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The Contribution of Mental Deficiency Research to Psychiatry

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In choosing the subject for this Lecture commemorating the august personality of Henry Maudsley, I did not intend to be exhaustive and I had in mind two things. I would like to discuss how the study of intellectual defects and their objective measurement may contribute to the knowledge of what Maudsley (1895) called the pathology of mind. Also, I would like to draw attention to the way in which clinical work in mental deficiency may help to explain some of the causes of mental illnesses. The first topic is psychological, the second is biological.

In surveying the works of classical psychiatrists, it is rare to find more than passing mention of the problems of mental deficiency. We look in vain for a volume by Kraepelin on the idiot. Freud had very little to say on the subject other than to insist that hysterical symptoms do not imply the underlying mental inferiority which Janet had postulated (Breuer and Freud, 1895). Looking through recent text books we find (with one or two notable exceptions), somewhere near the end or crowded into an introductory chapter, a brief account of certain clinical types of mental deficiency. Descriptive emphasis is quite properly related to complexity in the symptoms presented, and so the account of defectives tends to be relegated to the least inspired part of the book.

There is little agreement about the exact definition of mental deficiency, but it is clear that intellectual deficit is a critical feature in diagnosis. This is a quantitative character, and it differs from other features in psychiatric diagnosis which for the most part are qualities, signs and symptoms considered either to be present or absent. They vary, it is true, in their intensities, but they are not easily measured. Perhaps some of them should be, like depth of depression, strength of paranoid conviction or of obsessional compulsion. The point which I wish to stress, however, is that intellectual capacity, in its various aspects, is measurable and that it provides a peculiar dimension in the description of the mental state of a patient.

The study of this quantitative dimension of the mind has always been in a special category. In ancient times the distinction between defect and insanity was clearly recognized, as indicated by our English statute of the reign of Edward III. So marked was the clinical difference that, in the development of psychiatry during the last century and a half, problems of deficiency have always been considered separately from the rest and sometimes almost forgotten. The meeting-point has usually been in the description of insanity in children. Indeed, in very young patients it may be extraordinarily difficult to distinguish between intellectual defect and severe psychosis. Early clinicians also had the same difficulty with adult patients. The term "idiotisme", used by Pinel (1801), compared psychotic withdrawal of an extreme kind with the incapacity of a low grade defective. The distinction between *amentia*, the state of those who, in Maudsley's words, "have to face the world without the capital of a fit mental structure", and *dementia*, which characterizes a mind once competent but impaired or destroyed, still presents a real problem because "there is not an instance of mental derangement which is not essentially an example of more or less mental weakness".

The distinction between the two diagnoses

is, however, gradually becoming clearer, because in the last 30 or 40 years the aetiologies of so many specific diseases which are associated with amentia have been elucidated. Most of the genetically and environmentally determined conditions which have been recently defined lead to deficiency, not psychosis. Just at the moment when opportunities for differentiating defects of the mind from other psychiatric states are unprecedentedly favourable, the law demands that the distinction be abolished. Mental deficiency is now classified as a disorder. After 650 years of legal clarity, confusion has suddenly descended. But all is not lost. The integration of two previously isolated branches of psychiatry has been approaching for some time and mental deficiency specialists can now be legitimately called psychiatrists.

