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Looking Backward: The Twentieth Century Revolutions in Psychiatry, Law and Public Mental Health

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Looking Backward: The Twentieth Century Revolutions in Psychiatry, Law, and Public Mental Health

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I. INTRODUCTION

Do histories of psychiatry make a difference—or have legal implications—in the present? Does our current situation help explain what historians say about psychiatry's past? Focusing on the past half century—the era of medications—this paper explores the reciprocal relationship between the present and the past in psychiatry.[1]

Part II sketches the medical developments that constitute the subjects of any history of psychiatry. This Part also examines related developments in law. Part III introduces some problems of psychiatric historiography and examines some historians' attempts to deal with them. Part IV analyzes the account of psychiatry's past contained in Edward Shorter's well-regarded book, *A History of Psychiatry: From the Era of the Asylum to the Age of Prozac*.[2] Finally, the conclusion suggests two relationships between legal developments and the writing of psychiatric history.

II. PAST AND PRESENT

A. Before Medications

In the latter part of the nineteenth century, American psychiatrists lost faith in the value of hospitalization alone and turned to physical interventions.[3] They gave sedatives to make patients more manageable. And they performed procedures with supposedly profound effects on mental illness. One of these, "malarial fever therapy," proved effective in some cases. Neurosyphilis, an infectious disease which manifested itself as insanity, sometimes responded well when patients were exposed to malaria. Physicians intentionally gave patients malaria and then, after a short interval, cured the physician-induced

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1. When I refer to "psychiatry" in this paper, I generally mean the branch of that discipline concerned with serious mental diseases, such as schizophrenia.

2. EDWARD SHORTER, A HISTORY OF PSYCHIATRY, FROM THE ERA OF THE ASYLUM TO THE AGE OF PROZAC (1997).

3. *See* SHORTER, *supra* note 2, at 190; Peter Sterling, *Ethics and Effectiveness of Psychosurgery*, *in* CONTROVERSY IN PSYCHIATRY 126 (John Paul Brady & H. Keith H. Brodie eds., 1978).

disease. Strangely enough, this procedure actually helped as many as fifty percent of patients. Its inventor received the Nobel Prize.[4]

Other treatments, which seem no less strange, were probably less effective. Prolonged sleep therapy, for example, was an invention of the last years of the nineteenth century. Using various drugs, physicians kept patients in deep sleep for days on end. Some patients supposedly responded well but about one in twenty died.[5] Also in the late nineteenth century, surgeons removed the ovaries of women in an effort to cure mental illness.[6] This procedure was both ineffective and invidious. Then, in the 1930s, psychiatrists used a variety of agents to produce convulsions or to put mental patients into comas.[7] These procedures followed the model of malarial fever therapy; hoping to ameliorate mental illness, physicians produced temporary pathological states in patients. One of these treatments, electroconvulsive therapy, has been shown effective in treating serious mental illness.[8] Whatever their effectiveness, patients greatly feared these procedures, for obvious reasons.

Lobotomy—the surgical cutting of brain connections—also appeared in the 1930s.[9] Its inventor, Egas Moniz, received the Nobel Prize for his achievement in 1949.[10] Yet within four years of that triumph, the operation fell into disuse in the United States. It produced permanent brain damage, and in the 1950s—after the revelations of Nazi atrocities and the awakened sensitivity to human rights resulting from the civil rights movement—the American public had become unwilling to tolerate such damage as an instrument of public health policy.[11] Litigation threatened the operation as well.[12]

The same moral and legal considerations that had undone lobotomy threatened the coma and convulsive therapies as well. They, too, seemed brutal and crude, even if the patients who survived suffered no obvious permanent damage. For that matter, the reaction to Nazism threatened the very existence of mental hospitals.[13] During World War II, conscientious objectors had served as mental hospital orderlies. After the war, they reported with horror on conditions at the hospitals: the deteriorated buildings, infested with

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4. SHORTER, *supra* note 2, at 194.

5. *Id.* at 205.

6. Sheldon Gelman, *The Biological Alteration Cases*, 36 WM. & MARY L. REV. 1203, 1214 (1995); Lawrence D. Longo, *The Rise and Fall of Battey's Operation: A Fashion in Surgery*, 53 BULL. HIST. MED. 244, 253, 259 (1979).

7. SHORTER, *supra* note 2, at 207.

8. *Id.*

9. *Id.* at 226.

10. Sterling, *supra* note 3, at 133.

11. SHELDON GELMAN, MEDICATING SCHIZOPHRENIA 23, 24-27 (1999); GERALD N. GROB, FROM ASLYUM TO COMMUNITY: MENTAL HEALTH POLICY IN MODERN AMERICA 70-92 (1991).

12. *See* GELMAN, *supra* note 11, at 131.

13. *Id.* at 23.

rodents and vermin; the abusive and uncaring staff; the pervasive smell of human waste; the naked, neglected, and deteriorated patients. Frequent comparisons were made with Nazi concentration camps.[14] By 1950, the governors of various states had tired of the constant mental hospital scandals and the ever-increasing costs of running the institutions.[15] The governors met and decided that drastic changes were necessary.[16]

B. The Appearance of Medications

In this social and political setting, medications made their debut. Chlorpromazine, the first antipsychotic drug, appeared in 1952 and 1953 under the trade name Thorazine.[17] Named after Thor, the classical god of thunder,[18] Thorazine was marketed as electroconvulsive therapy in a bottle—the thunder, as it were, without the flash of electricity. Psychiatrists also described it as a "chemical lobotomy."[19] Through the 1950s, psychiatrists usually regarded medications as a therapy that produced pathology and worked in the same way as the older biological treatments—convulsions, comas, and lobotomy—but in a more convenient form.[20]

Hospital wards with medicated patients became much calmer and more orderly. Many patients, and particularly those with schizophrenia, improved, sometimes dramatically.[21] And by the middle of the 1950s, the patient population in some state hospitals had declined, albeit very slightly.[22] Any decline, however, represented a historic achievement. Meanwhile, more antipsychotic medications appeared on the market and more patients received the drugs.[23]

C. How Medications Work

Medications are clearly effective against some schizophrenic symptoms. Clinicians, ignoring decades of research results, often exaggerate these benefits.[24] Thus, medications usually lessen the intensity of hallucinations or

14. *Id.*
15. *See generally id.*
16. GROB, *supra* note 11, at 70.
17. GELMAN, *supra* note 11, at 24.
18. *Id.*
19. *Id.* at 25.
20. *Id.*
21. *Id.* at 38-39.
22. GELMAN, *supra* note 11, at 40-41.
23. *Id.* at 40.
24. *See generally id.* at 13.

delusions.[25] Yet "a substantial proportion of patients are resistant to treatment with neuroleptics."[26] Moreover, little evidence indicates that medicated patients—even the majority who respond favorably—enjoy better lifetime outcomes than patients experienced before drugs, or that medicated patients' quality of life has improved. Indeed, some studies suggest that medicated patients fare worse in both respects.[27] Moreover, most investigators find standard antipsychotics ineffective against the negative symptoms of schizophrenia, such as withdrawal from social life.[28] Describing patients who had responded favorably to medication in a 1975 study, for example, three leading researchers found that although all patients were in remission from all psychotic signs, this left room for considerable psychopathology, as well as vocational and interpersonal difficulties.[29] Their patients ran the gamut from those with a complete lack of psychopathology and with no functional impairment to others with chronical dysphoria (unhappiness) and marked vocational and social ineptitude, chronic psychiatric invalids supported by public assistance who led dismal, empty, withdrawn lives.[30] But none had delusions, hallucinations, disorganized speech, or bizarre behavior.[31]

Along with symptom reduction, medications also reduce the risk of a patient relapsing and suffering another acute psychotic episode. Yet it is not true that all relapses result from the failure to take medication, as clinicians often claim. Nor will every patient relapse without medication. In fact, about thirty to forty percent of medicated patients, as compared to about sixty to eighty percent of patients who discontinue medications, relapse over a two-year period.[32] Although substantial, this difference should not obscure the fact that many patients will remain free of relapse for substantial periods off medication—and that many patients will relapse despite receiving treatment. Moreover, these relapse estimates probably exaggerate the real differences. Two distinguished researchers in 1995 reviewed the relapse literature and discovered that

> [a]n extraordinarily large (13-fold) excess of relapse risk arose within the first 3 months after discontinuing neuroleptic [antipsychotic] treatment (50% vs 3.8%). The cumulative relapse risk rose moder-

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25. *Id.*

26. Kenneth L. Davis et al., *Dopamine in Schizophrenia: A Review and Reconceptualization*, 148 AM. J. PSYCHIATRY 1474, 1474 (1991).

27. *See generally* GELMAN, *supra* note 11, at 189, 191.

28. *See generally id.*

29. *See generally* Arthur Rifkin et al., *Akinesia: A Poorly Recognized Drug-Induced Extrapyramidal Behavioral Disorder*, 32 ARCHIVES GEN. PSYCHIATRY 672 (1975).

30. *See id.*

31. *See id.*

32. GELMAN, *supra* note 11, at 186-91.

> ately for those continued on maintenance medication to an average of 28.5% by 2 years. In contrast, there was little additional gain of risk after the first 3 months after drug discontinuation, with a maximal risk of 54% to 62% by 12 months. The relative risk fell over time to less than twofold.[33]

These researchers posited an "iatrogenic-pharmacologic stress effect" befalling patients who started—and then stopped—medications.[34] This effect, which could be minimized by very gradually discontinuing medication, made patients worse off than if had they never started medications in the first place. The result was "inflate[d]"[35] estimates of the advantage conferred by medication and "clinical risks for morbidity or even mortality . . . [that] sometimes exceed those associated with the natural history of the untreated illness."[36] Put differently, patients who had never received medication would relapse at rates much closer to the relapse rate of patients who received continued medication.

D. Side Effects

Medications cause serious and frequent side effects.[37] They produce mental distress, which can become severe, in perhaps a quarter to a half of patients. Many feel anxious, uneasy, or tormented. Other patients lose will power or initiative; their heads feel fuzzy.[38] These last effects are common and they explain why psychiatrists once described medications as "chemical lobotomies."[39]

Comparable numbers of patients develop abnormal physical movements on medications. Fingers and limbs tremble, jaws move, and tongues protrude,[40] or else patients develop a "masked face" and stiff gait that resemble the symptoms of Parkinson's disease.[41] These physical effects, like the

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33. Ross J. Baldessarini & Adele C. Viguera, *Neuroleptic Withdrawal in Schizophrenic Patients*, 52 ARCHIVES GEN. PSYCHIATRY 189, 189 (1995).

34. *Id.* at 190.

35. *Id.* at 191.

36. *Id.*

37. The following discussion of side effects is based on the account in GELMAN, *supra* note 11, *passim.*

38. For discussion of drug induced mental distress, see Theodore Van Putten & Philip R.A. May, *Subjective Response as a Predictor of Outcome in Pharmacology: The Consumer Has a Point*, 35 ARCHIVES GEN. PSYCHIATRY 477, 479 (1978).

39. GELMAN, *supra* note 11, at 25.

40. *See id.* at 6.

41. SHORTER, *supra* note 2, at 253.

subjective ones, can range from mild to severe. There are also potentially fatal side effects affecting heart function,[42] but these seem to be rare.

Some involuntary movements that drugs induce—and particularly purposeless movements called dyskinesias—become persistent or permanent, reflecting medication-caused damage to the brain or nervous system.[43] "Tardive dyskinesia" is the best known effect of this kind. Because of its potential permanence, and because it is seen so often in medicated patients, tardive dyskinesia has been regarded for the last thirty years as the most significant side effect of medications.[44]

Tardive dyskinesia raised the prospect of widespread, permanent neurological damage from drugs. Once it became clear that the public reviled lobotomy, psychiatrists had stopped describing medications as a chemical form of the surgery. The last such description I have found occurred in the early 1960s. Yet tardive dyskinesia threatened to make drugs chemical lobotomies all over again—with the possibly profound public and legal consequences. Biological therapies other than lobotomy had produced only transient damage to body or brain—comas, for example, or convulsions—and even these therapies had become suspect in the eyes of the public and government regulators. For good reason, psychiatrists feared that widespread permanent brain damage—if it existed—would lead to severe limits on using medication.[45]

During the late 1950s, early reports about isolated cases of tardive dyskinesia had appeared in Europe.[46] Despite these reports and an editorial in the Journal of the American Medical Association (JAMA) about the possibility of permanent neurological damage from medication,[47] American clinicians and researchers generally ignored tardive dyskinesia through the 1960s. The most remarkable exception was George Crane, a National Institute of Mental Health (NIMH) researcher who published articles and convened conferences on the subject.[48] In 1967, at a conference attended by eminent psychiatrists, Crane announced his own findings: a quarter of the patients on a NIMH research ward, he said, had tardive dyskinesia; the symptoms often persisted after medications were withdrawn; the movements could be extremely bizarre; and it appeared that the disorder was related to a patient's exposure to

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42. GELMAN, *supra* note 11, at 104.

43. *Id.* at 6.

44. *See* C. Thomas Gualtieri & Robert L. Sprague, *Preventing Tardive Dyskinesia and Preventing Tardive Dyskinesia Litigation*, 20 PSYCHOPHARMACOLOGY BULL. 346 (1984).

45. I discuss the potential impact of tardive dyskinesia in GELMAN, *supra* note 11, at 33.

46. The account of tardive dyskinesia in this paragraph, and in the two paragraphs that follow, is based on *id.* at 77.

47. Editorial, *Irreversible Side Effects of Phenothiazines*, 191 JAMA 333, 333-34 (1965).

48. GELMAN, *supra* note 11, at 78.

medication.[49] Leaders of the profession at once attacked Crane's findings and his professionalism. Crane was threatening to undo the progress brought by drugs, one said, suggesting that flaws in Crane's personal make-up were responsible.[50] On the merits, the critics argued that Crane had not seen any movements at all, that he had seen only movements symptomatic of schizophrenia, that supposed tardive dyskinesia victims were merely licking their lips, that their movements could be cured with a program of "deconditioning," that the movements were reversible rather than persistent, or that they were the product of lobotomies.[51] None of these often inconsistent criticisms had merit.

Over the next five years, Crane wrote prolifically about tardive dyskinesia and urged drug companies and government regulators to recognize the disorder.[52] At the same time, a few other American researchers began reporting tardive dyskinesia rates as high, or even higher, than Crane's.[53] On the other side, leading psychiatrists did not attempt to refute these results. Instead, they ignored them. With the exception of a 1968 article by Nathan Kline claiming that the true tardive dyskinesia rate was 1 in 1,000,000[54]—an absurdity—no well-known psychiatrist had anything to say about tardive dyskinesia in print. Yet the weight of the research began to tell. In 1973—almost fifteen years after the initial reports—an association of psychiatric societies and government agencies issued a grudging, equivocal acknowledgment that medications might cause tardive dyskinesia.[55] This acknowledgment included gross underestimates of the disorder's prevalence, but it was accompanied by the decision of leading pharmaceutical companies to warn about tardive dyskinesia in drug package inserts.[56] Despite these developments, as late as 1976, Jonathon Cole, an eminent psychopharmacologist, argued that tardive dyskinesia almost always resulted from prior damage to the brain, not caused by drugs, and that the disorder had virtually no implications for drug prescribing.[57]

By 1980, most researchers had accepted the fact that medications caused tardive dyskinesia and that the condition often became permanent. In part, this

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49. *Id.* at 78-80; *see* George E. Crane, *Tardive Dyskinesia in Schizophrenic Patients Treated with Psychotropic Drugs*, 9 AGGRESSOLOGIE 209, 212, 216 (1967); George E. Crane & George Paulson, *Involuntary Movements in a Sample of Chronic Mental Patients and Their Relation to the Treatment with Neuroleptics*, 3 INT'L J. NEUROPSYCHIATRY 286 (1968).

50. GELMAN, *supra* note 11, at 81 (quoting Nathan S. Kline, *On the Rarity of 'Irreversible' Oral Dyskinesias Following Phenothiazines*, 124 AM. J. PSYCHIATRY 48, 51 (1968)).

51. *Id.*

52. *See id.* at 96-108.

53. *Id.* at 88.

54. *Id.* at 81 (quoting Kline, *supra* note 50, at 51).

55. GELMAN, *supra* note 11, at 96-98.

56. *Id.* at 103.

