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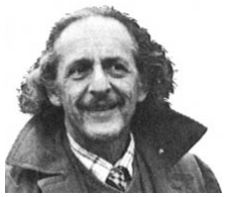

What Do We Know about the Group of Mental Disorders Called Schizophrenia? Part 2: Diagnosis and Treatment

Number 27 July 4, 1983

In the first part of this essay,[1] I reviewed several current theories on the causes of schizophrenia. Schizophrenia is one of the psychoses, or major mental illnesses involving disordered thoughts, emotions, and behavior. About ten million people worldwide suffer from schizophrenia, and between one and two percent of the US population will be diagnosed as schizophrenic in their lifetimes. Despite its prevalence and more than 50 years of intensive research, schizophrenia still is a poorly understood disease.

Part of the problem is that "schizophrenia," like "cancer," denotes a group of diseases, each of which may have different causes and outcomes. Researchers and clinicians have defined various criteria for diagnosing schizophrenia, but they differ on exactly what symptoms are more or less relevant to the disease. An individual might be labeled schizophrenic according to one diagnostic system, but not according to another.

This confusion frustrates the efforts of scientists studying schizophrenic patients. Experimental results reported on one group of schizophrenics may be difficult to confirm in another group diagnosed according to a different system. Disagreements on diagnosis also complicate the clinician's decision on how to treat schizophrenics. For example, a drug therapy successful in treating some patients may be ineffective in others. Many of the drugs used to treat this disease have serious side effects, and they should be prescribed only for those who will benefit. In this essay, I'll discuss how schizophrenia is diagnosed and treated.

Current diagnostic views of schizophrenia are based on the work of two European psychiatrists in the early 1900s. Emil Kraepelin, a German psychiatrist, considered the following to be characteristic symptoms of schizophrenia: lack of insight, emotion, and will; confusion; loosening of associations; deteriorated mental ability; hallucinations; delusions; and disturbed motor functioning, or catatonia. He also stressed a progressive deterioration, or "dementia," as central to the disease process.[2-4]

Eugen Bleuler, a Swiss psychiatrist, agreed with Kraepelin that loosening of associations and the "splitting" in thought processes and emotions from reality were primary symptoms of schizophrenia. But he regarded hallucinations, delusions, negativism, and catatonia as subordinate symptoms that were not necessary for a diagnosis of schizophrenia. He also rejected any consideration of the outcome of the disease—not all schizophrenics were inevitably demented, in his experience.[2-4]

Current diagnostic criteria carry on the Kraepelin-Bleuler debate as to which symptoms are primary to schizophrenia, and whether or not to take into account the course of the illness. Diagnoses based only on the present mental status of the patient are called "cross-

sectional." Those that also take into account the course of the illness and other factors, such as family history and the patient's condition before the onset of schizophrenia, are called "longitudinal" diagnoses.[5]

In 1959, Kurt Schneider, a German psychiatrist, provided the first set of detailed symptoms on which to make a cross-sectional diagnosis of schizophrenia. He described 11 "first rank symptoms" (FRS), any one of which was sufficient for diagnosing a patient as schizophrenic in the absence of neurological disorders, such as retardation or brain disease.[6] The first three deal with auditory hallucinations: voices repeat or anticipate the patient's private thoughts; they discuss or argue about the patient; or they keep up a running commentary on the patient's actions. Three more symptoms involve bizarre thinking: the belief that one's thoughts are removed by some outside force; that one's thoughts are magically broadcast for others to hear; and that thoughts are forcibly inserted into the patient's mind. Another three symptoms relate to the patient's experience of his or her feelings, acts, and impulses being under the control of some external agency, as if the patient were a robot or hypnotized. The patient also feels that he or she is a passive recipient of various sensations—heat, touch, or movement—imposed by outside forces. Finally, the patient's perceptions are deluded—what we would perceive as commonplace occurrences, the schizophrenic interprets as having a very special or profound personal significance.[2,4]

