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Many different and unrelated causes give rise to mental retardation.[1] Yet the single encompassing diagnosis of mental retardation has meaning in that it predicts, for the majority of affected children, limited development and a lifelong career of dependence. Epidemiology, which is the study of the determinants and distribution of health disorders in populations ("epi" meaning "upon," "demos" meaning "the people"), must recognize both the heterogeneous

causes and the common elements of mental retardation. In their concern with etiology and prevention, epidemiologists must recognize the heterogeneity of causes. In their concern with the planning and evaluation of care, they must recognize a condition of dependence that generates common needs. In the present chapter, weight will be given to both these concerns. Knowledge of causes and prevention depends mainly on studies of incidence; knowledge of planning and evaluation depends mainly on prevalence.

Incidence describes the frequency with which disorders arise in a population during a defined period of time. The search for causes of the trends and distributions of health disorders is best pursued by studies of incidence, because incidence relates disorders to circumstances that exist at or before the time of onset of a disorder, and time order is an essential

criterion in establishing causal relations.

Since prevalence describes the amount of disorder existing in a population at a particular time, regardless of time of onset, it affords a useful measure of the existing load of disorder to be provided for. But prevalence is less useful in the search for causes of the existing disturbance or disorder. It gives a cross-sectional view of a population's experience at one time and cannot establish with precision the circumstances in which disorders of long and variable duration arise.

Disorders of long duration have a good chance of appearing in a prevalence census. In severe mental retardation there is no recovery, and duration is synonymous with survival. Thus, the long-lived will swell and bias the numbers of the mentally retarded population who contribute to prevalence, while some who contribute to incidence will not live long enough to enter a count of prevalence. Hence, prevalence and incidence are not interchangeable terms for measuring frequencies. The divergence between incidence and prevalence is exaggerated where duration varies widely and where it changes through time. Both these circumstances hold for the duration of severe mental retardation. The relation between prevalence and incidence can be simply stated: The prevalence of a condition is a function of its incidence and its duration. When either incidence or duration is known, inferences about the other term can be drawn from prevalence studies.

A special problem in the epidemiology of mental retardation is the definition of the case. Cases, to be counted, must be distinguished from non-cases, but the lines of demarcation are blurred by confused definition. Recognized mental retardation is a social attribute. Recognition is a consequence of failures to perform the social roles demanded of individuals at each stage of life. The order of society determines how taxing these roles shall be. What is expected in particular social roles, therefore, varies with time and among societies, and among the classes of a single society.

The manifestation of mental retardation as a social attribute contains at least three components: organic, functional, and social. A primary organic component refers to a structural or physiological disorder; this we shall term "impairment." Impairment of the brain or its metabolism is diagnosed by the methods of clinical pathology and clinical medicine. A psychological or functional component, which we shall term "disability," arises from the individual's psychological reaction to the limitation imposed on function either by organic impairment or by psychic and social forces. In mental retardation, functional disability is expressed in intellectual deficit and is diagnosed by the methods of clinical psychology and psychological medicine. The social component of mental retardation is defined by the special social roles assigned to the retarded individual. This social limitation we shall term mental "handicap"; it describes a social role, the manner and degree in which primary impairment and functional disability alter expected performance.

Handicap is diagnosed by the methods of sociology and social medicine.

These organic, psychological, and social criteria yield different frequencies of mental retardation and make quite different contributions to our understanding of the condition. The components of mental retardation measured by each criterion do not have a one-to-one relationship with each other and are made apparent by different circumstances. Impairments that can be recognized at birth, and for which a one-to-one relationship with functional disability and mental handicap can be predicted, as in Down's syndrome, are not common. Cerebral palsy is an impairment recognized by the signs of brain damage. Only about one-third of all cases of cerebral palsy suffer the functional disability of subnormal intellect or are assigned the special social role of the handicapped person.

Conversely, recognized functional disability cannot always be related to definitive organic lesions. In a large proportion of cases of mental retardation, even with severe intellectual deficits, a specific clinical diagnosis cannot be made. In these cases, the presence of organic impairment is merely assumed.[2] Severe mental retardation of unspecified diagnosis thus describes a residual class, a dump heap of cases that is heterogeneous in terms of origins and types of organic impairment. Yet it is a homogeneous class in that all members share a degree of functional disability and social handicap.

In mild mental retardation, on the other hand, the intellectual deficit and functional disability of the cultural familial syndrome is preceded by no detectable organic impairment at all, and is not always accompanied by the social role of mental handicap. If it is accompanied by handicap, the role may often be temporary, between the phases of pubescence and young adulthood. The social role of mental handicap is occasionally assigned to individuals who have neither impairment of the brain nor intellectual disability. Their social roles are inadvertently acquired by their admission to "treatment" because of a combination of behavior disorders and lack of social support. Thus, a proportion of the inmates of many institutions for mental retardation have neither detectable clinical lesions nor IQ scores below the normal range.

In studies of incidence, the order of usefulness of each criterion ascends from social, through psychological, to organic. The practical reasons for this order emerge from the information each criterion yields when analyzed by age. To take first the social dimension of mental handicap in early life, incidence is a difficult measure to apply, or even to conceptualize. Failures in role performance emerge gradually without a sharp point of onset. In the dependent state of infancy, role failures may go unrecognized, or may even be denied by some parents, and they become apparent only at school ages, when social roles are better defined. The incidence of functional disability is also difficult to measure at young ages. Psychometric measures in infants do not predict adult intellectual function as well as measures in older children, and

early in the life cycle they may fail to identify the functional disability of new cases.

By contrast, some impairing conditions, for instance, Down's syndrome, are from the moment of birth the clear-cut entities that epidemiologists most desire for incidence studies. Yet even these conditions are not without measurement problems. The life of a unique individual begins with the formation of a zygote. Some aberrant chromosomal arrangements preclude zygote formation altogether; some permit the zygote to divide but are incompatible with fetal survival; other's permit a bare few days of extrauterine life; and the trisomy 21 of Down's syndrome is compatible with survival in spite of a high risk of death from fertilization onwards. Indeed, most of the impairments of severe mental retardation have their onset in intrauterine life. At best, therefore, incidence counts are based on the emergence of impairment at birth, and unknown numbers are aborted early in fetal life. It is an uncertain assumption that the impaired survivors fairly represent impairments among all conceptions. If the assumption is incorrect, the associations with impairment that are taken to point to causes may in fact be the misleading results of selective survival.