57. *Id.* at 106-07.

change caused some high-profile lawsuits, in at least one of which a federal judge castigated psychiatrists for refusing to acknowledge the disorder.[58] In litigation, the standard psychiatric tactic of ignoring tardive dyskinesia had proved disastrous; it seemed perverse when psychiatrists failed to notice what was apparent to everyone else in the courtroom. The litigation, in turn, was based on the increasing number of research reports about the disorder.[59] At this time, although many research estimates of prevalence remained in the range of ten to twenty percent, a few psychiatrists offered much higher figures.[60] Cole himself did a sudden about-face in the late 1970s, at one point urging that half of patients should be withdrawn from medications because of the risk of tardive dyskinesia and because of the lack of benefits from drugs.[61]

Throughout the 1980s and early 1990s, research estimates of tardive dyskinesia's prevalence rose steadily. By 1993, the authors of the Yale Tardive Dyskinesia Study reported that "[a]bout two of every three patients maintained on neuroleptic treatment can be expected to develop persistent TD [tardive dyskinesia] within 25 years of continued exposure."[62] The authors also explained why published prevalence estimates were lower, even in the 1990s, and many of these reasons were hardly flattering to psychiatry.[63] For one, researchers "often classify mild cases as noncases."[64] For another, "TD cases may be less likely than noncases to be selected [by researchers] for prevalence studies."[65] Moreover, "the proportion of patients in most cross-sectional studies with more than 10 years of exposure [to medications] is small, possibly because [such] . . . patients [are] less likely than patients with less exposure to be selected for these studies."[66] For those reasons, "prevalence findings may indicate very little about the occurrence . . . of TD in specific populations at risk."[67] It appeared that, even after the almost two decade delay in recognizing tardive dyskinesia, many researchers still took pains to produce misleadingly low estimates of its extent.

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58. Rennie v. Klein, 476 F. Supp. 1294, 1300 (D. N.J. 1979), *modified and remanded*, 653 F.2d 836 (3d Cir. 1981), *remanded*, 458 U.S. 1119 (1982); Rogers v. Okin, 478 F. Supp. 1342, 1353 (D. Mass. 1979). I represented the *Rennie* plaintiffs during the trial and appeals.

59. Sheldon Gelman, *Mental Hospital Drugging-Atomistic and Structural Remedies*, 32 CLEV. ST. L. REV. 221, 230 (1983-84).

60. GELMAN, *supra* note 11, at 97.

61. *Id.* at 109 (citing George Gardos & Jonathan O. Cole, *Maintenance Antipsychotic Therapy: Is the Cure Worse Than the Disease?*, 133 AM. J. PSYCHIATRY 32, 36 (1976)).

62. William M. Glazer et al., *Predicting the Long-Term Risk of Tardive Dyskinesia in Outpatients Maintained on Neuroleptic Medications*, 54 J. CLINICAL PSYCHIATRY 133, 137 (1993).

63. *Id.* at 138.

64. *Id.*

65. *Id.*

66. *Id.*

67. Glazer, *supra* note 62, at 138.

The clinical picture was worse, if anything. Clinicians continued to ignore tardive dyskinesia even after researchers had recognized it. In 1978, a lawsuit examined prescribing practices in the state hospital system of New Jersey and, as of that date, no patient in the entire four hospital system— containing, at any one time, over five thousand patients—had ever been acknowledged to have tardive dyskinesia.[68] Yet George Crane, who testified in the case as an expert, had found over a hundred tardive dyskinesia cases, including severe ones, during a two-day visit to some wards.[69] Crane also testified that the failure to acknowledge tardive dyskinesia was typical of psychiatric practice nationally.[70] Again, in 1980, a "task force" of the American Psychiatric Association published a major report about tardive dyskinesia, making recommendations about drug treatment and dosages.[71] Four years later, two researchers reported that the Task Force's guidelines were "honored more in the breach than the keeping" and that researchers' recommendations "seem to have had little, if any, effect on actual physician behavior."[72] To the same effect, researchers reported in 1991 that eighteen percent of chronic patients in a California state facility suffered from tardive dyskinesia, and all of them—without exception—received medications continuously.[73]

Clinicians slighted other side effects too. Hospital charts might record that a patient paced back and forth or suffered from stiffness, and doctors often administered other medications in hopes of ameliorating these effects. Whether the side effect abated or not, however, it was very rarely thought to preclude the use of medication.

During the late 1970s and early 1980s as some researchers studied tardive dyskinesia, others turned their attention to the subjective side effects of medications.[74] They found that patients frequently experienced unpleasant reactions and intensely disliked them, while clinicians generally ignored patients' distress.[75] Researchers also found that patients who experienced distress generally did poorly on drugs and suggested withdrawing medications

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68. For discussion of Crane's findings in the *Rennie* case, including citations to the trial record, see Gelman, *supra* note 59, at 231-34.

69. *Id.* at 233.

70. GELMAN, *supra* note 11, at 136.

71. AM. PSYCHIATRIC ASS'N, TARDIVE DYSKINESIA: REPORT OF THE AMERICAN PSYCHIATRIC ASSOCIATION TASK FORCE ON LATE NEUROLOGICAL EFFECTS OF ANTIPSYCHOTIC DRUGS (1980).

72. Gualtieri & Sprague, *supra* note 44, at 347.

73. John Sramek et al., *Prevalence of Tardive Dyskinesia Among Three Ethnic Groups of Chronic Psychiatric Patients*, 42 HOSP. & COMMUNITY PSYCHIATRY 590, 591 (1991).

74. Theodore Van Putten, *Why Do Schizophrenic Patients Refuse to Take Their Drugs*, 31 ARCHIVES GEN. PSYCHIATRY 67 (1974); *see generally* Philip R.A. May et al., *Predicting the Outcome of Antipsychotic Drug Treatment from Early Response*, 137 AM. J. PSYCHIATRY 1088 (1980); Van Putten & May, *supra* note 38, at 480.

75. Van Putten & May, *supra* note 38, at 1089.

(or at least drastically reducing the dose) in such cases.[76] Reviewing the question in 2002, David Healy called patient distress "the darkest side of antipsychotics,"[77] and observed,

> Senior figures in the field . . . readily agreed that akathisia [drug induced nervousness and pacing] and the dysphoria [unhappiness or despondency], which were part and parcel of the effects of neuroleptics on extrapyramidal systems, were a more frequently occurring and more subjectively distressing problem than tardive dyskinesia . . . . For many there was little doubt that akathisia led to a toll of suicides and violence.[78]

Healy added that "the megadose regimes of . . . drugs used in the 1970s and 1980s minimized the problem because in high doses these neuroleptics degraded the capacity to act on any of the murderous or suicidal impulses that [medication induced] akathisia can give rise to."[79]

In fact, clinicians prescribe medication continuously to nearly everyone who has suffered an acute schizophrenic episode—even though research shows that many do not benefit. In 1976, Jonathon Cole concluded that "perhaps as many as 50% of [outpatients] might not be worse off if their medications were withdrawn."[80] Using the figures cited earlier,[81] if twenty percent of patients will not relapse off medication, and thirty percent will relapse despite medication, the fifty percent figure seems justified.

Clinicians also use grossly excessive doses. George Crane had urged substantial dose reductions in the early 1970s.[82] And as concern over tardive dyskinesia increased, so did warnings by researchers about dosage. In the face of such cautions, however, clinicians *increased* drug doses. Comparing prescribing practices at a number of institutions in 1973—the date a prestigious professional group first warned clinicians about tardive dyskinesia—with prescribing practices at the same institutions in 1982, researchers reported that "the overall mean dose doubled at each center"[83]—a finding that "confirm[ed] the impression of many clinicians and investigators that higher doses of antipsychotics are routinely being used."[84] Another study, by Julie

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76. May et al., *supra* note 74, at 1089.

77. DAVID HEALY, THE CREATION OF PSYCHOPHARMACOLOGY 275 (2002).

78. *Id.*

79. *Id.*

80. Gardos & Cole, *supra* note 61, at 35-36.

81. *See supra* text accompanying note 61.

82. GELMAN, *supra* note 11, at 103. Crane's views are described in *id.* at 102-06.

83. Gerard T. Reardon et al., *Changing Patterns of Neuroleptic Dosage Over a Decade*, 146 AM. J. PSYCHIATRY 726, 727, 729 (1989).

84. *Id.* at 729.

Magno Zito, examined prescribing at a New York hospital in the 1980s.[85] Zito found patients receiving doses "clearly in excess of current guidelines"[86] and psychiatrists increasingly using medications whose side effects were less visible, but also more distressing to patients.[87] Summarizing matters in 1995, two researchers wrote that

> [y]ears of a narrow treatment focus on psychosis and rehospitalization (rather than broad psychopathologic and quality-of-life assessment), . . . and the false hope that increasing drug dose will increase efficacy have led to excessive medication for most patients despite adverse effects, high rates of noncompliance, and patient dissatisfaction.[88]

E. Atypical Antipsychotics

A newer class of medications, "atypicals," came into wide use during the 1990s. Clozapine, the first of these medications, had originally appeared in the 1960s.[89] It was recalled from the market, however, because some patients developed a serious blood disorder.[90] In the late 1980s, clozapine became generally available again. Within a few years other atypicals—which did not produce the blood disorder or, it seems, all of the same benefits—joined clozapine on the market.[91]

Compared to standard medications, atypicals affect a different profile of brain neurotransmitters and, in turn, produce a different profile of side effects. Stiffness, tremor, and movement disorders appear less frequently than with standard drugs.[92] Evidence suggests that tardive dyskinesia occurs less frequently too.[93] At the same time, currently available atypicals are more likely to produce weight gain, seizures, and life-threatening heart disorders than standard drugs.[94] Despite the seriousness of these effects, they are

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85. Julie Magno Zito et al., *Pharmaco-Epidemiology in 136 Hospitalized Schizophrenic Patients*, 144 Am. J. PSYCHIATRY 778 (1987).

86. *Id.* at 782.

87. *Id.* at 781.

88. William T. Carpenter Jr. & Carol A. Tamminga, *Why Neuroleptic Withdrawal in Schizophrenia?*, 52 ARCHIVES GEN. PSYCHIATRY 192, 192 (1995).

89. HEALY, *supra* note 77, at 243-44, 251-64.

90. *Id.* at 239-41, 242.

91. *Id.* at 251-64.

92. GELMAN, *supra* note 11, at 205-06.

93. GELMAN, *supra* note 11, at 206; Daniel E. Casey, *Tardive Dyskinesia and Atypical Antipsychotic Drugs*, 35 SCHIZOPHRENIA RES. S61, S65 (1999).

94. Ross J. Baldessarini & Frances R. Frankenburg, *Clozapine: A Novel Antipsychotic Agent*, 324 NEW ENG. J. MED. 746, 751 (tbl. 3) (1991); Brian C. Lund et al., *Clozapine Use in Patients with Schizophrenia and the Risk of Diabetes, Hyperlipidemia, and Hypertension: A Claims-Based Approach*, 58 ARCHIVES GEN. PSYCHIATRY 1172, 1174 (2001) (describing weight gain and higher risks of diabetes in

supposed to occur rarely enough for many psychiatrists to prefer atypicals.[95] Many patients prefer them as well. Treatment with atypicals costs many times more than treatment with older, standard agents, however. In fact, atypicals appeared at a time when the patients on some older antipsychotics were expiring.[96]

The side effects of atypicals are becoming a more serious concern. In 2001, FDA physicians warned of an almost one hundred fold increase in certain serious heart disorders when patients take clozapine.[97] Other atypicals may, or may not, resemble clozapine in this way. By the same token, they may cause more tardive dyskinesia and other side effects than clozapine.

Psychiatrists have claimed two advantages for atypicals, in addition to a more benign profile of side effects. First, some patients who respond poorly to standard drugs improve dramatically on atypicals. Second, atypicals have been said to produce improvements in the negative symptoms of schizophrenia —social withdrawal for example.[98]

In larger studies, however, the newer drugs do not appear more effective than older drugs overall—even if some treatment refectory patients do respond well to clozapine.[99] Moreover, claims for atypicals' effectiveness against negative symptoms have become increasingly suspect.[100] Indeed, David Healy and others have argued that claims for the superiority of atypicals on this score generally result from comparing low doses of the newer drugs to excessively high—and more harmful—doses of standard medications.[101]

A 1988 study supposedly showed clozapine superior to a standard drug in treatment refractory patients;[102] that result led to clozapine's return to the

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younger patients). Authorities on the question of heart disorder are cited *infra* note 97.

95. *See* GELMAN, *supra* note 11, at 29.

96. *See id.* at 205.

97. The warning by FDA physicians appears in Lois La Grenade et al., Letter: *Myocarditis and Cardiomyopathy Associated with Clozapine Use in the United States*, 345 NEW ENG. J. MED. 224 (2001) (reporting that patients on clozapine suffered two serious heart complications at eighty times the rate of the general population). On the side effect profiles of clozapine and the "atypical" drugs, see HEALY, *supra* note 77, at 263 (noting that atypicals, unlike clozapine, appear capable of inducing tardive dyskinesia in tests); Andrew Herxheimer & David Healy, *Arrhythmias and Sudden Death in Patients Taking Antipsychotic Drugs: High Doses and Combinations of Certain Drugs are Best Avoided*, 325 BRIT. MED. J. 1253 (2002) (noting that at least one atypical drug, as well as some standard antipsychotic drugs, produced two to five times the rate of cardiac arrests and ventricular arrhythmias—and patients deaths—as clozapine); Carol E. Koro et al., *An Assessment of the Independent Effects of Olanzapine and Risperidone Exposure on the Risk of Hyperlipidemia in Schizophrenic Patients*, 59 ARCHIVES GEN. PSYCHIATRY 1021, 1024 (2002) (reporting that one of two atypicals tested produced significantly higher rates of hyperlipidemia).

98. For discussion, *see* GELMAN, *supra* note 11, at 207.

99. *See* Baldessarini & Frankenburg, *supra* note 94, at 751.

100. *See id.* at 752.

101. HEALY, *supra* note 77, at 270 (comparing clozapine with chlorpromazine).

102. John Kane et al., *Clozapine for the Treatment-Resistant Schizophrenic: A Double-Blind*

marketplace. However, this study had compared "moderate doses" of clozapine to high doses of the standard medication.[103] Commenting three years later, Ross J. Baldessarini argued that some of the apparent benefits from clozapine may have resulted from relief from the side effects caused by the standard drug.[104] In his recently published account of the medication era, David Healy argues that

> there was no excuse for the clinical trials of the other atypicals, which came after the work of Baldessarini . . . . The trials with olanzapine, quetiapine, and risperidone compared these new compounds to haloperidol [an older, standard drug] in doses approaching 20 milligrams per day [a relatively high dose]. Even so, they were not obviously more effective than haloperidol, except for their marginal benefits on negative symptoms.[105]

In Healy's view, the rapid rise of atypicals has resulted from "wishful thinking and aggressive marketing" by drug companies.[106] In fact, according to Healy "the changes that patients and clinicians were witnessing"[107] in patients on atypicals,

> were often the consequences of changing from the equivalent of more than 5,000 milligrams of chlorpromazine per day to a dose of clozapine or a newer [atypical] agent equivalent to 300 milligrams of chlorpromazine per day. There were obvious difficulties for clinicians in accepting that even part of the benefits they were witnessing with the new drugs might stem from the fact that they were not now poisoning their patients to the same extent as previously . . . . As patients recovered from drug-induced negativity, the resulting benefits in turn seemed to validate the concept that atypicals had unique effects on negative states.[108]

Finally, Healy notes that "[b]y the year 2000 a number of academic centers were running studies randomizing patients who had not responded to clozapine or other 'atypicals' to adjunctive treatment with standard neuroleptics."[109] These investigations, which "reverse[d]" the 1988 study that had made

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Comparison with Chlorpromazine, 45 ARCHIVES GEN. PSYCHIATRY 789, 796 (1988).

103. Baldessarini & Frankenburg, *supra* note 94, at 750.

104. *Id.*

105. HEALY, *supra* note 77, at 269.

106. *Id.*

107. *Id.* at 270.

108. *Id.*

109. *Id.* at 274.

clozapine as the drug of choice for treating refractory patients,[110] prompted Healy to suggest a counterfactual: "had haloperidol been withdrawn because of [a serious blood disorder, as clozapine was withdrawn], . . . and [had] clozapine-like compounds been left to dominate the marketplace, haloperidol might later have been rediscovered as a drug that, given in astonishingly low doses, could produce remarkable results in managing treatment-resistant schizophrenia."[111]

With regard to both standard and atypical medication, Healy suggests that the customary methods of drug evaluation, testing and marketing—combined with the approach to disease classification in recent editions of the *American Psychiatric Association Diagnostic and Statistical Manual*—have led psychiatrists to judge the effectiveness of medications in terms of standardized symptom checklists, rather than their overall effect on patients. Since the 1960s, Healy believes psychiatrists' thinking has been influenced significantly by the marketing efforts of large pharmaceutical companies—a criticism echoing George Crane's thirty years before.

