Brain-Disabling Treatments in Psychiatry

Second Edition
Peter R. Breggin, MD, has been called “the conscience of psychiatry” for his efforts to reform the mental health field, including his promotion of caring psychotherapeutic approaches and his opposition to the escalating overuse of psychiatric medications, the oppressive diagnosing and drugging of children, electroshock, lobotomy, involuntary treatment, and false biological theories.

Dr. Breggin has been in the private practice of psychiatry since 1968, first in the Washington, D.C., area, and now in Ithaca, New York. In his therapy practice, he treats individuals, couples, and children with their families without resort to psychiatric drugs. As a clinical psychopharmacologist, he provides consultations and is active as a medical expert in criminal, malpractice, and product liability lawsuits, often involving the harmful effects of psychiatric drugs. He has been an expert in landmark cases involving the rights of patients.


At various stages of his career, he has been decades ahead of his time in warning about the dangers of lobotomy, electroshock, and, more recently, antidepressant-induced suicide and violence as well as many other recently acknowledged risks associated with psychiatric drugs. His views have been covered in major media throughout the world including The New York Times and The Wall Street Journal to Time and Newsweek, and from Larry King Live and Oprah to 60 Minutes and 20/20.

In 1972, Dr. Breggin founded the International Center for the Study of Psychiatry and Psychology (ICSPP; http://www.icspp.org). Originally organized to support his successful campaign to stop the resurgence of lobotomy, ICSPP has become a source of support and inspiration for reform-minded professionals and laypersons who wish to raise ethical and scientific standards in the field of mental health. In 1999, he and his wife, Ginger, founded ICSPP’s peer-reviewed scientific journal Ethical Human Psychology and Psychiatry. In 2002, they selected younger professionals to take over the center and the journal, although Dr. Breggin continues to participate in ICSPP activities.

Dr. Breggin’s background includes Harvard College, Case Western Reserve Medical School, a teaching fellowship at Harvard Medical School, 3 years of residency training in psychiatry, a 2-year staff assignment at the National Institute of Mental Health, and several teaching appointments, including in the Johns Hopkins University Department of Counseling and the George Mason University Institute for Conflict Analysis and Resolution.

Brain-Disabling Treatments in Psychiatry

Drugs, Electroshock, and the Psychopharmaceutical Complex

Second Edition

Peter R. Breggin, MD

SPRINGER PUBLISHING COMPANY

New York
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Springer Publishing Company, LLC
11 West 42nd Street
New York, NY 10036
www.springerpub.com

Acquisitions Editor: Sheri W. Sussman
Project Manager: Julia Rosen
Cover design: Mimi Flow
Composition: Apex Publishing, LLC

08 09 10 11/ 5 4 3 2 1

Library of Congress Cataloging-in-Publication Data
Breggin, Peter Roger, 1936–
  Brain-disabling treatments in psychiatry : drugs, electroshock, and the psychopharmaceutical complex / Peter R. Breggin. — 2nd ed.
p. ; cm.
  Includes bibliographical references and index.
  1. Psychotropic drugs—Side effects. 2. Brain—Effect of drugs on.
  3. Electroconvulsive therapy—Complications. 4. Iatrogenic diseases.
  5. United States. Food and Drug Administration. I. Title.
RC483.B726 2008
616.89'122—dc22

Printed in the United States of America by Edwards Brothers, Inc.
WARNING

_Psychiatric Drugs Are Dangerous to Take and Dangerous to Stop_

The psychiatric drugs discussed in this book are far more dangerous to take than many doctors and patients realize, but they can also become hazardous during the withdrawal process. In short, it is dangerous to start psychiatric drugs and dangerous to stop them.

Many are addictive, and most can produce withdrawal symptoms that are emotionally and physically distressing and sometimes life threatening. Tapering off psychiatric drugs should usually be done gradually with the aid of experienced clinical supervision.

A book cannot substitute for individualized medical or psychological care, and this book is not intended as a treatment guide. It provides a critical analysis of biological treatments in psychiatry written from a scientific, ethical, psychological, and social viewpoint.

Peter R. Breggin, MD
Professional Books by Peter R. Breggin, MD

College Students in a Mental Hospital: Contributions to the Social Rehabilitation of the Mentally Ill (Jointly authored) (1962)
Electroshock: Its Brain-Disabling Effects (1979)
Psychiatric Drugs: Hazards to the Brain (1983)
Toxic Psychiatry: Why Therapy, Empathy and Love Must Replace the Drugs, Electroshock and Biochemical Theories of the “New Psychiatry” (1991)
Beyond Conflict: From Self-Help and Psychotherapy to Peacemaking (1992)
Talking Back to Prozac (coauthor Ginger Breggin) (1994)
Psychosocial Approaches to Deeply Disturbed Persons (coeditor E. Mark Stern) (1996)
Brain-Disabling Treatments in Psychiatry: Drugs, Electroshock, and the Role of the FDA (1997)
The Heart of Being Helpful: Empathy and the Creation of a Healing Presence (1997)
The War Against Children of Color: Psychiatry Targets Inner City Children, Updated (coauthor Ginger Breggin) (1998)
Reclaiming Our Children: A Healing Solution to a Nation in Crisis (2000)
Talking Back to Ritalin, Revised Edition (2001)
The Antidepressant Fact Book (2001)
The Ritalin Fact Book (2002)
Medication Madness: True Stories of Mayhem, Murder and Suicide Caused by Psychiatric Drugs (2008)
For Ginger Breggin
My wife, best friend, partner in life, most trusted advisor,
last human resort in all crises, and playmate
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Preface

A WORD ABOUT WORDS

Throughout this book, I use diagnostic terms such as attention-deficit/hyperactivity disorder (ADHD), bipolar disorder, major depressive disorder, and schizophrenia. If I were to express my scientific skepticism toward these terms each time I used them, the book would be marred by constant interruptions. Instead, I want to establish from the beginning that I am using these diagnostic terms only for the purpose of consistency with current usage in the various sources on which I am drawing, such as clinical studies, research reports, and Food and Drug Administration (FDA)-approved drug labels.

As the book will indicate, these diagnostic categories do not reflect valid diseases or illnesses comparable to Alzheimer’s disease, stroke, or diabetes. Despite claims to the contrary, these psychiatric disorders have no proven genetic, chemical, or biological basis. They cannot be diagnosed with physical symptoms or laboratory studies.

Of course, no one denies that people can become highly irrational, lose touch with ordinary reality, or become suicidal or violent; but an extreme emotional response, however destructive, in itself does not demand an explanation rooted in biological dysfunction. Without any underlying medical disorder, human beings have the capacity for extreme psychological reactions, especially under stress.

Of course, genuine diseases or disorders of the brain, such as endocrine disorders or dementia, can change and disrupt human behavior. In this book and in Medication Madness (in press), I describe how psychiatric drugs cause brain disorders that lead to mayhem, murder, and suicide. Indeed, the FDA at long last has begun to confirm observations that I made long ago concerning antidepressant-induced mental and behavioral abnormalities. However, except for the brain dysfunction and biochemical imbalances caused by psychiatric drugs, there are no known abnormalities in the brains of people who routinely seek help from psychiatrists and
who become diagnosed with disorders like ADHD, schizophrenia, and major depressive disorder.

