American Handbook of Psychiatry

MENTAL RETARDATION

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e-Book 2015 International Psychotherapy Institute

From *American Handbook of Psychiatry: Volume 4* edited by Silvano Arietti

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I. Nature and Manifestations

Mental retardation is an arbitrary concept. It is often discussed in a medical and psychiatric context as if it were a homogeneous clinical entity, a disorder, or even a disease. This clinical viewpoint is reinforced somewhat circularly for physicians by the inclusion of the rubric in the classification of diseases in medicine and psychiatry. From a rigorous clinical standpoint, however, mental retardation is an imprecise, lumping term rather than a single, homogeneous entity. Unfortunately, a discussion of the "nature and manifestations" of mental retardation may further reinforce the notion that it is a naturalistic phenomenon.

The individuals who are assigned to the category of "mentally retarded" through clinical processes vary widely with respect to their genotypic, etiologic, anatomic, neurophysiologic, psychometric, cognitive, prognostic, and most other characteristics. For many, if not most, traits the scale of the phenotypic variation within the retarded population approximates that in the population at large. Since most traits vary continuously rather than discontinuously between the two populations, the retarded and nonretarded groups tend to merge without a sharp line of demarcation. Their separation ultimately depends upon the application of arbitrary criteria. While the differences between the populations are primarily those of degree rather

than of kind, there is a group of persons with mentally retarded behavior which represents a separate population. This group is set apart chiefly by its association with genetic mutations, cytogenetic abnormalities, and biomedical disorders of the central nervous system.

The heterogeneity within the category of mental retardation sharply limits the utility of the term for medical-psychiatric, educational, psychological, administrative, and scientific purposes. Because of the large numbers of variables which influence behavior within the total population of retarded individuals, different samples of the population tend to in-elude differing mixtures of subgroups. Even when carefully matched on a selected set of variables, homogeneous comparison groups are difficult to obtain. Over the years, this problem undoubtedly has confounded the interpretation and replication of innumerable studies in the field of mental retardation. The heterogeneity of the population highlights the fact that mental retardation is not a medical, psychological, educational, or sociological entity. For this reason, in part, mental retardation cannot be fully understood from the perspective of any one discipline.

Mental retardation does not stand alone as a field for scientific investigation. Its scientific roots lie within the subdivisions of developmental psychology, e.g., intelligence and cognition, attention and habituation, learning theory and behavior principles, language, and psycholinguistics. The

understanding of mental retardation is advanced only as far as investigations in the component fields of developmental psychology. Although particular developmental issues may be studied to advantage within the retarded population, developmental psychology is the mirror within which mental retardation must be viewed for scientific purposes.

Definition and Classification

An awareness of two constructs is necessary for an understanding of mental retardation. The first construct pertains to intelligence. It is assumed heuristically here that quantities of "intelligence" are normally distributed in the population. Within this framework, psychometric tests have been constructed to yield a normal distribution of test abilities with respect to a reference population. These tests provide an objective basis for measuring and defining low intelligence. Mental retardation is a second construct which is dependent upon the preceding one but is not identical with it. This construct is derived, in part, from the nontest behaviors of individuals at the low end of the distribution of measured intelligence. Within this framework, mental retardation may be attributed to persons with low measured intelligence whose adaptive behavior is also judged to be impaired or unintelligent. *Accordingly, low measured intelligence is not always synonymous with mental retardation.* From the perspective of learning theory, mental retardation reflects both insufficient and inappropriate learning. In the final analysis, however, "mental retardation" is a clinical term which refers to a reduced velocity and deviant direction of behavioral development.

The formal definitions of mental retardation, which are pertinent to clinical practice in the United States, are discussed in two reference books. These definitions reflect a consensus of prevailing professional views and are

subject to periodic revisions as concepts change. The *Manual on Terminology and Classification in Mental Retardation,* 1973 Revision, of the American Association on Mental Deficiency (AAMD) defines mental retardation as follows: "Mental retardation refers to significantly subaverage general intellectual functioning existing concurrently with deficits in adaptive behavior, and manifested during the developmental period." The interrelated *Diagnostic and Statistical Manual of Mental Disorders* (DSM-II, 1968) of the American Psychiatric Association (APA), which adapts the *International Classification of Diseases* (ICD-8, 1968) of the World Health Organization to American usage, defines mental retardation in somewhat similar terms as follows: "Mental retardation refers to subnormal general intellectual functioning which originates during the developmental period and is associated with impairment of either learning and social adjustment, or maturation, or both."

These definitions assign two essential properties to mental retardation, namely, low intellectual functioning and deficient adaptive behavior. In keeping with these definitions, dual criteria are required for an inference of mental retardation at an operational level, namely, measured intelligence at or below a selected level *and* demonstrated deficiencies in adaptive behavior. Strictly speaking, only the combination of a low IQ score plus deficient adaptive behavior equals mental retardation. Any other combination of impairment and nonimpairment in these two areas does not equal mental

retardation. “Learning disabilities,” many poverty-associated learning “failures,” and other forms of intellectual growth deflections are automatically excluded from the clinical category of mental retardation by these criteria, even though causal and functional continuities may exist between them.

Table 18-1. Levels of Severity of Mental Retardation by Measured Intelligence (AAMD)

		REPRESENTATIVE IQ SCORES	
DESCRIPTIVE LEVELS	STANDARD DEVIATION RANGES	STANFORD-BINET AND CATTELL (S.D. 16)	WECHSLER SCALES (S.D. 15)
Mild	—2.01 to —3.00	68-52	69-55
Moderate	—3.01 to —4.00	51-36	54-40
Severe	—4.01 to —5.00	35-20	39-25*
Profound	Below	19 and below	24 and below*

* Extrapolated.

