COLLECTED PAPERS

BY THE

MEDICAL STAFF

OF THE

NEW JERSEY STATE HOSPITAL AT TRENTON

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VOL. II.

1912–1913

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SOME PROBLEMS IN THE STUDY OF HEREDITY IN MENTAL DISEASES.* †

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Without doubt, from the standpoint of prophylaxis, at least, the most important aspect of psychiatry has always been the factor of heredity. It has been known for years, in a general way, that heredity has played an important rôle in the etiology of the psychoses, but we have been very far from having any definite knowledge of the subject. We have, until quite recently, been satisfied with vague opinions regarding "insanity in the family," of those mentally affected, without due regard to the nature of the malady in other members of the family.

The records of the patients in insane hospitals, even in those hospitals pretending to do modern scientific work, are woefully incomplete, inadequate and often inaccurate. In the older methods of examination the statistical data regarding heredity, as well as etiology in general, was based upon the statements of the committing physician. No attempt was made to inquire systematically into these questions, and every one knows how unreliable such statements received must have been. Even with modern methods, but very little progress was shown, as the source of information from the families of patients was also limited. The most conscientious work of the assistant physicians would fail to bring out all the important factors of heredity. Often the husband or wife of a patient was the only one who came to visit the patient, and usually they knew very little of the family history of each other. Hence, statistics made up from such sources, while much more

* Elaboration of a paper read by title at the sixty-seventh annual meeting of the American Medico-Psychological Association, Denver, Colo., June 19-22, 1911.

† This paper forms contribution number one of the Worcester State Hospital (Mass.), Series of 1912, offered in compliment to Dr. Hosea Mason Quinby on the event of his retirement from the Superintendency after 20 years of service.

accurate and complete than statistics collected as above, at the same time the best obtainable by this method were open to serious and just criticism.

We have been content until quite recently with the loose methods of investigating this important subject, and the fact that insanity occurred in the family in such and such a proportion of our patients was considered enough for our present knowledge.

It is usual to see in statistics of insane hospitals a summary of the number of cases in which heredity was a factor, such heredity being merely insanity in the family. We have been aware that certain forms of insanity exhibited more heredity than others. But we have had no accurate knowledge of the nature and type of mental disease in ancestors and immediate families of our patients. We must all plead guilty to our lack of interest in this subject, and we must acknowledge our indebtedness to one outside of our work who has succeeded in arousing our interest and stimulating our endeavors in this field.

Prof. Charles B. Davenport, of the Eugenics Section of the American Breeders' Association, and in charge of the Eugenics Record Office at Cold Spring Harbor, has been the one to stimulate our interest in this field.

In this office, in which Prof. H. H. Laughlin has been associated with Prof. Davenport, the modern ideas of the study of heredity had its birth. The office has been in existence less than three years, but has already accomplished a great deal, and through this agency many state hospitals, as well as other institutions for the care of epileptics and the feebleminded, have been supplied with competent and well-trained field workers, who are now engaged in vigorously attacking the problem in a score of centers. The workers have been prepared by means of a summer school, which lasts six weeks. During this time systematic instruction is given in collecting data in the field, tabulating such data, and in making heredity charts. The workers are usually chosen from among college graduates, and those having some experience in social service work, preferably women.

It is with some pride that we note that New Jersey has been the pioneer in this special line of work. The first systematic study of the question of heredity in the feebleminded was the work of Prof. Johnstone and Dr. Goddard, of the Training School, at Vineland,

and Dr. Weeks, of the Epileptic Village, at Skillman, has contributed the first important contribution based upon systematic field work on the question of heredity in this class of patients.

Dr. Everett Flood, of the Epileptic Colony, at Monson, Mass., has also done valuable work in this field. These can be said to have been pioneers in this work.

The King's Park State Hospital, in the State of New York, was the first state hospital for the insane to employ field workers in connection with their study of heredity, but such work has not been continued. We believe the New Jersey State Hospital at Trenton was the first to organize a permanent department of "field work," with a special appropriation to carry on the work, in connection with systematic "after care" work, and has now two trained field workers. By combining the heredity work with the "after-care" work, we feel that a direct benefit comes to the patient, and that we can show results of a practical immediate character as well as results which might be unjustly termed of theoretical importance only.

