The Stanley R. Dean Research Award, given annually to an outstanding scientist for major contributions to basic research on schizophrenia, was awarded May 4 this year to Dr. Leo Kanner at the American Psychiatric Association meetings in New York. Honored for his pioneering work on infantile autism, Dr. Kanner presented the following lecture as the Dean Award recipient.

INFANTILE AUTISM AND THE SCHIZOPHRENIAS

by Leo Kanner

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In a paper published in 1943, entitled "Autistic Disturbances of Affective Contact," I reported from the Children's Psychiatric Service of the Johns Hopkins Hospital observations of 11 children (8 boys and 3 girls) who had in common a pattern of behavior not previously considered in its striking uniqueness. The symptoms were viewed as a combination of extreme aloneness from the beginning of life and an anxiously obsessive desire for the preservation of sameness. I concluded the discussion by saying: "We must assume that these children have come into the world with an innate inability to form the usual, biologically provided affective contact with people, just as other children come into the world with innate physical and intellectual handicaps. If this assumption is correct, a further study of our children may help to furnish concrete criteria regarding the still diffuse notions about the constitutional components of emotional reactivity. For here we seem to have pure-culture examples of inborn autistic disturbances of affective contact."

In my search for an appropriate designation, I decided in 1944, after much groping, on the term *early infantile autism*, thus accentuating the time of the first manifestations and the children's limited accessibility.

The term autism was introduced by Eugen Bleuler, who wrote: "Naturally some withdrawal from reality is implicit in the wishful thinking of normal people who 'build castles in Spain.' Here, however, it is mainly an act of will by which they surrender themselves to a fantasy. They know that it is just fantasy, and they banish it as soon as reality so demands. I would not call the effects of these mechanisms 'autism' unless they are coupled with a definite withdrawal from the external world."

This definition does not quite account for the status of our patients. For one thing, withdrawal implies a removal of oneself from previous participation. These children have never participated. They have begun their existence without the universal signs of infantile response. This is evidenced in the first months of life by the absence of the usual anticipatory reaction when approached to be picked up and by the lack of postural adaptation to the person who picks them up. Nor are they shutting themselves off from the external world as such. While they are remote from affective and communicative contact with people, they develop a remarkable and not unskillful relationship to the inanimate environment. They can cling to things tenaciously, manipulate them adroitly, go into ecstasies when toys are moved or spun around by them, and become angry when objects do not yield readily to expected performance. Indeed, they are so concerned with the external world that they watch with tense alertness to make sure that their surroundings remain static, that the totality of an experience is reiterated with its constituent details, often in full photographic and phonic identity.

All this does not seem to fit in with Bleuler's criteria for autism. There is no withdrawal in the accepted sense of this word, and a specific kind of contact with the external world is a cardinal feature of the ill-

ness. It may therefore appear at first glance that I followed the example of the pseudo-etymologists who claimed that the Latin word for dog derives from the animal's inability to sing (*Canis a non canendo*) and the word for grove from the absence of light (*Lucus a non lucendo*). Nevertheless, in full recognition of all this, I was unable to find a concise expression that would be equally or suitably applicable to the condition. After all, these children do start out in a state which, in a way, resembles the end results of later-life withdrawal, and there is a remoteness at least from the human portion of the external world. An identifying designation appeared to me to be definitely desirable because, as later events proved, there was danger of having this distinct syndrome lumped together with a variety of generalized categories.

It can be said in retrospect that the brief history of infantile autism can be separated into three consecutive phases.

1. While the case reports, their phenomenology, and their etiologic implications almost immediately received the attention of the profession, it naturally took some time before similar observations could be made and communicated. For this reason, the earliest reactions dealing with the issue did not appear in print for several years. Meanwhile, studies were continued and intensified at the Johns Hopkins Hospital. For a period of approximately one year a special ward was set aside at the Henry Phipps Psychiatric Clinic for close investigation and therapeutic experimentation. In a 1946 paper, I discussed the peculiarities of metaphorical and seemingly irrelevant language of autistic children, and in a 1949 article, having by that time become acquainted with 55 patients who could be so diagnosed with reasonable certainty, I tried to set forth my ideas about the problems of nosology and psychodynamics of early infantile autism.