To label children with ADHD or to label adults with schizophrenia or major depressive disorder is to stigmatize them with damaging, discouraging labels and to encourage or coerce them to submit to biopsychiatric interventions such as drugs and electroshock. In my own psychiatric practice, I do not think in conventional diagnostic terms or tell patients that they have so-called mental disorders. Instead, I try to understand the life story of each individual—his or her personal biography—in all its subtle complexity. Often, I involve loved ones and family to help them understand each other. On this basis of genuine understanding, instead of cookie-cutter diagnoses, I am far more able to help individuals lead more satisfying, successful lives.
Acknowledgments

Springer Publishing Company published my first medical book, Electroshock: Its Brain-Disabling Effects, a long time ago, in 1979. Now, almost 30 years later, this new edition of Brain-Disabling Treatments in Psychiatry comes at a time when the public’s perception of psychiatric treatments has come closer to many of the seemingly controversial positions taken in my earlier Springer books. Even within the health care professions, there is growing recognition that the risks associated with psychiatric drugs and shock treatments are greater than originally anticipated and that their effectiveness is more limited than hoped.

None of the basic assertions in the original edition of this book or in its precursors, Psychiatric Drugs: Hazards to the Brain (1983) and Electroshock (1979), have been proven wrong. Instead, a mountain of new evidence supports the main themes that I have been developing over the last decades. In a number of areas, the Food and Drug Administration has confirmed assertions in the first edition that once seemed especially controversial, for example, that antidepressants are ineffective in children and increase the rate of suicide attempts and that they also increase suicidality in young adults. Many other conclusions made in my earlier books have been adopted by the mainstream, including recent confirmation that electroshock treatment causes permanent brain damage and dysfunction.

When Springer Publishing Company decided to bring out my first two medical books, Electroshock (1979) and Psychiatric Drugs (1983), it required courage. The president of the company, Dr. Ursula Springer, and the senior editor at the time, Carole Saltz, had to be concerned about publishing a viewpoint so critical of seemingly established concepts of treatment. The opportunity they gave me has helped to encourage a lifetime of work in the field. From then until the present, nearly all of my publications have drawn energy and direction from these first two books.

I am grateful that Dr. Springer and her company found my first two medical books of sufficient merit and importance to take the risk of
publishing them. If they had not, my career might have taken a different and ultimately less useful direction.

Nearly three decades later, and after the retirement of Dr. Springer, Springer Publishing Company and Sheri W. Sussman, Senior Vice President, Editorial, have continued to support my work with a new paperback edition of *The Heart of Being Helpful* (1997b) and now with this new edition of *Brain-Disabling Treatments in Psychiatry*.

Springer Publishing Company also worked with me and my wife, Ginger, in developing the peer-reviewed scientific journal *Ethical Human Psychology and Psychiatry*, sponsored by the International Center for the Study of Psychiatry and Psychology (ICSPP; http://www.icspp.org). The journal is now enjoying a decade of publishing under the leadership of younger professionals and provides a unique opportunity for scientists and clinicians to publish independent research in the light beyond the shadow of the psychopharmaceutical complex.

I also want to thank the many members of ICSPP who have been so supportive of my work and each other's work in the reform movement.

As in many of my books, my research assistant Ian Goddard continued to provide much-needed help obtaining original articles, sometimes under considerable time pressure, often delivering them along with a big dose of his own original ideas and remarkable insights. Beyond that, he read the entire manuscript and made many useful editorial observations. This new edition is a better book because of Ian.

And now, approaching 25 years together, my wife, Ginger, continues to provide the strength and often the inspiration behind so much of what I do. It is because of Ginger's encouragement that the book now has two concluding chapters on treatment and my 20 guidelines for therapy with disturbed patients. She insisted that I needed to write them, and then she helped to edit them.
Introduction

Confirming the Science Behind
the First Edition

This book is aimed at professional audiences, but it is hoped that it is written with sufficient clarity and explanation to be read by nonprofessionals. The current edition has been very thoroughly revised, but the basic scientific thrust remains essentially the same. The past several years have confirmed the brain-disabling principle of psychiatric treatment, and many of the author's seemingly controversial conclusions have become more widely accepted.

A THOROUGH UPDATE OF THE SCIENCE

For this edition of the book, the concept of brain-disabling treatment has been updated and expanded with the additional concept of "medication spellbinding (intoxication anosognosia)." The neuroleptic chapters have been updated to include much more material on the newer, atypical drugs as well as new information on the neurotoxicity and cytotoxicity of all antipsychotic drugs. A massive amount of new information about antidepressant drugs and the stimulant drugs has resulted in an additional chapter on each drug.

The new edition concludes with two entirely new chapters on treatment—one on how to safely withdraw from psychiatric drugs, and the other about psychosocial and educational approaches to very disturbed people, including 20 guidelines for therapy. I am pleased to include how-to treatment information in the book for the first time.
GROWING CONFIRMATION OF THE
PREVIOUS EDITION

My observations that antidepressant drugs cause a spectrum of stimulant or activation effects—including agitation, hostility, aggression, and mania as well as crashing into depression and suicidality—have been elevated to the status of official dogma in the new Food and Drug Administration (FDA)-mandated changes in antidepressant labels. The concept that psychiatric drugs are neurotoxic is now a widely accepted principle in scientific research, especially concerning the antipsychotic drugs and mood stabilizers, and research has mounted up that demonstrates similar neurotoxic effects in all categories of psychiatric drugs. Many other medical experts have now joined in my criticism of the FDA’s failure to do its duty and my concern about the corrupting influence of the drug companies on the theory and practice of psychiatry. Put simply, I am no longer quite such a lonely voice crying in the wilderness.

CONFIRMING THE LONGER VIEW
STARTING IN 1983

The lineage of this new edition began in 1983 with *Psychiatric Drugs: Hazards to the Brain*, a book that broke new ground with the first extensive review of the subject of neuroleptic-induced dementia. It also took a firm stand on the view that neuroleptics frequently cause tardive dyskinesia (TD) in young people. TD in children has become an accepted reality, and so that section has been reduced in size. Tardive psychosis is gaining increasing, if slow, recognition. Tardive dementia remains controversial—although it should not be—and an increasing amount of evidence supports my earlier observations on the cognitive deficits caused by neuroleptics. In addition, the neurotoxicity of psychiatric drugs is being studied more openly in laboratories.

In the 1970s, when I first began offering detailed critiques of psychiatric drugs, the medical model, and the psychopharmaceutical complex, I was, in many cases, breaking new ground, and initially, there were few supporters. By the time of the first edition of *Brain-Disabling Treatments in Psychiatry* in 1997, I could already cite many books that voiced strong criticism of the biological model and physical treatments from a variety of perspectives (Armstrong, 1993; Breeding, 1996; Caplan, 1995; Cohen, 1990; Colbert, 1995; Fisher et al., 1989; Grobe, 1995; Jacobs, 1995; Kirk et al., 1992; Modrow, 1992; Mosher et al., 1989; Romme et al., 1993; Sharkey, 1994).

Especially in the last few years, an escalating number of authors, many from within the medical establishment, have been offering strong
criticism of that conglomerate of powerful interest groups, and especially
the dominating influence of the pharmaceutical industry (Abramson et al.,
2005; Kean, 2005, 2006; Medwar et al., 2004; Moncrieff, 2006a, 2006b; O’Meara, 2006; Rost, 2006).