The necessity for dual criteria for a “diagnosis” of mental retardation reflects a distrust of either criterion alone as a basis for identification. An exclusive reliance on intelligence test scores would result in the labeling of many individuals whose behavioral adequacy could be demonstrated in a variety of circumstances. In addition, minority-group members would be inappropriately included within the clinical category of mental retardation in appreciable numbers. In a sense, *the adaptive behavioral criterion is intended to provide a double check on the psychometric criterion in the clinical labeling*

process. Unfortunately, the relative lack of precision of adaptive behavioral scales in separating retarded from nonretarded behaviors at borderline levels leaves considerable room for subjective judgments in this area. These judgments may ultimately be crucial in the labeling of large numbers of children, especially in minority groups. Because of the subjective latitude which enters into the application of the adaptive behavioral criterion, the category of mental retardation cannot be clearly delineated throughout its entire range. Except for measured intelligence, the available diagnostic tools lack sufficient discriminatory power to be useful for identification in many instances.

The APA manual distinguishes five levels of mental retardation, based entirely upon measured intelligence in this system; adaptive behavior is not considered. Levels are delineated by ranges of IQ scores (test unspecified) as follows: (1) borderline, IQ 68-85; (2) mild, IQ 52-67; (3) moderate, IQ 36-51; (4) severe, IQ 20-35; and (5) profound, IQ less than 20. The borderline level in this classification has been widely criticized because it would result in the potential inclusion of at least 16 percent of the total population within the category of retardation.

The AAMD manual, on the other hand, classifies the severity of mental retardation independently in the areas of measured intelligence (i.e., test behaviors) and adaptive behavior (i.e., nontest behaviors). In each behavioral

domain four levels of severity are distinguished. The criterion for mental retardation is fulfilled with respect to measured intelligence by scores which are more than two standard deviations below the mean of a standardized test. In the AAMD system, the controversial borderline category of the APA manual is eliminated. The remaining four levels of severity with respect to intelligence test scores are delineated by standard deviation intervals as shown in Table 18-1.

The adaptive behavioral criterion is fulfilled in the AAMD system when rating scales and observer judgments lead to the conclusion that adaptive behavior is significantly below the population norms for the age group. Adaptive behavior here refers to the behavioral phenomena which contribute most strongly to the social perception of retardation. Areas of interest in this context include self-help and personal independence, sensorimotor development, communication, socialization, reasoning, and judgment in meeting societal expectations, academic skills pertinent to community living, self-direction and responsibility, occupational attributes, maladaptive behaviors, and the like. Levels of adaptive behavioral deficiency are labeled mild, moderate, severe, and profound. At its upper limit the mild level of deficiency corresponds to a significant, negative deviation from population norms (analogous to -2 standard deviations for intelligence test scores, but lacking in this degree of precision). At its lower limit the profound level of deficiency corresponds to an almost complete lack of adaptation. The AAMD

manual provides descriptive patterns of adaptive behavioral functioning by age and level which may be helpful in estimating severity. It is intended, however, that standardized scales, e.g., the Vineland Social Maturity Scale and the AAMD Adaptive Behavior Scales, will be used in combination with clinical judgment to classify levels of adaptive behavioral deficiency.

Biomedical "Causation"

In a strictly logical framework, medical diagnoses, diseases, and pathological processes do not define mental retardation; only behavioral criteria do this. Conversely, behaviors which are labeled mentally retarded do not define or signify pathological processes or medical disorders; only biomedical criteria do this.

There are no direct relationships between specific medical diagnoses and specific learning or adaptive behavioral characteristics. Many interacting variables, including innumerable environmental contingencies, are interposed between biomedical phenomena and particular behaviors. From this perspective, a medical diagnosis or disorder can never be viewed as the sole or proximate "cause" of mental retardation. Lately, the behaviorists have been particularly vocal in indicating that the explanatory power of medical diagnoses for mentally retarded behavior is limited and that the techniques of behavioral intervention are independent of the dictates of medical etiologies.

General associations are noted, however, between mental retardation and various medical diseases, syndromes, disorders, findings, and events. Presumably, these associations are mediated through pathological processes which affect the response capacities for learning and predispose individuals to behaviors which may be labeled abnormal. The correlation between a medical diagnosis and mental retardation approaches unity in the case of

Down's syndrome or mongolism. Here, the association is so strong that the recognition of the medical syndrome at birth is considered tantamount to the identification of mental retardation, even though the behavioral criteria may not be fulfilled until some time in the future. A number of additional medical disorders are correlated with retardation in this way at different levels of probability. The AAMD manual includes a separate medical classification for the coding of conditions which are presumed to be etiologically associated with retardation. Similar coding requirements are contained in the APA manual.

When viewed from a medical perspective, the mentally retarded population is roughly separable into the following two groups: Group 1, a small group with abnormal medical findings, and Group 2, a large group without abnormal medical findings. The divisibility of the retarded population into two groups on the basis of medical findings has been noted in the literature for many years. In the past, various terms have been applied to these groups as follows: extrinsic and intrinsic, secondary and primary, exogenous and endogenous, organic and subcultural, pathological and physiological, and the like. Although these terms imply a binary view of causation which is no longer tenable, the separation into two groups is useful for medical diagnostic purposes. The relationships between these two groups and the major etiological categories which are postulated within the retarded population are shown in Table 18-2.

Table 18-2. Relationships between two medically distinguishable groups within the retarded population and postulated etiological categories

MEDICAL DIAGNOSES (20-25 percent of total retarded population)	MULTIFACTORIAL-POLYGENIC (75-80 percent of total retarded population)	
Single mutant genes (metabolic diseases)		
Malformation syndromes (including cytogenetic)	Complex causation	Tail of "normal" distribution of intelligence.
	low socioeconomic status	
	learning experiences	
Sequelae of prior disease (prenatal, perinatal, postnatal)	disadvantaged or predisposing to retardation	
	subclinical or undiagnosed prior disease	
Miscellaneous progressive neurological diseases		
Group 1. Abnormal medical findings: includes more than 20-25% of the total retarded population.	Group 2. No abnormal medical findings.	