2. This state of affairs changed abruptly in 1951. No fewer than 52 articles and one book were concerned specifically with the subject between then and 1959. The first European confirmations of the existence of the syndrome came in 1952 from van Krevelen in Holland and from Stern in France. In the same year, Clemens Benda included in his book, *Developmental Disorders of Mentation and Cerebral Palsies*, a brief chapter with four illustrations, entitled "The Autistic Child". In it, he wrote: "The great question is whether autism is a part of the schizophrenic syndrome complex or should be considered a separate entity. A decision of this question cannot be made without a more thorough discussion of what constitutes childhood schizophrenia."

This sage advice was not heeded by many authors. While the majority of the Europeans were satisfied with a sharp delineation of infantile autism as an illness *sui generis*, there was a tendency in this country to view it as a developmental anomaly ascribed exclusively to maternal emotional determinants. Moreover, it became a habit to dilute the original concept of infantile autism by diagnosing it in many disparate conditions which show one or another isolated symptom found as a part feature of the overall syndrome. Almost overnight, the country seemed to be populated by a multitude of autistic children, and somehow this trend became noticeable overseas as well. Mentally defective children who displayed bizarre behavior were promptly labeled autistic and, in accordance with preconceived notions, both parents were urged to undergo protracted psychotherapy in addition to treatment directed toward the defective child's own supposedly underlying emotional problem.

By 1953, van Krevelen rightly became impatient with the confused and confusing use of the term infantile autism as a slogan indiscriminately applied with cavalier abandonment of the criteria outlined rather succinctly and unmistakably from the beginning. He warned against the prevailing "abuse of the diagnosis of autism," declaring that it "threatens to become a fashion." A little slower to anger, I waited until 1957 before I made a similar plea for the acknowledgment of the specificity of the illness and for adherence to the established criteria.

To complicate things further, Grewel, in the hope of avoiding confusion between true autism and other conditions with autistic-like features, suggested the term pseudo-

autism for the latter. Even this term came to be employed haphazardly, and conditions variously described as hospitalism, anaclitic depression, and separation anxiety were put under the heading of pseudo-autism.

All this resulted in the need for a careful evaluation of the reports of cases presented as samples of autism. A sifting of the literature of the 1950's compels one to eliminate many alleged illustrations as descriptive of something other than that which they were intended to portray.

3. The 1960's have witnessed a considerable sobering up. The fashion deplored by van Krevelen has gradually subsided. This is perhaps caused in part by the fact that those who go in for the summary adoption of diagnostic clichés have now found another handy label for a variety of abnormalities. Instead of the many would-be autistic children who are not autistic, we have the ever-ready rubber stamp of "the brain-injured child." While this certainly is regrettable, it has at least driven the acrobatic jumpers onto another bandwagon and has left the serious study of autism to those pledged to diagnostic accuracy. Hence, it is easier to single out properly designated cases, not lost in the shuffle of a peculiarly miscellaneous deck, for an investigation of their pathognomonic characteristics. And indeed, in the past few years, the diagnoses made have been more uniformly reliable and the discussion has been considerably less obfuscated by the smuggling in of irrelevant materials.

However, the question of nosological allocation of infantile autism has continued to be a matter of puzzlement. This is especially true of the formulations regarding its relation to schizophrenia.

Anyone attempting such a formulation ought to bear in mind Clemens Benda's quoted suggestion that a decision will have to depend on a discussion of what constitutes schizophrenia. One cannot get away from the need for semantic clarification and for a historical and ideological review of the meaning attached to all that is involved when this term is used. Has the meaning been stationary since the word was coined or have there been fluctuations and modifications? Is schizophrenia to be conceived as a unitary disease or as a generic noun encompassing a variety of kindred entities? Would all psychiatrists respond to these questions with unanimity?

In the 1890's, Kraepelin undertook the magnificent architectural job of erecting a solid structure of psychiatry from the many building stones lying around in disarray. Guided by the search for basic similarities and dissimilarities, he found a common denominator in a number of psychotic conditions which impressed him as sharing a "deteriorating process." Among them he included the catatonia of Kahlbaum (1863), the hebephrenia of Hecker (1871), the simple deterioration of Pick (1891) and Sommer (1894), and paranoid states associated with disorganization. He subsumed the whole deteriorating group under the term dementia praecox. As soon as this was accomplished, Kraepelin found it necessary to retain the above syndromes, not as the separate units as which they had been previously presented but as subdivisions of the *specific* disease, dementia praecox.

