Round Table Discussion

THE PROBLEM OF MENTAL DEFICIENCY IN CHILDREN

HERMAN YANNET, M.D., Southbury, Conn., Chairman
M. A. PERLSTEIN, M.D., Chicago, and ALBERT J. SHELDON, M.D., Santa Ana, Calif., Secretaries

Chairman Yannet: Mental deficiency may be defined as an interference with the development of intellectual capacity resulting in social inadequacy. This may be so severe as to necessitate complete custodial care indefinitely; it may be so slight, that a simple change in environment or program may allow for relatively normal community activity with minimal to no supervision. For these different degrees of disability a wide variety of terms have been used. In decreasing order of disability, the terms most frequently employed including their definitions in terms of intelligence quotients are as follows:

1. Borderline: I.Q. 70-80
2. Moron or high grade: I.Q. 50-70
3. Imbecile or middle grade: I.Q. 20-49
4. Idiot or low grade: I.Q. 0-19

Such terms as feeblemindedness and mental retardation are generally used interchangeably for mental deficiency.

Obviously, not all children failing to adjust socially or follow normal paths of development are so because of basic mental deficiency. Certain other conditions, either superficially or strikingly resembling mental deficiency states, must be differentiated. These latter conditions require a different and sometimes highly specialized therapeutic approach and any considerable delay in their recognition may adversely affect subsequent adjustment. The most common of these conditions include the following which are briefly described:

1. Delay in Educational Maturation: There is considerable variation in the time at which children of normal intellectual capacity reach the stage at which they properly react to the usual educational procedures, normally introduced at about 6 years of age. This so-called period of "educational readiness", for want of a better term, includes many aspects such as attention span, personal relations and socialization status, and may occasionally be delayed 2 or 3 years. This makes it difficult if not impossible for the child to adequately profit from the usual early educational experiences. If such a cause for delayed school progress is erroneously attributed to basic intellectual deficiency and so handled, the child may be more or less handicapped as regards future emotional development and adjustment. Adequate psychologic study readily allows for proper recognition.

2. Specific Defects: Gross visual and auditory defects need no special discussion. Minor degrees including high tone deafness occasionally present diagnostic problems. Less common are visual defects of cortical nature such as congenital or acquired word blindness. Also aphasias, which are language disabilities, due to cortical abnormalities, are being recognized with increasing frequency and represent important conditions to be differentiated since their treatment and handling differ radically from that of the mental defective. Whether the aphasia is primarily expressive or receptive in type, its presence may be suspected by the striking disparity between the lack of verbal accomplishment and the relatively normal levels of motor and social aspects of the child's development. Confirmation is obtained by complete psychologic evaluation.

3. Psychogenic Factors: Deep-seated emotional disorders and accompanying personality and behavior disturbances may seriously interfere with the normal processes of intellectual development to the extent that the resulting clinical picture may occasionally be mistaken for a fundamental intellectual deficiency. In such cases, as well, psychologic study including projective technics will usually suggest the basic cause for the developmental difficulty.

4. Psychotic States: While relatively rare, psychotic states in comparatively young children do occur, and because of the bizarre and unexpected behaviorisms may superficially resemble the more severe grades of mental deficiency. In most cases the history of a relatively normal early development in all spheres merging almost imperceptibly and without physical cause into grossly abnormal
behavior patterns associated with the tendency to lose most of the already acquired verbal accomplishments and to withdraw from personal contacts suggest the diagnosis, in many cases. Other clinical manifestations of this syndrome in children do occur, however, and prolonged observation is frequently necessary for differentiation.