In studies of prevalence, the order of usefulness of the social, psychological, and organic criteria is reversed. We have noted that prevalence best establishes current needs for treatment and care. In mental retardation,

these needs rest principally on the social component of the condition, which embraces all the other components. The prevalence of the social role of mental retardation is more easily established than is the prevalence of disability and impairment. The individuals among children and adults who are socially defined as mentally retarded, by their backwardness at school or by their failures in occupational roles, can often be identified from records and interviews. To determine levels of functional disability requires psychometric testing. For school children, test scores may be no more difficult to come by than their educational performance, but for adults, tests will usually have to be specially undertaken among a reluctant population. To determine organic impairment requires still more elaborate clinical examinations and more laborious surveys.

The distinctions among the organic, functional, and social dimensions of mental retardation have theoretical as well as descriptive significance. For instance, the observation that all organic impairments need not be expressed in functional disability leads to the hypothesis that learning can fully compensate for impairment. Thus, any demonstration that retarded brain growth in fetal or infant life need not be accompanied by measurable intellectual disability in later life has significance for hypotheses about the basis of functional intelligence. Again, the demonstration that functional deficits need not be directly translated into social handicap confirms that social roles can accommodate a wide range of performance around the

average. The demonstration that functional deficits at pubescence need not persist leads to the hypothesis that assigned roles and achieved social status may themselves improve and maintain levels of role performance. These hypotheses have significance for preventive programs and methods of care.

Another important distinction to make in the epidemiology of mental retardation is between severe and mild degrees of mental retardation. Severe retardation comprises at most one-quarter of the total. Usually diagnosed at an early age, its many known and unknown causes, rooted in persisting organic impairment of the developing brain, are distributed fairly evenly across the social classes. Mild retardation comprises at least three-quarters of cases, is diagnosed later during childhood or even during pubescence and adolescence, and no more than one-quarter of those affected are rooted in organic impairment. The remainder, with functional disability and social handicap but without definite organic impairment, are concentrated in the lower social classes and associated with poverty and its concomitants. Thus, the origin and prevention of much severe retardation must be sought in impairing preconceptual and perinatal factors that affect all classes; the origin and prevention of much mild retardation must be sought in postnatal factors that influence the acquisition of functional disabilities and social roles.

In the section on incidence that follows, some proven and some possible causes of mental retardation will be discussed. Some are rare, but we have

concentrated on causes that could be prevented by a comprehensive program for mental retardation. The review is selective, and many causes have not been dealt with, for instance, those of genetic origin. The causes we shall consider are (1) chemical and physical agents in the environment; (2) infections; and (3) sociomedical factors including poverty, prenatal and perinatal factors, nutrition, and demographic factors. In the section on prevalence, we discuss the prevalence of severe and mild retardation separately, and then consider the implications of the trends for treatment and care.

This discussion has so far elaborated the simple and emphasized the complex side of epidemiological measures. These complex issues must be respected, but they are intended to clarify, not to invalidate, the accumulated knowledge on which advance in the field must always be based. Our review will proceed with that object in mind.

Causes and Incidence

Technological development has added to the possible causes and perhaps to the incidence of mental retardation. The side effects of medical treatments, industrial processes, and warfare expose people to pervasive chemical and physical agents. We therefore begin with a brief outline of some chemical and physical antecedents of mental retardation.

Chemical Factors

Such drugs as lysergic acid diethylamide (LSD) can affect the chromosomes. Because chromosomal abnormalities underlie some types of mental retardation, notably Down's syndrome, drugs attract suspicion as possible causes of mental retardation. Such drugs as thalidomide, and perhaps LSD, affect later fetal development, and they also attract suspicion as causes of mental retardation. Proof is lacking, however, that drugs, taken during gestation for therapy or for psychedelic effects, cause mental retardation.

The heavy metals, mercury and lead, are proven causes of mental retardation, and people have been exposed to them by ingestion of drugs, and by pollution of food, water, air, and housing. In addition to the "hatters' shakes" and other forms of mercury poisoning, there is little doubt that mercurials in teething powders still in use after World War II were responsible for the infantile neuropathy known as "pink disease" (acrodynia) . Mercury levels high enough to cause alarm have recently been found in fish in the United States and Europe. The well-documented Minamata Bay outbreak in Japan in the 1950s justifies the alarm. Over a period of several years a number of women who during pregnancy habitually ate shellfish taken from the bay bore children afflicted by cerebral palsy and mental retardation. These shellfish concentrated mercury discharged from an

industrial plant. Control of the mercury contamination promptly reduced the incidence of the condition. In the United States, during 1969, at least one family suffered severe mental effects from mercurial poisoning, and the mother of the family, pregnant at the time, gave birth to a child with neurologic defects and perhaps mental retardation. In this incident, the family had eaten the meat of pigs fed on mercury-treated grain, a treatment intended to protect the planted grain from pests.

Lead poisoning has long been known to produce lead encephalopathy. Often fatal in young children treated tardily, a proportion of those who survive the encephalopathy are mentally retarded. A history of pica is common, and usually the source of lead can be identified in peeling leaded paint in the home. Children from two to four years of age are most often affected, as might be expected from the association with pica.

Acute lead encephalopathy is uncommon. A question of great concern, however, is whether more chronic and milder forms of lead intoxication give rise to mild degrees of mental retardation, neurological disorders, and behavior disturbances. In Chicago, Cleveland, New York and Baltimore, lead encephalopathy occurs mainly in particular areas, the so-called lead belts. Numbers of children from the lead belts have high levels of lead in their blood, sometimes associated with lead deposits and radiological signs in bones and basophilic stippling of red cells. The peak age for these syndromes

is the same as for lead encephalopathy, namely two to four years, and many have a history of pica. Some cities, in an effort to detect dangerous sources of lead and to treat those with high blood lead levels, have initiated mass blood testing programs for children in affected areas.

The effect on mental function of lead intoxication without encephalopathy, as indicated by high blood levels, is not yet established, but there is good reason to assume that high levels are noxious. In areas where children are at risk, the only safe course for cities to follow is to enforce housing standards, educate parents to the danger, and systematically screen the infants at risk. Although the assumptions of screening programs are not proven and need testing, programs intelligently applied can test their own assumptions in the course of application.

Physical Factors

Among known physical agents that can cause mental retardation, by far the most serious is ionizing radiation. The sources studied have been, first, therapeutic and diagnostic irradiation and, second, the atomic bombs exploded over the populous cities of Hiroshima and Nagasaki, at the order of President Truman, as Japanese resistance was collapsing at the end of World War II.

