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MENTAL RETARDATION IN THE INFANT AND PRE-SCHOOL CHILD

DIAGNOSIS AND TREATMENT*

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The subject of mental retardation is such an extensive one that for the purposes of this paper I am confining my remarks to two aspects of the problem in the infant and pre-school child—the diagnosis and treatment.

DIAGNOSIS

There is a difference of opinion whether the future intelligence of a child can be predicted in the first two years. On the one hand, Bowlby (1951) wrote: "Mental tests have no predictive value in the first eighteen months of life." Kirman (1953) wrote: "It is only very seldom that a child of less than 2 can safely be diagnosed as definitely mentally defective." Bailey (1933) compared the score achieved by 49 children in 200 test situations in infancy with their intelligence quotient at intervals till they were 3, and found little correlation. Her patients, however, were selected children of high intelligence. Linfert and Hierholzer (1933) and Furfey and Muehlenbein (1932) found a similar lack of correlation between tests in infancy and intelligence four years later. The opinion given by Bowlby and by Kirman was apparently based on their experience and on the work of others. The opinions expressed in the remaining three papers were based on carefully conducted studies, but on purely objective tests which would fail to take into consideration the child as a whole, including particularly his interest, alertness, and concentration, against the background of his environment.

On the other hand, Gesell (1943) wrote: "Practically every case of mental deficiency can be diagnosed in the first year of life, excluding, of course, the small number of exceptional cases which occur from secondary causes in later infancy or childhood." Gesell *et al.* (1930) wrote that they had carried out developmental examinations on 10,000 infants, and many thousands of babies have been examined since that date. Gesell's opinion, therefore, is based on considerable experience, and it is obvious from his books and from observation of the work in his clinic that his studies are based on painstaking records and follow-up examinations. Hallowell (1932) and Nelson and Richards (1938) found good correlation between the Gesell tests in the first year and Stanford-Binet tests at the age of 3 to 4 years.

It is surely obvious to anyone accustomed to the developmental examination of children that babies who are uniformly backward in development in the first year nearly always remain backward in later years; and that in mentally defective children there is always a history of lateness in passing the milestones of development in infancy, unless, of course, the mental deficiency resulted

from trauma, encephalitis, or other condition in later months. I have no doubt that, although there are many pitfalls in the diagnosis, mental retardation can be diagnosed in the first year of life.

The diagnosis is made in four essential stages.

1. Developmental History

It is often said that a developmental history is useless, because parents forget so readily the age at which their children acquired various skills. I disagree with this attitude and attach the greatest importance to the developmental history. In taking a history from anyone it is essential to be certain that each understands exactly what the other means, and then on the basis of one's experience and knowledge of the subject, and one's assessment of the truthfulness and memory of the patient (or his parents, in the case of children), to decide what one can accept or discard. For instance, one knows from experience that if a mother says that her child learnt to sit without support at 11 months and to walk without help at a year she is not speaking the truth or her memory has failed her. One has to balance one statement against another, and the total picture which she gives against one's subsequent objective findings. When one is doubtful about the accuracy of an answer it is often wise to come back to the question later, asking it in a different way, in order to see how the answers tally.

In the case of the developmental history, it is useless merely to ask a mother when her child first smiled, grasped objects, held the head up, sat, walked, talked, or acquired clean habits, for the questions are not precise enough, and the replies will be meaningless. A mother may consider a facial movement during sleep or a contortion of the face from wind to be a smile, whereas the only information of value is the age at which the baby first began to smile in response to her overtures (average 6 weeks). A baby will grasp an object when it is placed in his hand at the age of 3 months, but he is unlikely to go for an object and get it until about 5 months. A baby can hold his head up momentarily when held in the sitting position at 2 weeks of age, but he cannot lift his head up from the supine, lying on a firm surface, until he is about 6 months old. A child can be held in the sitting position at any age from birth onwards, and he can be propped in the sitting position from 4 months or so; he can sit apparently unsupported in a pram a good month before he can sit for a few seconds without support on the floor (average 7 months). He can walk with hands held at 9 or 10 months, but he cannot usually walk without support until 13 to 15 months.

*Based on a lecture given to the Hull Medical Society, November 19, 1954.

Babies at 6 months of age are thought to be "talking" when they say "mum, mm, mm" in crying, and at 8 or 9 months of age when they combine syllables to say "da da da" and "ma ma ma." These are essential steps towards speech, but the child does not usually say one or two words *with meaning* until the age of 10 or 11 months, and put two words together till 21 to 24 months. A child may be said to be clean and dry from the age of 3 or 4 months, but this is merely the result of "conditioning," and not voluntary control. A child does not usually tell his mother that he wants to urinate until 15 to 18 months, and this is the first milestone concerning sphincter control which is of value in diagnosis. Many more examples could be given to show the importance of care and accuracy in taking a developmental history.