Etiologic Classification

Factors or conditions responsible for the occurrence of mental deficiency may be operative during any phase of the prenatal period, at birth or during postnatal life. The relative importance of these 3 periods in determining the incidence of mental deficiency has not been adequately estimated for the general population at large. Data is, however, available in reference to institutionalized defectives. Such data, unfortunately, is by no means of random nature since admissions to mental defective institutions is dependent primarily on social considerations obviously not directly related to etiologic implications. For instance, one type of mental defective, the familial moron, is genetically determined and almost invariably springs from parents who are either themselves defective or at least of inferior or borderline intelligence. As a result of their limited intelligence and lower economic position, the community status of such families tends to accentuate the early social inadequacy of the offspring especially during early school years, and increases the likelihood of institutionalization. Thus the importance of genetic factors would tend to be over-emphasized in public institution statistics. Bearing this in mind, the data obtained at the Southbury Training School may be presented as representative of institutional experience. Thus, of approximately 2,000 admissions, in only 3% could injuries during birth be implicated. In 6%, postnatal factors such as central nervous system infections, trauma, immunizations, etc., were noted as causative agents. Over 90% were due to etiologic agents operative during the prenatal period. And, approximately half of these were definitely hereditary in nature. Of the remainder, most of the etiologic factors were either completely unknown or, like mongolism, of controversial nature.

It is possible at present to identify, either in etiologic groupings, or as recognizable clinical syndromes approximately 70% of all mental defectives. It is not the intention of this discussion to exhaustively present the various clinical conditions and syndromes with which mental deficiency is associated. Limitations of time make it necessary to simply indicate and briefly describe the pertinent details pertaining to the more common conditions.

Prenatal States

1. Genetic: As previously indicated, genetic factors play an exceedingly important role in the etiology of mental deficiency. It is possible to recognize various genetically determined conditions without too much difficulty even when no other similar cases have occurred to raise this question. The importance of early diagnosis of a genetically determined disease and some knowledge of the nature of the genetic transmission for the proper advice to parents needs no elaboration.

A. The familial type of mental defective, practically always of moron level, represents over 30% of the institutionalized population and close to 75% of the moron population at large, and is of considerable sociologic importance. The diagnosis can be made by the presence of a somewhat similar degree of mental retardation in other siblings, and defective or inferior intelligence in one or both parents. As a rule there are no clinical abnormalities of a neurologic nature and the configuration of head and body are not remarkable. The type of genetic transmission suggests a polygene mechanism not unlike that operative in the transmission of normal intelligence. Thus, from various reported studies, it has been found that when both parents are mentally defective, only 4% of the offspring are of average intelligence, the remainder being either of borderline or moron defective levels. When one parent is of moron and the other of borderline intelligence, offspring of average intelligence increase to 10%. When both parents are of borderline intelligence then about 28% of the offspring are of average normal levels and of the remainder, one-fifth are of moron level, and four-fifths, borderline. When one parent is normal and the other of borderline level, the incidence of moron defective levels is only 3% in the siblings, while 64% are normal and 33% are borderline. The familial moron represents one of the most important aspects of the entire mental deficiency problem primarily because of their numbers; and also because it is in this group that community failure in supplying specialized adequate education and training is particularly unfortunate. The relatively inadequate parents compound the community neglect, frequently resulting in serious antisocial behavior.
B. Phenylpyruvic oligophrenia was first described by Folling in 1934. It is a form of mental deficiency transmitted by a single autosomal recessive factor. Clinically it is characterized by a severe degree of mental defect, 80% being in the idiot or low imbecile levels, although in rare cases a borderline or dull normal level is encountered. There are no specific neurologic findings. The majority are poorly pigmented, over 80% being blond and blue-eyed. The metabolic defect is basically an inability to metabolize the amino acid, phenylalanine, presumably due to the absence of a specific enzyme. As a result, this amino acid accumulates in the blood reaching relatively high values. Before being excreted in the urine, it is presumably deaminized by the kidney, and appears as phenylpyruvic acid. This substance is easily identified by the addition of a few drops of 10% ferric chloride solution to previously acidified urine. A deep green color appears when phenylpyruvic acid is present. It can be found in very young infants as soon as milk is taken in reasonable quantities. The test in young infants can be carried out by the addition of the ferric chloride solution to freshly voided urine on the diaper. Although the absence of the specific enzyme prevents the metabolism of the phenylalanine, this amino acid can be normally incorporated into the body proteins. This has been shown by demonstrating normal levels in muscle and brain tissues examined at post mortem.