Pelvic irradiation of mothers early in pregnancy can lead to cerebral

damage in the child. Clinical observations date from 1929. Decisive epidemiological observations date from the Hiroshima bomb, August 6, 1945. A twenty-year follow-up has established that the effects of the bomb included shortened stature, microcephaly, mental retardation, and leukemia. Microcephaly has a high correlation with brain size, and it is tempting to use head circumference as an indicator to retarded brain growth and organic impairment. The main stimulus to the growth of the skull is thought to be the growth of the brain. As a manifestation of nuclear irradiation, microcephaly proved to be to some degree independent of the functional disability of mental retardation. This independence is reflected in the fact that three-quarters of the micro- cephalic subjects did not exhibit notable mental retardation. On the other hand, sixteen of eighteen cases of marked mental retardation, about 90 percent, had microcephaly. It seems not unlikely that microcephaly caused by nuclear irradiation reflected impaired brain growth and cell depletion, and that this organic impairment became apparent in dysfunction only in the one-quarter of subjects most severely affected.

The susceptibility of the fetus to irradiation varied with the dose and with fetal age, and there was some interaction between these two factors. Both microcephaly and mental retardation increased in frequency as the dose of radiation increased. Where fetal exposure occurred earlier than about twenty weeks after the last menstrual period, the incidence of both conditions was distinctly higher than with later exposure. Mental retardation was

concentrated particularly in the period about ten weeks after conception. A review of twenty-six case reports of fetal injury after medical irradiation has also shown the fetus to be most vulnerable at seven to fifteen weeks. With heavy doses of radiation, however, the difference in susceptibility between early and late gestation in both microcephaly and mental retardation was diminished although still present.

Effects of irradiation have been sought in children whose mothers or fathers were irradiated before the children were conceived. In the cohort of Japanese children who were born between 1948 and 1959, and whose parents had been exposed to the atomic explosions, effects of preconception exposure were not detected in the incidence either of congenital abnormalities or of mental retardation. Indeed, the cases of Down's syndrome found among 5,582 exposed mothers proved to be only half the expected number.

Because of the small numbers of cases that are found with cohort studies of rare conditions, the question of the relationship between irradiation of the gonads before conception and subsequent mental retardation is still a matter of debate. In a case-control study, Sigler, Lilienfeld, Cohen, and Westlake compared the radiation experience of mothers and fathers of 216 cases of Down's syndrome and 216 controls matched for maternal age. Mothers exposed to fluoroscopy or to therapeutic

radiation had a relative risk of seven to one. The information about irradiation was derived from family reports. The validation of these reports from clinical records was not extensive, and on this ground the result awaits confirmation.

The findings of Sigler et al. get support from a prospective Canadian enquiry by Uchida, Holunga, and Lawley. On the other hand, in other studies besides that of the atom bomb cohort, a relationship between preconceptual irradiation and Down's syndrome has not been found. If the findings of Sigler et al. are confirmed, the attributable risk (that is, the rate in the population attributed to maternal irradiation over and above the rate attributed to other causes) would not be large; however, the information could be used immediately to reduce incidence. Clarification should therefore be sought without delay.

Infections

The infections that influence the incidence of mental retardation are a mixed bag. They include bacterial meningitis, congenital syphilis, toxoplasmosis, and viral encephalitis. On the one hand, changes in infectious diseases have led to a fall in the incidence of organic impairment and of mental retardation generally, or can be assumed to have done so. On the other hand, survival from attacks that would once have been fatal has led to a

rise in the prevalence of impairments and to a consequent rise in the incidence of subsequent functional disability and mental handicap.

Known infections have probably never been responsible for more than a small minority of cases of mental retardation. Even rubella, so grave a hazard to the embryo, is an uncommon cause of mental retardation unless the syndrome is complicated by lesions of the sense organs. In an English epidemic, congenital rubella without complications of the sense organs did not lead to a reduction in mean IQ in affected children followed over ten years. Influenza infection during pregnancy has appeared on occasion to increase the incidence of esophageal atresia, of cleft palate, and anencephaly, but no association with intellectual deficit or mental handicap has been discovered.

In general, viral encephalitides contribute a small and sporadic number of cases to the pool of mentally retarded persons. In epidemics, among infants the rate of fatalities and of brain damage with subsequent mental retardation is high, but the condition is an uncommon one overall. Measles encephalitis, for instance, is a rare cause of mental retardation. In the United States, between 1960 and 1966, about 250 cases of measles encephalitis were reported each year. Eighty percent survived, and an estimated one-third of these were under two years of age. An estimated one-third of these young survivors (about twenty-two per year) might have been left with severe

mental retardation. These numbers depend on reporting and may underestimate by as much as a factor of ten.

The possibility exists that even uncomplicated measles may have mental sequelae. The postulated effects of measles, like the fatalities it causes in malnourished populations, may depend on the interaction of infectious agent and host characteristics. In one series of studies in the United States, rea(Jing readiness at school entry was used as an index of intellectual disability. In poor communities, children with a history of measles were retarded in reading readiness compared with controls. In better-off communities, children with a history of measles were not retarded. Better-off children are perhaps more resistant to damage from infection than those in poverty. In addition, it is likely that the better-off children are better able to compensate, by learning and favorable experiences, for whatever temporary or permanent organic impairment measles may cause.

With successful and widespread immunization, any organic impairments and functional retardation caused by measles will disappear. In the United States, reported measles morbidity reached a nadir in 1968 following on a new national program; the rise in the subsequent two years probably relates to reduced effort rather than to reduced efficiency of vaccination. The immunization that controls measles is itself a rare cause of encephalitis and mental retardation. Encephalitis with resulting mental

retardation can also follow immunization against pertussis. There is no doubt, however, that with regard to mental retardation, more is to be gained than lost by immunization against these diseases.

The history of tuberculous meningitis illustrates that the effects of treatment on incidence have not always been in one direction. During the first decade after World War II, new treatments changed tuberculous meningitis from a uniformly fatal condition to one that permitted survival. Because a proportion of the early survivors had gross impairments of the brain, they added to the incidence of disability and handicap from mental retardation. In the 1960s the incidence of primary tuberculosis and its complications continued to decline, and treatment also became more effective, so that fewer new cases of mental retardation due to tuberculous meningitis appeared.