The developmental history, in addition to being accurate, must be comprehensive, covering, so far as is possible, all fields of development, and particularly the general understanding shown by the baby. This is essential, for the *cardinal feature of mental retardation in an infant is the fact that he is retarded in all fields of development, except occasionally in sphincter control.* Conversely, no child is seriously mentally backward, provided that his defect is acquired before birth, unless he is backward in all fields of development, except perhaps sphincter control. The history, therefore, must cover not only the sensorimotor development, the age at which he learnt to grasp, roll, sit, crawl, creep, and walk, but, of much greater importance, his general understanding, the interest he shows in things and people, and his powers of concentration. Of value, for instance, is the age at which the baby began to turn his head to sound (average 3 months), to play games, such as peep-bo (average 24 weeks), pat-a-cake (average 40 weeks), to wave bye-bye (average 10 months), to imitate his parents in coughing, putting the tongue out, or knocking on the table (average 6 to 9 months), to excite when he saw the feed being prepared (average 4 to 5 months), to hold his hands out to be pulled up into the sitting position (average 24 weeks), to hold his arm out for the sleeve of a coat or his foot out for his shoe (average 12 months). In addition, it is important to determine the age at which he began to chew (average 6 months) and to feed himself and manage a cup, lifting it up and placing it back on the table without help (average 15 months). The mentally retarded child is late in learning to chew, and if the mother gives him solid foods before he can chew them he is likely to vomit. A history of repeated unsuccessful attempts to introduce solid foods into the baby's diet is very commonly obtained in these cases.

In the older child, from 15 months onwards, it is useful to ask about "domestic mimicry," the age at which the child began to imitate the mother in her domestic duties, sweeping, cleaning, and washing clothes and china. The child's doll play, the way she dresses and undresses it, "trains" the doll in toilet habits, and puts it to bed, is of value in assessing the child. The average child begins to imitate her mother in her domestic work at 15 to 18 months.

In assessing speech it is essential to realize that children learn the meaning of words long before they can articulate them. Some children who are only able to say four or five words can point out the names of dozens of objects in books—a sure sign of good intelligence in a young child.

Other inquiries concern the ability of the child to dress himself and to carry out simple errands in the house, his interests, and especially the degree of concentration which he shows in his play.

With a comprehensive history of this sort the diagnosis is often obvious before one has begun to examine the child. For instance, a 16-months-old baby was seen because he was not nearly walking, and had only just begun to sit up without support. He had learnt to grasp objects voluntarily at 8 months. He was saying several single words with meaning before the age of 12 months, and was playing games, imitating his parents, and anticipating, by laughter, when

they were about to play with him, long before his first birthday. The diagnosis was a mechanical difficulty, almost certainly cerebral palsy, involving mainly the lower limbs, with a normal mentality. The diagnosis was readily confirmed by physical examination.

Finally, the developmental history should include questions about the delivery of the baby, his condition immediately after birth and in the subsequent two or three weeks. Particular attention is paid to a history of difficult delivery, asphyxia at birth, cyanotic attacks, cerebral irritability, twitchings or convulsions, and undue drowsiness, with sucking difficulties. A history of birth injury, with either severe asphyxia or cerebral haemorrhage, should make one's prognosis in a retarded infant particularly guarded, for one occasionally sees such a child who is definitely backward in the first months of life catch up to the average level at the age of 1 or 2 years. It should be remembered that, since a mentally backward child is retarded in all fields of development in early infancy, he will correspond at birth, when born at term, with a mentally normal premature baby, so that, like the premature baby, he will be liable to asphyxia, cyanotic attacks, and sucking difficulties. I believe that, although cerebral palsy or mental deficiency can often be correctly ascribed to asphyxia at birth, in some cases the asphyxia may well be the result of underlying cerebral agenesis and not the cause of the defects. It should be noted that the great majority of babies showing severe asphyxia at birth develop normally, without any defect.

One often finds a history of undue drowsiness in the newborn period of retarded children, with defective sucking, disinterestedness in feeds, and frequent regurgitation. Like the normal premature baby, the full-term retarded baby may sleep for the greater part of the day—more than the average intelligent full-term baby does. This tendency to sleep more than the average normal baby often persists for the first few weeks, though, of course, some normal babies sleep much more than others in this period. Mothers of such babies are often very pleased because he is "no trouble at all," or "such a good baby." This often explains the confusing story that "he was quite all right until he was 9 months old."

2. Family and Environmental History

Lateness in certain fields of development, especially walking, talking, and sphincter control, often runs in the family, and therefore a history of this should be sought.