Literature.

Although Gregor Mendel published his discoveries of laws regarding heredity, which now bear his name, and are so well known, as early as 1866, it was not until 1900, when his work was rediscovered by De Vries and others, that the importance of his work in this field was recognized. It was mainly through the work of Bateson and other Englishmen that his work received the recognition in England and this country. Since 1900 many investigators have been occupied with the problems of heredity, especially in plant and animal life. To some extent the problems of human heredity have been investigated, and the science of eugenics has now grown to considerable proportions and found an important place in our studies of the human family.

But the relation between the science of eugenics and psychiatry has but recently been established, and the literature upon the subject is as yet extremely meagre, and, with one or two exceptions, is confined exclusively to this country. In only one other country can it be said that systematic study of heredity in insanity by means of field work has been established, and that is Germany. It is all the more surprising that this country, where so much

valuable research in psychiatry had been carried on, had neglected this phase of the subject, especially as Mendel was a German, and his work should have been familiar.

And up to the present only one investigator has been occupied in this field in Germany, Dr. E. Rüdin, Oberartz of the Royal Psychiatric Klinic in Munich, under the direction of Prof. Kraepelin. His work is decidedly a most important contribution to the subject. His work is of such importance to the study of heredity in mental diseases that it is worth our while to review it more in detail, which will be done later. Since 1909, Rüdin has been Oberartz of the Psychiatric Klinic at Munich, and during this period he has personally collected material for the study of the complex problems of this question. (This valuable contribution of his is published in the Zeitschrift fur Gesammte Neurologie and Psychiatrie, Seventh Vol., 5th Part, Nov. 18, 1911.)

What makes the work of Rüdin so remarkable and noteworthy is the fact that he has done field work personally, and at his own expense. He gives up his position at the clinic for several months each year and goes into the field to collect this data. He has copies of the rosters of all the Bavarian institutions, and is able to get fairly good records of the patients who were in these institutions, although patients were committed a great many years ago. The fact that he has access to accurate records is a great advantage, especially in case of preceding generations.

From the majority of records of the hospitals in this country, even up to within a short time ago, it would be impossible to make any sort of a diagnosis of the patients admitted to them. In some cases one can get more accurate information upon which to base a diagnosis from the description of the cases who never come into the hospital than in patients who are admitted.

Another fact of importance connected with Rüdin's work is that he does his field work himself, and, consequently, is able to get better descriptions, to observe, and to " size up " the members of the family and classify them accordingly. Without financial assistance, and against difficulties which would seem insurmountable to us, he has achieved some wonderful results. He has accurate family histories in a great number of cases, representing various types of mental disease. Although he has so much material at his disposal, he feels that he has not done enough work to

justify any general statements or formulation of laws regarding the heredity factors of insanity.

The Eugenics Record Office has issued bulletins from time to time, based upon the work done by their field workers in various institutions.

The first bulletin is on "The Heredity of Feeblemindedness," by H. H. Goddard, Ph. D., of the Training School, at Vineland, and is an extremely interesting and valuable contribution to the subject. Fifteen charts are shown. While it is a preliminary report of the work being done at Vineland, it is well worth reading by those interested.

Bulletin No. II, from the same office, is a compilation by Dr. Davenport, Prof. H. H. Laughlin, Dr. Weeks, Prof. Johnstone and Dr. Goddard, on the "Study of Human Heredity," and methods of collecting, charting and analyzing data. It has been the object of those interested in this subject to have a uniform method of charting which could be adopted by all institutions, so that the various institutions would understand, without difficulty, the work that is being done in other institutions.

Bulletin III is by Gertrude L. Cannon and A. J. Rosanoff, M. D., of the King's Park State Hospital, and is a preliminary report of the work done at the King's Park State Hospital, New York. This work is principally an outline of the methods used, and a brief description of the Mendelian laws.

