should the use of neuroleptics be severely limited?[1]

Peter Breggin

In 1954 chlorpromazine (Thorazine) broke like a tidal wave across the state mental hospitals of the Western world. A dozen new neuroleptics were introduced into the market, and their use became focused on the psychoses, especially schizophrenia and acute mania. Several hundred million people worldwide have now received them, often involuntarily.

The neuroleptics drugs have gradually become promoted as agents with a specific "antipsychotic" effect on schizophrenic symptoms. Meanwhile, psychosocial approaches have fallen into disrepute among many psychiatrists. Patients have been instructed to remain on neuroleptics for a lifetime and told that it was safe to do so. The public was told that the "miracle" drugs had emptied the hospitals and returned millions of patients to normal lives.

the reality

In 1973, psychiatrist George Crane gained the attention of the medical community by disclosing that many, and perhaps most, long-term neuroleptic patients were developing a largely irreversible, untreatable neurological disorder, tardive dyskinesia (Crane, 1973). The disease, even its mild forms, is often disfiguring, with involuntary movements of the face, mouth or tongue. Frequently, the patients grimace in a manner that makes them look "crazy", undermining their credibility with other people. In more severe cases, patients become disabled by twitches, spasms, and other abnormal movements of any muscle groups, including those of the neck, shoulders, back, arms and legs, and hands and feet (American Psychiatric Association, 1992; Breggin, 1983; 1990; 1991). The muscles of respiration and speech can also be impaired. In the worst cases, patients thrash about continually.

The rates for tardive dyskinesia are astronomical. The latest estimate from the American Psychiatric Association (1992, p. 68) indicates a rate for all patients of five per cent per year, so that 15 per cent of patients develop tardive dyskinesia within only three years. In long-term studies, the prevalence of tardive dyskinesia often exceeds 50 per cent of all treated patients and is probably much higher. The disease affects people of all ages, including children, but among older patients rates escalate. In a controlled study, 41 per cent of patients aged 65 and older developed tardive dyskinesia in a

Peter Breggin is a psychiatrist and Director of The Centre for the Study of Psychiatry, Bethesda, Maryland.

CCC 0263-8371/96/010062-05

[1]This article was first published in *Controversial Issues in Mental Health* edited by Stuart A. Kirk and Susan D. Einbinder (pub. Allyn and Bacon) and is reprinted with kind permission. It is an updated version of a talk given for the Shropshire Mental Health Institute.

mere 24 months (Yassa et al., 1988). Hundreds of thousands of older people receive these drugs in nursing homes and state hospitals.

Other closely related, untreatable neurological disorders have now been recognised as variants of tardive dyskinesia. Tardive akathisia involves painful feelings of inner tension and anxiety and a compulsive drive to move the body. In the extreme, the individual undergoes internal torture and can no longer sit still. Tardive akathisia often develops in children who have been treated for "hyperactivity", ironically and tragically subjecting them to permanent inner torture. Tardive dystonia involves muscle spasms, frequently of the face, neck and shoulders, and it too can be disfiguring, disabling and agonizing.

There are no accurate surveys of the total number of patients afflicted with tardive dyskinesia. There are probably a million or more tardive dyskinesia patients in the United States today, and tens of millions have been afflicted throughout the world since the inception of neuroleptic treatment (Breggin, 1991). Despite this tragic situation, psychiatrists too often fail to give proper warning to patients and their families. Often psychiatrists fail to notice that their patients are suffering from tardive dyskinesia, even when the symptoms are flagrant (Brown and Funk, 1986; Breggin, 1991).

In 1983 I published the first in-depth analysis of the vulnerability of children to a particularly virulent form of the tardive dyskinesia that attacks the muscles of the trunk, making it difficult for them to stand or walk. This is now an established fact. In the same medical book, I offered the first detailed documentation showing that many or most tardive dyskinesia patients also show signs of dementia—an irreversible loss of overall higher brain and mental function. Indeed, it was inevitable that these losses would occur. The basal ganglia, which are afflicted in tardive dyskinesia, are richly interconnected with the higher centres of the brain, so that their dysfunction almost inevitably leads to disturbances in cognitive processes (for the functional neuroanatomy, see Alheid et al., 1990). Since my observations, a multitude of studies have confirmed that long-term neuroleptic use is associated with both cognitive deterioration and atrophy of the brain (Breggin, 1990; Gualtieri and Barnhill, 1988). While defenders of the drugs sometimes claim that this mental and neurological deterioration is caused by schizophrenia itself, their position is untenable. More than 100 years of autopsy studies of patients labelled as schizophrenic failed to show any such deterioration, until the recent advent of neuroleptics.

