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TOWARD A THEORY OF EARLY INFANTILE AUTISM

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This paper hypothesizes that in early infantile autism, fetuses with an inherited high potential for intellectual superiority are developmentally ready for imprinting with human behaviors much before birth. Instead of being imprinted postnatally at critical periods in cerebral development as are most children, these neurologically advanced fetuses are imprinted on the restricted uterine environment. Such atypical imprinting leads to the syndrome of early infantile autism. Suggestions for research are offered.

Since Kanner (1943) wrote his paper in which he described a group of children who were characterized by "extreme autistic aloneness," the construct of early infantile autism has become engulfed in a morass of semantic confusion. Although Kanner believed that these children were special cases included in a larger group of children with childhood schizophrenia, subsequent investigations have used the terms schizophrenia, psychosis, atypical child, brain injured, and many others, to describe Kanner's group. The semantic problem has become further confounded by the fact that experimenters have purported to be investigating autism when their subjects were not at all like those Kanner discussed. For example, a study may report that a particular drug is useful with autistics when in fact the investigation failed to include a single child in the study who met Kanner's criteria.

The remainder of this paper, then, will discuss children designated by Kanner as being characterized by (*a*) inability to relate to people from the *beginning of life,* (*b*) failure to use language to communicate, (*c*) an obsession with maintaining sameness, (*d*) preoccupation with objects, and (*e*) evidence of good cognitive potentialities.

Parental Intelligence

Based largely on his observation of their educational and social status, Kanner (1965) inferred that the parents of autistics are often remarkably intelligent. His statements have been questioned on the basis that wealthier, better educated, and presumably more intelligent parents are more likely to seek psychiatry than those of lower status (Bettelheim, 1967; Creak & Ini, 1960).

In spite of the doubts regarding Kanner's beliefs concerning the intelligence of parents of autistic children, evidence is accumulating to support his contention. For example, Rimland's (1964) careful analysis of the American-European literature containing descriptions of the parents of autistic children led him to state that the evidence overwhelmingly supports Kanner's early findings. Rutter (1969) points out that there are significant differences between the social class status of autistics and schizophrenics. In an epidemiological study, Lotter (1967) found that a high proportion of parents of autistic children are above average in intelligence and of superior socioeconomic status. Lotter is careful to show that this finding is not an artifact of referral policies, and is supported by Treffert (1970) who has found that highly educated parents do not identify their children as disturbed more often than less-well-educated parents.

Recent data from Treffert (1970) further supports the inference of high parental intelligence based on educational achievement. In his longitudinal study of 280 cases of childhood schizophrenia and infantile autism, Treffert found the educational level of parents with autistic children to be significantly higher than that of parents with schizophrenic children. Treffert states that his verification of this persistent finding

> could well point toward a genetic view of the illness with the autistic child as seen as the mutant of especially intelligent progenitors [p. 434].

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Not all studies support the suggestion of high parental intelligence (Levine & Olson, 1968; Lowe, 1966; Schain & Yannet, 1960), but these, as well as similar investigations, are difficult to evaluate because of their failure to adhere to the definition of autism as proposed by Kanner. Included in some of these studies are chronic undifferentiated schizophrenics and autistic/symbiotics (Lowe, 1966), autistic "mental defectives" (Schain & Yannet, 1960), and undifferentiated psychotics (Creak & Ini, 1960).

When investigators are careful to differentiate autism from other conditions that may simulate it, and when referral policies to clinics are properly accounted for, evidence is emerging that the inference of parental intellectual superiority is justified. Treffert's (1970) hypothesis that autistic children are mutants of highly intelligent parents appears entirely plausible. Interestingly, such a hypothesis lends credence to Kanner's (1943) observation that autistic children show evidence of good cognitive potentialities, and Rimland's (1964) review of the literature in which the finding of very special memory and manipulative abilities in autistics was commonplace.