The fourth bulletin, by Drs. Weeks and Davenport, is the result of systematic field work at the Epileptic Village, and is a valuable contribution to the inheritance of epilepsy. The authors give the following conclusions as the result of their work:

1. The method of field-study of epileptic families combined with the modern biological methods of analysis of hereditary data constitute a vastly improved means of inquiry into inheritance of epilepsy.

2. Epilepsy and feeblemindedness show a great similarity of behavior in heredity, supporting the hypothesis that such is due to the absence of a protoplasmic factor, that determines complete nervous development.

3. When both parents are either epileptic or feebleminded, all their offspring are so likewise.

4. The conditions, named migraine, chorea, paralysis, and extreme nervousness, behave as though due to a simplex condition of the protoplastic factor that conditions complete nervous development; *i. e.,* persons belong-

ing to these classes usually carry some wholly defective germ cells. Such persons may be called "tainted."

5. When such a tainted individual is mated to a defective, about one-half of the offspring are defective.

6. When a simplex normal is mated with a defective, about half the offspring are normal; the others defective or neurotic.

7. When both parents are simplex in nervous development and "tainted," about one-quarter (actually 30 per cent) are defective.

8. The proportion of tainted offspring is not noticeably higher when both parents show the same nervous defect.

9. Normal parents that have epileptic offspring usually show gross nervous defect in their close relatives.

10. While we recognize that "epilepsy" is a complex, yet there is a classical type numerically so preponderant that, in the mass, "epilepsy" acts like a unit defect.

11. Our data point to a poisoning in slight degree of germ cells by alcohol, but the evidence is hardly crucial.

12. There is evidence that in epileptic strains the proportions of epileptic children in the latest complete generation is double that of the preceding; but there is no evidence that in these epileptic strains the average number of children in a fraternity is greater than in the population at large. Provided matings continue as at present, and no additional restraint is imposed, the proportion of epileptics in New Jersey would double every thirty years.

13. The most effective mode of preventing the increase of epileptics that society would probably countenance is the segregation during the reproductive period of all epileptics.

Dr. Rosanoff and Florence I. Orr, B. S., are the authors of Bulletin No. V, entitled, "The Study of Heredity in Insanity in the Light of the Mendelian Theory." The conclusions of the authors are based upon the investigation of about 73 cases, and the heredity charts in these cases are reproduced. This represents 206 different matings, total, 1097 offspring. A table is given, showing the proportion of normal and neuropathic offspring, which resulted from various types of matings, compared to the theoretical expectations according to the Mendelian theory. The authors have considered that the neuropathic constitution, in reality, consists of a series of units, which are distinct, at least from the standpoint of clinical definition, though at the same time in manner related to each other. One is forced to emphasize here the fact that it is necessary to keep an open mind in regard to these problems. In other words, that we must investigate the facts of the heredity of psychoses as they exist, and not to be too

prejudiced towards the Mendelian laws. Following are the conclusions given by the above authors:

1. The neuropathic constitution is transmitted from generation to generation in the manner of a trait, which is, in the Mendelian sense, recessive to the normal condition. Rules of theoretical expectation are accordingly as follows:

a. Both parents being neuropathic, all children will be neuropathic.

b. One parent being normal, but with the neuropathic taint from one grandparent, and the other parent being neuropathic, half the children will be neuropathic and half will be normal, but capable of transmitting the neuropathic make-up to the progeny.

c. One parent being normal and of pure normal ancestry and the other parent being neuropathic, all the children will be normal, but capable of transmitting the neuropathic make-up to their progeny.

d. Both parents being normal, but each with the neuropathic taint from one grandparent, one-fourth of the children will be normal and not capable of transmitting the neuropathic make-up to their progeny, one-half will be normal, but capable of transmitting the neuropathic make-up, and the remaining one-fourth will be neuropathic.

e. Both parents being normal, one of pure normal ancestry and the other with the neuropathic taint from one grandparent, all the children will be normal, half of them will be capable, and half not capable of transmitting the neuropathic make-up to their progeny.

f. Both parents being normal and of pure normal ancestry, all the children will be normal and not capable of transmitting the neuropathic make-up to their progeny.

