MENTAL DEFECT.

By L. S. PENROSE, M.A., M.D.Cantab.,
The Ontario Hospital, London, Ontario.

I. GENERAL.

A SCRUTINY of the papers published during the years 1940–42 and, indeed those published in 1939 and 1943, reveals an increasing trend towards clinical inquiry. Few articles are to be found in which it is assumed that mental deficiency is itself a simple concept, about which little remains to be discovered, and writers are now more cautious in suggesting the best methods of control and treatment than they were wont to be ten or twenty years ago. It is even realized that there are no fully satisfactory methods of describing what is meant by mental defect. Kuhlmann (1941) gives this definition: "Mental deficiency is a mental condition resulting from a subnormal rate of development of some or all mental functions." The specific functions which are weak or strong in a given case are determined by tests and other observations. As Doll (1941) points out, there seems to be no escape from basing the diagnosis of mental deficiency upon a variety of different criteria, the main symptom still being social incompetence. Standards of social behaviour at different ages have been incorporated by Doll (1942) in the Vineland Social Maturity Scale; he recommends its use in classifying cases for training. The problem of deciding what constitutes mental defect is acutely brought out when differences in education or culture have to be considered. Sparling (1941), for example, showed that American Indian children, whose mean Binet I.Q. was on the borderline between normality and defect, had a mean I.Q. of 108 on the Porteous Maze test. In New York State, Malzberg (1943) found that even allowing for educational differences, mental defect was commoner among negroes than among whites. Nevertheless, as shown by Zubin and Scholz (1941), recognized mental defect (judged by the number of admissions to institutions) is commoner among whites than among negroes in the Southern States. This effect is partly due to the fact that more attention is paid to problems of mental defect in predominantly white than in predominantly coloured populations. Conversely, prisons contain unduly high proportions of coloured inmates according to both North American and South African statistics (Penrose, 1943). The question as to how far mental defect is a contributory factor in cases of crime has received attention from Frankel (1939), who reported that 15 per cent. of 1,000 murderers came into this category, and that the mean mental age for 722 of such criminals was 11 years. Among 309 adolescent delinquents, Stefanescu-Goanga (1939) found 99, or 32 per cent., mentally defective; 47 per cent. of the children committed for homicide were in this category, and 34 per cent. of those committed for theft. Selling (1942) has

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investigated the degrees of intelligence in offenders under Road Traffic Acts, and finds that although uncomplicated defect is compatible with safe driving, many accidents are caused by emotionally unstable cases with subnormal intellectual capacity, and that such people should not be allowed to drive motor vehicles.

Uncertainty as to the precise nature of mental defect has tended to make plans for eugenic eradication of the disability seem increasingly utopián. Wholesale sterilization of defectives has been somewhat discredited as a panacea by virtue of its close association with the methods of dictatorship. Moreover, a review of the prospects of making any practical diminution in the frequency of hereditary factors responsible for low intelligence by sterilization programmes, planned or already in operation (Price and Halperin, 1940), leads to pessimistic conclusions. Assortative mating is seen to be capable of producing changes which are out of all proportion to the possible effects of sterilization. The public, it is held, need to be more fully informed about the absence of relationship between sterilization and taxation. Advocates of preventive eugenic measures, including euthanasia, however, have received recent support from Kennedy (1942), who holds that idiots and imbeciles are not worth the educational training bestowed upon them, and recommends euthanasia—" a soft, gentle-sounding word "—for those hopeless ones who should never have been born—" Nature's mistakes." The converse of this view is urged by Kanner (1942), who emphasizes that nearly all the harm done in the world is attributable to people who are normally intelligent but distorted in mind or grossly ignorant. Too much emphasis is placed upon the I.Q., he thinks, as a discriminating tool. Society derives great benefit and could derive much more from the training and allocation of defectives and, in any case, idiots have committed no sin in being born, and have just as much right to enjoy life as those better endowed with the I.Q. commodity.