Let us consider the question of infantile psychosis for a moment because this illustrates the overlapping of disciplines very strikingly. To distinguish between primary psychosis, which Sancte de Sanctis (1906) named dementia praecocissima, and secondary psychosis grafted on an intellectually deficient mind—so-called "Pfropfschizophrenie" (Kraepelin, 1913: "Pfropfhebephrenie")—is no easy matter. If there are specific neuropathological signs, organic brain disease can be diagnosed, but when there is no clear evidence of cerebral damage child psychiatrists are free to attribute mental illness in infancy to early psychological trauma. Mental deficiency experts are usually very cautious in expressing opinions on the aetiology (Richards, 1951). However, the strictly scientific investigation of the causes of what is now called, after Kanner's (1935) recommendation, autism in children, from the genetical and psychological points of view, has, I think, been neglected. In such patients, we know far too little about biochemistry, chromosomes and family histories. This should be a field in which co-operation between specialists in remote corners of psychiatry could combine to make new discoveries. The existence of these cases illustrates the fact that we must not expect to be able to classify all patients as either defective or insane. The distinction between amentia and dementia is conceptually accurate, but in a given patient the two states can be present simultaneously. In fact, to put it crudely, we should not ask whether the patient has amentia or dementia, but find out how much of his condition is deficiency and how much is psychosis.

Not everyone would go so far as Earl (1934), who held that nearly all patients with gross intellectual defect were psychotic, but there is no doubt that classical signs can frequently be demonstrated. In mongolism, for example, waxy flexibility, echolalia and echopraxia can be found. Manneristic movements are characteristic in phenylketonuria and many other diseases. Negativism in idiocy is almost too common to be mentioned. There is also a wide prevalence of epileptic manifestations of all kinds, especially among the more severely handicapped cases. When signs of psychosis appear they are coloured by the low mental level. Thus, depressions are comparatively rare and of shorter duration than in patients with normal intellects. Complex delusional formations are never found. Pure paranoia and classical manic-depressive insanity have often been considered as hazards only for those whose intellects surpass the average. However, chronic mania with visual hallucinations has been thought to occur only in the mildly subnormal. Nevertheless, on the whole, a striking feature of the intellectually handicapped is their amiability, their freedom from emotional stress and their willingness to co-operate with others.

The general relationship between intellectual level and mental illness still remains obscure, although some hints have been provided by clinical observations and by statistical studies. The fact that a large proportion of the mildly subnormal or borderline cases of defect who drift into hospitals have psychopathic, neurotic or psychotic symptoms gives little information about the association of these characteristics in the community. The very large majority of adults with I.Q., say, between 70 and 100, are well adjusted and make good citizens. Those in hospitals or at clinics have been selected on account of social failure.

In a direct survey of the incidence of mental instability of all kinds in 30,000 men whose capacities had been measured by an Army intelligence test, Dewan (1948) found definite

evidence of greater instability in low scorers than in high scorers. This observation disagrees with the views of Binet and Simon (1916), but follows those of many accepted authorities such as Tredgold (1937) and Pollock (1944). However, since illness tends to depress scores, the results of such surveys are inconclusive. Nevertheless, the problem of separating the effects of low intelligence from those of mental illness is not, I believe, insoluble.

When the idea of mental measurement, relatively so successful in deficiency assessment, is imported into other branches of psychiatry, it meets with many difficulties and many objections. Most people are content to fall back on personality inventories of various kinds, from Bernreuter (1931) to Eysenck (1947), which are, in effect, efficiently mechanized interviews. A serious limitation in all kinds of mental testing is that the patient's co-operation is essential. It is, however, surprising how large a proportion of "disordered" patients are willing to co-operate provided that the test situation is designed to make their work as congenial as possible.

It must, at the outset, be conceded that the concept of a basic entity called intelligence, supposedly measurable by testing, is only a convenient fiction. Test scores are rather poor indices of special abilities which they are designed to explore. A test like the Binet is, nevertheless, a combination of many different special measurements. The result is comparable to an estimate of the fictitious entity, body size, by taking stature, weight, arm length, head size, and so on, and adding all the readings together. A total test score tells us something about the size of the mind but nothing about its shape, any more than a conglomeration of physical readings would tell us about the shape of the body. For psychiatric purposes we need to know, not only the total intellectual capacity of our patient, but the patern of his abilities also.

