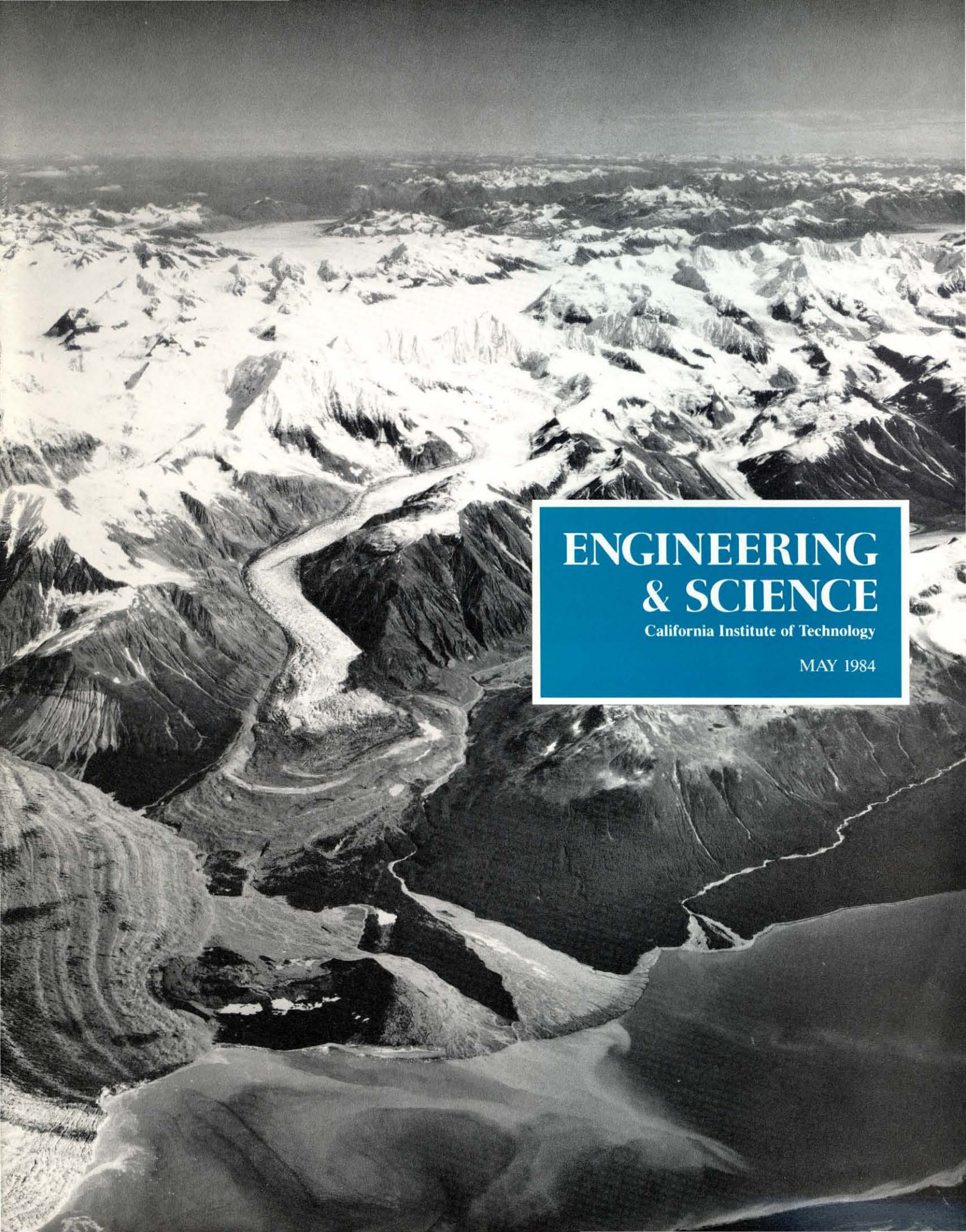
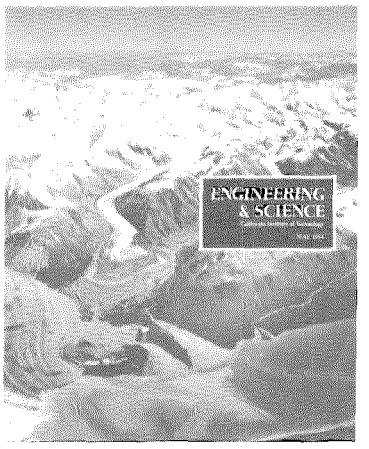


ENGINEERING & SCIENCE

California Institute of Technology

MAY 1984

In This Issue



Galloping Glacier

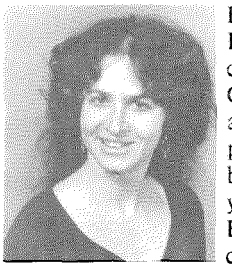
On the cover — view from above of Alaska's Variegated Glacier (the elbow-shaped ribbon just left of center) with the St. Elias Range in the background. The picture was taken last August after the glacier's surge sent it "galloping" forward, chopping it up into the deep crevasses visible even from the air.

The view for someone camped *on* the glacier was still more impressive — and more than slightly harrowing. But the surge provided a rare opportunity to study the mechanisms of glacier flow, and since Barclay Kamb had been watching and monitoring Variegated Glacier for 10 years in anticipation of the event, he and his team were not about to be deterred by a few crevasses. Some of their experiences and what they discovered about surging glaciers are described starting on page 6 in "The Surge of an Alaskan Glacier: A Moving Experience."

This is not the first glacier to appear on the cover of *E&S*. The January 1958 and February 1965 issues featured Blue Glacier in Olympic National Park, Washington, on the cover; Kamb was also involved in that project.

In between camping expeditions on glaciers, Kamb has spent a lot of time at Caltech. He received his BS here in 1952 and PhD in 1956. A member of the faculty ever since, he became professor of geology and geophysics in 1963; from 1972 to 1983 he was chairman of the Division of Geological and Planetary Sciences.

Immune Response



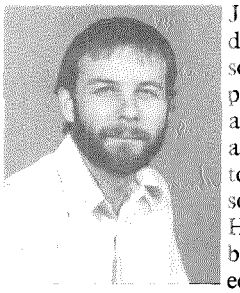
Ellen Rothenberg came to Caltech as assistant professor of biology two years ago. Her research concerns the rich and complex field of cellular immunology. Beginning on page 11, her article, "Innocence and Experience in the Immune System," describes some of this research as well as some background about the major actors in the immune system and the "education" for their roles.

Rothenberg earned her AB in biological sciences summa cum laude from Harvard in 1972 and a PhD in molecular biology from MIT in 1977. In the five intervening years before setting up her lab here, she was a research associate at MIT's Center for Cancer Research, research fellow at the Memorial Sloan-Kettering Cancer Center, and assistant research professor at the Salk Institute for Biological Studies.

The article here is adapted from her Watson lecture in January, at which Ray Owen, professor of biology, emeritus, introduced her as "a congenial colleague, an effective and caring teacher, and a concerned member of the larger Institute community."

Safety Man

Large dams and the possibility of large earthquakes keep uneasy company with each other in many parts of the world, making major losses of life and damage to property an ever-present threat. Fortunately, civil engineers and modern computers are also getting together to bring increasing sophistication into the search for ways to design new dams and to evaluate the safety of existing ones. In "Dams and Earthquake Safety," which begins on page 17,



John Hall discusses some of the problems and new approaches to their solution. Hall has been interested in structural analysis and design for quite a while. One of his early summer jobs, in fact, was as a highway and bridge inspector for the West Virginia Department of Highways. He got his BS in civil engineering from West Virginia University in 1972, his MS from the University of Illinois in 1973, and his PhD from UC Berkeley in 1980. He then came to Caltech as a research fellow in earthquake engineering and became assistant professor of civil engineering in 1983.

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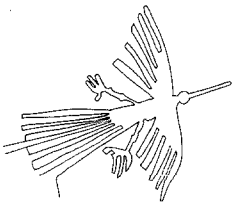

The Travel Program Of

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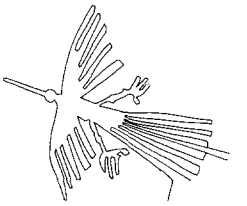

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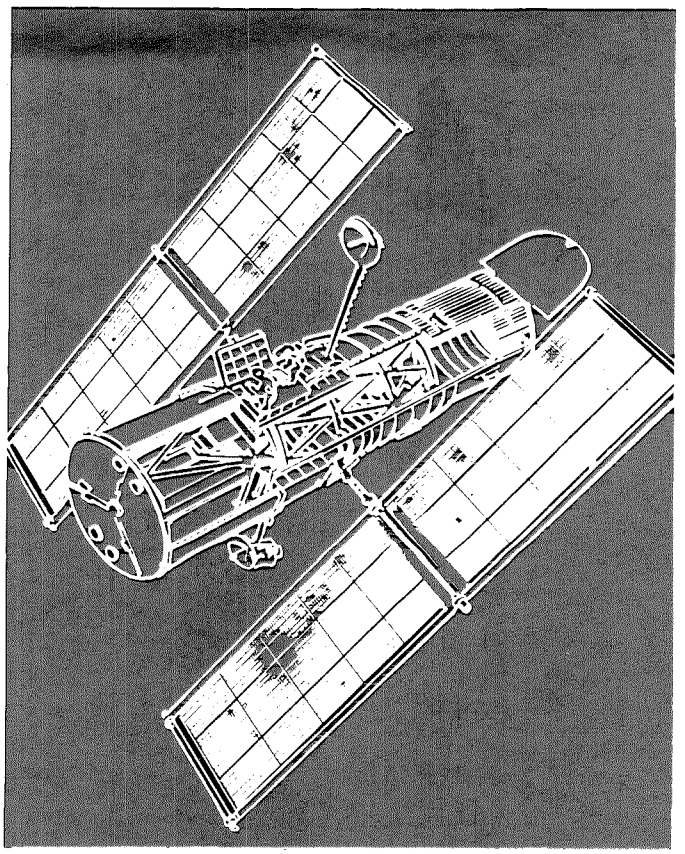

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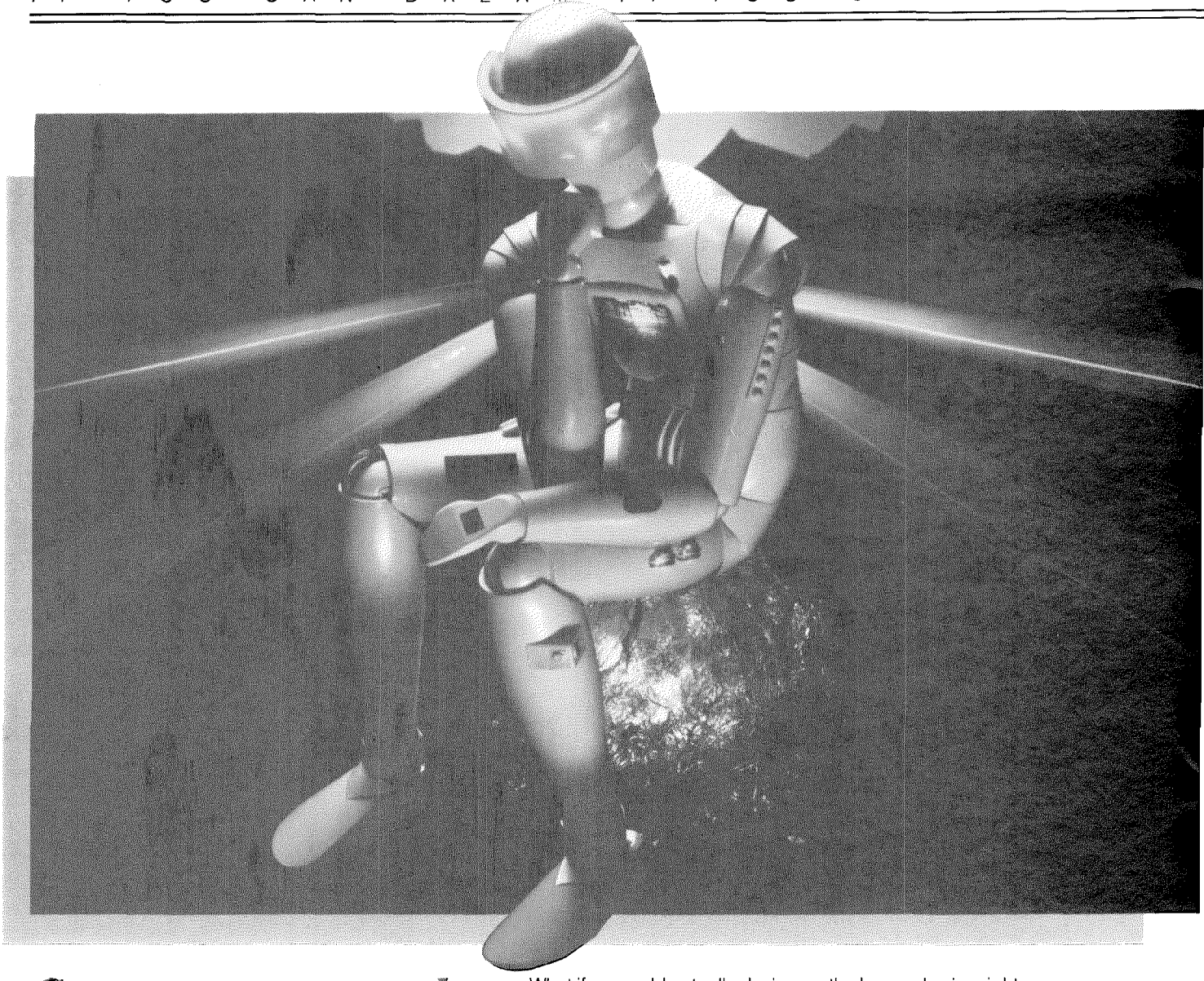

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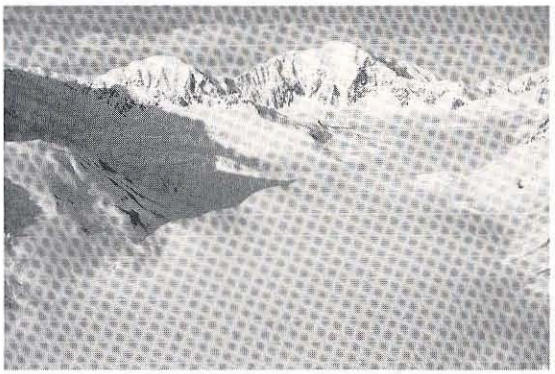
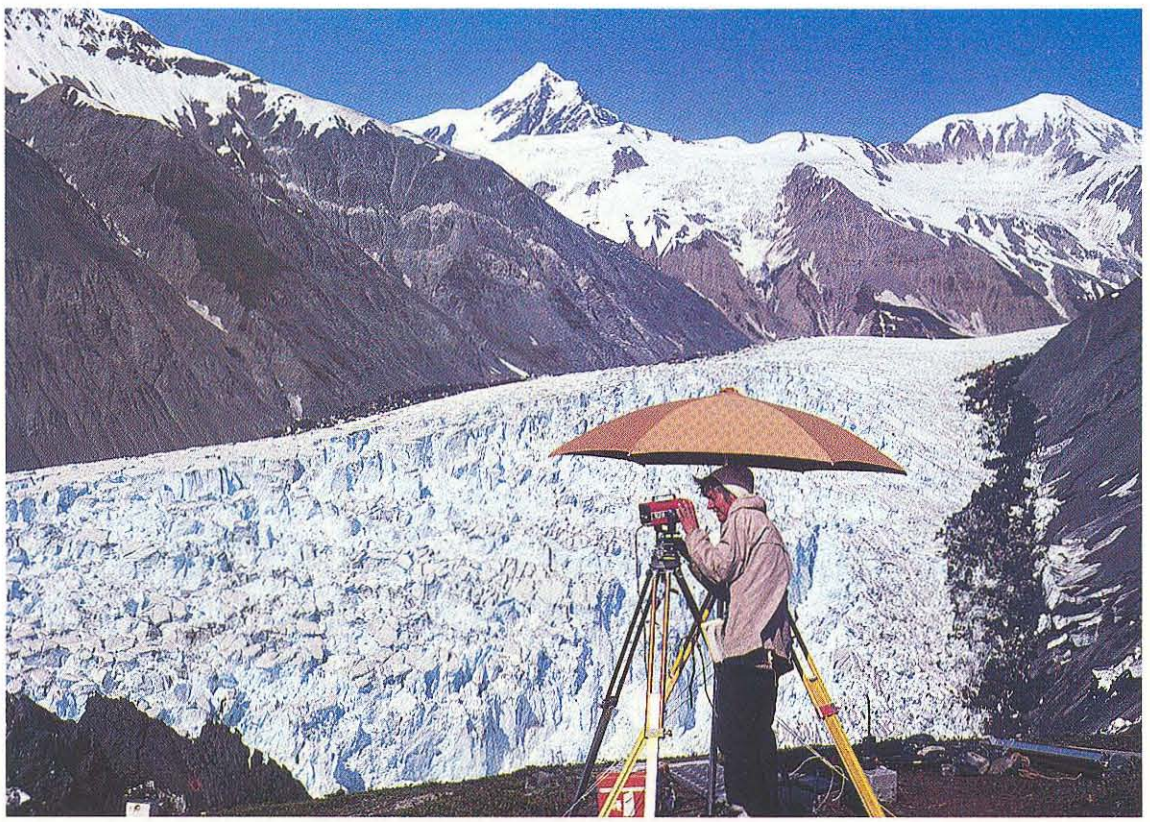


The Surge of an

Variegated Glacier before . . .