Schneider's FRS are still regarded by many clinicians as the definitive criteria for making a diagnosis of schizophrenia. They are commonly used in Europe, and had a significant impact on British researchers and clinicians in particular.[7] But studies show that FRS occur in about 50 percent of clinically diagnosed schizophrenics. That is, half of the patients labeled schizophrenic do not show any FRS. Also, FRS occur in other psychiatric disorders, especially mania, so they are not exclusive to schizophrenia.[5]

Other cross-sectional diagnostic systems are more or less based on Schneider's FRS, but they include other symptoms as well. For example, the New Haven Schizophrenia Index (NHSI),[8] developed in 1972 by B.M. Astrachan and colleagues, Yale University, New Haven, Connecticut, lists more than 20 symptoms, including visual hallucinations, confusion, catatonia, suspiciousness, and other paranoid signs. The flexible system,[9] designed in 1973 by W.T. Carpenter and J.S. Strauss, National Institute of Mental Health (NIMH), Bethesda, Maryland, itemizes 12 relevant symptoms of schizophrenia, including poor rapport and insight, incoherent speech, and absence of early waking, elation, and depression. The presence of either five or six symptoms are necessary for a diagnosis of schizophrenia with the flexible system. The CATEGO system,[10] a computer program used to process data from the Present State Examination,[11] was developed by J.K. Wing and colleagues, Medical Research Council, London, in 1974. It allows for *six* separate diagnoses: definite or uncertain schizophrenia; definite or uncertain paranoia; and definite or uncertain "other" psychoses, such as simple and catatonic schizophrenia.

At the same time that these cross-sectional diagnostic systems were evolving, other researchers and clinicians developed a longitudinal approach. In addition to considering present symptoms, they examined the patient's past history—whether or not other relatives were schizophrenic, the patient's work or school performance before the onset of the disease, the age at onset and dura-

tion of the illness, and so on. Their goal was to distinguish between "true" or chronic schizophrenics and patients with an acute form of the disease. Chronic schizophrenics, who develop symptoms earlier in life and over a longer period of time, have a poor prognosis. That is, there is little chance that they will recover. But acute schizophrenics, who develop symptoms suddenly, often recover spontaneously.

The *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-III*)[12] is the most current example of a longitudinal diagnostic system. *DSM-III* is a standard reference work in the US, and was published in 1980 by the American Psychiatric Association. It evolved from two earlier longitudinal diagnostic criteria: the Feighner criteria,[13] defined in 1972 by J.P. Feighner and colleagues, Washington University, St. Louis, Missouri; and the research diagnostic criteria (RDC),[14] developed in 1975 by R.L. Spitzer and colleagues, NIMH.

DSM-III draws a distinction between three classes of symptoms. "Prodromal" symptoms may develop before the onset of schizophrenia. They indicate that the individual's previous level of functioning is deteriorating—social withdrawal, personality changes, eccentric behavior, neglected hygiene and grooming, and diminished performance at work or school are listed as prodromal symptoms in *DSM-III*.[5,15,16]

"Active" symptoms indicate the psychotic phase of schizophrenia. Again, they are derived from Schneider's FRS—delusions, hallucinations, and thought disorders. Any one of these symptoms is necessary for a diagnosis, but thought disorders alone are not sufficient. Thought disorders—incoherence, loose associations, and poverty of speech, for example—must occur with delusions or hallucinations, catatonic behavior, or flat or inappropriate emotions for a *DSM-III* diagnosis of schizophrenia.[5,15,16]

"Residual" symptoms may persist after the active phase of schizophrenia. They are similar to the prodromal symptoms, but emotional flattening and impaired role functioning are more common in the residual phase.

In addition to these symptoms, *DSM-III* requires several longitudinal factors: onset before age 45 and continuous illness for at least six months, for example. Also, a diagnosis of schizophrenia is not allowed if the patient has a history of manic or depressive disorders, or brain disease and retardation.