THE SITUATION IN PSYCHIATRY WORSENS

Although many of my critiques and criticisms of biological psychiatry
and the psychopharmaceutical complex have a broader acceptance, in
many ways, the situation has deteriorated as the strength of the drug
companies has grown. In the process, my predictions about the growing
power of the psychopharmaceutical complex have come true.

The last two decades have seen escalating reliance on psychiatric
drugs, not only within psychiatry but also throughout medicine, mental
health, and even education. In private-practice psychiatry, it is common
to give patients a medication on the first visit and then instruct them that
they will need drugs for their lifetimes. Family practitioners, internists,
and other physicians liberally dispense antidepressants and benzodiaz-
epine tranquilizers. Nonmedical professionals, such as psychologists and
social workers, feel obliged to refer their patients for drug evaluations.
Managed care aggressively pushes drugs to the exclusion of psychother-
apy. Adult medications are increasingly prescribed to children. Hospitals
force psychiatric drugs on patients against their will.

There is a successful movement within psychiatry, implemented in
many states, that makes it easy to force clinic outpatients to take long-
acting injections of drugs. Under these outpatient commitment laws, if
the person refuses to come to the clinic, mental health workers can come
to the home to administer the injections by force. At the same time, there
is a movement to screen schoolchildren, and even preschoolers, for so-
called mental illness. This potentially disastrous movement is driven by
drug company money and aims at increasing the market for their products.

Laypersons have joined in the enthusiasm for drugs. Because of me-
dia support for medication as well as direct advertising and promotion
to the public, patients frequently arrive at the doctor’s office with the
name of a psychiatric drug already in mind. Teachers often recommend
children for drug evaluation or treatment.

This drug revolution views psychiatric medications as far more help-
ful than harmful, even as an unmitigated blessing. Much as insulin or
penicillin, they are vigorously promoted as specific treatments for specific
illnesses. Often, they are said to correct biochemical imbalances in the
brain. These beliefs have created an environment in which emphasis on
adverse drug effects is greeted without enthusiasm, and criticism of psychiatric medication in principle is uncommon heresy.

Drug companies heavily promote that unproven speculation that the problems they treat are biological in origin and result from biochemical imbalances. Advertising slogans are used to justify the prescription of medications. For example, Janssen (2005), the manufacturer of the antipsychotic drug Risperdal, offers a section “About Bipolar Disorder,” downloaded from its Web site in February 2006. It declares,

Mental illness is a medical illness, just like high blood pressure or heart disease.

The Janssen Web site goes on to say, “It is also thought that bipolar disorder may be caused by a genetic predisposition to the illness because it tends to run in families.” Notice again that no claim to scientific veracity is made. But the repetition of these unscientific biochemical and genetic speculations nonetheless conditions people to believe that psychiatric drugs are specific treatments for genetic, biochemical disorders, much like antihypertensive drugs for high blood pressure or insulin for diabetes.

This book takes a decidedly different viewpoint from that of biological psychiatry. It provides theory and evidence that psychiatric drugs achieve their primary or essential effect by causing brain dysfunction and that they tend to do far more harm than good. I will show that psychiatric drugs are not specific treatments for any particular so-called mental disorder. Instead of correcting biochemical imbalances, psychiatric drugs cause them, sometimes permanently.

Health care providers and the general public have also been bamboozled by the much-advertised speculation that brain scans can demonstrate the existence of mental disorders, and even diagnose them. In reality, no psychiatric disorder is demonstrable or diagnosable by brain scan (Jackson, 2006a) or by any other medical or biological means.

This second-edition book discusses how to stop taking psychiatric drugs and presents 20 guidelines for therapy. Considerably more information on how to help disturbed and disturbing people without resort to drugs or electroshock is readily available elsewhere (Breggin, 1991a, 1992a, 1997; Breggin et al., 1994a, 1996, 2002). Chapters in Reclaiming Our Children (2000b), Talking Back to Ritalin (2001c), The Antidepressant Fact Book (2001a), and The Ritalin Fact Book (2002b) also deal with therapeutic approaches. The best overall summary of my approach to helping people can be found in The Heart of Being Helpful (1997b). Finally, Medication Madness: True Stories of Mayhem, Murder and Suicide (in press) can be viewed as a companion to this book, providing real-life cases of the devastating impact of these drugs on individual lives.
CHAPTER 1

The Brain-Disabling, Spellbinding Effects of Psychiatric Drugs

Modern psychiatric drug treatment gains its credibility from a number of assumptions that professionals and laypersons alike too often accept as scientifically proven. These underlying assumptions qualify as myths: fictions that support a belief system and a set of practices. In contrast to these myths, this book identifies principles of psychopharmacology that are based on scientific and clinical evidence as well as on common sense.

Together, these form the brain-disabling principles or the brain-disabling concept of biopsychiatric treatment. While the book in its entirety provides the evidence for these principles, this chapter will summarize them, including the new principle of intoxication anosognosia, or medication spellbinding (Breggin, 2006d, in press).

In essence, the brain-disabling concept as a whole states that all psychiatric treatments—drugs, electroshock, and lobotomy—work by disrupting the function of the brain and mind, creating effects that are then interpreted (or misinterpreted) as improvements. Medication spellbinding is a brain-disabling effect that renders individuals unable to perceive the degree of their drug-induced impairment; causes individuals not to attribute any change in themselves to an adverse drug effect; often makes individuals believe that they are doing better than ever, when they are
doing worse; and in the extreme, drives them into compulsive activities that harm themselves and others.

**THE BASIC FOUR BRAIN-DISABLING PRINCIPLES**

I. All biopsychiatric treatments share a common mode of action: the disruption of normal brain function.

   Pharmacologists speak of a drug’s *therapeutic index*, the dosage ratio between the beneficial effect and the toxic effect. The first brain-disabling principle of psychiatric treatment reveals that the toxic dose is the therapeutic dose—that brain disability causes the seemingly therapeutic effect. This same principle applies to electroshock and psychosurgery.

   The brain-disabling principle states that as soon as toxicity is reached, the drug begins to have a psychoactive effect; that is, it begins to affect the brain and mind. Without toxicity, the drug would have no psychoactive effect.

   Psychoactive drugs, including psychiatric drugs, vary in their toxicity. However, all of the major categories of psychiatric drugs—antidepressants, stimulants, tranquilizers (antianxiety drugs), mood stabilizers, and antipsychotics—are neurotoxic. They poison neurons, and sometimes destroy them.

II. All biopsychiatric interventions cause generalized brain dysfunction.

   Although specific treatments do have recognizably different effects on the brain, they share the capacity to produce generalized dysfunction with some degree of impairment across the spectrum of emotional and intellectual function. Because the brain is so highly integrated, it is not possible to disable circumscribed mental functions without impairing a variety of other functions, typically causing generalized dysfunction of the brain and mind. For example, even the production of a slight emotional dullness, lethargy, or fatigue is likely to impair cognitive functions such as attention, concentration, alertness, self-concern or self-awareness, and social sensitivity. These changes can be subtle, and the spellbound individual may fail to perceive them, but the changes nonetheless adversely affect the person's quality of life.