. . . and after (or rather during) surge. Almut Iken, who is checking the glacier's forward movement, does not really imagine that she's at the beach; the unbrella protects heat-sensitive surveying equipment from the sun.



GLACIERS DON'T ALWAYS move at a glacial pace. Some of them, after flowing very slowly and steadily for a number of years, suddenly surge forward at up to a hundred times their normal rate, cracking into crevasses as they travel and threatening to overrun an occasional highway, pipeline, Alaskan roadhouse, or scientific camp in their path. Although many glaciers are considered capable of surging, few such "galloping" glaciers have actually been observed since the 1906 discovery of this dramatic phenomenon in the Variegated Glacier in Alaska.

Variegated Glacier surged again in 1947, in 1964-65 — and probably did so in 1926 also (while no one was watching) — a recurrence period of about 20 years. In order to monitor the glacier during its normal period and follow the buildup to the next anticipated surge, research teams, including that of Barclay Kamb, professor of geology and geophysics, have been watching it closely since 1973. Two former Caltech students, Charles Raymond (PhD '69), of the University of Washington, and William Harrison (PhD '66), now at the University of Alaska, have also been camping on the glacier and studying it over the past decade.

While there are many reasons for studying glaciers, including why they're there in the first place, their role as geological agents in modifying the earth, and what they imply for climatic change, Kamb is particularly interested in the

Alaskan Glacier: A Moving Experience

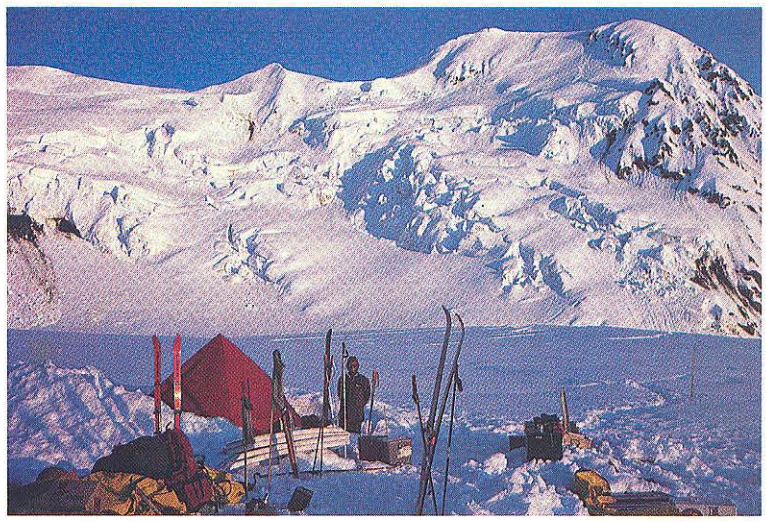
tectonic aspects of glacial flow, the flow phenomenon being in some ways similar to that of mantle convection in the earth's interior. His work has been supported by the National Science Foundation.

Variegated Glacier, which gets its name from the looping bands of different colored rock types in its moraines, is located where the Alaskan panhandle begins, at the head of Yakutat Bay, alongside the much larger Hubbard Glacier, which drains a large part of the St. Elias Range. It's 20 kilometers long and normally flows at a rate of about 0.2 m (meters) per day in the upper part and about 0.1 m per day in the lower glacier. Measurements between 1973 and 1981 showed that during this time the velocity tripled in the upper glacier and doubled in the middle. This slow, steady increase in speed was accompanied by thickening, or increase in elevation, of the ice surface in the upper part of the glacier and thinning downstream.

Normal forward motion of glaciers depends about equally on two different mechanisms — viscous or pseudoviscous creeping of the ice mass internally and basal sliding over its bed. The internal deformation mechanisms are fairly well understood, but what actually goes on at the bottom of the glacier has been more difficult to get at. In 1978 Kamb's group (which has included Hermann Engelhardt of the University of Münster in Germany, formerly a senior research fellow at Caltech, and grad students Melinda Brugman and Keith Echelmeyer, now a postdoc) began a program of drilling boreholes through the ice to study basal sliding, suspected as the prime mover in surging. Drilling the holes required pumping into the ice mass a jet of hot water (under pressure of about 1000 psi), which melted its way to the bottom. Measurements of the tilting of the boreholes provided data on the glacier's movement, which by 1978 was starting to become quite interesting. Short spurts of increased speed were noticeable during the summer.

Kamb called these peaks "mini-surges" because they looked like the beginning of a surge that quite literally didn't get off the ground. In the summers of 1979-1981 his team monitored and documented these mini-surges, four or five of which occurred early each summer. The flow velocity increased rapidly (over an hour or two) to 1 to 3 m per day and then declined back to original speed of 0.4 per day in 10 to 20 hours. This was accompanied by an increase in seismicity (icequakes) and by an abrupt rise in the water level in the boreholes, from normal depths of about 150 m to within 20 to 40 m of the ice surface, strongly implicating basal water pressure in the phenomenon. The hypothesis that the glacier was being temporarily pushed up and "floated" was strengthened by measurements of a simultaneous uplift of a few centimeters in the glacier surface.

The mini-surges propagated down the glacier, and by comparing the spikes in the level of borehole water and the times they occurred at various holes, the researchers determined the



The geologists' campsite on the upper glacier looks idyllic in the relatively calm spring of 1982.

With jets of hot water, boreholes were drilled to the bottom of the glacier to measure its movement and the basal water pressure. Here Barclay Kamb poses amid the pieces of drilling equipment, which occasionally turned temperamental and got stuck in the ice.

speed of the wave as 300-400 m per hour. Mini-surges had not been so closely observed before; Kamb thought they were premonitory to a real surge, which, based on his observations of the early summer activity (and the greater availability of water), he presumed would begin in the summer.

It didn't. In January 1982 the surprising onset was detected by a seismometer. At first no one was sure whether to believe this instrument message from the glacier, but Raymond's on-site measurements of average flow velocities of 2.2 m per day in the upper glacier in March and April forced the geologists to take it seriously. Kamb's group (a total of nine) began arriving toward the end of May. Setting up a scientific camp on an isolated glacier is not the most convenient way to do research. It took 8 helicopter flights from Yakutat to the glacier itself and, in addition, 14 airplane flights to a beach near Hubbard Glacier, and from there 15 more helicopter flights to get all the people and equipment to their camp.

The velocity continued to rise gradually to a crest of 8.7 m per day. Then, on the morning of June 26, the flow velocity suddenly dropped in just a few hours to less than half of what it had built up to. Five major pulses of movement occurred after that, superimposed on a generally decreasing trend of velocity. By the end of July the speed had decreased to 1 m per day, which seemed slow, but was still fast compared to the normal, pre-surge speed.

During this time, until mid-August, Kamb's team plumbed Variegated Glacier for all possible clues to the surge movement. They set up a system of seismometers the length of the glacier, drilled boreholes to measure the glacier's movement and the basal water pressure, and attempted to trace the subglacial flow with injected dye and to install survivable pressure transducers at the bed. Besides not being the easiest places to get to, glaciers are not the easiest places to work, either. Icequakes on the surging glacier every few minutes made sleep difficult. ("This was somewhat unnerving," says Kamb, "especially when crevasses were opening up under the tents.") The process of melting snow for the drilling water was time-consuming; drilling equipment got stuck in the ice, forcing cancellation of the transducer experiment; the dye experiment was inconclusive (and trying to pour 60 lbs. of dye down a borehole was "a very messy business"). But the problems brought insights into better ways to do it next time around.

There was indeed a next time. The surge began to build up again in the upper glacier the following October and continued through the winter and spring of 1983. A seismometer and automatic cameras recorded the glacier's movement, and the scientists visited periodically over the winter. The speed gradually increased over the winter, reaching about 5 m per day in the middle of February and about 10 m per day by early May. Kamb and Echelmeyer spent several days on the glacier in February and returned with their team on May 5.

In May and June 1983 the surge front propagated (as a kinematic-type wave, Kamb has concluded) down into the lower glacier; the previous year's surge had been limited to the upper glacier. Ahead of the sharply defined surge front the glacier moved at its normal slow pace; behind it the flow velocity was high — dramatically high, in fact, reaching flow velocities of from 40 to 60 m per day in the lower glacier during June. (The highest recorded was 65 m per day for two hours on June 9.) As the surge front moved through it, the ice in the lower glacier grew up to 100 m thicker, while in the upper glacier it dropped by as much as 50 m from its pre-surge thickness. Large, complex oscillations in flow speed propagated down the glacier at speeds of 600 m per hour.

The strain of the surge now cracked the surface of the glacier into a vast jumble of crevasses, making camping on top of it "very exciting," according to Kamb. In June, as they were forced to abandon one camp that was being sliced apart by crevasses, the rescuing helicopter pilot remarked, "It looks like nature has eaten your camp." Subsequently the researchers located their camp below the surge front, where life was perhaps less fragmented but no less exciting, as the huge wall of ice, the surge front, swept toward them. Much of their

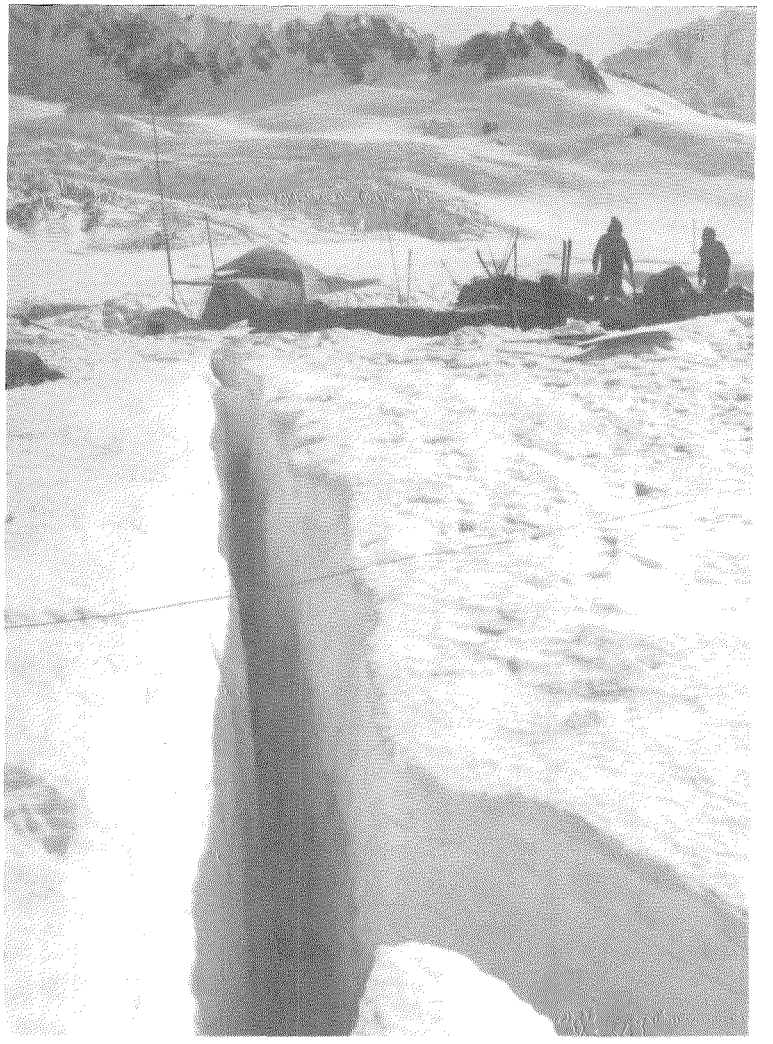
work on the surging glacier was carried out from a helicopter, which sometimes could find a flat piece of ice large enough to land on. Other times their ice-movement markers had to be set from the craft as it hovered over the seracs (pinnacles of ice). Many of the markers disappeared into the crevasses as the glacier surface broke up.

Suddenly in the afternoon and evening of July 4, the surge stopped abruptly. In just a few hours, the glacier's speed dropped to a quarter of what it had been; by July 26 it was down to essentially its pre-surge velocity, even less in the upper glacier, where the ice had been thinned by the surge, and somewhat more in the lower glacier where it was thickened.

What was, then, actually happening at the glacier bed that had precipitated the surge motion and was now terminating it? Kamb's team once again set about trying to find out. Measuring the shape of boreholes (how far they inclined over several days) demonstrated what had previously been assumed but never actually observed — that essentially all of the surge movement is due to basal sliding, while the internal motion was roughly the same as normal. Boreholes also provided measurements of the water level and thereby the water pressure at the bottom of the glacier. Kamb and his group found that during the surge the basal water pressure was consistently within 3 to 5 bars of the pressure of the overlying ice at the bed. (Pressures are normally 8 and sometimes as low as 16 bars below the overburden pressure.) The surge's sudden stop coincided with lowering of the water pressure. This was very reminiscent of what had been observed earlier in the mini-surges, where peaks of movement coincided with peaks in water pressure.

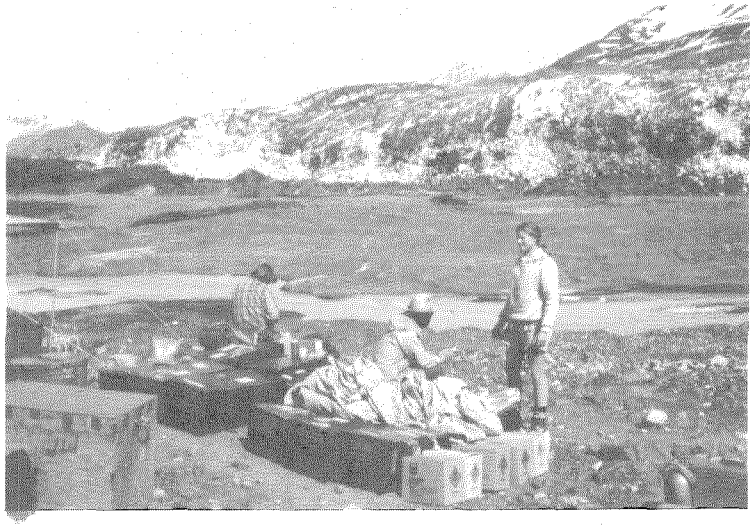
The emergence of a lake at the edge of the lower glacier implicated basal water pressure still further. Its water welled up from the basal water system through a large crevasse 100 m in from the glacier's edge and then flowed outward to the margin. The lake emptied literally overnight on July 4-5, coinciding with the abrupt end of the surge.