As you can see, there is considerable room for disagreement on exactly what schizophrenia is and how it can be diagnosed. I should point out that I haven't discussed several other diagnostic systems for reasons of space—those of G. Langfeldt[17] and M.A. Taylor and R. Abrams,[18] for example. But there is no reason to believe that any one system is "better" than another.[5] The situation would be considerably improved if diagnoses were based on biological "markers" of schizophrenia.[15] In Part 1 of this essay, I reviewed current research on several biological causes of the disease—hypersensitive dopamine receptors, structural and physiological abnormalities in the brain, and so on. Researchers and clinicians may eventually be able to diagnose subtypes of schizophrenia according to these biological factors.

The vast majority of patients diagnosed as schizophrenic are treated with a group of drugs called "neuroleptics." It is important to understand that neuroleptics do not *cure* schizophrenia. They simply reduce the more obvious symptoms—hallucinations, paranoid behavior, agitation, combativeness, sleep disturbance, and so on. But neuroleptics have little or no effect on other symp-

toms—impaired judgment, withdrawal, lack of insight, and poor motivation, for example. In addition, neuroleptic drugs reduce the risk of relapse. Two out of three patients who discontinue drug use reexperience schizophrenic episodes within one year. Drug maintenance reduces the relapse rate to between 20 and 30 percent.[19]

There are five categories of neuroleptics, classed by chemical structure. The phenothiazines include chlorpromazine, marketed under the trade name Thorazine; triflupromazine or Vesprin; mesoridazine or Serentil; thioridazine or Mellaril; fluphenazine or Prolixin and Permitil; perphenazine or Trilafon; and trifluoperazine or Stelazine. Another group, the thioxanthenes, includes chlorprothixene or Taractan, and thiothixene or Navane. Loxapine, marketed as Loxitane and Daxolin, is a dibenzoxazepine. Haloperidol, or Haldol, is a butyrophenone. Finally, molindone, or Moban, is an indole.[20]

The neuroleptics differ in their potency, or the dose required to have an effect on schizophrenic symptoms. High potency drugs, such as Haldol, Prolixin, Stelazine, and Moban, are prescribed in low doses. Thorazine, Mellaril, Taractan, and other low potency drugs are given in higher doses. The high potency drugs are not more effective or faster acting than low potency agents—they give comparable clinical results, but with fewer milligrams per dose. Typically, it takes between ten days and two weeks for the neuroleptics to show therapeutic results, but some schizophrenics respond within two or three days.[19]

As with any oral medication in tablet or liquid form, there is the risk that patients will not comply with the doctor's prescription. I've discussed patient noncompliance in a separate essay.[21] Among schizophrenics in hospitals, noncompliance has been estimated at about 20 percent. For schizophrenic outpatients, it increases to between 40 and 70 percent. Injectable long-acting neuroleptics have been developed to treat noncompliant schizophrenics. For example, fluphenazine decanoate injections have a duration of action between three and four weeks.[19]

While neuroleptics are very effective in treating schizophrenic symptoms, they have unpleasant side effects for some patients which contribute to noncompliance. Before I discuss these side effects, let me point out that most researchers and clinicians consider the neuroleptics to be no more "dangerous" than other drugs used to treat physical disorders in general medical practice. Also, not all schizophrenics on neuroleptic therapies suffer side effects. The benefits of neuroleptics in treating symptoms of schizophrenia usually outweigh their side effects.

