Chapter 1

The Psychobiology of Schizophrenia

Biological and scientific information about schizophrenia is included in a book on a psychosocial treatment for two reasons. First, the psychoeducational and family behavioral approaches are based on the core concept that schizophrenia is a biologically based vulnerability to stress, manifest as positive symptoms and cognitive deficits. All clinicians who work with people who have schizophrenia should have a working knowledge of that biology, in order to understand symptomatic behavior and to focus treatment efforts on specific deficits. A basic grasp of what the brain does and how brain abnormalities affect functioning makes the work not only more effective but also more satisfying. Second, this treatment approach requires that all clinicians understand psychiatric disorders well enough to educate those individuals' families. Much of this material is shared with families, so that they, too, can base their interactions and plans on a solid, empirically based, and relatively consistent body of knowledge.

The information presented here is relatively new, and it is constantly in flux and under development. Some of it is still controversial. Most critically, few findings apply to every person with the disorder. In fact, research now suggests that it is likely that there are several diseases that cause the disorder. Thus, schizophrenia is looking more like diabetes or mental retardation: a serious illness mediated by psychosocial stresses and caused in large part by a host of biological disorders of several types. With that caveat, however, an increasingly consistent picture is evolving across studies and is increasingly confirmed by rigorous empirical studies.

A BRIEF HISTORY OF THE CONCEPT OF SCHIZOPHRENIA

Concepts of schizophrenia and psychosis have changed over time in ways that mirror the philosophical and social predilections of the respective eras that spawned them. That is a problem for persons suffering this disorder and their families. For at least two generations in the United States, most clinicians have assumed that mental difficulties stem from the experiences that one has in one's family in the first few years of life and/or from the intrapsychic conflict that ensues. This view, of course, began with Freud and some of his earlier writings on paranoid psychosis, and was amplified repeatedly by American clinicians and psychological theorists. These assumptions were challenged by empirical and clinical evidence that conventional individual and family therapies are not effective in alleviating symptoms or improving the course of the disorder. Those theories and therapies are being replaced as the study of schizophrenia shifts from phenomenological and theory-driven methods to scientific methods, with dramatic and often surprising results. The psychoeducational approach is part of this larger enterprise. The dominant paradigm, supported by tens of thousands of research reports, is that schizophrenia is a disorder of brain function and, in many cases, the result of major alterations in normal brain anatomy.

With that biological understanding as a given, this family supportive approach arose largely from experimental evidence that clarified the role that family interaction plays in relapse and recovery, and the devastating impact that the disorder has on families. Because the illness itself leaves its victims exquisitely sensitive to sensory stimulation and cognitive overload from any source, families may unintentionally stimulate the very symptoms that they are usually trying to alleviate. The psychoeducational multifamily group model is an approach that, among several other goals, intends to alter ongoing brain function by altering the psychosocial environment to fit more closely the specific needs and biological vulnerabilities of people with schizophrenia. In fact, it is assumed that clinical improvement and rehabilitation can only occur if abnormal brain functioning is compensated by psychosocial and pharmacological means.

THE CLINICAL SYMPTOMS OF SCHIZOPHRENIA

Most readers of this volume will be familiar with the diagnostic symptoms of the disorder: hallucinations, delusions, and thought disorder, accompa-

nied by functional deterioration lasting at least 2 weeks in the acute form and 6 months in a residual form.[5] These have been termed positive symptoms, because they are added to usual mental functions. While they are dramatic and lead to relapse, hospitalization, and occasionally violence, they generally respond to antipsychotic medications and in many patients tend to diminish naturally over time.[6–10]