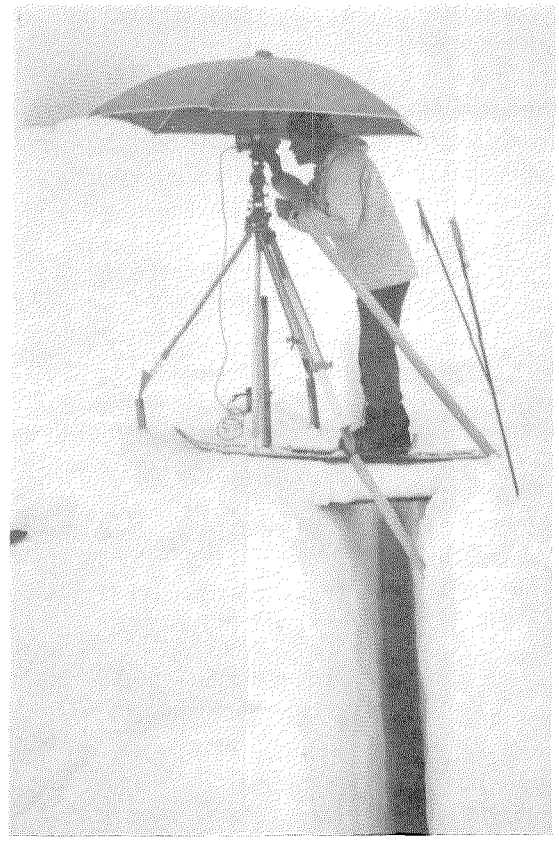
One of the most striking observations was a correlation between marked slowdowns in the surge speed and large floods in the subglacial river emerging from the glacier at its terminus. When the surge ended, a particularly spectacular flood of water emerged. Turbid water gushed and spurted out of cracks and crevasses along the leading edge of the surge front. Kamb thinks this represents the release of stored water from under the glacier, the same stored water



A crevasse advances ominously on the camp before the campsite was moved.

Everyone practices crevasse rescue — just in case.





(Above) With the camp safely relocated on the lower glacier, these members of the research team appear oblivious to the surge front, which looks like a dirty wave in the background, bearing down on them at a speed of up to 60 meters per day. (Right) When a crevasse interrupted his surveying, Kamb improvised a bridge and continued his work.

that was "levitating" the glacier off its bed during the surge.

To track the subglacial water flow, Kamb's group again injected tracer dye through a borehole to the glacier bed and determined how long it took to reach the main outflow stream down at the glacier terminus. They injected the dye once in June during the surge and again in July after it ended. Results clearly demonstrated a dramatically slower water flow during surge. In the first instance the dye took a mean transit time of 100 hours, corresponding to an average speed of 0.02 m per second; after the surge, it went through in 4 hours at an average speed of 0.7 m per second. *Something* was happening during the surge to retain water under the glacier.

On the basis of their discoveries of the behavior of subglacial water and measurements of uplifts and drops in the surface of the glacier during speedup and slowdown of the surge, Kamb thinks the surge mechanism involves the opening and closing of cavities under the glacier — cavities that fill and retain water during surge and that close and release water when the surge ends. These cavities are created, he believes, as increased basal water pressure causes the glacier to slide over its bed with increasing speed. He sees the cavities as forming an interconnected system of small conduits in a complex network that extends far and wide at the bed of the glacier and is kept open by the high water pressure. In contrast to this, in the glacier under non-surging conditions, as in "normal" glaciers, melt water flows at the bed of the glacier through a single large tunnel, or at most a few large tunnels.

In relating the behavior of the basal water pressure to the mechanism of the surge, Kamb theorizes that a surge is caused when water pressure at the bed approaches within 3 to 5 bars the pressure of the overlying ice, causing it to begin sliding rapidly and causing many cavities to open. If the water pressure exceeded that of the ice, which sometimes does occur in mini-surges, the glacier would literally float off its bed and slide at an increasing rate without stopping. Mini-surges are of such short duration, however, that the high water pressure doesn't have a chance to spread over the whole bed and precipitate an unstoppable slide.

Variegated Glacier's 1983 surge provided a unique opportunity to investigate the mechanisms of glacial flow. But there are still unsolved mysteries. For example, what causes the change in the basal water system that sets the glacier into surge in the first place? What is the switchover process that terminates the surge in summer? Where does the water come from, and how does it become available first in midwinter, when there is no melting to furnish it? And how does the surge mechanism control the propagation of the surge front and of the still unexplained, complex oscillations that accompanied the surge?

The glacier did not surge again in the winter of 1984. But the unanswered questions will continue to draw Kamb and other geologists back to Variegated Glacier even in less "exciting" times. □ — *JD*

Innocence and Experience in the Immune System

by Ellen Rothenberg

THE IMMUNE SYSTEM is clearly useful as a major line of defense against disease, but it is also an extremely interesting biological system. From birth till death, contacts with our environment make an indelible impact on the cells and molecules that comprise it. This must involve many separate reactions that we will need to understand in order to piece together the mechanisms that allow the system to respond in such a flexible and interesting way.

Many different types of cells make up the immune system. Among the major actors is the macrophage, an important accessory cell, which in certain circumstances also acts as an ultimate effector cell. A macrophage doesn't "know" the difference between one component of its environment and another, but it does "know" the difference between being in an activated state and being in a non-activated state. When they become active, macrophages engulf bacteria and foreign particles and destroy them by digestive mechanisms. Macrophages and related cells are the terrorists at sites of inflammation.

But the most interesting cells are the lymphocytes. There are two profoundly different classes of these cells — T and B lymphocytes — which look similar but vary both in their differentiation histories and in the functions that they perform. The T cells go through part of their "education" (or maturation) in an organ called the thymus, and are responsible for a complex of responses called cell-mediated immune responses. These involve direct killing of virus-infected cells or foreign cells as well as a host of regulatory activities. The B cells, on the other hand, never pass through the thymus but are responsible for secreting the antibodies that are a major component of the immune response.

B cells and T cells share an unusual property in the body — each cell is a unique individual. Each B or T cell has a unique cell-surface receptor encoded by that cell's chromosomes, which will fit only one type of structure it could encounter in the environment. So each cell is committed to recognize only one type of target structure, or antigen, binding it to its cell-surface receptor, and thereby becoming activated. B and T cells start out with the same genetic information as every other cell in the body. Each eventually commits itself to make a particular receptor by shuffling around bits of the DNA in its chromosomes that encode the receptor structure. Two segments out of a large range of possible combinations are brought together to form a complete receptor gene, in a way that is unique to each cell. This creates an immune system of tremendous diversity. In any human being or other animal, different B or T lymphocytes can recognize up to 10 million different antigenic structures.

After a B lymphocyte has been activated by binding the correct antigen, it starts to secrete a large amount of its cell surface receptor molecule. This is an antibody, or immunoglobulin molecule, which serves to sequester the antigen or bind to it and make it an appetizing target for the macrophages to eat.

T cells, on the other hand, don't have antibody molecules for their receptors, and different kinds of T cells work in different ways. Certain T cells are specialized for the function of killing other cells. To do this they bind tightly to the target cell, which they have recognized as being foreign through the membrane receptors. Under the correct conditions, the killer T cell then delivers a lethal hit. It dumps vicious little macromolecular complexes onto the surface of the target cell, special protein assemblies that punch holes in its membrane. And the killer T cell then disengages and goes on its way (perhaps to kill again), while the target cell is punctured and destroyed. The killer T cell's functional role depends on having both specific receptors to recognize its target cells and the correct kind of molecules to make the lytic (attack) complexes that perforate the membrane.

There are other kinds of T lymphocytes — suppressor T cells and helper T cells, which perform somewhat more complicated and equally crucial functions in the immune system. The mode of action of suppressor T cells is quite controversial, but much more is known about helper T cells. These are T lymphocytes that respond to the binding of antigens to their cell-surface receptors by secreting a series of

hormones that stimulate growth and differentiation.

Some of these hormones act on non-lymphoid cells, specifically macrophages and other cells related to them, by making them more active. A virus-infected cell can be sitting among a bunch of macrophages that are not aware that there is anything wrong, until a helper T cell comes along and recognizes the virus-infected cell as being foreign by binding to it. The T cell then secretes activation factors that induce the macrophage to swell up and start to secrete lytic enzymes and active oxygen radicals into the environment of the infected cell. These are extremely toxic to it and cause it to die. There is, however, no specificity to the activation of this macrophage; that is, at the same time that it's been induced to kill the virus-infected cell, it will also kill anything else in the general area. A lot of the pus that collects at the site of an inflammation is due to the activation of macrophages, which kill many of the body's perfectly normal cells that just happened to be innocent bystanders. The idea is that it's better to get rid of the infected cells even at the cost of some self components. This is also the kind of action involved in a poison ivy or poison oak response; a helper T cell recognizes the oil from the poison ivy as an evil substance and activates the local tissue macrophages to inflame the skin.

But the most interesting function of helper T cells has to do with the specific growth factors that the activated helper T cells secrete that act only on other T or B lymphocytes. Even if a B cell or a killer T cell recognizes another cell as being diseased, if it hasn't previously been activated, nothing much may happen. It can just sit there. But if a helper T cell in the same vicinity is also activated by binding to an antigen, perhaps on the same diseased cell, this helper T cell secretes growth and differentiation factors for both B cells and for other T cells, which cause them to proliferate and to become active. The B cells start to secrete antibody at a high rate, the killer T cells may become more energetic in the attack process, and the numbers of both will increase. While the helper T cell isn't directly secreting antibody or killing targets, its role is very important in making these other cells express their functional potentials.

Obviously, it's important for the helper T cells to activate the other lymphocytes to become effective killers or antibody secreters. But this differentiation from a resting state to an active state is not the only important change that the helper T cell causes. When a lymphocyte is activated by binding antigen and receiving an activation factor from a helper T cell, it will kill or make antibody for a certain length of time but will eventually die. Often its lifetime is fairly short. Imagine that a T lymphocyte with a unique target specificity is stimulated by an influx of antigen into its territory. If it simply does its best to kill the antigenic target, ultimately that lymphocyte is going to be exhausted. And there could still be a lot of antigen around. Worse, there are only a finite number of lymphocytes in the body but as many as 10 million different kinds. With only one in 10 million lymphocytes able to attack any particular antigen, this would be a pretty feeble way to defend the body against anything.

But we know that this doesn't happen. The key is the proliferation that is stimulated by the helper T cell's growth hormones. When the lymphocyte comes in contact with the antigen, it's not only stimulated to deal with the antigen directly but also to reproduce itself into a large clone — hundreds of thousands of cells. These can then kill off all the antigen; moreover, there will be excess cells, or memory cells, with that particular antigen-binding specificity left over. So the next time that that same antigen comes into the body, it will be very easy to deal with because there will be an increased number of cells primed to take on that particular challenge. This is why people who survived the flu epidemic of 1918 are still immune to certain strains of flu today that are related to the 1918 strain. These people have memory cells that have survived all this time and are still present in sufficiently amplified numbers to protect the body against a new bout of the disease. Furthermore, if it weren't for this memory phenomenon, experimentalists would have no way of even knowing that there is specificity in the immune system. The tangible evidence that recognition is specific is the fact that after the first exposure to an antigen, that antigen and no other becomes easier to eliminate.

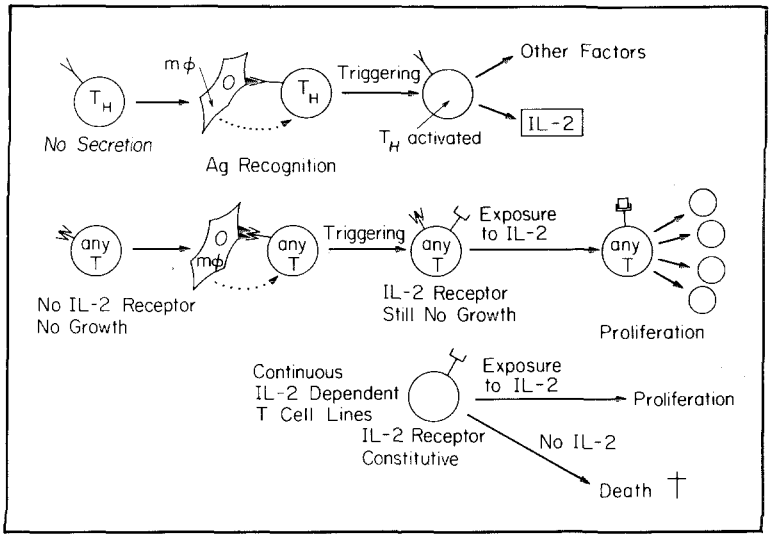
The proliferation mechanism is crucial, so the helper T cells are crucial, and any state of the body in which helper T cells are paralyzed or destroyed is going to have a catastrophic effect on immune responses. It means that you go back to a situation where you don't have enough cells to deal with a challenge. This drastic depletion of helper T cells is characteristic of the extreme depression of immune responsiveness in AIDS (acquired immune deficiency syndrome) victims.

We know most about the mechanism of action of the growth hormone that works specifi-

cally on T lymphocytes — a molecule called interleukin-2 (IL2). The human IL2 molecule is 153 amino acids long. In the case of the mouse, which is my lab's experimental system, it's a somewhat longer amino acid chain. There is nothing hypothetical about this hormone. Human IL2 has been purified to homogeneity, the gene for it cloned, and the cloned gene put into bacteria, and the IL2 that the bacteria make from the cloned gene works. It's now possible to produce virtually unlimited quantities of this hormone.

IL2 acts only on stimulated T cells that have bound antigen; it does not act on resting T cells that have not yet seen their own proper antigen; and it doesn't act at all on non-T cells. So in the presence of infinite amounts of IL2 the only cells that will be able to respond by growing are T lymphocytes that have seen their correct antigen. IL2 itself, however, has no antigen-binding activity at all. It also has no specificity. One helper T cell that is stimulated with one antigen will make IL2 that can work on another T cell that was stimulated by whatever antigen was appropriate for it. It does not need to be the same as the first antigen.

When a helper T cell is circulating innocently around in the body, it does not secrete any IL2. It has a receptor on it that will allow it to bind its proper antigen if and when that antigen enters the body. This may be never. If and only if it binds the correct antigen, the helper T cell is triggered and will then start to secrete IL2 as well as other growth and differentiation factors. As for responsiveness, all T cells, killers and helpers, have the *potential* of responding to IL2, but only when activated. They have antigen receptors, but until they are activated, they do not have IL2 receptors, so they can't proliferate even if there is an ocean of IL2 around them. Once a T cell is triggered by contact with its correct antigen, it also acquires the receptor for IL2, a specific polypeptide that binds IL2, which is now expressed on the surface of that cell. Still, although it has bound antigen, it cannot proliferate unless there is a helper T cell around to make the IL2 it needs. This two-cell cooperative system can be thought of as a sort of fail-safe mechanism in the immune system, so that if either a helper or killer T cell should mistakenly get activated by something that wasn't really a threat to the organism, there wouldn't necessarily be an explosive proliferative response. An immune response won't begin in earnest unless at least two types of cells in the same place both encounter some sort of challenge.