The short-term side effects of neuroleptic drugs include urinary hesitation, constipation, blurred vision, dry mouth, decreased blood pressure, and exacerbation of glaucoma. Thus, elderly patients and those with heart disease are more vulnerable to these side effects.[19,22]

Neuroleptic drugs also have long-term side effects for some patients that are disabling, sometimes irreversible, and even fatal. Soon after the neuroleptics were introduced in 1952, clinicians observed odd jaw movements, grimacing, and other "dyskinetic" movements in the oral region of some schizophrenic patients.[23] In 1960, this syndrome was named "tardive dyskinesia,"[24] and it has proved to be the most frequent and feared of long-term risks of neuroleptic drug therapy.[22]

The clinical signs of tardive dyskinesia include sucking or smacking of the lips, moving the jaw from side to side, puffing out the cheeks, and occasionally thrusting the tongue out of the mouth. In addition, jerking movements of the arms and

extremities, contractions of the neck and back, and writhing movements of the fingers, toes, or ankles are observed.[19,22,25] These involuntary and grotesque movements obviously are a source of distress and embarrassment for schizophrenics, and they may decide to stop taking the medications that cause them.[25]

Tardive dyskinesia usually appears about two years after drug treatment is started, but the syndrome can occur within three months. Only about 20 percent of older, chronic patients with a long history of neuroleptic medication are afflicted. The incidence is *much* lower in younger, acute schizophrenics.[19] It is most frequently observed in elderly females, in particular those with a history of brain disease.[25] When drugs are discontinued, tardive dyskinesia is reversed in about 30 percent of patients.[19]

Pseudoparkinsonism is another long-term side effect that appears weeks or months after the start of drug therapy, most frequently in elderly females. The symptoms are muscle fatigue and weakness, tremor in the hands and fingers, rigidity, drooling, and heat intolerance. Almost all cases of pseudoparkinsonism remit within a month or two after medication is discontinued.[25]

Akathisia often occurs in combination with pseudoparkinsonism, but this long-term side effect can occur alone. It is marked by the inability to sit still—restless movements, rocking back and forth while standing, and tapping the feet while sitting are observable several weeks or months after drug therapy begins. Again, akathisic symptoms disappear after drug therapy is stopped.[25]

Acute dystonic reactions most frequently afflict children and young adults, especially males, within three days after drug therapy is started. Dystonic means abnormal muscle tension—the patient suffers spasms of the face and throat muscles, curling and sticking out the tongue, grimacing and other facial distortions, and unusual posturing of the head, neck, and jaw. In addition, the patient's eyes may be fixed in an upward gaze. These symptoms come and go while drug therapy continues, but they remit about a week after drugs are stopped.[25]

The neuroleptic malignant syndrome (NMS), a potentially lethal and rare complication, was first noticed in 1960. The symptoms are apparent anywhere from a few hours to several months after neuroleptic therapy begins. Once it starts, NMS develops very rapidly over the next 24 to 72 hours. The symptoms are rigidity and akinesia, elevated temperatures as high as 110-20 degrees Fahrenheit, stupor and coma, rapid heartbeat, unstable blood pressure, and perspiration. Young adult males are predominantly affected, but NMS can strike patients of all ages and either sex.[26]

Only 60 cases of NMS have been documented in the literature. Of these, 12 patients died. The fatalities resulted from respiratory or kidney failure and cardiovascular collapse, and they occurred from three to 30 days after the onset of symptoms. The patient recovers about a week after drugs are discontinued, or two to three weeks later if long-acting injectable neuroleptics were prescribed. Interestingly, some patients were treated with drugs on several different occasions before NMS developed. Also, NMS sometimes does not recur after drug therapy is restarted. Thus, neuroleptics alone are not sufficient to trigger NMS. The patient's state of health at the time of medication may have considerable impact on the onset of NMS.[26]

The long-term side effects of neuroleptic drugs make it desirable to develop alternative treatment strategies. Also, you should remember that 25 percent of schizophrenics do *not* respond to neuro-

leptics.[27] Propranolol, a drug used to treat high blood pressure and migraine headaches, has been used in combination with the neuroleptic chlorpromazine.[28] But more controlled studies on larger populations of schizophrenics are needed to test the efficacy of propranolol, and to see what its long-term side effects are.