   Shock treatment and psychosurgery always produce obvious generalized dysfunction. Some medications may not obviously produce these effects in their minimal dose range, but they may also lack any substantial so-called therapeutic effect in that range.
Biopsychiatric treatments exert their therapeutic effect by impairing higher human functions, including emotional responsiveness, social sensitivity, self-awareness or self-insight, autonomy, and self-determination. More drastic effects include apathy, euphoria and mania, and lobotomy-like indifference.

Higher mental, psychological, and spiritual functioning are impaired by biopsychiatric interventions as a result of generalized brain dysfunction as well as specific effects on the frontal lobe, limbic system, and other structures. Commonly, the result is a lobotomy-like indifference to self and to others—a syndrome that I have called deactivation. Recent research confirms that these effects occur with the SSRI antidepressants, such as Prozac, Zoloft, and Paxil; the stimulants, such as Ritalin, Concerta, and Adderall; and the newer antipsychotics, such as Risperdal and Zyprexa (see chapters 2, 4, and 7). Chronic use of any psychoactive or psychiatric drug, including the benzodiazepines and mood stabilizers, will produce a degree of deactivation.

Spontaneous, self-generated, autonomous or voluntary activity is the vital essence of living creatures, and especially human beings. It can be viewed as the highest expression of human activity. Because it requires a fully functioning brain, impairment of spontaneous behavior occurs following any injury to the highest centers of the brain, including the frontal lobes and limbic system, as well as the deeper reticular activating system. Because higher brain functions are fragile and dependent on overall physical well-being, a deactivating loss of spontaneous, self-generated behavior is often the first sign of any physical impairment or illness, from head injury and chronic fatigue to flulike illnesses, hormonal disorders, and brain tumors. Similarly, deactivation is one of the earliest and most essential effects of any psychoactive drug—that is, any drug that disrupts the function of the brain and mind—including all psychiatric drugs.

A variety of adverse drug reactions can be subsumed under the broader concept of deactivation. Some of these reactions include drug-induced diminished initiative, indifference, apathy, lethargy, psychomotor retardation, and loss of interest. Drug-induced depression, sedation, drowsiness, emotional dulling or blunting, malaise, and passivity often reflect a degree of deactivation. In the animal literature concerning psychiatric drug effects, deactivation is described as reductions in overall activity, spontaneous activity, social interactions, and exploration.

Biopsychiatric treatments are deemed effective when the physician and/or the patient prefers a state of diminished brain function, with its narrowed or shallower range of mental capacity or emotional expression. If the drugged individual reports feeling more effective and
powerful, it is most likely based on an unrealistic appraisal, impaired judgment, or euphoria associated with medication spellbinding. When patients on so-called maintenance doses do not experience noticeable effects, either the dose is too low to have a clinical effect, or the patient is unable to perceive the drug’s impact, again characteristic of medication spellbinding.

IV. Each biopsychiatric treatment produces its essential or primary brain-disabling effect on all people, including normal volunteers and patients with varied psychiatric diagnoses.

Despite the deeply held convictions of drug proponents, there are no specific psychoactive drug treatments for specific mental disorders. There is, of course, a certain amount of biological and psychological variation in the way people respond to drugs, shock treatment, or even lobotomy or an accidental head injury. However, as a general principle, biopsychiatric interventions have a nonspecific impact that does not depend on the person’s mental state or condition. For example, it will be shown that neuroleptics and lithium affect animals and normal volunteers in much the same way as they affect patients, in part by subduing their overall emotional responsiveness.

ILLUSTRATIVE RESEARCH CONFIRMING THE BASIC FOUR BRAIN-DISABLING PRINCIPLES

The first four principles are the heart of the brain-disabling concept: basically, that all psychiatric drugs cause a generalized impairment of brain function that reduces overall mental and emotion function; that this disabling effect occurs, as well, in normal volunteers; and that the effect has no specificity for any psychiatric disorder.

On occasion, research studies directly confirm the brain-disabling principle, but without intending to do so and without acknowledging it. In some ways, this is the most objective kind of research in that the researchers are unaware of the principle that they are testing. The following three studies involve the second-generation or atypical neuroleptic risperidone (Risperdal), which is widely prescribed to children and adults.

Peter Liddle and his colleagues (2000) used positron emission tomography (PET) to study the effects of risperidone on the rate of metabolism on the ventral striatum, thalamus, and frontal cortex. Their subjects were eight neuroleptic-naïve patients diagnosed with their first episodes of schizophrenia.
First and foremost, Liddle et al. (2000) found that “a single dose of risperidone produced decreases in metabolism in ventral striatum, thalamus and frontal cortex.” The authors identified this region as the cortico–striato–thalamo–cortical feedback loop. This encompasses much of the emotion-regulating centers in the limbic system and higher mental centers in the frontal lobes. Dopaminergic neurotransmission plays a significant role in this system and is profoundly blocked by risperidone. Clearly, this confirms that risperidone, like all neuroleptics, causes a chemical lobotomy, with the inevitable production of relative degrees of apathy and indifference.

Moreover, according to Liddle et al. (2000), “after six weeks’ treatment with risperidone, the decreases in frontal lobe metabolism were more extensive.” In other words, the risperidone produced a progressive chemical lobotomy with suppression of frontal lobe function.

In keeping with the brain-disabling principle, Liddle et al. (2000) were able to correlate a progressive suppression of symptoms with the exposure to risperidone. Although they tested for a variety of symptoms, they only reported a decreased severity of reality distortion. Reality distortion turns out to be a global clinical impression of the patient’s delusions and hallucinations. There is certainly no question that a chemical lobotomy (or a surgical lobotomy) reduces the individual’s expression of delusions and hallucinations. It does this by suppressing limbic system and frontal lobe function, causing apathy and indifference. The patients no longer care enough to express their more florid symptoms, but they also no longer care about anything. It is a global deactivation.

Liddle et al. (2000) try to correlate the reduction in reality distortion with suppression of a presumably overactive region of the hippocampus, but this is a huge stretch of the imagination. The facts are simple: The PET shows a global suppression of metabolism, and hence function, in the limbic system and frontal lobes, with increasing impact on the frontal lobes over a 6-week period, correlated with the patients no longer communicating as much about their symptoms. This is a demonstration of the brain-disabling concept of neuroleptic treatment.

Again using PET, Ngan et al. (2002) measured cerebral metabolic activity in patients before neuroleptic exposure, after an initial dose of risperidone and after 6 weeks of treatment. They found a reduction of frontal lobe function, and, in keeping with my suggestion in the 1997 edition of this book, they called it deactivation. They concluded that this decrease in frontal lobe metabolism is a function of the drug and not “schizophrenia” and that the mechanism of antipsychotic drug action is a “reduction in cortical metabolism,” especially in the frontal and temporal regions. This is a pillar of the brain-disabling concept: that psychiatric drugs work by disabling the higher centers of the brain. The authors
pointed out that a healthy control group is needed to further demonstrate that the drug's primary effect is separate from the patient's disorder and would occur in any group of individuals, normal or abnormal.

Lane et al. (2004) conducted a related study that could have been planned for the specific purpose of testing the brain-disabling principle. Using PET, they measured changes in regional metabolism produced by a single 2-mg dose of risperidone and by placebo, administered in a randomized, double-blind study of nine healthy subjects. Their results confirm that risperidone has the same effect on normal people as people labeled schizophrenic and that it acts by reducing brain function in areas critical to overall mental functioning. They stated,

**Results:** Compared with placebo, risperidone produced reductions in metabolism in the left lateral frontal cortex and right medial frontal cortex in healthy subjects. Conjunction analysis reveals that these changes occurred at locations similar to the loci of change produced risperidone with schizophrenia.