F. Public Mental Health and Deinstitutionalization

By the late 1970s, a new public health system had emerged.[112] No longer were patients confined in long-term state hospitals as a matter of course. Instead, people with schizophrenia received antipsychotic medications in the "community."[113] They returned to hospitals only for relatively brief stays and only so that psychiatrists could adjust—or reinstitute—a regimen of medication.[114] This new system, known as "deinstitutionalization,"[115] commenced in earnest in the late 1960s—after medications had been available for a decade and a half.

Yet not all deinstitutionalized patients would take—or respond favorably to—medications. These patients lived and died in a deteriorated state outside of the hospitals.[116] Some became homeless, with increasing numbers hallucinating on the streets of major American cities.[117] Others lived under grossly substandard conditions in boarding or nursing homes.[118]

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110. HEALY, *supra* note 77, at 274.

111. *Id.*

112. On deinstitutionalization and the new system of public mental health, see GELMAN, *supra* note 11, at 38-43, 166-67.

113. *Id.* at 139.

114. *Id.*

115. *Id.* at 5.

116. *Id.* at 167.

117. GELMAN, *supra* note 11, at 167.

118. *See* RAEL JEAN ISAAC & VIRGINIA C. ARMAT, MADNESS IN THE STREETS: HOW PSYCHIATRY AND THE LAW ABANDONED THE MENTALLY ILL 101 (1990).

Today, the system of community medication has become more firmly entrenched. State hospitals, and the amount of time patients stay in them, have continued to shrink. Homelessness has increased, along with boarding and nursing home scandals.[119] Some states have taken small steps toward increasing the number of available hospital beds, but the basic tendencies in public mental health remain unchanged.

G. Litigation

As deinstitutionalization got underway in the late 1960s, state hospitals suddenly appeared on the radar screens of courts. Spurred by public minded lawyers citing constitutional law, numerous courts ordered enhanced hearings and stricter legal standards for hospital commitment.[120] Lower courts also proclaimed a constitutional right to treatment and, by way of enforcing the right, ordered substantial improvements to the public hospitals' physical plant, operations, and staffing.[121] Moreover, starting in 1978—about a decade after the initial decisions on commitment law—some courts recognized a limited (and in my opinion, usually ineffective) right to refuse medications.[122] With these last cases, the revolution in constitutional law collided head-on with the revolutions in clinical psychopharmacology and public mental health. Many patients wanted to refuse drugs, but the new public health system could hardly survive if they did.

The conflict did not last long. In a series of decisions handed down between 1979 and 1990, the U.S. Supreme Court brought the legal revolution to a halt.[123] While evading some basic questions,[124] these decisions clearly signaled that lower courts had gone too far. In general, the Court required

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119. *See id.* at 4, 327.

120. *E.g.*, Lessard v. Schmidt, 349 F. Supp. 1078 (E.D. Wis. 1972), *remanded* 414 U.S. 473 (1974). For discussion of litigation developments generally, see GELMAN, *supra* note 11, at 121-40, 167-77.

121. The leading decision was Wyatt v. Stickney, 344 F. Supp. 373, 378-79 (M.D. Ala. 1972).

122. Rennie v. Klein, 476 F. Supp. 1294, 1307 (D. N.J. 1979); Rogers v. Okin, 478 F. Supp. 1342 (D. Mass. 1979), *aff'd in part, rev'd in part, and remanded*, 634 F.2d 650 (1st Cir. 1980), *vacated and remanded by* Mills v. Rogers, 457 U.S. 291 (1982), *and overruled as explained in* Cuesnongle v. Ramos, 835 F.2d 1486, 1500 n.12 (1st Cir. 1987).

123. Washington v. Harper, 494 U.S. 210, 222, 227 (1990) (allowing prisons to forcibly medicate even competent inmates provided that a panel that included a warden and prison doctors approved); Youngberg v. Romeo, 457 U.S. 307, 321-23 (1982) (mandating deference to psychiatrists' "professional judgment" in hospital decision-making and declining to recognize a right to treatment independent of the right to physical liberty); Parham v. J.R., 442 U.S. 584, 604-05 (1979) (no prior hearing required when minors are committed to a state institution).

124. In particular, the Court has never formally decided on the procedures required when adults are committed to mental hospitals, or when children remain in the hospital after they have been committed, nor has the Court decided whether a free standing right to treatment exists, apart from patients' right to liberty.

deference to the professional judgment of psychiatrists.[125] The culmination of this line of cases came in 1990, when the U.S. Supreme Court upheld the practice of forcibly medicating state prison inmates—and, by implication, patients in state hospitals—whenever a warden and prison doctor believed that medication would serve a vaguely defined "medical interest" of a prisoner and more importantly, the needs of the institution.[126]

III. ADDRESSING THE PAST

When psychiatrists present the face of their profession to the world—or when they look at their profession as if in a mirror—the image certainly does not include neurologically damaged patients, out-of-control clinical practices, or researchers inventing imaginary treatment benefits. Thus, a detailed history of the field can mar psychiatry's picture of itself. For that reason, such histories often strike psychiatrists as calculated insults.

The profession's interest in its past goes beyond professional pride. The discipline's success greatly depends on how non-psychiatrists perceive it. Insurers may fail to cover psychiatric services. Legislators may impose burdensome, demeaning restrictions or restrict research funding. Judges can award injunctions and damages. Other physicians can demean psychiatry. Medical students can shun it. Patients may stay away. For these reasons, psychiatry is unusually sensitive about its image. Insecurity is an occupational hazard of psychiatrists.

Psychiatry is rightly concerned about history. Just as past acts influence our perception of the character of an individual, history influences our perception of institutions, including professions. Based on past behavior, we make judgments about an individual's competence and character. Based on an institution's history, we do much the same thing.

Of course, it rarely makes sense to blame a profession, such as psychiatry, for the past acts of its practitioners. But even if moral blame is inappropriate, and even if no real entity—e.g., psychiatry—exists to receive such blame, history still may tell us about the present capacities of an institution, discipline, or profession. If someone says they blame psychiatry for some past episode—lobotomy perhaps, or the removal of women's ovaries in the late nineteenth century—they are probably speaking figuratively. They mean we have reason to distrust psychiatrists' actions now, not that some entity called psychiatry exists that warrants punishment for something in the past. When psychiatrists cited their profession's achievements in neuroscience during the 1990s, they typically did so as part of an implicit argument that psychiatry *today* is a

125. *Romeo*, 457 U.S. at 323.
126. *Harper*, 494 U.S. at 225.

competent and thriving discipline. It is no different, however, if someone draws negative inferences from past lapses by psychiatrists, instead of positive inferences from past triumphs. Thus, questions about psychiatry's past tend to become questions about its present, heightening historical controversy.[127]

Not surprisingly then, some psychiatrists have produced histories of the medication era—and in particular, of the profession's response to tardive dyskinesia—that attempt to explain away psychiatry's failures. According to one such account, a general historical law—the so-called law of the new drug—explained what happened.[128] According to this "law," physicians in every medical specialty receive new therapies over-enthusiastically when they first appear.[129] There follows a stage of overreaction, during which physicians become too critical of the formerly new therapy.[130] Finally, a state of balanced judgment arrives, when physicians gain a realistic understanding of the treatment.[131]

As applied to tardive dyskinesia, the law of the new drug supposedly accounted for the psychiatric defaults of the 1960s and 1970s. Enthusiastic about a new treatment, psychiatrists predictably and understandably had overlooked a side effect. If the law of the new drug was correct, cardiologists and urologists—indeed, all physicians—would have acted in the same way. Taken literally, the theory meant that psychiatry's failure to recognize tardive dyskinesia actually *strengthened* its claim to be an integral part of modern medicine, since psychiatrists had acted in typical medical fashion.

The law of the new drug is specious applied to tardive dyskinesia, as I have shown elsewhere.[132] It conflicts with the facts for one thing. Contrary to what the theory predicts, psychiatrists in the 1950s were more ready to recognize serious side effects—including persistent effects and including dyskinesias—than psychiatrists became in the '60s or '70s. Thus, "over-enthusiasm" appeared not at the outset of the drug era, as the theory supposes, but closer to the middle. Moreover, proponents of the theory assumed that prevalence estimates in the thirty to fifty percent range had to be excessive since these estimates appeared during the second period of the drug era when

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127. When psychiatrists make predictions about someone's dangerousness, they are doing much the same thing—at least if their prediction relies on the person's past acts. Moreover, as is true of judgments about institutions, predictions of dangerousness do not carry moral opprobrium.

128. *See* Daniel E. Casey & George Gardos, *Introduction* to TARDIVE DYSKINESIA AND NEUROLEPTICS: FROM DOGMA TO REASON ix (Daniel E. Casey & George Gardos eds., 1986) [hereinafter TARDIVE DYSKINESIA AND NEUROLEPTICS]. For further discussion of the law of the new drug, see GELMAN, *supra* note 11, at 197.

129. GELMAN, *supra* note 11, at 197.

130. *Id.*

131. *Id.*

132. *Id.*

physicians were supposedly overly critical of a treatment.[133] Yet the higher estimates were essentially correct. In fact, proponents assumed that the "second period" of overreaction was underway only because they wanted to declare those higher estimates wrong. These psychiatrists had reached conclusions about the extent of an observable neurological disorder by consulting historical laws, rather than medical research or their own eyes. Self-interested professional history had replaced serious medical judgment.[134]

Another flawed attempt at history came from Paul Appelbaum, a psychiatrist who has specialized in legal topics. Appelbaum starts with the assumption that medications are indicated for every seriously mentally ill person. It follows that a legal right to refuse treatment must lead to medical error and, for patients, personal calamity. In the late 1970s, when Appelbaum believed that the law might recognize a substantial right to refuse medication, he angrily denounced it as the "right to rot."[135] In 1994, after it had become clear that the law would not mandate significant changes in medication practice, Appelbaum wrote a book analyzing the previous decades' developments in law and psychiatry.[136] Here, he expressed satisfaction that relatively few patients had exercised a right to refuse, noting that things had not turned out as badly as he had feared.[137] Yet Appelbaum's overriding and unchanging assumption—that every patient should receive medication—is simply false on purely medical grounds, and it undermines his various accounts of the era.

Assuming that every patient should receive medications, Appelbaum still might have discussed side effects in a serious way. He did not do so, however. Indeed, tardive dyskinesia earned only two entries in the index to Appelbaum's book.[138] The first reference occurs as Appelbaum is explaining why a lawyer, who had filed a right to refuse treatment case, thought patients should enjoy that right.[139] Appelbaum does not explain the lawyer's concern about tardive dyskinesia or comment on it; he simply mentions the side effect in passing. The second reference, also in passing, occurs as Appelbaum describes a trial of that right to refuse case. "Detailed descriptions[,]"[140] Appelbaum writes,

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133. This mistake appears in Thomas E. Hansen et al., *Is There an Epidemic of Tardive Dyskinesia?*, *in* TARDIVE DYSKINESIA AND NEUROLEPTICS, *supra* note 128, at 1, 6.

134. GELMAN, *supra* note 11, at 202.

135. Paul S. Appelbaum & Thomas Gutheil, *"Rotting with Their Rights on": Constitutional Theory and Clinical Reality in Drug Refusal by Psychiatric In-Patients*, 7 BULL. AM. PSYCHIATRY & L. 306 (1979); Paul S. Appelbaum & Thomas Gutheil, *The Boston State Hospital Case: "Involuntary Mind Control"; the Constitution, and the "Right to Rot"*, 17 AM. J. PSYCHIATRY 720 (1980).

136. PAUL S. APPELBAUM, ALMOST A REVOLUTION: MENTAL HEALTH LAW AND THE LIMITS OF CHANGE (1994).

137. *See id.* at 134.

138. *Id.* at 233.

139. *Id.* at 116.

140. *Id.* at 123.

"were offered [at the trial] of many of the side-effects of antipsychotic medications, especially tardive dyskinesia."[141] Judging from these references, tardive dyskinesia seems significant to Appelbaum only in relation to lawyers, not patients—and not very significant, even at that. Appelbaum adds that the "core" of the Boston court's eventual opinion focused on "legal arguments"—as if tardive dyskinesia was an irrelevance.[142]

Although Appelbaum does not mention it, the Boston case was preceded by a no less publicized ruling from a New Jersey federal court.[143] The New Jersey decision found that the state hospitals had ignored tardive dyskinesia and had systematically refused to diagnose it.[144] Unlike the Boston case, which relied on medical literature, the New Jersey litigation produced proof of numerous patients with tardive dyskinesia and other side effects.[145] Had Appelbaum described this case, a question might have arisen about physicians' medical judgment. He avoided those questions by ignoring the case. The "law of the new drug" theorists had at least acknowledged that psychiatrists overlooked tardive dyskinesia; Appelbaum does not concede even that.

Accounts like Appelbaum's are remarkable. Nor does the fact that he was analyzing legal developments explain his neglect of tardive dyskinesia. Just the opposite is true. Consider an alternate history of medication refusal litigation, a history like Appelbaum's, but written from the other side. In this alternate account, the benefits of medications would receive only two passing mentions. The first would occur in connection with a particular lawyer's decision to represent a psychiatrist whose drug prescribing had caused a severe case of tardive dyskinesia. The second reference would note that some testimony was given about medication's benefits in that case, but the testimony had little to do with the "core" of the court's eventual ruling. Such a history seems unlikely to be written, nor should it be. Yet accounts like Appelbaum's slight side effects occur in just the same way and are considered mainstream.[146]

Could a professional historian do better than Paul Appelbaum or the law of the new drug theorists? Edward Shorter, a professional historian, published a full length history of psychiatry in 1997 entitled *A History of Psychiatry:*

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141. APPELBAUM, *supra* note 136, at 123.

142. *Id.*

143. The Boston case was Rogers v. Okin, 478 F. Supp. 1342 (D. Mass. 1979); the New Jersey case, Rennie v. Klein, 476 F. Supp. 1294 (D. N.J. 1979).

144. *See Rennie*, 476 F. Supp. at 1300, 1302.

145. *E.g.*, *id.* at 1302.

146. Histories driven by assumptions—even rigid assumptions—are not always unhelpful. Appelbaum, for example, offers useful information about right to refuse treatment decisions and their implementation. APPELBAUM, *supra* note 136, at 123.

From the Era of the Asylum to the Age of Prozac.[147] To that history, we now turn.

IV. EDWARD SHORTER'S *HISTORY OF PSYCHIATRY*

According to Shorter, a contest between two ideas defines the history of psychiatry.[148] One is the idea of mental illness as a biological disease.[149] The other is the idea that mental illness is not a biological disease but something else, such as a problem resolvable by psychoanalysis.[150] In the late twentieth century, Shorter argues, the biological idea decisively—and rightly—prevailed.[151]

A. The Three Stages of Psychiatric History

Shorter divides the history of psychiatry into a prelude and three stages. During the prelude, in the early nineteenth century, psychiatry was "born" as a discipline and the first asylums appeared. Stage number one, which Shorter calls "the first biological psychiatry," emerged during the latter part of the nineteenth century.[152] It represented "a movement of ideas rather than an exercise in bricks and mortar"[153]—a movement of scientifically correct ideas as psychiatrists came to understand that "major mental illnesses have a heavy biological and genetic component."[154] This period was supplanted by stage two, "the Psychoanalytic Hiatus," during the early twentieth century.[155] Shorter thinks of this period as a dark age, a time when psychoanalysis obscured the truths of the first biological psychiatry. History righted itself, however, during stage three—the "second biological psychiatry"—which emerged by 1970.[156] At that time, "the progress of science within psychiatry"[157] and the success of medications had relegated psychoanalysis to the margins and restored the "biological" paradigm to professional prominence.[158]

It is noteworthy that when he constructs his stages of history, Shorter gives a decisive role to an extremely general medical belief—a belief in the biological basis of mental illness—rather than to treatments, treatment

147. SHORTER, *supra* note 2.
148. *Id.* at 69.
149. *Id.*
150. *Id.* at 145 ("Psychoanalytic Hiatus").
151. *Id.* (describing psychoanalysis as "an interruption").
152. SHORTER, *supra* note 2, at 69.
153. *Id.*
154. *Id.* at 93.
155. *Id.* at 145 (title of chapter five).
156. *Id.* at 239.
157. SHORTER, *supra* note 2, at 145.
158. *Id.*

outcomes, or the experiences of patients. So long as psychiatrists believed in the biological basis of mental illness, the field was on the right track in Shorter's view. It does not matter whether the reigning biological ideas were scientifically sound—something they were unlikely to be before the mid-to-late twentieth century when psychiatrists learned more about brain neurotransmitters. No matter how crude a nineteenth century theory, if it presumed a biological basis for the patient's disorder, it belonged to the first biological psychiatry.