Before we proceed, it is important to pause for an examination of the meaning of the word "specific." Some of the dictionaries offer among their definitions two which, if used interchangeably, are apt to produce—and are indeed producing—semantic quandaries. One says: "Designating a definitely distinguishable disease." The other says: "Of or pertaining to a species, or group, of which the members have common characteristics and are called by a common name."

Kraepelin viewed dementia praecox with its subdivisions essentially as specific in the sense of the first-quoted definition. When Bleuler suggested the term schizophrenia in 1911, he announced that he looked upon it as a common name for a species and emphasized his point by speaking not of schizophrenia in the singular but of the "group of the schizophrenias." He declared significantly: "This concept may be only of temporary value inasmuch as it may later have to be reduced," adding parenthetically, "(in the same sense as the discoveries in bacteriology necessitated the subdivision of

the pneumonias in terms of various etiologic agents)."

This prediction indicates a profound grasp of medical history and may prove to be prophetic in the long run. For much of the progress of medicine has been characterized by the singling out of circumscribed diseases from a welter of ill-defined generalities, by the gradual transition from the assumption of the homogeneity to the recognition of the heterogeneity of conditions which have certain broad aspects in common. We are far removed from the time when learned treatises were published, entitled *De febribus* or *De pestibus*, dealing with febrile illnesses and contagious diseases as if they were all of them identical in nature and origin. The falling sickness, once regarded as a single entity, is now divided into a variety of dysrhythmic conditions of different provenances. Before Langdon Down's description of mongolism in 1866, mental defectives were thought of as if, to paraphrase Gertrude Stein, the feebleminded were the feebleminded were the feebleminded; since then, neuropathologic, metabolic, genetic, and psychological studies have managed to do away with the illusion of the sameness of all mental deficiency.

There is at present cause to believe that similar developments are in store for the concept of schizophrenia, resulting in the "reduction" envisioned by Bleuler. Moreover, concrete demonstrations of this trend are beginning to be supplied in the area of child psychiatry.

When Kraepelin created the concept of dementia praecox, he did so entirely on the basis of his work with adults. At no time was there any reference to its occurrence in children. He mentioned once, as a hunch rather than on grounds of careful statistics, that of 1,054 patients 3½ percent had shown signs of "psychic weakness" before ten years of age. All that Bleuler had to say about this in his sizable monograph is contained in a footnote, which reads: "The disease rarely becomes manifest in childhood. Yet there are cases in which a primary schizophrenia can be traced back to the earliest years of life." On the page above this footnote Bleuler reported an estimate according to which the onset of schizophrenia could be found in 4 percent of the case histories to go back to the age of "before 15 years." Obviously, when any thought was given to children at all in connection with dementia praecox or schizophrenia, it was done so largely in terms of retrospect and not as a result of direct examination at the time of the anamnestically recorded incipiency. Childhood schizophrenia was not something seen and clinically investigated as such but rather merely hinted at as an occasional prelude remembered by the relatives of adolescent and adult patients.

Between 1905 and 1908, Sante de Sanctis tried to accomplish for children what Kraepelin had done for adults. He gathered a number of cases which had in common symptoms of lack of affect, negativism, stereotypy, talkativeness, delirium, hallucinations, and catatonic features. As etiologic factors he enumerated hereditary predisposition, acute or chronic toxic diseases, and "factors inherent in child development." He combined all cases under the name dementia praecocissima, a sort of miniature version of dementia praecox. His reports were followed by additional illustrations of his own and other Italian investigators, and a few cases were published under this caption by German, French, and Swiss authors. De Sanctis felt that the clinical picture was indistinguishable from dementia praecox but he was not sure that the adult and the infantile forms had the same causative background.