Less commonly recognized and understood are negative, or deficit, symptoms: normal functions that are lost, usually after an acute episode, and lost more profoundly as the illness worsens over months and years. In fact, evidence suggests that these symptoms accumulate with each succeeding acute episode.[11, 12] They include anhedonia, alogia, apathy, amotivation, and attentional deficit. What is lost encompasses a broad range of human experience, including most of what is enjoyable, or at least interesting, about being conscious. In the most severe form, the deficit syndrome involves a flatness of feeling, thought, sensation, and desire that is hard for most of us to comprehend.[13] It contributes to difficulties in taking initiative or carrying out responsibilities. Since it includes loss of pleasure and sensation, actions that are actually carried out are not accompanied by internal reward. The schizophrenic attentional deficit includes a general inability to direct and maintain mental focus on external and internal objects and processes. Bleuler emphasized these negative symptoms as the fundamental disorders in schizophrenia, because they were more consistent over time and led more directly to disability, personal misery, and family and clinician distress.[14] They are what patients themselves most often complain about, especially the absence of pleasure in events, interactions, and achievements. Family exasperation can be linked to negative symptoms; family members attribute them to character flaws such as laziness or manipulativeness.[15, 16] Understanding negative symptoms accurately and gradually alleviating them are two of the central goals of psychoeducational multifamily groups. They are also important because they are now well known to reflect the more fundamental functional and structural aberrations of the schizophrenic brain. In fact, their presence is more predictive of later positive symptoms than positive symptoms themselves.[17] Thus, a scientific perspective on schizophrenia regards negative symptoms as markers of the primary disorder.

SCHIZOPHRENIA OVER TIME

The Course of Recovery from an Acute Episode

For many younger patients, alleviation of psychotic symptoms is a rapid

process, usually occurring 5 to 15 days after medication is started. However, closer analysis of risk for relapse over time[18] and of return of cognitive function[19] suggests that negative symptoms increase during the acute episode and then, if relapse does not occur, slowly decline over the ensuing 12 to 36 months. This course may be reflected in loss of nerve cells in prefrontal cortex during unstable periods and in cortical cellular breakdown early in the illness before drug therapy.[20] The implication is that tolerance for stress and readiness for rehabilitation are low after an acute episode and increase very gradually thereafter.

A Review of the Various Courses

Strauss emphasized that schizophrenia takes one of several course types over the lifetime of those afflicted. Some people have one episode, recover, and never have another, though little is known of the middle and later years of these more fortunate lives. A few begin deteriorating functionally early in childhood and remain profoundly disabled and psychotic for most of their lives. The range in between is occupied by myriad variations. The most common, statistically, is the person who has repeated episodes in early adulthood, losing functional capacities and gaining negative symptoms with each episode, often becoming chronically though mildly psychotic on a permanent basis. (Repeated relapses have become common with deinstitutionalization and the inability to guarantee that medications are used adequately and consistently enough to prevent relapse and deterioration.) However, several studies have found that about 50% of all those with schizophrenia recover to the degree that they can function independently and have few or no positive symptoms by the time they reach their fifties.[9, 10, 21] Another 25% are able to function, but with persistent symptoms. Only about 25% across these studies manifest a poorer course indefinitely.

A similarly encouraging view is now emerging with regard to the fate of persons having their first episode. It appears that the earlier the impending or emerging psychosis is recognized and the earlier it is treated, even if with low doses of medication, the better the long-term outcome.[12, 22, 23] A plausible interpretation is that the fewer days a given person spends in a psychotic state, the less chance there is for development of long-term deterioration and treatment resistance.

THE BRAIN AND ITS ALTERATION IN SCHIZOPHRENIA

Alterations in brain function have consistently been shown to be associated

with schizophrenia. By the end of this chapter, the reader will have a working knowledge of a multilevel, empirically derived model of brain function and dysfunction that can be highly useful in guiding family and patient education, adaptation to community life, and rehabilitation. This is termed the biosocial model, because it assumes reciprocal influences of both the social environment on the brain and the dysfunction of the brain on the social environment. Leaders of multifamily groups should be expert in the interaction of these seemingly distant spheres.

The Brain Stem, the Midbrain, and Their Activation Centers

We start with the brain stem and its upper portion, the midbrain. Over the course of evolution, more complex functions have generally been added by laying new structures on top of older structures. So, it is not an oversimplification to say that the most basic, though primitive, functions are mediated by the brain stem, which lies at the base of the skull, just above the spinal cord, and that the most elegant and complex functions are mediated by the prefrontal cortex, which, as the name implies, lies just behind the forehead. Likewise, during fetal development, the lower centers are organized first, while higher order centers, especially the prefrontal cortex, arise last and involve the migration forward and upward of cell groups that start life directly adjacent to the brain stem. Migrating cells maintain important physical connections, mediated by electrical and chemical activity, with cells in the structures that gave rise to them. So prefrontal cells have important connections to cells in the brain stem and in the diencephalon, especially the principal sensory integration center, the thalamus. Their functions are interrelated as well. Schizophrenia affects parts of all these structures.