2. Various clinical neuropathic manifestations bear to one another the relationship of traits of various degrees of recessiveness; in a most marked way recoverable psychoses, though recessive as compared with the normal condition, are dominant over epilepsy and allied disorders.

3. Various other clinical neuropathic manifestations bear to one another the relationship of neuropathic equivalents; that is to say, they are conditions of the same degree of recessiveness, varying in their clinical manifestations with the personality of the subject, environmental conditions, etc.

4. All the neuropathic children, which result from a mating of the fourth type (both parents normal, but each with the neuropathic taint from one grandparent), can have, theoretically, only equivalent defects and not defects of different degrees of recessiveness.

5. Among the actual results from such matings the following have been met with:

a. Brothers and sisters suffering from clinically identical neuropathic manifestations.

b. Psychosis in one subject and peculiar or abnormal disposition, but no actual psychosis in brothers and sisters.

c. Psychosis in one subject and isolated, but clinically related symptoms in brothers or sisters; we find with particular frequency dementia præcox—fainting spells or convulsions in childhood.

d. Psychoses clinically not known to be related; senile deterioration—peculiar hysteriform psychoses.

6. Neuropathic conditions show only in about one-fourth of the cases indications for commitment to sanitariums or public institutions. The total incidence or neuropathic conditions may be roughly estimated as affecting between 1.5 and 2 per cent of the general population.

7. It is further estimated that about 30 per cent of the general population, without being actually neuropathic, carry the neuropathic taint from their ancestors and are capable under certain conditions of transmitting the neuropathic make-up to their progeny.

The methods of assuming facts, when they do not exist, is open to just criticism, and the fact that it was necessary for the authors to assume the fact of the simplex inheritance in places where information was not available, is open to criticism. It is true that these cases where the simplex inheritance is dissimilar has been treated separately and distinct from the other material. It is self-evident that the question of human inheritance presents many difficult problems, and there will be many cases that apparently do not follow any given law. The lack of matings and the absence of children in many families, or the usual "two-children" families, offers serious difficulties in making out definite laws regarding inheritance.

To some extent feeblemindedness can be considered a unit, but here one is forced to recognize the fact that imbecility itself is far from being a unit, and may be caused by entirely different factors. The grades of feeblemindedness, as outlined by Goddard, viz.: highest types, morons, next grade, feeblemindedness, and then imbecility and idiocy, are practical for a clinical classification, but from the standpoint of etiology these types may not be so distinct, and there certainly can be a distinct difference between the members of any one class. No one will deny that in feeblemindedness we have the purest form of inheritance, and that feebleminded parents, as found by Dr. Goddard, will certainly produce feebleminded children. At the same time, a no small number of cases of feeblemindedness may be the result of external causes, and not altogether due to heredity features. Infectious diseases in child-

hood, especially scarlet fever, and perhaps other fevers, cause feeblemindedness, both directly and indirectly, by arresting development through the direct action upon the cerebral tissues, and indirectly produce arrested development through deafness and other disturbance of the sensory organs.

When discussing the cause of epilepsy, one has to be especially careful not to consider this disease as a unit, a fact recognized by Weeks and Davenport. It is far better to consider the group as "the epilepsies" rather than to consider the disease as a unit. So it is even necessary, in discussing the inheritance of these simple forms of disease, to be guarded in not considering them entirely as units. At the same time, we recognize the fact that inheritance plays an important part in the production of feeblemindedness and "the epilepsies" and that the laws concerning same will be much simpler than the laws regarding other psychoses. In fact, we are forced to consider that these two diseases are subdivisions of insanity as such, and for this reason one readily sees the error in considering insanity as a unit or uniform disease.