A review of the contemporary and subsequent literature shows that the notion of dementia praecocissima proved to be of limited viability and has now been discarded altogether as a valid collective designation. Most of the patients turned out to be specimens of an assortment of neuropathologically identifiable, more or less progressive, congenital or acquired anomalies of the central nervous system. Some of them (e.g., Schilder's disease) could not have been known to De Sanctis because they were not isolated until after the time when he incorporated them in his classification. Clinical neurological tests, biopsies, or autopsies removed them from the category; the concept of dementia praecocissima continued to be

"reduced" to a point where it lost its justification altogether. In view of later attitudes, it is significant to note that the finding of a clearly definable organic disorder automatically excluded an ailment from the diagnosis of childhood schizophrenia.

This became evident again when the Viennese educator (*Heilpädagoge*) Heller described in 1908 a group of children presenting "an almost photographic identity of course" with the following features: After normal development during the first two or three years of life, there was a rapid change of behavior with anxiety, motor restlessness, loss of speech and of sphincter control, and general regression, leading in a short time to complete dementia, while the children showed no clinical signs of physical disturbance and retained an intelligent facial expression. Heller's disease or dementia infantilis (a term coined by Weygandt), though extremely rare, was observed by others as well and was entered in the textbooks as the earliest form of childhood schizophrenia. It remained there until Corberi in 1931 discovered in four brain biopsies "acute diffuse degeneration of the ganglion cells.' Schilder averred categorically in 1935: "I assume as a matter of course that dementia infantilis has nothing to do with schizophrenia but is an organic process."

This is a pivotal statement around which hinges the whole philosophy of the distinction between organic and functional psychoses. Schizophrenia was classed as a functional psychosis by definition. Ergo, if you find in a patient signs of an organic process his condition logically has nothing to do with schizophrenia.

Two departures are possible from here. One goes in the direction of viewing the term functional as a temporary, perhaps even a bit embarrassing admission of the inability to find an organic substratum; this spurs a search for one so that, if it is discovered, the adjective functional can be dropped. This is, after all, the gist of Kraepelin's postulate of a metabolic disorder, of Bleuler's prophecy, and of much that is happening today in the realm of schizophrenia research. The other departure goes in the direction of regarding "functional" as synonymous with nonorganic; this has encouraged a search for environmental, psychogenic noxa as the explanation of schizophrenic phenomena. The one focuses on the quest for internal, centrifugal springs of psychotic behavior; the other on the shaping influence of external, centripetal forces. Until recently, the twain did not meet and were bogged down in an irreconcilable antithesis.

This antithesis did not become conspicuous until the early 1940's. Until that time, when there was talk of childhood schizophrenia, curiosity was extended mainly to patterns resembling adult syndromes. Strohmayer's treatise on the psychopathology of childhood, which appeared in 1910, and the 1926 (second) edition of Ziehen's textbook of the mental diseases of children discussed juvenile (hardly ever infantile) schizophrenia in Kraepelinian terms. Strohmayer did not hesitate to say that, except for senile, arteriosclerotic, and true paranoid psychoses, all mental illnesses known in adults can be encountered in children with the same symptomatology. Ziehen viewed schizophrenia as an "acquired defect psychosis," together with paralytic, epileptic, traumatic, meningitic, and toxic dementia, in contrast to diseases not resulting in intellectual defect. He spoke of the latter (e.g., mania, melancholia, delirious states, paranoia, and obsessive psychosis) as "functional." This runs counter to the usual inclusion of schizophrenia among the functional psychoses.

However, in the late 1920's and in the 1930's more and more voices were raised in favor of a distinction between adult and childhood schizophrenia, attributable to maturational and experiential factors. There was growing agreement that the Kraepelinian subdivision was not suited for the preadolescent years, and efforts were made to find a grouping more in harmony with direct observations. In Germany, Homburger, one of the pioneers of child psychiatry, decided to use the type of onset and course of illness as a starting point, rather than the symptoms noted at any given time. He thus suggested two different groups, one with acute onset and another with insidious onset. Partly in consequence of Homburger's lead and in part independently, Ssucharewa

in Russia, Lutz and Tramer in Switzerland, and Despert in this country underlined this grouping in their discussions of childhood schizophrenia. Patients of the first group, mostly older children, have seemed to make a good adjustment prior to the appearance of recognizable psychotic symptoms. In the second group, there is a gradual withdrawal from contact with reality, a progressive loss of interest in play, an increasing tendency to brood, a preoccupation with abstractions, and obsessively repetitious ruminations. It was deemed essential for the diagnosis in both groups that a period of relative normalcy had preceded the beginning of the illness.

