# Theory and Treatment of Childhood Schizophrenia

**Théorie et traitement de la schizophrénie infantile – Theorie und Behandlung der Schizophrenie im Kindesalter – Teoria y tratamiento de la esquizofrenia infantil**

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Childhood schizophrenia is seen as an early manifestation of schizophrenia as it is recognized in adults. It is an endogenous disorder of the integration of the total organism, inherited as a predisposition which may remain latent or develop into a clinical illness by decompensation. When this occurs in childhood, it is the result of a combination of inherited tendencies and noxious or traumatic events, intrauterine or paranatal, resulting in a global disorganization in the patterning of all functions, which are seen as embryonic immaturities or maturational lags (BENDER 1967).

FRANZ KALLMANN's hereditary data and theories speak of the inheritance of a specific vulnerability for the disorder and of a complicated pattern of compensation. When confronted with a possible schizophrenic child one looks carefully in the family history for schizophrenic forebears, even if compensated and ambulatory. Often both sides of the family are affected, especially with our most severely handicapped children, and also siblings may be suspect. One looks carefully at the reproductive history of the mother concerning all of her pregnancies, as well as the pregnancy of the child involved, with regard to deviations in the mother's health in early pregnancy, the length of the pregnancy, and the early condition of the infant. These data can be compared with other pregnancies whether they result in normal or deviate offspring. One often can get some clue from the course of illness or compensation of other members concerning the prognosis of the child under treatment. This assumes we take seriously KALLMANN's second postulate, that a compensatory pattern is also inherited. Undoubtedly a home with schizophrenic parents will have some effect on a young child, though it can never cause schizophrenia or even autism, and the effect need not be unfavorable.

Since decompensation occurs in the cases of childhood psychosis or autism as the result of intrauterine or paranatal events, such children usually also show evidence of developmental defects (especially small heads), brain damage or at least minimal brain damage. It is a part of current medical thinking to assume or hope that all developmental deviate children are schizophrenic. This arises from the false concept that such conditions, if schizophrenic, are caused by psychological events or deprivation and may therefore be treated and cured by psychological and educational methods.

The real problem is whether a child with developmental problems from early infancy may have schizophrenia (determined by inherited vulnerabil-

ity) as well as the developmental problem of brain damage. One may say almost categorically that every child who has a severe developmental problem from infancy, even when schizophrenic features seem evident, has also brain damage or a gross disorder in development. For the most part these children have a bad prognosis. Nevertheless, a child with a minimal brain damage occurring paranatally, who does not have schizophrenic vulnerability, often has a good prognosis. So from time to time even a child, autistic from infancy, with minimal brain damage as the decompensation factor and no gross developmental defect, may have a favorable prognosis. It is important, however, to differentiate between such not only to treat the child adequately but also to avoid exposing parents to unnecessarily paralyzing programs of treatment and false hope.

In order to establish a proper program of treatment for a schizophrenic child it is important to understand what is primary both to schizophrenia and to minimal brain damage and what is secondary or defensive (therefore probably compensatory), and what is the natural course of schizophrenia in childhood to adulthood, and what can be modified by treatment measures.

The paranatally minimally brain damaged child shows disorganization and maturational lags usually without structural defects. Anxiety may be severe, for which neurotic defenses are a healthy response. The prognosis is often good. The potential schizophrenic child, decompensated by the same minimal brain damage, has the additional problem of a life course of schizophrenia. This may lead to the unfavorable defense of withdrawal into early autism, from which there may be no recovery, especially when it is associated with more severe pathology, which is often the case.

There is a natural course for schizophrenia in childhood when it is associated with only minimal brain damage which three decades of experience have taught us. The course is different for the two sexes, there are five to seven times as many prepuberty boys showing clinical schizophrenia as girls. The schizophrenic infant is fetal in its somatic and physiological lack of pattern, unresponsive (autistic) to contact stimuli and withdraws from distant stimuli. The schizophrenic boy lags in maturation. He is infantile, disorganized and anxious. In prepuberty he often presents a typical picture of childhood schizophrenia, at puberty his condition spontaneously evolves into a good remission, perhaps succumbing in late adolescence to pseudopsychopathic or pseudoneurotic defenses which may maintain him if he does not decompensate into an adult schizophrenic psychosis.

A few little girls may have a frank psychosis at five to seven years with a remission in latency and a pseudoneurotic or psychotic break after adolescence or during adulthood, postpartum or menopause. If we know the course to expect in our psychotic and neurotic children who have not been severely damaged in utero or paranatally, we can take advantage of critical life periods for guidance and treatment.

A program of treatment requires a theoretical understanding of primary and secondary symptoms and their meaning in the development of the child.

