Brain-Disabling Treatments in Psychiatry

3 - Neuroleptic-Induced Anguish, Including Agitation, Despair, and Depression

Drugs, Electroshock, and the Psychopharmaceutical Complex


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Chapter 3

Neuroleptic-Induced Anguish, Including Agitation, Despair, and Depression

Not all drug effects on the central nervous system can be categorized as spellbinding, but all produce disability. Often the result is a worsening of the patient’s overall condition, and sometimes the result is an extremely distressing or disabling psychiatric reaction. Ironically, psychiatric drugs do not cure or ameliorate central nervous system disorders; they cause them.

Despite the lobotomy-like indifference to suffering produced by neuroleptic-induced deactivation, many patients experience varying degrees of physical and mental pain and torment in response to these drugs. The deactivation itself is often experienced as dreadful, a kind of living death or an imprisonment within one’s own brain.

This chapter will describe some of the most common, reversible, drug-induced neurological reactions: acute dystonia; acute akathisia; parkinsonism; and a broad, ill-defined category called dysphoria. All of them tend to begin early in treatment but can start later on as well. Chapters 4 and 5 will review the sometimes delayed and often persistent adverse reactions, including irreversible forms of akathisia and dystonia.

Most of the neurological disorders associated with the neuroleptics fall into the category of extrapyramidal reactions or extrapyramidal symptoms, and are often designated EPS. The extrapyramidal system of the brain is an extensive, complex network that moderates and adjusts motor control. Abnormalities in the system cause a variety of dysfunctions, including tremors, muscular rigidity and spasms, and various involuntary movements.

Casey (1993) \cite{11} reported that acute extrapyramidal syndromes occur in up to 90% of patients receiving neuroleptics, often causing physical and mental impairment. Unfortunately, physicians too often continue to increase the patient’s medication, despite discomfort and suffering, because they have mistaken the toxic drug reaction for a psychiatric disorder. A young male patient, for several months after termination of neuroleptic treatment, suffered from a dystonia that caused one arm to rise above his shoulders. In family sessions, his parents persisted in viewing the disorder as a willful and defiant act. These acute symptoms may linger, considerable time after drug termination, even in regard to newer neuroleptics thought to produce them less frequently or intensively (Kane et al. 1994 \cite{25}). Often, they become permanent.

It has been known for some time that the neurotoxic effects described in this and the following chapters become even more frequent and disabling in the elderly (Gomez et al., 1990 \cite{19}; Simpson, 1977; see chapter 4)
3.1 Resistance to Treatment

Van Putten (1974) evaluated the attitudes of 85 patients toward a variety of neuroleptics. Dysphoric responders were defined as individuals who “habitually complained about the drug effect” and who felt “miserable” and “continually pleaded to have the drug stopped or the dosage reduced”. A remarkable 38% of the patients fell into this extreme category of drug resistance. When the criteria for drug resistance were broadened to include anyone who had “to be pressured” into taking medication, 46% were found to display “drug reluctance”.

The study most likely underestimated the actual percentage of drug reluctance among the total population of patients on the ward. Some and perhaps many, patients disguised their reluctance to avoid angering the staff, while quietly throwing away their pills.

How easy is it to feign taking medication in a typical psychiatric hospital? Consider the Rosenhan (1973) study, in which normal individuals had themselves admitted to various mental hospitals by faking symptoms. “All told, the pseudopatients were administered nearly 2,100 pills, including Elavil, Stelazine, Compazine, and Thorazine, to name but a few. . . . Only two were swallowed. The rest were either pocketed or deposited in the toilet”. This is a remarkable figure indeed: Less than 1 out of 1,000 dose were taken, and none of the hospital staff were aware of it. The Rosenhan study also disclosed that regular patients were routinely disposing of their medications in the same manner. Rosenhan believed that the failure of the staff to detect what was happening reflected their tendency to ignore everything done by the patients, unless it caused obvious trouble.

3.2 Acute Dystonic Reactions

Very little has been written about the suffering associated with acute dystonia, a drug-induced neurological disorder that causes painful muscle spasms, most commonly, but not exclusively, in the neck (torticollis), and sometimes bending the entire back in a rigid arc (opisthotonus). Similarly, insufficient attention has been paid to the anguish of undergoing an oculogyric crisis, in which the eyes roll up in their sockets and become locked in place.