There are no specific pathologic changes in the brain although considerable architectural distortion is present in the various cell layers. There is, as yet, no explanation for the mechanism responsible for the mental deficiency. In view of the fact that there is no obligate relationship between the degree of metabolic defect and the severity of the mental deficiency, it is unlikely that nutritional manipulation could in any way influence the course. Investigations along these lines have as yet been without significant results. There is the suggestion that the mental deficiency and the metabolic anomaly may be due to separate functions of the same gene or of 2 closely related genes appearing together but having possible differential expressions. Phenylpyruvic oligophrenia occurs in about 1% of institutionalized defectives. It is estimated that 1 to 2% of the general population may be carrying this abnormal gene.

C. The term "congenital ectodermoses" covers 3 closely related conditions, namely, tuberous sclerosis, neurofibromatosis and cerebral angiomatosis. Genetically they have been considered as Mendelian autosomal dominants with extreme variability in expression. Because of this, especially for the first 2 conditions, the clinical pictures are extremely variable. Mental deficiency is only one of the manifestations and occurs in only a small percentage of those exhibiting evidence of carrying the abnormal gene. Among the mental defective population, tuberous sclerosis is the most common of the 3. In very young infants, delay in development may be the only presenting finding. Usually, however, rather characteristic skin lesions are also present during early infancy or appear soon thereafter. These consist of slightly raised, small, pinkish white soft lesions first seen on both cheeks, and occasionally scattered over the trunk. These pathologically are fibroangiomata, the intensity of the pink or red color depending on the degree of angiomatous involvement. Another finding often seen in these young infants is the rather typical mulberry-like retinal tumor or phakoma. As the child grows older, other phenomena appear. These include sebaceous adenoma, especially at the naso-labial folds, intracranial calcification most often around the ventricles, and extracerebral neoplasia. Convulsions may be an early manifestation or may appear later. All degrees of mental defect may be seen, and when present is usually not progressive.

Neurofibromatosis is so closely related to tuberous sclerosis as regards its manifestations in infancy and childhood as to suggest a unitary genetic cause. In some children, features ordinarily described for either of these conditions may appear simultaneously. The neurofibromata may vary from the smallest, barely perceptible cutaneous growths as described above to grotesque overgrowths in limbs resulting in elephantiasis.

Cerebral angiomatosis (Sturge-Weber) is less closely related to the preceding conditions although bearing some superficial similarities. It is characterized by extensive cutaneous hemangioma of the face, forehead and scalp usually on one side, associated with angiomatic neoplasm on the cortex. Probably because of pressure of the vascular tumors, the underlying cortex undergoes necrosis and early calcification. This results in characteristic radiographic findings of skull examination consisting of serpentine calcified areas that tend to follow the cortical gyri. Unlike the calcification in tuberous sclerosis which rarely appears before the seventh or eighth year, I have seen the typical x-ray findings in the skulls of children with cerebral angiomatosis soon after birth. Mental deficiency is relatively uncommon and when it does appear is usually of the moron level. Practically all develop some manifestation of convulsive disorder.
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D. Hereditary idiocy, due to an autosomal recessive gene, unlike the preceding conditions, has no other significant finding upon which to base a diagnosis except for a severe degree of mental defect appearing in 2 siblings and unexplained by other causes. There are no abnormal neurologic findings. Fortunately it is rare. Its importance stems from the inability of the physician to exclude it as a diagnosis in the case of the severely retarded child born to normal parents without adequate explanation.

E. Heredo-degenerative cerebral diseases may involve primarily either the white matter or the ganglion cells. They include a large variety of clinical conditions that in only a small number of cases can be differentiated during life, especially in infancy. Among the ganglion cell degeneration variety, Tay-Sach's disease and gargoylism are the most common. These are well described in many neurologic and pediatric texts. The white matter degenerative diseases are even more confusing in their terminology, and cover a wide variety of pathologic states that seem to have been described and named on the basis of isolated family studies, and variable periods of onset. These include the cerebral scleroses of Pelizaeus-Merzbacher, Scholz, Krabbe and others. Both the ganglion cell and white matter degenerative diseases are genetically determined and usually behave as autosomal recessives. They presumably represent metabolic defects of highly specialized enzymatic nature. Their importance in our discussion rests on the fact that delay in development may represent the first clinical manifestation of the condition in the very young child.