The brain stem regulates the level of activation of the nervous system, primarily through the action of three neurotransmitters—dopamine, serotonin, and noradrenaline. Four small structures, composed of groups of neurons and lying near each other in the midbrain (the upper portion of the brain stem), regulate the activity of other parts of the brain using these neurotransmitters. Those neuronal groups are the ventral tegmentum, the substantia nigra (both use dopamine), the raphe nuclei (serotonin), and the locus coeruleus (noradrenaline)(see Figure 1.1). They determine the level of consciousness as well as the varying levels of activity in almost all other higher brain structures.

The dopamine system controls the level of activity in both the prefrontal cortex and in the limbic cortex and other limbic structures, including the hippocampus and the amygdala, all of which are affected in schizophrenia (discussion follows). Dopamine was the first neurotransmit-

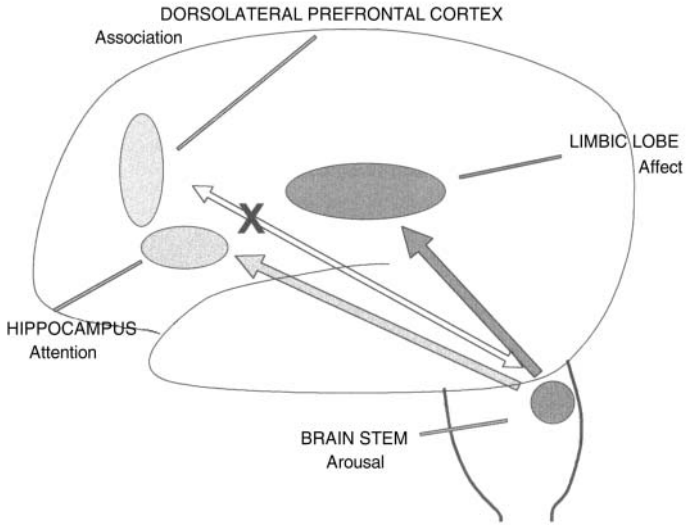

FIGURE 1.1. A simplified view of the interaction of the principal anatomical components of the dopamine system in the schizophrenic brain.

ter to be linked directly to schizophrenia, because antipsychotic drugs decrease dopamine activity. These medications mimic the molecular shape of dopamine sufficiently to occupy the receptors, thus preventing the usual activation of this system.

Thus, it should be no surprise that schizophrenia is characterized by excess levels of dopamine activity during the acute episodes and periods of clinical instability, and by abnormally low levels when negative symptoms and extreme apathy predominate.[24, 25] One effect is that the limbic system tends to be overstimulated during acute episodes. Dopamine activity normally also increases the activity of the prefrontal cortex, but this does *not* occur in schizophrenia. Usually, when the dopamine system is activated, so is the noradrenaline system, with the result that heart and respiratory rates, blood pressure, anxiety, and agitation are also increased.

It is too early in the progress of brain research to provide a clear understanding of the serotonin system and its role in schizophrenia. However, serotonin is widely distributed in the entire cortex and to many subcortical structures. It appears to inhibit the dopamine system, modulating its influence.[26] Also, in the schizophrenic dorsolateral prefrontal cortex, there are increased neurotransmitter receptors for some forms of serotonin and decreased receptors for others.[27] It is clear that drugs that block serotonin at the receptor, such as clozapine, have better negative symptom outcomes

and fewer Parkinson's disease-like side effects, reflecting complex effects on a variety of receptor subgroups and brain areas. The rest of the serotonin story is very complex and as yet far from clear.