The researchers then concluded that there is a link between this reduced metabolism (a brain-disabling effect) and the reduction of clinical symptoms in patients diagnosed with schizophrenia:

Because the reduction in metabolism in the medial frontal cortex produced by risperidone is associated with alleviation of positive symptoms in patients with schizophrenia, the observation of a reduction in metabolism at a similar site in healthy subjects supports the hypothesis that the antipsychotic effect of risperidone arises, at least in part, from a physiologic effect that occurs in both patients with schizophrenia and healthy subjects.

The positive symptoms found in patients diagnosed with schizophrenia, such as hallucinations and delusions, can be suppressed by any brain-disabling trauma, from electroshock and lobotomy to neuroleptic drugs. This is in contrast to the negative symptoms, such as apathy, which are worsened by disabling or suppressing brain function. If it had been measured, the deactivation of the frontal lobes would also have correlated with a reduction in all spontaneous mental activity and verbal expressions, which is a commonly observed clinical phenomenon during neuroleptic treatment. This suppressive effect is often identified as psychomotor retardation, parkinsonian symptoms, or an apathylike syndrome of indifference.

Studies such as these three involving risperidone completely confirm the brain-disabling principles of psychiatric treatment. There should no longer be any scientific doubt about the correctness of the brain-disabling
Brain-Disabling, Spellbinding Effects of Psychiatric Drugs

concept, although its general acceptance requires letting go of numerous myths surrounding psychiatric treatment.

SIX ADDITIONAL BRAIN-DISABLING PRINCIPLES

The last series of brain-disabling principles describe clinical phenomena associated with treatment-induced brain disability.

V. Patients respond to brain-disabling treatments with their own psychological reactions such as apathy, euphoria, compliance, or resentment.

There is some variation in the way individuals respond to drugs. For example, the same antidepressant will make one person sleepy and another energized. Ritalin quiets many children but agitates others.

It can be very difficult to separate out drug-induced from psychologically induced responses. For example, all antidepressants can cause euphoria and mania. At the same time, some of the people who receive these drugs have their own tendency to develop these mental states. Similarly, a variety of drugs are capable of generating agitation and hostility in patients, yet people can develop these responses without medication. The docility and compliance seen following the administration of neuroleptics can be caused by the drug-induced deactivation syndrome but can also result from the patient’s realization that further resistance to psychiatric authority and control is futile or dangerous.

VI. To the extent that a physical disorder of the brain afflicts the individual, currently available biopsychiatric interventions will worsen or add to the disorder.

The currently available biopsychiatric treatments are not specific for any known disorder of the brain. One and all, they disrupt normal brain function, without correcting any brain abnormality. Therefore, if a patient is suffering from a known physical disorder of the brain, biopsychiatric treatment can only worsen or add to it. A classic example involves giving Haldol to control emotionally upset Alzheimer’s patients. While subduing their behavior, the drug worsens their dementia (chapters 2–4).

After psychiatric drugs are developed and marketed by drug companies, attempts are made to justify their use on the basis of correcting presumed biochemical imbalances. For example, it is claimed that Prozac helps by improving serotonergic neurotransmission. Even electroshock
and lobotomy are justified on the grounds that they correct biochemical imbalances. There is no likelihood that these intrusions correct a biochemical imbalance. A wide variety of brain-disabling agents are used to treat the same or similar disorders—everything from Prozac to Xanax to electroshock is prescribed for depression—and each treatment ends up disrupting innumerable brain functions. In reality, all currently available biopsychiatric interventions cause direct harm to the brain and hence to the mind, without correcting any known malfunction.

The pharmaceutical industry has lobbied hard to convince the U.S. Congress, the health professions, and the public that emotional problems such as depression and anxiety are biological in origin. The supposed biological basis of psychiatric disorders is then used to justify the widespread sale of their products, psychiatric drugs. But even if one or another psychiatric disorder someday turns out to have a biological basis, that in no way would justify inflicting psychiatric drugs on these patients, thereby compounding their underlying brain disorder with drug toxicity.

VII. Individual biopsychiatric treatments are not specific for particular mental disorders.

It is often said that psychiatry has specific treatments for specific diagnostic categories of patients, for example, neuroleptics for “schizophrenia”; antidepressants for depression; benzodiazepine tranquilizers for anxiety; lithium for mania; and stimulants, such as Ritalin, for attention-deficit hyperactivity. In actual practice, many individual patients are given all of the above categories of drugs at one time or another, and, increasingly so, all at once. Often the recommended use of a drug changes over the years. While there is a general tendency for patients labeled schizophrenic to be initially treated with neuroleptics or for depressed patients to be initially prescribed antidepressants, this is, in part, a matter of convention within the profession.

When a drug seems more effective for a particular disorder, it often depends on whether it has a suppressive or an energizing effect on the central nervous system. For example, if depressed patients are already emotionally and physically slowed down, giving them a neuroleptic that causes psychomotor retardation would tend to make them look worse. These patients are more likely to seem improved when artificially energized. Conversely, if patients diagnosed with schizophrenia become agitated and difficult to control, it would not make sense to give them stimulants. They are more likely to be judged improved when taking a neuroleptic that reduces or flattens their overall emotional responsiveness. Similarly, if a child is bored and restless in the classroom, stimulants
such as Ritalin, Adderall, and Strattera will suppress spontaneous behavior and enforce obsessive–compulsive behavior, giving an illusion of improvement (chapter 10). These gross behavioral effects, however, are a far cry from having a magic bullet for a specific disease.

VIII. The brain attempts to compensate physically for the disabling effects of biopsychiatric interventions, frequently causing additional adverse reactions and withdrawal problems.

The brain does not welcome psychiatric medications as nutrients. Instead, the brain reacts against them as toxic agents and attempts to overcome their disruptive impact. For example, when Prozac induces an excess of serotonin in the synaptic cleft, the brain compensates by reducing the output of serotonin at the nerve endings, by reducing the number of receptors in the synapse that can receive the serotonin, and by increasing the capacity of the transport system to remove serotonin from the synapse. Similarly, when antipsychotic drugs such as Risperdal, Zyprexa, or Haldol reduce reactivity in the dopaminergic system, the brain compensates, producing hyperactivity in the same system by increasing the number and sensitivity of dopamine receptors. All of these compensatory reactions create new abnormalities in brain function, sometimes causing irreversible disorders, such as antipsychotic drug–induced tardive dyskinesia (chapter 4).

It is difficult, if not impossible, to determine accurately the underlying psychological condition of a person who is taking psychiatric drugs. There are too many complicating factors, including the drug’s brain-disabling effect, the brain’s compensatory reactions, and the patient’s psychological responses to taking the drug. I have evaluated many cases in which patients have deteriorated under the onslaught of multiple psychiatric drugs without the prescribing physicians attributing the patients’ decline to drug toxicity. Instead, physicians typically attribute their patients’ worsening condition to “mental illness” when in reality the patient is suffering from adverse drug reactions.

