of spin. Finally changes in the rate of spin trigger earthquakes.

We must carefully state what we have here. As seismologist Lynn R. Sykes of the Lamont-Doherty Geological Observatory takes pains to point out, such a chain of events cannot *cause* earthquakes. Earthquakes are caused by stresses and strains within the earth. Nobody argues otherwise including Gribbin and Plagemann. They are saying that the chain of events they lay out could serve as a trigger, releasing an already overstrained portion of the earth's crust, and this is an idea that Sykes says cannot be dismissed out of hand.

Don L. Anderson, director of the Seismological Laboratory at the California Institute of Technology says the idea is "on its face not as ridiculous as it seems," but it does have serious weaknesses. There is a connection between the triggering of earthquakes and changes in the earth's rotation rate, but it is not yet clear which is cause and which effect. Gribbin and Plagemann have to assume that changes in the spin rate are the cause and earthquake triggering the effect. The weather does affect the spin rate, Anderson continues. and the sun does affect the weather.

The weakest link in the chain of reasoning, in Anderson's opinion, is the notion that the planets trigger sunspot activity. This is "not generally accepted by solar physicists," he says. "There is nothing to the planetary theory of sunspots." Anderson also points out that in 1803, the last time this planetary lineup occurred, no great increase in seismicity was recorded.

"I wouldn't dismiss it and say 'baloney,' " says Robert Hamilton, who is chief of the Office of Earthquake Studies of the U.S. Geological Survey. He too agrees that there is some connection between earthquakes and changes in the earth's spin rate, but he is unsure of planet-sun-atmosphere connections. He can neither pooh-pooh nor confirm Gribbin's and Plagemann's reasoning. Their picking of Los Angeles for a 1982 quake is "a leap" in Hamilton's view. They say they do it because the southern part of the San Andreas fault has gone longest (since 1857) without a major quake.

Meanwhile there is a countersuggestion that much of the San Andreas is safe from a major quake for a number of years to come, possibly as many as 25. It is by Max Wyss of the University of Colorado and appears in the Sept. 13 NATURE. Wyss's conclusion comes from a study of the velocity of P or pressure waves in the rock around the San Andreas. It has been noted that the velocity of such waves increases years before a major earthquake. Wyss finds no increases near the San Andreas and concludes that an earthquake of Richter magnitude seven is unlikely for seven years and one of magnitude eight for 25 years in the region between San Francisco and Parkfield.

The Gribbin-Plagemann prediction has stirred governmental interest. In a letter Sept. 18 to NASA Administrator James C. Fletcher, Sen. Frank E. Moss (D-Utah) asked NASA to look into it to see how plausible it is and what its impact may be. Moss, chairman of the Senate Aeronautical and Space Sciences Committee, believes the commitee may want to consider possible funding and research authorization. A member of the committee staff, Gilbert Keyes, who has been asking astronomers about the Jupiter effect, says most of them feel that the chain of events has a low probability of occurrence, but none would positively declare it impossible.

NASA's immediate response was that none of its astronomers thought much of the idea. It is too early to tell who in NASA will be assigned to answer Moss's questions, spokesmen say. The question is likely to prove a hot potato for NASA. Although Gribbin and Plagemann are reputable scientists—Gribbin is geophysics editor of NATURE, and Plagemann is an astronomer who does contract and consulting work—the idea is the sort of thing cultists like to take in their teeth and run with, and NASA already has trouble enough with cultists. On the other hand if the proposed scenario is acted out, NASA would not want to be caught with its expertise down on that great gettin'-up morning in 1982. □

# Right to treatment: A legal dilemma

In 1957 Kenneth Donaldson was diagnosed as paranoid schizophrenic and confined to the Florida State Hospital. During the next 14 years Donaldson brought 15 different legal petitions before state and Federal courts to request release. In the petitions, some of which went to the Supreme Court, the patient protested the conditions of his confinement and the lack of treatment —even though, as a Christian Scientist, he had refused the drug treatment that hospital authorities considered appropriate for his condition.