IL2 is not antigen-specific but promotes antigen-specific growth. *First line: a helper T cell (T_H) only* secretes *IL2 when it has encountered its own correct antigen, presented by a macrophage (mφ). Second line: all T cells can only* bind *IL2 and respond to it as a consequence of recognizing their own particular target antigens. Only after expressing a receptor for IL2 and binding IL2 to their surfaces can they proliferate. Third line: some tissue culture lines of T cells are frozen into a perpetually IL2-responsive state. They no longer need prior contact with antigen to allow them to bind IL2. When starved for IL2, however, they die.*

Using a molecule like IL2 that doesn't discriminate between one antigen and another, the immune response only maintains its specificity because of the geography in which the response takes place. Immune responses don't occur just floating around in the blood — or in tissue culture — as we are sometimes led to believe. Rather, they take place in specialized structures, one of the most important of which is the lymph node. There are many of these nodes distributed all around the body. Their role is to take in possibly antigenic molecules from tissue fluid and to filter them, in a kind of countercurrent distribution, past migrating lymphocytes that are circulating in search of their correct antigens. The lymphocytes migrate into the lymph nodes through blood vessels; once they're in the node they squeeze themselves out between the cells that form the blood vessel wall, and can then encounter antigens in the node. A correct antigen will trigger the cell and activate it; if that antigen isn't present, the cell just continues on its way through another duct and ultimately gets circulated back into the blood.

The lymphocytes move through the node, but in a rush-hour traffic pattern — all jammed together. This wouldn't be surprising in an organ such as the liver where all the cells are stationary, sitting together like tiles on a floor. But the T and B cells are actually *circulating* in very tight contact. Within this tight pack the T cells tend to be all clustered together and the B cells likewise, although there are some key T cells in the B area and vice versa. This means that when a single cell begins to produce a growth hormone such as IL2, it won't need to diffuse very far in order to find another T cell that may be stimulated by the same antigen. The growth hormone doesn't need to dilute itself through-

out the whole circulatory system. So, in a lymph node in which multiple lymphocytes are all being activated by a foreign antigen, it's likely that their responses are all against the same biological challenge. A non-antigen-specific mediator like IL2 can pump up the response to maximum strength. If a person is suffering from a helper T-cell deficiency and has a problem dealing with an immunological challenge, one of the ways that you could amplify his T-cell response would be to inject a high dose of IL2 locally, which would allow any cells that recognized the antigen to start proliferating.

How do cells get to the point where they can carry out these responses in such specific and interlacing ways? To understand this at the level that we would like, we need to know what special kinds of molecules are made in the differentiation of these lymphocytes from their precursor cells. Lymphocytes are, of course, derived from the same fertilized egg that gives rise to all the other tissues in the body, and we'd like to know which genes are turned on to make lymphocytes unique. We would also like to know what signals from the body drive their differentiation in particular directions, and what triggers turning on first one gene and then another.

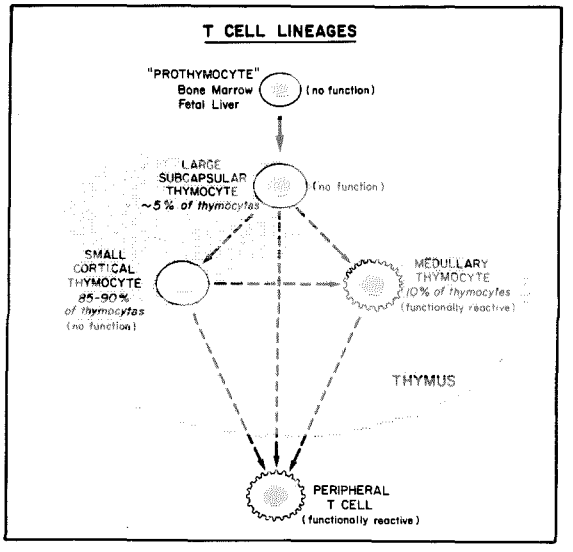
We know a lot about the kinds of molecules that B lymphocytes use as receptors — the antibody molecules, or immunoglobulins — and about the process of shuffling DNA segments to produce them. But we don't know very much about the particular domains of the body in which B-cell differentiation takes place. B cells at early stages in their differentiation show that they are B cells by rearranging their DNA, but we know almost nothing about the sorts of cell types involved in stimulating them to do this. We also know very little about whether there are different lineages of B cells or if all B cells go through an identical early education.

With T cells the situation is almost exactly the reverse. Just in the last six months we have learned what sort of molecule is used by the T cell for its receptor. And although it is likely that it also undergoes shuffling in its genes like the B cell receptor, we don't know very much about the details of that process. What we do know, though, is quite a bit more about the cell biology of how T cells become T cells. The distinguishing characteristic of T cells is that they're educated in the thymus — a white, two-lobed organ just above the heart. It's large before puberty, when it begins to shrink in size. Then it maintains itself at a reduced size and perhaps reduced capacity throughout most of life except during serious illness.

The thymus seems to be a necessary place for the precursors of T cells to mature into effective cells. These precursors come from the bone marrow (at least they do after birth) and enter the thymus. A fraction of these cells are then exported after they have been processed by the thymus to give rise to mature helper and killer T cells.

The thymus has two roles. One is as a site for differentiation and expansion of the total T-cell population. The cells going into the thymus don't look or act like T cells, but the cells coming out do. The other role, which has been possible to approach only by indirect experiment, is as the organ in which T lymphocytes apparently learn to discriminate self from non-self. This is, of course, a key aspect of the immune system, because if your lymphocytes don't know the difference between "self" parts of your body and foreign molecules, then they will attack your body. This is presumably the type of breakdown that occurs in autoimmune diseases. We know that at least a large part of T cells' education as to what constitutes self and non-self is dictated by the *particular* thymus in which they differentiate. Transplantation experiments in mice have shown that T-cell precursors differentiated through a foreign thymus come out thinking that the foreign tissue is self and not foreign.

When we study what actually goes on in the thymus when this education is taking place, we find cellular events that are practically melodramatic. A small fraction of cells in the thymus divide every eight hours — about as fast as any mammalian cell. They barely have a chance to finish copying all their DNA to make genetic information for their new daughter cells before



T lymphocytes differentiate in the thymus. *Precursor cells must migrate to the thymus before they can mature into functionally reactive T cells. The cells in the thymus fall into at least three groups. The large thymocytes in the subcapsular region and elsewhere are the only dividing cells, and the other cell classes are their descendants. The exact pathways of differentiation are still controversial (broken arrows). Our results suggest that a separate class of precursors exists for many of the medullary cells.*

they split and start again. They have almost no time for housekeeping functions at all. Most of the cells generated by this very hasty cell division accumulate in the thymus, making up the majority of cells in the thymus, and these progeny cells don't divide. They sit there in a part of the thymus called the cortex. What's interesting about these cells is that they have an almost negligible survival rate. The vast majority of the daughters of the rapidly dividing cells are killed; less than 5 percent of them survive to be exported. On the average 30 or 35 percent of them are destroyed every day, and the entire population is turned over every three or four days. There's an anomaly here: About 40 percent of all the cells in the immune system are the ones that are churned out as cortical thymocytes, yet most are never going to see any useful role in the body. We don't know if any survive.

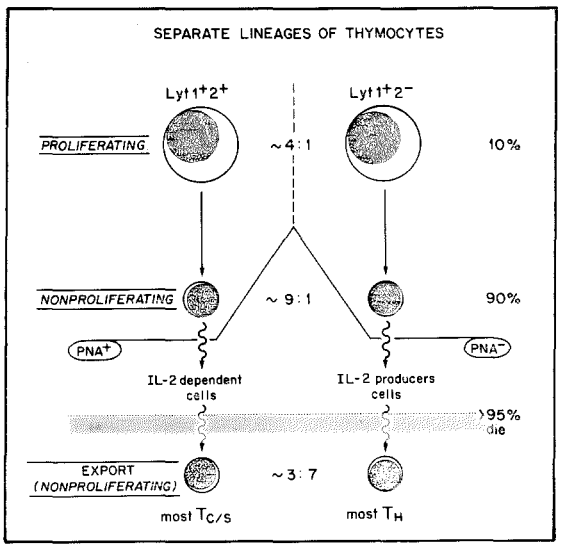
There is another population in the thymus, the medullary thymocytes, which make up about 10 percent of the total cells in the thymus. These do not divide as frequently as the large cells in the cortex, although perhaps a few of them do; it's still controversial. These cells seem to have a longer average lifetime in the thymus than cortical cells. They appear to be very similar to the peripheral T cells, and what we don't know is whether they are exported to become the precursors of the T cells that defend the body from disease, or whether this is just a reserve population that renews itself and stays in the thymus.

These, then, are the cells that may be on their way to becoming T cells. The questions we need to answer are: 1. Where in all this cell division and cell death is the differentiation taking place that allows a cell to act as a functional part of the immune system? 2. At what stage are these cells being educated to know the difference between self and non-self?

Over the last four or five years my lab has concentrated on learning to separate the different cell populations in the thymus and to find ways of telling them apart in terms of the way they look and behave. We are working up to the point where we can then assay each of these individual fractions for the changes in gene activity that might have to do with cellular function. To separate the different types of thymocytes from each other, we take advantage of a number of different physical properties. We can separate them on the basis of cell division behavior, because the dividing cells as a rule are much larger than the non-dividing cells, regardless of which compartment of the thymus they're in. Also, cells in different compartments of the thymus decorate their cell surface molecules with different complex sugars. Putting sugars on cell surface molecules is characteristic of all cells in the body, but it's unique that within this particular organ the cells in the cortex have different sugar structures on their surfaces than cells in the medulla. We can use proteins called lectins, which bind one type of sugar and not another, as a kind of handle to pull out and separate the cortical from the medullary cells. We can also use antibodies which react with different cells, either different lineages of T cells or T cells in various stages of their differentiation within a particular lineage. Like the lectins, the antibodies can be used as a handle to pull out the cells. Or we can put a fluorescent dye on the antibodies and bind them to the cells, which can then be sorted out using a fluorescence-activated cell sorter (*E&S*, March 1983).

We can resolve a lot of differences both among the molecules that these different types of thymocytes express on their surfaces and among the kinds of proteins that the cells are actively engaged in making at a particular moment. We don't know yet what these properties have to do with immune function, but my lab has just entered a new phase of looking at the functional properties of the developing T cells. We've been concentrating on the helper lineage of cells rather than the killer cells, because the helper T cells have the ability to make IL2 under appropriate stimulation — a property that we think we can follow fairly well.

In order to look at the ability of cells to make IL2, we need to get the cells to respond as if they had encountered their own specific target antigens. To activate all the helper cells, whatever antigens they recognize, we trick them



Major classes of thymocytes. *Left: Cortical thymocytes. Right: Pre-helper T cells, the majority class in the medulla. Note that while the cells in the thymus are mainly cortical type, most of the cells that survive to be exported are T_H type. It is not known, in fact, whether any of the cortical-type cells survive, for the mature cytotoxic and suppressor T cells ($T_{C/S}$) could come from separate precursors (not shown). Our results show that even primitive proliferating cells of the Lyt2$^-$ lineage can make the mature T_H product IL2.*

with a combination of well-defined compounds. One is the protein concanavalin A, which probably binds to their antigen receptors as well as other cell surface molecules. The other is a molecule called TPA (tetradecanoyl phorbol acetate) that provides an additional signal. The combination seems to mimic contact with each cell's particular antigen. So we can isolate thymocytes, or any other T-cell population to be tested, and put them in culture with concanavalin A and TPA overnight. It's not a long incubation; the cells that we treat are all still there in the culture at the end. We can then determine whether or not these cells make IL2 by asking whether or not the molecules that they secreted into their tissue culture medium are now capable of maintaining the growth of an indicator cell line, a cultured T-cell line that is frozen in an IL2-dependent state. These indicator cells always respond to IL2 and cannot survive without it. If you allow them to use up all the IL2 in their culture medium, they need to have a new dose within 24 hours or they die. So we can easily measure the difference between indicator cells that are making DNA to prepare for cell division, because they got IL2, and indicator cells that are dead, because they didn't get any. The assay is sensitive enough so that we can measure over a thousandfold range the concentrations of IL2 that thymus cells might have produced.

By applying these separating and assay techniques we have found that the cells of the different thymus populations make quite different amounts of IL2. The majority cell type in the thymus — the small cortical cells — make negligible amounts of IL2 per cell under these conditions, whereas the whole category of cells that we believe to be in the medulla produce quite a lot. The dividing cells in the cortex, the main precursor cells in the thymus, do not appear to make the hormone. But among cells that appear to be in the medulla, even the cells that are still actively dividing — one of the rarest cell types in the thymus — are very good at making IL2.

We find this result extremely interesting for the following reasons. The cells from the medulla that have *finished* their cell division in the thymus are what we would expect to be typical mature T cells. These would be cells that the thymus has finished educating, and they're ready to be exported. But this can't be said for the cells that are still dividing in the thymus. They are cells that are still being acted on by the hormones they come in contact with in the thymus, still being forced to divide, even though they're already competent as helper T cells in terms of their ability to make IL2. This finding, then, allows us to make a separation between the role of the thymus as a differentiation organ and its role as a cell division organ. The purpose of this rapid cell division cannot simply be to prepare the cells to become functional. They're dividing although they already seem functional, since they can make IL2. We think that the cell division and the death that attends many of the products of the cell division could be the means by which the thymus selects for cells that recognize the correct thing as self. Even though these large cells are already partially competent at least as helpers, they may still be acted upon by the thymus to fine-tune their specificity for self versus non-self.

This is a hopeful and speculative view of what these cells might mean. We still have to find out exactly what they are, exactly where they are, and when they arise in the development of an animal. My lab is now focusing on the cloning of the gene for IL2 in the mouse. We are also starting work on cloning the receptor gene for IL2, which will allow us to do a parallel study on when the cells first become responsive to IL2. We hope to use these cloned genes as probes to track down individual cells in the thymus glands of very young animals, before there are any mature T-cells, to see whether any of these genes are expressed. We can also ask whether these genes are turned on in the precursors of T cells even before they leave the bone marrow to come to the thymus.

Among my colleagues in the lab are research fellow Barry Caplan, who brought the functional assays into the lab, and research fellow Jim Lugo and graduate student Tom Novak, who are working on the isolation of the IL2 receptor gene. Rochelle Sailor, my lab manager and senior research assistant, has been instrumental in bringing in the most recent nucleic acid technology and is helping on cloning the IL2 gene. Previously, in my work at the Salk Institute, Dennis Triglia helped with much of the characterization of the types of cells in the thymus.