B. Treatments Before Medications

Major biological treatments—insulin coma therapy, electroconvulsive therapy, and lobotomy—date from the 1930s and appeared during the so-called psychoanalytic hiatus, not during the first or second biological psychiatry. For that matter, medications appeared during the psychoanalytic period too. In fact, Shorter portrays medications as one of the forces that undermined psychoanalysis. Thus, during the supposedly psychoanalytic period, major biological therapies thrived.

Shorter handles the mismatch between treatment and historical stages by using a literary device. Rather than examine each treatment in the context of the historical stage when it appeared, he devotes a separate chapter, called "Alternatives,"[159] to all of the biological treatments that preceded medications. "In the first half of the twentieth century," Shorter writes, "[p]sychiatrists could warehouse their patients in vast bins . . . . [Or they could employ] psychoanalysis, a therapy suitable for the needs of wealthy people desiring self-insight, but not for real psychiatric illnesses."[160] "Caught between these unappealing choices," Shorter observes, "psychiatrists sought alternatives"[161] —and these alternatives took the form of biological treatments. Psychiatrists discovered these therapies "serendipitously" as Shorter himself points out;[162] they were not based on any knowledge of how the brain operated.

Discussing the detriments of these procedures, Shorter offers only the briefest, capsule accounts. Comparing one early sedative to another, for example, he notes that it was superior by virtue of "not tasting unpleasant, by having few side effects, and by acting at therapeutic levels far beneath the toxic dose."[163] Sleep therapy, he reports, produced mortality rates of five

159. *Id.* at 190.

160. *Id.*

161. *Id.*

162. SHORTER, *supra* note 2, at 198 (describing the process of "drug discovery largely by serendipity" that held sway until "the designed drug discovery of the second biological psychiatry").

163. *Id.* at 202 (comparing barbital to potassium bromide).

percent as patients developed pneumonia or cardiac complications.[164] Insulin coma therapy was "a dangerous procedure with a mortality [rate] of almost one in a hundred."[165] Describing camphor, a drug that induced convulsions, Shorter remarks that "patients hated the feeling of anxiety preceding the fit, the vomiting that camphor caused, and the pain in the muscles where it was injected."[166] Discussing another convulsive drug, cardiazol, Shorter quotes a psychiatrist who abandoned the treatment because of the "'agonizing fears of dying and crumbling away'"[167] that it produced in patients. This psychiatrist had always "sought to get away from the room"[168] where the treatment was administered because "'[t]he sight of the artificially produced attack of epilepsy, especially of the contorted blue faces, was so awful.'"[169]

Shorter's discussion of the effectiveness of these procedures is even more brief and rather remarkable. Repeatedly, Shorter says that these treatments "appeared" or "seemed" to be effective, and he praises them for their effect on *psychiatrists'* state of mind and for anticipating later psychiatric therapies. Shorter appears more interested in how various treatments affected psychiatrists and psychiatry than in how patients fared.

Of sleep therapy, for example, Shorter writes that it "seemed to be a promising new therapy,"[170] and

> [f]or the first time in the history of psychiatry, a drug therapy had been described that seemed to alleviate major psychiatric illness with a physical procedure. Whether . . . bromide sleep really did cure patients is beside the point: A hint had been inserted into the profession's collective thinking that some kind of cure with drugs might be possible.[171]

Shorter's assessment of other biological therapies follows the same general lines, sometimes in almost the same words. Insulin coma treatment, he writes, "seemed to be a procedure that actually worked, at least for the short term, without the extreme dangerousness of sleep therapy."[172] Later, Ladislas von Meduna pioneered the practice of inducing epileptic fits in psychiatric patients.

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164. *Id.* at 205.

165. *Id.* at 212.

166. *Id.* at 216.

167. SHORTER, *supra* note 2, at 216 (quoting Max Müller, ERINNERUNGEN: ERLEBLE PSYCHIATRIEG ESCHICHTE 244 (1982)).

168. *Id.*

169. *Id.*

170. *Id.* at 205 (describing a form of sleep therapy used in 1919 and 1920).

171. *Id.* at 202.

172. SHORTER, *supra* note 2, at 214.

Did patients improve? According to Shorter, "[t]he answer seemed to be yes."[173]

Yet "seeming to work" apparently differs from actually working. When Shorter describes medications, he writes that they, "truly worked,"[174] "did work,"[175] or represent "[t]he first drug that worked."[176] At another point, Shorter manages to create the same ambiguity about whether treatments "worked" using different phrasing. "Barbiturate narcosis, insulin coma, and Metrazol convulsion,"[177] he writes, "extend[ed] the promise of lasting remissions and even cures."[178] Was the "promise" of "lasting remissions" actually fulfilled? Shorter does not say.

The sources that Shorter cites in connection with insulin coma therapy shed additional light on how therapies "seem to work." After noting that insulin treatment "seemed . . . to work,"[179] Shorter added something more substantial: "In the long term, it was discovered that insulin coma had about the same success rate as barbiturate-sleep therapy. Both represented a substantial improvement on what was available before, which is to say, nothing."[180] A single footnote, referring to two sources, appears after this passage. The first source, a 1957 article by Brian Ackner, Shorter cites in support of his claim that "barbiturate narcosis" and insulin coma had comparable success rates.[181] The second source is a 1987 article about insulin treatment by W.A. Cramond that, according to Shorter, provides a "not unfavorable historical evaluation of insulin coma therapy."[182]

Shorter cites the Ackner and Cramond articles to support his claim that insulin coma therapy "seemed" to work, but both authors concluded that the coma produced no therapeutic benefit whatsoever.[183] They attributed the apparent success of insulin to the fact that staff, concerned about the danger of fatalities, paid especially close attention to patients who had undergone it.[184] Ackner's article, a classic, describes an experiment in which some patients received standard insulin coma therapy and other patients were put into a deep

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173. *Id.* at 215.

174. *Id.* at 238.

175. *Id.* at 268.

176. *Id.* at 246.

177. SHORTER, *supra* note 2, at 218.

178. *Id.*

179. *Id.* at 214.

180. *Id.* (internal footnote omitted).

181. Brian Ackner et al., *Insulin Treatment of Schizophrenia: A Controlled Study*, 2 LANCET 607, 607-11 (1957). For discussion of Ackner's article, see GELMAN, *supra* note 11, at 230-31.

182. SHORTER, *supra* note 2, at 386 n.93 (citing W.A. Cramond, *Lessons from the Insulin Story in Psychiatry*, 21 AUSTL. & N.Z. J. PSYCHIATRY 320, 320-26 (1987)).

183. GELMAN, *supra* note 11, at 231.

184. *Id.* at 230.

sleep designed to look like a coma.[185] The two groups of patients fared equally well after six months.[186] Although Ackner had used barbiturates to induce deep sleep, Shorter's reference to "barbiturate narcosis" treatment is misleading. In fact, Ackner used the barbiturate-induced sleep as a placebo, a device to keep patients and staff from learning who had received coma treatment and who had not.[187] The sleep was brief, and the placebo procedure had no connection to the "sleep therapy" that kept patients asleep for days at a time. Shorter says that insulin coma and "barbiturate narcosis" proved equally effective, but in light of Ackner's article, that means only that insulin coma treatment had proved no more effective than a placebo.

Ackner himself described the significance of his experiment as follows:

> The results . . . do not demonstrate that the coma regime has no therapeutic effect. During insulin therapy patients are subjected to powerful group influence and receive increased medical and nursing care in a special setting. Daily they are exposed as a group to the threat of being rendered unconscious. Daily they are brought back to consciousness by doctors and nurses on whom they become dependent. The new relationships so built up cannot be ignored, for it could be that the coma regime is helping to establish that lost capacity for relationship with others. But the results suggest that insulin is not the specific therapeutic agent of the coma regime as has so often been claimed.[188]

It appeared that, in Ackner's view, anything that "built up" relationships to the same extent would prove equally effective.

Writing thirty years later during the medication era, Cramond accepted Ackner's conclusion that staff attention—and not the physical intervention—had benefited insulin patients. Cramond wrote: "As I look with anxiety at my schizophrenic patients coping with the neurological deficits occasioned by neuroleptic drugs, I not infrequently regret the passing of insulin and wish we could use some other process to provide [the old effects]."[189] Cramond thereby suggested that a placebo effect—indeed, any effect that attracted staff attention —was preferable to medications.

Thus, Ackner and Cramond conclude that the insulin coma's sole benefit lay in its psychological and social effects, and that biologically, it did nothing. If anything, Ackner's account makes the procedure sound more like a

185. *Id.*; Ackner, *supra* note 181, at 607.

186. GELMAN, *supra* note 11, at 231; Ackner, *supra* note 181, at 611.

187. GELMAN, *supra* note 11, at 231; Ackner, *supra* note 181, at 609-10.

188. GELMAN, *supra* note 11, at 230-31 (quoting Ackner, *supra* note 181, at 611).

189. *Id.* at 233 (quoting Cramond, *supra* note 182, at 320).

psychoanalytic experience than a biological therapy, as patients "built up" relationships with staff.[190] Shorter, on the other hand, leaves the impression that there was good reason to think insulin coma effective and cites Cramond and Ackner—who regard the procedure as a placebo—in support of his position. His citation of Ackner and Cramond highlights the misleading ambiguity in Shorter's oft-repeated claim that older therapies "seemed" to work. Had Shorter said that the treatments *only* seemed to work—but actually did not—his book would read very differently.[191]

Moreover, if Ackner and Cramond are right, the entire premise of Shorter's "Alternatives" chapter is mistaken. Biological treatments did not offer the only hope available to psychiatrists before the drug era. Indeed, once properly understood, they offered no hope at all. The real hope for patients, according to Ackner and Cramond, lay in staff-patient relationships and other non-biological techniques.[192]

The effectiveness of two other treatments, electroconvulsive therapy (ECT) and lobotomy, receives somewhat more consideration from Shorter. He believes that the forces of antipsychiatry pilloried ECT unfairly and, for that reason, he wants to demonstrate the procedure's usefulness.[193] Lobotomy, on the other hand, crossed an ethical line in his view.[194] Thus, ECT and lobotomy both have contemporary relevance: alone, among all the pre-medication biological treatments, ECT remains in wide use while lobotomy is the treatment best remembered as a mistake.

To establish ECT's efficacy, Shorter describes one case, the first use of the procedure by its discoverer, Cerletti. "After eleven applications of ECT,"[195] Shorter writes, "the patient . . . did get well and was discharged from the [hospital]."[196] Cerletti reported that after a year, the patient remained "per-

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190. *See* Ackner, *supra* note 181, at 611.

191. Healy observes that insulin coma treatment had undoubted effects on the brain. HEALY, *supra* note 77, at 53-56. While acknowledging Ackner's study and also the likelihood that insulin coma affected staff patient relations for the better, Healy leaves open the possibility that some clinical benefits from the procedure resulted from its neurological effects. "[S]omething probably was going on in the [insulin] patients that called forth placebo responses in the staff . . . ." *Id.* at 54. Healy also suggests, as Shorter did, that "[p]erhaps both [barbiturate narcosis and insulin coma treatment] worked." *Id.* Thus, Healy—unlike Shorter—defends his conclusions about insulin coma treatment; he does not rely on ambiguities to make his point. At the same time, I do not believe that Healy gives Ackner's results their full due. If a brief sleep had the same therapeutic benefits as a coma, it seems extremely unlikely that the coma's effects on the brain—which, of course, are nothing like the effects of sleep—were producing the clinical benefits. For the rest, I do not believe that Healy improves on Ackner's analysis.

192. *See* GELMAN, *supra* note 11, at 231.

193. *See* SHORTER, *supra* note 2, at 221-24.

194. *Id.* at 229.

195. *Id.* at 221.

196. *Id.*

fectly well,"[197] even though the man's wife described episodes in which he heard voices.[198] Based only on this single case, Shorter concludes that "ECT was not a cure for schizophrenia. But it represented a great alleviation of the disabling symptoms of psychotic illness, and permitted individuals to function more or less normally."[199] In similar fashion, by describing the case of a single patient in 1977, Shorter illustrates the efficacy of ECT against depression.[200] Shorter also summarizes the 1990 judgment by an American Psychiatric Association task force that "'ECT is an effective treatment for . . . major depression,' for manic depressive illness[,] . . . mania, and psychotic schizophrenia."[201] Forgetting his own scheme of historical stages (ECT in fact dates from the psychoanalytic hiatus),[202] Shorter concludes that "[t]he ability of the new biological psychiatry to make individuals [like this one] better represents an accomplishment of historic dimensions."[203] If there was an "achievement of historic dimensions," as Shorter says, it occurred well before the second biological psychiatry began, and the achievement had no relationship to any deeper understanding of the brain. Nor does Shorter tell us enough about ECT or the vast literature about it to justify his claims about an historic feat. Older treatments, including simple hospitalization, have also proved effective against depression. ECT may be superior to these older methods or provide relief more quickly than they do, but Shorter does not even attempt to explain how.

If ECT survived the advent of medications because it worked so well, lobotomy did not survive, in Shorter's view, because it was unethical. "Although lobotomy did tend to tranquilize the raving patients who were management problems,"[204] Shorter writes, "it generally deprived them of their judgment and social skills."[205] According to a medical text quoted by Shorter, "'[i]t is probable that every individual after the operation is happier than before, but this may be brought at too great a cost, not only to himself but to society . . . .'"[206] Shorter himself reaches the conclusion that "[i]n retrospect, frontal lobotomy was indefensible for ethical reasons."[207] Lobotomy is, in fact,

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197. *Id.*

198. SHORTER, *supra* note 2, at 221.

199. *Id.*

200. *Id.* at 286-87. The patient was Norman Endler, a psychologist who had opposed the use of ECT until he himself underwent the procedure.

201. *Id.* at 285 (quoting AM. PSYCHIATRIC ASS'N, THE PRACTICE OF ELECTROCONVULSIVE THERAPY: RECOMMENDATIONS FOR TREATMENT, TRAINING AND PRIVILEGING: A TASK FORCE REPORT 7-8 (1990)).

202. ECT was over fifty years old at the time of the American Psychiatric Association report.

203. SHORTER, *supra* note 11, at 287.

204. *Id.* at 227.

205. *Id.*

206. *Id.* (quoting WILLIAM SARGANT & ELIOT SLATER, AN INTRODUCTION TO PHYSICAL METHODS OF TREATMENT IN PSYCHIATRY 145 (1944)).

207. *Id.* at 229.

the only major treatment in psychiatry that Shorter criticizes in this—or, for that matter, any other—way.

Despite—or perhaps, because of—his judgment about the unethical character of lobotomy, Shorter examines the benefits of the procedure. Although he discusses these briefly, they still get more attention than the benefits conferred by other pre-medication treatments. Apparently, Shorter wants to show that lobotomy was not bad enough to raise questions about psychiatry's bona fides as a medical science. Doing so, however, he goes too far, leaving it unclear why lobotomy was ethically indefensible in the first place. Thus, he notes that "'[t]here were some dramatic responses [to lobotomy], and some long-standing satisfactory responses—all better than the restraint of those same patients in back wards of mental institutions.'"[208] "Follow-up studies,"[209] according to Shorter, "found that about a third of all psychosurgical patients had been discharged from [the] hospital and were living at home."[210] "Yet,"[211] he continues,

> [M]any of these patients would sooner or later have recovered spontaneously. And the irreversible damage to their brain and spirit must be weighed against the extra months or years with which they would have encumbered the institutional system. "Not all so-called mental disorders were so severe that it was worth exchanging them for an organic brain syndrome," concluded one student. True, lobotomy reached the most difficult of the difficult in the back wards. Yet unlike any of the other physical therapies, it caused deep uneasiness within the profession of psychiatry, and would be the first of these therapies to be abandoned as the new antipsychotic drugs came in."[212]

None of these arguments, however, explains why Shorter considers lobotomy "indefensible for ethical reasons."[213] In fact, they often suggest the opposite. In some cases, lobotomy produced benefits, and, in Shorter's words, it was "better" than the alternative of staying in the hospital.[214] Why was the "better" alternative unethical then? The "unethical" course of action would appear to be withholding a "better" treatment, not administering it. Again, if it was true that "not all . . . mental disorders" reached a level of severity that warranted

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208. SHORTER, *supra* note 2, at 229 (quoting Eben Alexander, *A Perspective of the 1940s*, 28 SURGERY & NEUROLOGY 320 (1987)).