F. Muscular dystrophy of the childhood type, also described as pseudohypertrophic muscular dystrophy, is associated with mild degrees of mental deficiency in a few cases. The nature of the relationship between the muscular dystrophy and the cerebral malfunction is not understood. Genetically, the condition is probably a sex-linked recessive, which is practically confined to males.

G. Primary microcephaly is a distinct condition genetically determined and transmitted as a Mendelian autosomal recessive. It should be differentiated from a wide variety of other conditions either genetic or acquired in which diminution in head circumference is found. These include perinatal infections, degenerative diseases, birth traumas and congenital cerebral palsy. The appearance of the head is characteristic. The circumference may only be slightly decreased; the major diminution is in the cerebral volume due to an extremely low cranial vault. The forehead is strikingly sloped backwards. The facial bones, especially the mandible, age of normal size and by comparison appear enlarged. As a rule, there are no specific motor defects or neurologic abnormalities. The mental development usually is found in the high imbecile levels.

H. The craniosynostoses include those conditions in which one or more of the cranial sutures prematurely close. The evidence in favor of including these conditions among those genetically determined is strong. The condition behaves irregularly, in some families as a Mendelian dominant, in others, as a recessive. The configuration of the skull will depend on the sutures involved. The 2 most common varieties are oxycephaly in which the coronal suture is affected, and the head is short rising to a blunt top; and scaphocephaly, affecting the sagittal suture and resulting in a narrow and long head. Bizarre shapes will result if more than one suture or parts of a suture are involved. Occasionally other parts of the skeleton are defectively developed. In some cases of craniosynostosis, definite pressure symptoms appear. These include papilledema, optic atrophy, exophthalmos and increased cranial digitations. Mental deficiency may be present and may vary in degree from the most severe to a mildly involved moron state. There is considerable evidence to indicate that the mental defect, when present, is probably an associated symptom rather than secondary to the premature suture fusion. Regardless as to whether mental deficiency is a secondary phenomenon or not, however, operations designed to prevent the disfiguring effects are definitely indicates for cosmetic purposes at least, in many cases.

1. Hypertelorism is an uncommon cranial anomaly in which the eyes are widely separated and the root of the nose depressed. Varying degrees of mental deficiency may be associated. Basically, the configuration of the skull is due to the overgrowth of the small wings of the sphenoid, although other anomalies of development at the base of the skull are seen. There is no direct correlation between the degree of skull anomaly and the intensity or even the presence of mental deficiency since certain of these children may be of relatively normal intelligence. There is considerable evidence in the literature to indicate a genetic basis for this disorder. It appears to behave as an irregular dominant in some families and recessive in others. As in other conditions, mutations may explain the frequent sporadic appearances. Also of interest is the high incidence of congenital heart anomalies in this condition.
2. Infections: Prenatal infections as a cause of mental deficiency have recently been stressed. Although the whole story is far from told, we can only definitely recognize at present 3 distinct etiologic agents of any consequence. These include rubella, toxoplasmosis and congenital syphilis. In all 3, mental deficiency represents only one aspect of what may be a complicated clinical picture. Because the fetal infection with rubella virus occurs as early as the first trimester, the degree of abnormality both cerebral and otherwise is usually the most extensive. The clinical picture may be quite variable. When associated with the more severe degrees of mental deficiency, the clinical manifestations include microcephaly with or without motor defects, cataacts, congenital heart disease and deaf-mutism. Prenatal infection with toxoplasma apparently occurs later in pregnancy. Occasionally infection may take place in the paranatal period. The presenting symptoms are variable depending in part on the time of infection. Mild hydrocephalus or microcephalus usually with intracranial calcification, and almost always associated with bilateral chorioretinitis is the most common picture. The intracranial calcification is widely disseminated in the peripheral cortex, and tends to be granular in appearance. In this respect it greatly differs from the calcification seen in tuberous sclerosis which tends to be periventricular and conglomerate in appearance. Congenital syphils is now a very rare cause of mental deficiency. In addition to the usual signs of congenital syphils, in these children there is also neurologic evidence of central nervous system involvement including changes in the C.S.F. The mental defect is usually in the moron levels.