All of our studies are aimed at trying to understand something about the education of those cells that become helper T cells and those that respond to the IL2 that helper T cells secrete. By understanding the kinds of influences these cells are exposed to during their education, we might begin to have some clues as to how these mechanisms go awry to create immune deficiency and autoimmune conditions. □

Dams and Earthquake Safety

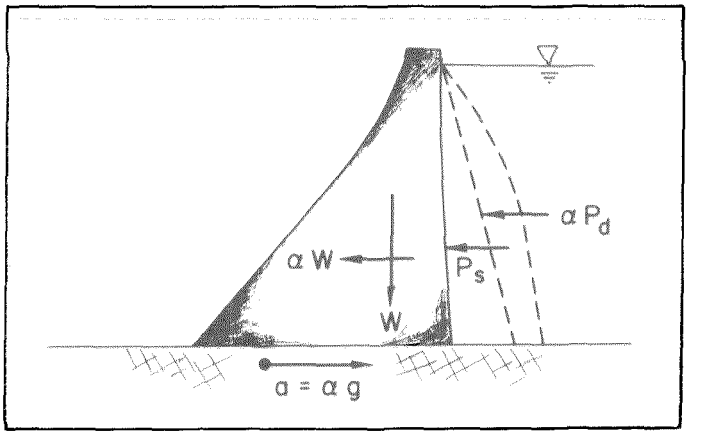
by John F. Hall

WILL DAMS SURVIVE severe earthquake shaking? Finding an answer to this question is one of the important goals for many engineers today. How are they going about including earthquake shaking as an element in dam design and in the evaluation of existing dams? In the past, earthquake effects may have been treated too lightly in dam design. Are such dams safe, and how have they fared in previous earthquakes? There are three major types of dams — embankment and concrete gravity and concrete arch — but this discussion will be limited to some of the findings about the two concrete types.

Concrete gravity dam design was, and still is, based on two-dimensional idealizations (as illustrated in the figure at the right) because gravity dams, which are generally located in wide river valleys, are long and nearly uniform in cross section. Water loading from the reservoir behind the dam seeks to overturn or slide the dam downstream; and the dam's own weight resists this action. A proper choice of dam cross section provides stability. In addition, since concrete is weak in tension and since no steel reinforcing is employed, engineers equated the presence of tensile stresses with failure. If their computations showed tensile stress at any point, they redesigned the cross section. Stress analysis was performed by treating the dam cross section as a beam of variable thickness cantilevering from the valley floor.

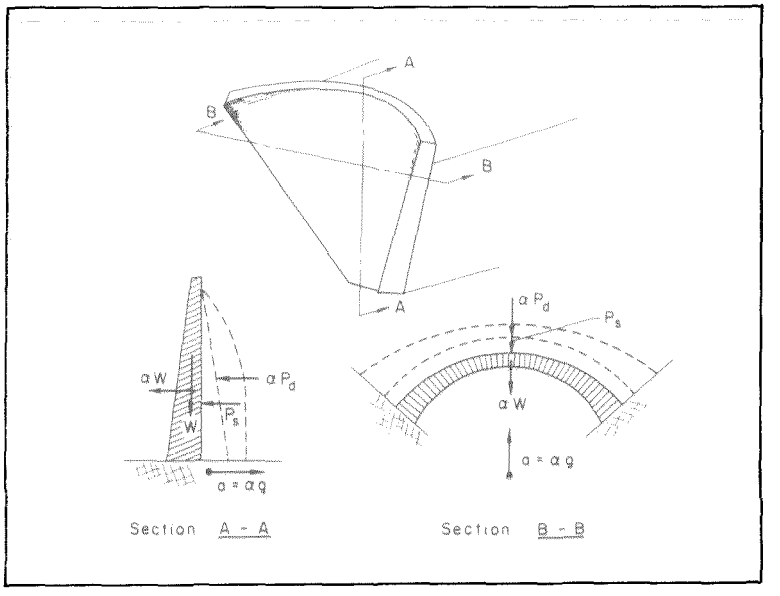
Arch dams are built in narrow canyons, and they are true three-dimensional structures. They resist the water load by combining cantilever bending from the canyon floor with arch thrusting to the abutments (lower right). Usually their proportions are much thinner than those of concrete gravity dams. Tensile stress was again avoided in the design, but engineers found stress analysis much more complicated. An iterative relaxation method applied to independent arch and cantilever sections was developed, which produced many rooms full of engineers grinding out stress calculations.

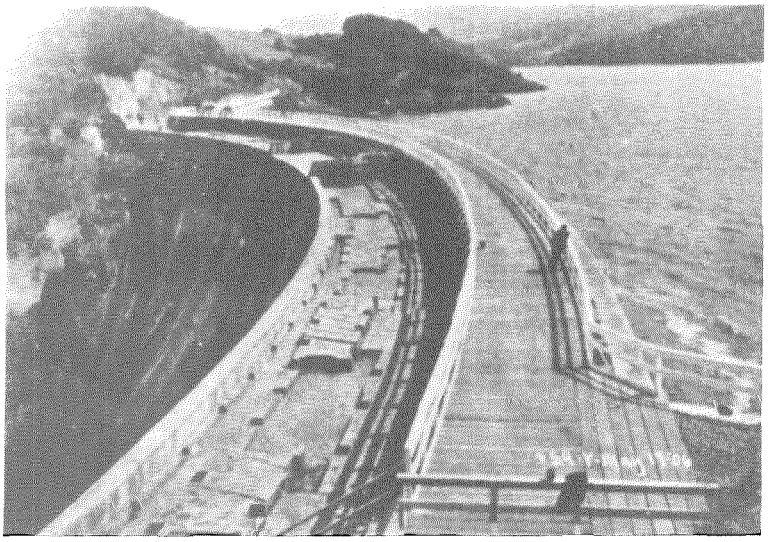
Engineers recognized early in this century that earthquake shaking introduced additional forces into their analyses. A horizontal acceleration in the upstream direction seemed most critical because it increased the overturning forces. The assumption was that the dam, which appeared to be a stiff structure, would move rigidly with the ground. Thus, if the ground accelerates at a fraction, designated α (alpha), of gravity, an inertial force of magnitude α times the dam weight is created and acts on the dam in the downstream direction. Moreover, additional water pressure is generated, proportional to the acceleration of the dam into the reservoir if water incompressibility is assumed. This feature was recognized in 1933, and it has been included in dam design ever since. Typical values of α were 0.05 to 0.15, and inclusion of earthquake effects still allowed



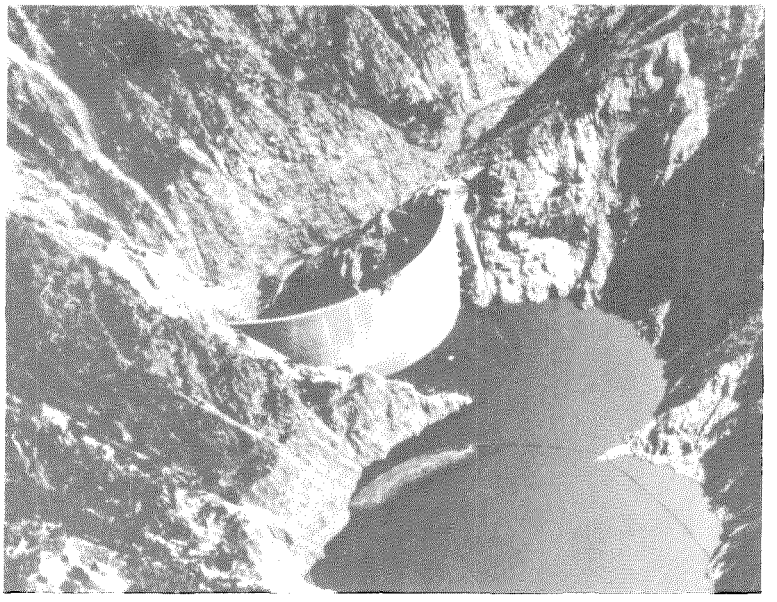
Design of a concrete gravity dam (above) is based on a two-dimensional idealization. The design loads include the dam weight W, the static water force P_s and the ground acceleration α (given as a fraction α of the gravitational acceleration g). The ground acceleration produces an inertia force αW on the dam and an additional water force αP_d (where P_d is the water force caused by a unit acceleration of the water into the reservoir).

Design of a concrete arch dam (below) must take into account the three-dimensional structural action.

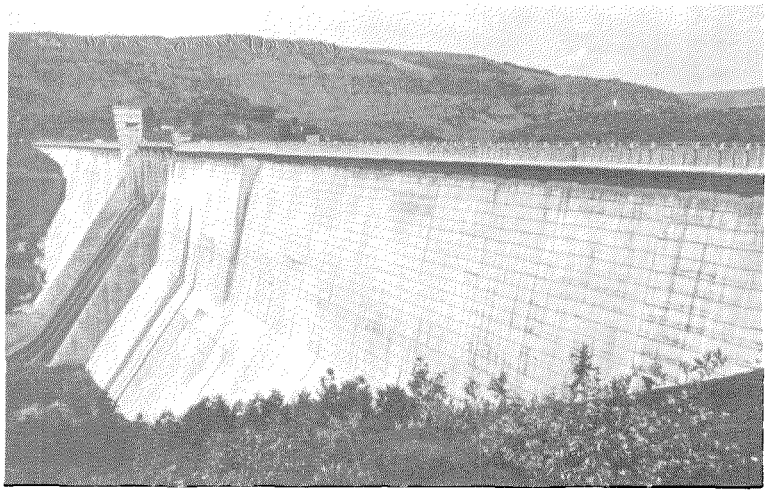




Crystal Springs Dam (a curved gravity type shown above) was undamaged by the 1906 San Francisco earthquake. Furthermore, earthquake accelerations in the range of 1.0 g during the 1971 San Fernando earthquake failed to damage . . .



. . . Pacoima Dam (above), a 370-foot-high arch dam. The upper portion of Koyna Dam (a gravity dam pictured below) cracked through from upstream face to downstream face during an earthquake in 1967. No failure resulted.

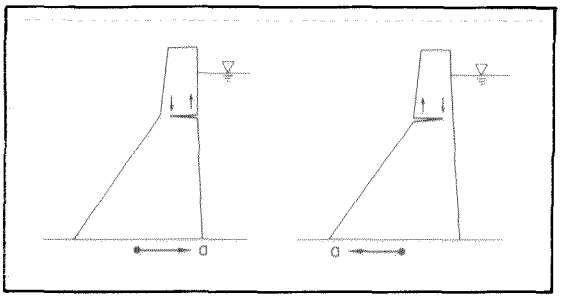


the no-tension criterion to be observed in the design.

Early design procedures were obviously great simplifications of reality. Dams are not really rigid; they are flexible structures that vibrate on their own when excited by ground motion. Stress analysis methods were approximate, and the maximum ground accelerations used were only fractions of what could occur. Vertical and cross-stream components of ground motion were neglected. But the pertinent question is, of course: How have dams designed by the methods described performed during past earthquakes? And the answer is: Fairly well, although there have been a few surprises.

The first significant event occurred in 1906 during the San Francisco earthquake. Crystal Springs Dam, a 145-foot-high curved gravity dam (pictured at the left), was located adjacent to the fault break, and it survived undamaged even though earthquake loading was not considered in its design. This good performance was attributed to a high reserve strength; the cross section was designed as a gravity section, but the curved plan also enabled arch action to carry a portion of the load. Another dam, Pacoima, performed well during the magnitude 6.4 San Fernando earthquake in 1971. Pacoima Dam (shown at the left) is an arch dam 370 feet high, 10 feet thick at the top, and 99 feet thick at the base. Earthquake accelerations measured above one abutment peaked at the remarkable value of 1.25 g. Yet the dam survived undamaged, possibly because of a low water level in the reservoir.

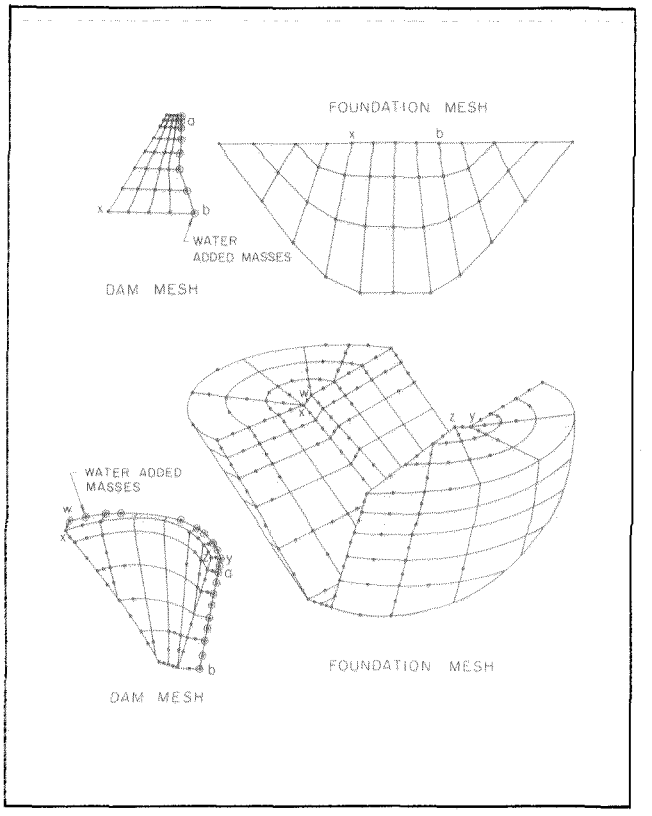
In 1962 Hsinfengkiang Dam, a 344-foot-high concrete dam near Canton, China, was shaken by a magnitude 6.1 earthquake. Considerable longitudinal cracking occurred in the upper portion of the dam, but no failure resulted. This event had a twofold significance. First, it showed that concrete tensile stress could be present (which cast doubt on the accepted methods of dam design). Second, it showed that considerable cracking does not necessarily imply failure. These two lessons were reinforced in 1967 during a magnitude 6.5 earthquake near Poona, India, which shook the 338-foot-high concrete gravity Koyna Dam (lower left). Maximum ground acceleration at the site measured 0.63 g. Again, extensive longitudinal cracking appeared in the upper portion of the dam, and again, failure did not occur. Researchers believe that the dam cross section cracked completely through from upstream face to downstream face, and the block above the crack rocked back and forth during the earthquake (as



shown in the figure above). Fortunately, this post-cracking stability is a real phenomenon, and there has come to be general agreement that arch dams, because of their combined cantilever and arch actions, possess a considerable amount of it.

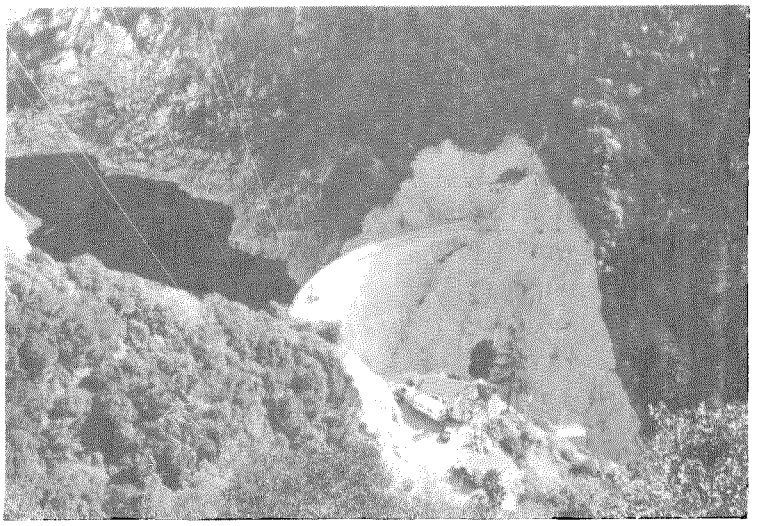
Although no earthquake-related failure of a concrete dam has occurred to date, no large concrete dam with a full reservoir has ever been subjected to really severe ground shaking. Such a possibility has many groups concerned, including the Division of Safety of Dams, a California state agency responsible for assuring the safety of California dams. The DSD has the power to order an updated seismic check of a dam if new information rises or if better analysis techniques are developed by researchers. In the early 1970s, two events led the DSD to initiate a program to perform seismic checks on all major dams under its jurisdiction. The first event was the near collapse of Lower San Fernando Dam, a large earthen dam, during the 1971 earthquake; and the second was the development of the finite element method, a tool for computerized stress analysis.