209. *Id.*

210. *Id.* (citing GROB, *supra* note 11, at 131).

211. *Id.*

212. *Id.* (footnotes omitted) (quoting STANLEY FINGER, ORIGINS OF NEUROSCIENCE: A HISTORY OF EXPLORATIONS INTO BRAIN FUNCTION 294 (1994)).

213. *See* SHORTER, *supra* note 2, at 229.

214. *See id.*

lobotomy, that hardly meant no mental disorders did. Not all heart conditions are severe enough to require surgery, for example, but that hardly means no heart patient should receive surgery or that surgery is unethical. Again, Shorter remarks that "many . . . patients would sooner or later have recovered spontaneously" without treatment, but he does not notice that this argument constitutes an objection to any treatment whatsoever.[215] It also conflicts with the basic point of Shorter's chapter on "Alternatives," namely, that psychiatry was "caught in a dilemma" in the 1930s because the existing therapeutic choices—warehousing patients or psychoanalysis—were "unappealing."[216] If that dilemma explains and justifies other physical treatments in this period, it should do the same for lobotomy. Moreover, as applied to the "back ward" patients that Shorter describes, the claims about "spontaneous" recovery were most likely untrue. Finally, if "irreversible damage to . . . brain and spirit must be weighed against . . . extra months or years" of hospitalization as Shorter says, it would appear that the balance should favor surgery in some cases—particularly when Shorter himself, in the very same paragraph, has described surgery as "better" than staying in the hospital.[217]

The incoherence, and even contradictions, in these arguments suggest that Shorter had difficulty reconciling lobotomy with his larger themes. Other features of his book suggest the same thing. Thus, the lobotomy discussion appears in the "Alternatives" chapter, but Shorter describes it as something quite different from an alternative. He writes instead about the "[l]obotomy [a]dventure,"[218] as if the treatment was wild and uncharacteristic of psychiatry. And he implies much the same thing about lobotomy's inventor, Egas Moniz.[219]

According to Shorter, Moniz reported great success with his first twenty lobotomy cases, claiming seven "cures" and seven improved patients.[220] Shorter points out, however, that Moniz provided "little detail to support these claims"[221] and provided an "account of the development of his procedure . . . [which] was filled with windy speculation about hypothetical mechanisms."[222] One gets the sense that lobotomy constituted an "adventure," rather than an alternative, because of Moniz's personal recklessness. Shedding additional light on Moniz's motives, Shorter notes that he "had twice been nominated for

215. *See id.*
216. *Id.* at 190.
217. *See id.*
218. SHORTER, *supra* note 2, at 225.
219. *Id.* at 226.
220. *Id.*
221. *Id.*
222. *Id.* at 390 n.153.

a Nobel prize"[223] for work in radiology, rather than psychiatry, but was "twice turned down."[224] Remarkably, Shorter neglects to add that Moniz later won the Nobel Prize for inventing lobotomy—a point that would be inconsistent with Shorter's portrayal of lobotomy as an uncharacteristic "adventure" for psychiatry.

Describing the first reports of success for other biological treatments, such as deep sleep or camphor, Shorter had expressed no comparable objection and offered no criticisms about lack of "detail."[225] Shorter knows, however, that those reports were no better than Moniz's. Thus, after criticizing Moniz for providing "little detail," Shorter adds the following: "[I]n fact, this kind of grandiose communication characterized the first notice of almost all the physical therapies, their authors eager to ascertain their priority in history and unaffected as yet by the rigorous statistical tests and follow-up studies that would later be demanded."[226] In the case of those other therapies, favorable "first notice[s]" led Shorter to conclude that the treatment "seemed" to work. Using the same approach, he should have concluded that lobotomy "seemed to work," too. Moreover, once Shorter conceded that Moniz's reports did not differ from Cerietti's or those of other biological pioneers, the idea of Moniz as an adventurer collapses as well. Once again, we find lobotomy in the mainstream of mid-twentieth century psychiatry, exactly where Shorter does not want it.

Lobotomy was, in fact, a dreadful procedure, but because of Shorter's determination to portray it as an aberration—an "adventure"—he misses the real criticisms of it. Peter Sterling has demonstrated that lobotomy did not produce real benefits, that it caused enormous harm, and that psychiatrists' optimistic portrayals of its supposed successes—the kind of success that Shorter posits in the tortured paragraph reproduced above—were usually bogus and outlandish.[227] Nor was it true that psychiatrists limited it to "back ward" patients; in fact, lobotomists preferred to operate on patients with little history of hospitalization.[228]

Shorter will not offer such criticisms, however. He limited his point about "grandiose communications" to the first reports of new treatments—a kind of law of the new drug for inventors of treatments.[229] If numerous psychiatrists, and not just Moniz, routinely offered bogus evaluations of lobotomy, reporting that badly damaged subjects had improved, then psychia-

223. SHORTER, *supra* note 2, at 226.
224. *Id.*
225. *Id.*
226. *Id.*
227. *See generally* Sterling, *supra* note 3.
228. *See generally id.*
229. *See* SHORTER, *supra* note 2, at 226.

trists' evaluations of any treatment—from sleep therapy to medications—could be equally bogus. Shorter struggles mightily against that conclusion.

As an ethical matter, lobotomy was "indefensible," just as Shorter says. However, if a procedure that becomes unethical because "irreversible damage to . . . brain and spirit"[230] can outweigh "extra months or years with which . . . [patients] would . . . encumber . . . the institutional system,"[231] then a strong case for medications being unethical in the same way can be made. In fact, George Crane made precisely that case in the 1970s when he criticized psychiatry for ignoring irreversible brain damage produced by drugs.

For Shorter, however, lobotomy must be *sui generis*. Lobotomy represented "a blip in the history of psychiatry," he writes, "a . . . study in medical hubris"[232] that "faded away in the early 1950s almost as abruptly as it had risen up."[233] No other procedure, from sleep therapy to insulin coma treatment, resembled it. No other procedure raised inherent "ethical" issues—not prolonged sleep therapy, despite the death rate associated with it, not drug-induced convulsions that terrified patients out of their minds, and not any of the other dangerous treatments that patients were forced to undergo despite a lack of convincing evidence of efficacy. Apart from the "lobotomy adventure," on Shorter's account, psychiatry boasted an unbroken history of over a hundred years of treatments that "seemed to work" or "truly worked" and that involved no ethical problems.[234]

Shorter's account of lobotomy highlights two additional aspects of his historiography. First, focusing on the dispute between psychoanalysts and biological psychiatrists, he ignores debates and disagreements over particular biological treatments. Even in the case of lobotomy, which some psychiatrists touted and others felt "uneasy" about, Shorter does not examine the actual debates.

Second, Shorter portrays medical treatments and ideas as if they develop autonomously, uninfluenced by public attitudes, government regulations, or laws. Virtually everything revolves instead around pure science, a science that reduces itself to a matter of being right (biological psychiatry) or being wrong (psychoanalysis). "Ethics" constitutes the only other force in Shorter's scientific universe—lobotomy was effective, yet "unethical"—but ethics plays a very small role. It is small because only one development—lobotomy—implicates ethical questions and because Shorter treats ethics as little more than conforming to the prevailing scientific consensus. Moreover, Shorter's

230. *Id.* at 229.
231. *Id.*
232. *Id.* at 228.
233. *Id.*
234. *See generally* SHORTER, *supra* note 2, at 228.

ethical considerations do not significantly affect the course of history. In his view, medications would have supplanted lobotomy during the 1950s even if no ethical question about the procedure had existed just as medications supplanted insulin coma therapy.

Shorter is wrong, however, about the cause of lobotomy's demise. The use of lobotomy had declined dramatically before medications ever appeared —and years before medications became a standard treatment.[235] It happened because public confidence in the procedure had collapsed.[236] After World War II and the revelations of Nazi atrocities, the nation's moral climate changed. Concepts of individual right and dignity had become more robust, and permanent brain damage had become correspondingly more suspect as a psychiatric or governmental technique. In this climate, large jury verdicts against lobotomists loomed as a real threat. For this reason, the law undid lobotomy more than medications did. Shorter cannot acknowledge this, however; it belies his view of medical developments as autonomous and driven by pure science. Shorter's unsatisfactory attempt to fill the gap is what he calls "ethics."

C. The Medication Era

Shorter approaches the medication era in the way one would expect given the rest of his book. He says little about the effectiveness of medications, except that they "work," and even less about drug side effects, including tardive dyskinesia. The bitter debates that had George Crane at their center receive no attention nor do law and public attitudes.

Just as he had in the case of other biological therapies, Shorter begins with anecdotes about early drug successes. These anecdotes do not include accounts such as that of the first psychiatrist to take thorazine during the drug's development, who reported feeling "that I was becoming weaker, that I was dying. It was very painful and agonizing . . . . I experienced an illness

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235. GELMAN, *supra* note 11, at 25; Sterling, *supra* note 3, at 135. Shorter acknowledges that "in Britain, the rate of lobotomies began to decline even before the introduction in the mid-1950s of the new antipsychotic drugs." SHORTER, *supra* note 2, at 228. Nonetheless, he asserts that "in both Britain and the United States, it was unquestionably the advent of these drugs in the spring of 1954 that killed off lobotomy." *Id.* In fact, Peter Sterling has shown that lobotomy went into a rapid decline in the United States before medications appeared. Sterling, *supra* note 3, at 133. Without citing sources, Shorter acknowledges that the number of lobotomies declined sharply after the early 1950s, but he ignores the fact that medications did not come into wide use immediately; it took time. *See* SHORTER, *supra* note 2, at 228. Based on what happened, one could more plausibly argue that medications spread in the vacuum left by the abandonment of lobotomy (and other biological therapies).

236. GELMAN, *supra* note 11, at 23.

more pronounced than depression . . . . I had felt all along that I was going to die, but this new state left me indifferent . . . ."[237]

Psychiatrists generally called the drugs "major tranquilizers" because patients became indifferent when taking them. Shorter uses a different term "antipsychotic,"[238] which did not come into general use until the 1960s or later[239] but which better fits his themes. Many psychiatrists during the 1950s theorized that the medications worked by producing a brain disease or state of brain dysfunction in patients, just as fever therapy and lobotomy had.[240] Shorter fails to mention that either. Indeed, Shorter does not write anything that could raise an "ethical" or even a serious medical issue concerning medications nor does he suggest that past psychiatric practices, such as lobotomy, could in any way color or taint the medication era.

Predictable in outline, Shorter's account includes some noteworthy errors and misinterpretations. These occur because Shorter's larger themes blind him to what happened during the medication era. The mistakes range from subtle to egregious.

On the subtle end of the scale is Shorter's comment on a French researcher's suggestion in 1952 that chlorpromazine might find its psychiatric use "in connection with barbiturates in a deep-sleep cure."[241] The researcher's suggestion was "perhaps tongue in cheek,"[242] Shorter writes. Yet the early French investigators did not think of chlorpromazine as an antipsychotic in the way Shorter does. Moreover, the drug was developed to potentate anesthesia. Suggesting that the chlorpromazine would find a use in deep sleep therapy was perfectly reasonable under the circumstances. Shorter raises the "tongue in cheek" possibility only because of his own conception of chlorpromazine as an effective antipsychotic agent, a therapy different in kind from all earlier treatments. In general, psychiatrists during the 1950s did not think that way.[243]

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237. JUDITH P. SWAZEY, CHLORPROMAZINE IN PSYCHIATRY: A STUDY OF THERAPEUTIC INNOVATION 117-18 (translating a report by psychiatrist C. Quarti) (1974). Quarti went on to say that the "painful feeling of imminent death" soon gave way to a sense of "euphoric relaxation" and "extreme feeling of detachment from myself and . . . others . . . everything was filtered, muted." *Id.* These changes lasted for about a week. *Id.*

238. *See, e.g.*, SHORTER, *supra* note 2, at 248.

239. *See* GELMAN, *supra* note 11, at 65.

240. *Id.* at 25-26.

241. SHORTER, *supra* note 2, at 249 (quoting Henri Laborit et al., *Un nouveau stabilisateur végétatif (le 4560 RP)* 60 PRESS MÉDICALE 208 (1952)).

242. *Id.*

243. For discussion, see GELMAN, *supra* note 11, at 24-29. Earlier in his book, Shorter suggested that another psychiatrist had spoken "tongue in cheek" when observing that "[o]ne may question whether shock treatments do any good to the patients but there can be no doubt that they have done an enormous amount of good to psychiatry." SHORTER, *supra* note 2, at 224 (quoting Louis Casamajor, *Notes for an Intimate History of Neurology and Psychiatry in America*, 98 J. NERVOUS & MENTAL DISEASE 607 (1943)). In this

More serious errors appear in Shorter's cursory discussion of side effects. Quoting Heinz Lehmann's early observations of medicated patients in 1953, Shorter writes that the patients "walked with a peculiarly stiff gait"[244] and "had that peculiar mask-like face"[245] suggestive of Parkinsonism.[246] According to Shorter, these "symptoms would later be called tardive dyskinesia"[247]—which is simply not the case. Dyskinesias are abnormal movements. Stiffness is not a dyskinesia. Moreover, tardive dyskinesia generally occurs late in treatment —hence the word "tardive"—rather than at the early treatment stages that Lehmann was observing. Neither in 1953 nor today would those symptoms be described as either "tardive" or as "dyskinesias." Shorter's mistake is quite remarkable for a serious history, and it suggests a profound lack of interest on his part in medication side effects.[248]

The balance of Shorter's account of side effects is also flawed. In discussing the phenomenon of deinstitutionalization, he writes: "[T]he antipsychotic medications that in hospital had provided such effective relief were often not taken once the patients were on the street because of tardive dyskinesia, the troublesome side effect that caused facial twitches and other involuntary movements."[249] Yet that certainly is not what happened. Side effects besides tardive dyskinesia—and notably the capacity of medications to make many people feel horrible—led patients to stop taking medications. Before the late 1970s, few people in the mental health system even knew about tardive dyskinesia, and in most cases, patients do not experience it as unbearably distressing. Shorter possibly cites tardive dyskinesia because he had reverted to the idea that that disorder includes all abnormal, drug-induced states, or he may have confused psychiatrists' view of tardive dyskinesia—that it is the only side effect with wide potential significance for prescribing practice—with the views of patients. This represents one of very few passages in the book concerned with patients' feelings and actions, as opposed to psychiatrists', and

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instance, too, nothing suggests that the remark was tongue in cheek. In fact, it parallels Shorter's own suggestion that some therapies were good for psychiatrists, whatever they did for patients.

244. SHORTER, *supra* note 2, at 253 (quoting Heinz Lehmann, *The Introduction of Chlorpromazine to North America*, 14 PSYCHIATRIC J. U. OTTAWA 263, 265 (1989)).

245. *Id.*

246. *Id.*

247. *Id.*

248. George Crane and others came to believe that drug-induced Parkinson's symptoms, like stiffness and tremor, might later evolve into tardive dyskinesia but this position was not widely held—and, in any case, it would be meaningless to hold it if Parkinsonism *was* "tardive dyskinesia." *See* George E. Crane, *Two Decades of Psychopharmacology and Community Mental Health, Old and New Problems of the Schizophrenic Patient*, 36 TRANSACTIONS OF THE N.Y. ACADEMY OF SCI. 644, 646 (2d ser. 1975).

249. SHORTER, *supra* note 2, at 281. Immediately after his initial reference to tardive dyskinesia, see *supra* text accompanying note 44, Shorter used similar language, writing that deinstitutionalized patients would "stop taking their medication" in order to avoid the "side effect" called "tardive dyskinesia."

Shorter certainly is in the habit of viewing everything from the psychiatrists' point of view.