The mother's attitude and management of the child may be important in establishing the diagnosis. Excessive fear of spoiling the child, for instance, may well lead to emotional deprivation and so to some degree of retardation. A child who is left lying out of doors in a pram for the entire day, from the age of, say, 6 months to a year or more, may be retarded in play and social behaviour when compared with a child who has been given normal love and play stimulation. A certain amount of delay in sitting may result from the mother's unfounded belief that the spine would be weakened if the baby were to be propped up in the pram. Some retardation in standing and walking results from the mother's belief that rickets, knock-knee, or bow-legs will result if the child is allowed to bear his weight. Severe illness will retard the development of sitting or walking. Excessive efforts to train the child in clean habits are likely to retard the development of sphincter control. I saw a child of 3 years with incontinence of urine and faeces resulting from excessive toilet training, the child having been soundly smacked from the age of 3 weeks onwards if she did not have a motion or pass urine when placed on the chamber. Most babies who have been brought up from the earliest infancy in an institution are retarded considerably in all fields of development, and it is essential to take this into consideration in assessing such children for adoption. They usually make very rapid strides forward and quickly catch up to the average, if of normal intelligence, when they are placed in a good foster-home.

3. The Examination

The routine physical examination includes inspection for the signs of mongolism, cretinism, microcephaly, and hydrocephalus, examination of the optic fundi for choroidoretinitis, optic atrophy, or other abnormalities, examination for hearing, where relevant, and examination of the urine for phenylpyruvic acid. Signs of cerebral palsy may be detected in this part of the examination, but they will be more likely to be detected during the developmental examination. I personally pay little attention to many of the so-called stigmata of mental deficiency, such as abnormalities of the ear or a high arched palate, because they may occur in otherwise normal children. The date of eruption of teeth is of no value in diagnosis. The date of closure of the fontanelle is so variable that no reliance can be placed on it as a sign of normality of development.

The developmental examination must cover all fields of development if errors are to be avoided, owing to the fact that many normal children are backward in certain fields, such as sitting, walking, or talking, and not in others. The developmental examination was described in detail by Gesell and Amatruda (1947), and was summarized elsewhere (Illingworth, 1953). Ruth Griffiths (1954) recently described a battery of tests for the assessment of babies. Tests for the older pre-school child are described by Gesell *et al.* (1940). Space will not permit of a discussion here of the methods of examination. The examination must be performed at the child's normal playtime, when he is neither tired nor hungry.

As the mentally backward child is retarded in all fields of development there is no need to describe here the usual findings in such a child. Some features, however, are worthy of mention. The first sign of abnormality noted is likely to be the child's lateness in watching his mother's face when she talks to him (an average 4-weeks-old baby watches his mother's face intently as she speaks to him, often opening and closing his mouth and bobbing his head up and down). He will be late in smiling, and in head control, as shown by pulling him to the sitting position, placing him in the prone, or holding him off the couch with the hand under his abdomen. The average 6-weeks-old baby when he is held in the latter position should be able for a few seconds to hold his head up so that its plane is in that of the long axis of the trunk. As he grows older his defective head control becomes progressively more obvious. It is the most important early objective sign of abnormality.

Other features of note in the retarded baby, other than the obvious signs of general retardation, include the lateness in following with the eyes and of taking notice, which so often leads to an erroneous diagnosis of blindness; lateness in turning the head to sound (3 months in an average baby), which often leads to an incorrect diagnosis of deafness; lateness in stopping to drool and to mouth objects (both usually achieved at about 12 months in an average baby, with occasional exceptions in the succeeding weeks); the persistence of the reciprocal kick, long after the age of 9 months, when it is normally lost; and, more important than any of these, the general disinterestedness of the baby, the lack of prolonged concentration, and the slowness with which acts are performed.

On completion of the examination one estimates the level of development reached by the baby in the various fields studied, making, of course, an allowance for prematurity, for if the baby were born, say, two months before term an addition of two months must be made to his age before his level of development can be related to his age. Further examination at an interval or intervals will then show how far the child has progressed in the intervening period, and a much more firm conclusion can be reached than after a single examination. As Kirman (1953) emphasized, delay in diagnosis of mental deficiency is seldom of much importance, except in the case of cretinism. Until one is certain of one's diagnosis, nothing is said to the parents about one's suspicions.

4. Interpretation of the Findings

After the developmental, family, and general histories have been taken, and the physical and developmental examinations have been completed, it remains to interpret the findings, and here serious mistakes can readily be made. Each item in the developmental examination is considered against the background of the history and physical examination, and every possible extenuating factor which would account for a poor performance in an item is taken into consideration. Special attention is paid to lack of opportunities to learn, illness, institutional care, and physical difficulties, such as partial deafness or abnormalities of muscle tone. The tremendous individual variations in the age at which skills are learned are particularly remembered. Special note is taken of a family history of lateness in acquiring skills in certain fields, such as speech. One will not be misled by the child's tiredness, hunger, bad behaviour, uncooperativeness, or shyness in the tests, proper allowance being made for these in interpreting the results. One will discard those items which are clearly influenced by extraneous factors, such as those mentioned above, and in the final assessment one will use only those items which seem to be true indications of the child's development.