It is now considered likely that other neurotransmitters are involved in the disorder, including gamma-aminobutyric acid (GABA), glutamate, and *N*-methyl-D-aspartate (NMDA), a subtype of glutamate. The last appears to be reduced in activity in the cortex in general, in some subcortical areas,[28] and in connections between the thalamus and the cingulate cortex, both of which are involved in processing more complex sensory information.[29, 30] Drugs that reduce activity in the glutamate system have been found in normal volunteers to induce thought disorder and negative symptoms that cannot be distinguished from those of patients on structured interviews of thinking.[31] Alterations of glutamate and/or NMDA have been found in the prefrontal and superior temporal cortex, and in the hippocampus, the areas most directly implicated in schizophrenia.[32–34] Remarkably, amino acids that regulate this neurotransmitter system, glycine and D-serine, have been found to improve positive and negative symptoms substantially for patients already taking antipsychotic medication.[35, 36] GABA precursors (messenger RNA [mRNA]) and levels of GABA itself have been found to be markedly reduced in the prefrontal cortex, reflecting reduced neuronal activity in this important region.[37] Recently integrated models for the interaction of some of these neurotransmitter systems have been proposed, explaining both biochemical findings and the differences in effects and efficacy among various antipsychotic drugs.[29, 32, 38–40]

The key concept is that the midbrain is impaired in its ability to adjust the activation of the brain and nervous system in ways that are normal or appropriate to the situation at hand. Although the means by which this adjustment occurs are unclear, the brain under normal circumstances can very precisely change its activation to match the environmental, social, or internal demands imposed by exercise, infection, metabolic imbalance, and cognitive and emotional input. In schizophrenia, the dopamine and norepinephrine systems are both unstable, tending toward overactivity when stress is present. This abnormal activation then radiates its effects through at least three other neurotransmitter systems to the rest of the brain in ways that are complex but that nearly always undermine mental and emotional well-being. At the other extreme, negative symptoms tend to be associated with underactivity of dopamine-driven systems,[24] as well as reduced activity on the part of activating neurotransmitters, especially GABA and the glutamate/NMDA system. Studies of the excitatory neurotransmitters—

glutamate, NMDA, and GABA—have great promise for development of new drug and perhaps nutritional treatments for this disorder.

The Limbic System and Thalamus

The next higher strata, above the brain stem, include the limbic cortex and the thalamus, as well as several other nearby structures. The limbic system is a way station among the prefrontal cortex, the thalamus, and the temporal lobe. Two of its components, the amygdala and the cingulate cortex, generate more primitive and life-sustaining affects and emotions, especially anger and fear, the classic fight-or-flight pattern of response. It is the limbic system that is most directly affected therapeutically by antipsychotic medications; that effect is mediated by dopamine blockade. The drugs alleviate labile emotions and delusional thinking, which are more extreme forms of fear and suspiciousness. The limbic system has consistently been found to be hyperactive during psychosis and underactive in conjunction with negative symptoms.

Another limbic structure of great importance is the hippocampus. This tiny, seashell-like structure mediates all short-term memory registration and many crucial components of attention, especially establishing and maintaining focus. In schizophrenic patients it has been found to be atrophied and its very cell structure disorganized.[41, 42] The effect is a partial, but consistently confirmed, disability in directing attention appropriately, focusing attention, and ignoring distracting stimuli when necessary. Furthermore, this defect creates marked difficulties with storing information long enough to be transferred for longer-term retention, resulting in subtle and erratic memory deficits.

The thalamus, which serves as the central control system for integrating sensory input, has been found to be underactive in schizophrenia, resulting in an inability to screen out sensory stimuli and a tendency for all sensory information to be experienced as excessive, inappropriately generalized, and overwhelming.[43, 44] One recent study has found that portions of the thalamus are reduced in size in schizophrenia, suggesting an anatomical basis for this impairment.[45] In schizophrenia, defects in the limbic and thalamic systems create a state of sensory hypersensitivity, combined with a tendency toward excessive levels of fear, suspiciousness, and anger.[46–48]

Disorders of the limbic, thalamic, and midbrain systems are functionally linked in schizophrenia; that is, as arousal increases in response to outside or internal sources of stimulation, attention deteriorates. As attention

deteriorates, arousal increases reactively, leading to a downward spiral that ends in hyperarousal in the entire limbic system, with resulting extreme states of primitive emotion, increasingly heightened sensory sensitivity, and severely limited attentional capacity. This cycle is termed the distraction–arousal hypothesis and is central to the psychoeducational approach.[49]

Higher Cortical Areas Affected: Prefrontal, Superior Temporal Gyri, and Postcentral Areas