A major contribution to the psychology of mental illness was made some thirty-five years ago and is now scarcely noticed. I refer to the work of Babcock (1930). Following Yerkes (1917), she found that in mental illness there was often a very marked differential score on mental tests. Vocabulary, in particular, was retained when reasoning power had deteriorated. Myers and Gifford (1943) later confirmed that the Stanford Binet test could be broken down into items of differential significance for psychosis. Working with the Canadian Army test battery, Penrose and Myers (1941) were able to show that in mental illness the appreciation of pictorial absurdities was far below the level expected by the ability to do arithmetic and especially by the extent of the vocabulary. This differential pattern of mental capacity is found in an extreme form in some cases of organic brain disease; it is very noticeable in G.P.I., for example. Age and sex of the patient also must carefully be taken into account in evaluating the result. Nevertheless, the same type of differential is highly characteristic of schizophrenia, particularly the catatonic variety. Mentally subnormal patients, though their pooled total scores on tests may be not far below those of psychotics, for the most part show entirely different scoring patterns; often they excel in non-verbal tasks. Their results tend to be similar to those of normal children, and disabilities are fairly evenly distributed over the various specific tests. An example of differences in scores of psychotics and subnormals is shown in Table I and Fig. 1. By methods of weighted scoring of such sub-tests, the distinction between amentia and dementia is capable of succinct objective demonstration.

It seems probable, however, that something more than deterioration is involved in mental illness, and particularly in what is called schizophrenia. The same differential is found in both affected children and adults, and it is lessened in remission and recovery. As Bleuler (1912) put it, the psychotic patient lives in a dream. This condition he named "autism", to some extent agreeing, as he explained, with Jung's concept of introversion. Autism is a non-logical thought process which appears in dreams, day dreams, myths, poems and hysterical manifestations. It is part of the normal mind, but in schizophrenia it predominates.

Assuming that the Bleuler hypothesis is correct, it may be supposed that the Babcock phenomenon is in part produced by autistic thinking. Many years ago, it occurred to me

TABLE I

Mean Scores of Males on Sub-tests of Canadian Army General Examination M (see Penrose, 1945a)

Sample Number	Control (±S.D.) 1500	Psychotic 100	Subnormal 50
Sub-test:			
1. Picture completion	14·5 (±3·4)	9·6	10·1
2. Pictorial absurdities	13·1 (±3·9)	8·3	9·8
3. Figure construction	12·4 (±4·7)	7·4	4·7
4. Tool recognition	20·7 (±6·8)	15·5	11·6
5. Mechanical information..	21·2 (±7·9)	15·4	6·8
6. Arithmetic	9·7 (±3·6)	7·5	2·1
7. Vocabulary	15·5 (±7·4)	14·2	5·4
8. Verbal analogies	24·4 (±8·4)	17·7	11·1
Total score	131·5 (±38·2)	95·6	61·6

The psychotics fail at tests which require judgment (1, 2 and 3) but hold their own in routine procedures, especially retaining vocabulary. The subnormals excel relatively at detecting visual absurdities but they are very weak in arithmetic.

