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 healthylooking 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 flulike 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 Sci-ENCE 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 Etreated 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."

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