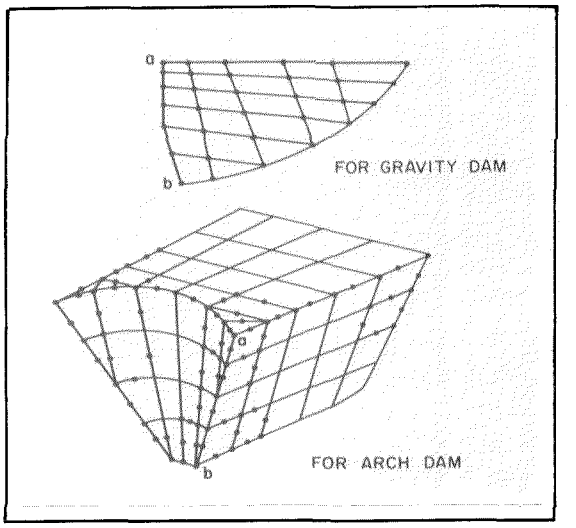
The finite element method transforms the governing differential equations (the equations of solid mechanics in the case of a dam) to a matrix equation that is solved on the computer. The structure to be analyzed is meshed into elements (see figure above right), which are connected at nodal points. Associated with these nodes are displacement degrees of freedom, which become the unknowns of the matrix equation. Solution of the matrix equation yields the structure displacements, from which the stresses are easily computed. As long as the governing differential equations are linear, the finite element method produces remarkable solutions. Nonlinearities, however, are much more difficult to handle. An example of nonlinearity in dam behavior is the formation of cracks or opening of built-in joints due to the presence of tensile stresses. Even today, finite element techniques have not progressed to the point where this type of nonlinearity can be handled.



As a part of the DSD program, a standard procedure using the finite element method was developed for computing the (linear) response of concrete dams. The drawing above illustrates this procedure. A finite element model is constructed of the dam and of a portion of the foundation region that extends out to an artificial boundary where earthquake motions are applied. The motion specified by the engineer is actually the free-field motion (that is, the motion that would occur at the dam-foundation interface if the dam were not present), and the engineer must back-calculate what motion to apply at the foundation boundary. Since the foundation boundary produces wave reflections that contaminate the computed dam response after a short time, the foundation mesh is usually assumed to be massless. The alternative is to place the foundation boundary far away from the dam, but this results in a large, expensive-to-solve matrix equation. The water is included in the analysis by an added-mass approach. An appropriate volume of water is assigned to move with each horizontal, nodal degree of freedom at the upstream dam face. Treating the water in this manner neglects water compressibility (which can be important for deep reservoirs) and ignores the additional pressures generated by the vertical and cross-stream components of earthquake ground motion along the reservoir boundary.

The top block of Koyna Dam (far left above) rocked back and forth but did not overturn. Above, an illustration of how improved earthquake safety evaluations of dams have been made possible through use of finite element analyses. A common procedure employs a dam mesh, a mesh of a finite region of the foundation, and added masses on the upstream dam face to represent the water.





Finite element analysis revealed that Santa Anita Dam (above), a 230-foot-high arch dam, would have difficulty surviving severe ground shaking if its reservoir were full. Above right, finite element models of the water in the reservoir accurately represent its effect on the dam response to earthquake shaking. These models include water compressibility and the additional pressures generated by vertical and cross-stream motions of the reservoir boundaries.

Nevertheless, this procedure has found wide application, because it can be readily implemented by a number of available structural analysis computer programs. Unfortunately, the assumption of linear behavior (no cracking) has been shown to be usually invalid. Consider, for example, the DSD-required check of Santa Anita Dam (pictured above left). It is a 230-foot-high arch dam, 7 feet thick at the top, and 62 feet thick at the base, located in a canyon above Monrovia, California. The purpose of the check was to determine if the dam could survive a hypothetical magnitude 7.0 earthquake on the nearby Sierra Madre fault. The free-field ground motion employed contained peak accelerations of 0.7 g and 0.45 g in the horizontal and vertical directions, respectively. Results of the linear, finite element analysis showed peak tensile stresses in the upper portion of the dam of 1400 psi, which is more than twice the tensile strength of concrete. Recognizing that Santa Anita Dam could take a reasonable amount of cracking and remain stable, the engineers faced the difficult task of judging the safety of the dam using an analysis that assumed no cracking — but that clearly showed significant cracking would occur. This remains the major dilemma today. The final decision on Santa Anita Dam was to lower the water level to a safe point until the dam could be strengthened.

Obviously, the most pressing research need is for computational techniques that accurately model the cracking behavior, but little progress has been made to date. Recently, however, some headway has been reported on improved modeling of the dam foundation and of the water in the reservoir. The artificial foundation boundary can be replaced by mathematical transmitting boundaries, which reflect only a small fraction of an incident wave. For the water, I have developed finite element models (shown above) that include water compressibility and the additional pressures generated by vertical and cross-stream motions of the reservoir boundaries. Both of these effects have been shown to influence the earthquake response of concrete dams significantly.

In order to provide guidance in the development of future mathematical models, the civil engineering group at Caltech has recently received a two-year grant for experimental research from the National Science Foundation. We plan to conduct an extensive series of shaking tests on actual dams using our 5000-pound shaking machines. Measurements will be made to define each dam's resonance and damping characteristics, the additional water pressure generated by the dam motions, and the stiffness characteristics of the foundations. Small-scale model tests will also be performed. Because calculations have shown that water pressures near the dam can reduce to water's vapor pressure during the shaking, we plan to study at reduced scale the mechanism by which this cavitation takes place. An interesting series of small-scale, shaking-table tests on precracked dam models will provide insight into the post-cracking stability of concrete dams. Future mathematical models should greatly benefit from these next two years of experimental research.

What will happen to dams during severe earthquake shaking? It is obvious that at present engineers cannot answer this question with any certainty. But we are very much aware of the threat of disastrous losses of life and damage to property if dams should fail, and we are making great effort to increase our understanding of this complex topic. □

Lewis Thomas, Global Habitability, and Earth Satellites

by S. I. Rasool

S. Ichtiaque Rasool is currently Distinguished Visiting Scientist at Caltech's Jet Propulsion Laboratory and a visiting associate in planetary science on the campus. He also holds a Fondation de France chair at the Ecole normale supérieure in Paris and previously spent 17 years with NASA, his last position there being Chief Scientist for Space and Terrestrial Applications at NASA headquarters in Washington, D.C. For the past few years he has been particularly interested in promoting the use of space information for earth sciences and, as chairman of the International Committee on Space Research (COSPAR) Commission on Studies of the Earth's Surface, Meteorology and Climate, has been instrumental in organizing the international workshops and program of research described below.

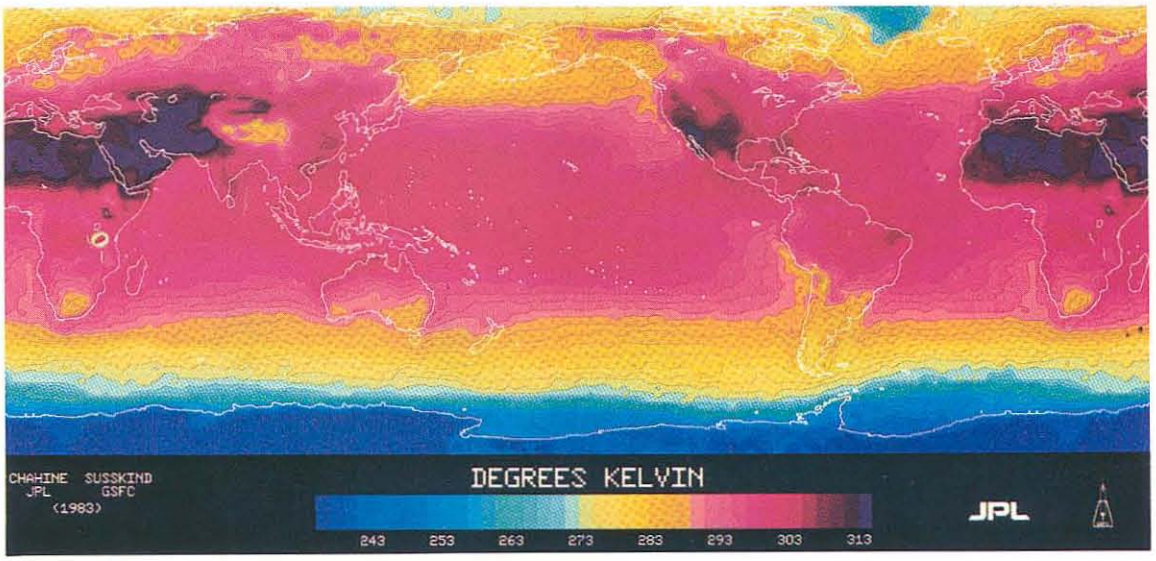
On reading Lewis Thomas's discussion of the Global Habitability project in the last issue of E&S, *he was inspired to respond on the state of the program and why it's not as simple a task as it may sound. Born in India and educated in France (PhD in atmospheric physics), he is as interested in the questions of "international politics" that Thomas raises as in the technical problems.*

In his essay ("Science and Social Science," *E&S*, March 1984) Lewis Thomas eloquently and convincingly argues that NASA as an agency should begin to apply the sophisticated space technology it developed for planetary exploration to monitor and study the "anatomy, physiology, and pathology of the earth itself." He writes of his hope that the Global Habitability project stands high on NASA's list of priorities and urges its funding.

Dr. Thomas is quite right — the problems of earth are fundamental, and satellites and the imaging technology developed for space exploration can help us understand and in some cases even solve these problems. He writes: "It is possible now to begin monitoring this planet, spotting early on the evidences of trouble ahead for our species or for others, especially the kinds of trouble for which we humans are responsible. I cannot think of a better work for the international science community on the ground or out in space, and I hope we will get on with it."

We are indeed getting on with it. But whether it is "possible now to begin monitoring this planet" is another question. There is still a lot of homework to do before the current space technology can really be used to observe consistently the seasonal or year-to-year changes in the character of the earth's surface on a global scale. Even though the imaging from space is constantly improving, and we can "see" more and more clearly, we still very often don't know exactly *what* we are seeing and what it means, especially in the case of the earth's surface. We have to be able to convert images into meaningful and quantitative scientific data over time, and this presents some real problems. Images taken by satellites in the visible, infrared, and microwave regions of the spectrum will first have to be validated with ground "truth"; that is, teams of researchers will have to compare what the satellite sees from space with what can actually be confirmed on the ground. For example, what are we really seeing in a "vegetation index" determined by a satellite? Is it the leaves, the humidity of the plants and soil, the chlorophyll in the biomass, or what?

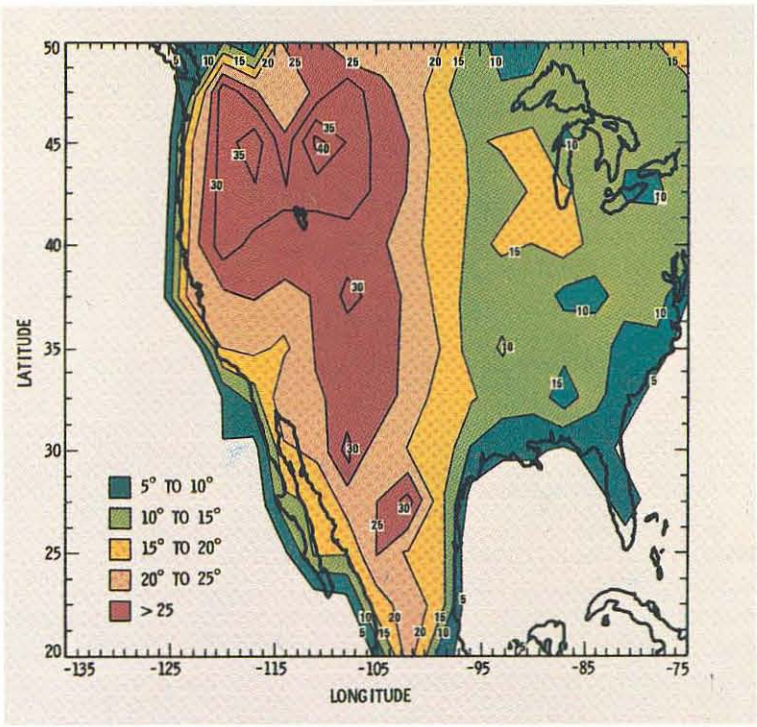
The physical, chemical, and biological state of the whole globe cannot be monitored all the time by a hypothetical Global Habitability satellite. Also, NASA isn't going to be able to do it alone. To start with, we will have to use the satellite systems that exist today and that are already scheduled for the rest of the decade.



A NOAA satellite provided temperature data that, when corrected to remove interference from clouds and atmosphere, yields this image (made by Chahine/Susskind at JPL) of the mean "skin" temperature (averaged over day and night) of the earth in July 1979. To determine whether the earth as a whole is warming or cooling over time, at least 10 years of such data would need to be analyzed.

Using the same satellite data that generated the world map above, S.I. Rasool plotted the difference between day and night temperatures for the United States, dark green representing the smallest difference and dark brown the greatest. Since moist soil retains the day's heat while deserts do not (and consequently grow colder at night), this map can be seen as a "wetness" index, which could also have something to say about vegetation. Before this could have any real meaning, however, it would have to be verified on the ground.

For any new system we will have to wait until the early 1990s. But what does already exist are the weather satellites operated by the National Oceanic and Atmospheric Administration (NOAA), which observe mainly the cloud and storm systems and measure atmospheric temperatures and moisture but have some capability to look at the surface; the land-observing satellites, such as NASA's Landsat and the French SPOT (to be launched next year), whose task is to map the geology and monitor the health and acreage of crops; and NASA's research satellites, which attempt to measure the chlorophyll in the oceans, the dust in the stratosphere, and the extent of the polar ice.



Today six different nations launch these kinds of satellites with very specific, and often only local, objectives in mind. The *immediate* challenge, therefore, is to use these existing systems and try to extract from them information on global changes in biomass over the last decade, so that we can understand, for example, as Thomas describes it, "the cyclic exchanges of carbon, nitrogen, phosphorous, and sulfur between land and oceans," the year-to-year fluctuations in global rainfall patterns, variations in soil humidity and in surface temperatures on a continental scale, changes in ocean productivity, and, of course, attempt to resolve the big question of whether the earth as a whole is warming up or cooling down.

Because these satellites are multinational and because any one of them lasts only two or three years on the average, calibrating the different systems into a standardized interpretation over a decade is a major task. But it's not impossible. Dr. Thomas writes that the Global Habitability program will involve more than just congressional wrangling over NASA's budget. "It will require, as well, collaborated efforts by researchers from many different disciplines in science and engineering and from virtually every country on the face of the earth, which means international politics at its most difficult."

These collaborated efforts have already begun with the blessing of NASA, NOAA, the French Space Agency (CNES), the European Economic Community, and the United Nations Environment Program (UNEP). Following are excerpts from an article about these preparations from the February 1984 (Vol. 65, No. 2) *Bulletin of the American Meteorological Society*, which I wrote with H. J. Bolle, president

of the International Association of Meteorology and Atmospheric Physics. (Since this was published, NASA has sponsored another workshop in March in Washington, D.C., bringing the total of international scientists involved in the project to more than 200.)

More than 100 geophysicists and space scientists, drawn from a variety of disciplines and from 22 countries, deliberated in two week-long workshops held in Boulder, Colorado, and Innsbruck (Austria) and in a two-day briefing session in New Delhi (India) this summer as part of the United Nations Environment Program (UNEP) supported International Satellite Land-Surface Climatology Project, within the World Climate Impact Studies Program.