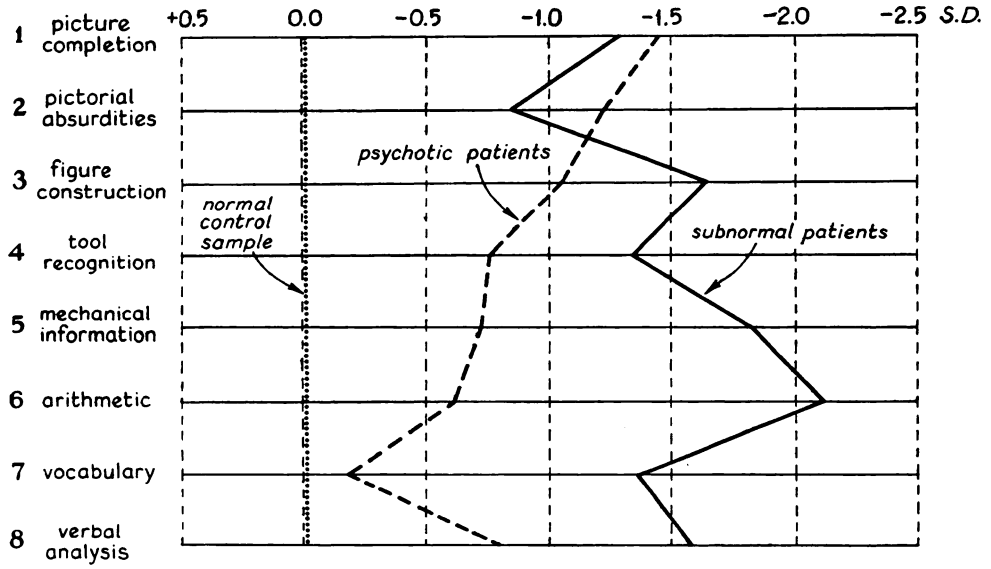

FIG. 1.—Mean sub-test scores of psychotic and subnormal male patients expressed in standard deviation units and compared with control means.

that it might be possible to design specific tests for schizophrenic thought. Uncontrolled association tests, the verbal batteries of Kent and Rosanoff (1910), like those of Jung (1918), and the visual stimuli of Rorschach (1932) and of Morgan and Murray (1935) are all difficult to score. Now, it is well known that colour-blindness can be quickly diagnosed by the Ishihara plates on which abnormals see things hidden from those with normal colour vision. Could not something be devised, nearly as automatic, to detect reality-blindness? I would like to quote here one or two experimental examples. The idea was to make use of an

intelligence test like Thurstone's (1937) classification of words, and within its framework to construct items with significant ambiguity. The normal or modal answer would differ from the answer given by the psychotic because choice would be based on a different kind of mental process. Emotional tone or clang would be decisive.

Here are some items from a series which is deliberately presented in the guise of an intelligence test. The subject is told that, in each row, there are four words whose meanings, to some extent, agree. He is asked to underline the word which does not fit in with the others. We start with something very easy:

2 aunt uncle father mother locomotive

Then ambiguity is intentionally introduced. Here is an item in which clang association leads to an illogical answer:

22 friendship fellowship steamship comradeship society

Emotional tone is used for distraction in:

29 sorrow anxiety delight dentist pain

or, probing into deeper associations,

5 ink pen water blood coffee

By such straightforward items as No. 2, the patient's verbal intelligence level is investigated. No. 22 proved quite a good trap for psychotics, some of whom are carried away by the clang association. No. 5 was even better because of the preponderance of "blood" as a choice with psychotics about six times as frequently as with normals. The apparently promising No. 29 had a strong sex difference (in that females tended to choose "delight" and males "dentist") which at first obscured its psychiatric significance.

Encouraged by preliminary success, I proceeded to try non-verbal material (see Fig. 2). The same type of result was obtained, but patients seemed less inclined to co-operate than with verbal tests. One man agreed, as a great favour, to complete one item a week! Fortunately this test had only eight items. In No. 1 some psychotics prefer to eliminate the fifth figure. In No. 3 the middle figure was picked out by intelligent schizophrenics, but not by the feeble-minded. The first figure in No. 5 was chosen

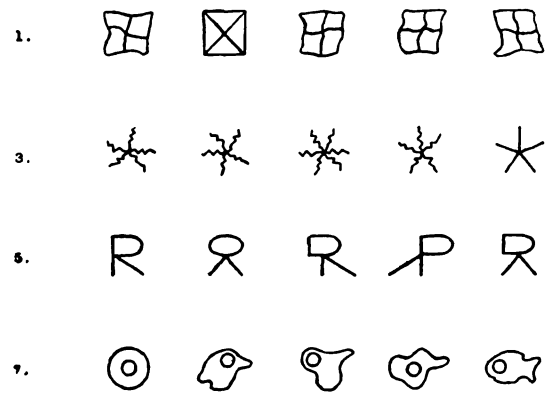

FIG. 2.—Items from an experimental non-verbal classification test. In each row the subject is required to choose one figure which does not agree with the remaining four.

preferentially by psychotics (the second is the modal choice for controls) and the last figure in No. 7, where the first figure is modal.