II. Genetics.

A summary of the position of human genetics at the beginning of the period under discussion is to be found in Roberts's textbook (1940*b*); other authoritative accounts emphasizing the biochemical, biological and psychological aspects of heredity have been given by Haldane (1941), Snyder (1940) and Burlingame (1940) respectively. The advances made in animal and plant genetics in the last few decades have totally transformed the basic conceptions of heredity to which any plausible theories of inheritance of mental defect must conform. Phrases like " progressive degeneration of the stock through inbreeding " and " neuropathic taint " are becoming less frequent in the literature as it is becoming realized that accurate descriptions are necessary. Moreover, as described by Muller (1941), the gene or basic unity of heredity has been clearly shown to have physico-chemical reality, and has ceased to be merely a convenient descriptive concept. Among the most important recent basic investigations into human heredity is the study on the frequency of consanguineous marriages in England compiled by Bell (1940). It is now possible to determine with a much greater degree of confidence than formerly whether or not, in a given series of cases, consanguineous parentage occurs more frequently than in the general

population. This information often proves to be of great value in determining the exact mode of inheritance of particular conditions. It also enables studies to be made which indicate, in a general way, how far recessive factors contribute to the aetiology of mental defect. Duff and Dingee (1941) surveyed 2,082 institutional cases of mental defect of all grades and found consanguineous parentage in 60 instances, which corresponds to a rate of 2·9 per cent.—a figure in agreement with that found in previous surveys, and significantly higher than that found by Bell for patients in general hospitals, namely, 0·6 per cent. Still higher consanguinity rates were found in the sub-group of cases at the idiot level and in the clinical groups, spastic paralysis and endocrine dysfunction. The offspring of incestuous parental unions tended to be found among high-grade cases of defect, probably because such unions occur almost exclusively among persons of low intellectual capacity. More detailed investigation of the families in which such unions occur might give valuable genetical information. The suggestion has also been made that the search for consanguinity between grandparents on one or other side of the family may be useful in the study of aetiology of defects because of the possibility that relatively inbred stock may conceal defective chromosome structures (Penrose, 1940), which may manifest themselves when there is outbreeding.

The extensive work of Murphy (1939) on the relationship between maternal age and the incidence of congenital malformations has been supplemented by other studies. Dayton and Truden (1940), in a survey of 23,422 families examined by mental health clinics in Massachusetts, concluded that the maternal age of 35 to 49 years was an unfavourable factor in the child's mental development, especially at the dull normal and imbecile levels. Primogeniture is also still to be reckoned with as a possible cause of malformation of the nervous system (Penrose, 1939). The question as to how far such unfavourable maternal influences can be nutritional has been investigated by Murphy and Bowes (1939), who concluded that, though inadequacy of diet was found more frequently among mothers of cases of congenital malformation than in a control group, it was doubtful whether either poor diet or anaemia in the mother was a predisposing cause.

The discovery (Murphy, 1939) of malformed offspring born to mothers who had been subject to pelvic irradiations during pregnancy has led to much subsequent speculation on this question. Maxfield (1941) described a case of a microcephalic imbecile born to a mother aged 42 years, who had received radiation therapy for uterine haemorrhage up to a time within four months of the birth, but did not commit himself on the question of causation of the defect. Another somewhat similar case was reported by Murphy, Shirlock and Doll (1942), in which pelvic irradiation had been applied in the third month of pregnancy with a view to producing an abortion. The child, at 14½ months, was undersized, asymmetrical, and had spastic paralysis of the limbs; mental development was retarded and the head was small. The cases tend to confirm the view previously put forward by Goldstein and Murphy (1929) that irradiation during pregnancy is very harmful to the foetus.

On the question of the degree to which intelligence can be said to be inherited, the continuation of the analysis by Roberts (1940) of data, obtained in a com-

prehensive survey of 3,362 children in Bath, give rise to some important figures. The degree of likeness of sibs in respect of mental ability measured by the correlation coefficient was +0·535 for 608 pairs of sibs. Parents probably also resemble one another closely in degree of intellectual capacity, and this has the effect of raising the coefficient of likeness between sibs. If a fairly high degree of interparental resemblance is allowed for, the observed fraternal likeness is of the correct order of magnitude to support the assumption that multifactor inheritance is largely responsible for the determination of intelligence level, as measured by the Otis or Binet tests. Other recent investigations bearing on the same problem include that by Preda and Mates (1939), who found a close association between I.Q. level of children and paternal mental grade as judged by occupation. On the whole, evidence continues to increase in favour of the view that the genetic background of intellectual defect is multifactorial, when the special clinical types are excluded. Brugger (1941) still holds, however, that uncomplicated defect is not a graded character, but a genetic entity which is recessively determined. Goddard (1942), in a reply to criticism of his work on the inheritance of feeblemindedness and the Kallikak family, implies that he still believes in the essential soundness of the recessive hypothesis, and the classification of persons either as normal or defective without intermediate descriptions. Frets (1940) emphasizes the importance of heredity in mental defect, but doubts the validity of the recessive hypothesis to explain 39 extensive pedigrees. That hereditary constitution is not the only factor which determines intelligence level has been clearly demonstrated by Hobbs (1941), who reported two uniovular female twins, one normal (Binet I.Q., 105), and the other defective (Binet I.Q., 57).