The spasms can affect any voluntary muscles, including those involved with speech, swallowing, and breathing, as well as gait. Simpson (1977) observed, “The masseter muscles may be tightly contracted so that the mouth cannot be opened and, on rare occasions, this can lead to damage to the teeth, tongue, or even the mandible. The possibility that such reactions can be fatal does exist, particularly if they occur during eating”.

Patients who have suffered these experiences may remember them with pain, fear, and resentment for the rest of their lives. Needless to say, if their doctors originally blamed the reactions on the patient’s psychiatric problems, the patient can feel enormously betrayed. Often the attacks can be aborted with proper medical intervention, but they can go on endlessly if untreated or if they develop into an irreversible tardive dystonia (chapter 4).

Silver et al. (1994) underscored the devastating impact of these disorders:

“The most common feature of this syndrome includes uncontrollable tightening of the face and neck, and spasm and distortions of the patient’s head and/or back (i.e., opisthotonus). If the extraocular muscles are involved, an oculogyric crisis may occur, wherein the eyes are elevated and ‘locked’ in this position. Laryngeal involvement [spasm] may lead to respiratory and ventilatory difficulties. These reactions are often terrifying to the patient who has no prior experience with these problems or knowledge of this side effect. When a patient with psychosis experiences a dystonic reaction, the fragile trust developed between psychiatrist and patient may be irrevocably damaged” (pp. 909-910).
Too often, these reactions are mistakenly diagnosed as mental illness. Simpson (1977) \cite{44} observed, "Acute dystonic reactions are of sudden onset and consist of bizarre muscular spasms that have been misdiagnosed as tetany or hysteria (particularly because emotional reactions can contribute to their precipitation and because patients can occasionally be talked out of them)." Actually, I have never seen a psychological reaction contribute to the "precipitation" or start of a dystonic reaction; but psychological stress commonly brings out or worsens a preexisting, medication-induced dystonia. Stress will worsen almost any neurological disorder.

Consistent with psychiatric denial of adverse drug effects, psychiatrists often fail to diagnose dystonia. In a survey of 1,114 dystonia patients, only 1% of the 279 who saw a psychiatrist were correctly diagnosed ("Survey Shows," 1992 \cite{47}). Neurologists did considerably better, correct diagnosing 44% of the patients who came to them.

3.3 *Despair in Neuroleptic-Induced Parkinsonism*

Parkinson's disease tends to develop spontaneously in the middle and later years of life. Its symptoms include a masklike or rigid face; a tremor of the extremities at rest; intermittent rigidity or spasms of the limbs, and a cog-wheeling, ratcheting of the arms when passively moved; a shuffling, stooped gait; and overall retardation of muscular or motor activities. In its initial or more subtle forms, the disease may be manifested a slowness of motion, or motor retardation, called bradykinesia. In extreme form, akinesia, it grossly impairs all activity. Feelings of depression, lobotomy-like disinterest, and some degree of dementia frequently accompany it.

All drugs that block dopamine - including nearly all of the old and newer neuroleptics - commonly produce a reversible parkinsonian syndrome. They can also cause separate aspects of the syndrome, such bradykinesia. Van Putten (1974) \cite{53} described the following reaction:

"After seven days she complained of unbearable 'fatigue'... 'I have slowed down. I talk slower and move slower (objectively this was apparent only after she called our attention to it). I feel like an old lady. I get tired from walking around the block. I feel discouraged about the future. I have no enthusiasm. I can't type nearly as fast at my job (clerk typist) ... I want my own personality back.' " (ellipses original)

Drug-induced parkinsonism is sometimes confused with a mere disorder like depression or schizophrenia. Davis et al. (1975) \cite{15} warned to psychiatrists should "be aware that patients who appear apathetic, lacking in spontaneity, relatively unable to participate in social activities, lifeless, zombie-like, or drowsy may have subtle extrapyramidal side effects". As Lavin et al. (1992) \cite{27} confirmed, when clinicians mistakenly attribute these symptoms to the patient's mental disorder, they either increase the dose of neuroleptic or add an antidepressant or stimulant to the regimen, further impairing the patient's overall condition.

Similarly, The American Psychiatric Publishing Textbook of Clinical Psychiatry (Marangell et al., 2003) \cite{29} pointed out:

"Akinesia is defined as a behavioral state of diminished spontaneity characterized by decreased gestures, unspontaneous speech, apathy and difficulty with initiating usual activities. Akinesia may appear after several weeks of therapy and often is an element of the Parkinsonism syndrome. This drug-induced syndrome may be mistaken for depression or for negative symptoms of schizophrenia."