A somewhat comparable effect may be observed with other meningitides. There has been a sharp decline in the case-fatality rate of bacterial meningitis since modern treatments were introduced. Although the proportion of survivors with mental sequelae may also have declined, the larger overall number of survivors could have generated a number of cases of mental retardation as large in the years subsequent to chemotherapy and antibiotics as in the years before.

Infections that occur at and around birth, some caused by agents

discovered only in recent years, have attracted suspicion as neurological pathogens. Cytomegalovirus has been implicated as a cause of mental retardation. It is also associated with infantile spasms and fits, and possibly with microcephaly, both common in mentally retarded populations. The pathogenesis is not clear; the virus seems to be harmless in the majority of the newborn from whom it is isolated. Mycoplasma, virus-like agents, have also been suspect as a cause of mental retardation. Firm evidence is lacking. Congenital toxoplasmosis is a rare but well-documented protozoal cause of mental retardation. Antibodies to toxoplasma developed at the third and fourth months of gestation in 2 per 1,000 of a large series of pregnancies studied longitudinally in the cities of the eastern United States. Congenital toxoplasmosis was observed in five offspring of mothers who developed antibodies, an incidence of 0.25 per 1,000. Since antibodies in mothers are detectable, prevention of this cause of mental retardation by aborting the fetus is technically within reach.

The incidence of mental retardation due to known infections has surely declined. Social change has improved host resistance and immunity, effected environmental control, and provided the techniques to prevent or to treat such infectious diseases as syphilis, bacterial meningitis, pertussis, measles, mumps, and rubella. Further gains from the control of infectious diseases seem to be within reach. A reduction in incidence of small but uncertain magnitude should follow better understanding of the encephalitides,

toxoplasmosis, and cytomegalovirus.

Social Class and Poverty

An association between mild mental retardation and social class has been recognized at least since the beginning of the century. Observers have been in doubt about which was the antecedent variable, that is, which was cause and which consequence. As the selective racial theories of social Darwinism lost force, so poverty of the environment has been given greater prominence as a causal antecedent rather than a consequence of mental retardation. The cultural-familial syndrome associated with poverty is a form of mild intellectual disability without detectable clinical lesions and is by far the most common type of mild mental retardation.

In two industrial cities of Lancashire, England the incidence of the condition (defined by an IQ between 50 and 80 and the absence of detectable clinical lesions) was not more than 0.26 per 1,000 among children in publicly financed schools of high social standing; the rate was 3.8 per 1,000 among the schools of lowest social standing, a relative risk at least fifteen times higher. In Aberdeen, Scotland, a similar distribution was also found to hold good. A family study in Lancashire demonstrated that the syndrome was familial. It was virtually specific to demotic families. These were families of the lowest social strata, defined by occupation and education, that showed no signs of

upward social mobility in the occupational and educational experience of three generations. Among aspirant working-class and higher-class families, which did show signs of upward social mobility, the typical syndrome was not found.

The specificity of the syndrome for families typed by cultural characteristics legitimizes the "cultural-familial" label. Other features suggest that it is also cultural in origin. A degree of intellectual and social recovery tended to occur in young adulthood. Unlike those with neurological lesions, those with the syndrome were found to have made IQ gains in young adulthood. They made social gains as well. Provided they came from functioning nuclear families, most were adapted to normal social roles. Only those from nonfunctioning dysmorphic families broken in early childhood were institutionalized or otherwise handicapped.

Much other evidence indicates that poor cultural environment can depress intelligence test scores. This environment effect best explains the fact that the tail end of the normal IQ distribution is substantially lower among the lowest social classes, which include demotic families, than among higher classes.

Seen in this context, IQ scores in the range 50-80 are the pathological outcome of adverse cultural environment. The mental retardation attributed

to the demotic culture presumably occurs in individuals endowed with relatively low innate intelligence or high sensitivity to the environment. Adverse environment depresses their performance below the threshold for normality in the society at large. The genetic component in mental performance probably explains a large proportion of individual variation but a much smaller proportion of the variation between groups.

Although the cultural-familial syndrome is a characteristic of certain types of family, this does not require that the causes of the condition reside within the environment of the families alone. Members of demotic families share, outside the family, the same harsh social and physical environment and impoverished educational experience. The external as well as the internal familial environment could contribute to the association of mild mental retardation and social class.

Compared with those better off, the children of the poor have experienced higher prematurity rates at birth, more infectious diseases with less treatment and more severe effects, more malnutrition; they have enjoyed less enduring family ties and been burdened with a greater frequency of all kinds of functional disabilities and physical handicaps. They have been poorly housed and poorly schooled, have suffered discrimination in social and public life, and have had a high liability to conviction for crime. These elements, too numerous to receive adequate treatment in this chapter, could each

contribute to intellectual disability and to the assignment to members of the affected classes of the role of mental handicap. In the sections that follow, two attributes of the poor, the high risk of having prenatal and perinatal complications and inadequate nutrition, have been selected for discussion.

Prenatal and Perinatal Factors

Relationships between circumstances of birth and later intellectual function have been much studied, but the causal connections are still not clear. We exclude from discussion those chromosomal and genetic abnormalities, fetal malformations, and intrauterine infections that are in themselves both antecedents of mental retardation and of perinatal difficulties. The list that remains is long, since it includes factors related to the mother as well as the child, for instance, maternal factors like toxemia, short and long labor, mal-presentations, unattended deliveries, antepartum hemorrhage, low birth weight, multiple births, neonatal anoxia, and bilirubinemia. The discussion will be limited to three factors for which there are substantial data: (1) low birth weight, (2) toxemia, and (3) multiple births. Even with regard to these three common and easily recognized conditions, the causal connections are difficult to disentangle.

1. Studies of perinatal mortality show that birth weight is either in itself a crucial factor in survival or an indicator of other crucial factors that lead

both to death and to retarded prenatal development. Low birth weight can account for the great part of perinatal mortality. Low birth weight also has associations with mental retardation, and similar associations hold for short gestation and for retarded intrauterine growth measured by combining the two indices.

Low birth weight cannot be assigned a causal or a direct mediating role in mental retardation without equivocation. Low birth weight itself has heterogeneous causes, known and unknown. Its association with mental retardation is reduced when those cases are excluded in which the causes of mental retardation antedate birth weight and retard development, as with Down's anomaly and phenylketonuria. With very low birth weights, preexisting congenital defects seem to account for much of the high risk of mental retardation. Where congenital defect does not accompany low birth weight, brain damage appears to be a necessary intervening factor between low birth weight and mental retardation. In one series of infants of very low birth weight, the risk of cerebral palsy was high among those with short gestation periods. The risk of mental retardation was raised only among those with cerebral palsy, but not in the remainder.