Shorter's account of deinstitutionalization is flawed not just in its details but at its core. He deems deinstitutionalization a disaster, rightly calling it "one of the greatest social debacles of our time."[250] However, Shorter misplaces the responsibility for the "debacle." He lays the chief blame on the "antipsychiatry" movement, which was mainly made up of nonpsychiatrists. Antipsychiatrists believed that "psychiatric illness is not medical in nature but social, political, and legal."[251] Academic writings by the movement's intellectual leaders during the 1960s and 1970s—Michel Foucault, Thomas Szasz, and Erving Goffman—proved "influential among university elites,"[252] Shorter observes, "cultivating a rage against mental hospitals and the whole psychiatric enterprise."[253]

Hospital populations began a slight decline during the mid-1950s, shortly after the appearance of antipsychotic medications.[254] Since the antipsychiatrists had not yet published their books, Shorter allows that "[i]n a strict sense . . . deinstitutionalization was a consequence of the second biological psychiatry and not the antipsychiatry movement."[255] Nonetheless, the principal fault lies with the movement:

> Yet if drug therapy kicked deinstitutionalization off, what kept it going, driving patients of all kinds into the community whether they were treatable with drugs or not? It was the combined pressure of the antipsychiatry movement outside of medicine and of the ideology of community psychiatry within medicine. The antipsychiatry movement preached that mental hospitals as such were wicked, given that there was no such thing as mental illness. And well-meaning psychiatrists who had absorbed the teachings of [community psychiatry theorists] . . . believed that "therapeutic communities" could be constituted out there in the cold streets of big cities . . . .[256]

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250. SHORTER, *supra* note 2, at 277.

251. *Id.* at 274.

252. *Id.* at 275.

253. *Id.*

254. For discussion, see *supra* text accompanying note 24.

255. SHORTER, *supra* note 2, at 280. This observation conflates medications with the second biological psychiatry, even though elsewhere in the book Shorter dates the second biological psychiatry from the 1970s.

256. *Id.* In the "Alternatives" chapter, Shorter had described the development of social and community psychiatry theory, which occurred predominantly in England. *See id.* at 229-38. In Shorter's system, this theory was not part of the "second biological psychiatry."

Shorter's explanation hardly seems credible, however. In fact, large-scale deinstitutionalization resulted from a political decision by state governors and legislatures to reduce—and eventually all but eliminate—state mental hospitals. Judges and commitment law reforms possibly contributed something to the process as well. For its implementation, deinstitutionalization relied on clinical, that is to say, biological, psychiatrists. Working at state institutions and community agencies, these physicians discharged patients from hospitals and, in many instances, refused to re-admit them when they became ill again. Moreover, as George Crane had observed, deinstitutionalization depended on medicating virtually every patient—again, something only psychiatrists could do.[257] Leading biological psychiatrists were among the pioneering theorists of deinstitutionalization as well.[258]

To accept Shorter's analysis, one must believe that the tracts of anti-psychiatrists and the writings of British psychiatrists who theorized about "therapeutic communities" had cast a spell over governors, legislators, judges, and over the psychiatrists who implemented deinstitutionalization. That simply did not happen, and it borders on fanciful to suppose it did. Political officials and judges hardly spent their time plodding through antipsychiatrist texts. Rather, as New York Governor Averill Harriman explained when he decided to authorize widespread use of medications in state hospitals, the goal was saving money.[259] Governors had also tired of the endless scandals that engulfed state hospitals.[260]

It is true that some of the lawyers who sued mental hospitals in the 1960s and 1970s espoused "antipsychiatrist" views.[261] These lawyers did not decide the cases however, judges did. Nor did these lawyers settle cases on behalf of states and then implement the settlement by discharging of patients wholesale. The states did that. Moreover, whether they denied existence of mental illness or not, the lawyers did little at critical junctures to check the use of medication, something essential to deinstitutionalization.[262] In their actions, then, the lawyers were more pro-deinstitutionalization than "anti-psychiatry." Indeed, since psychiatry largely supported deinstitutionalization—despite Shorter's claims—the lawyers were actually aligned with psychiatric goals, not opposed to them.[263]

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257. *See infra* text accompanying note 269.

258. GELMAN, *supra* note 11, at 72-75.

259. *Id.* at 40.

260. *Id.*

261. *See* Nancy K. Rhoden, *The Limits of Liberty: Deinstitutionalization, Homelessness, and Libertarian Theory*, 31 EMORY L.J. 375, 403 (1982).

262. For discussion, *see* GELMAN, *supra* note 11, at 121-34.

263. *Id.*

Crane himself receives a single mention from Shorter in a footnote that, unsurprisingly, badly distorts his views.[264] The footnote cites a 1973 article by Crane to support Shorter's argument that because of tardive dyskinesia, patients "out on the street"[265] stopped taking medications, which "had provided such effective relief"[266] in the hospital. According to Shorter, Crane's article was "an early *prise de conscience* of this problem."[267]

Crane's article did focus on deinstitutionalization and on tardive dyskinesia, a disorder that, according to Crane, psychiatrists "seem[ed] to be completely unconcerned about."[268] But Crane, unlike Shorter, never imagined that medications "provided such effective relief" in hospitals. To the contrary, Crane's article reported that "fewer than 50 percent of patients hospitalized for several years improve in response to neuroleptics."[269] Combined with the high risk of serious side effects, drugs' limited effectiveness made the practice of medicating every patient indefensible,[270] Crane argued. Quite naturally, Crane held psychiatrists and clinical psychopharmacology responsible.[271] Indeed, he considered their view of medications an essential part of deinstitutionalization. Shorter's blaming "antipsychiatrists" would have struck Crane as absurd.

Shorter gets deinstitutionalization wrong in part because of his insistence that psychiatric ideas and debates—exclusive of legal, social, or political concerns—explain everything in the history of psychiatry. For that reason, he ignores the powerful political impetus to dismantle the state hospital system. Shorter also gets deinstitutionalization wrong because he will not concede that psychiatry is capable of any systematic wrong. If deinstitutionalization represents a debacle—and Shorter says it does—then the fault must lie with antipsychiatrists, not psychiatrists.

The idea that psychiatry is incapable of systematic wrong to patients—anything more sustained and serious, that is, than a "blip" in history—lies at the heart of Shorter's approach to his subject. Medications, like other biological therapies, benefit from this principle. It is almost inconceivable that the profession Shorter portrays could have performed during the medication era in the way Crane described. If Shorter is right about psychiatry's history, Crane had to be wrong about medications. By the same token, if Crane was right about medications—and Crane *was* right—then something is very wrong in Shorter's history.

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264. SHORTER, *supra* note 2, at 406 n.153.
265. *Id.* at 281.
266. *Id.*
267. *Id.* at 406 n.153.
268. George E. Crane, *Clinical Psychopharmacology in Its 20th Year*, 181 SCIENCE 124, 127 (1973).
269. *Id.* at 125.
270. *Id.* at 126-27.
271. *See id.* at 125.

Shorter's belief in the absolute autonomy of psychiatric ideas and in the blamelessness of psychiatrists leads to a kind Manichean history, one in which a battle between two forces—right and wrong—determines everything. Shorter's scheme of three periods frames this as struggle of biological psychiatry versus psychoanalysis. However, as befits a Manichean history, at bottom, Shorter sees a struggle between psychiatry and antipsychiatry.

Antipsychiatrists consider psychiatry a social, political, and legal creation, nothing more. That position leads them to overlook distinctively medical developments and to presume that psychiatry can do nothing right. Shorter, on the other hand, is an "anti-antipsychiatrist." He views psychiatric developments and debates as completely free from social, political, and legal influences.[272] Thus, he attributes the "debacle" of deinstitutionalization to an academic mistake by professors of history and sociology about the nature of psychiatry and mental illness.

On principle, then, Shorter ignores psychiatry's problems and highlights antipsychiatry's. Just as psychiatry can do no wrong, antipsychiatry can do nothing right. Since Shorter allocates deinstitutionalization to the realm of *antipsychiatry*, he readily acknowledges the problems that affected that policy. But since psychiatry actually caused those problems, Shorter's account of deinstitutionalization includes some remarkable facts that his approach to *psychiatry* had ruled out. From these examples, we learn that the shortcomings in Shorter's history are not due to lack of knowledge on his part; instead, they result from his unwillingness to fault psychiatry.

The paragraph that blames antipsychiatrists and community theorists for "driving patients . . . into the community *whether they were treatable with drugs or not*,"[273] is one example. The cryptic phrase "treatable with drugs or not" is Shorter's only acknowledgement that medications have limited effectiveness. It is difficult to say which is more remarkable: the fact that Shorter blames antipsychiatrists, who do not believe in mental illness, for failing to appreciate that some patients should remain drug-free, or the fact that Shorter writes at length about medications but ignores the limitations of the treatment, except for those he associates with the antipsychiatrists.

The second example relates to Shorter's basic conception of biological treatments from the 1920s and 1930s, as justifiable "alternatives" to "warehous[ing]" patients."[274] "At the outset,"[275] Shorter writes,

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272. Remarkably, Shorter says in the introduction that he has tried to write a "social history" of psychiatry. SHORTER, *supra* note 2, at viii. By social history, however, Shorter apparently means no more than an attempt to "recaptur[e] the lives of some of the major players." *Id.*

273. *Id.* (emphasis added).

274. *Id.*

275. *Id.*

> [A]ll of these alternatives had an aura of desperateness about them, seemingly radical and possibly quite dangerous innovations. This desperateness must be understood in the context of the time. The asylums were filing, and psychiatry stood helpless in the face of disorders of the brain and mind. In these years, the profession reached the nadir of its descent from the therapeutic promises that had beckoned so brightly a century before . . . . [T]he center of gravity of psychiatry lay in the mental hospitals. In these snake pits, a bleakness prevailed that would have turned away any but the most resolute young medical graduate.[276]

Shorter takes a different view of matters, however, when antipsychiatrists —or those Shorter associates with them—criticize mental hospitals. Books published in the late 1940s by a journalist named Albert Deutsch,[277] and films such as *The Snake Pit* dating from the same period, depicted what Shorter himself calls the bleakness of mental hospitals. Describing these developments from the 1940s, Shorter writes:

> Midst this horrendous publicity for psychiatry, on which the antipsychiatric movement would later feed, several basic realities were obscured. One is that most patients younger than 65 were discharged relatively rapidly from mental hospitals: They did not experience prolonged stays to say nothing of lifelong incarceration . . . . Second, much of the bizarre posturing and disordered movement that Deutsch and later antipsychiatric writers ascribed to "hospitalism," meaning the iatrogenic results of institutionalization, turned out to be an inherent biological feature of such illnesses as schizophrenia that, in affecting the entire brain, affect the entire nervous system as well. Third, even though conditions in mental hospitals were unsettling enough, there were worse alternatives. One was being tossed to the mercy of the streets."[278]

Thus, antipsychiatrists and their allies commit fundamental errors by ignoring patients who fared relatively well in hospitals and by overlooking the possibility of "worse alternatives" than institutions. Yet Shorter himself commits exactly those errors when discussing "alternatives" to hospitalization in the 1920s and 1930s—that is, the biological therapies such as prolonged

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276. *Id.* at 190.

277. ALBERT DEUTSCH, THE SHAME OF THE STATES (1948).

278. SHORTER, *supra* note 2, at 278-79 (internal references omitted). Interestingly, Shorter made a similar point about hospitalization as an alternative to lobotomy; see discussion *supra* text accompanying note 210.

sleep and coma therapy. When considering those treatments, Shorter never mentions that some patients might fare relatively well in the hospital nor does he consider the possibility that those treatments represented a "worse alternative" than doing nothing. The supposed difference between these situations does not rest on any facts; it simply follows from Shorter's premise that psychiatrists can do nothing wrong and the critics of psychiatry can do nothing right. It is not that psychiatrists escape blame for harsh therapies because no alternative existed. Rather, no alternative existed in Shorter's view because, as a matter of principle, he will not blame psychiatrists.

D. Progress and Revolutions

During the 1960s, 1970s, and 1980s, many psychiatrists came to regard medication as a revolutionary treatment that had transformed their discipline and rewritten its history.[279] On this view, there was the medication era in psychiatry and an earlier, virtually irrelevant, professional past.[280] Nothing existed in between.

Thinking this way carried obvious—and for psychiatry, highly desirable—implications. It made psychiatry's history before 1953 irrelevant in evaluating the profession or its capabilities. Not only did nineteenth century procedures, like removing patient's ovaries, become relics of the irrelevant past, so did mid-twentieth measures like lobotomy, which the public had come to revile.

This idea of medications as revolutionary plays some role in Shorter's book. He even refers to a medication "revolution."[281] More generally, Shorter believes in scientific progress. He portrays the first and second biological psychiatries as similar in fundamental ways, but also recognizes that the later period boasted real scientific knowledge about the brain, not just a vague biological orientation. Nor are medications the same thing as prolonged sleep therapy in Shorter's view. In addition, Shorter occasionally considers earlier treatments in light of the medication revolution to come. Writing about the older therapies, he observes that "[s]ome . . . proved to be dead ends and were discarded; others became the basis of a new vision of psychotherapy; still others laid the groundwork for the revolution in drug therapy . . . after World War II."[282] Thus, Shorter places favored therapies on the path of progress.

Yet Shorter is not a full-fledged revolution historian. He does not believe that medications fundamentally changed psychiatry. The kinship he sees

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279. GELMAN, *supra* note 11, at 70. Views of the "medication revolution" are described in *id.* at 70-71, 223-29.

280. *Id.*

281. SHORTER, *supra* note 2, at 190.

282. *Id.*

between the first and second biological psychiatries reveals a basic continuity in psychiatric history, not revolutionary change. Whether in 1890 or 1980, the biological psychiatrists were right, and everyone else, wrong, in Shorter's view. Moreover, Shorter acknowledges that physicians discovered medications serendipitously; their discovery did not build on scientific work by sleep therapists or insulin coma physicians.

Nor is Shorter a simple "progress" historian. Progress historians see the present as a kind of purpose that the past aimed to achieve. They justify the past because it produced the present. Shorter, however, allows older therapies only the most tenuous connections to modern treatment. Often, the older therapy contributed little more than hope. Shorter writes, for example, that fever therapy demonstrated one kind of insanity (neurosyphilis) to be "curable."[283] Prolonged sleep therapy "offered the prospect of cure,"[284] "address-[ed] the brain,"[285] and, apparently for that reason, "marked the beginning of a revolution in psychiatry."[286] Drug-induced convulsions represented steps on the path to electroconvulsive therapy, which remains in use.[287] Insulin coma gives Shorter difficulty on this count, though he emphasizes that it represented a "substantial improvement" over doing nothing, and he makes a halfhearted effort to connect it with ECT.[288] None of these connections seem substantial —wouldn't psychiatrists have wanted to cure psychosis if fever therapy and prolonged sleep had never existed?—and Shorter in fact makes relatively little of them. They represent little more than bows in the direction of progress.

The older biological treatments stand on their own merits, as Shorter views them, with no need for the progress rationale. The treatments "seemed to work," according to Shorter, and were superior to available alternatives. Reading his book, one simply does not get the sense that prolonged sleep therapy and coma treatment would have been unjustifiable had they not led (in some obscure way) to medications and ECT. Progress historians value the past because it produced the present. Shorter generally values psychiatry's past for its own sake.[289]

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283. *Id.* at 196.

284. *Id.* at 206.

285. *Id.* at 205.

286. SHORTER, *supra* note 2, at 207.

287. *See id.* at 214.

288. *Id.* Shorter writes that insulin coma "seemed to be a procedure that actually worked" and "represented a substantial improvement on what was available before, which is to say, nothing." *Id.* He goes on to say that the drug metrazol, which "produced convulsions without coma," represented "the true beginning of convulsive therapy." *Id.* Shorter's attempt to link insulin with metrazol, and his strange suggestion that comas were a poor precursor to convulsions, both bespeak an effort to place insulin coma treatment on one of the paths of progress.

289. Without changing a great deal, Shorter might possibly recast his narrative to better fit the progress mold. In order to do that, Shorter could present the "psychoanalytic hiatus" as something that

An example will illustrate the role that progress and revolutions play in Shorter's *History*. Discussing the introduction of medications in 1953, Shorter quotes the recollections of Heinz Lehmann, a pioneering Canadian clinical psychiatrist. Before medications appeared, Lehmann found it "pretty horrible to work under . . . [mental hospital] conditions."[290] Being "convinced that psychotic conditions . . . had some sort of biological substrate,"[291] Lehmann had "kept experimenting."[292] He treated patients with "very large doses of caffeine."[293] He "injected sulfur suspended in oil,"[294] producing only pain and fever. He "injected typhoid antitoxin,"[295] hoping to duplicate the success of malaria therapy. He "even injected turpentine into the abdominal muscles"[296] in order to "produce . . . a huge sterile abscess."[297] All these measures had been suggested by other psychiatrists, but Lehmann found "[n]one . . . had any effect."[298]

Then, in 1953, a pharmaceutical salesperson gave Lehmann some medication samples and sales literature.[299] Lehmann suspected the new drug was only a sedative.[300] But the sales literature claimed that medications "'acted like a chemical lobotomy,'"[301] which impressed him. On that basis, and because he considered the psychiatrists who had discovered medications "very sophisticated,"[302] Lehmann tried the new drugs.[303] The rest was history.