F. W. Mott (Brain, Part 2-3, Vol. XXXIV, Nov., 1911), "Inborn Factors of Nervous and Mental Diseases," discusses the question of inheritance in general, and in particular the hereditary features of insanity. He discusses at length the laws of Galton, and is inclined to agree with his views, which laws, as we know, are opposed to those of Mendel. He also discusses the Mendelian principals at length, and gives a detailed explanation of these laws. He firmly believes in the law of sex limitation in certain types of diseases, such as color blindness and hæmophilia, and, in the field of nervous diseases, pseudohypertrophic paralysis. This form of inheritance is not only discontinued or interrupted for successive generations, but the disease is limited to one sex, although it is to be noted that the disease is transmitted by the sex in which it does not appear. Thus, it is the males who are affected in hereditary sex-limited diseases, and it is the females who transmit the disease. Mott also gives considerable space to "Nature and Nurture," and shows, conclusively, how in a great many conditions the environment and experience due to environment may have a very important bearing on inheritance. Also that a neurotic tem-

perament may be manifested in many different ways by conduct and behavior, and this neurotic temperament may be the first evidence of any degeneration in the stock. It is well that he has emphasized this important fact, for these characteristics must be looked for in collecting data for pedigrees of the insane, as it has been found that they are of as much importance as the pure mental disease in the ancestors. It is true, as he states, "that unsound stock may have successful men in the eyes of the world, but these may really form the first step in the process of degeneration, for avarice and normal guile, which made them pillars of society, may come out in the next generation as gross criminality or insanity. Mott is of the opinion that inborn factors partly, if not wholly, can account for the appearance of insanity in the stock."

Of considerable interest is the discussion of the Law of Anticipation, which was defined by Nettleship as "a manifestation of the morbid change at an earlier period of life, either in members of each succeeding generation as a whole, or as successively born children of one parentage." He gives examples of the truth of such a law. His observation, "that there is a general tendency for insanity not to proceed beyond three generations, either because of regression to the normal, or from the fact that the stock dies out," is important. But his explanation, that not infrequently the stock dies out through the inborn tendency of insanity manifesting itself in the form of congenital types, such as imbecility, or in the insanity of adolescence, is open to criticism, for it is not always true that children of insane parents are defectives. Types of insanity, of course, have to be considered, but even children of dementia præcox are frequently entirely normal, and the brothers and sisters of such patients may also be normal, although many of them appear to be peculiar. Mott gives some interesting statistical data regarding familial character of insanity, but here one is forced to call attention to the uselessness of such statistics, where insanity is considered as a unit. We call attention elsewhere to the necessity of considering various types separately, at least until we can establish some sort of relation between the various forms, especially as regards hereditary features. He gives statistics of 2246 individuals where one or more members of the family were inmates of an institution. This is all right as far as it goes, but

in our field work we frequently find evidences of mental disease in members of the family where these individuals have never been inmates of an institution. Especially is this true in early generations, where a very small percentage of those who were insane have been committed to a hospital. So that, to be accurate, one must consider these cases as well as members of the family who have been in institutions. It is also important to note that statistics based on hospital admissions alone would not truthfully represent the facts.

Mott gives the conclusions of Dr. Edward Shuster, who made the study of inheritance of the same types of insanity in 1910. They are as follows:

1. A periodically insane son or daughter is more likely to be associated with a periodically insane mother or father than if one if differently affected. In the case of two offspring in the insane there is even a greater tendency for a periodically insane male or female to be associated with a periodically insane brother or sister than with one differently affected.

2. In case of delusional insanity, the tendency for the affection to run in families is very strongly marked, and the correlation between members of the same co-fraternity is more strongly marked than between parents and offspring.

3. In the instance of primary dementia of adolescence, there is a strong correlation between members of the same co-fraternity. There is also a decided tendency indicated for the brothers and sisters of imbeciles to be also imbeciles.

4. There is no indication of general paralysis running in families. This is not surprising, as it is now recognized to be an acquired disease due to syphilitic infection. Both conclusions would seem to be justified from our knowledge at the present time. The chief criticism of Mott's work is that he has not gone carefully enough into the question, as he has practically taken only the cases which have been admitted to the institutions as a basis for his statistics, thus leaving out of consideration a large number of important individuals.

It is conclusively demonstrated in this country that only by the help of well-trained field workers can we expect to collect valuable data regarding this complex question, and, secondly, in considering the inheritance of insanity, he is inclined to treat the disease as a unit, rather than to closely differentiate the various types. We must first establish the same rules between the various types of psychoses before we can justly consider them similar or dissimilar as regards the form of inheritance. Mott's article, in all prob-

ability, represents the best work that has been done by the English in this field.