Modern imaging methods (CT [computed tomography], PET [positron emission tomography], SPECT [single photon emission tomography], and MRI [magnetic resonance imaging] scans) have demonstrated in schizophrenia an increase in the size of the ventricles, the usually small channels through which flows cerebrospinal fluid from brain to spinal cord to bloodstream.[50] Increased ventricular size usually indicates loss of cerebral tissue, though it says little about the site of loss; that is, in many people with schizophrenia, living brain tissue has been replaced by spaces filled with fluid. These studies show that in many persons with schizophrenia, the cerebral cortex is reduced in size and has less total volume.[51–55] Indeed, some studies, though not all, show that the entire brain tends to be smaller on average.[56] The changes are similar in type to dementias, though far less dramatic and less progressive over time. The most pronounced changes are concentrated in prefrontal, medial and superior temporal, and cingulate cortices. The differences noted do not seem to be acute, because there is only the weakest correlation with age or number of episodes. These changes are present even at the first episode in young adults.[57] Studies of children with schizophrenia, followed into adolescence, show some enlargement of ventricles over a 2-year time span, suggesting that some degradation of brain structure may occur in the early years of adolescence in cases with a very early onset.[58] Many studies have shown correlations of ventricular enlargement with negative symptomatology and cognitive impairment.[47, 59] *This indicates, perhaps more clearly than any other single set of findings, that functional disability in some persons with schizophrenia is secondary to structural defects in the brain itself.*

PET and functional MRI scans provide an increasingly coherent picture in several studies. Most important, the prefrontal cortex has been found to be less active than normal, especially when the subject is challenged to do complex and frustrating mental tasks, such as the Wisconsin Card Sort, a test of abstracting and problem-solving capacity under social

duress. The dorsolateral prefrontal cortex appears to be the brain area that activates to accomplish the test. However, in schizophrenia, there is dramatically lower activation to the test and poor performance as well.[60] Recent work has shown that the prefrontal area is less active in proportion to the degree of negative symptoms, verbal task demands, and cognitive impairments, and in the presence of delusions, hallucinations, and stereotyped ideas.[46, 48, 61–63]

The left superior temporal area tends to be overactive in association with thought disorder, negative symptoms, and verbal tasks, even while having reduced physical volume. However, it is less active in the presence of delusions and hallucinations.[64–68] This last insight is particularly useful, suggesting that this area may be deficient in processing, inhibiting, and modulating auditory stimuli, predisposing patients to alterations and misperceptions in the auditory sphere. It is not clear how verbal hallucinations are formed, but given what is known, it may be that reduction in temporal brain tissue removes some key monitoring functions and leaves open the way to spontaneous verbal perceptions.

Another key finding is that activity is increased in the posterior portions of the cerebral cortex, in the parietal and occipital areas. These have long been known to be the processors of visual and other nonverbal sensory input, elaborating, correlating, and interpreting sensory data. A possible model is emerging: The thalamus is impaired and releases the parietal and occipital cortical areas to overprocess, underinhibit, and overreact to sensory information, creating a tendency toward relative overactivity, in turn leading to the well-demonstrated heightened sensitivity to sensory stimuli so characteristic of this disorder.

To summarize, a picture is emerging from hundreds of studies, using scanning techniques, metabolic studies and the direct examination of brain tissue and cells. Physical and biochemical abnormalities correlate with symptoms and functional difficulties. Specifically, the functional axis comprising the midbrain, the thalamus, and the limbic, superior temporal, and prefrontal cortexes is disordered and in many patients is clearly but not severely damaged, with secondary effects on the parietal–occipital/sensory cortical areas. The neurotransmitters involved in this axis—dopamine, serotonin, noradrenaline, glutamate, GABA, and some neuropeptides—tend to be deranged complexly. Dopamine in excess appears to mediate psychosis and, when decreased, mediates the deficit state, while excess serotonin that may be serving as an antipsychotic in reaction to excess dopamine activity may be deficient in some patients and in some receptor subsystems. The antipsychotic drugs act by down-regulating dopamine in the limbic

cortex and perhaps serotonin in the prefrontal cortex. However, this is a partial and, in some areas of research, confusing picture. It is sure to be revised and expanded in the near future.