One cannot, in other words, write down a score for each of 20 items, add the figures up, divide by 20, and conclude that the figure so obtained represents his present level of development. By trying to be accurate and scientific I believe that one may well arrive at an entirely wrong conclusion. I believe that the most accurate assessment of a child's development is best achieved by a judicious guess, based on a thorough painstaking history and a full physical and developmental examination, interpreted in the light of common sense and experience. No laboratory studies can replace this method of examination. Not even a pneumoencephalogram is sufficient, for Amatruda (1942) demonstrated a lack of correlation between changes in the pneumoencephalogram and developmental performance.

A comprehensive study of the type described will prevent one being misled by a child's peculiar face, epicanthic folds, mongoloid facies, or other physical features which may well lead the unwary into an incorrect spot diagnosis of mental deficiency. Even a considerable degree of hydrocephalus is not incompatible with a relatively normal level of intelligence. One will not be misled by the common lulls of development in speech or other items which occur for no apparent reason when other skills are being actively learnt, or by the extreme stereotypy of behaviour with regard to a single toy or occupation (Gesell, 1943), often seen in a mentally defective child, which superficially resembles a high degree of concentration. Nor will one be misled by apparent advancement in a particular field, which so often impresses parents, in a child who, considered as a whole, is quite severely mentally defective.

The examination will pick up the rare case of infantile autism, the child who superficially looks to be mentally defective because of his remarkable lack of interest in people and his lack of response to affection and social overtures, but who achieves a good score in the developmental examination. One will not make the mistake of overestimating the intelligence of a child on account of his good and attractive appearance. One will be helped to avoid making the mistake of diagnosing mental defectiveness when in fact the child's bad behaviour and absence of speech is due to partial or complete deafness. High-frequency deafness is particularly apt to be missed, because the child hears many ordinary noises and discriminates well between them, but cannot hear sufficiently well to learn language. When a mother is told that such a child is just "lazy," and advised to ignore him unless he speaks, most troublesome behaviour problems are produced, just as they are in children with normal hearing who are late in speaking and who are treated in this way. It is very easy to make the mistake of diagnosing mental defectiveness in these children unless a really full history and examination is made.

One will not rely on intuition, clinical impression, or physical stigmata, but consider the child as a whole. Admittedly any doctor can make an instantaneous diagnosis of mongolism, though there are mistakes. Thanks to the many studies of mongolism, he can predict with considerable accuracy what the child's future achievements will be. Parents, however, are not impressed by such spot diagnoses. They think, quite naturally, albeit wrongly, that the doctor cannot possibly know so much about an individual without examining him. In Arnold Gesell's clinic at Yale no "spot" diagnosis of this kind was ever made. Although the diagnosis and prognosis was immediately obvious as soon as the child was seen, Gesell and his staff treated mongols and other severely defective children in the same way as other children, taking a full detailed history and carrying out a detailed developmental examination, thereafter in conference interpreting the findings. Then and then only did the parents feel that their child had been properly investigated, and as a result not only were they grateful for the trouble taken but they paid real attention to the doctor's verdict. Much of the so-called "shopping round" by parents of mentally defective children from one doctor to another, and their refusal to accept the verdict of mental deficiency, is due to the doctor's inadequate examination and discussion.

Real difficulty is presented by the very occasional slow starter, who subsequently catches up to the average, and who in my experience has always had asphyxia or birth injury, and by the child with severe cerebral palsy, who is retarded in almost all fields of development. It will be impossible to detect the child who, after a period of normal development, will suddenly begin to deteriorate as a result of a degenerative disease of the nervous system or as a result of trauma or encephalitis. With a proper examination, however, I believe that the mistakes made will be few.

TREATMENT

Breaking the News to the Mother

When one is certain of one's diagnosis, and not before, the mother must be told, but not necessarily immediately. It is a matter of opinion how soon the child's retardation should be discussed with her. When a mongol is born it is probably wrong to tell the mother in the first two or three weeks after delivery, when she is in a particularly emotional state, but it may be wise to tell the father, so that when she does hear he will be able to give her some moral support. There is something to be said for allowing the mother to discover the child's backwardness herself: in that case she is likely to discover it between 9 months and a year, when the child is late in learning to sit. On the other hand, this might well cause her to repress her fears and worries about the child before she eventually tells her husband about them. This is undesirable, and she may feel considerable resentment against the doctor for not having told her about the real position sooner. The alternative, which I think is the right one, is to tell her three or four weeks after the baby's birth. It is a matter of opinion whether it is better to dash her hopes and ambitions for the child early or late. I am certain, however, that whatever is decided, it is absolutely wrong to tell lies about the child, saying that he is merely lazy and will eventually be normal or, worse still, that there is nothing wrong, and nothing to worry about. Deception of this nature never does any good. I remember witnessing in Gesell's clinic the resentment and anger of a professional man against his doctor when Gesell told him about his child's mental defectiveness. The child was an 8-year-old mongol, and the parents, at the cost of great personal privation, had paid very high fees for the boy's education in a private school. Only after three or four years did the parents realize that the boy was hardly learning anything, and then they consulted Gesell. No one had given them any idea that there was anything wrong with the boy.