Mendelian Laws in Relation to Insanity.

In recent literature we find explanations of the principles of heredity as formulated by Mendel, but the clearest exposition of the subject is found in the work of Rüdin.

We are acquainted with the facts that the total inheritance of an individual from his parents is certain human characteristics, each of which is inherited independently of all the rest, and the inheritance of any such character is believed to be dependent on the presence in the germ plasm of a substance called the determiner.

With reference to any given character the condition of an individual may be dominant or recessive: the character is dominant when, depending upon the presence of its determiner in the germ plasm, it is plainly manifest. It is recessive when, owing to the lack of its determiner in the germ plasm, it is not present in the individual under consideration.

The symbols D and R in the following table represent the dominant and recessive conditions. In other words, D stands for the presence of the determiner of the trait, and R stands for its absence.

The following formula for six types of matings and their resulting offspring:

Type 1. $(D+D)X(D+D)=4DD.$
Type 2. $(D+D)X(D+R)=2DD+2DR.$
Type 3. $(D+D)X(R+R)=4DR.$
Type 4. $(D+R)X(D+R)=DD+2DR+RR.$
Type 5. $(D+R)X(R+R)=2DR+2RR.$
Type 6. $(R+R)X(R+R)=4RR.$

We speak of the inheritance of a character from both parents as duplex inheritance, designated by DD.

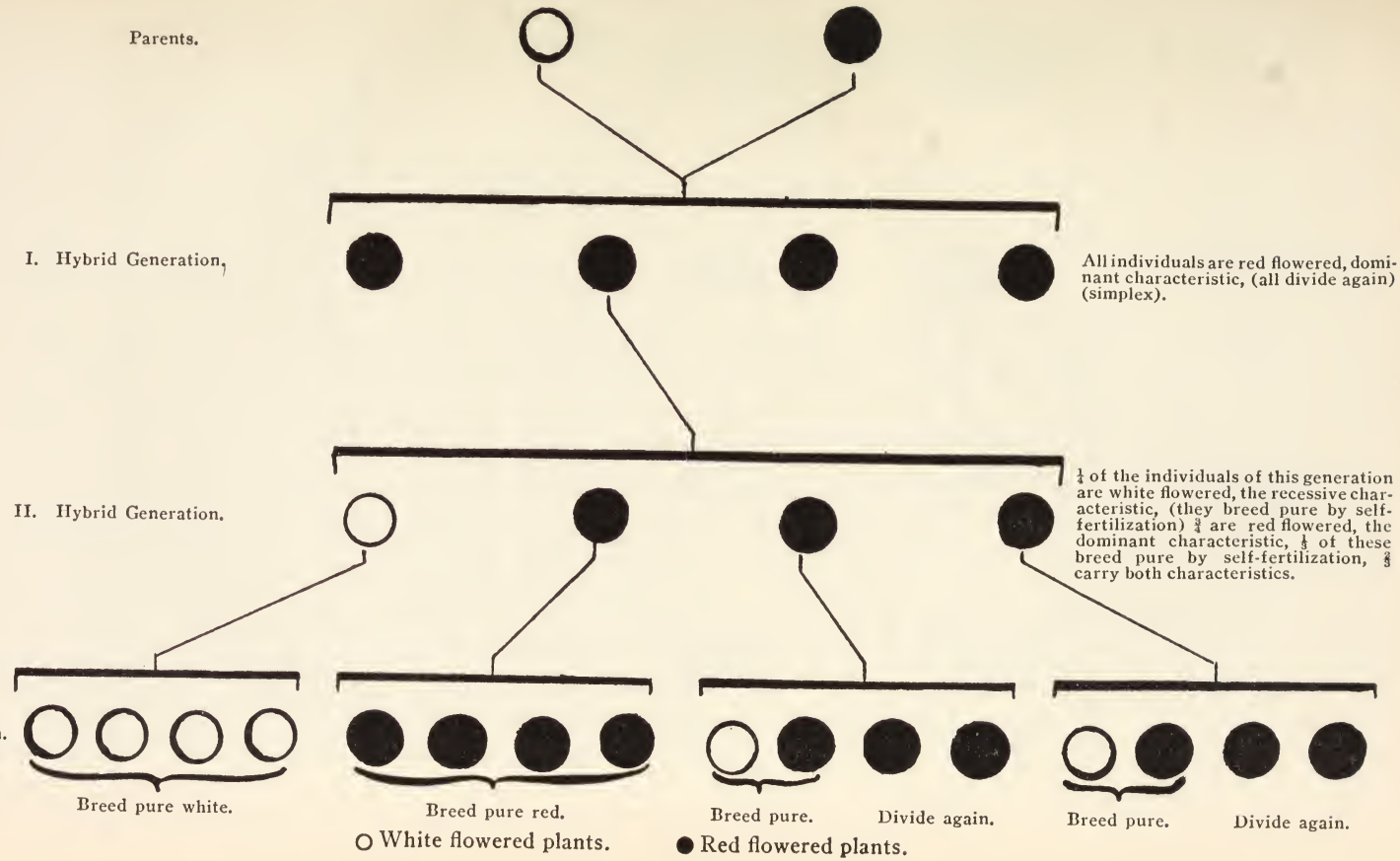
The case of inheritance of a character from one parent is spoken of as simplex inheritance, designated by the symbol DR.