In an effort to quantify a test of the theory and diagnostic indications in childhood schizophrenia (BENDER and HELME), it was found that

1. Childhood schizophrenia is characterized by a disturbance in the regulation of maturation processes at both the biological and psychological levels.
2. The dominance of tonic-neck-reflex motility is a particularly striking example of the maturational lag and unevenness in development.
3. The schizophrenic child has basic difficulties in boundaries and segregation in physiological and psychological subsystems.
4. The disorder is characterized by its pervading to some extent virtually all major areas of functioning.
5. The concept of plasticity characterizes the behavior and organization of the schizophrenic child in virtually all these areas.

The schizophrenic child shows poor tissue and muscle tone, bodily dependence, clinging or motor compliance, and cohesiveness, tending to use the adult's body and motility in order, it would seem, to help establish his own body boundaries and center of gravity. This also points to the fluidity or plasticity in the perception of motility, boundaries, and stability. The schizophrenic child in a symbiotic relationship offers the more extreme example of these features, but they can also be demonstrated in autistic schizophrenic children.

Perceptual experiences are derived from the fetal reaction to gravity by responses in tone, at first global. The vestibular system is the most ancient mechanism for perceptual motor responses. This is the beginning of awareness of the self as separate from the non-self, of all object relationships, and of awareness of reality. This is also the mechanism for the patterning of tone in muscles.

In schizophrenic children, where the perceptual motor patterns remain undifferentiated, there is retained a primitive plastic pattern of tone in all muscles and tissues. This contributes also to the autonomic nervous disorders in the vascular bed, intestinal tract, etc., and to the non-gratifying motility experiences leading to dependency, symbiosis, etc. Also, the infant fails to respond to changes in position when lifted or cuddled by the mother, nor does it learn to anticipate the approach of the mother by toning its muscles. Thus, object relationship is impaired, resulting in withdrawal and autism. Defects in awareness of reality and in establishing boundaries develop and contribute to body image disturbances.

I have frequently emphasized that lack of patterning, fluidity, maturational lags, and plasticity in all these areas of behavioral, homeostatic, motility, perceptual, ideational, and interpersonal functions, characterize the primary symptomatology in the child with schizophrenia.

Anxiety, which is a core problem in schizophrenia, arises secondarily as a result of the failure in differentiation and patterning in perception of internal, that is somatic and visceral sensation, as well as external perception, and in the failure to experience reality.

The anxiety leads to tertiary symptoms or defense mechanisms. There is a great variety of these and they may indeed determine the clinical picture and are often mistaken for the primary pathology. They include autism, symbiosis, various neurotic syndromes referred to as "pseudo-neurotic schizophrenia" (HOCH and POLLATIN), psychopathic acting-out behavior, especially in adolescence (BENDER 1959), or a frank psychosis.

Our treatment programs should include both specific and non-specific methods. Specific methods of treatment should have as their goals: 1) stimulation of maturational lags, 2) patterning of plasticity especially in the vascular tone, in smooth and striped muscle tone, in the autonomic nervous system functions and in perceptual patterns, 3) relief of anxiety, 4) protection and promotion of useful defense mechanisms.

With these goals in mind we have tried various forms of biological therapies. This has included metrazol convulsive treatment when it was in use. A twenty-five year follow-up (BENDER 1964) indicated favorable results. Electric convulsive treatment has been extensively used both at Bellevue Hospital (BENDER 1947) and Creedmoor State Hospital (FARETRA and GRUGETT). There has been evidence that this treatment does give a spurt to lagging maturation, it stabilizes the functions of the autonomic nervous system and the electroencephalogram. Psychological tests given before and after treatment show less variability in psychological functioning, a breaking up of preshock bizarre schizophrenic patterns in body image (human figure drawings) and gestalt drawing (BENDER, Visual Motor Gestalt Test, 1937), and improved level of intelligence (BENDER and KEELER; COBRINIK and MESSIS). Personality development improves, symptoms diminish and the prepuberty child is more normal (BENDER 1955) while remissions occur in puberty or early adolescence.

Electric convulsive therapy is most appropriate for the young autistic child who shows some favorable signs of free anxiety and capacity to develop defenses and language; for prepuberty children with neurotic manifestations; and puberty and early adolescent children with acute psychotic episodes.

Subcoma insulin therapy is an effective way of stimulating poorly developed and nourished regressed autistic children and other children of retarded development. It stimulates appetite and general well being. A close personal relationship with a physician during the period of recovery from the insulin, while the child is fed sweets, may encourage a child to talk and it may be generally therapeutic.