From a practical standpoint, the yield of medical diagnoses will be limited to Group 1. Some individuals in this group will have specific medical diagnoses which can be currently and directly verified; included in this category will be the metabolic diseases, malformation syndromes, and certain progressive neurological diseases, e.g., subacute sclerosing panencephalitis. Other individuals in this group will have medical disorders which cannot be

currently or directly verified, because the findings represent sequelae of conditions which were active for a limited time only during the prenatal, perinatal, or postnatal periods. In these instances, the applicable diagnoses must be inferred retrospectively; the validity of these inferences will depend upon the degree of specificity of the residual findings and/or of the available historical data. If neither the findings nor the history justify a specific diagnostic inference, the abnormal findings must be considered to be idiopathic or undiagnosable. Individuals with idiopathic findings contribute to the size of Group 1 in indeterminate numbers. It is commonly estimated, however, that persons with assignable medical etiologies account for 20-25 percent of the total retarded population. Table 18-2 shows the proportionate representation (estimated) of medical findings and diagnoses within the retarded population. Table 18-3 presents a simplified medical classification which can serve as a framework for the medical diagnostic approach. Representative medical disorders which are included in Table 18-3 for illustrative purposes will not be discussed here.

Group 1 is delineated by five sets of medical findings which provide access to the etiologic categories in Table 18-3. Since these findings are always presumptive of a medical etiology or "cause" for mentally retarded behavior, the medical examination should be directed toward their identification. These findings are not mutually exclusive and may occur in any combination in a single individual. The five sets of findings with a high

diagnostic payoff in relation to the etiologic categories in Table 3 are the following: (1) a cluster of abnormal neurological signs; (2) neurological deterioration or developmental regression; (3) a malformation cluster; (4) positive laboratory tests for abnormal concentrations of metabolites in body fluids; and (5) a documented neurological disease in the past. These findings and their diagnostic implications will be discussed briefly below.

Positive neurological signs are, perhaps, the most frequent indications of medical abnormality which are associated with Group 1. Significant here are one or more localizing or major signs; two or more nonlocalizing, minor, or "soft" dysfunctional signs; or a developmental or IQ which is three standard deviations or more below the mean of a standardized test (roughly equivalent to a score of 50 or below). Since the latter finding is regularly associated with major pathology of the central nervous system (CNS), a low IQ per se at these levels is usually interpreted as an abnormal neurological sign. Identifiable medical diseases and syndromes are heavily concentrated among individuals at these low IQ levels. If identified, positive neurological signs dictate additional investigations for disorders in the categories of metabolic abnormalities, malformation syndromes, and progressive neurological diseases, as shown in Table 18-3.

Neurological deterioration or developmental regression points to currently active diseases. Evidence for deterioration should be obtained from

documented, longitudinal observations whenever possible, but lacking this, from the history as provided by family members or other significant nonprofessionals. Rates of regression may be rapid or exceedingly slow and difficult to recognize, either intermittent or continuous. If abnormal neurological signs are identified in a person with mental retardation, active disease of the CNS must be considered. Evidence for progressive neurological deterioration points to the metabolic disorders and active neurological diseases as shown in Table 18-3. Evidence for nonprogressive neurological abnormalities (especially when accomplished by a continuous advance in developmental level) points to the residual effects of prior disease. In these instances, the etiology may or may not be assignable.

Table 18-3. Simplified Classification of Medical Disorders Which May Be Etiologically Associated with Mentally Retarded Behavior

METABOLIC DISEASES SINGLE MUTANT GENES	MALFORMATION SYNDROMES AND CLUSTERS, CYTOGENETIC AND NONCYTOGENETIC	NEUROLOGICAL SEQUELAE OF TIME-LIMITED DISORDERS: PRE-, PERI-, OR POSTNATAL
Phenylketonuria	Malformation cluster with a cytogenetic anomaly; pattern of anomalies may or may not be specific	Prenatal
Histidinemia	Down's syndrome	Teratogenic agents
Homocystinuria	E_1-trisomy (47,18+)	Maternal phenylketonuria
Maple syrup urine disease	D_1-trisomy (47,13+)	Rubella virus
Argininosuccinic acidemia	Cat-cry syndrome (46, 5P-)	Cytomegalovirus
Glycosaminoglycans: mucopolysaccharidoses	Malformation cluster without demonstrable cytogenetic	Herpesvirus hominis
Galactosemia		Perinatal
Gangliosidoses:		Low birth weight

Tay Sachs, etc.	abnormality; recognizable pattern of anomalies Acrocephalosyndactyly (Apert's syndrome) Oral-facial-digital syndrome Rubenstein-Taybi syndrome Cornelia de Lange syndrome Malformation cluster; no demonstrable cytogenetic abnormality; no recognizable pattern	prematurity; small for gestational age Malnutrition Asphyxia; acidosis Hypoglucosemia Rh isoimmunization and kemicterus Postnatal Child abuse Intracranial trauma Meningitis Encephalitis

Malformation clusters are important contributors to the abnormal medical findings which characterize Group 1. Significant here is the combination of two and, especially, three or more malformations, either of major organs or of minor structures, including dermatoglyphics. In some instances, the cluster of anomalies may be associated with a cytogenetic abnormality. In other instances, the cluster of anomalies may conform to a recognizable syndromic pattern in the absence of a demonstrable cytogenetic abnormality. In still other instances, the cluster of anomalies may be unclassifiable and unassociated with a cytogenetic abnormality. Malformation clusters suggest disordered embryogenesis. If accompanied by mental retardation and abnormal neurological signs, they generate an inference that structural abnormalities of the CNS are also present. Regardless of the

specificity of the clinical pattern, malformation clusters provide an indication for cytogenetic studies. Several useful catalogues are now available to aid the physician in the clinical identification of malformation syndromes.

Positive laboratory tests for abnormal concentrations of metabolites in body fluids, or for deficient enzyme activity, point directly to the genetic disorders of metabolism. Pre-determined protocols of qualitative or semi quantitative screening tests for metabolic diseases, especially for the amino acid disorders, are often applied in the medical evaluation of individuals with mental retardation. Positive results from these tests must always be confirmed by specific procedures. Assays for enzyme activity are undertaken selectively in situations in which a specific disease, e.g., a ganglioside storage disease, is suspect on the basis of the family history or clinical findings.