Because the brain attempts to compensate for the effects of most psychoactive drugs, patients can have difficulty withdrawing from them. Physically, the brain cannot recover from the drug effect as quickly as the drug is withdrawn so that the compensatory mechanisms can require weeks or months to recover after the drug has been withdrawn. Sometimes, as in tardive dyskinesia, the brain fails to recover. In some cases, patients who have taken the newer antidepressants such as Prozac, Paxil, Zoloft, and Celexa for months or years cannot withdraw from them owing to the emotional instability and physical symptoms produced by drug-induced changes in the brain.
Physicians who prescribe biopsychiatric interventions often have an unrealistic appraisal of their risks and benefits.

An entire book could be written about how little physicians appreciate the risks associated with the psychiatric drugs that they prescribe and how much they overestimate their effectiveness. The Food and Drug Administration (FDA), medical and psychiatric associations, experts with a vested interest in promoting drugs, and the pharmaceutical industry—the psychopharmaceutical complex—combine to push doctors to prescribe psychiatric drugs to children and adults.

What about the clinical judgment of individual physicians? The individual physician is not in a good position to assess the effectiveness of psychiatric drugs. In recent years, doubt has even been thrown on the objectivity of controlled clinical trials, in which drugs are compared to placebo or to alternative medications (see chapters 6–7). Too often, the investigators are influenced by their conscious or unconscious biases.

If clinical and scientific studies can be distorted by bias, it is even more likely that routine clinical practice will be affected by the hopes and expectations of the prescribing physician. Physicians in great numbers have prescribed drugs with unbounded enthusiasm for years before the agents have proven to be worthless or unacceptably dangerous. Amphetamines, for example, were freely dispensed for many years to millions of patients for both depression and weight control, without regard for their lack of efficacy, long-term hazards, and addictive potential (chapter 11). Although there has been some increased caution in recent years, benzodiazepines such as Valium and Xanax have been overly prescribed for anxiety, despite the fact that they worsen anxiety in long-term use, cause persisting memory and mental deficits, and frequently produce abuse and dependence (chapter 12). Antidepressants continue to be given freely to children and adolescents, even though the FDA itself has admitted that multiple studies have failed to prove them useful (chapter 6). Indeed, the effectiveness of antidepressants in treating depressed adults is also in doubt ( chapters 6–7), while their adverse effects can be life threatening and make withdrawal impossible, yet most physicians think of them as very safe and efficacious. In even more extreme examples, both psychosurgery and electroshock continue to be utilized, despite obviously devastating effects on the mental lives of the patients and the absence of proven efficacy ( chapter 9).

Patients subjected to biopsychiatric interventions often display poor judgment about the positive and negative effects of the treatment on their mental and emotional functioning, often causing intoxication anosognosia (medication spellbinding).\footnote{4}
Generalized brain dysfunction tends to reduce the individual’s ability to perceive the existence or impact of the dysfunction. This incapacity lies at the heart of spellbinding effects of drugs and is one of the main reasons that patients continue to take psychiatric medications when the drugs are doing more harm than good.

Anosognosia refers to the capacity of brain damage to cause denial of lost function. Anosognosia is a hallmark of central nervous system disability from any cause (Breggin, 2006d; see subsequent sections).

Human beings are physically and psychologically complex, with varying reactions to drugs. As a result, no two cases of medication spellbinding are identical, they vary widely in intensity, and not all cases will display every characteristic. Nonetheless, spellbinding is a readily identifiable clinical phenomenon that probably characterizes all cases of drug intoxication from mild to severe and probably can be found to some degree whenever a psychoactive agent is having an impact on brain and mind.

The following four characteristics of medication spellbinding are taken from this author’s book *Medication Madness* (Breggin, in press):

First, spellbound individuals fail to perceive the degree of mental or emotional impairment that the drugs are inflicting on them.

Second, spellbound individuals tend to rationalize and justify their drug-induced mental distress, typically blaming negative feelings on themselves or on something else, sometimes leading to violence against themselves or others.

Third, spellbound individuals often feel as if they are doing better than ever when in reality they are doing worse.

Fourth, extreme spellbinding produces medication madness in which the individual feels driven or compelled to behave in out-of-character and potentially disastrous ways—to murder her beloved mother like Emily Ashton or to drive his car into a policeman like Harry Henderson. The spellbound actions are typically carried out without the individual realizing that he or she is drug impaired and without the individual stopping to consider or grasping the disastrous consequences.

To practice applying the four principles of spellbinding, the reader can simply recall how individuals act when intoxicated with alcohol. Typically, people intoxicated with alcohols do not realize how impaired they have become; when they become emotionally distressed, they blame it on someone or something other than alcohol intoxication, often becoming depressed or belligerent; they often think that they feel better than ever when they are in reality mentally impaired and behaving badly; and finally, they can do stupid things and even perpetrate violence that is wholly out of character for them when sober.
Many individuals who chronically smoke marijuana believe that it improves their overall psychological and social functioning, but if they withdraw from the drug, it may become apparent to them that their memory, mental alertness, emotional sensitivity, and social skills have been impaired while using the drug. People intoxicated with stimulants, such as amphetamine, may feel they have superior or even superhuman capacities, when they are often seriously impaired. The same is true of all psychiatric drugs. Often the patient will have little appreciation for the degree of mental or emotional impairment until the drug has been stopped for some time and the brain has had time to recover.

In my clinical practice and in my work as a medical expert in legal cases, I often find that people are dismayed at how much better they function when they have been safely withdrawn from psychiatric medications. Many of these patients have remained for years in severe states of intoxication from one or more psychiatric drugs without realizing it. Attributing their condition to their own emotional reactions or to stresses in the environment, they have asked their doctors for more medication.

Owing to brain damage–induced spellbinding, even after a devastating series of shock treatments or psychosurgery, patients may fail to understand the iatrogenic source of their mental dysfunction and instead believe that they need repeated interventions.

**THE BIOLOGICAL BASIS OF MEDICATION**

**SPELLBINDING**

Some degree of spellbinding is characteristic of any compromise of frontal lobe function. Beer et al. (2006) noted that orbitofrontal damage is “associated with objective inappropriate social behavior.” The patients “were aware of social norms of intimacy” but “they were unaware that their task performances violated these norms.” The authors call this an impairment of self-monitoring and self-insight. Bach and David (2006) pointed out that self-awareness deficits are very common in patients with traumatic brain injury and key to the development of behavior disturbances: “Our research found that lack of social self-awareness predicts behavioural disturbance in acquired and traumatic brain injury independent of cognitive and executive function.”

Lobotomized or electroshocked patients as well as patients chemically lobotomized by neuroleptics have greatly impaired self-awareness. They often fail to perceive their mental dysfunction and will neglect warning signs of physical illness in themselves. Consistent with spellbinding, they are likely to report that they are doing better than they are. A study of the atypical neuroleptics, including risperidone, olanzapine,
and quetiapine, found that these patients unrealistically rated themselves as improved in quality of life (Voruganti et al., 2000): “These perceived benefits, however, were not reflected in the clinician rated (objective) measure of psychosocial functioning and quality of life.”

These gross disruptions of the frontal lobes, including neuroleptic toxicity, usually subdue individuals, making them docile, thereby preventing dangerous disinhibition that might otherwise occur in the absence of self-monitoring and self-insight. However, many psychoactive drugs, including antidepressants, benzodiazepine tranquilizers, and stimulants, can markedly disinhibit and/or energize and drive the individual to act in a compulsively destructive manner, sometimes leading to criminal behavior, suicide, and violence (Breggin, 2006d, in press). When neuroleptics cause akathisia, they can also drive individuals toward out-of-control behaviors.