Lithium is another drug used in combination with neuroleptics to treat schizophrenics who do not respond to neuroleptics alone. E. Fuller Torrey, St. Elizabeth's Hospital, Washington, DC, says, "Lithium is being used quite widely as an ancillary drug in schizophrenia. Lithium is well established in the bipolar [depressions], but many are starting to use it on treatment resistant schizophrenics in conjunction with antipsychotics. Its use is spreading quite quickly."[29] I previously reviewed the literature on lithium as a treatment for depression.[30]

I should point out that large doses of vitamins have been suggested as a treatment for schizophrenia for more than 20 years now.[31,32] But recent controlled studies have not supported the claims that megavitamin therapy is useful in treating this disease. Torrey concluded, "Unfortunately, this therapeutic regimen has not been shown to be effective in controlled studies of schizophrenics. It is possible that it may be effective for some subgroups, but no evidence has been offered to date."[33]

The endorphins also have been tested as a treatment for schizophrenia. When beta-type endorphins were used on chronic schizophrenics, no clinically significant improvement in symptoms was observed.[34] In fact, symptoms worsened when beta-endorphin was tested on acute schizophrenics.[35] This finding suggests that too much of these opiates in the brain might actually be a cause of schizophrenia. Thus, reducing or blocking the effects of endorphins might improve symptoms. Several studies tested the effects of naloxone, an opiate antagonist, on schizophrenics. But the balance of research results indicates that naloxone has little or no effect on this disease.[36,37] I recently discussed naloxone in a separate essay on Jack Fishman and Harold Blumberg, who won the 1982 John Scott Award for their synthesis and investigation of this drug.[38]

More recently, gamma-type endorphins were studied for their effects on schizophrenics who did not respond to neuroleptics. Half of the patients tested showed clinical signs of improvement. More intensive research on gamma-endorphins is needed. If it proves successful in treating some schizophrenics, the lack of side effects would give it a distinct advantage over neuroleptic medication.[39]

Interestingly, research on endorphins and schizophrenia has revived the controversial practice of treating psychiatric patients with hemodialysis. Hemodialysis is used on patients whose kidneys have failed in order to clear the blood of toxic substances. In 1977, an endorphin was reportedly found in excess amounts in the blood of schizophrenics.[40] This finding, combined with anecdotal evidence that schizophrenic symptoms improved after hemodialysis, suggested that dialysis might be a useful treatment strategy.[41] But recent controlled studies have shown dialysis to be ineffective in treating schizophrenia.[42-44]

One of the earlier treatments for schizophrenia was electroconvulsive therapy (ECT). In fact, the first patient treated with ECT, in 1938, was a schizophrenic.[45] Chronic schizophrenics or patients who have been ill for more than two years do not respond well to ECT. However, acute schizophrenics, especially those with catatonic symptoms, benefit from ECT—they are discharged from the hospital earlier, and their self-care behavior improves. The number of

schizophrenics treated with ECT who improve has been reported at between 40 and 80 percent. But relapse rates are high.[45,46] I've previously discussed the use of ECT for schizophrenia and other mental disorders.[47]

Like many patients with major psychiatric disorders, schizophrenics are also treated with psychotherapy. Studies comparing the effectiveness of psychotherapy to other treatment strategies show that it is most useful in combination with drugs and/or ECT. Schizophrenics treated with ECT, drugs alone, or drugs and psychotherapy spent less time in the hospital than those undergoing psychotherapy alone. Psychotherapy designed to support and rehabilitate the patient's social skills was more effective than psychotherapy aimed at improving the patient's insight and understanding of the psychodynamics of schizophrenia.[48]

Other forms of "talk therapy" include the schizophrenic's family in the treatment plan. Family treatment programs evolved in response to research showing that stress in the home environment increases the rate of relapse in patients. Family members who have to cope with the emotional demands of dealing with a schizophrenic relative can be critical of, and hostile toward, the patient. Or they feel somehow guilty for the patient's condition and are overanxious and protective of the schizophrenic. About 50 percent of patients who return to "high-stress" home environments relapse within the year. The relapse rates improve significantly when patients continue taking medication *and* spend less time with high-stress family members.[49]