An interesting discussion on the genetical relationship between mental defect and psychosis is provided by Kallman and others (1941). The writers hold that the view often found in psychiatric literature that schizophrenia and defect have a common genetic background is false. After presenting several cases of twins they conclude that these two conditions are due to specific and unrelated factors. Often the more intelligent of a pair of twins is the more psychotic. Rosanoff's conception of " unspecific inheritance " as the cause of inherent cerebral vulnerability is repudiated, and the practical importance of distinguishing between mental disease and mental defect is emphasized. Brown's records (1942) of the relatives of psychoneurotic patients did not indicate the likelihood of any genetic relationship between defect and mental disorder; only four of 492 sibs of psychoneurotic patients were rated defective. Psychopathic personality, according to Humphreys (1940), occurs in defectives just as frequently as it does in normals and in persons of superior ability; consequently, special institutional supervision should be made for psychopathic defectives.

III. Clinical Types.

With respect to some of the specific diseases which are frequently associated with mental defect, there are numerous recent investigations to report. Haldane (1941) analysed the data collected by Bell (1939), consisting of families in which there was more than one case of spastic diplegia in the sibship. A recessive type of inheritance was shown to be prevalent in rather more than

one-quarter of the cases studied. Furthermore, the tendency in these families for those affected to be all boys or all girls was so marked that Haldane postulates that the recessive factor is partially sex-linked. Dominant spastic diplegia does not appear to be partially sex-linked, however. These interpretations need to be evaluated in relation to the different clinical types of familial diplegia. An investigation of the cerebral histology of 50 cases by Stewart (1942) revealed that no invariable or specific changes are associated with diplegia. In four instances no abnormality could be found in any part of the cortico-spinal path, while in others there were gross pathological changes. The brains were mostly under the normal weight. Though prenatal origin must be postulated, there is no single common cause for cerebral diplegia, and in some cases it may be due to foetal malnutrition. Two cases showing diplegia associated with microcephaly were examined in detail histologically by Benda (1941).

Microphthalmia associated with mental defect, usually low grade, is not very uncommon. It is a condition which lends itself readily to genetic investigation because of its easy recognition. A sibship containing four affected sibs, two imbecile twin males, and a male and female of normal intelligence was reported by Kallman, Barrera and Metzger (1940). The parents were of Persian origin and they were second cousins. This recessive type of inheritance contrasts with the sex-linked type exhibited by the cases described by Roberts (1937), one of which died and was examined, post-mortem, by Whitnall and Norman (1940). The optic nerves, chiasma, tracts, lateral geniculate bodies and one corticovisual area were found to be grossly deformed and the eyeballs completely disorganized.

A sibship containing six cases of juvenile amaurotic idiocy was described by Jervis (1941), and the age of onset of symptoms in all instances was between 5 and 6 years of age. Norman and Wood (1941) described a congenital form of the disease in which the only child of unrelated parents survived but a few days after birth; the brain showed extreme microcephaly with a lipoid dyscrasia and pachygyria. Cases of the dystrophy named "gargoylism" by Ellis, Sheldon and Capon (1936) have attracted attention from many observers. Ross, Hawke and Brown (1941) gave descriptions of four cases. Jervis (1942) described a family in which the cases were somewhat atypical, and argued that the disease was closely related to amaurotic idiocy. Halperin and Curtis (1942) have shown that gargoylism is due to a single recessive gene. Osteochondrodystrophia deformans (Morquio's disease) was noted in two sibs with related parents by Farrell and others (1942), and in three patients by Einhorn, Moore, Osborn and Rowntree (1941). The family described by Shafar (1941), who relates an irregularly dominant type of brachyphalangy to Morquio's disease, is of considerable genetical interest. Here the condition was mild and evidently heterozygous, whereas in Morquio's disease it is severe and homozygous. Possibly the union of two brachyphalangeous parents would give rise to osteochondrodystrophic children. Two defective male sibs with anhidrotic ectodermal dysplasia were reported by Halperin and Curtis (1942), and analysis of cases in previous literature showed sex-linked inheritance to be probable.