Again there were marked differences in male and female responses, as might have been expected from studying the results of the Terman and Miles (1936) pictorial scale for sex discrimination. In spite of the need occasionally to apply refined statistical methods in scoring (Smith, 1946), the choices made in these experimental tests provided some indication of psychotic thinking. They did not distinguish between types of illness, however; schizophrenics behaved like those with affective disorders, but more so, and deviations associated with organic insanities were even more extreme.

When tests of intelligence were first applied to the study of mental deficiency, the resulting distributions demonstrated that deficiency was not a separate disease entity but a graded deviation from the normal (Pearson and Jaederholm, 1914). Similarly, the measurement of insanity by objective methods leads to the conclusion that derangement is also a graded character. There are all degrees of dementia. Incidentally, the same applies even to the mental processes generally believed to be specific to males and females. The distributions of abilities of men and women are graded and they overlap, but this is not inconsistent with an underlying polarity based upon a biological

distinction. Similarly, though the signs and symptoms of mental illnesses and defects merge into the normal, the underlying causes of deviation from the normal can be discrete.

In medicine, tradition insists that one single cause must be found for every diseased condition. In surgery and in some parts of medicine this rule probably works well, but in psychiatry it is very difficult to apply. For example, in the field of mental defect, division into "primary" and "secondary", "exogenous" and "endogenous" types was an attempt to simplify the situation and to separate genetically from environmentally determined diseases. This has had to be resisted strongly by research workers, for although basic causes can be predominantly environmental or hereditary the end result is a mixture of many diverse influences. We have to get used to the idea of variable manifestations and at the same time continue to search for precise underlying causes with contributory influences. The existence of inborn susceptibility to specific drugs or foods is now well established. A typical example is favism caused by G-6-P-D enzyme deficiency. In an analogous way, specific sensitivity to mental or physical stress of an unusual kind may predispose to psychotic disorder.

Researches into basic origins of mental defects have been, in general, much more successful than similar investigations on the mentally ill. I think this is mainly because the clinical phenomena in mental retardation are more concrete and less capricious than in psychosis. The first necessity in both fields is the separation of conditions likely to have specific causes from the pooled sample of patients all with approximately the same diagnosis. The method has been successful in mental deficiency, because types like mongolism and true microcephaly can be distinguished morphologically, whereas, in spite of Kretschmer's (1925) efforts, this is not possible in psychosis. Nevertheless, when there are no obvious morphological peculiarities, inborn biochemical differences may still exist.

The search for biochemical changes characteristic of mental illness has not yet led to the discovery of clearly defined peculiarities. This is partly because the tendency has been not to look for inborn errors but to try to explain acute symptoms by examining physiological and pathological concomitants (Gjessing, 1932, 1936, 1939). The underlying metabolic peculiarity producing the instability might be of a very different nature indeed—an unexpectedly slight deviation from the normal, present alike in the healthy pre-psychotic stage, in acute disease and during remission.

In his survey of pedigrees of mental disease, Myerson, as early as 1923, noted that occasionally idiocy occurred in families where mental illness was present in both parents or their close relatives. This is, in fact, a rare situation but explicable in modern genetics by the suggestion that recessive traits are not perfectly recessive. Mild signs may be found in heterozygous carriers of homozygous recessive disabilities. So far, the homozygous type of Alzheimer-Pick disease is unknown and also that of Huntington's chorea. It could help greatly in understanding the pathology if the homozygotes could be found. They may be inviable, but would almost certainly show severe mental changes at an early age. In the much commoner heterozygous dominant predispositions which cause some manic-depressive psychoses, the homozygotes should be not too rare.