With moderate degrees of low birth weight, a consistent association with mental performance also is not found. The interconnectedness of the postulated causal factors obfuscates the contribution of birth weight to

measured intelligence. Thus, the social-class gradients of birth weight and IQ are sharp and parallel, to the disadvantage of the lower classes. When the closest possible control of social class has been applied, by comparing sib pairs, only a small difference in IQ has been found between sibs of different birth weight (excepting the few pairs with gross weight differences between members). This finding suggests that among the complex of variables associated with social class, a moderate difference in birth weight is not the likely intervening link between social environment and measured intelligence in school children. Similarly, within families with a mildly retarded child, the affected child is no more likely than his sibs to have been of low birth weight or small for the period of gestation. These observations strengthen the view that certain forms of mild mental retardation, and particularly the cultural-familial syndrome, depend primarily on membership in families of distinctive character and milieu.

The distinction between organic impairment and dysfunction may be at the root of the apparent incoherence of the relationship of mental performance with low birth weight. Most factors causing functional disability act postnatally. If prenatal factors cause mild impairment, a plausible hypothesis is that this impairment can be compensated for by acquired functional abilities. Compensation may occur only in favorable circumstances. Some studies, but not all, point to interaction between the effects of low birth weight and social class on mental retardation. In these, moderately low birth

weight was associated with mental retardation or lowered IQ only among the lower classes and not among the higher. Moderately low birth weight seems to be neither a sufficient nor a necessary cause of mild mental retardation, but it may be a contributory cause in unfavorable circumstances.

2. Preeclamptic toxemia is often associated with other obstetric complications. It is difficult to isolate its role as a cause of mental retardation because other complications confound the results. Another difficulty is that the epidemiology of toxemia is far from clear. Some studies indicate that toxemia is less frequent in times of starvation and among the lower social classes. Studies of the relationship of toxemia with retardation must control for such factors, which may themselves produce the observed variations. Thus in Aberdeen, Scotland, children with preeclamptic toxemia scored above the normal in IQ, but when social class was controlled, the infants of toxemic mothers were at a slightly higher risk of mental retardation. In that result, however, the toxemia was almost always associated with other obstetric complications. One Canadian study, which controlled for both social and obstetric factors, also concluded that a small amount of intellectual handicap could be attributed to toxemia.

3. Multiple births are associated with a raised risk of mental retardation. In some cases, as with low birth weight, retardation can be attributed to evident fetal abnormality. Twins and triplets experience many perinatal

complications that relate to mental retardation more often than singletons: curtailed gestation, low birth weight, intrauterine growth retardation, long and short labor, toxemia, malposition, antepartum hemorrhage are all more common among them. Their intellectual retardation, however, is greater than can be accounted for by these factors alone.

Explanations of the mental retardation of twins must also weigh the contributions of prenatal organic impairment and postnatal experience. Among single survivors of twin births, mean IQ is approximately normal at eleven years of age, whereas among pairs of survivors, mean IQ is several points lower. Single survivors differ from pairs of survivors in their postnatal experience, and their better intellectual performance can be attributed to postnatal causes rather than to chromosomal, intrauterine, or intranatal causes.[3]

In sum, low birth weight, toxemia, and multiple births are all closely associated with perinatal mortality and organic brain impairment. Where impairment is gross, mental retardation is often severe. Where impairment is not gross, its contribution to intellectual function in later life is overshadowed by postnatal experience. This is not to deny that these three natal circumstances have some predictive power in relation to mental retardation. This predictive power rests in the main on their association with other factors, such as congenital anomalies, neurological lesions, or socioeconomic

status. In assessing causes in the individual case, these related factors always need evaluation.

Nutrition

In recent years, malnutrition has been strongly canvassed as a cause of mental retardation. In animal experiments, diets sufficient only to keep the animals alive damage the central nervous system. Effects include neuromotor and behavioral abnormalities as well as reduced brain weight and changes in the histology and biochemistry of spinal cord and brain.

The period of most active growth of a target organ (whether measured by cell numbers or by cell size and differentiation) is supposedly the time of its greatest vulnerability to insult. The theory of the critical period postulates that to interdict growth processes of an organ at the time of maximum growth and vulnerability will not only retard the processes but will prevent their taking place at later life stages. In the human brain, two periods of maximum growth are thought to be early in gestation and in the months just before and after birth. The proliferation of neuron and glial cells takes place in the early phases of gestation, and growth in their size in the later phases.

In humans, nutritional deprivation can seldom be pinpointed in time, as a test of this hypothesis requires. No evidence bears on malnutrition during the postulated period of maximum vulnerability early in gestation, and that

which bears on late prenatal and early postnatal life is scant. Starvation of the mother during the late prenatal period leads to low birth weight in the infant, and other forms of malnutrition may possibly also cause low birth weight. According to one investigator, study of a few infants who died of acute malnutrition during the first year of life showed a marked deficiency of cell number in the brain. The deficiency of cell number was more marked in infants of low birth weight, which pointed to a process initiated prenatally. Infants who died of acute malnutrition in the second year of life did not have a deficiency of cell number in the brain, which suggested that after early infancy malnutrition did not affect proliferation.

In published reports of mental retardation supposedly induced by malnutrition, the earliest episodes of malnutrition have been in the postnatal months. Thus, among sixteen marasmic infants admitted to hospital in Santiago, Chile, during the first six months of life, usually when a diet of flour and water had been substituted for breast feeding, several were found to be severely retarded later in childhood. During the second and third years of life, an association of malnutrition and mental retardation has not been demonstrated as yet, and in adulthood severe malnutrition has not been associated with mental retardation, aside from pellagrous dementia.

If mental retardation is caused by malnutrition, this cause is likely to be associated with forms of mental retardation that are commoner among the

poor. In industrial societies food deficiencies, like mild mental retardation without organic impairment, are concentrated almost exclusively among the poor. The more severe degrees of mental retardation show no more than a slight predilection for the poor. In the United States, Sweden, and Great Britain, therefore, it would be safe to predict that any malnutrition-induced syndrome would be a form of mild mental retardation and would be reflected in indicators of function and role, rather than of impairments. In the developing countries of Asia, Africa, and South America, little is known of the distribution of the different forms and degrees of mental retardation. In these harsher environments, malnutrition-induced cases of severe mental retardation may conceivably occur.