All of Donaldson's petitions were either denied or refused by the courts so he started a class action suit on behalf of the patients on his ward in the hospital. The thrust of the suit was an attack on Florida's civil commitment laws. It asked that the state provide adequate treatment for those committed to its hospitals against their will. Shortly after the right-to-treatment suit was initiated, Donaldson was discharged from the hospital. But this did not end the legal action. The class action suit was dropped and Donaldson's lawyers initiated a personal action suit against two doctors who had been in charge of the case at various times. They were charged with knowingly, intentionally and maliciously confining the patient against his will without adequate treatment. Even though the psychiatrists were following the rulings of 15 different court decisions to keep the patient in the institution, Donaldson was awarded $38,500. His suit was upheld last spring by the U.S. Court of Appeals.

The American Psychiatric Association disagrees with the decision. APA President John P. Spiegel announced this week that the association will ask the Supreme Court to review the case next month. The APA does not argue that Donaldson got adequate treatment. In fact, it admits that most large state institutions are not funded, equipped or staffed to provide good treatment. Alan Stone of Harvard Law School is the chairman of the APA Judiciary Committee. He and Spiegel both say that conditions in the Florida hospital were deplorable and horrible. The institution was so overcrowded that staff psychiatrists could afford to spend only three minutes per day with each patient.

In such situations should individual psychiatrists be held responsible? The APA feels that the institution and ultimately the state has the legal obligation to supply treatment for involuntarily committed patients. And this is what the APA wants the Supreme Court to rule on. State institutions would then be forced to go to the legislature for funds to ensure adequate treatment. In this way psychiatrists who have 500 or up to 1,000 patients to treat would not be held responsible for the shortcomings of the system. Psychiatrists would still, Spiegel points out, be responsible for their own clinical decisions and still be open to malpractice suits.

If the Supreme Court hears the case and rules against the APA's position, things can only get worse, Spiegel and Stone warn. Psychiatrists in state institutions will have two options. They could release any patient who complains about treatment or they could protect themselves by going to work somewhere else. If patients are released early, they will still not be getting adequate treatment. Released patients will end up on welfare or in already overcrowded nursing homes, says Stone. And if the few psychiatrists available leave the state institutions, those left behind will only be more overworked and less able to offer adequate treatment.

The APA's attempt to shift the burden

of responsibility to the state sounds reasonable in some respects, but it actually does nothing but name a different scapegoat. The states are no more able than the psychiatrists to get legislatures to appropriate enough money for mental health institutions. "What is really needed," says Spiegel, "is major institutional change. . . . Perhaps," he goes on, "the present mental health treatment crisis is good. It might force some changes in a system that has been too long without change and bring in a new breed of psychiatrist." □

# Preventing sudden infant deaths

After the neonatal period, the sudden death syndrome is the greatest single cause of death during the first year of life. The typical victim is a healthy-looking infant who dies silently during sleep. The causes of sudden infant deaths are obscure. Now scientists in Britain have identified some of the risk factors for sudden infant deaths and have shown that the knowledge can be used to prevent such deaths.

To determine the means of identifying high-risk babies at or soon after birth, R. G. Carpenter of the London School of Hygiene and Tropical Medicine and J. L. Emery of The Children's Hospital, Sheffield, studied the obstetric and perinatal histories of 254 infants born in the same hospitals. One hundred and thirty-five of the infants were healthy; 119 had died suddenly.

The investigators found that the babies who had died suddenly had shared certain experiences. Their mothers had often been young, had had blood groups O, B or AB and had not breast fed them. They had more likely than not been born prematurely and had been far along in birth order. There was also a good chance that their mothers had had a urinary infection during pregnancy.

Having identified these risk factors, Carpenter and Emery then set up a study to see whether they could use the risk information to prevent sudden infant deaths. They examined 6,003 newborns during 1973 for the risks. They then selected half the newborns at high risk and followed them closely. The infants were examined 48 hours after birth, five weeks after birth and received 10 home visits during the first 20 weeks of life. The other half of the newborns at high risk were not followed up clinically.