3. Irradiation: This is extremely rare clinically, and results from the application of therapeutic doses of x-ray or radium to the pelvis of a pregnant woman during the early months of gestation. Severe fetal cerebral malformations with symptomatic microcephaly and marked degrees of mental defect are the most common manifestations in the subsequently delivered infant. Considerable experimental work in animals has confirmed these observations. Eye and skeletal defects have also been produced in the experimental animals.

4. Isoimmunization: Maternal Rh or ABO isoimmunization may result in fetal cerebral damage. It is a relatively uncommon cause of mental deficiency, being responsible for approximately 1% of the institutionalized mental defectives. In addition to mental deficiency, the clinical manifestations include microcephaly with or without motor defects, cataracts, congenital heart disease and deaf-mutism. Prenatal infection with toxoplasma apparently occurs later in pregnancy. Occasionally infection may take place in the paranatal period. The presenting symptoms are variable depending in part on the time of infection. Mild hydrocephalus or microcephalus usually with intracranial calcification, and almost always associated with bilateral chorioretinitis is the most common picture. The intracranial calcification is widely disseminated in the peripheral cortex, and tends to be granular in appearance. In this respect it greatly differs from the calcification seen in tuberous sclerosis which tends to be periventricular and conglomerate in appearance. Congenital syphils is now a very rare cause of mental deficiency. In addition to the usual signs of congenital syphils, in these children there is also neurologic evidence of central nervous system involvement including changes in the C.S.F. The mental defect is usually in the moron levels.

5. Miscellaneous Conditions:

A. Mongolism is one of the most common of classifiable conditions among mental defectives. It represents approximately 10% of institutionalized defective children. It occurs about once in every 500 to 700 births. It can be diagnosed at birth, although many times with difficulty, the condition having begun to differentiate very early in fetal life probably around the sixth to the eighth week. While the diagnosis requires the presence of the peculiar and characteristic skull deformity which results in the upward and outward slant of the eyes, the disorder is widespread. Associated anomalies include defective skeletal growth, cardiac abnormalities, congenital eye disorders, and many others. Mental defect is most commonly in the imbecile range and always present.

While there has been considerable research and discussion concerning etiology, no viewpoint has been generally accepted. For myself, the available evidence favors the hypothesis that the defect is basically genetic but that its expression is influenced by various environmental factors such as maternal age, hormonal deficiencies, local uterine abnormalities and others. If this is so, the possibility of prevention may lie in our ability to recognize and modify these environmental factors. As regards treatment, there is no good evidence that the expected course of the condition can in any way be beneficially influenced by any available medicinal therapy, endocrine or otherwise.

B. Cretinism probably represents the only endocrine disorder that seems to be specifically related to mental deficiency. While many other endocrine disorders can be found among defective children these are associated abnormalities rather than a direct cause of the mental state. So-called congenital myxedema or cretinism appears to be of 2 types which may have different pathogenic mechanisms, or may merely represent a matter of degree of thyroid deficiency. Although both types usually require a few months for recognition, in one, the administration of thyroid seems to completely correct the disorder and allow for subsequent normal development both physical and mental. In the other, thyroid may stimulate physical growth effectively but a completely normal child, particularly mentally, is not obtained. The etiology is not clear in every case. While genetic factors probably do play a role in some cases, sporadic examples are numerous, especially in this country and may be environ-
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mentally determined. Obviously, thyroid is indicated in every case and early diagnosis should be sought.

C. Approximately 30% of institutionalized mental defectives can be recognized as being prenatally conditioned but are completely devoid at the present time of differentiating characteristics that would allow for accurate classification. To this large group, the term "undifferentiated" is applied. Its size indicates the tremendous gap in our knowledge of prenatal injurious factors.

Natal and Postnatal Conditions

As was indicated earlier, on the basis of institutional statistics only 9% of mental deficiency states are due to factors operative at birth or during infancy. Diagnosis is usually readily made at the time of the acute condition. However, etiologic diagnosis made in retrospect many years after birth presents considerable difficulty and is frequently unreliable. The criteria demanded for retrospect diagnosis varies considerably in different clinics and by different investigators. This undoubtedly explains the striking discrepancies in the incidence of acquired factors in the various studies which have appeared. It is unnecessary to do more than list these possible factors which should be considered in reaching an etiologic diagnosis.