A remarkable paper describing hereditary examples of lumbosacral syringo-

myelia by Van Epps and Kerr (1940) shows that altogether now 71 familial and 9 sporadic cases have been reported. The condition is only rarely associated with mental defect, but may be of value in studying the mutation-rate in man. Cases of hyperostosis frontalis interna continue to be discussed in the literature, though the origin of the disease is still obscure; Stewart (1941) shows that the new bone formation in the skull is not due to brain atrophy. Ferriman (1940) discusses the irregularly dominant type of inheritance found in oxycephaly and acrocephalosyndactyly. Both diseases, which are significantly associated with mental defect, are often sporadic in occurrence, especially acrocephalosyndactyly. Mental defect associated with cutaneous naevi may occasionally appear in more than one member of the same family. A sporadic case was recently described by Ironside and Hill (1941). Full references are to be found in the paper by Nussey and Miller (1939). Multiple angiomata affecting the retina and cerebellum (Lindau-Von-Hippel disease) in four patients, one of whom had an affected parent, were described by Craig, Wagner and Kernohan (1941). Here, again, some cases may be due to new mutation. Disagreeing with the views of some investigators who have considered myotonia atrophica to be inherited as a simple dominant character, Maas (1937) and Maas and Paterson (1943) point out that there is a large variation in degrees of severity in affected members of the same pedigree. This disease is evidently commoner than is usually supposed. The writers infer from analysis of material, which covers 94 families, that the worst affected cases appear in the latter part of the sibship, and they maintain the viewpoint, now generally discredited, but also held by Ravin and Waring (1939), that the disease tends to become earlier in onset in each succeeding generation.

Many aspects of the subject of mongolism continue to afford scope for the research worker. It is becoming more widely accepted that two unknown factors, one associated with advancing maternal age rather than with birth order, and the other a genetic influence, irregularly dominant and possibly a chromosome abnormality, are aetiologically significant. Jervis (1942) gives a very useful summary of the literature on mongolism over the last decade, but does not commit himself on the question of aetiology. Studies of twins have so far revealed that in all, certain cases where both twins are affected, they are uniovular. To the possibly exceptional cases of mongol twin pairs, which have been recorded as dizygotic and both affected, Jervis (1943) has added another pair. There, however, as in McKaye's cases (1936), a main physical difference noted was eye colour. Jervis's cases had separate foetal membranes and placenta, but were of the same blood group B. In contra-indication to this, in the pair described by Gordon and Roberts (1938), the twins were undoubtedly binovular, but quite atypical with respect to mongolism. A study by Ford and Frumkin (1942) demonstrates that finger and palm prints can be valuable aids both to diagnosis of monozygosity and understanding of the nature of the disease. Thus, the patterns found on the hands of both cases, in a pair of twins reported, were very similar to one another, and were of types found much more frequently on hands of mongolian imbeciles than on those of normal persons. These unusual patterns, first discovered by Cummins (1936), are considered to indicate a disturbance of growth at a very early stage in

development. Wolff and Rollin (1942) have attempted to relate the grosser abnormalities in the creases of mongol palms to different types of personality shown by the patients.

Further information about the nature of the growth disturbance characteristic of mongolism has been obtained by Benda (1939), who analysed the results of measuring a series of cases from 2 months to 34 years, and showed that the skull growth is retarded with especially early arrest of growth in the basal structure. Benda (1939) also holds that there is some evidence of foetal hyperthyroidism and subsequent involution of the thyroid gland in mongols. He also found an excess of eosinophilic cells in the pituitary glands. Benda (1940) reports further that the maldevelopment of the brain in mongolism is due to hormonal dysfunction. A number of biochemical studies were made by Bixby (1939, 1940, 1941, 1942), and, according to her, protein or mineral metabolism are ruled out as sources of abnormality in mongolism. Blood or serum cholesterol, plasma fibrin, serum albumin, globulin and total protein are found to be normal. Glucose tolerance curves, however, show late peaks and slow returns to the fasting level, though after giving insulin, mongoloid blood sugars fall normally. This suggests that pituitary failure is not characteristic of mongolism, since the glycotropic hormone effect is unimpaired. Himwich and Fazekas (1940) investigated the cerebral metabolism in mongolism and also in phenylketonuria, using a special method for obtaining blood samples, and they came to the conclusion that less than the normal amount of oxygen and sugar is utilized in both types of case. The writers hold that there is a real diminution in metabolism and not merely a retarded blood-flow. A survey of defectives by Hess (1943) showed that cases of mongolism, together with diplegics, epileptics and hydrocephalics frequently showed arrested descent of the testes—an abnormality closely related to loss of the cremasteric reflexes and due to deep-seated disturbance in the brain and spinal cord.