This was the situation around the start of the 1940's, at the time when I published the first cases and introduced the concept of infantile autism. One year earlier Bender had summed up the general professional attitude in a few sentences which deserve to be quoted because of their clarity and succinctness. She wrote: "There are those who do not believe in childhood schizophrenia, not having seen a case. At the best, none of us has seen very many cases in which we could make a definite diagnosis, not knowing the acceptable criteria. There are others who, having seen certain types of mental disorders in children, prefer to call them schizophrenia-like psychoses of childhood."

While Bender and others began to look for acceptable criteria, a sizable group of workers, temporarily influential especially in this country, joined in a chorus chanting the refrain, *Cherchez la mère* (which I tried in vain to silence in 1941 in my book, *In Defense of Mothers*). Poohpoohing description as an obsolete pastime of atavistic nosographers, they started out with interpretations in which the mother-child relationship was put on the pedestal as the only valid etiologic consideration. The underlying idea was: Why bother about questions of genetics, organicity, metabolism, or anything else if we can proceed promptly with the psychogenic denominator common to all disturbances of the ego? Thus arose a tendency to set up a pseudodiagnostic waste basket into which an assortment of heterogeneous conditions were thrown indiscriminately. Infantile autism was stuffed into this basket along with everything else. On the East Coast, Beata Rank, creator of the notion of "the atypical child," comprised in this hodgepodge "all more severe disturbances in early development which have been variously described as Heller's disease, childhood psychoses, childhood schizophrenia, autism, or mental defect." On the West Coast, Szurek announced: "We are beginning to consider it clinically fruitless, or even unnecessary, to draw any sharp dividing lines between a condition that one could call psychoneurotic and another that one could call psychosis, autism, atypical development, or schizophrenia." Such looseness threw all curiosity about diagnostic criteria to the winds as irrelevant impediments on the road to therapy which was applied to all comers as if their problems were identical. The therapeutic cart was put before the diagnostic horse and, more often than not, the horse was left out altogether. With a perfunctory bow in the direction of "heredity and biology," we were urged to give up the concern for the differentiation of any kind of behavioral deviation. By decree, mother-infant involvement was to be accepted as the sole key to everything that goes on within and around the neonate; it alone was supposed to determine his destiny. Modify it therapeutically by using the right technique, and the child has a chance to become adapted to the requirements of suburban propriety.

In contrast to this summary disavowal of biological factors, Bender, assuming the possibility of an underlying diffuse encephalopathy, defined childhood schizophrenia as follows: "A disorder in the regulation of maturation of all the basic behavior processes, represented in children by a maturation lag at the embryonic level, characterized by a primitive plasticity in all patterned behavior, determined before birth and activated by a physiological crisis, such as birth." With anxiety as the organismic response to such crises, secondary symptoms were called forth as defense mechanisms which served Bender as a basis for the grouping of childhood schizophrenia into three types: (1) the pseudodefective or autistic type; (2) the pseudoneurotic or phobic, ob-

sessive, compulsive, hypochondriacal type; (3) the pseudopsychopathic or paranoid, acting-out, aggressive, antisocial type.

This formulation accomplished a number of things. It managed to dispose of the idea that childhood schizophrenia must be viewed as *either* functional *or* organic. It implied that at the present state of our knowledge one can—and must—take into consideration the likelihood of a fusion of innate physiological and postnatal emotional factors. It suggested a grouping based on observed phenomena differing in character and in course.

Questions have arisen, however, with regard to the ease with which the diagnosis was suddenly bestowed upon a relatively vast contingent of patients. Bender, who in 1942 had, as she said, "not seen very many cases in which we could make a definite diagnosis," announced later that by 1951 "over 600" schizophrenic children had been studied in one single psychiatric unit, that of the Bellevue Hospital in New York. By 1954, she had as many as 850 cases on her list, which means an addition of about 250 in the short span of three years. It is highly improbable that all of them would be acknowledged as being schizophrenic by many other experienced child psychiatrists, and yet it cannot be denied that Bender has made careful investigations and has conscientiously adhered to her established criteria.