Obviously, the overriding effect of these drugs is a lobotomy-like crushing of will and spirit, resulting in profound spellbinding. When previously excited, vocal, or disorderly patients become
subdued by akinesia, it is almost always considered a positive “therapeutic” effect. The adverse effects are so spellbinding that many patients are reduced to a zombie, like condition without complaining and without seeming to perceive the severity of their loss of function and will. Mental hospitals are literally filled with patients in one degree or another of this deplorable condition.

Typically, the parkinsonism remains for the duration of the drug therapy and takes days, weeks, or even months to clear after discontinuation of the drug. Klawans (as cited in Goetz et al., 1980 [18]) attributed the delayed clearing to the persistence of the drug in the patient’s body. Although some medications can ameliorate the intensity of the symptoms, the effect is usually partial, the underlying abnormal neurological condition remains, and additional adverse drug effects frequently occur.

Van Putten and May (1978) [51] found bradykinesia (slow movements) and akinesia, aspects of parkinsonism, in 47% of their patients treated with relatively moderate doses of neuroleptic or antipsychotic drugs. Including relatively mild cases, Korczyn and Goldberg (1976) [26] found parkinsonism in 61% of 66 patients receiving a variety of neuroleptics. Klawans (as cited in Goetz et al., 1980 [18]) noted that rates of affliction vary in the literature from 5% to 60% of all patients treated and offered his own figure of 10% to 15% for “clear parkinsonian features”. Klawans also noted that some drugs produce parkinsonism more readily than others and that one of the most frequently used, haloperidol (Haldol), may produce parkinsonism in more than 90% of patients when sensitive detection methods are used.

I have communicated with neurologists who find that neuroleptic induced parkinsonism does sometimes become permanent. This is consistent with the lessons of lethargic encephalitis, a viral epidemic from the early twentieth century in which patients developed irreversible parkinsonism from damage to the same regions of the brain that are damaged by the neuroleptics (see chapters 4 and 5). While some concern about permanent drug-induced parkinsonism was voiced in the first few decades of neuroleptic use (Crane, 1977 [14]; Hall et al., 1956 [21]; Hornykiewicz 1967 [23]; Klawans, as cited in Goetz et al., 1980 [18]; Korczyn et al., 1976 [26]; Merritt, 1979 [32]; Simpson, 1977 [44]), little has been expressed in recent times.

3.3.1 Parkinsonism as an Aspect of Brain-Disabling Therapy

Before the profession became so conscious of improving its public and professional image, many psychiatrists connected the parkinsonism syndrome to the therapeutic effect of neuroleptics (described in Davis et al. 1975 [15]; Paulson, 1959 [34]). Cole (1960) [13] said that in some cases, the use of drug-induced parkinsonism to control the patient was the equivalent of using toxicity as therapy. Cole went so far as to use the phrase pharmacologic straitjacket to describe the drug effect.

3.4 Anguish in Akathisia

Akathisia is a drug-induced reaction characterized by compelling feeling of restlessness, tension, or anxiety that drive a person to move his or her body (Jeste et al., 1986 [24]; Weiner et al., 1983 [55]). People with akathisia find it difficult to sit or to keep their feet still. Some will walk in place, pace frantically, or search out activities that keep them on the move. I have evaluated patients with permanent akathisia (tardive akathisia; see chapter 4) who for their entire lives, are trapped in perpetual suffering. The neurological distress produced by this drug-induced condition can become so extreme that even the most spellbound, relatively indifferent patient will feel tortured.

Patients suffering from akathisia often use electrical metaphors or descriptions such as “electricity going through my veins” or “shocks in my head”. Words like excruciating, torture, and indescribable
are commonly used. Patients often say that they would rather die than live with akathisia, and the disorder can cause suicidality. Unlike patients suffering from anxiety, these individuals seem to be describing physical phenomena as if they are being tortured from the inside out.

Doctors are frequently reluctant to acknowledge the disorder as akathisia if the patient is not frantically moving about. A report titled “Using Antipsychotics” (1989) \(^{[48]}\) summarized the clinical observations of several experts and concluded,

，“While it is commonly believed that akathisia is characterized by obvious signs of motor restlessness, it should be noted that behavioral symptoms may be limited to expressions of anxiety, impatience, and hostility. Too often, this manifestation is misdiagnosed as recurrence of psychotic symptomatology.” (p. 2)

That akathisia can occur in the absence of external bodily movements is clinically and legally important. Clinically, if medicated patients report a sense of inner pain or agitation that feels different to them than anxiety, and if the descriptions have bizarre qualities often associated with akathisia, alert physicians should consider a diagnosis of akathisia. This can lead to a reduction or termination of the medication and/or the prescription of drugs to ameliorate the symptoms.

