

Soviet psychiatrist describes abuse

In February 1981, Soviet psychiatrist Anatoly Koryagin was arrested and sent to a prison camp after he examined several political dissidents confined to a mental hospital and concluded that they had no psychiatric disorders. Koryagin and his family were allowed to leave the Soviet Union last month and now live in Switzerland. At a press conference, Koryagin called for the establishment of an international tribunal to monitor psychiatric abuse worldwide and recounted his experiences as a prisoner of conscience.

From his first days in the prison camp, Koryagin says he protested the conditions, particularly the poor medical care and nutrition. He went on periodic hunger strikes, and as a result was put in solitary confinement. A tube was run through his nose and into his stomach so he could be force-fed nutrients mixed with powerful antipsychotic medications, as well as drugs that induced rapid heartbeats and irregular heart rhythms. The tube inserted in his nose was coated with a corrosive substance that caused extreme pain. He was handcuffed during force-feeding.

"I was virtually cut off from any news of the outside world," Koryagin said through an interpreter. Occasional breaks in this isolation, such as a visit from his wife after his first hunger strike with news of international efforts to free him, "gave me an enormous boost."

Koryagin knows of 183 "victims of psychiatric oppression" still in the Soviet Union, but adds that there are certainly many others he is not aware of. He says there are now 16 special psychiatric facilities for political dissidents, up from 11 in 1977.

"My personal view is that no psychiatric association should have any contact with official Soviet psychiatry," says Koryagin. Private contacts with Soviet psychiatrists should, however, be encouraged, he adds. "Only the pressure of world opinion will help to stop the abuse of psychiatry in the Soviet Union," he says.

The official Soviet psychiatric society withdrew from the World Psychiatric Association in 1983 (SN: 2/19/83, p.116). Controversy persists in the American Psychiatric Association over whether to resume formal contacts with the Soviet group.

The aging of immunity

Communication between the brain and the immune system appears to be more complex than many scientists have assumed. For example, in initial studies Steven J. Schleifer of Mount Sinai School of Medicine in New York City and his colleagues found that some immune responses of bereaved men and severely depressed patients are weaker than those of healthy controls (SN: 2/16/85, p.100). But the same researchers now report that immune measures are far more likely to decline in depressed individuals who are middle-aged or older.

Schleifer and his co-workers used two substances to stimulate lymphocyte reproduction among 88 severely depressed patients and 88 healthy controls. Subjects ranged in age from 18 to 80. Depressed patients in their mid-40s and older had a smaller proliferation of lymphocytes than age-matched controls, while younger patients had the same or, in a few cases, greater lymphocyte responses than controls. In addition, the numbers of T-helper cells and natural killer cells, two components of the immune system, were markedly lower only among older depressed patients. The most severe cases of depression were associated with lower immune measures at all ages.

"We don't know what's going on in the immune systems of depressed people in relation to age," says Schleifer. For instance, he explains, depression that begins during adolescence may alter immune function differently than depression that begins during middle age. "There's no simple relationship between immunity and depression," adds Schleifer.

New tissue eases Huntington's disease

University of Cincinnati researchers report that transplanting brain tissue from healthy rat fetuses into rats with chemically induced Huntington's disease "can produce remarkable recovery of function of both locomotor and more complex psychological tasks." The results confirm and extend earlier reports, which were more preliminary and narrower in scope, according to psychiatrist Paul R. Sanberg and his colleagues.

The investigators injected the rats' brains with normal cells from the striatal portion of fetal brains. The striatum appears to be the primary site of disease in the brains of Huntington's victims. In this and other rat experiments, the striatum was substantially damaged — and Huntington's was simulated — by injections with kainic acid. Although previous work had established varying degrees of recovery with such transplants, the Cincinnati scientists performed several transplantations on each rat "in order to determine if more complete recovery of function than that previously found could be obtained."

At three, six and nine weeks following the transplant injections, the rats were tested on various motor and other activity measures. The results were compared with those of Huntington's diseased rats that received "sham" transplants of adult sciatic nerve and with those of healthy rats that also received sciatic transplants.

The researchers found that while the sham-transplant rats with Huntington's remained "consistently hyperactive" during the nine-week testing period, "the hyperactivity exhibited by the striatal transplant group prior to transplantation decreased gradually following implants until they reached control levels nine weeks later." And, when injected with amphetamines, the experimental rats did not display the "exaggerated" response of the sham transplant group. Other, more complex movements such as rearing up and rotation also appeared to improve in the transplanted rats.

Moreover, the researchers report, examination of the brains following the experiment revealed that "the striatal transplants reconstructed much of the gross morphology of the lesioned [damaged] striatum in recovered animals."

"The present, preliminary findings," they say, "confirm [previous] results and extend them by demonstrating that large fetal transplants can completely reverse some aspects of . . . locomotor activity in rats with large . . . striatal lesions."

Caffeine jolt for ECT

While the debate continues over whether or not electroconvulsive therapy (ECT), or shock therapy, should be used in the treatment of severe depression, those psychiatrists who do use the technique have encountered a frequent problem: As the course of ECT progresses, some patients seem to become more tolerant of the shock, and the length of their seizures decreases. Theoretically, this would weaken any "therapeutic effect" derived from ECT.

If the electric charge were strengthened to combat this problem, it would carry with it some obvious, inherent dangers, says C. Edward Coffey, a psychiatrist at Duke University Medical Center in Durham, N.C. Coffey's answer? Caffeine.

In a study with 20 patients hospitalized due to severe, or "major," depression, Coffey reports that pre-ECT injection with 250 to 750 milligrams of caffeine resulted in an average increase of seizure duration of 127 percent. He found that higher doses of caffeine resulted in longer seizures. Although "there were no associated adverse effects or prolonged post-ECT disorientation," says Coffey, three patients did suffer from temporarily heightened anxiety.

Based on these and similar results in a double-blind study, Coffey concludes that "caffeine injections may be a potentially safe and highly effective technique to augment ECT."