Out of this emerges a rather disturbing dilemma. We seem to have reached a point where a clinician, after the full study of a given child, can say honestly: He is schizophrenic because in my scheme I must call him so. Another clinician, equally honest, can say: He is not schizophrenic because according to my scheme I cannot call him so. This is not a reflection on anyone in particular. The whole concept has obviously become a matter of semantics.

It is not unreasonable to hope that the *bracketing out of the syndrome of infantile* autism portends a way out of the dilemma. Returning to the two definitions of the word "specific," we can state unreservedly that, whether or not autism is viewed as a member of the species schizophrenia, it does represent a "definitely distinguishable disease." This disease, specific—that is, unique, unduplicated—in its manifestations, can be explored per se. Unimpeded by the perplexities of nosological assignment, investigators can agree on its own phenomenology, search for its own etiology, and follow its own course in ordinary and experimental settings.

This is in keeping with the trend to which Eugen Bleuler's son, Manfred, referred when in a comprehensive review of theory and research between 1941 and 1950, he wrote: "The formulation of schizophrenia has rid itself during the past ten years of the hypothetical idea that a disease termed schizophrenia is available for investigation; instead, it has turned its focus on the study of separate diseases within the group of the schizophrenias."

Once it is acknowledged that infantile autism is a separate disease (and then only), the controversy about its rightful position in any hierarchic nomenclature becomes a matter of personal preference and will remain so until we have acquired more substantial knowledge of fundamentals and depend less on speculation and dialectics.

There are those who insist that infantile autism is one of the schizophrenias, even though this means giving up the original idea that childhood schizophrenia develops after a period of relative normalcy. There are some who, because of the poor response by many patients to psychometric assessment, want it placed among the mental deficiencies. And there are those who refuse it a domicile in either group.

Rimland (1964, p. 68) believes "that there is sufficient information at hand to demonstrate clearly that early infantile autism is *not* the same disease or cluster of diseases which has come to be called childhood schizophrenia, and that autism can and should be distinguished from it at all levels of discourse." Further on (p. 76), he states that "it is clearly accurate and desirable *to treat infantile autism and childhood schizophrenias* as separate and quite unrelated disease entities." But is childhood schizophrenia a separate disease entity?

The singling out of autism has been followed by a number of other attempts to describe specific conditions lifted out of the

schizophrenic package. Mahler reported in 1949 a syndrome which she named "symbiotic infantile psychosis," distinguished by a symptomatology which she thought to be centered around a desperate effort to avert the catastrophic anxiety of separation. In the same year, Bergman and Escalona discussed "children with unusual sensitivity to sensory stimulation." In 1954, Robinson and Vitale introduced a number of "children with circumscribed interest patterns." It may well turn out that this is just a beginning and that other syndromes will be detected and studied on their own merit.

There is, indeed, no "disease entity" called childhood schizophrenia, just as there is no disease entity called mental deficiency. It would not occur now to anybody to look for a uniform background for such anomalies as phenylketonuria, galactosemia, and familial oligoencephaly. By the same token, it is hardly feasible to house together an assortment of dissimilar phenomenologic conditions grouped loosely as childhood schizophrenia, be that on the basis of genetics, neuropathology, biochemistry, psychoanalysis, existentialism, or what have you.

Infantile autism serves as a paradigm. Unfortunately, cause and effect are not as easily ascertainable as Fölling has been able to make them for phenylketonuria. But we have at least a well-defined clinical picture of beginnings, symptoms, and course which in their totality are unmatched and therefore specific in the same sense as phenylketonuria is specific—that is, "a definitely distinguishable disease." Efforts have been made recently by Polan and Spencer and particularly by Rimland (1964) to refine the diagnostic criteria and to compile a check list of symptoms as a guide for differential diagnosis. Since my own publication in 1946, valuable studies have been made of the language peculiarities of autistic children, especially in the German monograph by Bosch, *Der frühkindliche Autismus*, which appeared in 1962. In 1951, I reported observations on the conception of wholes and parts in early infantile autism. In 1954, I attempted to review autism from the point of view of genetics or at least genealogy. Others have investigated the effects of drugs on autistic children. There have been a number of follow-up studies through, and some beyond, adolescence. A valuable contribution was made in 1953 by Ritvo and Provence about form perception and imitation. The occurrence of autism in twins has been reported.