The biological bases for the individual’s failure to perceive adverse drug effects on his or her mental life include the following interrelated phenomena:

- **Drug-induced confusion.** Almost all biopsychiatric interventions can at times induce confusion, impairing the patient’s awareness of the drug-induced mental dysfunction.

- **Drug-induced short-term memory loss.** Psychoactive drugs frequently impair recall and also disrupt the order of past memories, making it more difficult for individuals to recognize how a drug has been affecting them.

- **Drug-induced mental disturbances, especially various degrees of apathy and mania.** All psychiatric drugs can produce either indifference or euphoria, and many—for example, the newer antidepressants, the stimulants and the benzodiazepine Xanax—can produce both. Apathy and indifference make people less aware of and less concerned about drug-induced impairments. If the person is suffering a great deal, the apathy may be welcomed. Euphoria and mania override any sense of impairment, instead making the individual feel better, stronger, and more able than ever.

- **Drug-induced confabulation.** Confabulation is a symptom of generalized brain dysfunction with marked memory impairment. The patient uses rationalizations and various cover stories to hide the extent of mental dysfunction from himself and others. Confabulation is well understood in psychiatry and neurology but is generally ignored in regard to treatment-induced effects. Many patients confabulate good results from drug therapy, although they are obviously impaired by it.
Psychological influences also play a role in the patient’s tendency to misperceive or misjudge the effects of drugs, but they are not central to the concept of medication spellbinding, which is biologically based. Psychological influences include the following:

- **Psychological denial.** Individuals overcome by emotional suffering are likely to deny the degree of their psychological dysfunction. They do not want to admit to being severely mentally impaired. If they are hoping to feel better with the use of a drug, or if the drug initially caused euphoria or emotional anesthesia, their denial can be further reinforced.

- **Placebo effect.** Patients have faith that biopsychiatric interventions will be helpful, rather than harmful, encouraging them to disregard drug-induced dysfunction or to mistakenly attribute it to their emotional problems.

- **Compliance.** To an extraordinary extent, patients will tell doctors what the doctors want to hear. If a psychiatrist clearly wants to hear that a drug is helpful, and not harmful, many patients will comply by giving false information or by withholding contradictory evidence.

- **Psychologically induced confusion.** Emotionally upset individuals can easily lose their judgment concerning the cause of their worsening conditions. They can easily mistake a negative drug effect, such as rebound anxiety from a benzodiazepine tranquilizer like Xanax or Ativan or depression from a neuroleptic like Risperdal or Abilify, for a worsening of their emotional problems. Typically, they blame themselves rather than the medication. This confusion is abetted when the physician exaggerates the drug’s benefits and fails to inform the patient of its potential adverse effects.

**IATROGENIC HELPLESSNESS AND DENIAL IN AUTHORITARIAN PSYCHIATRY**

In the previous edition of *Brain-Disabling Treatments in Psychiatry*, I introduced the term *iatrogenic helplessness and denial in authoritarian psychiatry* to designate a guiding principle of biopsychiatric interventions (see also Breggin, 1983a). Although they may not recognize or admit what they are doing, biological psychiatrists use authoritarian techniques,
enforced by brain-disabling interventions, to produce increased helplessness and dependency on the part of the patient. In their journals and conferences they frequently speak of obtaining “medication compliance”—getting the patient to take drugs. In an effort to push their patients to take medications, biological psychiatrists convince them that they have biochemical imbalances, and even genetic disorders, that require treatment with drugs. This creates a submissive, dependent relationship with the prescribing physician. Physically, the psychiatrist prescribes multiple drugs or electroshock, causing brain damage and dysfunction that increases the patient’s tendency to be submissive and dependent. Often these doctors encourage their patients to enter mental hospitals, and sometimes they force them into hospitals or into outpatient commitment in which they are required to submit against their will to medication.

This may seem like a harsh indictment, but it is instead a harsh reality. While most psychiatrists may not realize that they are causing dependency and helplessness, millions of patients throughout the nation are misled into believing that they have biological and genetic defects that can be corrected by medication or electroshock, in effect making them feel helpless and dependent on their doctors and on physical treatments. When many of these patients become worse as a result of treatment, they are told that their underlying “mental illness” is surfacing. When multiple drugs lead to escalating adverse emotional effects, more drugs are added to the regimen, and too often the patient is hospitalized. Rarely do these doctors admit that the drugs are the source of the patients’ worsening problems and that a drug-free period of time may lead to recovery. Throughout the process, the patients remain so spellbound that they cannot perceive how badly they are doing or that the drugs are ruining their lives.

The concept of iatrogenic helplessness and denial includes the patient’s and the doctor’s mutual denial of the damaging impact of the treatment as well as their mutual denial of the patient’s underlying psychological and situational problems. Overall, iatrogenic helplessness and denial accounts for the frequency with which psychiatry has been able to utilize brain-damaging technologies, such as electroshock and psychosurgery, as well as toxic medications. Spellbinding explains how the biological impact of the medication reinforces iatrogenic helplessness and denial.

**RELATIONSHIP BETWEEN MEDICATION SPELLBINDING AND IATROGENIC HELPLESSNESS AND DENIAL**

The concept of medication spellbinding expands or elaborates on the concept of iatrogenic helplessness and denial. It specifically observes that
patients exposed to psychiatric drugs, electroshock, or lobotomy display the following indications of helplessness and denial: (a) impairment in their ability to perceive their treatment-induced mental dysfunction; (b) inability to identify that the drug, shock, or lobotomy is causing their deterioration and a tendency to attribute their distress to some other source, such as their own so-called mental illness or someone else’s distressing effect on them; (c) an unrealistic belief that they are doing better than ever, when they are doing worse; and (d) in extreme cases, the development of compulsive, destructive, ego-alien actions, sometimes of a manic quality.

Most people who seek psychiatric treatment are already vulnerable to becoming helpless and dependent. Before the potential patient encounters a psychiatrist, he or she has usually been feeling helpless for some time. In my formulation, as described in The Heart of Being Helpful (1997b), helplessness is the common denominator of all psychological failure. Helplessness is at the core of most self-defeating approaches to life. People who feel helpless tend to give up using reason, love, and self-determination to overcome their emotional suffering, inner conflicts, and real-life stresses. They instead seek answers from outside themselves. In modern times, this often means from so-called experts.

Iatrogenic helplessness and denial, and medication spellbinding, go far beyond relatively benign suggestion (as used in medicine and psychiatry, e.g., to help overcome physical pain). First, in iatrogenic helplessness and denial, including medication spellbinding, the psychiatrist compromises the brain of the patient, enforcing the patient’s submission to suggestion through mental and physical dysfunction. Second, in iatrogenic helplessness and denial, the psychiatrist denies to himself or herself the damaging effects of the treatment as well as the patient’s continuing psychological or situational problems.

Brain damage and dysfunction from any cause, including accidents and illness, frequently produce helplessness and denial, but only in psychiatry is damage and dysfunction used as treatment to produce these disabling, spellbinding effects.