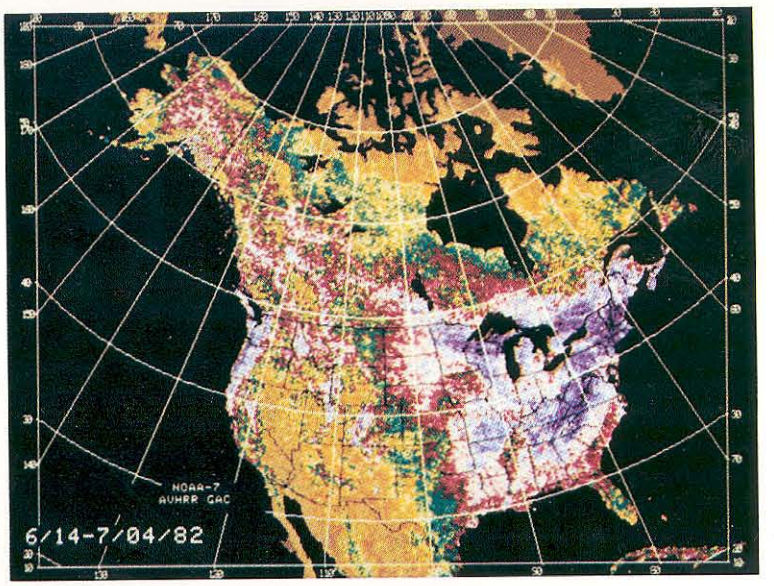
The principal motivation for such a project comes from three important considerations: 1) both climate induced and man-made changes on the earth's surface are known to be large and of profound significance; 2) satellites are ideally suited to measure these changes globally and over long-time scale (years, decades); 3) but there is currently no uniform methodology to derive consistent surface cover information from radiances measured by currently operating satellites.

The purpose of the workshops was to define a program of research which would lead, within five years, to agreed upon methodologies for converting satellite measured radiances into quantitative data concerning the earth's surface . . .

. . . Vegetation cover is the variable which is of greatest susceptibility both to the climatic fluctuations and human activities. Vegetation cover also has a direct bearing on the life of man on earth . . . Climatic fluctuations also change the extent of snow cover and ice sheets; natural climatic fluctuations along with human activity also affect the soil humidity and the boundaries of the desert. At the same time, these very changes on the earth's surface can themselves influence the climate of a region or even the globe. They certainly affect the energy and momentum transfer between surface and atmosphere as well as the soil hydrology . . .

. . . Currently, satellites appear to be capable of measuring a few of these land-surface variables with some precision; none of the parameters, however, is determinable with high enough consistency and accuracy that a long-term history of changes on the earth's surface can be documented. A careful assessment of the state-of-the-art of space technology at the workshops indicated that although the satellite sensors have become quite sophisticated, many important fundamental problems have yet to be solved satisfactorily before the radiances measured at the satellite could be adequately interpreted in terms of changes in surface properties of the earth . . . In spite of the fact that some ten years of quantitative satellite observations are available, these problems have not been solved to the extent required. It was the conclusion of the workshops that now we understand the problems well enough to plan an initial program to solve them . . .

. . . In summary, at this set of three workshops, it was abundantly clear that satellites can be a major tool to really assess globally the impact of climate and of man's activity on the character of the surface of the earth. It was also clear that if the satellites are to be an adequate tool, then one has to "calibrate" them with ground "truth" repeatedly in time and in space, and one scientist's interpretation of the measurement has to be compared with that of other workers. Only then can one visualize a homogeneous and authentic data set which can be used to assess quantitatively key parameters for climate studies. It was agreed that if this objective is to be accomplished before the end of the decade, the evaluation and validation studies as described briefly here must start at once because all the ingredients to initiate such a program are available now: more than a decade of satellite data, improved models of land surface-atmosphere interactions, ground observations at key locations, competent scientists and, very importantly, the motivation of appropriate international agencies to coordinate such an activity on a global scale. □



This "greenness" index for June 1982, using data from another satellite instrument, was made by Samuel Goward and colleagues at the University of Maryland and Goddard Space Flight Center. Since the map shows the amount of green, or chlorophyll (actually represented by colors ranging from purple to yellow), it might also be considered a vegetation index. But, like the wetness index, this too is really a measure of radiation, and, although the pattern is strikingly similar to the wetness index, such interpretations will have to be correlated and authenticated over a long period of time in order to be useful for habitability planners.

Henry Borsook

1897-1984

HENRY BORSOOK, professor emeritus of biochemistry, died of cancer in Santa Barbara, California, on March 6, 1984. He was 86.

Borsook was born in London, England, but moved with his family to Toronto, Canada, in 1907. He entered the University of Toronto as an undergraduate in 1917 and left in 1927 with a BA in physiology and biochemistry, a PhD in biochemistry, and the medical degree of MB (later converted to MD). His university career was marked by many honors, including the Faculty of Medicine Gold Medal on his graduation from medical school.

After graduating, Borsook spent two years as research fellow and lecturer in biochemistry at Toronto. In 1929 he was appointed assistant professor of biochemistry in the division of biology at Caltech, which had been founded the year before by Thomas Hunt Morgan. Borsook remained at Caltech until his retirement in 1968 at age 70. Not ready to give up work in the laboratory, he accepted an appointment at Berkeley, where he continued to do research until 1977.

The biochemistry of protein synthesis was one of the major themes of Borsook's scientific life. When he started his career, notions of the mechanism of protein synthesis were primitive by modern standards. No protein had been sequenced, the idea that the properties of proteins rested on unique sequences of amino acids was at best hypothetical, and the concept that an input of sequence information is essential for protein synthesis was still in the future. Borsook's earliest papers deal with the synthesis of a protein-like material by the action of the proteolytic enzymes pepsin and trypsin on a concentrated solution of the products of hydrolysis of egg albumin. The product, called "plastein," was thought to result from reversal of the hydrolytic action of the enzymes and to be relevant to the normal process of protein synthesis. Borsook himself later disproved this idea when, at Caltech, he initiated a program to measure the free energies of formation of biologically important compounds, in collaboration with Hugh M. Huffman. He found that energy is required for protein synthesis from amino acids and that no significant reversal of the action of proteolytic enzymes is possible under the conditions that obtain in cells. This was perhaps his most important contribution to the general theory of protein synthesis. Later in his life he worked extensively on the role of the hormone erythropoietin in the cellular transformations that lead to the synthesis of hemoglobin.

Human nutrition was another subject of interest to Borsook. In the 1930s he carried out studies on the effects on human beings of B vitamins, especially B-1, in collaboration with the Institute physician, Dr. M. Y. Kremers. Old-timers can remember the clinic that was held weekly in Dr. Kremers' office on the first floor of Kerckhoff Laboratories, where massive doses of vitamin B-1 were administered to patients with trigeminal neuralgia (tic douleureux). During the war, Borsook was appointed to the Food and Nutrition Board of the National Research Council and to numerous other boards and commissions advising federal and state government on nutritional questions. The table of Recommended Daily Allowances drawn up by the Food and Nutrition Board revolutionized the concept of nutrition, Borsook later pointed out, by showing that a good diet consists not of "food" but of certain amounts of specific nutrients — proteins, vitamins, calories, and the like. After the war, Borsook joined with Clifford Clinton, philanthropist and owner of Clifton's cafeterias in Los Angeles, in the production of Multipurpose Food, a low-cost, enriched food based on soybeans, that was used to sustain impoverished populations around the world. A non-profit organization, Meals for Millions Foundation, was set up to distribute the food. Borsook was research director and member of the board of the foundation. As of 1963, over 90 million meals had been distributed.

Borsook taught biochemistry at Caltech for 35 years. He was twice chairman of the faculty, and he also chaired the student health committee for years. Among his off-campus activities, the most memorable for old biology alumni was his sponsorship of the Anaximandrian Society, an organization that met monthly in the Borsook home on Constance Street to hear and discuss a student-written paper on some aspect of the history of biology. Interesting guests were often present, and Borsook was a brilliant conversationalist. These, along with the refreshments provided by Mrs. Borsook, made the meetings a welcome break from dormitory and rooming-house life. The society existed from 1935 to 1945. The bound papers are in the Archives.

Borsook's hobby was art history. He had a special interest in the rococo churches of Bavaria, Austria, and the Tyrol, which he photographed in annual trips to Europe. Borsook was a member of the American Society of Biological Chemists and several other professional societies. He received the Groedel Memorial Award of the American College of Cardiology in 1957.

He is survived by his wife, Lisl, and their daughter, Eve.

Norman H. Horowitz
professor of biology, emeritus

Research in Progress

Magnetic Monopoles

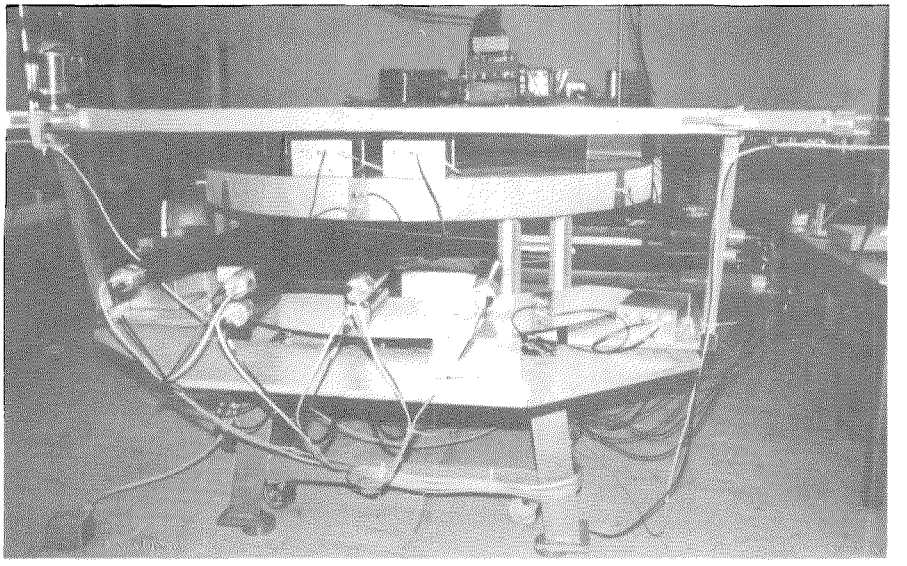
MAGNETIC MONOPOLES, which, as their name implies, are fundamental particles behaving as a single pole of a magnet, have never been observed. But there are enough hints of their existence to whet physicists' appetites to find them. The monopole, or carrier of a single magnetic "charge" corresponding to the electron, was the only lack of symmetry in Maxwell's equations of electric and magnetic fields. Half a century ago, Dirac predicted magnetic monopoles to explain the quantization of electric charge. And just a decade ago, grand unified theories, which seek to unify three of the four basic forces — electromagnetism, the strong force, and the weak force — suggested that magnetic monopoles could have been produced in the extreme high energy collisions of the Big Bang.

This recent prediction set off a new wave of searching, because the monopole's existence would help confirm the theories. One of those looking for the rare and elusive particle is Barry Barish, professor of physics. Or he is, like the other monopole hunters, at least trying to determine the best way to search.

The monopole described by the grand unified theories is extraordinarily massive for a fundamental particle — about as heavy as a speck of dust. It has 10 million billion times the mass of a proton but its volume is far smaller. And it's slow moving — about one-thousandth the speed of light.

No accelerators exist that are capable of the extremely high energies necessary to produce magnetic monopoles. That amount of energy existed only at the time of the Big Bang (in the first 10^{-43} second), so the only possibility of observing a monopole lies in finding one that has been around since then, lurking perhaps in the cosmic rays. How likely is this? How many monopoles might exist, if they exist at all?

Various cosmological calculations have come up with either too many or none at all, says Barish. There is, however, some important guidance from astrophysics. Upper limits on their abundance can be set by assigning monopoles responsibility for all the unseen mass of the universe; greater mass would have caused the universe to collapse. Also, the existence of the galaxy's magnetic field limits the concentration of monopoles that could exist; too many monopoles would have destroyed these magnetic fields. The astrophysical bounds, then, set the scale for a serious experimental search. Just to get below the upper bounds, to have the possibility of finding even a few monopoles in a year, would take a detector at least the size of a football field.



Detectors like this, but greater in size and sensitivity, may someday be listening for monopoles. The acoustic detector consists of two aluminum disks, 2 m in diameter and 10 cm thick, with tiny sensitive microphones attached along the sides to pick up the high-frequency signal of a monopole's ultrasonic shock wave when passing through the disks. Sheets of scintillation material above and between the disks, with cylindrical photocells attached, would provide additional signals to determine the presence of a monopole.

Barish's experimental ionization detectors are not yet that large. One recently dismantled in his laboratory consisted of a stack of sheets of plastic scintillation material 3 meters by 1.5 meters, surrounded by photomultipliers. If the detector is sensitive enough, a monopole passing through the scintillation material would produce a tiny, but detectable, flash of light. Barish is working on some of the fundamental aspects of the detector's design, including determining the optimal type of scintillating material. Eventually an array of such detectors could be constructed on the hypothetical "football field."

One of the practical problems is where to put such a huge, flat array. Physicists aren't likely to commandeer a football stadium anyway, but the main problem is to shield the detector from cosmic rays — from all of them that are not monopoles, that is, all but about 1 in 10^{10} cosmic rays. If the detector is underground, the intervening dirt will screen out most of the unwanted cosmic rays, and the rest could be dispatched with instruments. Barish recently returned from talks in Italy about a possible collaboration there. Italian scientists have acquired access to a 150-meter-

long, underground laboratory space in the Gran Sasso tunnel (an extra little boon from a highway project) and are serious about using it for magnetic monopole experiments. Over the next six months Barish will continue negotiations on joining the Italians in the tunnel.

An alternative to putting the detector underground is to use instrumentation to remove all the cosmic rays from an above-ground detector. This might, however, raise its cost considerably; the cost of the projected large scintillator array is probably at least $10 million. And even though the scintillator is generally considered the most likely technology for finding magnetic monopoles, the monopole's signature in such a detector is "not as unique as we would really like," says Barish. So he is simultaneously pursuing an alternate acoustic technique.

Barish theorizes that monopoles passing through a conductor produce eddy currents that would create an ultrasonic shock wave, detectable by piezoelectric transducers. A stack of aluminum disks with acoustic transducers (very sensitive microphones), attached around the edges to measure the speed of a particle passing through them, would produce an unambiguous signal from a slow-moving monopole. Sheets of scintillator material are mounted between and above the disks of the detector to give further information on the particle. A large-scale version of this system could be significantly cheaper than the scintillator and would identify a monopole more definitely, but Barish still has to make the technique 100 times more sensitive before it has a chance of finding a magnetic monopole.

Other attempts to find magnetic monopoles are under way from such diverse sources as x-ray emission from neutron stars and etched tracks in ancient mica. There have been a couple of false alarms, the most recent being the 1982 preliminary report of a monopole-like signal from a wire-loop apparatus in a Stanford lab. But since no more particles have been recorded on a more sensitive Stanford instrument, though it has run 68 times longer than the first experiment, it appears less and less likely that a magnetic monopole was actually observed. And it seems more and more likely that the first monopole will be seen as a tiny flash of light or heard as a high frequency ping in some giant detector yet to be built. □ — *JD*

Sickle Cell Anemia

AN ABNORMAL HEMOGLOBIN, known as hemoglobin S, characterizes sickle cell anemia, a genetically determined disease that seriously afflicts an estimated one in 600 Americans of African descent. About 8 percent carry the genetic trait for the disease but are asymptomatic.