Demographic Factors

Maternal age is a major factor in the incidence of Down's syndrome. The chances of bearing a child with Down's syndrome increase sharply with the age of the mother at the time she conceives. Parity and paternal age are closely linked with maternal age, but do not add to the risk of Down's syndrome.

The incidence rates at birth of Down's syndrome, for each maternal age, exhibit a high degree of stability at different times and places. The proportionate contribution of older women, however, varies with the

demographic trends affecting all births. The frequency of all births depends on the age- specific fertility rates among women of different ages and on the proportion of women of different ages comprising the child-bearing population. Both are highly variable. The proportion of all births with Down's syndrome, therefore, is also variable.

The natural variation in child bearing points to the possibility of reducing the frequency of retardation by voluntary restriction of fertility. Knowledge and techniques are available with which to reduce significantly the incidence at birth of Down's syndrome. Women in their later years of fertility are clearly a salient target group for a preventive program. So too are those few at risk of transmitting the condition genetically. Public attitudes and laws about contraception and abortion are changing, methods of prenatal diagnosis have been developed, and the problem is ripe for attack. The hazard to older women needs to be made widely known and contraceptive advice and methods made fully available to older couples. For older women who fall pregnant, whether by accident or design, prenatal diagnosis could contribute an important part to a preventive program. The existence of a fetus with Down's syndrome can be diagnosed by amniocentesis, with reasonable accuracy and apparent safety, fourteen to sixteen weeks after conception. Should a diagnosis of Down's syndrome be made by intrauterine aspiration, it should be mandatory to offer the mother the choice of a therapeutic abortion. Where the laws permit, there seems good reason to advise elective abortion.

The affected embryo can be identified in the amniotic fluid, just as in Down's syndrome, in a growing number of genetic diseases. Several are causes of mental retardation. Theoretically, these forms of mental retardation can also be reduced in incidence by identifying women at high risk and monitoring their pregnancies by amniocentesis. Some of the inborn errors of metabolism, enzyme defects inherited through autosomal recessive genes, serve as examples. Some defects in lipid metabolism affecting the mucopolysaccharides (Hurler syndrome, Hunter syndrome) and the sphingolipids (Tay-Sachs disease) and some defects in sugar metabolism are causes of mental retardation. In these conditions, the birth of an affected child is often the first indication, too late for prevention, that a couple is at high risk because each member is heterozygous for the gene. In some cases, the prediction of high risk can be made before conception. Information on kin and biochemical methods of detecting heterozygosity sophisticate prediction and permit prenatal confirmation and action. In other cases, notably the enzyme defect in protein metabolism expressed in phenylketonuria, high risk can be predicted before conception, but prenatal diagnosis has not been achieved. The risk is recognized from family and obstetric history and from phenylalanine levels in the blood compatible with heterozygosity for the gene. Action must therefore be taken before conception or after birth. Mothers with still higher levels of phenylalanine blood levels compatible with homozygosity have a high risk of giving birth to a child already affected by the

raised maternal level; in such cases the only successful prevention known is to prevent conception or induce abortion.

Table 31-1. Rates from Selected Prevalence Studies of Mental Retardation: By Period, Location

SOURCE OF REPORT	PERIOD	CASE SOURCE	LOCATION	AGE SPECIFIC RATES PER 1,000			AGE GROUP
				SEVERE <50 IQ	MILD >50 IQ	TOTAL	
Great Britain							
Royal Commission (Tredgold)	1904	Key informants, schools, agencies	Manchester, Glasgow, and Belfast			12.0 7.4 5.0	Elementary school children
E. O. Lewis	1925-1927	Key informants, and screening of schools; psychometric validation	3 urban areas 3 rural areas	3.76 6.14	17.14 33.56		7-14 7-14
Goodman and Tizard	1960	Official register	Middlesex, England, and Wales	3.61			10-14
Kushlick, Susser	1961	Official register	Salford	3.25			10-19
Kushlick, Susser	1961	Official register	Salford		6.60		15-19
Kushlick	1964	Special consensus of local agencies	Urban and rural Wessex	3.75			15-19
Innes and Kidd	1968	Official register	Northeast Scotland	3.70	9.50		15-19

Birch et al.	1970	Screening of schools, psychometric validation	Aberdeen	3.40	23.70	8-10
United States						
Lemkau et al.	1933-1936	Agency records, household sample census	Baltimore, eastern county	3.30	40.30	10-14
Oregon State Board of Health	1962	Agency records	Oregon	3.30	19.30 30.30	Under 20 12-14
Lemkau and Imre	1966	Complete household survey, school screening, psychometric validation	Maryland rural	13.89	71.00	10-14
Netherlands						
Hartogh	1954	Questionnaire to households	Gelderland, towns and villages	3.20	37.90	10-14
Sorel	1968-1969	Administrative and psychometric screening	Amsterdam	7.34	9.50	13
Australia						
Krupinski et al.	1963	Agency records	Victoria	1.91	4.56	12-14
Sweden						
Akesson	1964	Agency records, key informants	Western Sweden, rural	5.50		10-20

These various diagnostic advances point to the possibilities of screening programs. Large-scale screening has been practiced for some years in order to detect disease and prevent or treat it. For rare autosomal recessive genetic conditions, phenylketonuria programs serve as a prototype. In the course of the development of these large-scale screening programs, guiding principles for planning and evaluation have evolved. Screening tests must be sensitive enough to detect virtually all at risk and avoid false negatives. They must be specific enough to avoid false positives, each of which may entail extensive further diagnostic studies. Statistical tests are available to evaluate efficiency in terms of sensitivity and specificity. The influence of frequency of the condition on the efficiency of screening should also be taken into account. Given efficient detection, its timing must be early enough to permit intervention while prevention or treatment is possible. Finally, intervention must be shown to be not only possible but effective, often a difficult challenge. Most screening programs proceed on these assumptions; all too few actually test them.

Prevalence

Prevalence studies of mental retardation, like those chosen for illustration in Table 31-1, are difficult to interpret and compare. The difficulties reside in differences in the methods used to find cases and in the criteria used to define them. It can be seen from Table 31-1 that the disparity

between surveys is much reduced provided comparisons are limited to specific age groups with the gross impairment of severe mental retardation. When there is little or no impairment, as with mild mental retardation, and ascertainment depends on functional and social criteria that differ from one survey to another, the disparities between rates remain marked. Apart from these methodological considerations, variation in rates is to be expected. The mentally retarded population, sensitive like other populations to the social environment, is both dynamic and unstable. It is dynamic in that there is continual recruitment of new individuals and loss of old ones. It is unstable in that the balance between losses and recruitment is changing. The time trends for severe and mild mental retardation diverge sharply and will be considered separately.