Looking at the problem in reverse, there is some evidence that carriers of the phenylketonuria gene may be predisposed to develop depression in late life. No investigations of this sort have been made in other recessive biochemical defects. Insanity of very early onset (e.g. child psychosis) might sometimes be the homozygous manifestation of much commoner heterozygous diseases of late onset.

The chance of finding an inborn metabolic error is much greater in homozygotes than in heterozygotes, for the action of the abnormal enzyme is then seen at an early age; and the patient's mental disability is more likely to appear as defect than as insanity. The heterozygotes who carry only one abnormal gene are, as a rule, likely to have sufficient normal enzyme to compensate until later on in life. Age of onset, indeed, is a very important datum and can be used as a marker in genetical studies. The gradual accumulation of toxic material in or between the cells of the central nervous system may be a critical factor in producing mental

breakdown. The same sort of process occurs early in life in the cerebromacular degenerations and in Wilson's disease, which, of course, are homozygous conditions.

Mental deficiency research teaches us that we must get used to the fact that quite different causes can lead to very similar clinical results. Not infrequently clinical differences become noticeable only after the causes have begun to be sorted out. As soon as phenylpyruvic acid is detected in the urine of a patient, in a previously untested group, typical clinical signs first become evident. The dilution of hair and eye colour, the mannerisms and brisk reflexes, together with the family history indicating recessive inheritance, when recognized, can be seen to be almost sufficient for diagnosis.

Another example is the recognition of the specific type of spastic athetoid condition which is the result of icterus gravis neonatorum caused by incompatibility. The patient's history and serological analysis in the mother can make very clear the meaning of the neurological signs. The same kind of diagnostic improvement has occurred in the appreciation of the effects of maternal rubella, and also in the recognition of the sequelae of infections like toxoplasmosis and cytomegalic inclusion disease.

Even more dramatic are the previously unsuspected clinical entities which can emerge from chromosomal analysis. Mongolism has been relatively easy to diagnose in about 90 per cent of cases, but there has always been a doubtful group. The problem of this borderline group is now being resolved by the cell culture techniques which enable mosaic or partial mongols to be accurately described.

Occasionally an entirely new clinical type suddenly becomes visible among undifferentiated defectives, as, for example, in the so-called "cri du chat" syndrome (Berg, Delhanty, Faunch and Ridler, 1965). More problematic is the question of what the patients are like who have some cells triploid with 69 instead of 46 chromosomes (Böök and Santesson, 1960). They are not unattractive, although syndactyly, asymmetry and physical weakness accompany mental retardation; but a specific clinical type is yet hardly recognizable (Ellis, Marshall, Normand and Penrose, 1963).

Autosomal aberrations seem to produce defect rather than insanity, but the situation is somewhat different with respect to the sex chromosomes. The effects of sex chromosomal aberrations are more noticeable in relation to alterations in character and stability than they are in relation to intellectual loss. Most noteworthy is the Klinefelter syndrome, associated with an additional X-chromosome, making the sex complement XXY instead of XY. The condition is not very rare in the general population—about one in 400 males. It is commoner in mental deficiency hospitals, about one per cent (Shapiro and Ridler, 1960), but the patients are mostly in the mildly subnormal group. They tend to be admitted on account of behaviour difficulties; and psychotic signs are not unusual. It is not yet established how many are to be found in hospitals or clinics for mental illness, but the number may be assumed not to be negligible.

It is of great interest to note here that Mott (1919) reported frequently finding testicular atrophy in patients diagnosed as psychotic. The diminution of masculinity in schizophrenic patients as a whole can be demonstrated by mental tests (Penrose 1945b,), by hormone surveys (Hoskins and Pincus, 1949) and observations on their low marriage rate and restricted fertility (MacSorley, 1964). Sex quantity in experimental animals and plants (*Drosophila*, *Lymantria* and *Melandrium*) is a graded trait influenced by genes as well as by whole chromosomes. The same is likely to be true for man. If abnormal sex quantity tends to produce mental instability, clearly a wide and neglected field for psychiatric research is ripe for exploration.