Growing evidence indicates that these drugs produce tardive psychoses that are irreversible and more severe than the patients' prior problems. In children, permanent behavioural or mental disorders frequently develop as a result of the drugs (Gualtieri and Barnhill, 1988). Furthermore, drug withdrawal often causes rebound of the anticholinergic neurotransmitter system, resulting in a flu-like syndrome that includes emotional upset, insomnia, nausea and vomiting. Many patients find themselves unable to stop taking the drugs, suggesting that we should consider them as addictive (Breggin, 1989a, 1989b).

Shocking as it may seem, this brief review can only scratch the surface of neurological disorders associated with these drugs, let alone the vast number of other potentially serious side effects. For example, in a small percentage of patients the neuroleptic reaction goes out of control, producing neuroleptic malignant syndrome. The disorder is indistinguishable from an acute inflammation of the brain comparable to lethargic encephalitis (Breggin, 1990, 1991) and can be fatal.

Given that these are exceedingly dangerous drugs, what about their advantages? How do they "work"? It is well known that these drugs suppress

dopamine neurotransmission in the brain, directly impairing the function of the basal ganglia and the emotion-regulating limbic system and frontal lobes and indirectly impairing the reticular activating system as well. The overall impact is a chemical lobotomy—literally so, since frontal lobe function is suppressed (Breggin, 1983, 1991). The patient becomes de-energised or de-enervated. Will or volition is crushed, and passivity and docility are induced. The patient complains less and becomes more manageable. Despite the claims made for symptom cure, multiple clinical studies document a non-specific emotional flattening or blunting effect (reviewed in Breggin 1983, 1991).

There is no significant body of research to prove that neuroleptics have any specific effect on psychotic symptoms, such as hallucinations and delusions. To the contrary, these remain rather resistant to the drugs. The neuroleptics mainly suppress aggression, rebelliousness, and spontaneous activity in general. This is why they are effective whenever and wherever social control is at a premium, such as in mental hospitals, nursing homes, prisons, institutions for persons with developmental disabilities, children's facilities and public clinics, as well as in Russian and Cuban psychiatric political prisons. Their widespread use for social control in such a wide variety of people and institutions makes the claim that they are specific for schizophrenia ridiculous. (They are even used in veterinary medicine to bend or subdue the will of animals. When one of our dogs was given a neuroleptic for car sickness, our daughter observed, "He's behaving himself for the first time in his life".)

The neuroleptics are supposedly most effective in treating the acute phase of schizophrenia, but a recent definitive review of controlled studies showed that they perform no better than sedatives or narcotics and even no better than placebo (Keck et al., 1989). One psychiatrist (Turns, 1990) responded to these revelations with anguished questions: "Has our clinical judgement about the efficacy of antipsychotics been a fixed, encapsulated, delusional perception ... Are we back to square one in antipsychotic psychopharmacology?".

That the neuroleptics emptied the U.S. mental hospitals is a myth. The drugs were in widespread use as early as 1954 and 1955, but the hospital population did not decline until nearly ten years later, starting in 1963. That year the federal government first provided disability insurance coverage for mental disorders. The States could at last relieve themselves of the financial burden by refusing admission to new patients and by discharging old ones. The discharged patients, callously abandoned by psychiatry, received a small federal cheque for their support in other facilities, such as nursing or board and care homes. Some patients went home as dependents while others went onto the streets. Follow-up studies show that very, very few patients became independent or led better lives following these new policies (Mosher and Burti, 1989; Breggin, 1991).

But are there better psychosocial alternatives? Controlled studies by Loren Mosher have shown that patients diagnosed with acute schizophrenia improve better without medication in small home-like settings run by non-professional staff who know how to listen and to care (Mosher and Burti, 1989). The patients become more independent, and do so at no greater financial cost, because non-professional salaries are so much lower. As an enormous added benefit, the drug-free patients do not get tardive dyskinesia or tardive dementia, as well as other drug-induced and sometimes life-threatening disorders.

Controlled studies by Karon and Vandenbos (1981) indicate that even in traditional psychiatric facilities psychotherapy is the treatment of choice for patients labelled as schizophrenic. My own experience in psychiatry began as a

college student volunteer in a State mental hospital. We proved that untrained college students, with only minimal supervision, could work as case aides to help nearly all of our chronic patients leave the hospital (Breggin, 1991).

But isn't schizophrenia a biochemical and genetic disease? In reality, there's no convincing evidence that schizophrenia is a biochemical disorder. While there are a host of conjectures about biochemical imbalances, the only ones we know of in the brains of mental patients are those produced by the drugs. Similarly, no substantial evidence exists for a genetic basis of schizophrenia. The frequently cited Scandinavian genetic studies (Kety et al., 1975; reviewed in Breggin, 1991) actually confirm an environmental factor while disproving a genetic one. Such conclusions may seem incredible to readers who have been bombarded with psychiatric propaganda, and I can only hope they will personally review the literature and read *Toxic Psychiatry* for a review and analysis. But even if schizophrenia were a brain disease, it would not make sense to add further brain damage and dysfunction by administering neuroleptics.

