Dilatancy: Growing Acceptance as an Earthquake Mechanism

One measure of a field's scientific credibility is the number of presentations devoted to it at meetings, and by this measure earthquake prediction has clearly arrived. The papers given at the Washington, D.C., meeting of the American Geophysical Union on this subject have grown in number from a few a year ago to a veritable outpouring this year of evidence and calculations. Of particular interest is the dilatancy–fluid diffusion model of earthquakes and associated phenomena, which appears to be gaining wide acceptance as a basis for understanding and predicting the occurrence of these often destructive events.

Dilatancy is the swelling that occurs in rocks stressed almost to their breaking point, a phenomenon well known in the laboratory. That dilatancy also occurs in the earth's crust was only recently proposed (*Science,* 25 May 1973, p. 851). According to the model, the rocks along a fault dilate before an earthquake, opening microcracks and reducing the fluid pressure in rock pores. This has the effect of strengthening the rock and temporarily delaying the quake until water from surrounding regions can diffuse in, restoring fluid pressure and triggering the crustal rupture. Dilatancy is thought to cause surface deformation and a host of other premonitory phenomena, including a reduction of the speed with which *P* waves (compressional seismic waves) can propagate through the rock, which in principle can be used to give warning before the quake takes place.

Principle is rapidly becoming practice. A quake of moderate size near Riverside, California, on 30 January 1974 was successfully predicted about 3 months in advance by J. Whitcomb and his colleagues at the California Institute of Technology on the basis of the dilatancy-diffusion model. They noted a drop in the velocity of *P* waves passing through a region east of Riverside, an anomaly that disappeared after 13 months, indicating the likelihood of a quake. The investigators forecast a quake of magnitude 5.5, but what resulted was one of magnitude 4.1, although in the correct location and within the time period expected. Of more importance was the type of crustal fault on which the quake occurred. Earlier variations in seismic velocities, and hence indirect evidence of dilatancy, had been noted only in areas where one crustal block was thrusting underneath another. The Riverside quake, however, took place on a section of fault in which the crustal blocks are sliding past each other horizontally (strike-slip motion) and perhaps vertically, thus indicating that the dilatancy model may be applicable to essentially all types of quakes.

A wealth of other evidence for the prevalance of dilatancy phenomena in earthquakes was presented at the AGU meeting. R. Robinson of the U.S. Geological Survey, for example, reported that records of *P*-wave velocities on the San Andreas fault in central California showed variations similar to those reported by Whitcomb prior to three moderate 1972 quakes. The San Andreas fault is of the strike-slip type in the region of the quakes.

Crustal Deformation Measured

Variations of seismic velocities are an indirect confirmation of dilatancy, but more direct measures are available. A network of tiltmeters has recently been installed along part of the San Andreas fault to monitor crustal deformation. According to M. Johnston of the U.S. Geological Survey, preliminary results from these instruments indicate both long-term premonitory changes and short-term variations in the amplitude and direction of ground slope. The short-term effect was a gross change in the direction of tilt at the time of seven of eight quakes observed. Johnston's interpretation of his findings is conservative; he does not believe that the data fit a simple dilatancy model. But other geophysicists, who are excited about what appears to be direct evidence of dilatancy-induced deformation in the crust, do not agree. In any event, an abundance of data that should help to settle the question will shortly be available as the tiltmeter array is completed.

More specific confirmation of the dilatancy-diffusion model is provided by a study of the extensive data recorded during the earthquake swarms in Matsushir, Japan, from 1965 to 1967. The data on Japan include measurements of fault slip, geodetic changes, tilt, strain, water flow, seismicity, seismic velocity, electrical and magnetic variations, and gravity anomalies. The main features of these observations, according to A. Nur of Stanford University, were evidence of ground upheaval and later subsidence that were symmetric with respect to the fault, extensive flows of groundwater, and an increase and later decrease in the value of gravity measured at the surface.

These features, he believes, are exactly what would be expected from the dilatancy-diffusion model. Stress-induced swelling in the rock along the fault would cause a vertical uplift and a concomitant decrease in the value of gravity. Since the rock closest to the fault on either side should be the most stressed, the pattern of uplift should be symmetrical with respect to the fault. After a quake or series of quakes, these features should return to normal, and water which had diffused into the dilatant region should flow out again. Nur concludes that the model gives a good account of the physical phenomena, and that dilatancy and fluid flow are responsible for the observed variations in other measured quantities. And because the extent of fluid flow may vary from one region to another, depending on rock porosity, he suggests that changes in the gravitational field may be the easiest way of detecting dilatancy in practice.