The next question to decide is how to tell the mother about her child's retardation. The first essential is to possess

real sympathy and feeling for her, and to realize the appalling tragedy of mental defectiveness. We must admit that medical people, doctors and nurses, see so much human suffering that they become hardened to it. One sees all too often an attitude of complete indifference to a problem of a mentally defective child. I heard a doctor, referring to a teaching round for students, say, "By a stroke of luck there was a beautiful newborn mongol to teach on." When mental deficiency can be diagnosed at a glance there is a tendency for a doctor to give a consultation which is extremely brief, and merely refer the parents to the lady almoner for further information. The lady almoner can help a great deal by advising the parents about facilities for the institutional care of these children, but it is the responsibility of the doctor to discuss the numerous problems of the individual child with his parents, in particular with regard to the diagnosis and prognosis, and the pros and cons of institutional care. The lady almoner will also help the parents by amplifying what the doctor has said about facilities provided for these children in the way of special schools, occupation centres, and home teachers. Where necessary she will arrange help through various statutory and voluntary bodies. She can help a lot to prevent the parents from going away with the impression that nothing can be done to help them in their plight.

It is a good practice for the doctor, having formed his conclusions, to ask the parents what they really think of the child and even to ask them, in suitable words, what is their assessment of his level of development (Jensen, 1950). When they say that they know that the child is backward the doctor agrees with them.

Certain words must be absolutely avoided in the discussion because of the distress which they cause. They are "mental defectiveness," "mental," "imbecile," "feeble-minded," "moron," "idiot," "it's his brain." The best words to use are "backward" or "retarded." It is essential, however, to make it clear that this is not just a temporary backwardness which will right itself. They should know that he will always be backward, and that he will not "grow out of it." It is a mistake to raise false hopes which will subsequently be dashed to the ground. Zwerling (1954) gave a most valuable analysis of the views of parents of defective children on how they should and should not be told about the defectiveness. The paper emphasized the importance of being kind, sympathetic, and gentle in breaking the bad news. Kanner (1953) also discussed this problem.

Parents often feel guilt and bitterness about the defectiveness of their child, and think that the defect is due to some fault of their own. This idea should be anticipated and appropriately dealt with, making it quite clear that the trouble is not due to any infection or mistake in either parent, or to birth injury as a result of bad obstetrical care. In my opinion it is a mistake to delve too deeply into the family history for some evidence of mental deficiency. It does not help, but the unearthing of some case of mental deficiency in some distant relative on one or other side of the family may arouse resentment, conscious or unconscious, between husband and wife.

Every effort should be made to give the parents some idea of the prognosis, making it clear that it is very difficult to give this with certainty until the child is older. It is nearly always possible, however, to say whether the child will learn to walk and talk, and parents are greatly encouraged if they can be told that their child will do this. Only if it is obvious that the I.Q. is well below 50 can one say that it is unlikely that the child will graduate to a special school. In the case of mongols, it is most unlikely that they will be able to attend a school for educationally sub-normal children, for their I.Q. is usually well below 50. The fact that mongols deteriorate rather than improve should always be borne in mind (Gesell, 1943). Gesell wrote: "The typical mongol is developmentally at his best during the first year of life. In the first weeks some of his behaviour may be at a relatively normal level. But he soon

displays a retardation which increases as he grows older." The average mongol sits without support at 1 year, walks without help at 2, says single words at 3, feeds himself at 4, acquires clean habits at 5, and puts words into sentences at 6.

Zwering (1954) and others have emphasized the importance of not dwelling too much on the negative side, the difficulties which will have to be faced, and the problems which will arise, but rather of drawing attention to the assets, and to the possibility of the parents leading a relatively normal life in spite of the child's backwardness.

The doctor usually has to advise the parents about the risk of another child being affected. The risk depends in large part on the nature of the defect. In most cases of primary amentia, in which no sibling or other near relative is affected, it is reasonable to say that there are about 24 chances out of 25 that a further child will be normal. The risk of abnormality is much more than it is for the normal population, but a much smaller risk than many believe. The risk is, of course, greater if another child is affected, or if there is a strong family history of the same complaint. It is very unusual indeed for a second mongol to be born to a woman, but it has happened. It is much less likely to occur if the mother is young than if she is elderly. If, on the other hand, the child has some familial degenerative disease of the nervous system, such as Schilder's disease, or other condition with a strong hereditary tendency, such as epiloia, Tay-Sachs's disease, gargoylism, or phenylpyruvic oligophrenia, then the risk is much greater. In cases in which there is a considerable risk of a repetition of the tragedy, or in which the parents are unwilling to take the risk, the question of adoption should be seriously considered. The parents may want to know whether there is any risk that their other normal children will become affected as they grow older; they can be reassured about this, unless the child has a familial degenerative disease of the nervous system.

It is always better to see the parents a second time, not only in order that one can reassess the child, but also to answer further questions which may occur to them and to discuss points which they have not fully understood.