If the neuroleptics are so dangerous and have such limited usefulness, and if psychosocial approaches are relatively effective, why is the profession so devoted to the drugs? The answer lies in maintaining psychiatric power, prestige, and income. What mainly distinguishes psychiatrists from other mental health professionals, and of course from non-professionals, is their ability to prescribe drugs. To compete against other mental health professionals, psychiatry has wed itself to the medical model, including biological and genetic explanations, and physical treatments. It has no choice: anything else would be professional suicide. In providing psychosocial therapies, psychiatry cannot compete with less expensive, more helpful non-medical therapists, so it must create myths that support the need for medically trained psychiatrists.

After falling behind economically in competition with psychosocial approaches, psychiatry formed what the American Psychiatric Association now admits is a "partnership" with the drug companies (Sabshin, 1992). Organised psychiatry has become wholly dependent for financial support on this unholy collaboration with the pharmaceutical industry (Breggin, 1991). To deny the effectiveness of drugs or to admit their dangerousness would result in huge economic losses on every level from the individual psychiatrist who makes his or her living by prescribing medication, to the American Psychiatric Association which thrives on drug company largesse.

If neuroleptics were used to treat anyone other than mental patients, they would have been banned a long time ago. If their use wasn't supported by powerful interest groups, such as the pharmaceutical industry and organised psychiatry, they would be rarely used at all. Meanwhile, the neuroleptics have produced the worst epidemic of neurological disease in history. At the least, their use should be severely curtailed.

Beyond the specific issue of the neuroleptics, there is a much broader one—how are we to understand and to show care for people who undergo emotional pain and anguish (Breggin, 1991, 1992; Mosher and Burti, 1989). Are we to view them as defective objects or as human beings struggling with emotional and social problems and personal conflict? Are we to drug them into oblivion, or are we to understand and empower them? Giving a drug disempowers the recipient. It says, "You are helpless in the face of your problems. You need less feeling and energy, and less brain function". The true aim of therapy should be to strengthen and to empower the individual. People, not pills, are the only source of real help.

references

Alheid, G. F., Heimer, L. and Switzer, III, R. D. Basal ganglia. In G. Paxinos (ed.) *The human nervous system*. New York: Academic Press

American Psychiatric Association (1992) *Task force on tardive dyskinesia*. Washington DC: APA

Breggin, P. R. (1983) *Psychiatric drugs*. New York: Springer

Breggin, P. R. (1989a) Addiction to neuroleptics? (letter) *American Journal of Psychiatry, 146*, 560

Breggin, P. R. (1989b) Dr Breggin replies (follow-up letter on addiction to neuroleptics). *American Journal of Psychiatry, 146*, 1240

Breggin, P. R. (1990) Brain damage, dementia and persistent cognitive dysfunction associated with neuroleptic drugs. Evidence, etiology, implications. *Journal of Mind and Behavior, 11*, 425–64

Breggin, P. R. (1991) *Toxic psychiatry*. New York: St Martin's Press

Breggin, P. R. (1992) *Beyond conflict*. New York: St Martin's Press

Brown, P. and Funk, S. C. (1986) Tardive dyskinesia: barriers to professional recognition of an iatrogenic disease. *Journal of Health and Social Behavior, 27*, 116–32

Crane, G. (1973) Clinical psychopharmacology in its 20th year. *Science, 181*, 124–8

Gualtieri, C. T. and Barnhill, L. J. (1988) Tardive dyskinesia in special populations. In M. E. Wolf and A. D. Mosnaim (eds) *Tardive dyskinesia*. Washington DC: American Psychiatric Press

Karon, B. and Vandenbos, G. (1981) *The psychotherapy of schizophrenia*. New York: Jason Aronson

Keck, P. E. Jr., Cohen, B. M. and Baldessarini, R. (1989) Time course of antipsychotic effects of neuroleptic drugs. *American Journal of Psychiatry, 146*, 1289–92

Kety, S., Rosenthal, D., Wender, P., Schulsinger, F. and Jacobsen, N. B. (1975) Mental illness in the biological and adoptive families of adopted individuals who have become schizophrenic. In E. Fieve, D. Rosenthal and H. Brill (eds) *Genetic research in psychiatry*. Baltimore: Johns Hopkins

Mosher, L. R. and Burti, L. (1989) *Community mental health*. New York: Norton

Sabshin, M. (1992, 10 March) To aid understanding of mental disorders. *New York Times A*, 22

Turns, C. N. (1990) Effects of sedatives and neuroleptics (letter). *American Journal of Psychiatry, 147*, 1576

Yassa, R., Nastase, C., Camille, Y. and Belzile, L. (1988) Tardive dyskinesia in a psychogeriatric population. In M. D. Wolf and A. D. Mosnaim (eds) *Tardive dyskinesia*. Washington DC: American Psychiatric Press