Modern psychopharmacotherapy during the last ten years has become a most valuable addition to our treatment programs for schizophrenic children. To the extent that we hope and believe that we see some evidence of some of the drugs stimulating maturation and patterning in the behavior of children and stabilizing of autonomic and visceral-vascular functioning, psychopharmacotherapy may be considered a specific form of therapy for schizophrenia.

However, in the use of drugs for therapy, it should always be emphasized that it is the child we are treating in an attempt to modify behavior, relieve symptoms, and promote normal development. Because of the double-blind, controlled research in drug evaluation, there has developed an attitude that giving a child a drug is like placing a chemical in a test tube and that similar reactions should be obtained, otherwise it is thought that all unique reactions are the result of doctor (or nurse) child relationship or a placebo response.

There are a few general statements that can be made about the drug treatment of children. So-called "tranquilizers" or sedatives should not be used to tranquilize or sedate children. Neither should drugs be given to relieve single symptoms such as enuresis, hyperactivity, depression, etc. Extensive experience in the use of a wide range of drugs has shown us (BENDER and FARETRA) that children react differently than adults to those drugs which affect the central nervous system. Often the effects are paradoxical especially if larger doses are used which children tolerate well. They rarely develop the side effects which often interfere with treatment of adults. Furthermore, children tend not to develop tolerance and doses rarely need to be increased. Children can take drugs for long periods of time with benefit. On the other hand, it often happens that the child shows a new spurt of behavioral improvement when the drug is discontinued. And for the most part improved behavior continues after the drug is discontinued.

This last observation reemphasizes our experience that the drugs promote normal maturation of the total organism, in behavior and in physiological functions.

Although it is important for every physician to learn from his own experience which drugs are most effective in his hands and with the children he is treating, we can offer a summary of our experience with the drugs we have used most commonly, with indications and dosage:

*Phenothiazines:* Best in schizophrenic patients who are disorganized, anxious, showing neurotic symptoms, especially hallucinations and introjected bodies, poor reality contact. Effective in catatonic or "typical" adolescent schizophrenics. Good in aggressive children, especially when combined with reserpine. Children with organic impairment occasionally show lowered convulsive threshold. Not so effective in organics.

Thorazine: Usual doses 100–1000 mg a day.

Compazine: 20–80 mg a day.

Stelazine: 2–8 mg a day. More stimulating. Seems promising for autistic children. More side-effects.

Sparine: 100–1000 mg a day. Better for control of motor symptoms.

Benadryl: 40–400 mg a day in a disorganized, younger child, including organic children.

*Reserpine:* 4–8 mg a day (higher doses in severely disturbed children). Among the best drugs for autistic children. Often combined with one of the phenothiazines. Very good in disturbed, disorganized, "psychotic" children. Helpful in organic children.

*Meprobamate:* 1600–4800 mg a day. Disorganized, anxious, hyperactive children, especially the organic, with or without anticonvulsants.

*Prazine* (Equanil plus Sparine): 4–8 capsules a day. Excellent in controlling behavioral symptoms, especially the organic children.

*Benzedrine:* Up to 20 mg a day. Hyperactive, organic younger children. Learning disabilities. Sexual problems and preoccupations.

*Energizers* (Niamid, Nardil, Tofranil, etc.): Effective in withdrawn, depressed adolescents. Helpful with autistic children; they are more alert, make attempts to speak, begin to relate.

*Librium:* 20–80 mg a day. Similar in indications to meprobamate. Not more effective.

*Deaner:* There are many reports that it stimulates learning and controls behavior, but we have found it relatively ineffective.

*Anticonvulsants* (Dilantin, Tridione, Mebaral, Mysoline, etc.): often combined with meprobamate, sometimes with reserpine, prazine, benzedrine. Used in organic behavior disorders or children with abnormal EEG's, even without convulsions.

Our use of D-*lysergic acid diethylamide* (LSD-25) and a methylated derivative (*methysergide*, UML 491) best illustrates our theories, methodology and results in the drug treatment of children (BENDER et al. 1966).

Our interest in these drugs was due to theoretical considerations, because of their serotonin-inhibiting effect and because of their stabilizing effect on the autonomic and central nervous system. The effects of LSD and other hallucinogenic agents are known to be arousal and increased responsiveness to sensory stimuli, preponderance of sympathetic activity and increased skeletal muscle tone and activity. Of particular interest is their tonic effect on the vascular bed especially of the brain, as has been shown with UML in vascular headaches. The known effects of these drugs on perception further increases their interest in the treatment of schizophrenia.