A *documented disease or insult* to the central nervous system in the past may be crucial for the subsequent assignment of etiology in instances of nonprogressive neurological abnormalities. Presumably, abnormalities of this type represent the sequelae of prior time-limited diseases. Precise diagnoses of these diseases must be established during their active phase. Retrospective diagnoses, which are based entirely upon the recall of events, or nonspecific, residual findings, are at best speculative. Prenatal or fetal diseases are presumed to be major contributors to the residual category in Table 18-3. Since intrauterine disorders are exceptionally difficult to identify, either

concurrently or retrospectively, the etiology of many nonprogressive neurological abnormalities cannot be assigned. The findings are labeled idiopathic under these circumstances.

These five sets of findings are not the only presumptive indications for a medical etiology or "cause" in individuals with mental retardation. Many other pathological findings (e.g., cutaneous lesions of neuroectodermal disorders, chorioretinitis, microcephaly, intracranial calcifications, and the like) may be identified during the course of medical examinations and contribute to diagnostic or etiologic inferences. In addition to the neurological abnormalities which may be elicited by the medical examinations of physicians, psycholgists regularly find evidence for impaired cortical and neurointegrative functions during psychological and psychoeducational testing. In the future, neurophysiological techniques may also be applied clinically and permit the recognition of subtle biological abnormalities which now cannot be identified, e.g., in the areas of evoked potentials, attention and habituation, expectancy patterns, sleep patterns, and the like. Developments of this type may expand the numbers of individuals with retardation in whom biological abnormalities can be demonstrated.

Group 2 of the retarded population is distinguished by an apparent absence of the abnormal medical findings which characterize Group 1. Group 2 includes the majority, perhaps 75-80 percent, of the total retarded

population. The severity of the retardation here is limited to the mild level, whereas all levels are included in Group 1. Several etiologic subgroups undoubtedly exist within Group 2 which are not separable through medical technology. The causes of mentally retarded behavior here are believed to be multifactorial and to involve complex interactions between polygenic inheritance factors, experiential factors, and subclinical medical factors.

Polygenic "Causation"

Geneticists estimate that 70 percent of all mental retardation can be encompassed etiologically within the framework of polygenic inheritance of a graded characteristic. In this context, each of the segregating polygenes is assumed to have a small effect on trait variation by comparison with the total variation observed for the trait. Gene effects of this type presumably account for "physiological" trait variations which conform to a normal or continuous distribution. By contrast, major or single mutant gene effects may account for discontinuous or "pathological" traits.

Tests for the measurement of intelligence have been constructed and standardized to yield a normal distribution of test abilities or scores with respect to a reference population. These tests, in turn, generate the assumption that a trait "intelligence" exists which is normally distributed and polygenically determined. If normally distributed, 99.73 percent of intelligence test scores will predictably lie within three standard deviations above or below the mean of a standardized test. On the Stanford-Binet with a standard deviation of 16, scores from 52-148 would be included within this range; on the Wechsler Scales with a standard deviation of 15, scores from 55-145 would be included. These ranges presumably reflect the "physiological" spread of intelligence due to polygenic inheritance.

A normal frequency distribution also predicts that 2.14 percent of

intelligence test scores will lie between two and three standard deviations below the mean. Many individuals with scores in this range are labeled mildly retarded. Those who are distinguished by an absence of abnormal medical findings may reflect the low end of the continuum of intelligence due to the segregation of polygenes. The observed frequency of IQ scores in this range, however, exceeds the predicted frequency due, presumably, to medical pathology and other influences which will be examined subsequently.

The concept of polygenic inheritance in relation to measured intelligence has been supported by numerous studies which involve correlational pairings. These studies have indicated that intragroup resemblance in intellectual abilities increases in proportion to the degree of genetic relationship, i.e., the greater the gene overlap the greater the similarity in intelligence, whether or not the paired individuals have shared the same environment. Empiric risk figures for mild mental retardation, as derived from family studies, have also been consistent with a polygenic hypothesis. Finally, the distribution of IQ scores along a social class gradient has been regarded as consistent with the distributional expectations due to polygenic inheritance given conditions of social mobility and assortative mating.

Multiple studies in white populations in more or less uniform environments have estimated that heritability accounts for 60-80 percent of

the total variance in intelligence test scores. On the assumption that the variance attributable to heritability is equally high in all groups, it has been reasoned that differences in mean IQ scores between black and white populations or social classes are genetically determined, i.e., are due to differences in the distribution of genotypes for high and low. Environmentalists, on the other hand, have argued that statistical estimates of heritability apply only to a particular population in a relatively constant environment. Measures in another population with a different distribution of environmental characteristics do not necessarily show the same proportion of genetic and environmental variances. From this standpoint, it is hypothesized that heritability will account for more of the variance in optimal environments and less of the variance in suboptimal environments. Accordingly, differences in mean IQ scores between racial and social groups are attributed to environmental disadvantage. It is concluded in this framework that a phenotypic distribution of IQ scores neither reflects the distribution of genotypes for intelligence nor is immutable.

Obviously, psychometric instruments influence the phenomena which are measured and the conclusions which are reached concerning polygenic and sociocultural "etiologies." If cognitive capacity is not fully matched by cognitive competence or performance in response to the demands and conditions of a test, an artifact of measurement will occur. In recent years, interest in the concept of general intelligence has yielded somewhat to the

study of differentiated intelligence, i.e., of the various subabilities which are hypothesized to affect mental functioning. If not necessarily clarifying the genotypic issues, such studies provide a refined view of the differences in performance between subgroups of the population (or individuals) under specific demand situations. While some abilities may be more resistant to training and experience than others, *psychometric items are always culture-bound.*

Sociocultural "Causation"

As has been noted, the majority of individuals in the retarded category are classified as mildly retarded. Usually, no single etiological or causal influence can be identified in this group. Presumably, mental retardation at this level represents a final symptomatic pattern of IQ and adaptive behavioral effects due to multiple interacting variables. Because correlations with social class and ethnic minority status are high in this group, the terms "cultural-familial" and "sociocultural" are frequently applied to it.

Epidemiological and prevalence studies provide a useful perspective from which to view the social implications of mild mental retardation. It should be noted first that all studies of prevalence have shown that severe mental retardation (i.e., moderate, severe, and profound levels) is distributed across social classes without a significant gradient. Prevalence has been remarkably constant in all studies at approximately three to four per 1000 children between five and eighteen years of age. Virtually all individuals with severe retardation have significant signs of medical pathology. For this reason, biomedical causalities are thought to account for the excess frequency of low IQ scores (below 52-55) beyond the predictions of a normal frequency distribution.