THE SCHIZOPHRENIC BRAIN MANIFESTED IN PSYCHOLOGICAL FUNCTIONING

These biological abnormalities exert a major influence on the psychic state and psychological capacities of the person with schizophrenia. During periods of heightened activation and/or psychosis, arousal dyscontrol leads to pervasive anxiety and tension, often described as a sense of impending doom. Heart rate and respirations are more rapid than usual. In more extreme states, this can become fearfulness, then terror, then suspiciousness, and can end in delusional thinking and fixed delusional beliefs. Sufferers complain of difficulties focusing their attention. They say that minor distractions seem larger, and more intense and pressing than when they were well. Everyday experience becomes subject to hundreds of extraneous stimuli, which cannot be ignored, but which also cannot be processed and integrated. Psychophysiological research has explained this phenomenon by showing that the brain in schizophrenia cannot damp multiple, repeating stimuli, as can the unaffected brain, so that adaptation cannot occur as readily.[43, 69, 70] Reaction time is slowed and there are often minor degrees of difficulty with motor coordination and other "soft" neurological abnormalities.[71, 72] Perception is altered, leading to distorted and often very intense visual sensations and louder, hard-to-ignore auditory experiences. These can lead to frank hallucinations. Thinking becomes more fragmented and less under conscious control. Harrow and Quinlan demonstrated that loose associations are rather like computer output that has been cut into pieces then randomly put into a new and chaotic sequence.[73] As arousal increases, attention deteriorates, and anxiety and arousal rise further, often with psychosis as the end result.[49] This process can only be interrupted by medication or by unusually positive social support and isolation from stimuli, or preferably a combination of both.

In the aftermath of a psychotic episode, as negative symptoms predominate, there is less conscious thought altogether. It remains at a more rudimentary, concrete level, without affective meaning or expression. Motivation diminishes and *la belle indifférence* becomes the substitute for desire and concern. Capacities for problem solving, sequencing of behavior and action, planning, and even self-care are increasingly impaired. Emotional

interaction becomes bland or anxiety-provoking and engenders fearfulness or suspiciousness. The ability to recognize emotional states in others is lost, reducing the appropriateness of emotional responses.[74] All these cognitive deficits result in a significant loss of social skills and difficulties in working.[75] The end result is social withdrawal and cognitive disability that can become as enduring as it is pervasive. As stresses impose themselves, the process can begin again, traversing prodromal symptoms, mild then severe psychotic experiences, agitation and loss of behavioral control, and then on again, into the deficit state. Recent evidence strongly points to increasing negative symptoms, disability, and reduced responsiveness to antipsychotic treatment with each episode, and probably with each day spent in a psychotic state.[22, 76–78]

What cannot be forgotten in our increased understanding of the linkages between biology and psychology is that the psychotic experience also happens to an individual human being, whose unique personality and prior experience will influence how much control he or she gains over the illness. In particular, outcome will be influenced by the person's desire to regain sanity and stability, and his or her resilience in the attempt to retain and rebuild social relationships and a career. The less that psychotic symptoms and experiences totally replace the personality and erode intellectual abilities, the greater the chance that the process of recovery will not be undone.[79] Even more important influences in recovery, however, are the ability and willingness to participate in drug and other therapies, and the influence of the immediate environment, both social and physical. One of the most basic insights gained in the last two decades of research is that schizophrenia is a disorder of the capacity to tolerate, defend against, and manage sensory stimulation, negative social interaction, and the stresses of life, with its complex chain of events and demands. We turn in Chapter 2 to those influences and the research supporting that perspective.

PRESENT UNDERSTANDING OF THE CAUSES OF THE DISORDER

The causes of schizophrenia remain unknown at the present time. However, increasingly strong evidence for a variety of types of causes, from genetics to fetal viral infection, to autoimmune disorders, provide support for the view that schizophrenia can now be viewed as a neurological and/or developmental disorder, with the same kinds of causes as many other such disorders. For instance, evidence for genetic factors includes the fact that

identical twins have a high concordance for schizophrenia, at least 40% in most studies.[80–83] Recent studies using large population data sets and newer analytical methods put the variance explained by heritability at 83% and that of the environment at 17%.[84] Beyond genetics, an increasingly likely explanation for several of the documented brain abnormalities is that development of the prefrontal cortex is delayed or deranged just enough to lead to the disorder we know as schizophrenia, but not so much as to leave the person disabled intellectually or physically. In this model, events during the late first trimester through the second trimester leave the prefrontal cortex without key connections that become important in early adulthood, especially those related to higher cognitive functioning and complex information processing.[60, 85] Another proposed mechanism for these findings is that the normal pruning of cells, which occurs throughout the cortex as an integral part of brain development, is excessive and leads to the brain overreducing its cell census.[86, 87]