However, spending less time with critical or emotionally overinvolved family members often is not a practical alternative for the schizophrenic. Thus, researchers have tried to reduce the stress levels in the home environment through family counseling. The program involves educating the patient and family about schizophrenia and the use of medications. They are also trained in various methods of problem solving. For example, the family learns to be more positive and less critical of the patient. They are also taught various strategies to cope with difficult situations as they arise. In addition, the patient is encouraged to become more independent of the family, and to develop and maintain social skills and outside interests.[50]

Studies show that family treatment plans are a successful therapeutic approach to schizophrenia. Relapse rates are significantly reduced, as are the number of days spent in the hospital. Also, better compliance with drug treatments is associated with family management.[51,52]

Unfortunately, not all families of schizophrenics are willing to accept the patient after he or she is discharged from the hospital. Some schizophrenics simply may not have a family to return to. The problem of where to place schizophrenic patients has become a major concern over the last two decades. Since the early 1960s, state mental hospitals in the US have discharged hundreds of thousands of psychiatric patients. Part of the reason is due to the introduction of the neuroleptic drugs. Since they treated the symptoms so dramatically, it was felt that the patient's life would be improved if he or she returned to the community instead of being confined to the hospital ward.[53,54]

Also, there was growing pressure to develop more humane community-based treatment programs. The spirit of reform resulted in passage of the Community Mental Health Centers Act in 1963.[55] Although the act made provisions for a network of counseling and outpatient services, they were not fully implemented. Many mental patients were simply "reinstitutionalized" in nursing homes, homes for the aged, or

"welfare hotels" in the poorest sections of the city. Medical and psychiatric care was inadequate or nonexistent, and the discharged mental patients often wound up on the streets.[53,54] I've discussed the increasing phenomenon of street people and "bagladies" previously.[56]

In contrast, the Veterans Administration (VA) hospitals began to place their discharged mental patients in "foster homes" in 1951. Foster homes are small, private family houses in which the patient lives with at least one adult "sponsor." The patients pay for their care and are treated as members of the family. Social workers periodically look in on patients to make sure they are taking medication and to monitor their progress. The VA has placed more than 60,000 patients in foster homes since the program was started, and about 12,000 patients are currently in foster care.[57-59]

The relapse rate for schizophrenics in foster care after one year is about 36 percent. Keep in mind that chronic schizophrenics treated with drugs have a relapse rate of about 45 percent one year after discharge. Researchers compared relapsed and nonrelapsed patients in foster care to see if they differed significantly on the kinds of activities they pursued. Nonrelapsed schizophrenics spent more of their time reading, working in the garden or doing other outside activities, and caring for children. Relapsed patients tended to watch more television and movies.[59]

Several characteristics of the foster home itself were found to have an impact on relapse rates. Relapsed patients tended to come from larger foster homes with more than ten people. These homes also had more than two patients in foster care. Relapse rates also were higher if the home had no children in the family. That is, the more the foster home resembled an institution rather than a family environment, the higher the relapse rates.[58,59] In addition, relapse rates were higher when the sponsor initiated many social activities, or when the patient was closely supervised by the sponsor or social worker. Thus, when the schizophrenic was treated more as a patient than a family member, there was a greater chance of deterioration and relapse.[58]

The VA foster care program is patterned after a form of treatment for the mentally ill that dates back to the thirteenth century in Gheel, Belgium. According to tradition, the patron saint of the mentally disturbed, St. Dymphna, was murdered in Gheel by her father. In response to this tragedy, the church founded an infirmary to treat the increasing number of mentally ill people who made pilgrimages to her tomb in search of a miraculous cure. When the number of patients overflowed the wards, the townspeople began to accept them into their homes as members of the family.[60]