MENTAL AND EMOTIONAL SUFFERING ROUTINELY TREATED WITH BIOPSYCHIATRIC INTERVENTIONS HAVE NO KNOWN GENETIC OR BIOLOGICAL CAUSE

Keep in mind that the validity of the brain-disabling concept does not depend on the origin of psychiatric disorders but rather on the known effects of biopsychiatric treatment. Even if one or another psychiatric disorder should turn out to have a biological basis, it would not justify using current medications, all of which disable the brain. Although most
people who seek psychiatric care have nothing wrong with their brain function, some may have an underlying physical disorder (not a mythical biochemical imbalance). If, for example, a patient has a thyroid disorder or diabetes that is causing feelings of depression, the patient should be given proper medical treatment to correct the underlying physical disorder and not antidepressant drugs.

So-called schizophrenia is usually put forward as the best model for a biological and genetically based psychiatric disorder. For critiques of the genetics of schizophrenia, see Breggin (1991b) and, more recently, Joseph (1999, 2004a, 2004b, 2006). There are many detailed criticisms of the brain disease model for schizophrenia (see, e.g., Siebert, 1999) and for biochemical theories of psychiatric disorders (Breggin, in press; Colbert, 2001).

Timothy Crow’s (2007) article “How and Why Genetic Linkage Has Not Solved the Problem of Psychosis: Review and Hypothesis” confirmed that even the genetic researchers admit they have not found a genetic linkage for schizophrenia. Meanwhile, the search for a biological basis, or a biological marker, for depression also continues to run aground.

“What Have We Learned About the Neurobiology of Major Depression?” by Maria Oquendo and Ramin Parsey (2007) demonstrated that as of April 2007, no genetic or biological causes have as yet been discovered. As always, the editorial talks about how the search must go on. All of this, of course, will feel intellectually jarring to most health care providers, who have been taught to believe that psychiatric disorders have known biological and genetic causes.

Despite more than 200 years of intensive research, no commonly diagnosed psychiatric disorders have been proven to be either genetic or biological in origin, including the diagnostic categories of schizophrenia, major depressive disorder and bipolar disorder, the various anxiety disorders, and childhood disorders such as attention-deficit hyperactivity.

At present, there are no known biochemical imbalances in the brain of typical psychiatric patients—until they are given psychiatric drugs. It is speculative and even naïve to assert that antidepressants such as Prozac correct underactive serotonergic neurotransmission (a serotonin biochemical imbalance) or that neuroleptics such as Risperdal or Seroquel correct overactive dopaminergic neurotransmission (a dopamine imbalance). The failure to demonstrate the existence of any brain abnormality in psychiatric patients, despite decades of intensive effort, suggests that these defects do not exist.

It seems theoretically possible that some of the problems treated by psychiatrists and other health practitioners could eventually be proven to have a biological basis. As already mentioned, mental function often improves when certain physical disorders, such as hypothyroidism...
or Cushing's syndrome, are adequately treated with appropriate medical interventions.

However, the vast majority of problems routinely treated as so-called mental disorders do not remotely resemble diseases of the brain or body. For example, they do not produce the cognitive deficits in short-term memory or abstract reasoning characteristic of brain disorders. They are not accompanied by fever or laboratory signs of illness. Unlike many neurological disorders, they are not degenerative. To the contrary, neurological and neuropsychological testing usually indicates normal if not superior brain function, and the body is healthy—until the brain-damaging treatments are begun. There seems little likelihood that any of the routinely treated psychiatric problems are based on brain malfunction, rather than on the life experiences of individuals with normal brains.

To claim that an irrational or emotionally distressed state, however extreme, in itself amounts to impaired brain function is simply false. An analogy to television sets and computers may illustrate why this is so. If a TV program or Internet site is offensive or irrational, it does not indicate that anything is wrong with the electronics of the television set or the hardware of the computer. It makes no sense to attribute the bad programming or the offending Internet site to bad wiring. Similarly, a person can be very disturbed psychologically, without any corresponding defect in the wiring of the brain.

However, the argument is moot since no contemporary biopsychiatric interventions can truthfully claim to correct a brain malfunction the way an expert can fix a broken TV set or computer. Instead, we blindly inflict toxic substances on a brain that is far more subtle and vulnerable to harm than the hardware of a TV or computer. We even shock or mutilate the brain in ways that would appall TV or computer repair persons or their customers, all of whom would instantly recognize that these treatments were ruining their TV sets or computers.

It is often suggested that persons suffering from extremes of emotional disorder, such as hallucinations and delusions or suicidal and murderous impulses, are sufficiently abnormal to require a biological explanation for their mental processes or behavior. However, the emotional life of human beings has always included a wide spectrum of mental and behavioral activity. Individual willingness or ability to remain rational and to control one's emotions varies enormously. That a particular mental state or action is especially irrational or destructive does not, per se, indicate a physical origin. If extremes require biological explanation, then it would be more compelling to ascribe extremely ethical, rational, and loving behaviors to genetic and biological causes since they are especially rare in human life.
The fact that a drug works—that is, influences the brain and mind in a seemingly positive fashion—does not confirm that the individual suffers from an underlying biological disorder. Throughout recorded history, individuals have medicated themselves for a variety of spiritual and psychological reasons, from the quest for a higher state of consciousness to a desire to make life more bearable. Alcoholic beverages, coffee and tea, tobacco, and marijuana are commonly consumed by people to improve their sense of wellness. Yet there is no reason to believe that the results they obtain are due to an underlying biochemical imbalance.

CONCLUSION

As I have discussed in earlier books (Breggin, 1991a; Breggin et al. 1994a, 1994b), I believe that the concepts of mental illness and mental disorder are misleading and that none of the problems commonly treated by psychiatrists are genetic or biological in origin. The terms attention-deficit hyperactivity disorder, schizophrenia, and major depressive disorder, for example, are based on concepts whose validity can easily be challenged. However, the brain-disabling principles remain valid, even if some of the mental phenomena that are being treated turn out to have a genetic or biological basis. All of the currently available biopsychiatric treatments—drugs, electroshock, and psychosurgery—have their primary or “therapeutic” effect by impairing or disabling normal brain function, causing iatrogenic helplessness and denial and, more specifically, intoxication anosognosia (medication spellbinding).

NOTES

1. The term euphoria as used in psychiatry indicates an exaggerated, irrational, or unrealistic sense of well-being. It can be psychological in origin but is commonly caused by brain damage or drug toxicity.
2. Because most laypersons and many physicians do not know the generic names for drugs, I will occasionally use trade names, such as Prozac and Risperdal, throughout the book. However, the appendix offers a list of psychiatric drugs by category, including both trade and generic names.
3. Euphoria is unusual in patients treated with the neuroleptics because of the suppressive effects on the central nervous system (see chapter 2). It is more common among patients treated with antidepressants, stimulants, and benzodiazepine tranquilizers, especially alprazolam. Drug-induced mania is an extreme of medication spellbinding.
4. The concept of medication spellbinding occurred to me when I was reviewing a lifetime of clinical and legal cases in the process of writing a new book, Medication Madness
(Breggin, in press), which describes approximately 70 cases that I had personally evaluated (see also Breggin, 2006e).

5. In the previous edition of this book, this subtitle was one of the brain-disabling principles, but I have removed it from the principles because, even if some future psychiatric disorder proves to have a genetic or biological basis, the current treatments in use will nonetheless remain toxic and cause brain disability.