Several studies have correlated complications at birth with later onset of schizophrenia.[88–94] Spinal anesthesia, low Apgar scores, hypoxia, and other seemingly less serious complications have also predicted schizophrenia.[95] This was especially true for women patients[96] and for those *without* genetic risk.[97] Furthermore, there appeared to be more people born with schizophrenia during the winter.[98] Further research found that this effect was largely restricted to those without a family history of schizophrenia.[99] The implication is that an environmental factor, especially an infection prior to or just following birth, was the likely culprit. Prominent among these possible causes is infection with influenza, especially during well-identified epidemics.[89, 96, 100–102] Another among the increasingly likely causes of schizophrenia is autoimmune disease.[103, 104] Most telling is the presence of antibodies to brain cells associated with other abnormalities, suggesting an autoimmune process directly involving brain structures known to be abnormal in schizophrenia.[105] Other recently documented causes include Rh incompatibility[106] and severe malnutrition.[107]

The parallels with understanding about the causes of mental retardation are most telling. That, too, was once viewed as a single disease, but there are now over 100 known causes. If schizophrenia is similar, it will be something of an embarrassment for a field that saw psychosis as rooted in early childhood experience or family interactional aberrations. However, the reward would be that many of those causes may be treatable, and a few may be preventable.

IMPLICATIONS FOR AN OPTIMAL ENVIRONMENT

The psychoeducational approach of Carol Anderson and her colleagues at the University of Pittsburgh advanced treatment outcomes, linking a biological understanding of schizophrenia with a design for the social and physical environment that specifically compensates for many of the known vulnerabilities and deficits.[108] This approach has proven to be especially acceptable for families and patients, while proving itself to be a powerful means of fostering adaptation to community life and guiding rehabilitation. The newer psychosocial methods, including the psychoeducational multifamily group approach, assertive community treatment (see Chapter 10), and atypical antipsychotic medications are achieving a different kind of illness course in younger adults.[109] Here, episodes become more and more rare, negative symptoms decrease slowly but steadily, and functional capacities and some degree of mental liveliness and ability to work and study return over time. We explore this in more detail in Chapter 4 but summarize it here.

- To compensate for difficulty in regulating arousal, the people closest to the susceptible person can create a relatively quiet, calm, and emotionally warm environment.
- They can attempt to protect against sudden intrusions, confrontational conversations, arousing entertainment, and simultaneous and multiple kinds of sensory input.
- To help with information-processing difficulties, conversations can be shorter, less complex, and focused on everyday topics.
- Complexity in the environment and stressful life events will overwhelm cognitive capacities: These need to be protected against and buffered as much as possible.
- The optimal emotional tone is in the middle range, not intense and especially not negative, but also not overly distant, cold, or rigid.
- To compensate for delusions, family and friends can be encouraged to change the subject and not dwell on delusional ideas, but rather focus on less stressful topics.
- Sensory overload can be avoided by these same means, and also, for example, by reducing background noise, keeping light levels moderate, and having only one conversation going at a time.
- Negative symptoms can moderate with time but not under conditions of high stress: Rehabilitation should be carried out in small,

careful steps, using reductions in negative and positive symptoms as indicators of safety and success.

- There is a biological and psychological relapse recovery process that cannot be accelerated without risking another relapse or at least stalling progress toward functional recovery; slow, careful, and steady rehabilitation can achieve remarkable degrees of functional improvement without relapse.
- Time, rather than an enemy that leads inevitably toward deterioration, is on the side of recovery.
- Stresses and demands are taken seriously and steps toward recovery are paced to keep stress below the threshold for symptom exacerbation.

Multifamily groups address the many complexities and difficulties in applying these principles through ongoing problem-solving techniques that help families to use and individualize these ideas.

Our experience has shown that different families can use and understand various aspects of this information. Although the knowledge requirements for each family seem to be unique, the overriding message is universal, essential, and powerful in its therapeutic impact: This is a complex, serious, and ultimately biologically based disorder that can be ameliorated by those who know and care about the person affected when their effort is combined with optimal drug therapy and psychosocial rehabilitation.

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