Shorter takes the view one would expect of Lehmann's pre-medication experiments. "The point,"[304] Shorter writes,

> is not that researchers such as Lehmann behaved inhumanely with their patients: They were searching in the best of faith for something

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interrupted the processes of progress in psychiatry. Such an account would have to include casual connections between the first biological psychiatry and the second one, which Shorter does not supply. It might also require that Shorter demonstrate progress as treatments evolved from prolonged sleep to insulin coma. Such a narrative would not be impossible to write. I do not think it would be convincing, however, nor, in my view, did Shorter attempt to write it.

290. SHORTER, *supra* note 2, at 247 (quoting Lehmann, *supra* note 244, at 263).
291. *Id.*
292. *Id.*
293. *Id.*
294. *Id.*
295. SHORTER, *supra* note 2, at 247 (quoting Lehmann, *supra* note 244, at 263).
296. *Id.*
297. *Id.* at 248.
298. *Id.*
299. *Id.* at 252 (citing David Healy's then-unpublished interview with Lehmann).
300. SHORTER, *supra* note 2, at 252.
301. *Id.*
302. *Id.*
303. *Id.*
304. *Id.* at 248.

> better to offer them. It is rather that, by the time in 1951 that [research began into antipsychotics,] . . . the ground had already been well prepared for the reception of new . . . drugs.[305]

This "point" conflicts, however, with some of Shorter's larger themes. Shorter claims that psychiatrists held lobotomy in disrepute at the start of the medication era. However, we now know that the drug company marketed medications as "chemical lobotomies"—and the marketing pitch resonated with Lehmann. If psychiatrists were truly concerned about the "ethical" issues surrounding lobotomy as Shorter says, this marketing strategy made no sense. Moreover, if insulin coma therapy really "seemed to work," and if ECT produced the successes that Shorter claims, it is unclear why Lehmann felt so discouraged about mental hospitals, or why he resorted to drastic measures like toxin and turpentine injections. Shorter repeats the "alternatives" rationale—psychiatrists like Lehmann were "searching in the best of faith for something better" —but he forgets that the original alternatives, the coma and convulsive treatments, seemingly had succeeded by this time.

These details aside, many readers will simply not accept Shorter's "point" about Lehmann. Instead, they will think Lehmann "behaved inhumanely" by injecting patients with sulfur, toxins, and turpentine. On that basis, readers may distrust all Lehman's judgments, including his subsequent assessment of medications. They may also distrust psychiatrists generally or at least psychiatrists like Lehmann. Such a reaction is natural. It involves looking at later events through the lens of earlier ones—or, if not that, looking at two events (injecting turpentine and injecting medications) as if they had occurred simultaneously. Critics of psychiatry—particularly antipsychiatrists—often employ such views, tarring the field with its history of crude treatments.

Shorter, like many of these critics, sees the same fundamental processes at work in psychiatry's present and its past. Critics argue that the same professional processes lie behind medications and prolonged sleep therapy—and that we should therefore consider medications as if they were like prolonged sleep therapy. Shorter agrees with critics' premise. He thinks that the same professional processes have been at work throughout psychiatric history, but he wants us to think of prolonged sleep therapy as a good thing, not a bad one. And that creates a problem for Shorter's position.

This problem can be explored with a simple model. Shorter treats the periods of psychiatry's history as if they were unfolding simultaneously in three different rooms inside a building marked "Science." Within Room #1, biological psychiatrists of the late nineteenth century proceed scientifically and pursue promising leads but have achieved no breakthroughs in understanding

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305. SHORTER, *supra* note 2, at 248.

the brain as of yet. In Room #2, the psychoanalysts are not attending to science at all; instead, they ramble on about their patients' parents and childhoods. In Room #3, biological psychiatrists proceed very much like those in Room #1 except that because of a serendipitous discovery, they have acquired a deeper understanding of the brain. The psychiatrists in Rooms #1 and #3 obviously practice the same profession and belong in the same building, but the same cannot be said about the psychoanalysts in Room #2.

Viewed in this way, Shorter's view is not inherently implausible. The existence of Rooms #1 and #2 does not make the scientific breakthroughs in Room #3 less likely or believable. This model, however, omits biological treatments. So instead of building marked "Science," imagine one called "Mental Hospital." Again, there are three rooms. Inside Room 1a, psychiatrists inject turpentine and toxins into patients, induce comas and perform lobotomies. In Room 2a, they talk about patients' childhoods and dreams. In Room 3a, they administer psychiatric medications. In this model, the existence of Room 1a might lead an observer to question the entire hospital and everything that happens within it, including in Room 3a. If a hospital harbors Room 1a, something is fundamentally wrong with the entire institution.

Shorter deals with this conceptual problem in three ways, two of which I have already described. First, he portrays the benefits of older treatments using ambiguous—and ultimately, misleading—phrases, such as "seemed to work," while largely ignoring side effects. This makes older treatments appear more benign, but it does not entirely eliminate the difficulty. One remains suspicious of inducing comas and convulsions in patients as a treatment for mental distress. Second, Shorter defines the periods of psychiatry's history—in our model, the rooms—solely with reference to general theories of mental illness. Pre-medication treatments do not unfold inside the theoretical rooms —Rooms #1, 2, and 3 in our model. Rather, Shorter places those treatments in their own chapter. Yet the treatments belong in psychiatry's history somewhere. If they cannot be found within the rooms, they must be located somewhere else in the building—in unmarked rooms perhaps, or in the hallways. Neither possibility helps Shorter very much.

Progress is Shorter's third way of dealing with the problem. Using the model of buildings and rooms, progress gives us a reason to focus on Room 3a (medications), and not Room 1a (turpentine, toxins, and lobotomy) when thinking about events in the "Mental Hospital" building. Knowledgeable and insightful observers might not need a heuristic aid-like progress. Without it, they may still understand that the events in Room 1a only appear "inhumane" on the surface. But many people, perhaps ruled by emotions, require something to help them understand—something like the idea of progress. With its help, one can think of Lehmann as the doctor who pioneered the use of medications after he had tried other treatments—and not as the psychiatrist

who treated patients, at different times, with toxins, turpentine, and medications.

E. Neoapologists and Revisionists

In matters of historiography, Shorter describes himself as a "neoapologist"[306] for psychiatry and his opponents as "revisionists."[307] Revisionist historians deny the existence of mental illness and presume that "doctors act not in the interest of their patients or of science, but to shore up their own sagging authority."[308] Neoapologists, on the other hand, hold that mental illness exists and that psychiatrists generally act scientifically in the interest of patients.[309] Thus, historical revisionists qualify as antipsychiatrists and historical neoapologists as biological psychiatrists. (Recall that in order to qualify as a biological psychiatrist, one only need believe in the biological basis of mental illness.) Remarkably then, Shorter sees historians divided along precisely the same lines as psychiatrists, over precisely the same basic issues —a view that makes the historiography of psychiatry something very much like a subdivision of psychiatry itself.

Shorter says that his first objective in writing the book was "to rescue the history of psychiatry from the sectarians who have made the subject a sandbox for their ideologies."[310] In light of the parallels between historiography and psychiatry, Shorter probably equates criticism of prevailing practice in biological psychiatry with sectarianism and ideology. But if anything deserves the label "ideological," Shorter's book does. It presumes that biological psychiatry can do no wrong. And its selection of facts and topics is rigidly determined by his assumptions about the autonomy and blamelessness of psychiatry.

Thus, we learn little about the side effects of treatments or, for that matter, about treatment benefits. The existence of side effects would threaten the assumption that psychiatry does no wrong, particularly since psychiatrists often ignored those effects. And treatment benefits require little attention because of the assumption that psychiatry is beneficial and benign. Thus, Shorter omits the very things that one would like to learn from a history of psychiatry.

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306. *Id.* at ix. Shorter distinguishes neoapologists from "real apologists" on the ground that the latter believe that "the rise of the asylum represented undiluted progress in the alleviation of human misery." *Id.* at viii. He might have added that neoapologists believe the same thing about developments in biological psychiatry.

307. *See id.* at ix.

308. *Id.*

309. Neoapologists also hold that psychiatrists' pursuit of professional self-interest and authority "ends up explaining little of a complex story." *Id.* at ix.

310. SHORTER, *supra* note 2, at viii.

The failings of Shorter's account, such as they are, do not result from an unwillingness on his part to make judgments about past actors and events. His is not a neutral history written without a point of view. Instead, Shorter makes numerous evaluative judgments about the past. He considers lobotomy unethical, for example, and the "psychoanalytic hiatus" as a colossal mistake. Antipsychiatrists qualify past historical actors too, and Shorter simply denounces them.

In Shorter's Manichean history, to be a biological psychiatrist was to be right in all times and places—even when biological theories were vapid and biological treatments crude. To criticize biological psychiatry was wrong, again in all times and places, even when biological theories had nothing to offer. Problems that psychiatry had obvious responsibility for—most notably, deinstitutionalization—Shorter blames on antipsychiatrists, as if harm could come only from them.[311] Facts inconsistent with his story of psychiatric triumph—for example, many patients do not respond to medications—Shorter also connects with antipsychiatrists, as if French philosophers and American sociologists were responsible for medical shortcomings.[312] Shorter justifies harsh psychiatric treatments on the ground that anything was better than asylums, and he criticizes antipsychiatrists for overlooking the fact that some things were worse than asylums.[313] Throughout, Shorter virtually ignores the harm caused by psychiatrists and psychiatric interventions. Lobotomy, an apparent exception, he dismisses as an aberration.

Imagine a history written to the same standards as Shorter's but defending opposite conclusions. This history would describe the harms older psychiatric interventions caused and report summarily that the treatments "seemed" *in*effective.[314] It would denounce psychiatrists' motives in using these treatments.[315] It would place the entire blame for deinstitutionalization on psychiatrists, ignoring the role played by states.[316] Regarding more recent developments, it would report that medications "*truly* caused brain damage,"[317] and describe two or three individuals seriously damaged by drug treatment. Drug benefits would receive passing mention in one or two sentences—sentences that misused the basic terminology.[318] Throughout, the successes

311. For discussion, *see supra* part IV(C).

312. For discussion, *see id.*

313. For discussion, *see id.*

314. For discussion of Shorter's treatment of this point, see *supra* text accompanying notes 170-77.

315. For discussion of Shorter's treatment of this point, see *supra* text accompanying note 305.

316. For discussion of Shorter's treatment of this point, see *supra* text accompanying note 251.

317. *Compare* Shorter's description of medications as a treatment that "truly worked," discussed *supra* text accompanying notes 174-77.

318. *Compare* Shorter's account of tardive dyskinesia, discussed *supra* text accompanying notes 247-48.

achieved by psychiatry would be ignored or else portrayed as aberrational. Of course, such a history would be sectarian and ideological but no more than Shorter's own history.

In either version, ideological history omits much that a history of psychiatry should include. Without knowing how well treatments worked and the harms they caused, we cannot assess psychiatrists' performance nor do treatments themselves tell the whole story. Potentially beneficial therapies may be abused, but we cannot know whether that happens without examining how psychiatrists actually use treatments.

For the medication era in particular, Shorter's approach produces a false picture. His historiography, as noted above, practically ruled out George Crane's criticisms of psychiatrists and medication practices. Crane had charged psychiatrists with systematic abuse of a potentially beneficial treatment when, for Shorter, the entire history of psychiatry made such abuse a virtual impossibility. We can now see something else in Shorter's approach —something even more basic—that precludes criticisms like Crane's. How psychiatrists perform—as opposed to what therapies psychiatrists possess or what they believe about mental illness—does not appear to be a legitimate question for Shorter. Certainly, Shorter does not ask it. Thus, Crane offered not only the wrong answer, but the wrong question—and "wrong" in every sense of the word.

Crane's critics regarded him as not just mistaken but as professionally deranged. For those critics and for Shorter alike, some questions simply do not get asked and to pose those questions marks one as anti-psychiatry, which is to say, is responsible for everything that goes wrong. (One of Crane's critics suggested that patients needed protection, not from side effects, but from Crane.)[319] Nor is it without significance that Shorter dates the triumphant emergence of the second biological psychiatry at the very time when leading psychiatrists were denouncing Crane and insisting on a tardive dyskinesia rate of 1 in 1,000,000. In this way at least, Shorter's suggestion of a fundamental identity between psychiatry and psychiatric historiography is correct. Shorter has written a history that goes hand in hand with the views of psychiatrists who denied tardive dyskinesia's existence at the birth of the "second biological psychiatry."

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319. GELMAN, *supra* note 11, at 82 (citing George E. Crane, *Tardive Dyskinesia in Schizophrenic Patients Treated with Psychotropic Drugs*, 9 AGGRESSOLOGIE 209, 218 (1967) (reporting an "emergency discussion" following the reading of Crane's paper). The psychiatrist Herman Denber charged that "sweeping, generalized conclusions" such as Crane's would "undo the past 15 years of work" in psychiatry —that is, the medication era—and he exhorted "each psychiatrist who treats patients to speak for and defend them." *Id.* In context, it seems clear that the patients needed to be defended against Crane. For discussion of Denber's criticisms, and of similar criticisms by Nathan Kline, see *id.* at 81-87.

F. Why Not a Revolution?

Although Shorter sheds little light on psychiatry's past, his approach is revealing about the writing of history and also about our present situation. Shorter did not take the easiest path for an apologist. He did not argue that a medication revolution had fundamentally transformed the field, making psychiatry's questionable past irrelevant,[320] nor did Shorter take the next easiest path, which is to argue that judgments about psychiatry's past are impossible from our present vantage point. Instead, as already noted, he makes all kinds of judgments about biological psychiatry, antipsychiatrists, and treatments.[321]

Why would an apologist endorse the history of sickening patients, convulsing them, and making them comatose when alternative historical techniques exist—techniques that wall off psychiatry's past? Possibly, Shorter's primary allegiance lies with history rather than psychiatry. For the sake of professional pride perhaps, he wants to invest the past with as much relevance as he can or perhaps he simply considers the older treatments justifiable.

Whatever Shorter's personal reasons, the history of apologetics for psychiatry over the past four decades suggests a different kind of answer. In 1964, a pivotal study of medications had suggested a vision of psychiatric history very much like Shorter's.[322] This study contributed greatly to the idea of medication as a revolutionary treatment—one that reversed schizophrenic processes in the brain. Yet the study's authors also portrayed older psychiatric treatments as effective, commenting that "it has been clear for many decades [prior to 1964] that acute schizophrenic patients had reasonable chances of improving with available treatments."[323] Moreover, like Shorter, the authors focused on psychiatrists' mindset and outlook and not merely on the outcomes of treatment for patients. Despite the effectiveness of older therapies, they noted, "there generally has been a cautious and skeptical, if not nihilistic, attitude [among psychiatrists] toward the prognosis of schizophrenia. However, in the past two decades the situation has greatly improved, and considerable optimism now attends the treatment of acute schizophrenia."[324] Indeed, they thought that the study's findings about medication "lend strong support

320. For discussion, see *supra* Part IV(E).

321. For discussion see *supra* this part.

322. NIMH Psychopharmacology Service Center Collaborative Study Group, *Phenothiazine Treatment in Acute Schizophrenia*, 10 ARCHIVES GEN. PSYCHIATRY 246 (1964).

323. *Id.* at 256.

324. *Id.*

to the rising optimism about and confidence in the treatment of acute schizophrenic psychoses."[325]

Compare this to Shorter's remarkably similar claims regarding the therapies of the1930s: "The historical record . . . shows that coma and shock gave psychiatrists powerful new therapies in a field dominated for half a century by nihilistic hopelessness, and it is against that sense of despair that they must be set."[326]

Looking back on the 1964 study today, its combination of faith in older treatments with a view of medications as a revolutionary advance is striking. Of course, there is nothing illogical about holding the two positions simultaneously. The existence of effective treatments for a disease does not preclude the possibility of better treatments emerging later. In the wake of the 1964 study, however, psychiatrists had embraced not only the idea of medications reversing schizophrenic brain processes, but also the idea of medications as revolutionary treatment that had transformed psychiatry and made the profession's past irrelevant—precisely because of medications' supposed actions in the brain.[327] It was as if medications cured not only schizophrenia, but the problems of psychiatry as well.