Such drugs were of interest to us for the treatment of childhood schizophrenia since our definition of this condition is a disorder in maturation characterized by an embryonic primitive plasticity in all areas of integrative brain functioning from which behavior subsequently arises. It was hoped that autism might be disrupted and that more normal autonomic functions

in the vascular bed, brain, intestines, skin and other organs as well as in perception would permit more normal development.

54 patients, ages six to fifteen, received LSD or UML daily for two months or more, with the usual dosage of LSD 150 micrograms daily, or UML 12 mg daily. The patients were boys and girls in the following groups: prepuberty autistic boys, autistic girls; postpuberty, "chronic" autistic boys; verbal psychotic pre- and postpuberty boys. The children were evaluated before, during, and after treatment, by clinical interviews, psychological tests, and biochemical studies, as well as routine reports and observations.

There were some differences in results in the various groups. In general, the younger autistic children became less anxious, less autistic and plastic, more aware and responsive, with some increase in verbalization. The girls and older autistic boys showed similar results, but much less marked. Verbal intelligent children showed improvement in general behavior, with marked changes in fantasy and bizarre ideation to more insightful, reality-oriented, though often anxious and depressive attitudes, and improved maturity and organization. They have made good adjustment outside of the hospital depending upon the social situation and school opportunities.

There were no major side effects, though a few patients on UML had muscular spasms and vasomotor changes in the legs, of a temporary nature. It is significant to note that while most of these patients had previously required tranquilizing or other medications, they could all be maintained only on the LSD or UML.

Parts of our therapeutic program may be considered non-specific since such programs are adapted to all of the emotionally disturbed, mentally ill or retarded children under our care. However, much of this program appears to stimulate maturation, improve behavior patterns and promote physiological stability also. These include – environmental change by removing the child from the anxiety-ridden, often distraught and disorganized home, into a hospital unit specifically planned for such children, which permits an opportunity for medical, neurological, psychiatric and psychological evaluation with daily school and activity programs suited to the child. It also gives him new group experiences with children, new relationships with adults, new routines and new stimuli with opportunities for new patterns of response.

Such a hospital program is essential if the child is undergoing convulsive therapy. It is also desirable in establishing a drug program especially for children who have not responded to a private doctor's office or out-patient clinic treatment or for children who do not have an adequate home. Many children respond to this program alone and may return home and be cared for in after-care clinics, even returning from time to time for short periods in the hospital. Other children move on to boarding schools, institutions for the retarded, correctional institutions, group living or foster care in the

community, depending on the basic underlying level of functioning of the child and the social background.

Close relationship with the child's home and community to which he belongs is an important part of treatment. Parents' visits are encouraged from the beginning and the child is permitted to go home for weekends, holidays, etc. Early discharge is encouraged even with frequent readmissions. The parents are encouraged and helped in organizing parent groups. They are seen in group discussion with psychiatrists and social workers. Volunteer workers from the community help with many of the activities in the hospital and visit and take a special interest in individual children who do not have families or visitors.

Psychotherapy for parents of children in hospitals should be supportive and helpful in interpreting their children's problems and what they have to accept for the future. Many parents of schizophrenic children, being schizophrenic themselves, may need psychiatric care at different levels. They should be referred to suitable treatment facilities. They should not be treated merely as the parents of a sick child. They easily become dependent on the institution or agency and taking their cue from current professional attitudes, demand attention and care which is not appropriate for the psychiatric facilities for children to give them. They need to become independent individuals in their own right.

Specific psychotherapy for schizophrenic children has been found to be inadequate by itself, except to tide some favorable cases through an episode where the prognosis was probably good in any case. In our experience (BENDER and GUREVITZ) it seemed that some young autistic children were helped to form more active neurotic defenses with clearer concepts of their own identity, body image, body functions and relationships to people and objects in the world by a close psychophysical relationship with the therapist. However, these children were in the hospital program also receiving convulsive therapy and appropriate drugs. The immediate results seemed good but later development was unfavorable in all but one of the children reported in this study.

Specific remedial procedures directed at specific areas of developmental lag, such as remedial tutoring for schizophrenic children with reading disabilities, has proved a promising therapeutic agent. The realistic interpersonal relationship with a skilled reading tutor facilitates the maturation of even the basic schizophrenic personality disorder as well as the learning disability (GOLDBERG).

A wide variety of therapeutic procedures have seemed to stimulate maturation, better organized development, behavior and learning and also the child-as-a-whole. Such therapeutic results we refer to as "specific" since we see childhood schizophrenia, at a clinical level, as a disorder in maturation and in patterned behavior.