Today, patients are placed with families from similar social and economic backgrounds. They are given their own room and have their meals at the family table. No more than two patients are placed in one home. They are free to come and go as they like, and can even get a job. Many of the patients have been with their families for 20 or more years. The townspeople treat the mentally ill as friends and neighbors. This is in contrast to most people in the US, who are suspicious or fearful of the mentally ill and avoid contact with them.[60]

The most difficult task in successfully treating schizophrenia may be to change our attitudes toward the mentally ill. Several groups in the US have been organized to improve the public image of the mentally disturbed. In addition, they help families cope with the pressures of caring for afflicted relatives, advocate the patients' rights to treatment and social services, and lobby for more

research funds. Many of them are local or statewide organizations, while others have joined to form national coalitions.

I can't discuss them all in detail, but Table 1 provides a partial list of family self-help organizations. The National Alliance for the Mentally Ill, shown in Table 1, is a coalition of about 200 family and friends groups. You can contact them for more information on these organizations in your part of the US. *The Caring Family*,[61] a book written for families with relatives who have emotional and psychological problems, lists more than 100 self-help organizations. Another book by Torrey, *Surviving Schizophrenia*,[62] which will be published in the near future by Harper & Row, identifies about 300 self-help groups.

Table 1: A partial list of private organizations providing self-help, treatment, emotional support, and/or advocacy on behalf of the mentally ill.

Alliance for the Mentally Ill
P.O. Box 1502
Madison, WI 53701
(608) 255-1695

American Schizophrenia Association
Huxley Institute for Biosocial Research
219 East 31st Street
New York, NY 10016
(212) 683-9455

Community Mental Health Organization, Inc.
93 West Palisade Avenue
Englewood, NJ 07631
(201) 567-0500

Families and Friends of the Adult Mentally Ill
980 6th Street
Boulder, CO 80302

Families Unite for Mental Health
P.O. Box 126
Oreland, PA 19075
(215) 572-1394

Mental Health Advocates Coalition
of Minnesota, Inc.
265 Fort Road (W. 7th St.)
St. Paul, MN 55102
(612) 222-2741

National Alliance for the Mentally Ill
1234 Massachusetts Avenue, NW
Suite 721
Washington, DC 20005
(202) 783-6393

Parents of Adult Schizophrenics
San Mateo County
P.O. Box 03333
San Mateo, CA 94403

Project Overcome
1900 Hennepin Avenue
Minneapolis, MN 55403
(612) 874-7600

Schizophrenia Association of Greater
Washington, Inc.
Wheaton Plaza Office Building North #404
Wheaton, MD 20902
(301) 949-8282

As you can see from this two-part essay, research on schizophrenia represents a broad field with many specializations—etiology, diagnosis, treatment, etc. In Part 1, I listed the *ISI/BIOMED*® [63] research fronts on the causes of schizophrenia. Table 2 shows the 1981-1982 *ISI/BIOMED* research fronts on the diagnosis and treatment of schizophrenia, and the number of core and citing papers in each. Research fronts are formed when a group of current papers cite one or more articles identified as core for that topic.

I've included both 1981 and 1982 research fronts in Table 2 to demonstrate the wealth of literature you can retrieve from the *ISI/BIOMED* data base. For example, if you are interested in the diagnosis of schizophrenia you can retrieve 159 current papers that cited the core literature on that topic. In 1981, 118 papers cited the core documents in research front #81-0323, "Diagnosis of schizophrenia." In 1982, 41 citing papers were included in research front #82-1534, "Diagnosis of schizophrenia and catatonia."

You should keep in mind that the core publications in these two research fronts do not overlap. Table 3 lists the core literature in the 1981 and 1982 *ISI/BIOMED* research fronts on diagnosis of schizophrenia. It's interesting to note that the core documents in the 1981

Table 2: *ISI/BIOMED®* research fronts on the diagnosis and treatment of schizophrenia. A=research front number. B=research front name. C=number of *core* papers in the research front. D=number of *citing* papers in the research front.