The prevalence of mild mental retardation, on the other hand, has varied between studies according to the criteria, instruments, population

characteristics, and sampling techniques employed, and the locale or country studied. Roughly, prevalence ratios by severity are as follows: mild twenty, moderate four, severe one. Aside from actual prevalence rates, however, three important epidemiological characteristics of mild mental retardation emerge from these studies.

The most striking epidemiological feature of mild mental retardation is its disproportionate social distribution, i.e., its social gradient. The discrepancy between the top and the bottom of the social scale is marked enough to suggest that this form of mental retardation is virtually specific to the lowest social classes. A dramatic increase in prevalence occurs particularly at IQ levels equal to or greater than sixty between social classes 3 to 5 (as determined by the occupation of the head of the household). As a generalization, it may be concluded that the prevalence of mild mental retardation increases by a factor of two for each step downward in social class.

The second outstanding epidemiologic characteristic of mild mental retardation is its disproportionate age distribution. This distribution has been designated as the "schologenic hump." Mild mental retardation has a low prevalence in the preschool years, increases rapidly in frequency during the early school years, peaks dramatically during puberty and adolescence, and declines sharply again thereafter, in part, as a reflection of the role of the

schools in identification. In some studies, the prevalence of mild mental retardation reaches twenty to forty per 1000 population in the age group 10-14 years. It is assumed that movement of many individuals into and out of the category of mild mental retardation accounts for these age specific prevalence rates. The importance of the behavioral demands of particular social settings and of the labeling process on the prevalence of mild mental retardation is highlighted by these studies. Since the label of mental retardation is difficult to remove, professionals should be exceedingly wary of stigmatizing anyone at the mild level who might move out of the category.

The third outstanding epidemiological characteristic of mild mental retardation is the frequent absence of abnormal medical findings. This feature was documented most clearly by Birch et al. in a superb epidemiological study of mental retardation in eight- to ten-year old children in Aberdeen, Scotland. In this homogeneous white population, one third of the children with IQ scores above 50 (up to 75) had abnormal neurological findings as defined by one localizing or two nonlocalizing signs. However, only one fourth of children with IQ scores equal to or greater than 60 (up to 75) had abnormal neurological findings as defined. The numbers of children with neurological abnormalities in the latter group were ten times greater than in a sample of normal children. In other studies, it has been estimated that 20-50 percent of mildly retarded children will present abnormal medical findings.

Medical findings and social class often interact to influence the prevalence of mild mental retardation. This relationship is demonstrated dramatically with respect to low birth weight and severe perinatal stress. Low-birth-weight infants (above 3% lb.) in upper social-class families reveal minimal IQ effects, while decreasing birth weight is associated with a marked increase in the frequency of low IQ scores in other social classes. Severe perinatal stress also appears to be compensable in good postnatal environments. The interaction between reproductive and environmental hazards produces a cumulative risk which has a profound effect on the IQ levels of lower class children who are exposed to the severest forms of perinatal stress.

Epidemiological evidence suggests that mild mental retardation is not randomly distributed within low social classes. In a study of prevalence in an American slum, for example, Heber demonstrated that mild retardation was concentrated in families of mothers with low IQ scores. Although mothers with an IQ less than 80 made up 40 percent of the total surveyed, they accounted for four fifths of the children with IQ scores less than 80. The offspring of these mothers experienced a marked decline in measured intelligence between three to five years which was followed by a further modest decline until the average measured IQ at twelve to fourteen years approximated the maternal average of 68. The probability of children testing between IQ 55-67 on the Wechsler Scales at twelve to fourteen years was

fourteen times greater if the IQ scores of their mothers were in this range than if the mothers' scores were at or above IQ 100. The mothers in this study were comparable in economic level, living conditions, educational background, and the like, and varied only on measured intelligence. This study suggested that a threshold level of maternal IQ might be correlated with most instances of mild mental retardation in low social classes.

Other studies have seemingly corroborated the existence of a progressive form of intellectual retardation (and mild mental retardation) which is associated with low social class for some children (See references 15, 27, 31, 32, and 55). Cross-sectional data have indicated that groups of disadvantaged children compare unfavorably with children of high socioeconomic status on intelligence test measures at least by four years of age, and differences tend to increase with increasing age. Groups of disadvantaged children also perform below the national average on all measures of school achievement at all grade levels, and the absolute discrepancies in age levels increase with time. Not all children in low socioeconomic circumstances, however, reveal indications of progressive intellectual or mental retardation.

It is commonly hypothesized that progressive retardation in low social classes results from a deprivation of experiences which are necessary for the development of cognitive competencies for academic learning. Untangling the

web of class and specific environmental differences which may contribute to differences in intellectual functioning, however, is a complicated process. Child-rearing practices which favor one cognitive style rather than another, specific types of class-related interpersonal communications which result in specific deficits in intellectual functioning, differences in linguistic styles and form which make mainstream English syntax and vocabulary a second language, differences in patterns of reinforcement contingencies, differences in motivation, differences in pregnancy, nutrition, and health factors, and the importance of social "models," all and more have been postulated and explored.

Race and social class contributions to the mildly retarded category also interact. White group mean IQ scores and black group mean IQ scores may differ by as much as one standard deviation or roughly 15 points. Among the blacks, 18 percent may score below IQ 70, while only two percent of whites score in this range. Mercer has shown, however, that low scores for black and Mexican-American children in Riverside, California, were correlated more with sociocultural status than ethnic or racial background. In each group, mean IQ scores converged progressively toward standard norms as sociocultural background characteristics were successively controlled for similarity to the dominant society.

