHLBI symposium, entitled "Oxygen Free Radicals" was held in Bethesda, Maryland, on 10 to 12 December 1986.

The superoxide radical is the usual free radical produced by cellular oxidation reactions, although its effects can be magnified because superoxide produces other kinds of cell-damaging free radicals and oxidizing agents. The therapies now being tested use two enzymes that normally help to protect cells against superoxide's effects. Superoxide dismutase converts it to hydrogen peroxide, which is then converted by catalase to water and molecular oxygen.

Myron Weisfeldt and his colleagues at Johns Hopkins University School of Medicine have found that treatment with these two enzymes limits the damage to dog hearts that are reperfused with blood after a period of deprivation. In these experiments, the investigators first clamp off one of the coronary arteries. Then, after 90 minutes, they restore the blood flow and at the same time administer superoxide dismutase and catalase into the coronary artery. This reduces the area of killed tissue by about one-third in the enzyme-treated animals compared with the nontreated control animals, Weisfeldt says. The Johns Hopkins workers find that the enzymes decrease the free radical concentrations in the treated dog hearts.

Weisfeldt plans to begin preliminary clinical trials of superoxide dismutase and catalase within the next 6 months in human heart attack patients who undergo reperfusion therapy. Other investigators, including Bennett Luchesi of the University of Michigan School of Medicine, expect to initiate similar trials.

Nevertheless, Eugene Braunwald of Harvard Medical School sounds a note of caution. He points out that the animal models in which the enzymes have been tested so far are significantly different from the situations of human heart attack patients. In the animals, the blood flow to a portion of the heart is stopped and restarted suddenly. For a brief period, the reflow of oxygenated blood is even greater than normal. This combination, oxygen deprivation followed by an excess, is ideal for free radical generation and may exaggerate the problem in the animal models.