Advice Concerning Management of the Child

It is scarcely possible for the fortunate possessor of normal children to realize how very difficult it may be to manage a mentally retarded child, particularly when he is of the destructive type.

It is very easy to ascribe much of the bad behaviour of backward children to the inherent retardation, whereas in fact it is due to parental mismanagement and lack of discipline. These children must be taught discipline like any others, but it is a slow and difficult process. Like normal children they must not be allowed to have all their own way, for overindulgence will do nothing but harm to them. They must not be overprotected, except in the sense that more than normal care has to be taken to prevent accidents. They must be taught independence. The natural reaction of the parents of children with any kind of defect, physical or mental, is to do everything for them, instead of teaching them to become independent. A good residential school for retarded children can bring about a great improvement in backward children by teaching them to feed and dress themselves and attend to their own toilet needs. The process will be slow, but it will be well worth while. The teaching of independence is the main theme of the book by Loewy (1951).

Retarded children need love and security, and much harm can be done by talking in front of them about their failings, or by making unfavourable comparisons between them and their siblings or friends. I once heard a mother say, "Mary has always been a big disappointment to us. She is not nearly as clever as her two sisters." This would inevitably cause a great deal of distress in the child's mind and make her feel unwanted, unloved, and insecure. No retarded child should ever be ridiculed for his failings. The greatest

care should be taken to avoid showing favouritism to the more intelligent siblings.

Another important aspect of management is the importance of not expecting too much of the child, by not trying to teach discipline before he is ready for it, of not demanding obedience, conformity, manners, and habits of which he is not capable. It is hard enough for parents not to expect too much of the normal 5- or 6-year-old child. It is very difficult for them not to expect too much of a 6-year-old child with a mental age of 2 or 3. The parents have to realize that the retarded child is not just lazy, and that he cannot be forced to learn. His concentration is poor, and excessive efforts to make him learn, by constant efforts to teach him, will do nothing but harm by causing discouragement and insecurity.

As for toilet training, many retarded children acquire clean habits at the normal age. Others are late and are difficult to train. Loewy (1951) has useful advice to offer and suggests that the children should be taught to retain their urine longer and longer during the day, avoiding, however, accidents.

The books by Loewy (1951) and Macdowall (1930) are helpful to parents. The leaflet by Majorie Shave (1951) and the pamphlet by the National Association for Mental Health (1950) will help parents. Parents are also helped by joining the local branch of the National Association for Parents of Backward Children (central address: 39, Queen Street, London, W.1).

Special Treatment

No drug treatment is of any value, except in the case of hypothyroidism. It has been adequately shown by many workers—for example, Loeb and Tuddenham (1950)—that glutamic acid is of no value.

Surgical treatment is of value only in special cases. McKissock (1953) performed a hemispherectomy in retarded children with cerebral palsy of hemiplegic type with fits which could not be controlled, achieving considerable improvement in their condition. Premature closure of the cranial sutures, also termed craniostenosis, is an important though rare cause of mental deficiency, and it is essential that it should be diagnosed early. It should be suspected whenever a baby or child presents with a deformity of the skull other than the flattening on one side of the head due to the baby lying on that side. The condition is cured by surgery (Faber and Towne, 1943), so that the normal growth of the brain can proceed. Even though retardation has already developed, the operation should be performed in the young child in the hope that further growth of the brain may occur. In the case of hydrocephalus something can be done to arrest further mental deterioration and to check the increase of the hydrocephalus by arachnoid-ureterostomy (Matson, 1953) and other operations. Proper treatment of subdural haematoma (Ingraham and Matson, 1944) falls more into the realm of prevention of mental retardation than treatment, but even in the presence of retardation a subdural haematoma should be properly dealt with in order to prevent further deterioration. The operation for anastomosis of the internal carotid artery to the internal jugular vein (Beck *et al.*, 1949), devised to increase the cerebral blood flow in mentally defective children, has now largely fallen into disrepute.

Fay (1932) advocated severe fluid restriction in cases of birth injury, in order to prevent further damage to the brain by reducing the intracranial pressure and to minimize the damage already done, but it is doubtful whether this treatment achieves anything. Bickel *et al.* (1954) suggested that a phenylalanine-low diet should be given to cases of phenylpyruvic oligophrenia, on the grounds that the high level of phenylalanine or its breakdown products in the blood and C.S.F. may be partly responsible for the mental retardation. Woolf *et al.* (1955) claimed good results by such treatment. It is extremely unlikely that speech therapy will help mentally retarded children to learn to speak, because they cannot speak until there is sufficient maturation

of the nervous system; but speech therapy does help when a retarded child has learnt to speak but is speaking indistinctly (dyslalia).

When no special treatment, especially surgical, is possible, as is nearly always the case, this should be made quite clear to the parents at the interview, so that they do not collect opinions from other doctors.

The Question of Institutional Care

The question of placing a child in an institution arises only with the severer cases of mental retardation, which include mongols. Once more it is most difficult for those who are fortunate enough to have normal children themselves to give a really balanced opinion on this subject, for they cannot fully appreciate the far-reaching effects a defective child has on the home and the parental reactions to them.