Mercer's study demonstrated the importance of the labeling process in

the determination of the category of mild mental retardation. As a result of her findings, she is currently developing differential ethnic norms for adaptive behavior by subculture in order to obviate the labeling of school children who meet social expectations and demands in their own cultural group. In Mercer's view, low IQ scores predict the need for appropriate educational supports, while adequate adaptive behavior in relation to an appropriate (nonschool) reference group should predict the capacity to fill an adult role acceptably. From this perspective, the category of mild mental retardation should be reserved for those children who are "comprehensively" retarded in terms of IQ scores and adaptive behavioral criteria as applied within a cultural context. Much of the confusion regarding the category of mild retardation and the prognosis for those assigned to it results from the unavailability of discriminating criteria for adaptive behavioral adequacy.

From a dynamic viewpoint, the test and nontest behaviors which define mental retardation always reflect the interaction of multiple variables, not only at mild levels but at all levels of severity. In this sense, the assignment of the cause of mental retardation to a single variable or etiology represents an oversimplification. At any given time, mentally retarded behavior may be conceptualized as resulting from an interplay between the person's *response potentialities* (i.e., the polygenetic, physiological, and pathological limitations imposed upon his reacting and coordinating systems), his *learned responses* or competencies (i.e., his cumulative or antecedent learning history as

acquired through interactions with his personal, cultural, social, and physical environments), and his *current stimulus-response environment* (i.e., the specific circumstances in which his present specific behaviors are arising).

Mutability of IQ

Logically, an increase in IQ scores above the criterion level for mental retardation should result in a "cure." IQ changes with time occur at all levels of retardation, either upwardly or downwardly and with or without intentional intervention. As noted previously, the progressive downward movement of group mean IQ scores of low socioeconomic status children corresponds with an increasing frequency of mild mental retardation at puberty and adolescence. In an institutional population, Fisher and Zeaman found the growth of mental age (MA) to be roughly linear between the ages of five and sixteen years at all levels of retardation. The IQ scores declined precipitously during this period, however, due to the slow rate of MA increase. Since the mildly retarded group continued to exhibit MA growth through the late thirties, IQ scores in this group subsequently rose after age sixteen years. Non-institutionalized children with culturally associated retardation have also been reported to make IQ gains in early adulthood.

In general, the greatest potential for IQ change is associated with the most unfavorable social and educational origins of a group. The frequency and extent of IQ changes in retarded children from advantaged environments remain moot questions, especially as consequences of training and education. Intelligence test scores, however, place a primary emphasis upon the products of cognition. During the past two decades, numerous investigations

have explored the process of learning among the retarded (See references 5, 7, 24, 51, 54, and 57). These studies have contributed to a growing technology of education which has permitted retarded individuals to learn complex tasks previously thought to be impossible. Even without IQ change, remarkable changes in performance can be achieved through strategies derived from discrimination learning, information theory, learning theory, and the like.

Some aspects of an effective educational program for the retarded child may involve the following: (1) the definition of specific objectives for learning in accord with the present stage and learning characteristics of the child; (2) the ordering of the sequential tasks necessary to achieve a learning objective; (3) the ordering of the physical and spatial environment to direct attention to the relevant stimulus dimensions of tasks; (4) the ordering of tasks in magnitudes of difficulty which insure a high probability of successful achievement; (5) the use of a range of reinforcing techniques which is appropriate to the task and child; (6) the provision of an appropriate response model which the child can also emulate and imitate; and (7) the creation of a social relationship which enhances motivation and encourages movement toward autonomy.

Mutability of Adaptive Behavior

Logically, an increase in adaptive behavior above the criterion level for mental retardation should also result in a "cure" even without an IQ increase. There are no generalized behaviors, however, which are specific to retardation. At mild levels, adaptive behavioral retardation is often defined in relation to the specific demands of a particular social setting, especially the schools. Rightly or wrongly, the child's responses in coping with the particular environment serve as the basis for the definition of retardation within that environment. Adaptive behavioral change under these circumstances requires a clear delineation of the behaviors which are contributing to the social definition and a specific strategy for intervention, an endeavor which is often questionable from an ethnic and subcultural perspective.

There are behavioral phenomena, however, which distinguish a child as atypical in virtually all social settings. Basic social skills which are appropriate to the chronological age of the child, for example, may be missing from the behavioral repertoire. These missing skills may relate to toileting, feeding, dressing, bathing, ambulation, play, and communication. The complexity of the skills which are lacking may increase at successive ages. These absent behaviors have the effect of depriving the retarded person of independence and movement through the expected range of social settings

for age. There are also maladaptive behaviors which distinguish a person as atypical. These behaviors may involve stereotyped or repetitive acts which consist of body rocking, head rolling, hand flapping, bruxism, twirling, pill-rolling, unusual limb posturing, object spinning, vocal sounds, and the like. A closely related group of self-injurious behaviors may also be observed which includes head-banging, face slapping, self-biting, trichotillomania, and eye poking. Tantrums, aggressive and destructive acts, explosive outbursts, hyperkinesis, lack of impulse control, unconcealed masturbation, inappropriate channeling of erotic feelings, compulsions, unusual fears, negativism, and withdrawal, all represent behaviors which are considered to be undesirable in most settings, and hence, maladaptive. Manifestations of these types are generally independent of social class and are closely correlated with IQ levels and abnormal medical findings. A number of scales are available for the assessment of adaptive behavior in these areas which may serve as useful guides for the specification of training objectives (AAMD Fairview, Cain-Levine, Balthazar, and Watson).

A vast experimental and clinical literature now supports the potency of the techniques of behavior modification for changing behaviors of the preceding types. Through these techniques, maladaptive behaviors can become less frequent; likewise, adaptive behaviors which are available to the child, but infrequent, can become more frequent. Developmentally appropriate behaviors which are not in the behavioral repertoire may also be

generated through the chaining of separate steps in a complex behavior or through shaping, i.e., the successive approximation of a terminal behavior. Through these techniques, even severely and profoundly retarded children can learn toileting, self-feeding, self-dressing, communication, and other skills. At the present time, therefore, it is probably appropriate to conclude that all retarded children can be helped to become less retarded in the area of adaptive behavior.