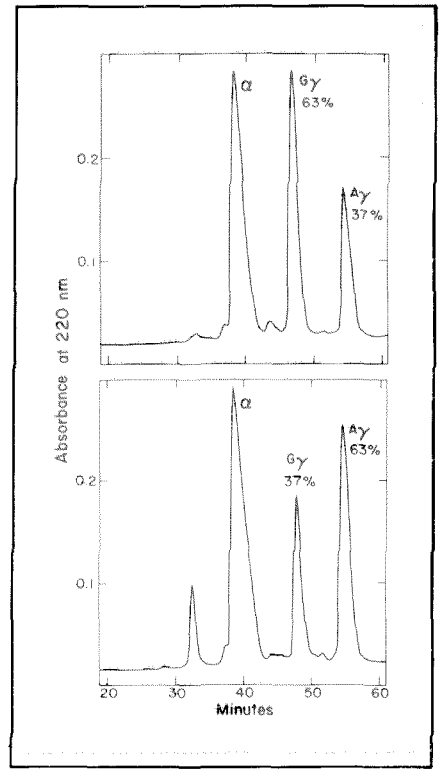
Hemoglobin carries oxygen throughout the body to be used in various metabolic processes. The problem in sickle cell anemia arises when oxygen leaves hemoglobin S. The molecules of hemoglobin S then tend to line up in long fibers, which distort the red cell into a rigid sickled shape that does not pass easily through the capillaries of the circulatory system. Even though much is known about sickle cell anemia and about hemoglobin, no cure or means of alleviating the symptoms of the illness has yet been found.

Walter Schroeder, senior research associate in chemistry, has been studying abnormal hemoglobins (with some lapses) since hemoglobin S was discovered by Linus Pauling and Harvey Itano here at Caltech in 1949. Recently, new, faster, and more sensitive tools for biochemical analysis, as well as current advances in DNA research, have added much information and may lead to the possibility of at least alleviating the severity of sickle cell anemia, as well as other genetic diseases of hemoglobin abnormalities, such as thalassemia.

The problems of sickle cell anemia begin to appear as fetal hemoglobin is replaced by adult hemoglobin (in this case, hemoglobin S; in the normal child this adult hemoglobin is hemoglobin A), a process that begins before birth and is completed by about six months of age. How the switch to adult hemoglobin takes place is a mystery. Fetal and adult hemoglobin differ in one of their two polypeptide chains, which are the products of particular genes. It's in the gene for that different chain, the beta chain, that the sickle cell abnormality is expressed in adult hemoglobin. Fetal hemoglobin has properties that are useful to the developing fetus but it also functions perfectly well in adults; in fact, it would be preferable to hemoglobin S.

Traces of fetal hemoglobin remain in the blood of adults. These traces occur in greater, but variable, amounts in individuals with sickle cell anemia and may have something to do with alleviating the symptoms. For example, Saudi Arabians with sickle cell anemia have rather a lot of fetal hemoglobin, and they are reported to have a generally less severe form of the disease. Consequently, it has long been a goal of many investigators to try to prevent the switch from occurring in infants diagnosed as having sickle cell anemia, or even to reverse the mechanism in adults and switch back to production of fetal hemoglobin. Schroeder thinks that 40-50 percent fetal hemoglobin might be needed to prevent the sickle cell fibers from forming. This conclusion comes from statistical studies with Darleen Powars of USC, which suggest an effective threshhold of 20 percent. In order to find a way to increase this percentage, Schroeder has done extensive analyses of fetal hemoglobin in search of the switch-over mechanism.

With Joseph DeSimone at the University of Illinois, Schroeder has collaborated on studies of baboons, whose hemoglobin synthesis is very similar to that of humans. The scientists have succeeded in treating the baboons chemically, under certain conditions, so that



Analyses of the concentration ratios of polypeptide chains in fetal hemoglobin of two sickle cell anemia patients shows opposite ratios for the two gamma chains (G and A). This ratio may affect the severity of the disease.

the baboons actually do produce large amounts of fetal hemoglobin. The conditions and complexities of this switch are still being investigated.

The Caltech chemist is also concentrating on analysis of the hemoglobin and DNA of sickle cell patients for biochemical factors that determine how severity of the illness varies. Some patients get much less sick than others; is there one piece of genetic information that is likely to tell why? Schroeder's main tool to determine the composition of hemoglobin in these studies is high performance liquid chromatography (HPLC). It's now possible to separate chemically two polypeptide chains that differ by only one methylene group (molecular weight 14) out of a total molecular weight of 16,000. Work that once took three days can be done with HPLC in two hours and with a sample of 0.1 milligram instead of 100.

The particular hemoglobin chains that he's interested in are the two types of gamma chains in fetal hemoglobin. What is called the alpha chain in fetal hemoglobin remains the same in the switchover to the adult form, but the two gamma chains switch to a single beta chain in adult hemoglobin. And it's in the beta chain that the sickle cell and thalassemia abnormalities are expressed.

The two gamma chains (called G-gamma and A-gamma) differ in only one amino acid in a particular position along the chain. The ratio of the concentrations of these chains is different in the hemoglobin of newborn infants than in the trace of fetal hemoglobin present in adults. Adult sickle cell patients, however, may have either ratio. The figure gives examples of HPLC data from two patients who have opposite ratios of the chains.

Similar data had previously been obtained with the old time-consuming methods in collaboration with Titus Huisman of the Medical College of Georgia. Huisman has also been examining fetal hemoglobin of patients in Georgia by HPLC and looking at certain aspects of DNA structure. He is seeing correlations between some properties of fetal hemoglobin and DNA that suggest certain regions of DNA as the control point of the switch. Schroeder is studying patients in southern California to discover whether there's a similar correlation of the ratio of the gamma chains with a difference in the DNA of these patients, whether the ratio of the gamma chains and differences in DNA influences the severity of the illness, and whether these factors are implicated in the switchover to adult hemoglobin.

Correlations of biochemical data with medical information has been aided by the Los Angeles Sickle Cell Center at the USC School of Medicine. Schroeder has been associated with the center since its founding in 1972. Some of his main collaborators at Caltech have been associate chemists Roger Shelton, Joan Shelton, and Lois Kay. □ — *JD*

Letters

Readers' Choice

In the January issue of E&S *we printed the picture at right of a 1936 Throop Club housewarming party, identifying the members of the group as follows: "We can't name the first and third men from the left, but the others are Frank Jewett, Bob Mahoney, K. Watanabe, Hugh Colvin, Wally Swanson, Paul Hammond, Howard Hamacher, and Ed Kasnika." Predictably, we got some help on the missing names from some of our readers. Probably equally predictably, they didn't entirely agree. Below are their letters, and we leave it to you to decide.*

Vacaville, CA

EDITOR:
Please refer to the photo on page 32 of the January issue of *E&S*. You state that you cannot identify the first and third men from the left. I feel quite certain that the first man is Frank Jewett (he always wore glasses). The second man is, I believe, Richard Pond. The third man is Jim Ritchey. Both Dick and Jim were 1939 classmates of mine. If they aren't as I have assumed, they are at least good doubles for those two.

FREDERICK C. HOFF

Mission Viejo, CA

EDITOR:
I think that Fred Hoff's observation is correct and that I am the third from the left. I didn't have much time in those days to take part in Throop activities, because I lived in Lynwood and worked full time at nights in the Firestone factory. I can't believe I was wearing a dark suit. Thank you for writing.

JIM RITCHEY

New Canaan, CT

EDITOR:
I think I can fill in the missing persons and help sort out the names and faces in that delightful picture under the heading "For Old Times Sake," *E&S*, January 1984, page 32. I believe the second person from the left is David K. Beavon, BS '38, not Frank Jewett as the caption indicates. I am the first person on the left. The third from the left, above Bob Mahoney, is, I believe, Evan A. Johnson, BS '38. If I am correct, all individuals are identified.

FRANK B. JEWETT

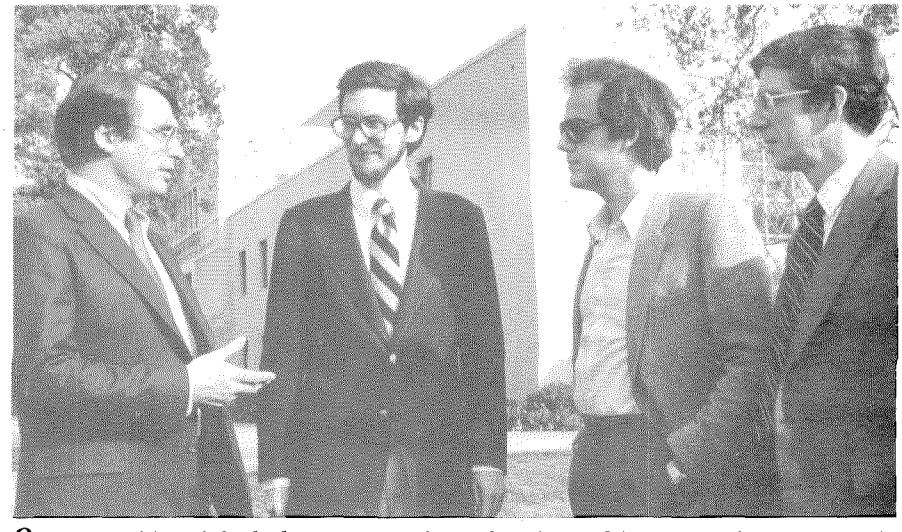
Random Walk

Women's Suffrage?

NOT LONG AGO we received the photograph reproduced above from John Benton, professor of history at Caltech. The photograph was actually taken by Martha Ward, assistant professor of art history, whose particular interest is 19th-century French painting. She and Benton, whose specialty is medieval history, were both working in Paris last summer when she had a camera and he wanted a picture taken. Benton felt the situation deserved a picture and a comment.

"The recognition of women in science comes in small steps," he says. "The Parisian street which houses the Ecole nationale supérieure de chimie was named after Pierre Curie in 1909, three years after his death and six years after he and Marie had been jointly awarded the Nobel Prize for Physics. Marie Curie was again awarded a Nobel Prize (for chemistry) in 1911, the first scientist to receive such an award twice, and she continued her distinguished work until her death in 1934. The street did not receive its present name of Rue Pierre et Marie Curie until 1967 — and the old sign naming Pierre Curie alone was still in place last summer when the photograph was taken."

Merit Increase



On a recent visit to Caltech, George Keyworth II (left), science advisor to President Reagan and director of the Office of Science and Technology Policy, met two Institute recipients of Presidential Young Investigator Awards, David Rutledge and Gregory Stephanopoulos. Bruce Abell (right) is assistant to Keyworth and a Caltech alumnus (BS'62).

WITH LIMITED means to compete, for several years universities have been losing more and more scientists and engineers to industry. This has meant a shortage of faculty to give students technical training, plus a shortage of graduates who decide to stay in academia. But some help seems to be forthcoming in the form of Presidential Young Investigator Awards. The names of the first 200 recipients were recently announced by the White House Office of Science and Technology Policy.

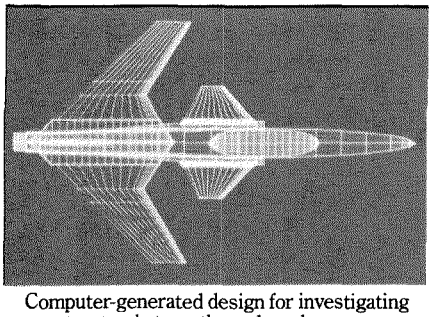
Eight Caltech faculty were included, putting the Institute behind only UC Berkeley's 12 and Stanford and Cornell's 11 each. The Caltech 8 are geologists Robert Clayton and Joseph Kirschvink, physicists Robert McKeown, John Preskill, and Thomas Prince, chemist Manfred Morari, electrical engineer David Rutledge, and chemical engineer Gregory Stephanopoulos.

This program, which will be administered by the National Science Foundation, provides for a grant to each researcher of $25,000 annually, renewable for up to five years. In addition, each year NSF will match any portion of an additional $37,500 that the Institute obtains from industrial sources, making the total possible support $100,000. It is expected that 200 new investigators will be named each year, which means that at the end of five years 1000 young scientists and engineers should be finding it easier to set up long-term research programs in academic settings — and more certainly rewarding than it has been for a long time.

Better Late Than Never?

THE MAIL recently brought us a kind of research report — a copy of an *E&S* Reader Survey, completely filled out, but originally mailed from our office in 1967. This is one delay that can't be blamed on the Post Office though, because our correspondent has had it all those years. In response to our 1967 questions, he gave us answers as of 1984, telling us that he is married, over 65, has a PhD in education, owns his home and two cars, is retired, prefers to travel by plane, carries bank credit cards, has a pension and Social Security income of under $20,000, and owns something under $10,000 worth of securities and about the same amount of life insurance. One thing he didn't tell us — and we hadn't asked — was his name.

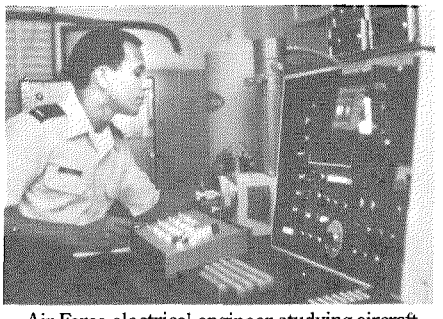
ENGINEERING TAKES ON EXCITING NEW DIMENSIONS IN THE AIR FORCE.



Computer-generated design for investigating structural strengths and weaknesses.

Developing and managing Air Force engineering projects could be the most important, exciting challenge of your life. The projects extend to virtually every engineering frontier.

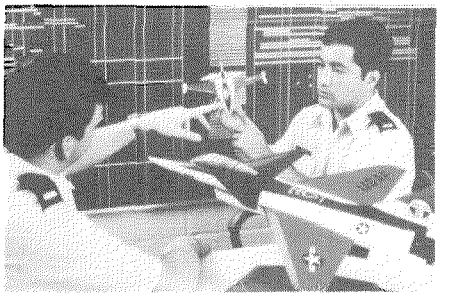
5 CAREER FIELDS FOR ENGINEERS



Air Force electrical engineer studying aircraft electrical power supply system.

Engineering opportunities in the Air Force include these five career areas: aeronautical, architectural, astronautical, electrical and nuclear. Hundreds of diverse specialties are included in a wide variety of work settings. For example, an electrical engineer may work in aircraft design, space systems, power production, communications or research. An aeronautical engineer might be included in wind tunnel testing, designing cockpit displays and flight testing.

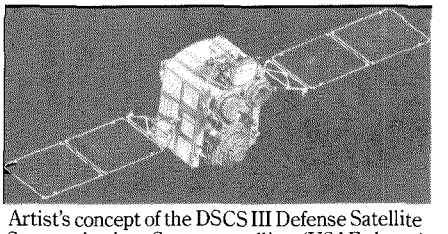
PROJECT RESPONSIBILITY COMES EARLY IN THE AIR FORCE



Air Force aeronautical engineers discuss flight characteristics of a fighter aircraft.

Most Air Force engineers have complete project responsibility early in their careers. For example, a first lieutenant directed work on a new airborne electronic system to pinpoint radiating targets. Another engineer tested the jet engines for advanced tanker and cargo aircraft.

OPPORTUNITIES IN THE NEW USAF SPACE COMMAND



Artist's concept of the DSCS III Defense Satellite Communications System satellite. (USAF photo.)

Recently, the Air Force formed a new Space Command. Its role is to pull together space operations and research and development efforts, focusing on the unique technological needs of space systems. You may have an opportunity to join the team that operates superior space systems as the Air Force moves into the twenty-first century.

To learn more about how you can be part of the team, as an officer, see your Air Force recruiter or call toll free 1-800-423-USAF (in California call 1-800-232-USAF). There's no obligation.

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