The study showed that there were 6.1 times more deaths among the endangered newborns who were not followed clinically than there were among the endangered infants who were. So Carpenter and Emery conclude in the Aug. 30 NATURE that identifying infants with a potential risk from sudden death, and keeping close tabs on them, helped prevent such deaths. □

# Hospital infections traced to hamsters

Hospital-induced infections are a major problem in health care (SN: 7/20/74, p. 44). Patients' infections have been traced to the germs in sinks, flower vases and pails. A report in the Sept. 27 SCIENCE traces the infection of several hospital personnel to a virus carried by research hamsters. This same virus caused, in the words of one physician, an "epidemic" of illness amongst pet hamster owners in California, Florida, Georgia and other states earlier this year and is now being studied by Government scientists.

Hamsters often are used in research laboratories to incubate human tumor cells. They can carry a virus infection called lymphocytic choriomeningitis (LCM), which is hard to detect in the animal and can cause the onset of flu-like symptoms in humans.

Five New York state health department scientists, John Hotchin, Edward Sikora, William Kinch, Alan Hinman and John Woodall, report a series of hamster-induced illnesses at Strong Memorial Hospital, Rochester, N.Y. The illnesses took place over several months, but have now subsided. Most of the approximately 50 cases occurred after hospital personnel visited a room in the basement of the radiotherapy department where a photocopier and 200 hamsters were kept. After several cases, LCM was suspected, and the serological tests confirmed the suspicion. No patients are believed to have been infected.

The findings are important for several reasons. First, Hotchin told SCIENCE NEWS, the lives of patients, weakened by immunosuppressants or diseases, might be jeopardized if they are exposed to the virus while in the hospital. Second, tumor research findings will be invalidated if hamsters are carrying the virus, because it prolongs the life of certain test animals with tumors. Third, and perhaps of broadest significance, Hotchin says, the hamsters came from a nationwide pet distributor, and more than 100 cases of pet hamster owners contracting LCM-like symptoms have been reported this year.

Paul D. Walter, a medical epidemiologist at the Center for Disease Control in Atlanta, is reviewing these cases and soon will publish results on the extent of the problem. Most of the cases occurred this spring, and "We are not aware that the epidemic is still going on," Walter says. Self-monitoring by the pet industry seems to have controlled the outbreak, but "the question of whether standards should be established by the Government or the pet industry still has not been settled."

A spokesman from Aquarium Supply Co. of Harrison, N.J., whose supplier was implicated in the distribution of the contaminated hamsters, says Government controls are unnecessary because the industry is now policing itself, and the supplier "is now shutdown." He agreed laboratory animals should be monitored regularly to prevent ruined experiments and danger to patients, but emphasizes that pet owners need not fear contamination. □

# Vitamin E retards cellular aging

Many virtues have been claimed for vitamin E, one of which is that it retards aging. Certainly there is some clinical evidence that it can. When antioxidants such as vitamin E were given to rodents, the drugs extended the animals' life spans 30 percent (SN: 12/23/72, p. 413). Now Berkeley physiologists have found that vitamin E can also retard the normal aging process in laboratory cultured human cells.

Most normal cells in culture have a definite lifespan. Human embryonic lung cells will divide and reproduce about 50 times before they die. Lester Packer and James R. Smith of the Lawrence Berkeley Laboratory added vitamin E to human embryonic lung cells. The cells have divided 120 times and are still dividing and appear young and healthy.

Packer and Smith have also obtained results that reinforce the theory that vitamin E, as a natural antioxidant, counters environmental pollutants before they oxidize (damage) molecules in cells. They exposed vitamin E-treated cells and control cells to two typical environmental stresses—oxygen and visible light. They found that although the stresses killed 90 percent of the control cells, the stresses killed only 35 percent of the treated cells.

On the basis of these findings, in press with the PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, Packer and Smith conclude that vitamin E is not a panacea for all aging processes. But they do believe that vitamin E will extend life where humans are subjected to severe environmental pollution. "It might," they speculate, "prevent an early death, or brain disease, heart attacks or senility." □