The professional framework in which a child is viewed may determine the interpretation or label which is applied to his adaptive behavior. Many of the behaviors which have been described here may be interpreted in a psychiatric context. In this light, several epidemiological studies have shown that mental retardation is associated with a wide range of psychiatric disorders. In general, the frequency of psychiatric diagnoses is inversely correlated with IQ levels. Although psychoses, especially in mentally retarded adults, are frequently noted, the psychiatric disorders of children with retardation are heterogeneous and nonspecific. The traditional psychiatric syndrome of infantile autism is highly correlated with mentally retarded behavior.

Conclusion

Mental retardation can be viewed from at least three perspectives, namely, medical, behavioral, and sociological. The *medical orientation* (including the genetic) tends to assign the causes for the behaviors which are labeled abnormal to deficiencies and pathological processes *within* the individual. In this context, the focus is often directed toward the assignment of etiology to biomedical diseases and events. Mental retardation may be considered to be a unitary condition here and the individual with retardation to be a patient who requires treatment to achieve health or normalcy. The *behavioral orientation* tends to ignore medical and other etiologies and relates the behaviors which are labeled maladaptive to learning experiences and current-stimulus response events within particular environments; in this context, many of the retarded behaviors are considered to be trainable, manipulable, or extinguishable under specified environmental conditions. The person with retardation is considered here to be one who needs environmental modification to permit appropriate learning and behavior. The *sociological orientation* focuses on differences in the distribution of socioeconomic-cultural variables and upon the labeling process per se. In this context, mental retardation is seen as an artifact of society, of social influences and of social organization. Mental retardation here is regarded as essentially non-clinical, and the burden of causation is placed *outside* the individual.

The heterogeneity of the retarded group defies a single, all-inclusive conceptualization. The medical or clinical perspective, however, limits the view of retardation to instances which are categorized through professional activities and clinical procedures. The sociological perspective broadens the scope of interest to include equivalent sets of behavioral phenomena whether or not they are clinically labeled. Although not mutually exclusive, these two orientations generate different views of the nature and manifestations of mental retardation and somewhat different strategies for intervention. Social change rather than individual change represents the primary difference in emphasis.

Bibliography

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders,* 2nd ed., Washington: American Psychiatric Association, 1968.

Ampola, M. G. "Phenylketonuria and Other Disorders of Amino Acid Metabolism," *Pediatric Clin. North Am.,* 20 (1973), 507-536.

Balthazar, E. E. *Balthazar Scales of Adaptive Behavior.* Champaign, Ill.: Research Press, 1971.

Baumeister, A. A. and R. Forehand. "Stereo-typed Acts," in N. R. Ellis, ed., *International Review of Research in Mental Retardation,* Vol. 6, pp. 55-96. New York: Academic, 1973.

Baumeister, A. A. and G. Kellas. "Process Variables in Paired-Associate Learning of Retardates," in N. R. Ellis, ed., *International Review of Research in Mental Retardation,* Vol. 5, pp. 221-270. New York: Academic, 1971.

Bergsma, D., ed. *Birth Defects: Atlas and Compendium.* Baltimore: Williams & Wilkins, 1973.

Berkson, G. "Behavior," in J. Wortis, ed., *Mental Retardation and Developmental Disabilities: An Annual Review,* Vol. 5, pp. 55-71. New York: Brunner/Mazel, 1973.

Birch, H. G. and J. D. Gussow. *Disadvantaged Children: Health, Nutrition and School Failure.* New York: Grune & Stratton, 1970.

Birch, H. G., S. A. Richardson, D. Baird et al. *Mental Subnormality in the Community: A Clinical and Epidemiological Study.* Baltimore: Williams & Wilkins, 1970.

Bortner, M. and H. G. Birch. "Cognitive Capacity and Cognitive Competence," *Am. J. Ment. Defic.,* 74 (1970), 735-744.

Burt, C. "Intelligence and Social Mobility," *Br. J. Statist. Psychol.,* 14 (1961), 3-24.

Cain, L. F., S. Levine, F. F. Elzey. *Cain-Levine Social Competency Scale.* Palo Alto: Consulting Psychologist's Press, 1963.

Cavanagh, J. B. *The Brain in Unclassified Mental Retardation.* Baltimore: Williams & Wilkins, 1972.

Crome, L. and J. Stern. *The Pathology of Mental Retardation.* London: Churchill, 1967.

Das, J. P. "Cultural Deprivation and Cognitive Competence," in N. R. Ellis, ed., *International Review of Research in Mental Retardation,* Vol. 6, pp. 1-53. New York: Academic, 1973.

Dingman, H. F. and G. Tarjan. "Mental Retardation and the Normal Distribution Curve," *Am. J. Ment. Defic.,* 64 (1960), 991-994.

Doll, E. A. *Vineland Social Maturity Scale.* Circle Pines, Minn.: American Guidance Service, n.d.

Drillien, C. M. *Growth and Development of the Prematurely Born Infant.* Baltimore: Williams & Wilkins, 1964.

Eastham, R. D. and J. Jancar. *Clinical Pathology in Mental Retardation.* Baltimore: Williams & Wilkins, 1968.

Ellingson, R. J. "Neurophysiology," in J. Wortis, ed., *Mental Retardation: An Annual Review,* Vol. 4,

pp. 123-139. New York: Grune & Stratton, 1972.

Erlenmeyer-Kimbling, L. and L. F. Jarvik. "Genetics and Intelligence: A Review," *Science,* 142 (1963), 1477-1479.

Farber, B. *Mental Retardation: Its Social Context and Social Consequences.* Boston: Houghton Mifflin, 1968.

Fisher, M. and D. Zeaman. "Growth and Decline of Retardate Intelligence," in N. R. Ellis, ed., *International Review of Research in Mental Retardation,* Vol. 4, pp. 151-191. New York: Academic, 1970.

----. "An Attention-Retention Theory of Retardate Discrimination Learning," in N. R. Ellis, ed., *International Review of Research in Mental Retardation,* Vol. 6, pp. 169-256. New York: Academic, 1973.

Fox, R. M. and N. H. Azrin. *Toilet Training the Retarded.* Champaign, Ill.: Research Press, 1973.

Gardner, W. I. *Behavior Modification in Mental Retardation.* Chicago: Aldine-Atherton, 1971.

Girardeau, F. L. "Cultural-Familial Retardation," in N. R. Ellis, ed., *International Review of Research in Mental Retardation,* Vol. 5, pp. 304-348. New York: Academic, 1971.