**Summary.** Childhood schizophrenia is seen as the early part of the continuum into adult schizophrenia. It occurs as a result of an inherited predisposition, decompensated by intrauterine or paranatal noxious or traumatic events. It is characterized by disorders in maturation and in patterning of physiological and psychological behavior, often associated with developmental defects or organic brain disorders. Anxiety is a core problem with defense mechanisms of a great variety cloaking the underlying schizophrenic features. Typical life courses are described. Treatment is directed at stimulating maturation and improving the patterning in every area of function, physiological and psychological, in behavior and the personality of the child as a whole. Various methods of treatment and their results are discussed.

**Résumé.** La schizophrénie infantile est considérée comme étant la première phase d'un processus aboutissant à la schizophrénie de l'adulte. Elle est le résultat d'une prédisposition héritée, décompensée par des événements nocifs intra-utérins ou paranataux, ou traumatiques. Elle est caractérisée par des troubles de la maturation et des modèles de comportement physiologique et psychologique, souvent associés à des défauts évolutifs ou des troubles cérébraux organiques. L'anxiété est un problème central avec des mécanismes de défense variés voilant les caractères schizophréniques sous-jacents. Des modalités évolutives typiques sont décrites. Le traitement vise à stimuler la maturation et le renforcement dans les domaines du fonctionnement physiologique et psychologique, dans le comportement et dans la personnalité infantile comme un tout. Diverses méthodes thérapeutiques et leurs résultats sont décrites.

**Zusammenfassung.** Die Autorin betrachtet die kindliche Schizophrenie als den ersten Teil eines kontinuierlichen Verlaufs, der in die Schizophrenie der Erwachsenen übergeht. Sie ist das Ergebnis einer erblichen Prädisposition, die durch intrauterine oder perinatale Noxen oder durch traumatische Erlebnisse dekompensiert wird. Sie ist durch physische und psychische Reifungs- und Verhaltensstörungen charakterisiert und oft mit Entwicklungsschäden und organischen Hirndefekten verbunden. Angst ist ein Kernsymptom mit verschiedenartigen Abwehrmechanismen, welche die schizophrenen Grundmuster maskieren. Typische Lebensläufe werden beschrieben. Die Behandlung richtet sich auf die Förderung der Reifung und die Verbesserung der Anpassungsmöglichkeiten im Verhalten und Charakter. Verschiedene Behandlungsmethoden und ihre Ergebnisse werden erörtert.

**Resumen.** La esquizofrenia infantil es vista como la fase precoz dentro del continuum de la esquizofrenia del adulto, originada como resultado de una predisposición hereditaria, descompensada por noxas intrauterinas o paranatales, así como por eventualidades traumaticas. Se caracteriza por desórdenes en la maduración y modulación de la conducta fisiológica y psicológica, a menudo asociados con defectos evolutivos o trastornos orgánicos del cerebro. La ansiedad es el problema medular, con mecanismos de defensa muy variados, que superponen las manifestaciones esquizofrénicas básicas. Se describen casos de curso vital típico. El tratamiento se dirige a estimular la maduración y a mejorar la modulación fisiológica y psicológica de cada área funcional, considerando la conducta y la personalidad del niño como un todo. Se discuten varios métodos de tratamiento y sus resultados.

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# Die Bedeutung der vergleichenden Berücksichtigung des Lebensalters für die Untersuchung der Verlaufsgesetzmäßigkeiten der Schizophrenie bei Kindern und Jugendlichen

**The Importance of Considering the Effect of Age for the Study of Fixed Rules in the Course of Schizophrenia in Children and Adolescents – La signification de la prise en considération comparative de l'âge chronologique pour l'étude des critères évolutifs de la schizophrénie des enfants et des adolescents – Importancia de la consideración comparativa de la edad para la investigación de las formas de curso en la esquizofrenia infantil y juvenil**

G. J. Sucharewa

Der Einfluß des Altersfaktors auf die Symptomatik und den Verlauf der Schizophrenie bei Kindern und Jugendlichen ist durch zahlreiche Untersuchungen einheimischer und ausländischer Psychiater erwiesen worden (Simson, Kudrewzewa, Schechowa, Oseretzky, Rümke, Sante de Sanctis, Homburger, Tramer, Lutz, Kanner, Despert, Bender, Leonhardt, Michaux, Koupernik, Duché, Kirman, Wieck). Die Besonderheiten der Formierung des klinischen Krankheitsbildes der Schizophrenie bei Kindern und Jugendlichen bildeten im Verlauf der letzten drei Jahrzehnte den Gegenstand von Untersuchungen in unserer Klinik (Witebskaya, Wischnewskaya, Wrono, Grebelskaya, Dejanow, Lapides, Mamzewa, Novlanskaya, Osipova, Perskaya, Scanary, Sossükalo, Schur, Iwic-