

## Mental illness from psychiatric drugs?

The powerful tranquilizers that are commonly used to control schizophrenic psychosis and to manage problem behavior may in some cases be doing as much damage as good. Two recent studies and a book-length review of the literature on psychiatric medication indicate that the popular class of drugs called "neuroleptics" may actually be causing serious mental and emotional problems that are unrelated to the original disorders. The hypothesis remains highly controversial, however, with critics arguing that the reported behavioral deterioration may well occur in the natural course of chronic mental illness.

Debate about the psychological effects of neuroleptic (or antipsychotic) drugs has been going on almost since the first, chlorpromazine, was successfully used in 1953. Long-term use of these drugs (which include Mellaril, Thorazine, and Haldol) has been associated with a physical disorder called tardive dyskinesia, a syndrome characterized by involuntary movements and thought to be caused by drug-induced changes in the movement coordination area of the brain; since the same physiological change, a supersensitivity to the neurotransmitter dopamine, could theoretically take place in the dopamine pathways of the brain's limbic system as well, it has been suggested that the drugs might affect mental and emotional functioning, too. The hypothesized limbic system counterpart to tardive dyskinesia has been called iatrogenic schizophrenia, tardive psychosis, subcortical dementia, and — most recently — "tardive dysmentia."

The term "tardive dysmentia" was coined by the late Ian C. Wilson, who as a psychiatrist at the Dorothea Dix Hospital in Raleigh, N.C., observed signs of emotional disturbance among schizophrenics during the 30-year period coinciding with the growing use of psychiatric drugs. When Wilson and co-worker James C. Garbutt investigated, they found (and report in the current *SCHIZOPHRENIA BULLETIN*) that the unusual behavioral syndrome — euphoria, unstable mood, and manic interactions with people — was closely associated with the worst cases of tardive dyskinesia — suggesting that the syndrome might be related to extensive drug use.

Another researcher, psychiatrist C. Thomas Gualtieri of the University of North Carolina in Chapel Hill, has found what he considers even stronger evidence for a behavioral analogue of tardive dyskinesia. Studying mentally retarded children and young adults, Gualtieri found cases of acute behavioral deterioration — including aggression, destructiveness, screaming and insomnia; as is often true with dyskinesia, these behaviors showed

up only after the drugs were discontinued and were qualitatively different from the original behaviors for which the neuroleptics were prescribed. Although it is not possible to know if the drugs caused the behavioral changes, he says, the most appealing explanation is that the behaviors resulted from dopamine hypersensitivity in the brain's cortex and limbic area.

Others remain unconvinced. According to psychiatrist Daniel E. Casey of the Veterans Administration Medical Center in Portland, Ore., it is too early to rule out an alternative hypothesis — namely, that behavioral changes are part of the chronic schizophrenic process. In his own research with monkeys, he notes, he has found that abnormal movements occur spontaneously with aging; in addition, he says, 19th century descriptions of schizophrenia include references to movement disorders very much like what is today called tardive dyskinesia. So it is not possible to blame even the dyskinesia entirely on drugs, much less to conclude that dyskinesia has a psychological counterpart. Psychiatrist Kenneth L. Davis of the Bronx VA Hospital agrees; he was among the first to hypothesize that there might be a limbic system equivalent of tardive dyskinesia, but he says that after three years of study the data are inconclusive.

Peter Breggin, a Bethesda, Md., psychiatrist and author of the recently published book, *Psychiatric Drugs: Hazards to the Brain*, argues that the evidence is overwhelming; psychiatrists, he says, simply cannot admit that they have effectively "lobotomized" millions of patients with chemicals that are toxic to the brain. The euphoria that Garbutt and Wilson report is a symptom of dementia, or general brain deterioration, Breggin says; in fact, he adds, the literature on tardive dyskinesia reveals that most people with the movement disorder are also demented. The euphoria changes to apathy as the brain continues to deteriorate, Breggin says, and ultimately the chemical damage to the frontal lobes accomplishes the same thing as psychosurgery. Only a lobotomized patient would be indifferent about a disorder like tardive dyskinesia, as most older chronic schizophrenics are, according to Breggin.

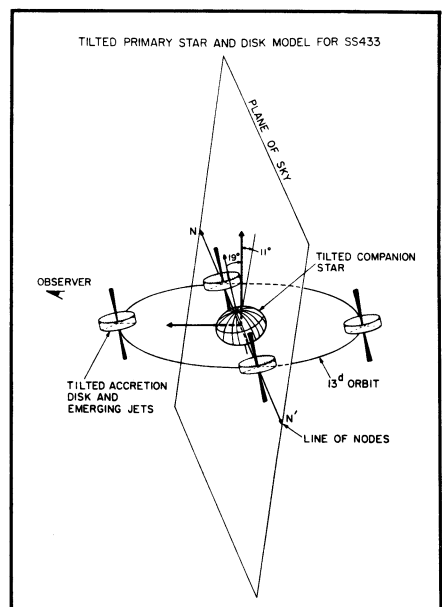
Even if it were possible to confirm a causal connection between neuroleptic drugs and tardive dysmentia, many psychiatrists say that some schizophrenics are so psychotic that they cannot function off drugs. But according to Gualtieri, neuroleptics are often used inappropriately — to manage rebelliousness in children, for example; in one recent study in which he withdrew children from drugs, he found that fewer than one in five required neuroleptic treatment. Psychiatrists, Gualtieri says, should be less aggressive in prescribing neuroleptics and more vigilant in monitoring the side effects of those that they must prescribe.

—W. Herbert

## X-raying the odd object SS433

SS433 was just a number in a catalog of peculiar astronomical objects until a few years ago when astronomers discovered that there are patterns of resonant emission lines in the spectrum of its light that move back and forth in wavelength in cycles (SN: 4/28/79, p. 277). Complicated motions in the source would produce such cycles. Astronomers rushed to observe SS433 in light, radio and X-rays and to devise models to explain the spectral data.

The Einstein X-ray Observatory satellite observed SS433 numerous times over an 18-month period. Jonathan E. Grindlay of the Harvard-Smithsonian Center for Astrophysics in Cambridge, Mass., and colleagues from the CFA, the Marshall Space Flight Center in Huntsville, Ala., and the Goddard Space Flight Center in Greenbelt, Md., will publish a compilation and analysis of these data in the Feb. 1, 1984 *ASTROPHYSICAL JOURNAL*. In it they state that the X-ray data generally support the model of SS433 that had been derived from optical and radio observations.



The lines in SS433's visible spectrum change according to two cycles, one of 13 days, the other of 164 days. SS433 is also a radio source consisting of a compact central object flanked by two teardrop-shaped lobes embedded in a more tenuous cloud that astronomers consider to be a remnant of a bygone supernova explosion. In X-rays it shows a similar configuration of a compact central object and flanking lobes.

The model that emerged is a binary star system in which a very compact object (a neutron star or a black hole) orbits around a companion, which is an ordinary star of some kind. The 13-day period is the binary orbit. The 164-day period is the precession period of the compact object's rotation. In