When the question of institutional care does arise there are many aspects to be considered. In the first place, the ultimate decision must be that of the parents. Many doctors make the mistake of being too dogmatic about what they consider to be a right or wrong course in this matter, and try to insist that the parents should have the child placed. It is the duty of the doctor to discuss all the pros and cons of the matter with the parents. Once more, he should not shelve the responsibility and leave it to the lady almoner. In a matter as important as this the doctor, with his knowledge, experience, and authority, should be the one to advise the parents.

The factors to be considered include the following:

(1) *Degree of Retardation and Type of Defectiveness.*—Although, in general, mongols are relatively easy to manage in the private house, not all are, some of them being aggressive and destructive. It is uncertain how many of the difficulties sometimes met with in these cases are due to mismanagement in the home. There is practically no alternative to institutional care for the hyperkinetic destructive idiot, who is a full-time occupation for the mother, and prevents her attending to the house and the rest of the family.

(2) *Parental Attitude.*—When it is clear that the parents are very closely attached to a defective child, such as a mongol, it is rarely wise even to broach the question of institutional care. Many good mothers feel that if they have given birth to a defective child, that child is entirely their responsibility, and they are determined to keep him at home, however bad he is. It is quite wrong to argue against such a laudable instinct, unless the child will cause trouble between husband and wife or have a bad effect on siblings (see below). If it is felt, however, that in future years the child is going to become more and more difficult for the mother to manage, then the matter will have to be discussed, because the waiting-list for institutions is such a long one—usually a matter of several years, and there is a lot to be said for putting the child's name on a list. By so doing the parents are not committing themselves to anything, though he will have to be "ascertained." If the parents are seen to be resentful and bitter, and to be rejecting the child, there is much to be said for institutional care. Such a child will lose little, whereas the much-loved mongol will lose by being separated from his parents. Kirman (1953) remarked: "The decision to place a child in an institution on account of mental defect is almost never in the child's interest, but it may be in that of another child or in that of the parents themselves."

(3) *Age of the Mother.*—An elderly mother is more likely to find it difficult to manage a defective child at home. On the other hand, an elderly mother who has no other children and on account of her age cannot have further ones—a common position in the case of mongols—may well find it much better to have even a defective affectionate child of her own than not to have a child at all.

(4) *Effect on Siblings.*—This depends partly on their age. If the siblings are younger than the defective child they may well suffer because the mother gives all her time and attention to the defective one and has no time left for the

normal children. Otherwise it is a matter of opinion how much siblings suffer from the presence of a defective child in the family. It has been said that it may help them to learn kindness to less fortunate people than them. They are likely to feel embarrassed at having an obviously defective child in the home, but it is doubtful whether the defective child should suffer on that account. There is no need to allow the presence of a severely defective child to prevent all possibility of holidays, for the child can be placed in an institution for two or three weeks in the year so that the family can get away, and it is important that they should have a break like this. Local authorities can make provision for such short-term stays; and if parents can afford to pay, it can be arranged with private institutions. It is important to consider what will happen to the defective child when the parents die. It may well be most undesirable and unfair to leave him on the hands of the siblings.

(5) *Effect on Prevention of Further Pregnancy.*—When the first child is severely defective parents are sometimes prevented from having further children, however much they would like to have them, because the mother finds her defective child a full-time occupation. There is much to be said in such a case for recommending institutional care for the child.

(6) *Availability of Institutions.*—The great difficulty of placing children is well known. It is easier when parents can afford to pay the fees of private institutions. It is doubtful, however, whether they are justified in paying very high fees for private institutions when the placement is made entirely for the sake of the child's education. The fees of a well-known institution in the United States vary from 3,600 to 6,000 dollars a year. A mongol will certainly learn something in a good private institution, but he learns so little more there than he would in a good home that it is very doubtful whether the expenditure is justifiable.

(7) *Social Circumstances.*—Overcrowding at home, and the economic and social circumstances, must be considered.

(8) *Time of Placement.*—In the United States it is a common practice to prevent the mother seeing a mongol when it is born, and it is immediately placed in an institution. Some mothers might well have a considerable feeling of guilt if that is done. There is much to be said in favour of parents keeping the child at home for a time, and if they find that, in spite of loving care and attention and every possible effort to manage him, he is unmanageable and learning nothing, they will be much less likely to have a feeling of guilt when the child is placed in an institution. It may be argued that it is more difficult emotionally for mother and child if placement is delayed. That may be partly true.

There is always a small chance of a doctor making an incorrect diagnosis, and it would be a tragedy if a baby were placed in an institution as a mongol when he is no such thing. The diagnosis is usually obvious in the newborn period, but it can be difficult.

Finally, parents who are finding it difficult to decide whether to part with a child or not, should read Pearl Buck's book (1951) *The Child Who Never Grew*.