Gravity measurements made before and after the 1971 San Fernando earthquake give additional support to this means of geodetic monitoring. According to H. W. Oliver of the U.S. Geological Survey, gravity changes of as much as 0.4 milligal due to the quake were found, and the changes corresponded well to changes in elevation (the ground was uplifted as much as 2 meters in some locations by the quake). Remeasurement since the earthquake shows that the area has partially collapsed, although Oliver finds some indication that the central area has begun rising again. Most existing equipment for field measurements of gravity is relatively primitive, but the technique may be less expensive than measuring ground level or seismic variations. The Survey measurements would seem to

confirm, as many geophysicists believe, that the technique has considerable potential for earthquake studies.

Other research presented at the AGU meeting included theoretical studies of the properties of dilatant rocks, models of dilatant phenomena, and more field studies. There is by no means agreement on the details of the dilatancy-diffusion model or on the extent to which it can be applied to widely varying geophysical situations. Many geophysicists, for example, question the accuracy of predictions based on any model that is not based on the measured properties of the rock in a particular location, and others believe that dilatancy on the scale necessary for a major earthquake is not likely. (In this regard the report by M. Wyss of the University of Colorado of *P*-wave velocity variations before a magnitude-7 earthquake in New Zealand, where the dilatant region was approximately 300 kilometers across, is of interest.) Nonetheless, it seems clear that most investigators are taking the model seriously and directing their research along lines indicated by it. The prospects for a better understanding of earthquake mechanisms and for earthquake predictions that may be of social and economic value both appear to be excellent, and it is correspondingly a time of considerable excitement for geophysicists.

—ALLEN L. HAMMOND

Tumor Immunology (I): The Host's Response to Cancer

No one worries about the growth of cancer cells in culture systems or in test tubes. Only when they grow in the living — human—organism is there cause for alarm. Culture systems are valuable for studying the basic mechanisms of oncogenesis, but it is the response of the whole individual to his disease that is of prime importance because this interaction between host and disease determines the patient's prognosis. Many investigators think that the immune system is a major component of an individual's response to cancer. They are now seeking the answers to two questions of fundamental importance: What is the role played by the immune system in the initiation and growth of tumors? And, how may the immune system be manipulated to cure or control cancers in humans?

Since deficiencies in the immune responses of cancer patients are well documented, there is little doubt that the immune system is somehow involved in oncogenesis. The uncertainty concerns its role—whether the deficiencies are the cause or effect of the disease and whether the immune system hinders or promotes tumor growth. Determining the nature of immune system involvement in cancer development is thus critically important for devising strategies for immunotherapy.

Immunotherapy—the manipulation of immune responses for cancer treatment—is considered by some investigators to hold the greatest promise for a cancer cure. Techniques employing surgery and radiation are restricted in application. They can eliminate the primary cancer but are of little value in controlling metastasis, the spread of cancer throughout the body. Chemotherapy, which aims to kill all cancer cells regardless of their location, has proved successful in controlling certain kinds of relatively rare cancers like Hodgkin's disease and some leukemias, but not for more common cancers like those of the lung and colon. Consequently, many investigators are turning to immunotherapeutic techniques. Some of their early clinical trials—and they emphasize the preliminary, experimental nature of the studies—have produced results that have encouraged them to proceed, but with caution.

The caution stems from observations that in some studies with animals, and possibly with humans, stimulation of the immune system produced enhancement, not inhibition, of tumor growth. Complexity appears to be the rule for cancer research, and tumor immunology is no exception. Immune responses require a number of components, including several different cell types and an assortment of "factors," which may or may not interact with one another. Thus, despite recent progress, immune response mechanisms are incompletely understood, and there is still uncertainty about how they can best be manipulated for the cancer patient's benefit.

The early history of tumor immunology research was inauspicious. Investigations at the beginning of this century purported to show that animals immunized with material prepared from a transplantable tumor resisted tumor growth when they were subsequently challenged with live tumor cells. The tumor cells grew and formed tumors in nonimmunized animals. The experiments suffered from a major flaw, however; at that time, there were no inbred strains of animals. Resistance to the tumor challenge was due, not to recognition and rejection of specific tumor antigens, but to an immune response directed against normal tissue antigens from a genetically dissimilar animal.

All cells carry genetically determined antigens. (Antigens are any substances that stimulate an immune response; most are chemically complex materials like proteins or nucleic acids.) An individual does not normally mount an immune attack on antigens of his own tissues, but cells with different antigens from another individual are recognized as foreign and attacked by the immune system. Identical twins can tolerate each other's cells because they are genetically the same. This is also true for members of the same inbred strain of laboratory animals, which have very similar, if not identical, genetic compositions. Development and use of these strains has greatly facilitated immunological research.

Scientists now think that most (but not all) tumor cells carry membrane antigens called tumor-associated antigens that do differ from those of the host's normal cells. Proving that these antigens are absolutely tumor-specific (found only in tumors and not in normal cells at any time during development) is extremely difficult and remains a major problem of tumor immunology. Investigators have established, in both in vivo and in vitro systems, that animals can mount an immune response against tumor cells. For example, after a chemically induced tumor is surgically removed from a mouse, the animal can resist tumor growth when viable cells of that same tumor are injected. It cannot