Cases of phenylketonuria, previously described as occurring in Norway, England, France and the United States, have now been reported from Switzerland (Brugger, 1942) and Canada (Penrose, 1941). Analysis by Munro (1940) of 47 sibships, containing cases of phenylketonuria, of which five showed parental consanguinity, confirmed the recessive origin of the disease. Further information about the nature of the biochemical abnormality involved has been obtained by studying the blood serum and tissues. Kondritzer (1940) studied the precipitation pattern of serum proteins, and found a small but significant increase in the globulin fraction as compared with the normal. Analysis of the amino-acid content of tissue proteins by Jervis, Block, Bolling and Kanze (1940), however, did not demonstrate any differences between normals and phenylketonurics, or "phenylpyruvic oligophrenic individuals," as these writers prefer to term them. Jervis and others (1940) also clearly demonstrated phenylalanine in the blood of such patients, confirming, by a new method, the work of Norwegian investigators. Especially high levels of blood phenylalanine were found after the patients had been fed on high protein diet. Phenylalanine was also found in the cerebrospinal fluid but no phenylpyruvic acid, nor was any phenylpyruvic acid found in the blood, as previously suggested by Jervis. These writers consider that the main abnormality is inability to break down

phenylalanine. While examining the ratio of creatine to creatinine excreted in the urines of defective patients, Pugh (1940) showed that phenylketonurics had definite creatinuria. High creatine excretion (Penrose and Pugh, 1939) was also found in cases of diplegia and congenital syphilis; it has always been regularly found in muscular dystrophy and hyperthyroidism,. Pugh (1940) further reported that Millon's reaction should be useful as a routine procedure in the detection of cases with defective ability to deal with protein metabolites.

A paper by Bender (1942) on congenital syphilis in relation to mental defect mentions that in congenital cases the blood serology is frequently negative. The diagnosis, which depends upon family history (including a study of sibs) and clinical signs as well as serology, is often difficult. In their text-book on congenital syphilis, Dennie and Pakula (1940) state that the mental retardation caused by the disease is due partly to direct injury by the spirochaete to brain cells, and partly to toxaemic conditions present at a very early developmental stage. Ironside (1940) described an interesting case of juvenile paresis in one of a pair of fraternal twins, whose mother also was syphilitic: mental retardation in the affected twins was present since birth; typical neurological signs developed and no stigmata were present.

IV. Psychology and Training.

The application of mental tests on a wide scale to predict military competence and to screen out defectives has led to the publication, during the war, of a large number of surveys bearing on this topic. There have been also some psychological investigations of independent interest, which should first be mentioned. Cutler, Little and Strauss (1940) studied the effect of benzedrine administration over long and short periods on the test scores of a group of mentally retarded children. Immediately after administration of the drug, improved scores were obtained on the Knox Cube form boards and the Porteous Mazes. No permanent benefit, however, was derived even in patients whose treatments were continued for six months. A somewhat more optimistic view is taken by Moskowitz (1941), who believes that in selected cases benzedrine raises the ability to a point where educational training can be facilitated. Kephart (1939) believes that by specific training programmes the intelligence levels of borderline and high-grade feeble-minded subjects can be raised; his results, which were based on retest data, were striking, but seem to require confirmation. The converse picture, the loss of intelligence due to disease and injury, has received attention from Hebb (1942), who found that lesions tended to produce low scores in both Binet and performance test tasks. Depression of test scores due to temporary dysfunction at an early age gives rise to higher performance test scores than Binet scores because of the learning experience implied in the Binet test. Lesions which develop late tend to produce a condition which approaches adult deterioration, with low performance and relatively high Binet and vocabulary scores. Notwithstanding, Hebb and Penfield (1940) emphasize how remarkably little effect on intelligence level is produced by removal of large quantities of cerebral tissue. In comparing the responses of defectives with brain injury, other types of defective, and normals, Werner and Strauss (1941) noted a special lack of ability to distinguish an