NEURAL DYSFUNCTION

There is substantial growing support for the suggestion that early infantile autism is associated with neurophysiological dysfunction of the reticular activating system. Working with autistic children who were selected on the basis of Kanner's criteria, Hutt and Hutt (1969) found that electroencephalogram (EEG) records of their subjects showed desynchronized activity. These findings, together with behavioral observations, led the investigators to hypothesize that the children were in a condition of high physiological and behavioral arousal. Ornitz and Ritvo (1969) postulate that the basic condition causing autism rests on faulty homeostatic regulation of perceptual input. Indirect evidence by Holzman (1969) shows that dysfunction in the final or proprioceptive-autonomic response portion of the total perceptual process may be a significant factor in the etiology of the schizophrenic syndrome. Finally, Mirsky (1969) found 13 recent studies supporting the assumption that all schizophrenia begins with a basic fault in the ascending reticular activating system.

BRAIN DAMAGE

While many investigators agree that autism has a neurophysiological basis, probably in the reticular activating system, and that a potentially superior organism is involved, the interaction of these two findings needs exploration.

Rimland (1964), who subscribes to both of the above concepts, neurophysiological basis and organismic superiority, states,

> If a potentially very intelligent child whose brain was continuing to develop at a rapid rate, were exposed to atmospheric oxygen (or worse yet, to relatively high concentrations of medical oxygen) while the vasculature was still immature, damage to the vasculature of his brain could result [p. 129].

Rimland's (1964) theory of oxygen insult crumbles under the simple logic that if the child is genetically superior and developing at a rapid rate, it, even more than a normally growing organism, should be able to withstand normal stresses. The circumstances of a superior, rapidly developing brain that is *more* sensitive to normal environmental insults seems contradictory and denies all we would logically hypothesize about the ability of superior biological organisms to survive physical trauma.

A number of investigations have suggested that autistic children are more likely to evidence various kinds of brain pathology than are other children. Even when it is indicated that the researchers have taken great care to differentiate autistics from other behaviorally impaired children, the findings are inconclusive. As Rutter (1969) points out,

> There are no published histopathological studies; the EEG studies are inadequate and contradictory; and metabolic investigations are still in their infancy [p. 397].

Experimenters who purport to find brain damage in autistic children may actually be observing dysfunction of the reticular activating system. As will be shown later in this paper, such atypical functioning is not

necessarily a result of brain damage but can result from sensory deprivation.

A review of "brain damage" as an etiologic factor in the production of autism is of little value. Rutter (1969) states,

> However, even if it is accepted that autism may sometimes, or perhaps often, develop on the basis of brain damage, the explanation is too general to be of much help in understanding how autism develops. The term "brain damage" includes a wide variety of disturbances. The vast majority of children with brain damage are not autistic, and it remains to be explained why the minority who become autistic do so. More specific biological hypotheses are required [p. 397].

SENSORY RESTRICTION

Until very recently, psychologists have largely ignored the prenatal *psychological* environment. The assumption seemed to have been in the past that all children start from a zero point at the moment of birth and few significant environmental influences, other than those affecting physical growth and development, have made an impression up until that time. Such an assumption has remarkable commonsense validity in view of the limited sensory experiences available to the fetus, but the question remains as to whether this restricted environment is the same for all fetuses. Does the prenatal psychological environment have a different effect on the cerebral structures of superior, rapidly developing fetuses than it does on those who develop more normally?

Part of the answer to this question is supplied by experiments concerned with the effects of sensory restriction. Schultz (1965) comments,

> restriction of early environmental stimulation in animals has produced drastic and enduring effects on emotionality, learning ability, activity level, social behavior, and perception. The optimal level of sensory variation of the individual is influenced by early postnatal levels of stimulation and the resulting level of cortical arousal as mediated by the reticular activating system. In other words, the level of stimulus variation to which the neonate is exposed functions to influence the optimal range of cortical arousal appropriate for adaptive behavior of the adult organism. The adaptation level of the ascending reticular activating system . . . is thus determined to some degree by the amount of early stimulation [pp. 25–26].