A	B	C	D
81-0323	Diagnosis of schizophrenia	4	118
81-0938	Social approaches to schizophrenia	2	67
81-1516	Des, tyrosine and gamma-endorphin in the treatment of schizophrenia	3	84
81-1722	Antipsychotics and schizophrenia	2	40
81-2122	Diagnosis, treatment and social adjustment for schizophrenia	2	70
81-2198	Studies of schizophrenia therapy	2	37
81-2669	Depression symptoms in schizophrenia	3	49
81-3078	Propranolol, chlorpromazine and other beta-blockers in schizophrenia	2	40
82-0628	Clinical studies and behavioral pharmacology of the endorphins; treatment of schizophrenia	39	254
82-1110	Prediction of response to pimozide therapy for schizophrenia based on response to treatment with dextroamphetamine	8	108
82-1534	Diagnosis of schizophrenia and catatonia	2	41
82-1718	Family management and social intervention in schizophrenia patients	5	54
82-1951	Effects of antidepressant drug therapy on dopamine receptors, uptake and metabolism of dopamine and on noradrenergic synaptic mechanisms, and the use of dopamine agonists in the treatment of schizophrenia	3	26
82-2380	Social factors in the diagnosis of schizophrenia, affective disorders, major depressive disorder and other psychiatric disorders	2	184
82-3536	Systems of diagnosis, types and prognosis of schizophrenia	2	19

Table 3: Core documents in *ISI/BIOMED®* research fronts on the diagnosis of schizophrenia. A=research front #81-0323, "Diagnosis of schizophrenia." B=research front #82-1534, "Diagnosis of schizophrenia and catatonia."

A

Astrachan B M, Harrow M, Adler D, Brauer L, Schwartz A, Schwartz C & Tucker G. A checklist for the diagnosis of schizophrenia. *Brit. J. Psychiat.* 121:529-39, 1972.

Carpenter W T, Strauss J S & Bartko J J. Flexible system for the diagnosis of schizophrenia: report from the WHO International Pilot Study of Schizophrenia. *Science* 182:1275-8, 1973.

Carpenter W T, Strauss J S & Muleh S. Are there pathognomonic symptoms in schizophrenia? An empiric investigation of Schneider's first-rank symptoms. *Arch. Gen. Psychiat.* 28:847-52, 1973.

Schneider K. *Clinical psychopathology.* New York: Grune & Stratton, 1959. 173 p.

B

Bleuler E. *Dementia praecox or the group of schizophrenias.* New York: International Universities Press, 1950. 548 p.

Kraepelin E. *Dementia praecox and paraphrenia.* Huntington, NY: Krieger, 1971. 331 p.

research front represent some of the more recent diagnostic systems—NHSI of Astrachan and colleagues,[8] Carpenter and Strauss's flexible system,[9] and Schneider's FRS.[6] But the core books in the 1982 research front are the work of Bleuler and Kraepelin, first published in the early 1900s. The frequent citation of these old sources by current researchers may indicate that real breakthroughs in the diagnosis of schizophrenia are yet to come.

I've pointed out several times that we still don't know what schizophrenia is or how to cure it, even after a half century of intensive research. But there is no reason to feel helpless or pessimistic about the schizophrenic's plight. It has been estimated that between 20 and 30 percent of schizophrenics recover completely, and another 40 percent improve significantly.[64] In the near future, there may be medical breakthroughs that reach chronic schizophrenics who do

not respond to drug therapy. Even though they may have little chance of being cured, we can at least improve the quality of their lives by being accepting and compassionate. More tolerance and understanding might create the atmosphere needed to gain support in Congress for basic research to help unlock the puzzle of schizophrenia.

* * * * *

My thanks to Linda Cooper and Alfred Welljams-Dorof for their help in the preparation of this essay.

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