In Fig. I, taken from Rüdin, we have exhibited diagrammatically the principles of the Mendelian prevalence and the rules of

FIG. I.

MENDELIAN PREVALENCE AND RULES OF DIVISION.

(Inheritance of the pea flower.)

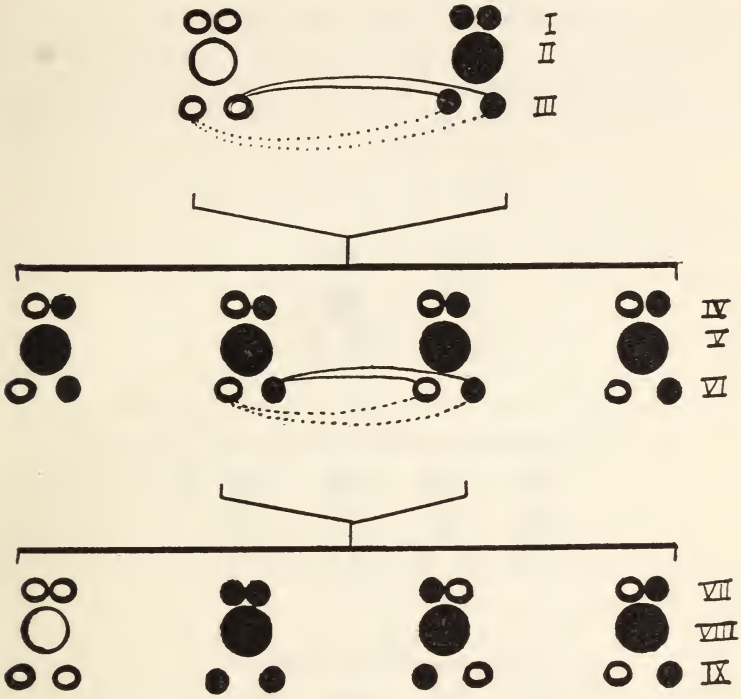


variance in the original experiment of the crossing of red and white peas. In the first hybrid generation we see that all individuals are red-flowered peas, although this generation contains also white-flowered characteristics, which are not manifest because the red is dominant. In the second hybrid generation we see the Mendelian proportions of one white to three red, in other words, one recessive and three dominant. In the third hybrid generation the white sweet pea breeds only white, one-third of the red peas breed pure red, while two-thirds of these red peas breed white and red in the proportion of one to three. This is the simplest explanation of this law. We see by this chart that the pure Mendel inheritance is not a mixed product composed of inherited characteristics, and these characteristics do not exist in a permanent combination, but always occur as separate unchanged characters in succeeding generations.