Schultz suggests that an impoverished sensory environment would prevent differentiation of cue functions, sensory modalities, and of events within modalities. Such a failure to differentiate leads to defects in the perceptual process.

Evidence that autism may result from sensory deprivation is supported by Rimland's (1964) review of the literature in which he concludes,

> there is obviously a good deal of behavioral similarity between the victims of early infantile autism and the victims of sensory deprivation [p. 104].

He notes that subjects who experience long periods of sensory deprivation are "detached from the external environment," and that they "stare right at you but never see you and just sit and look into space and not talk [p. 104]." The vacant "long eye" stare usually observed in these sensorially deprived subjects closely resembles the unfocused gaze of autistic children. Furthermore, Rimland's (1964) search of the literature has shown that the reticular formation is the most likely site in the brain to be associated with the phenomenon of sensory deprivation. He explains that there is sufficient reason for believing that autistic children are so perceptually impaired that sensory deprivation psychosis is entirely within the realm of possibility.

DEVELOPMENTAL READINESS

An additional concept needs to be considered in order to answer the question: Does prenatal psychological environment have a different effect on the cerebral structures of superior, rapidly developing fetuses than it does on those who develop more normally? This additional concept has to do with the developmental readiness of the fetal brain.

Differences in the maturational levels of full-term neonates as evidenced by variation in physical size, activity level, and the presence of developmental landmarks have been noted for decades. These same maturational differences exist prior to birth. Some fetuses have cerebral structures that are developmentally ready for sensory stimulation long before others. The neurophysiology of a fetus that is the product of intellectually su-

perior parents can reasonably be expected to show advanced maturation. As Ames (1969) has suggested, intellectual superiority tends to go along with advanced maturity.

The quantity and quality of early stimulation have enduring effects on the functioning of the brain. Profound environmental deprivation produces organic dysfunction (Rutter, 1969) and may *simulate* "brain damage." Significantly, the negative effects of such environmental deprivation are relatively greater for genetically brighter organisms (Schultz, 1965).

Given a fetus with a brain in an advanced state of developmental readiness for stimulation but residing within a restricted uterine environment, what would be the probable results? What are the effects of sensory restriction and failure to experience primary socialization when these two circumstances occur simultaneously?

PRIMARY SOCIALIZATION AND IMPRINTING

The striking similarities between the behavior of autistic children and that of nonimprinted or artificially imprinted animals are too great to ignore. The autistic aloneness of the child and his lack of interest in his own species has many parallels in studies demonstrating the species-ignoring behavior of animals that are not imprinted with their own kind. Writing about his experiments with geese, Lorenz (1957) says,

> Once their social relations are transposed to a human being their behavior does not change even if they are kept for years with other members of their species . . . [p. 105].

Moltz (1960) points out that once imprinting occurs, its effect cannot be reversed. The stimulus the animal experiences during the critical imprinting period becomes either the preferred stimulus or the only stimulus toward which the animal will move. Scott (1969) states in his discussion of the primary socialization in canine and human infants that an individual at the proper period in life will become attached to anything in the surrounding environment, both living and nonliving.

Addressing himself to the importance of the critical period concepts, Money (1969) writes,

> The essential elements of the concept are: 1) that a developmental readiness of the organism must be met by a suitable stimulus environment before the given phase of development can take place; 2) there is a time limit, after which the developmental opportunity is lost, often forever; 3) the events of the developmental phase leave a permanent, or at least a very durable, residue; 4) defective or impaired development may leave a permanent residual effect; and 5) as applied to social science, there is no clear distinction between body and mind for the events of the social life as assimilated by the brain which is affected by what it assimilates as well as effecting in turn, the events outside of itself in social life [p. 145].