The Prevalence of Severe Mental Retardation

Losses to the mentally retarded population are occurring at a slower rate than before because the death rate is declining and survival is improving. For evidence we turn to Down's syndrome, an excellent epidemiological index. The diagnosis is one of the least equivocal in medicine, and the population is one of the most complete and representative within the whole class of mental retardation. Life tables constructed for cases of Down's syndrome show an undoubted increase in longevity over the last generation. Prevalence at the age of ten years appears to have risen from 1 in 4,000 in

1929 to 1 in 2,000 in 1949 and to a possibly still higher rate in i960.

Improvements in medical and surgical treatments, in public health and the techniques of immunization against infectious diseases, and in the physical environment and accident control all reduce mortality in Down's syndrome. They prolong life without in any way modifying the accompanying mental retardation. The major cause of death in the past, respiratory infection, has been much reduced by antibiotic treatment. In most cases, death is now due to associated anomalies, such as those of the heart or gut. Leukemia kills a fraction (about 1 in 95). Half of the infants born with Down's syndrome die before their fifth birthday, but those who survive beyond it tend to continue in reasonably good health for several decades, so that in later life presenile symptoms and diabetes are coming to be recognized as common among them. In industrial societies, Down's syndrome now makes the largest single contribution to the prevalence of severe mental retardation both in the community and in residential institutions.

Downs syndrome serves as a paradigm for all forms of severe mental retardation. The total number of handicapped persons for whom families and communities must make provision has risen. Thus surgery prolongs the life of children with spina bifida and hydrocephalus, conditions accompanied often by severe physical handicap and sometimes by mental handicap. During the decade 1950 to i960, in the United States, the peak rate for deaths attributed

to all congenital anomalies shifted from the under-five age group to the five to fourteen age group.

Survival is reflected in the prevalence of all forms of severe mental retardation. In Salford, England, data compiled from community registers pointed to a life span for severely retarded people that was longer in than in 1948, and even longer in 1968. In the interval 1948 to 1963, there was an average increase in the rate of the registered population of 4.75 per 100,000 per year. Table 31-2 shows that this increase in the population was accompanied by aging. The most notable increment was to the older age groups. Within the short period of fifteen years, a longer life span had evidently produced a notable increase in the prevalence of severe mental retardation.

Table 31-2. Registered Severely Subnormal Population in Salford in 1948 and 1963 Compared: Age-Specific Rates per 100,000 by Ten-Year Age Groups[a]

	AGE GROUPS						
	0-9	10-19	20-29	30-39 40-49	50-59	60-69	N
January 1, 1948	70	290	270	221 96	49	39	147
January 1, 1963	152	209	182	198 180	113	61	238

a The increased rate in the 0-9 year age group is the result of an active case-finding program instituted about five years before 1963.

In general, the available evidence from developed countries points to a

changing trend in recruitment to the mentally retarded population. The trends suggest a fall in incidence as well as a rise in longevity. New cases of Down's syndrome are probably occurring less frequently than before because older mothers have curtailed their child-bearing period. New cases of other types of severe mental retardation have probably declined even more dramatically. In England, the prevalence of Down's syndrome at ten years of age observed in i960 by Goodman and Tizard was higher than that observed by Lewis thirty-five years earlier; the prevalence of other kinds of severe mental retardation at the same age in i960 was only two-thirds as high. In the face of prolonged survival for all kinds of severe subnormality, a decline in the prevalence of mental retardation other than Down's syndrome most likely came about through a considerable decline in incidence.[4]

To sum up, in severe mental retardation advances in public health, medicine, and surgery, and social change generally have reduced the incidence of impairment and intellectual disability at a slower rate than they have increased survival and prevalence.

Prevalence of Mild Mental Retardation

Longevity is not of major significance in the prevalence of mild mental retardation. The social handicap of mild mental retardation consistently has maximum prevalence in the second decade, building to a peak at adolescence,

with a sharp decline into early adulthood and through the third decade. Death rates at these ages have been

low during recent times; the room for variation is too little to affect prevalence to the same degree as in severe retardation.

The age distribution of mild mental retardation is the result of the limited duration of the state of mental handicap in the majority of cases of this condition. Diagnostic labels are assigned only after role failures are recognized, usually at school, and the child is referred for psychometric and medical review. In early adulthood, the labels tend to be removed once the affected individuals resume normal occupational or domestic roles.

The duration and age distribution of mild mental retardation are not solely a matter of the assignment of the role of mental handicap to individuals with intellectual deficits. It is also a matter of the duration of intellectual dysfunction. The dysfunction that ordinarily underlies mild mental handicap seems to be, as noted above, of two broad types: One type is associated with organic impairments, usually prenatal in origin, and accounts for about one-quarter of cases; the other type is not associated with organic impairment and accounts for the remainder.

The intellectual deficit associated with organic impairment seems to be stable and permanent. The undemanding roles of early childhood are within

the capacities of these affected individuals. At pubescence, as educational demands in particular become more exacting, deficiencies in capacities for role performance become relatively greater. Social recognition leads to the assignment of the diagnosis of mild mental retardation and the role of handicap.

The deficits associated with no detectable impairment, on the other hand, are not stable. A synthesis of the evidence suggests that the duration of these deficits runs concomitantly with the duration of handicap. Thus, the high frequency of mild mental handicap after pubescence can be linked with a decline in IQ. A number of studies suggest that the intellectual performance of groups at a marked social disadvantage grows progressively poorer up to adolescence, an outcome that can be attributed to a cumulative effect of exposure to an adverse environment. Similarly, the sharp fall in the frequency of mild mental handicap in early adulthood can be linked with IQ. As noted above, persons with the cultural-familial syndrome have been shown to make gains in IQ in adolescence and young adulthood.

More needs to be known about the distribution of IQ losses and gains and the size of the contribution of age changes to the frequency of mild retardation. We can conclude that for children at a marked social disadvantage pubescence is a highly vulnerable period. The handicap of such children appears greatest at just those ages when a competitive school system

and specific socialization to the adult world begin to make their greatest demands. Although in cultural retardation a degree of intellectual and social recovery occurs without special treatment, it is uncertain how much permanent damage remains.

