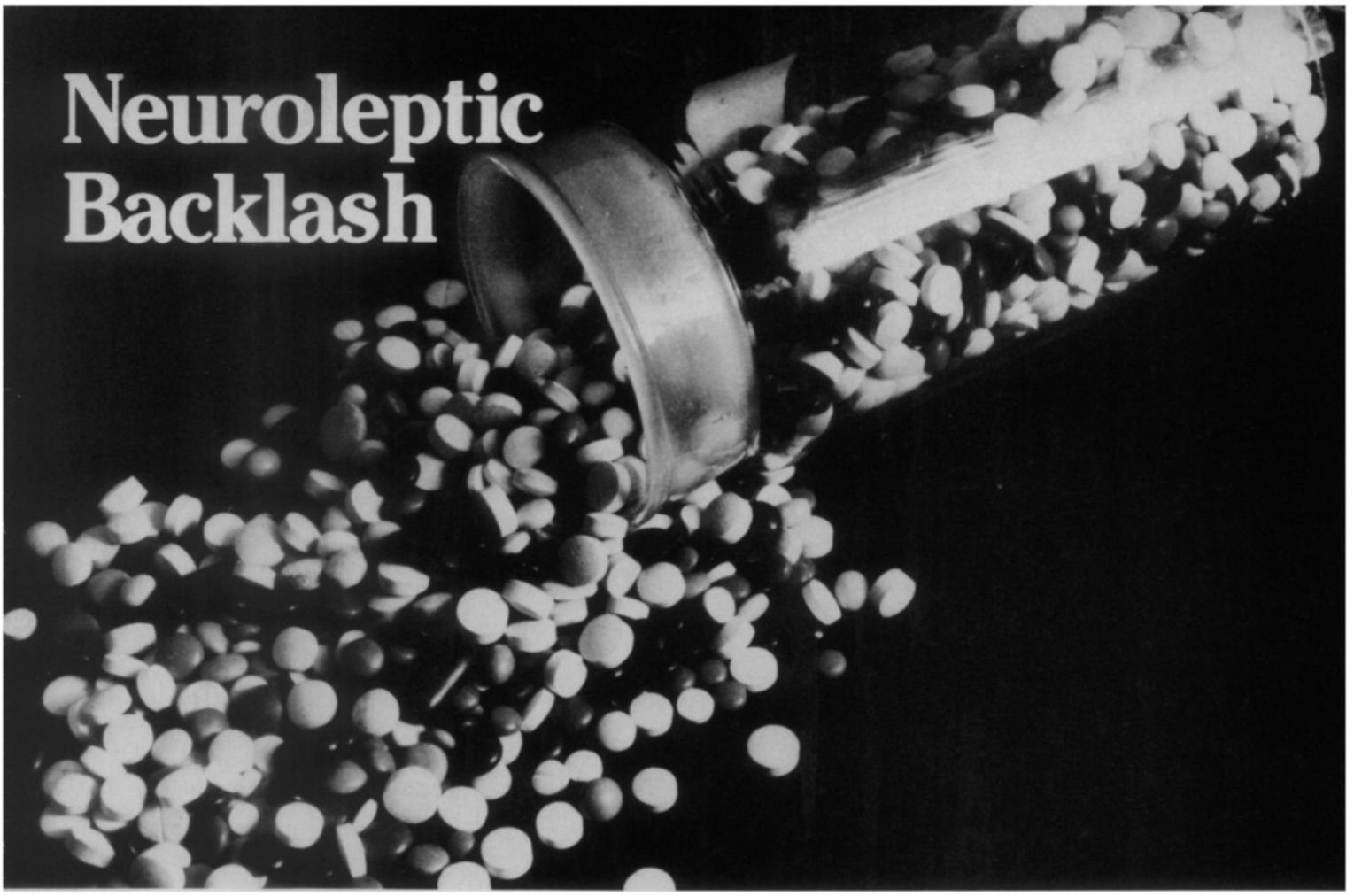
Neuroleptic Backlash



By BRUCE BOWER

Psychiatrists are getting an increasingly bad case of jitters over tardive dyskinesia (TD), a movement disorder associated with the powerful drugs that are often used to ease psychotic symptoms. Their dis-ease is being stoked by several successful malpractice suits filed by patients who developed TD after long periods of treatment with antipsychotic drugs, also known as neuroleptics.

TD is characterized by a rapid, involuntary twitching or writhing of the mouth, lips, tongue, arms, legs or trunk. The abnormal movements often appear after at least six months of drug treatment. Many cases are mild and do not get worse. However, severe, disabling forms of the disorder occur in both adults and children.

A few of the more severe cases have resulted in hefty, court-ordered malpractice payments. A mentally retarded man developed TD after receiving neuroleptics at an Iowa state institution and won a $760,000 lawsuit in 1982; the same year, a Michigan woman who had been treated with the drugs after two psychotic episodes was awarded over $1 million because several psychiatrists failed to diagnose her TD and continued to administer neuroleptics in the face of severe abnormal movements and the woman's objections; and a Minnesota Veterans Administration hospital was ordered this year to pay nearly $2.2 million in damages to a man who was not regularly monitored by physicians while taking antipsychotic drugs that led to severe TD.