1. Birth trauma includes various injurious mechanisms such as cerebral hemorrhages, thromboses especially of the cerebral sinuses, subdural hematomas, both unrecognized and those unsuccessfully treated, and prolonged asphyxia, either due to delay in initiation of adequate pulmonary exchange or interference with umbilical blood supply.

2. Postnatal conditions include infections of the central nervous system either suppurative or non-suppurative including postimmunization reactions; acute cerebral trauma resulting from accidental falls and head injuries; cerebral vascular thromboses arterial or venous, many at present spontaneous and unexplained, a few secondary to severe infantile dehydration; spontaneous intracranial hemorrhages on the basis of ruptured congenital aneurysms or mycotic aneurysms; asphyxia associated with drowning accidents, gas poisonings including anesthesia accidents and acute tracheobronchial obstructive conditions; certain central nervous system poisons accidentally ingested, and probably others.

Treatment

A. Organic Methods: The last few years has witnessed an increasing interest in possible organic methods for stimulating or accelerating the mental development in established states of mental retardation. These have included the use of large doses of glutamic acid, a preparation of calf pituitary for mongoloid children, excess vitamin B6, and a surgical procedure (cerebral revascularization) designed to increase cerebral blood flow. Two of these have been adequately tested under reasonably well controlled conditions and have failed to live up to the original expectations (glutamic acid and cerebral revascularization). It is likely that their employment for the original purpose of improving basic intellectual status will diminish and soon disappear. Too few reported results are available for the remaining methods on which to base any conclusions. On the available evidence my reaction is pessimistic as to obtaining eventual significant results. It is heartening, however, to note the increasing interest being given to the direct organic therapeutic approach to the problem of mental deficiency.

B. Educational and Social Procedures: The foundation of treatment, using the term in its broadest sense, for mental defectives still remains based on the following 3 fundamentals:

1. Specialized education consistent with the intellectual capacity.
2. Industrial training to prepare the individual for useful work if possible, and
3. Providing, at all times if necessary, an environment that will allow satisfactory personality and emotional development.

While children of practically all degrees of defect except for the most seriously retarded could profit from such a program, it finds its greatest application to the so-called "trainable group." These represent children with intellectual capacities in the ranges of the high level imbecile, the moron and the borderline grades. Under ideal conditions, the proper facilities and environment would be supplied by the home, public schools and community. Unfortunately, these conditions are not always available, especially in the smaller communities. It should be one of the responsibilities of the medical profession to help stimulate the local school systems to make adequate provisions for the care and training of these children. As is true for the normal child, the trainable defectives are best handled in their own homes. This is particularly important during the early years of childhood.
The adequacy of the early home and school experiences will in great part determine the success of later industrial and community adjustment. Studies in communities supplying such specialized educational facilities have indicated the excellent prognosis to be expected. (Trainable mental defectives: Comment on prognosis, J. Pediat. 37: 816, 1950.) The institutionalization of the trainable defective should be necessary in only a small proportion of these cases. The indications should include one or more of the following: (a) a home situation that is grossly undesirable; (b) a community completely devoid of facilities; (c) the development of serious behavior or personality difficulties in the child. Of these, the most common is unsatisfactory home environment, most frequently due to inadequate parental adjustment and understanding. Much of the subsequent behavior difficulties of the occasional child so exposed can be traced to this factor. Although early parental training and advice will partially solve some of the problems and reduce the need for institutionalization, this is not always possible. It is, of course, apparent that should it be necessary to remove such a child from the home for institutional care, one must at least assure himself that a suitable educational and training program will thereby be made available. Too many of our state schools for mental defectives, as well as many of the private schools, fail strikingly to supply these needs. Another possibility to be explored in these cases, prior to institutionalization, is the use of suitably situated foster homes, in communities with adequate facilities. Our experience repeatedly confirms the oft repeated observation that the finest of residential training schools and institutions is still inferior, as a child raising agency, to adequate homes, personal or foster.