What, then, is the chief contribution which experience in mental deficiency research can offer to psychiatry? I believe it is the suspicion that the great clinical groups of traditional psychiatry, which were amplified by Kraepelin, and which still determine psychiatric teaching to a large degree, do not represent the true aetiological grouping. It was Maudsley himself who "shunned the use of many learned names—of Greek, Latin and Graeco-Latin derivations—which have been invented in appalling numbers often to denote simple things, and sometimes,

it may be feared, with the effect of confounding apprehension of them". The classification of mental defect into the grades of idiocy, imbecility and feeble-mindedness has been less misleading.

If researches have clarified diagnosis, what good are all these results to the patient? This inevitable question must be answered. The study of chromosomes, like all genetical investigations, in the first place only leads to a better understanding of individual cases. A patient who is found to have some peculiarity in this respect is likely to receive increased interest and attention, and these by themselves can be therapeutically useful. Otherwise, neither treatment nor prognosis is changed. There is, however, definite value from the point of view of prevention if a parent or sib can be warned that he or she carries an abnormal gene, a balanced chromosomal translocation or a mosaicism which can prove dangerous for the mental development of the offspring.

Considerable therapeutic advances have been made when the cause of defect is found to be a homozygous recessive enzyme abnormality. The idea that mental deficiency is always a consequence of an inferior cerebral cortex was very popular until 1934, when Fölling described the first cases of phenylketonuria. I can remember Dr Douglas Turner's great delight at the news that one of his patients had something wrong with his liver rather than with his brain. It was thirty years ago that we tried a low phenylalanine diet on this patient, consisting mostly of gelatine, sugar, dripping and vitamins. The patient, a low grade idiot aged 17 years, did not improve much mentally, and he deteriorated physically. However, I am glad to say that he survived—and I believe is still alive and well. Things are very different now that cases can be diagnosed soon after birth and treatment of an effective nature started immediately.

Other successes are recorded in the treatment of galactosaemia, homocystinuria, probably the "maple syrup" disease and several others. Easiest of all should be the treatment of sex-linked diabetes insipidus simply by prescribing excess fluids. All these diseases have early onset, but recessive conditions with later onset, like Wilson's Disease, can also react favourably to rationally planned treatments to neutralize the effects of inborn errors. These advances in no way diminish the value of concurrent improvement in educational methods.

Treatment of mental illness has also a great number of facets. Psychotherapeutic and pharmacological treatments are now in vogue—they seem to be displacing the more violent shock procedures of induced convulsions and particularly of psychosurgery. A legacy of psychiatry to mental deficiency, the treatment of idiocy by leucotomy, is best forgotten. As Engler (1948) discreetly observed, "the results have been rather unsatisfactory".

Insanities resulting from injury or infection are dealt with more efficiently as time goes on. However, rational basic rather than symptomatic treatment of the so-called endogenous diseases is not yet feasible. Though clearly these are largely genetically determined, study of the genes and the chromosomes so far gives us little help. The contribution of mental deficiency research to this great problem would seem to be, by analogy, to indicate that there are included here, under clinically similar behaviour patterns, a variety of quite different genetical peculiarities. Some dispositions may be caused by chemical errors which act slowly by piling up toxic metabolites over a long period of years. If we knew what the errors were, prophylactic diets might be prescribed. Other susceptibilities may be caused by chromosomal aberrations not discerned with our present techniques.

I think I have said enough to emphasize the point of view that the various branches of psychiatry all have potential value for each other. Provided that we do not neglect or despise the data derived from one another's disciplines, there are advances to be made in the diagnosis, prevention and treatment of apparently most intractable mental diseases.

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