Concern about drug-induced movement disorders and the need for cautious use of neuroleptics is spreading beyond the courts. The American Psychiatric Association (APA) will soon send a letter to its 30,000 members summarizing the latest information on TD and recommending careful monitoring of drug treatment and periodic discussions of medication risks and benefits with patients and their families. Beginning June 1, the Food and Drug Administration required the 19 neuroleptics approved for use in this country to carry a detailed statement about TD and its risks; the drugs previously carried a brief warning.

Despite the recent flurry of activity, TD has been described in professional journals since 1957. A 1980 APA report included guidelines to help prevent TD, but they have been honored "more in the breach than the keeping," says psychiatrist C. Thomas Gualtieri of the University of North Carolina at Chapel Hill. One gauge of neuroleptic use, the number of prescriptions charted by the FDA, remained about the same in 1983 as it was in 1973: 17 million to 19 million. It is estimated that up to 3 million people in the United States, from schizophrenics to the mentally retarded to autistic children, take antipsychotic medication each year.

Psychiatrists at the recent APA annual meeting in Dallas filled in more of the current TD picture.

"It's very difficult to study risk factors for tardive dyskinesia," says Nina Schooler of the National Institute of Mental Health in Bethesda, Md. "One possibility, though, is to manipulate the drug treatment itself." For example, preliminary studies indicate that, for many schizophrenics, an antipsychotic drug known as fluphenazine is most effective at doses up to one-tenth of those usually prescribed (SN: 11/10/84, p. 297). But the patients most likely to develop severe movement disorders cannot be identified, she notes.

Another difficulty for researchers, adds Schooler, is the paradoxical nature of TD. "The drugs that cause tardive dyskinesia in a sense treat it by masking its appearance," she explains. For an as-yet-unknown reason, movement disorders most often begin to appear when neuroleptics are discontinued.

To make matters more perplexing, TD does not always get worse as drug treatment continues. Daniel E. Casey of the Veterans Administration Medical Center in

Portland, Ore., reports that the movement disorders of 48 American and Danish psychiatric patients improved or remained stable three to 10 years after standard neuroleptic treatment was switched to low doses. "This is a rare example of good news about tardive dyskinesia," he says. "We can manage psychosis [with medication] and cut down the risk of TD."

Estimates of the percentage of neuroleptic-treated psychiatric patients with TD have varied widely, from 0.5 percent to 56 percent. John M. Kane and his colleagues at Long Island Jewish Hillside Medical Center in Glen Oaks, N.Y., recently surveyed four psychiatric hospitals in the New York City area and found that about 8 percent of the patients had moderate or severe TD after almost three years of drug treatment. Half of the patients displayed mild types of movement disorders. Approximately 20 percent of all mental hospital patients probably have TD, says Kane, but the prevalence rises to at least 40 percent among elderly, long-term patients. In Kane's sample, the number of patients with TD rose by 4 percent annually.

Still, says Gualtieri, neuroleptics are often effective at moderating psychotic symptoms. The drugs can calm an excited patient, bring a catatonic out of a withdrawn state and dampen the intensity of hallucinations and delusions. He cautions, however, that medication does not relieve some of the most deep-seated problems of schizophrenic patients, such as the inability to interact with others, deal with day-to-day stress, live independently and hold down a job.

Neuroleptics are also prescribed for about 1 million nonschizophrenic adults annually, says George Gardos of McLean Hospital in Belmont, Mass. They include individuals with severe depression, organic brain disorders, mental retardation and a variety of medical or surgical conditions. Aggressive and assaultive behavior is often quelled with these drugs.

Neuroleptics have been "grossly overused" in group homes for the retarded and nursing homes, notes Gardos. Retarded persons and patients with organic brain disorders may have a slightly greater chance of developing TD than schizophrenics, he says. Gualtieri estimates that 34 percent of mentally retarded individuals withdrawn from neuroleptics have TD.

Yet researchers immersed in the study of neuroleptics have not conclusively demonstrated how the medication causes severe twitching and writhing. A widespread assumption is that the biological root of TD lies in a neuroleptic-caused "supersensitivity" of dopamine receptors in the brain that elevates levels of this neurotransmitter. Several investigators speculate that other chemical messengers in the brain, such as norepinephrine and gamma aminobutyric acid (GABA), play a more crucial role in movement disorders.

Some critics charge that the drugs "lobotomize" patients and result in brain damage (SN: 10/1/83, p. 214), but there is little research to support this or any other contention, says Gualtieri.

The causes of physician overreliance on neuroleptics are clearer, he asserts. "American medicine is oriented toward acute, high-tech care for people with insurance coverage," maintains Gualtieri, while schizophrenics and the mentally retarded often need structured group homes where social and vocational skills are taught. Such facilities are few and far between, and the services they provide are not usually covered by medical insurance.

High-tech medicine is not about to fall from dominance, but continuing TD malpractice suits may well put a dent in neuroleptic prescriptions, especially since improved medications are not expected soon. An antipsychotic drug known as clozapine has been touted by some European researchers as less likely to cause TD than other neuroleptics. Clozapine may be "cleaner" than its pharmaceutical brethren, says Jonathan Cole of McLean Hospital, but "I don't know of any anticipated new neuroleptics within the next five or 10 years that are better than what is now available." □