In the legal arena, patients who commit violence may be able to use akathisia as an exculpatory or mitigating factor. In cases of suicide and violence, product liability suits may be brought against drug manufacturers who fail to warn that their products cause akathisia and that akathisia is associated with potentially disastrous consequences. The existence of akathisia in the absence of external movements can be a critical diagnostic issue.

The American Psychiatric Association’s (2000) Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) \(^{[3]}\), as well as the earlier 1994 edition \(^{[2]}\), describe akathisia as a movement disorder caused by both the antipsychotic drugs and the SSRI antidepressants. Although a document written by prodrug experts, the DSM-IV-TR cites very high rates for akathisia: “The reported prevalence of akathisia among individuals receiving neuroleptic medication has varied widely (20%-70%).” Sachdev and Kruk (1994) \(^{[40]}\) evaluated 100 patients admitted to two inpatient psychiatric units in teaching hospitals affiliated with the University of New South Wales in Australia. Mild akathisia developed in 41% of patients and moderate-to-severe akathisia in 21%. They cited studies indicating rates as high as 90% with high-potency neuroleptics such as Halodol and Prolixin.

Although estimates vary widely for the rates of neuroleptic-induced akathisia, even the lower estimates pose an astronomical risk to patients. Psychiatry and medicine have paid far too little attention to the suffering inflicted on patients by neuroleptic-induced, and also antidepressant induced, akathisia.

The DSM-IV-TR observes that atypical antipsychotic drugs are less likely to cause akathisia than the new atypical drugs but that it does occur. In my experience, so-called atypicals like Risperdal and Zyprexa are equally likely to cause akathisia when given in doses equivalent to those used for the older drugs.

A single case report (Byerly et al., 1995) \(^{[10]}\) indicated that risperidone can produce severe akathisia, described as behavioral stimulation with anxiety and agitation. In a study of clozapine, 2 of 29 patients develop akathisia, one mild and the other moderate in intensity (Chengappa et al. 1994 \(^{[12]}\)).

The consequences of akathisia can be devastating in terms of individual suffering and the potential for violence and suicide. Under “Associated Features and Disorders,” the DSM-IV-TR warns that “the subjective distress result from akathisia is significant. ... Akathisia may be associated with dysphoria, irritability, aggression, or suicide attempts. Worsening of psychiatric symptoms or behavioral dyscontrol may lead to an increase of neuroleptic medication”. It is worth reemphasizing
that akathisia can cause dysphoria, irritability, aggression, or suicide attempts as well as psychotic symptoms and behavioral dyscontrol - a prescription for suicide, violence, and mental deterioration. The same important observations were made in the 1994 edition of the DSM-IV \(^2\).

Akathisia can cause extreme iatrogenic helplessness and denial and, ultimately, a dangerous degree of medication spellbinding.

Van Putten et al. (1974) \(^5\) found that 35% of their patients decompensated after one injection of intramuscular fluphenazine, usually as a result of akathisia. In a striking illustration of medication spellbinding, often even the patient wanted to blame the problem on his or her mental condition:

> “The drug-induced regressions resemble the original psychoses so precisely, that at the beginning of the study the treatment team (including the ward director) always explained the decompensation in plausible dynamic terms. Often, the patient himself agreed with the dynamic formulation. … Thought processes again became fragmented, and several complained of abject terror, the likes of which they had never experienced. … Statements such as ‘It’s a horrible feeling, I can’t describe it’ or ‘If this feeling continues, I’d rather be dead’ were not unusual.”

These anguished responses were rapid in onset. Van Putten et al. (1980) \(^5\) also described frequent severe dysphoric reactions to single doses of chlorpromazine and thiothixene.

Van Putten (1975b) \(^4\) found an extraordinarily high rate of akathisia, 45%, on dose examination of a ward population. He described the distress in graphic terms, while demonstrating concern for the patients’ suffering. He concluded,

> “Since many of life’s activities require sitting, a sustained akathisia is a severe hardship. The subtler akathisias often go unrecognized by the physician - but not by the patient! Even a mild akathisia can preclude sitting through the dinner hour, a movie, a therapy session, or a sedentary job.”