An early synthesis of those ideas appeared in a work of history interestingly enough. In a 1974 book about the development of medications, Judith Swazey had described the 1964 study as "the most definitive demonstration of a fact gradually perceived by psychiatrists during the 1950s: that CPZ [chlorpromazine] is not just a 'glorified sedative,' a drug uniquely effective in controlling psychotic excitation, but a true 'antischizophrenic' drug, with highly specific actions against the range of symptoms characterizing the schizophrenic illness."[328]

Yet little reason existed to believe that medications exert "highly specific" actions against schizophrenia.[329] Swazey was creating a medication revolution more than she was describing one. Nonetheless, the view of medications as antischizophrenic treatment—and therefore a revolutionary treatment—took hold in the late 1960s. Before then, it had seemed perfectly reasonable to describe medications as a major advance and also to suppose that no medical revolution had occurred in psychiatry. It was the very success of the 1964 study in portraying medications as "antischizophrenic" that makes its historical vision seem odd today.

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325. *Id.* at 257.
326. SHORTER, *supra* note 2, at 208.
327. For discussion, see GELMAN, *supra* note 11, at 59-76.
328. SWAZEY, *supra* note 237, at 16-17.
329. GELMAN, *supra* note 11, at 53-54; GROB, *supra* note 11, at 152-53.

If the 1964 study's portrayal of history and pre-medication treatments now appears anomalous, what of Shorter's book, which adopts a similar historical view? Like the study, Shorter both regards medications as a major advance *and* posits a basic continuity in psychiatric history. He does not perceive the introduction of medications in 1953 as the beginning of a new era; his second biological psychiatry does not commence until a decade and a half later. Also like the study, Shorter focuses on the way that treatments affected the outlook of psychiatrists (as noted above).

The authors of the study wrote under conditions very different from Shorter's, which makes the similarities between them even more remarkable. The 1964 study offered a pioneering demonstration of medications' effectiveness. With very few exceptions, previous studies of medication had involved about the same degree of rigor as studies of lobotomy had. Planning for the study dated back almost a decade and those responsible for it had the lobotomy debacle firmly in mind. They did not want the profession to endure a similar disaster because of drugs.[330] In short, the study was born out of defensiveness.

The study's authors could not have known how successful their conception of "antischizophrenia" medication would become or how quickly medications would dominate psychiatry. Moreover, the senior and middle generations of psychiatrists in 1964 had used the older biological therapies freely during their careers. In all likelihood, they did not want to hear that medications had relegated their previous work to the dustbin of medical history.

When Shorter's book appeared in 1997, the situation was different. Medications had taken over psychiatry, their superiority an article of faith. The senior generation of psychiatrists had always used medications. For good or ill, the treatment had transformed the public mental health system.

No less significant in the late 1990s was the fact that prevailing practice in psychiatry had survived serious threats, both internal and external. Internally, there had been George Crane and the profession's failure to acknowledge tardive dyskinesia—a side effect—and a professional failure that many had feared would produce a lobotomy-like public reaction against medications. Externally, psychiatric practices had survived the litigation of the 1970s and 1980s, which had threatened drastic changes. By 1990, it had become clear that courts would do little, if anything, to interfere with the use of medications.

In the wake of these developments, another conception of psychiatry and its history emerged. According to Robert Michels and Peter Marzuk, psychiatry had "undergone a profound transformation in recent years"[331] as its "focus

330. GELMAN, *supra* note 11, at 53; GROB, *supra* note 11, at 152.

331. GELMAN, *supra* note 11, at 208 (quoting Robert Michels and Peter M. Marzuk, *Progress in*

of research . . . shifted from the mind to the brain"[332] and its conception of psychiatric illness "shifted from a model of . . . disorders based on maladaptive psychological processes to one based on medical disease."[333] Noting that many key research conclusions remained unconfirmed and that "only a few have been translated into clinical practice,"[334] Michels and Marzuk nonetheless concluded on a triumphant note:

> A 1975 review . . . described psychiatry as "the battered child of medicine . . . born in witchcraft and demoniacal possession, feared by the public, often scorned by the family of medical specialists, and dependent for much of its existence upon handouts from public agencies." One of the findings of research on high-risk children is that the majority do very well. Fortunately, psychiatry is in this majority. Its basic research is among the most exciting in contemporary medicine, its diagnoses are reliable, its treatments are effective, and the stigma that has long marked both its patients and its practitioners is rapidly disappearing . . . . The battered child [of psychiatry] has been transformed by a tumultuous adolescence into a vigorous and successful young adult.[335]

Michels and Marzuk anticipate much of Shorter's account. Like Shorter, they regard biological research—not treatment or clinical practice—as the defining feature of modern psychiatry. Like Shorter, they praise modern treatments without focusing on them. And, like Shorter, they date the birth of modern psychiatry, not from the appearance of medications in 1953, but sometime later. Shorter dates the second biological psychiatry from 1970. Michels and Marzuk say the transformation occurred in "recent years."

From psychiatrists' point of view, this conception was superior to the idea of a medication revolution in a number of respects. An old revolution is hardly something to brag about in science or medicine, and medications had appeared forty years earlier. Moreover, medications had also lost much of their luster since the 1970s. The very research that Michels and Marzuk touted had demonstrated that medications were not a magic bullet against schizophrenia. There were also serious side effects—including fatal ones—that psychiatrists could not deny. At the same time, many people with schizophrenia remained desperately ill despite drug treatment. The public mental health system based on medications was performing badly. And though psychiatric

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Psychiatry (parts 1 & 2), 329 NEW ENG. J. OF MED. 552 & 628(1993)). For discussion of Michels' and Marzuk's views, see GELMAN, *supra* note 11, at 208-09.

332. *Id.*

333. *Id.* at 209.

334. *Id.*

335. Michels & Marzuk, *supra* note 331, at 635 (footnotes omitted).

practice had survived, the battles over tardive dyskinesia and the litigation had surely taken a toll.

Michels' and Marzuk's conception solved all these problems in a single stroke. Psychiatry's stature—indeed, its identity—now rested on brain research not on treatment, clinical practice, or public health. It followed that any problems in those realms—treatment-resistant patients and serious side effects that get systematically ignored, a public mental health disaster—should not diminish psychiatry's stature. If Michels and Marzuk were right, psychiatry had become immune from future criticism, except on account of its research. And who would, or could, take issue with that?[336]

The new conception also removed any threat from psychiatry's past. With the profession transformed into a brain science, events before the transformation—particularly events in the realms of clinical practice or public health—could have little relevance to presentday. More than just the standard conception of a revolution, Michels and Marzuk propounded a future-oriented vision, one forever focused on tomorrow's research advances. What happened yesterday—indeed, what is happening today—pales in bright light from the future. And just in case one explores the past anyway, Michels and Marzuk offer their developmental metaphor for psychiatry. The profession endured a "battered childhood" they say—remarkably, Michels and Marzuk identify *psychiatrists* as the prime victims in psychiatry's history—but it grew nonetheless into a "vigorous and successful young adulthood."[337] Nothing suggests that anyone outside the profession—patients, for example—ever suffered harm at the hands of psychiatrists. But if they did, it was, at most, a juvenile offense by the young and battered profession.

Shorter's history is best understood as a variation of Michels' and Marzuk's. Both accounts offer a triumphalist view of psychiatry—one that all but immunizes the profession against criticism. Their common features—psychiatry as primarily a brain science and research enterprise, a de-emphasis on clinical practice, and a supposed historical transformation occurring well after the appearance of medications—are described above. Their differences involve events *before* the recent transformation of the field. For Michels and Marzuk, what happened was the profession's "battered childhood." For Shorter, what happened was the first biological psychiatry, the fall from grace represented by psychoanalysis, and the series of treatments that "seemed to work." Michels and Marzuk would as soon forget psychiatry's past, seeing no

336. One answer to this not entirely rhetorical question is "David Healy." Healy faults research pertaining to "atypical" antipsychotics and antidepressants and laments the influence of drug companies on psychiatric research. HEALY, *supra* note 77; for examples of Healy's views, see *supra* text accompanying notes 77-78, 89-91, 192.

337. *See* Michels & Marzuk, *supra* note 331, at 635.

point in dredging up old memories. Shorter, on the other hand, embraces psychiatry's past and its treatments.

Shorter is a historian. Unlike Michels and Marzuk, who are psychiatric researchers, if Shorter writes off psychiatry's past, he writes off his own profession. Nor can historians remain true to their vocation and focus, as Michels and Marzuk suggest, on the future. Thus, Shorter had to take psychiatry's past seriously. At the same time, he did not have to view it as he did or adopt so much of Michels' and Marzuk's triumphant account.

In fact, Shorter offers an even more triumphant account than Michels and Marzuk do. They glory in psychiatry's present and future by dismissing its past. But Shorter finds glory in that past as well. Except for psychoanalysis and antipsychiatry, he sees no occasion for anything but pride in what happened. In my view, his contribution is noteworthy precisely because it goes farther in this respect than other recent apologies for psychiatry.

Imagine two versions of a nation's, rather than a profession's, history. Questionable episodes and worse dot that nation's past. But one view of the nation ignores its past on the ground that the country has undergone a transformation and that its future is bright. Beyond a vague reference to the nation as a victim ("battered national childhood"), the past virtually disappears from this account. The other account, in contrast, does not overlook the past. Instead, it glories in what happened. This second account either selectively ignores questionable episodes, or explains them away on the ground that whatever the nation did, represented the best "alternative" at the time. Questionable episodes get attributed to the nefarious actions of an "anti" group opposed to everything the nation holds dear.

The first account, which parallels Michels' and Marzuk's, would justify great freedom of national action. It says, in effect, that the nation has much to offer, that it has a destiny, and that it will do the right thing. The second account, which parallels Shorter's, justifies even greater freedom of action. It says, in effect, that the nation virtually always does the right thing— even when it might appear otherwise—and that the nation is beset by mortal enemies. From that, it is only a short step to the conclusion that whatever the nation does, it is right.

The same kind of implications, I believe, inhere in these accounts of psychiatry. Michels' and Marzuk's version suggests that psychiatrists should have great freedom of action, which would mean great freedom from legal regulation. (It also means that psychiatrists should have the necessary financial resources.) Shorter's version suggests the same things but more strongly. If virtually nothing warranted criticism in psychiatry's past, what could possibly deserve criticism in its future? Someone who refuses to even concede psychiatry's "troubled childhood"—and Michels and Marzuk surely did not mean that the "trouble" was caused by psychoanalysts and antipsychiatrists—will probably admit to no faults whatsoever, regardless of what happened in

the past or might happen in the future. Portraying psychiatry's past as he does, Shorter does not need a revolution.

V. CONCLUSION

This brings us back to our starting point—the law. Given Shorter's assumptions about the autonomy of psychiatry—his idea that psychiatry develops independently of any social, political, or legal influence—it is hardly surprising that he says virtually nothing about legal matters. Shorter does observe that ECT faced legislative and judicial restrictions in the 1980s, which he blames on patients' rights groups. The story has a happy ending, however, because ECT made a comeback in the 1990s, gaining increasing professional support.[338] Shorter drops the legal thread in that story, but one gets the sense that the law only reacted to antipsychiatric agitation—again, the autonomy of psychiatric developments—and that the legal mistake was short-lived.

Shorter also describes an early 1980s lawsuit against Chestnut Lodge, a private mental hospital that favored psychoanalytic treatments.[339] A patient brought a malpractice action against the hospital because doctors had *failed* to treat him with medications.[340] This litigation attracted enormous attention from psychiatrists before being settled.[341] Shorter rightly observes that, despite the lack of definitive court ruling, clinicians who treated patients without drugs now realized that they "ran the risk of incurring heavy [legal] penalties."[342] He is also right that the case pushed psychoanalysis further from the medical mainstream. Since the law conformed to psychiatric developments in this instance—at least, as those developments are portrayed by Shorter—the case comports with Shorter's ideas about the autonomy of psychiatry. I said at the outset that the law and the writing of psychiatric history were related in two ways. Despite its negligible attention to legal matters, Shorter's history illustrates these relationships.

The first connection involves the motives for writing about psychiatry's past. Legal concerns—political and social concerns as well—lie at the heart of many histories, even when law barely gets mentioned. Histories like Shorter's represent extended arguments for psychiatry remaining free from legal and social regulation. The implicit argument is: Why should the law interfere when things are going so well? The law may as well mandate an orchestra violinist play better as dictate to a psychiatrist—at least if psychiatrists perform in the way Shorter says they do.

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338. SHORTER, *supra* note 2, at 285.
339. *Id.* at 309 (citing accounts of the case, which is unreported).
340. *Id.*
341. *Id.* at 309-10.
342. *Id.* at 310.

At the present moment, the second connection between law and psychiatric historiography is even more significant. The triumphant claims of Shorter and others are possible only because courts and laws failed to control —and barely acknowledged—psychiatrists' excesses during the medication era. The amount of unnecessary suffering by patients during this period is beyond calculation as is the affront to patient dignitary occasioned by psychiatrists' systematic denials of drug-caused harms. As millions of patients suffered permanent damage, psychiatrists acted—and today historians write—as though nothing of importance happened. George Crane described psychiatrists' performance during the medication era as a professional default without precedent in the history of medicine, and I have no reason to disagree.

Had the law held psychiatrists to account, histories like Shorter's would be inconceivable. He portrays a period of unprecedented professional default as an era of medical triumph. As noted already, Shorter even dates this triumph—the emergence of the second biological psychiatry—to 1970, the very time when Crane was being reviled for calling attention to tardive dyskinesia. Only because of the default of the legal system—its failure, for whatever reason, to document the problem and intercede—are the claims of historical triumph taken seriously. Indeed, they are regarded as self-evident.

Although the courts have spoken on these subjects, it is worth considering the natural of effect of a history like Shorter's on judicial deliberations. For a variety of issues affecting patients' rights, the U.S. Supreme Court has decided that courts should "defer" to the professional judgment of psychiatrists. Yet what of the Court's decision to adopt a deferential stance in the first place? In part, it must have rested on a decision that psychiatry warrants deference. Were psychiatrists performing wholesale lobotomies, for example, it is unlikely that courts would defer to the profession.

If Shorter is right, an extremely deferential stance by courts is proper. With psychiatrists performing well, legal interference is uncalled for. As noted already, however, Shorter's judgments do actually rest on a serious assessment of psychiatry's performance. Instead, his judgments of particular events are dictated by the premise that psychiatrists do not commit systematic wrongs.

Interestingly, and I think not coincidentally, the U.S. Supreme Court has taken an approach like Shorter's in two pivotal cases. A 1982 decision, *Romeo v. Youngberg*,[343] announced the "deference to professional judgment" standard.[344] In *Romeo*, the Court observed that a deferential test was necessary "to enable [psychiatric] institutions . . . to continue to function."[345] Here, the

343. 457 U.S. 307 (1982).
344. *Id.* at 323.
345. *Id.* at 324.

opinion was referring to state institutions for persons with developmental disabilities but the general point is the same as Shorter's—psychiatric autonomy from law. The Court did not reach its conclusion based on any perceived successes of psychiatric institutions. Indeed, the institution in question was notoriously bad. Rather, the Court was deferring to psychiatry, not because of its treatments but just because it was psychiatry—Shorter's, Michels', and Marzuk's point exactly.

The second case is *Washington v. Harper*,[346] a 1990 decision upholding forced medication of prisoners. Reaching that result, the Justices did not hesitate to describe the side effects of treatment, including tardive dyskinesia,[347] nor did the Court assert that medications reversed any disease processes in the brains of prisoners. "The purpose of drugs,"[348] Justice Kennedy wrote, "is to alter the chemical balance in a patient's brain, leading to changes, intended to be beneficial, in his or her cognitive process."[349] Leaving out "chemical balance" phrasing, Justice Kennedy could have written the same thing about lobotomy. Leaving out lobotomy—and only lobotomy—Shorter could have written the same thing about any biological treatment in psychiatry.

346. 494 U.S. 290 (1990).
347. *Id.* at 229-30.
348. *Id.* at 229.
349. *Id.*