Gottesman, I. I. "Genetic Aspects of Intelligent Behavior," in N. R. Ellis, ed., *Handbook of Mental Deficiency: Psychological Theory and Research,* pp. 253-296. New York: McGraw-Hill, 1963.

Grossman, H. J., ed. *Manual on Terminology and Classification in Mental Retardation,* 1973 Revision. Washington: American Association on Mental Deficiency, 1973.

Gruenberg, E. M. *"Epidemiology,"* in H. A. Stevens, and R. Heber, eds., *Mental Retardation: Review of Research,* pp. 259-306. Chicago: University of Chicago Press, 1964.

Haywood, H. C., ed. *Socio-Cultural Aspects of Mental Retardation.* New York: Meredith, 1970.

Haywood, H. C. and J. T. Tapp. "Experience and the Development of Adaptive Behavior," in N. R.

Ellis, ed., *International Review of Research in Mental Deficiency,* Vol. 1, pp. 109-151. New York: Academic, 1966.

Heber, R. F. "Culture and Familial Retardation," in M. Cohen, ed., *International Research Seminar on Vocational Rehabilitation of the Mentally Retarded,* Special Publications Series, no. 1. pp. 313-324. Washington: American Association on Mental Deficiency, 1972.

Heber, R. F. and R. B. Dever. "Research on Education and Habilitation of the Mentally Retarded," in H. C. Haywood, ed., *Socio-Cultural Aspects of Mental Retardation,* pp. 395-427. New York: Meredith, 1970.

Holmes, L. B., H. W. Moser, S. Halldorsson et al. *Mental Retardation: An Atlas of Diseases with Associated Physical Abnormalities.* New York: Macmillan, 1972.

Jensen, A. R. "How Much Can We Boost I.Q. and Scholastic Achievement?" *Harvard Educ. Rev.,* 39 (1969), 1-123.

----. "A Theory of Primary and Secondary Familial Mental Retardation," in N. R. Ellis, ed., *International Review of Research in Mental Retardation,* Vol. 4, pp. 33-105. New York: Academic, 1970.

Kennedy, W. A., V. Van De Riet, and J. C. White, Jr. A *Normative Sample of Intelligence and Achievement of Negro Elementary School Children in Southeastern United States,* Monographs of the Society for Research in Child Development, 28 (1963), no. 6.

Leland, H. "Mental Retardation and Adaptive Behavior," *J. Spec. Educ.,* 6 (1972), 71-80.

Lewontin, R. C. "Race and Intelligence," *Bull. Atomic Scientists,* 26 (1970), 2-8.

McKusick, V. A. *Mendelian Inheritance in Man: Catalogs of Autosomal Dominant, Autosomal Recessive, and X-Linked Phenotypes.* 3rd ed. Baltimore: The Johns Hopkins University Press, 1971.

Menolascino, F. J., ed. *Psychiatric Approaches to Mental Retardation.* New York: Basic Books, 1970.

Mercer, J. R. *Labeling the Mentally Retarded: Clinical and Social Systems Perspective on Mental*

Retardation. Berkeley: University of California Press, 1973.

Nihira, K., R. Foster, M. Shellhaas et al. *Adaptive Behavior Scales.* Washington, C.: American Association on Mental Deficiency, 1969.

Reed, E. W. and S. C. Reed. *Mental Retardation: A Family Study.* Philadelphia: Saunders, 1964.

Ross, R. T. *Fairview Self-Help Scale (1970); Fairview Social Skills Scale (1972).* Costa Mesa, California: Research Department, Fairview State Hospital.

Rutter, M. L. "Psychiatry," in J. Wortis, ed., *Mental Retardation: An Annual Review,* Vol. 3, pp. 186-221. New York: Grune & Stratton, 1971.

Rutter, M. L., ed. *Infantile Autism: Concepts, Characteristics, and Treatment.* Baltimore: Williams & Wilkins, 1971.

Scarr-salapatek, S. "Race, Social Class, and Q.," *Science,* 174 (1971), 1285-1295.

Scriver, C. R. and L. E. Rosenberg. *Amino-Acid Metabolism and Its Disorders.* Philadelphia: Saunders, 1973.

Sersen, E. A. "Conditioning and Learning," in J. Wortis, ed., *Mental Retardation: An Annual Review,* Vol. 1, pp. 28-41. New York: Grune & Stratton, 1970.

Skinner, B. F. *Technology of Teaching.* New York: Appleton-Century-Crofts, 1968.

Smith, D. W. *Recognizable Patterns of Human Malformation.* Philadelphia: Saunders, 1970.

Spitz, H. H. "Consolidating Facts into Schematized Learning and Memory System of Educable Retardates," in N. R. Ellis, ed., *International Review of Research in Mental Retardation,* Vol. 6, pp. 149-168. New York: Academic, 1973.

Stein, Z. and M. Susser. "Mutability of Intelligence and Epidemiology of Mild Mental Retardation," *Rev. Educ. Res.,* 40 (1970), 29-67.

Stevenson, G. S. in *New Directions for Mentally Retarded Children.* New York: Josiah Macy, Jr.,

Foundation, 1956.

Stevenson, H. W. *Children's Learning.* New York: Meredith, 1972.

Tharp, R. G. and R. J. Wetzel. *Behavior Modification in the Natural Environment.* New York: Academic, 1969.

Watson, L. S., Jr. "Application of Operant Conditioning Techniques to Institutionalized Severely and Profoundly Retarded Children," *Mental Retardation Abstracts,* 4 (1967), 1-18.

----. *How to Use Behavior Modification with Mentally Retarded and Autistic Children: Programs for Administrators, Teachers, Parents, and Nurses.* Columbus, Ohio: Behavior Modification Technology, 1972.

Werner, E., J. M. Bierman, F. E. French et al. "Reproductive and Environmental Casualties: A Report on the 10-year Follow-up of the Children of Kauai Pregnancy Study," *Pediatrics,* 42 (1968), 112-127.

World Health Organization. *International Classification of Diseases,* 8th Revision. Geneva: WHO, 1968.