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ASPIRIN AND ULCER

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It has been known for many years that the taking of aspirin may cause dyspepsia. The observations of Douthwaite (1938), Douthwaite and Lintott (1938), and Hurst and Lintott (1939) indicated that serious gastric haemorrhage could also occur following the use of aspirin, particularly on an empty stomach.

Douthwaite and Lintott made a detailed gastroscopic study of the reactions of the gastric mucosa to the ingestion of gastric irritants, with particular reference to aspirin. They examined 16 unselected patients shortly after the administration of 15 gr. (1 g.) of crushed aspirin in 1 oz. (28 ml.) of water and found that 13 of them developed a mucosal reaction varying from slight hyperaemia to intense congestion with submucous haemorrhages. These reactions were localized to the area immediately surrounding adherent aspirin particles and were maximal on the lesser curvature. A further five patients were gastroscopied after the administration of 15 gr. (1 g.) of calcium aspirin, and one showed a very mild reactive hyperaemia only. To ascertain if free salicylic acid, known to be present in certain brands of aspirin tablets, was responsible for the reaction, two further patients were examined by the same technique, substituting salicylic acid for aspirin. Both showed a positive reaction, but in neither case was it more intense than that which had frequently been seen to result from aspirin alone.

Hurst and Lintott gastroscopied a patient with a history of repeated haematemesis and actually saw bleeding occurring from the intensely hyperaemic areas surrounding two large portions of aspirin tablet.

Wolf and Wolff (1943) were unable to produce an aspirin reaction in the gastric mucosa of their subject Tom, and Paul (1943) was unable to find any evidence of gastric irritation from aspirin on carrying out 1,000 gastroscopies. This latter work has recently been criticized by Douthwaite (1954) on technical grounds.

The present investigation, prompted by the personal experience of one of us, was started in 1947. It was decided to investigate fully the possible harmful effects of aspirin on the stomach, particularly in patients with peptic ulcer.

Experimental

A group of 20 patients, each suffering from radiologically confirmed peptic ulceration, were selected, and in each of them three standard caffeine-citrate fractional test meal examinations were performed at intervals of a few days. The first test was done normally to serve as a control. After the removal of the fasting juice during the second and third meals, two bland calcium lactate tablets and two 5-gr. (0.32-g.) aspirin tablets were given respectively, uncrushed, with a little water. Specimens were removed at half-hourly intervals until the stomach was empty. Each specimen was tested for free and total acidity and examined for blood chemically and by naked eye. In 15 of these patients faecal occult blood tests before and after the aspirin test meal were made by the Gregersen method. The simple fractional test-meal was chosen with a full knowledge of its limitations in place of the more accurate serial test meal, since the chief interest in each specimen was the presence or absence of blood and not its chemical content.

It will be seen from Table I that aspirin tended to increase gastric acidity and that evidence of gastric irritation, as shown by the presence of blood-stained mucus in the test-meal specimens, was commonly present.

Thus in five patients (Cases 1, 3, 5, 6, and 20) blood-staining of the specimens was observed during the aspirin test meal alone. In one (Case 7) it was much greater than had been observed during the calcium lactate test meal; in two (Cases 10 and 15) it was similar; and in one (Case 14) it was less. The blood seen with the control test meals, presumably traumatic in origin, was often fresh red blood appearing in the syringe at the end of withdrawal of a specimen. The blood found in the aspirin test meals was in the form of blood-stained mucus and clots of altered blood, and once it had appeared it remained constant and sometimes increased to the end of the test.

A further point of interest was the conversion by aspirin of a previously consistently negative test for faecal occult blood to a strongly positive one in three patients (Cases 3, 5, and 6).

Pathology

In order to study directly the effect of aspirin on the stomach two uncrushed 5-gr. (0.32-g.) aspirin tablets with a little water were given to a series of patients approximately two hours prior to gastrectomy for peptic ulceration. Shortly after their removal the specimens were examined macroscopically for evidence of gastric irritation, and later microscopically. An initial 20 gastrectomy specimens were studied before these investigations to gain some knowledge of the effect of trauma, the presence of gastritis, etc., in routine gastrectomy specimens. In the first 20 patients an ordinary commercial brand of aspirin was given, in the second 20 a specially hard type of aspirin tablet was used (see below), and in the last 20 soluble calcium aspirin ("disprin") was given. Gentle gastric aspiration, but not lavage, was done by the anaesthetist just before operation, but it was felt that this could not alter significantly any evidence of gastric irritation.

Of the 20 patients who received ordinary commercial brand aspirin, 8 showed no evidence of mucosal irritation. A little mucosal oedema and congestion, a few scattered petechial haemorrhages, especially near the more massive interstitial haemorrhages of clamp marks, and some flakes of adherent mucus were present, but these were also present in a few of the initial control group and were disregarded. In the remaining 12 patients evidence of mucosal irritation was present macroscopically in the form of an acute gastritis. In 9 of these the condition was relatively mild, but