object from its background on the part of the brain-injured cases. The failure was not due to impairment of sensory function, but was possibly an example of the tendency of these cases to be attracted by irrelevant details. Sirkin and Lyons (1941) studied the incidence of speech abnormality in defectives, and showed that 43 per cent. of high grade cases, 74 per cent. of imbeciles and all idiots had speech defects; much benefit could be derived from the intensive treatment of such defects in the higher grade cases, particularly as an aid to rehabilitation.

An investigation among Army personnel of the relationship between skin diseases and mental capacity was made by Hodgson (1941). Here subnormal intelligence as judged by the Matrix tests was found prevalent in all groups referred for skin conditions, especially infestation (scabies and pediculosis) and venereal disease. Much work has been done on the mental abilities of cases referred by the armed forces on account of psychiatric or behaviour problems, but no great proportion of such cases have usually been found defective. Sutherland (1941) reported that the mean mental age in 45 cases of war neurosis was 12 years on the Matrix tests and 14 years on the Binet. Anderson (1940) pointed out that defectives may sometimes make more satisfactory naval ratings than their more intelligent brethren, provided that no psychopathic features are present. The general principle, however, in most services has been to exclude defectives at the outset, as likely to give unsatisfactory service. The standards have varied in different places, and there is difficulty in making comparisons because of the widespread use of the concepts of mental age and I.Q.; these terms are only correctly applicable to children and, for adults, the use of standard scores is to be preferred. Mecker, Plesset and Grava (1942) state that, although the I.Q. of 60 or over is required to meet U.S. Army induction standards, an I.Q. of 75 judged by the Binet or Kent test should be the lower limit. Esher (1941) considers all cases below 7 years and 11 months mental age, which corresponds to an I.Q. of 50 to 60, untrainable. In a sample of 100 cases specially studied, referred by the British Army, he found that 80 per cent. had relatively high mental ages, i.e. from 9 to 11 years. The drop in intelligence test scores which normally occurs with advancing age is commented upon, and Esher further recommends paying careful attention to school standards. The use of the Kent Test has been widespread because of its convenience and brevity; Rudolf (1941) examined the performances of 367 adult defectives, and showed that male responses differed somewhat from female responses using Earl's adaptation. The Kent emergency test was also first used by Atwell, Bloomberg and Wells (1941) in the U.S. Army, but later replaced by batteries including non-verbal elements. These writers state that the basic question is not one of mental age, still less of intelligence quotient, but is the determination of the subject's capacity with respect to army demands. Myers (1942) believes that intelligence tests for screening purposes should be preferably non-verbal and supplemented by "job analysis." It is evident that although the standards of intelligence required for success in the armed services and success in civilian life are not exactly the same, the experience in the use of tests during the war will have important future bearings upon the concept of mental deficiency in civilian life. It must not be inferred, moreover, that the mentally defective cannot adapt themselves to wartime strains. Benjacar (1940) reports that

institutional cases co-operate well with A.R.P. work and useful constructional tasks. They show no more liability to panic than normals and are very susceptible to efficient leadership; their reactions to war vary from blissful ignorance to moderately intelligent patriotic interest.

The use of tests for selecting personnel in institutions for defectives is advocated by Buck (1941) and Hadley (1940) to increase the efficiency of the training programme. Some of the results of training delinquent feebleminded children are described by Dwybadd (1941). Many interesting ideas for making institutional life more profitable have been voiced; for example, the teaching of languages to high-grade girls has been found useful (Angilillio, 1942). Discussions on the problems of home training by Nugent (1940) and Horsefield (1942) give outlines of the methods in use which have been found satisfactoryin this field.

In these times, when enthusiasm for all forms of shock treatment for mental cases is high, it was hardly to be expected that defectives would escape attention, and Humphreys, Vassaf, Menzel and Howe (1943) give details of the application of insulin therapy to three morons and two imbeciles on account of behaviour difficulties without, however, being able to report anything more than very slight changes, not always in a favourable direction.

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