I was greatly impressed from the start by an observation which stood out prominently and that I made a point of in my first report. Among the first 11 cases, the parents of my patients were for the most part strongly preoccupied with abstractions of a scientific, literary, or artistic nature, and limited in genuine interest in people. Even some of the happiest marriages were rather cold and formal affairs. I remarked: "The question arises whether and to what extent this fact has contributed to the condition of the children. The children's aloneness from the beginning of life makes it difficult to attribute the whole picture exclusively to the type of the early parental relations with our patients." As time went on and more autistic children were seen, the coincidence of infantile autism and the parents' mechanized form of living was startling. This was confirmed by most observers. These were realities which were impossible to ignore. Yet there were some exceptions. Approximately 10 percent of the parents did not fit the stereotype. Besides, those who did reared other normal or, at any rate, nonpsychotic offspring. Moreover, similarly frigid parents are often seen whose children are not autistic.

Aspects of interplay between patients and their parents have been studied by various investigators, and on the basis of these studies four viewpoints have emerged. One theory regards parental behavior as a reaction to the children's peculiarities and of no etiologic significance; this would be justified if it were not for the established fact that the parents' personalities had displayed the characteristic traits long before the arrival of their autistic children. At the other extreme parents, particularly mothers, are considered the basic cause of pathogenicity; the assumption is that a healthier maternal attitude would have precluded the disorder. A third group feels that the patient, endowed with an innate disability to relate to people,

is further influenced adversely by the parents' emotional detachment and the resulting manner of handling him; this in no way discounts the possibility of a reciprocal awkwardness of living together. A fourth theory looks upon the children's psychoses and the antecedents' emotional aloofness as stemming from a common, biologic, genetically determined source; some of the parents indeed give one the impression of autism that has escaped psychotic propensities.

Without going into further details, all of which are fascinating and instructive, the following points can be made with regard to the present state of affairs.

1. It is now generally agreed that a unitary disease entity, schizophrenia, does not exist. Analogously, there is no such unit as childhood schizophrenia.

2. For the time being, however, this caption cannot be discarded. It is "specific" only in the sense that it "pertains to a species, or group, of which the members have common characteristics and are called by a common name." After all, we still speak of mental deficiency, knowing well that the term, a semantic convenience, includes etiologically and clinically heterogeneous conditions.

3. Bender's pioneering work has helped do away with the either-or antithesis of functional and organic by recognizing the fusion of innate as well as experiential components.

4. Attempts have been made to subdivide clinical varieties comprised under the term childhood schizophrenia: cases with acute and insidious onset; "organic and nonorganic" (Goldfarb & Dorsen, 1956); pseudodefective, pseudoneurotic, and pseudodelinquent types. These, I believe, have important though only temporary value in that we may anticipate a splitting off of specific syndromes—"specific" in the sense of a "definitely distinguishable disease."

5. Infantile autism has been split off from the cluster and offers itself for investigation of its unique features. It has an identity of its own.

6. Some of the remaining controversies are based more on semantics than on intrinsic essentials.

In closing, I would like to quote from a paper, entitled "Schizophrenia as a Concept," which I presented at a symposium in 1959. I said there: "Child psychiatry is showing the way to the practical application of Bleuler's vision of the plurality of the schizophrenias. It is encouraging to note that similar attempts are beginning to be made with regard to the adult schizophrenias. The smug certainty about a disease schizophrenia has been definitely sloughed off. For the time being, there is still much groping and more or less emotionally tinted clinging to cherished opinions. But so long as facts are scarce, it is inevitable that there be differences of opinion about the delineation of the concept itself, about etiology, and about therapeutic procedures. It is my opinion that in the foreseeable future the same thing will happen to the schizophrenias as has happened to the hyperpyrexias, the insanities, and the amentias and that, when we stop searching for an identical cause and treatment of different ailments tied together in the schizophrenia bundle, we may expect the opening up of new and clearer vistas. But this, also, is only an opinion."

REFERENCES

Rather than list the voluminous literature on infantile autism and childhood schizophrenia, I would like to call the reader's attention to three good bibliographic sources on the topic.

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