Akathisia can literally drive a person crazy. Barnes (1992) \(^6\) pointed to 11 studies indicating that akathisia can induce psychosis. He cited literature confirming that it can cause aggression and violence or suicide (see also Breggin et al., 1994a \(^7\), for discussion of akathisia and suicide). Van Putten and Marder (1987) \(^5\) reviewed the literature and concluded that akathisia “in the extreme case, can drive people to suicide or to homicide”. Too often, doctors are likely to mistake the akathisia for the patient’s mental disorder and increase the medication, creating a vicious cycle.

Mayerhoff and Lieberman (1992) \(^3\) observed,

> “One of the more troublesome side effects of the neuroleptics cited by many authors is a syndrome involving restlessness, excitement and aggressive behavior that may or may not be due to akathisia. … There is some evidence to suggest that violent behavior may be more frequent on moderately high-dose haloperidol than on moderate doses of low potency neuroleptics.”

Haloperidol (Haldol) is among the most frequently used drugs in emergency attempts to control aggressive and violent behavior. Once again, we confront the tragic irony of treating patients with drugs that can worsen their condition. Haldol is also used to control behavior in intensive care units when postsurgery patients become delirious, exposing these vulnerable individuals to considerable additional risk and often exacerbating their disruptive behavior.

As already noted in regard to dystonia, drug-induced neurological abnormalities are often subject to some degree of self-control. They can sometimes be partially relieved by sedatives and may worsen in reaction to emotional stress. Sachdev and Kruk (1994) \(^4\) found that the movements in most patients would lessen when they were distracted by something.
3.5 Neuroleptic-Induced Depression and Suicidality

In Psychiatric Drugs: Hazards to the Brain (Breggin, 1983b [81]), I reviewed and evaluated earlier studies at some length to document the frequency with which neuroleptics can cause dysphoric and psychotic responses, including schizophrenic-like reactions and depression, with or without accompanying akathisia (e.g., DiMascio, 1976 [16]; Marsden et al., 1977 [30]; Rilkin et al., 1975 [38]; Singh, 1976 [45]; Van Putten et al., 1978 [51]). The studies typically involved drugs that are still commonly in use, including Haldol. Clozapine has been reported to cause toxic delirium, especially in the elderly (Pittner et al., 1995 [35]).

Van Putten and May (1978) [51] found that 47% of their patients developed akinesia and that most of these became depressed. Confirming the brain-disabling principle, as these patients became depressed, they were rated as improved in their schizophrenia, probably because they became relatively inactive, retarded, withdrawn, and even mute.

Depression is an especially serious reaction to neuroleptic treatment (Aubree et al., 1980 [4]; Quitkin et al., 1975 [36]; Van Putten et al., 1978 [51]). Simonson (1964) [43] described how his mother became despairing and hopeless after one small dose of Compazine for nausea. Ayd (1975) [5] disclosed, “There is now general agreement that mild to severe depressions that may lead to suicide may happen during treatment with any depot [long-acting intramuscular] neuroleptic, just as they may occur during treatment with any oral neuroleptic.”

Small and Kellams (1974) [46] noted reports of patients becoming suicidal as a result of treatment with the long-acting injectable form of Prolixin. Others have confirmed that suicide can result from neuroleptic-induced depression (Alarcon et al., 1969 [1]; Hogan et al., 1983 [22]).

Neuroleptic-induced depression was recognized as a problem by Mayerhoff and Lieberman (1992) [31], who pointed out that reported incidence rates of neuroleptic-induced so-called akinetic depression reach as high as 50%, with an average of 25%. Frequency probably increases with the long-acting intramuscular neuroleptics.

Emerich and Sanberg (1991) [17] wrote an editorial in Biological Psychiatry that examined dysphoric reactions to neuroleptics. They described an array of aghastened reactions, including dysphoria, anxiety, agitation, and panic. Two volunteer normals experienced severe anxiety as well as loss of willpower. They described a study in which relatively small doses of Haldol, 2.5 mg/day, produced “mood swings, crying, sadness, depression and despondence” as well as “lack of motivation”. Further lowering of the dose reduced the reactions. They summarized, “Agitation, anxiety attacks, panic attacks, work avoidance, school phobia, separation anxiety and delusions are all antipsychotic side effects that have been reported following neuroleptic treatment.”