A decline in the prevalence of mild mental retardation should be expected. The social conditions that underlie the substantial fraction of cases without impairment are undeniably less harsh for the populations of industrial societies taken as a whole. In this instance, countervailing rises in longevity would affect only the prevalence of the smaller fraction of cases with impairment. Longevity would not affect the substantial social component that gives rise to disability and handicap of limited duration. Available evidence indeed points to a decline in prevalence. This appears from cross-sectional surveys made in England over an interval of about four decades, and from the Scottish surveys of national intelligence in 1932 and 1947. In Sweden, too, after World War II, there was a decline in rejections for induction into the army because of poor mental performance.

Since social change included improvements in nutrition, employment, social mobility, and education as well as reduction in mortality and morbidity rates, the decline in the prevalence of mild mental retardation cannot be related to any specific element of the social change. Improvement in the social environment of groups at a marked social disadvantage, however, can be

expected to bring about a further decline in the prevalence of mild mental retardation. It seems likely that the greatest advantage will come from a serious attack on poverty and its cultural concomitants.

The Prevalence of Psychiatric Disability and Mental Retardation

The interrelationships between psychiatric disability, mental retardation, and organic impairment have been analyzed, particularly in a recent study in the Isle of Wight, in the south of England.

Among children with physical disorders, the proportion with psychiatric disability was about double the proportion in the general population. Among children with neuro-epileptic symptoms and signs, the proportion was about five times as high. Among children with intellectual disability (measured by IQ), the proportion with psychiatric disability was still higher. The more severe the intellectual disability, the higher the rate of psychiatric disability. No single diagnosis among the mentally retarded population accounted for the high rate. Children had a high rate of psychiatric disability whether or not their intellectual disability was accompanied by signs of brain damage.

A prevalence study gives inadequate grounds for determining the time order of associations between manifestations, and therefore of the causal sequence among them. The bare facts do not permit a sure distinction to be

made among conjunctions of physical, cerebral, intellectual, and psychiatric disorders that, on the one hand, have a common source in some antecedent underlying impairment and, on the other, arise successively in consequence of one another. In this study, a plausible interpretation seems to be that the high rate of psychiatric disability that occurred together with neurological disability (indicated by fits or spasticity) and severe intellectual disability had a common source in brain damage. In mild mental retardation in the absence of brain damage another explanation must be found for the raised rate of psychiatric disability. The excess of psychiatric disability that occurred both with mild mental handicap and physical handicap seems more likely to have been a consequence of the handicapped condition.

Implications for Care

The existence in the population of an increasing number of impaired individuals who will survive into later childhood and adulthood with severe mental retardation makes urgent the need to develop and apply new knowledge in its amelioration. The need focuses on three objectives: (1) to limit functional disability and social handicap in impaired persons; (2) to cultivate the maximum intellectual and social potential of those affected; and (3) to provide families and communities with appropriate supportive services to carry the burden of dependency.

The reduction of the prevalence of intellectual disability and mental handicap can be approached in two main ways. In some conditions, treatment of the underlying impairments can control their impact on intellectual function and role performance. In other conditions, the direct improvement of intellectual function and role performance can be attempted.

The effect on intellectual disability of controlling underlying impairment has been best elaborated, if still imprecisely, in phenylketonuria. Here, dietary treatment in infancy and childhood has reduced the degree of intellectual disability, and perhaps its prevalence, in the cohorts of phenylketonuric infants identified at birth.

That the prevalence of functional disability can be reduced by means that develop intellectual function is indicated by several studies among the mildly retarded. First, longitudinal data on adolescents admitted to an institution for the mentally deficient showed that they continued to make IQ gains into their late twenties. Second, in a cohort study of subjects labeled "educationally subnormal" at school, IQ gains were found in young adulthood among those with cultural-familial retardation. Third, a special education program, begun in the preschool years and maintained for about three years, improved the performance of mildly retarded children. Fourth, an experimental program of social and sensory stimulation, which started with pregnancy in retarded mothers and continued for four years, is reported to

have shown remarkable benefits for stimulated children compared to controls. Thus, improvement in the intellectual function of mildly retarded individuals is known to occur, and the application of pedagogic and social techniques offers promise of accelerating the improvement. Widespread use of these methods would be necessary if there were to be any hope of inducing changes in the prevalence or in the distribution of the degree of disability.

The severity of mental handicap can be reduced by developing the full social potential of mentally retarded individuals. In adults, social adequacy can often be achieved by those with mental handicap. We have noted that the sharp decline in the age-specific prevalence of mild mental retardation from the mid-twenties coincides with the somewhat delayed adoption of adult roles by mildly retarded persons. The young man or woman who marries, or who acquires economic independence through working, no longer occupies the social role of a retarded person. According to follow-up studies of mildly retarded men and women, two-thirds to three-quarters have settled well in the extramural environment. To socialize individuals affected by mild mental retardation to adult roles can be expected to advance their recoveries, and thereby to reduce the prevalence of handicap.

The concept of socialization can equally well be applied to severely retarded individuals. Severely retarded children living in a large institution, when they were removed to a smaller home and given more individual

attention, improved both their mental scores and their social skills compared with matched controls. Among adults, when moderately retarded people were encouraged to achieve semi-independence by living in residential hostels and taking up supervised work, the level of dependency declined accordingly. On the other hand, resettlement of residents from an institution into the outside world has often proved difficult; the difficulties point to the need for support and assistance beyond the immediate transition.

Comprehensive and coordinated programs aimed at pedagogic, social, and residential improvements are needed to reduce the severity of mental handicap. These programs, being recent, can be expected to do more in the future if they are properly planned and funded.

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Notes

1 A number of terminologies are available and in use in different classifications. The use of the term "retardation," not entirely satisfactory, relates to attempts to evade the stigma that has come to attach to such other terms as mental deficiency or mental subnormality (the latter introduced, for the same reason, in the British legislation of 1959). Unlike these terms, retardation does not necessarily imply a permanent state of dysfunction. It better describes that particular syndrome of mild mental retardation from which recovery does occur than all the conditions it has been made to designate.

2 In severe mental retardation, brain lesions have been found in about 90 percent or more at autopsy.

3 It is possible but unlikely that both the survival of single twins and their better performance are the outcome of environments more favorable than those in which pairs survive.

4 Another possibility is that cases of mental retardation that would once have been classified as severe are now classified as mild. If so, in order to accommodate such reassignment, the decline in the prevalence of mild mental retardation must be even more marked than we suggest.