The other category of mental defectives, the custodial, includes those children whose mental capacity will make it impossible to attain a condition of even partial self support in the community regardless of the highest level of training and care. These include all of the idiot level, and most of the imbecile or middle grade defectives. Approximately 20 to 25% of all defectives fall into this category. Unlike the trainable group, a reasonably large number of these children can be recognized as custodial almost from birth: for example, the mongoloids and the primary microcephalics. The problem for physicians, as regards planning for future care, and parental advice, will therefore present itself either soon after birth or during infancy. Moreover, the expected high mortality rate of preantibiotic days in these children, which tended to encourage procrastination on the part of the physician as regards discussion of future care with parents, has been strikingly reduced. While the parents will and should make the final decision, it is the responsibility of the physician to present all the facts in such a way that the parents' decision will be based on real understanding. What are these facts? They will be discussed using the mongoloid child as an example, since this represents the most common custodial type that can be recognized soon after birth; and from 3 points of view, namely, the child, the family, especially the mother, and economic considerations.

1. The mongoloid infant will undoubtedly receive better care from the exclusive attention of its mother than it can expect in any institution. Also, as is true for the normal child, the mongoloid child may be expected to profit considerably, developmentally speaking, from the security, affection and attention afforded by its parents and family during the early years spent in its own home. Any possible benefits, as they may affect the parents or family, to be expected from removing the mongoloid infant from its home soon after birth or during early infancy, should be weighed against these considerations. As the mongoloid child grows, and its deviation from the normal becomes increasingly evident, the possibilities for childhood companionship and play become increasingly limited in his own home. Thus there comes a time when what the mongoloid child gains from home care is greatly overshadowed by what he misses in the opportunities to explore human relationships and develop at his own rate. A point of diminishing returns is almost invariably reached with adverse effects on both the child and family. The time when this point is reached is quite variable, depending on the home environment and the degree of developmental retardation exhibited by the child. In some, it may be at 2 years or earlier. In others, it may not be before 6 or 8 years of age. If the family is adequately educated to this fact, they can usually determine this point for themselves. The institutionalization of the child at this time becomes advisable for his own sake. With few exceptions, a family, especially the mother, who has been informed of the plan from early life and has been a partner in reaching a decision, has little difficulty in adjusting to the removal of the child from the home to another environment more suitable for the child's emotional well being.

2. There are only 2 valid reasons that a physician can give for advocating the removal of the mongoloid child from the home soon after birth. First is the possible adverse effects on the family, especially siblings, in having a custodial problem in the home. Second is the possibility that the
mother or family will become so emotionally attached to the child that they will not be able to allow the child to leave the home when the proper time is reached. Experience has shown that both are valid reasons; but, I believe they apply to only a small percentage of cases. Here again, the physician, in frank discussion with the family, can help decide jointly if such possibilities might be reasonably expected to occur in spite of whatever psychotherapeutic help the physician can give through his contacts with the family. It may be mentioned as well that, from my own experience, I have no reason to believe that the emotional development of normal children is adversely affected by the presence of a custodial type of defective child in the properly oriented family.

3. We may reasonably expect that at no time will there be enough public institutions to care for all custodial children of this kind from early infancy. In the absence of available public facilities, advice to remove these infants from the home makes it necessary to use private institutional care. Should any considerable time elapse before a bed is available in a state institution, the cost to the family may represent a considerable burden. Only if a physician can convince himself and the family that the financial sacrifice is commensurate to the benefits to be derived from such a procedure should the expenditure be advised.

C. Sterilization: Sterilization is an important auxiliary procedure in the management of mentally defective individuals, especially females in the trainable category. Although it has eugenic implications, its greatest value lies in the social sphere as regards the over-all problem of mental deficiency. The prevention of parenthood in the defective is indicated socially on 2 grounds. First, the mentally defective individual will make, as a rule, a poor, ineffectual parent regardless of the mental status of the child. Second, the problems of pregnancy in the unmarried and parenthood in the married are frequently responsible for adversely affecting the social adjustment of the defective in the community necessitating return to the institution, or requiring an original commitment in a previously well adjusted situation.
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