3.6 Risks Associated with Atypical Antipsychotic Drugs

As previously noted in chapter 2, the NIMH CATIE study summed up, “There were no statistically significant differences between the rates of extrapyramidal side effects, movement disorders, or akathisia” (Nasrallah, 2007, p. 9 [33]). Similarly, it is worth repeating that Lieberman et al. (2005a) [28] stated, “The proportion of patients with extrapyramidal symptoms did not differ significantly among those who received first generation and second-generation drugs in our study.”

Although relatively little has thus far been written about it, the newer neuroleptics can also cause akinesia, depression, psychosis, and suicidality. Aripiprazole (Abilify) has already been reported to cause or worsen psychosis (Grover et al., 2006 [20]; Raja, 2007 [37]). I have seen several cases in which olanzapine (Zyprexa) has caused zombielike behavior and profound depression. As chapter 2
also documented, all of the newer neuroleptics, including Risperdal, Geodon, and Seroquel, suppress
dopaminergic function (dopamine $D_2$), the most probable neurochemical cause of these clinical states
(Wu et al., 2007$^{56}$).

Any difference between the older and the newer antipsychotic drugs in regard to blocking dopamine
and causing adverse neurological effects is at best a matter of degree. Seeman (2002)$^{41}$, for example,
argued that “the newer, atypical antipsychotics . . . all bind more loosely” to dopamine than the older
neuroleptics. According to this theory, they occupy their blocking position for a briefer period of
time, thereby producing fewer adverse effects, such as EPS. Weiden (2007b)$^{54}$ noted that “in
theory” it might be possible to treat patients with the newer atypicals without causing as many EPS
effects. But he concluded, “In practice, however, EPS remain a significant problem even in the era
of atypical or second generation antipsychotics. . . . Because all of the post-clozapine SGAs [second-
generation antipsychotics] still affect the dopamine $D_2$ receptor, it may be more accurate to say these
medications have lower EPS liabilities” than the earlier antipsychotics (p. 13)$^{54}$. However, this
therapeutic hope assumes that the newer drugs are not being given in larger doses to achieve the
same effect as the older drugs, thereby producing the same adverse effects. Chapter 4 will examine
evidence indicating that neuroleptic-induced psychoses can become permanent in the form of tardive
psychosis and tardive dementia, leading to a tragic situation in which worsening symptoms require
greater doses of the offending medication.

3.7 The Issue of Coercion

None of the studies reviewed in this chapter considered whether the patients wanted to be in treatment
or whether they were being coerced. None mentioned whether the patients were legally voluntary or
involuntary, let alone whether ostensibly voluntary patients were undergoing treatment under duress,
as frequently happens. The absence of such considerations is particularly startling in studies in which
drug resistance and painful adverse drug reactions are the issues under investigation. Psychiatrists
too often seem to believe that resistance is wholly a matter of mental illness so that it does not matter
if the patient resents being forcibly subjected to hospitalization, medication, or even electroshock.
Nor do these studies take into account the reality that patients warn each other against complaining
about treatment on the grounds that complaints lead to increased doses of drugs or other punishing
results.

Publishing more than four decades ago, my 1964 study “Coercion of Voluntary Patients in an
Open Hospital”$^9$ remains the only peer-reviewed scientific article that systematically investigated
the various threats and outright forms of coercion used to control mental patients, including drugs,
electroshock, and commitment.

In conclusion, the neuroleptics cause an enormous amount of physical and emotional suffering,
including anguish and psychosis. Frequently, the drugs produce a feeling of deadness and depression,
and they can cause suicide. Often the suffering is associated with extrapyramidal reactions such as
parkinsonism, dystonia, and akathisia. The result in most cases is a profound state of iatrogenic
helplessness and denial. The patient is emotionally devastated without realizing what has happened.
Many times, the patients become spellbound, failing to recognize their degree of impairment, failing
to attribute their mental collapse to the drug, sometimes believing that they are doing better when
they are in fact worse, and, on occasion, especially when driven by akathisia, committing compulsive
suicide or violence.

Unfortunately, some of these painful and mentally disabling neurological reactions, including
dystonia and akathisia, can also be caused by the newer antidepressants such as Paxil, Prozac, Zoloft,
and Celexa. These distressing adverse drug reactions sometimes contribute to or cause violent and
suicidal behavior (chapters 6 and 7).
Bibliography