Does the autistic child's insistence on sameness in the environment, his repetitive and stereotyped behaviors, his hand regarding, and his failure to develop speech suggest that he was imprinted by an atypical environment at a critical period in his primary socialization? In this essentially unchanging environment, the fetus has only the heartbeats of himself and his mother, and perhaps the other rhythms of their bodies, to affect him. Is it possible that the autistic child is so firmly imprinted with his relatively featureless, secluded, and limited world during critical developmental periods, when he should be vividly stimulated and experiencing primary socialization, that he in fact seems to be living "in his own world" after birth? Does it explain his apparent need to maintain environmental sameness?

What are the effects of rhythmic heartbeats and body movements on a developmentally superior fetus? Do these rhythms account for the repetitive and stereotyped behaviors observed later in life? Does such "rhythmic imprinting" explain Rimland's (1964) findings that astonishing musical ability has been reported in at least nine studies of autistic children?

Is the fetus "kinesthetically imprinted" on his own hands? They constitute the major movable stimuli in his dark prenatal world. Does this account for the frequently observed exceptional manipulative skills shown after birth?

Rutter (1969) notes that at 16 weeks, the normal infant is definitely responding meaningfully to human speech. What happens

when a neurologically advanced, developmentally ready fetus does not receive early speech stimulation because it is unavailable to him?

Psychological literature contains many reports of feral children who remained wild after many years of association with humans. Was their inability to assume human characteristics due to a failure to experience primary socialization with humans at a critical period in their development?

Spitz (1965), reporting on the work of M. Von Senden, notes that several congenitally blind persons whose sight was restored between the ages of 3 to 40 never learned to "see" although they had vision. Had the critical period for imprinting for meaningful visual perceptions passed? Was their behavior similar to autistic children who "look through" persons but never "at" them? Do humans *as* humans have any meaning for autistic children, or are they similar to lower animals who have been prevented from being imprinted on their own species?

Are autistic children, by their frequent obsessions with inanimate objects, demonstrating Scott's (1969) finding that an individual at the proper time in life will become attached to both living and nonliving things in the surrounding environment?

The period for primary socialization in the human infant extends from approximately six weeks to six months after birth. It is during this time that the infant forms his first meaningful responses to his environment (Scott, 1969). This is the critical period for much of his important species imprinting. What happens when a fetus, who has inherited superior neurological potential from his parents, approaches his ninth month? He is hardly an "average" fetus. If his eventual IQ is projected to be 170 or more, his unstimulated brain has already "passed through" the first six months of what would ordinarily be his postnatal life. Consequently the optimal period for his primary socialization has passed.

Are autistic children "mutants of especially intelligent progenitors" as Treffert (1970) suggests they may be? Are autistics not just superior organisms, but do they have potential intellectual endowment so rare that it might occur in only the upper extreme limits of the distribution of intelligence? Interestingly, the prevalence of infantile autism is approximately 2 per 10,000 (Treffert, 1970). This prevalence is almost identical to the number of persons thought to have rates of intellectual development which might be expressed as quotients of more than 170 if tests were available with which we could measure them.

Suggestions for Research

The most obvious research need for testing the hypotheses stated in this paper revolves around the necessity for determining the intellectual levels of parents who have autistic children. These investigations should follow Kanner's definition of autism carefully to avoid contaminating the results with scores of parents whose children have "autistic symptoms" but are not truly early infantile autistics. It may be necessary for researchers to pay particular attention to the quality of parental intellectual activity as well as the quantity. This seems especially advisable in view of Kanner's (1965) observation that the parents of autistic children, while they may have meager incomes, are preoccupied with abstractions of a scientific, literary, artistic, or mathematical nature.

More research is needed to test the neurophysiological aspects of autism. In this respect, the work by Hutt and Hutt (1969) mentioned earlier seems an especially appropriate area of investigation. Their experiments led them to hypothesize that autistic children are in a high state of physiological and behavioral arousal. This paper has suggested that in autism it is believed that the fetus becomes habituated to a low level of stimulus variations. Reference was made previously to Schultz's (1965) statement that the level of stimulus variation the organism experiences early in its development is an important determinant of the optimal cortical arousal for adaptive behavior. Because there is little stimulation prenatally, the child's optimal cortical arousal is low. After birth, normal levels of environmental stimulation constitute an unaccustomed overload with consequent cortical hyperarousal.

Hutt and Hutt (1969) present data to support this hypothesis in their statement that autistic children

> will actively explore new objects or toys only when they have been exposed to the same situation on two or three occasions. Moreover, during prolonged or repeated exposure to the same environment, the EEG may gradually change from a low voltage, irregular, or desynchronized record to a medium/high voltage record characterized by a well-established theta/Alpha rhythmicity. . . . Both behavioral and electrophysiological abnormalities may be considerably reduced by either simplification of the environment or a prolonged period of familiarization with a constant situation [pp. 8–9].

Hutt and Hutt (1969) report two cases in which the administration of drugs known to repress the activity of the reticular system helped these children. More data, such as that supplied by Hutt and Hutt, would be extremely valuable.

Finally, there needs to be additional study of the relative organic and behavioral effects of environmental deprivation on primates of different intellectual potential during critical periods of primary socialization.

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A COMMENT ON D. J. MOORE AND D. A. SHIEK'S "TOWARD A THEORY OF EARLY INFANTILE AUTISM" [1]

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D. J. Moore and D. A. Shiek's theory concerning the origins of infantile autism is examined and found implausible. The theory is seen as based on the misuse or misunderstanding of two key concepts—imprinting and intelligence. Their notion of imprinting is mechanical and does not take into account the necessary environmental interaction which is characteristic of social development in man and other mammals. More serious is Moore and Shiek's belief that intellectual potential would be indicative of developmental precocity. Extensive empirical study indicates that there is no relation between the behavior of infants at birth and later IQ. It is suggested that all theories of autism based on the presumed precocity of children be abandoned as unproductive.

In their recent paper on early infantile autism, Moore and Shiek (1971) hypothesize that: "fetuses with an inherited high potential for intellectual superiority are developmentally ready for imprinting with human behaviors much before birth . . . [and become] imprinted on the restricted uterine environment [p. 451]." Their theory deserves comment as a potential source of confusion. While novel theories are clearly needed in the difficult and important field of early infantile autism, it is unlikely that Moore and Shiek's formulation will prove useful. Their paper implies a notion of development which does not do justice to the complexities of the process. While other points might be examined, this note will concentrate on Moore and Shiek's misunderstanding or misapplication of two fundamental concepts—imprinting and intelligence.

The realization that early human social development is based on instinctive processes that are related to similar mechanisms in other animals is an insight of profound importance (cf. Bowlby, 1969, for a review). However, as Bowlby and other have shown, primary socialization in humans (as well as other mammals) occurs well after birth and depends on extensive environmental interaction. For example, considerable perceptual organization must occur before the infant can begin to distinguish strange from familiar faces. Also, several relatively independent systems must be organized and focused on important social figures. Considered in these terms, the socialization process is better compared to Piaget's analysis of the construction of the permanent object than to elicited following in ducks. Also, Moore and Shiek fail to note that social responsiveness persists with no sharp cutoff for some time after the six-week to six-month period they postulate (Bowlby, 1969, p. 321 ff.). Thus, it is unlikely that any aspect of the normal human socialization process could function in the uterine environment to produce the profound and lasting effects of autism.

Moore and Shiek's misconceptions about the nature and development of intelligence are even more serious. They write: "If his [the autistic child's] eventual IQ is projected to be 170 or more his unstimulated brain has already [in utero] passed through the first six months of what would ordinarily be his postnatal life [p. 455]." It is a plausible hypothesis (though not well established) that autistic children have (or had) considerable intellectual potential, but the projection of an IQ of 170+ is totally unfounded. Moreover, their logic would seem to imply that all children with potential IQs of 170+ should be autistic, and that all children with $100 < IQ < 170$ should be ready for "imprinting" before the period of normal socialization. Such great variability in early development is, in fact, not seen, and there are nonautistic, highly intelligent people about.

These considerations indicate that Moore and Shiek hold an outmoded and incorrect notion of what intelligence is. First, there is an implicit assumption that social precocity could be predicted from a measure of IQ or

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mental age, and this is not likely to be true. Second, there is an erroneous assumption that presumed intellectual potential would have some implications for the state of the infant at the time of birth or before.

The idea that an IQ taken at any age can give an accurate mental age at any other age holds only if mental age/chronological age is the proper conceptualization of intelligence and if IQ is constant at all ages. Neither prerequisite is likely to be correct. One could argue that there is no necessary relationship between precocity and intelligence and that, in fact, the two notions are conceptually unrelated. There is, of course, an empirical relation between the acquisition of intellectual material and intelligence in the years of childhood. Regardless of the truth of this argument, however, one can safely say that there is no demonstrated relationship between behaviors measured in the first year or so of life and later intelligence, despite a long and extensive search for such relations (Bayley, 1970, p. 1173 ff.). In fact, factor-analytic studies suggest that the abilities which eventually contribute the majority of variance to intelligence do not emerge clearly until age four or so (Hofstaetter, 1954). Thus, even if autistic children were of high intellectual potential, there would be no grounds for assuming there was anything functionally different about them at birth.

Realization of these limitations on theorizing is important, since Moore and Shiek are not alone in confusing precocity and later intelligence. Rimland (1964), for example, bases his early brain-damage hypothesis on an assumption of intellectual potential. He postulates that the more rapidly developing brain of the highly intelligent child is more likely to suffer injury at birth. Moore and Shiek's rejection of this theory on an argument of biological adaptability is incorrect, since the brain is more susceptible to damage when it is undergoing rapid change. Rimland's hypothesis is gratuitous, however, for the same reasons that applied to Moore and Shiek's above. It is to be hoped that all theories of autism that attribute a critical role to the infant's intellectual potential will be abandoned, and that research on this difficult problem will examine potentially more profitable areas.

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A RESPONSE TO R. A. WEBB'S COMMENTS

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Webb (1972) accepts as a plausible hypothesis that autistic children have (or had) high intellectual potential. He also agrees that normal human social development has its basis in instinctive processes and is preceded by considerable perceptual organization. He further agrees that such perceptual organization is unlikely to occur in utero. Having agreed with all the major tenets of the theory, Webb proceeds to argue its conclusions by citing truisms from normal development as his evidence. He conveniently ignores the fact that early infantile autism is not a normal state.

Webb anticipates that if the theory is valid there should be degrees of autism. He notes correctly that such variability is not observed. The IQ limit of 170+ was not chosen capriciously or arbitrarily. As hypothesized in the original paper, a projected intellectual level of such magnitude is necessary for the critical six months to pass in utero. Less potential permits adequate opportunity for imprinting and/or perceptual organization after birth. There are probably time limits for imprinting in humans as in lower animals. After expiration of these limits, the developmental opportunity is lost. Imprinting seems to be an all-or-nothing-at-all phenomenon. In autism, it is hypothesized to be a nothing-at-all phenomenon. Without such basic perceptual organization, the human socialization process fails to proceed normally. Hence, Webb is correct in his observation that early infantile autism does not exist in degrees, but he is correct for reasons other than those he states.

Finally, Webb rejects the theory's rejection of Rimland's (1964) early brain damage hypothesis on grounds that the brain is more susceptible to damage when it is developing rapidly. His restatement of this commonly accepted truism lends nothing to the discussion. Significantly he failed to comment on the fact that the negative effects of sensory deprivation are relatively greater for genetically brighter organisms.

The theory demands flexibility of thought, rigidity of logic, and a renunciation of many time-worn and erroneous beliefs regarding autism. Hopefully, experimenters and theoreticians will scrutinize past, present, and future knowledge regarding the status of autistics in the light of new hypotheses. One avenue of exploration might attempt to determine how biochemical differences that appear to exist in the reticular activating systems of autistics approximate those found in normals who have undergone sensory dprivation.

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