In Fig. II (after Rüdin), we see a diagrammatic explanation of this law of the regular appearance of the dominant and recessive characteristics. We see in Row I the germ plasm from which the parents develop. On the right the two black dots represent the homozygous gameten, because the germ plasm or the fertilized germ cells, gameten, has only one characteristic, that is, either for white or red. The germ plasm of these separate individuals, *i. e.*, the father and mother, are also considered as pure homozygous. By mating this pair, and following the lines in Row III, we will see there is an equal number of red and white gameten. In Row IV, which produces offspring shown as red in Row V, but, although this generation is red entirely, the gameten are not homozygous but heterozygous, that is, made up of both red and white characteristics, but because the red is dominant, the white characteristic does not appear. Now, by mating two of this generation, and noting the lines indicating combinations in Row VI, we see the reason for the proportions in the second hybrid generation. We have one white individual made up of homozygous gameten, one red homozygous and two heterozygous. Because of the red being dominant, the white characters do not show. Then in the germ plasm of these individuals we have the white producing pure white, and the red producing pure red, and the heterozygots producing both white and red.

FIG. II. (After Rüdin.)

The Experimental Crossing of the Red and White Flowered Pea.
(The "anlage" combination of the Gametes and zygots.)



○○ = Germ plasm which carries the tendency (anlage) for white flowers only.

●● = Germ plasm which carries the tendency (anlage) for red flowers only.

○● = Germ plasm which carries tendency for red as well as white flowers.

Row I. Germ plasm from which the parents are derived, they carry the one character. White or red, they are pure homozygous.

Row II. The two parents which result from the above germ plasms.

Row III. Pure, homozygous germ plasm which is produced by the parents.

Row IV. Result of the union of the unit characters from the parental germ plasm, heterozygous are combined germ plasms (simplex).

Row V. Here the tendency for red in the germ is dominant over the tendency for white. The flowers appear red, the first hybrid generation.

Row VI. The two characters, which are present in the heterozygous germ plasm and are derived from the parents, divide again, so that half of the germ cells bear the tendency (anlage) for white, the other half the tendency for red.

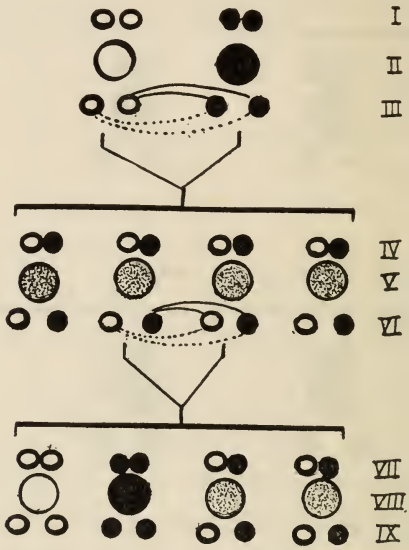
Row VII. The fourth combination of characters which occurs through the crossing of two hybrids of the first hybrid generation, two heterozygous and two homozygous.

Row VIII. Where the tendency for red is dominant over white, red flowered and white flowered individuals result from the four named germ plasms in the proportion of one to three.

Row IX. The above-named combination of germ plasms from the first hybrid generation will again divide. The one white individual, because it is produced from germ plasm with a tendency (anlage) for white only, produces germ cells with a tendency for white; one red individual with germ plasm with tendency for red only and two red individuals with both characters.

There is still another form of inheritance, which is shown in Fig. III, which is known as incomplete inheritance of the dominant characters or intermediate inheritance. This figure illustrates the mating of the red and white " wunder blume " or maribilis jalappa. By mating the red and white plant of this species we get in the

FIG. III. (After Rüdin.)
Scheme of Mendelian Inheritance of the Intermediate Type.



∞ Germ plasm, which carries the tendency (anlage) for white flowers only.
●● Germ plasm, which carries the tendency (anlage) for red flowers only.
○● Germ plasm, which carries the tendency for red flowers as well as for white flowers.
○ White flowered individual.
● Red flowered individual.
⊕ Pink flowered individual.

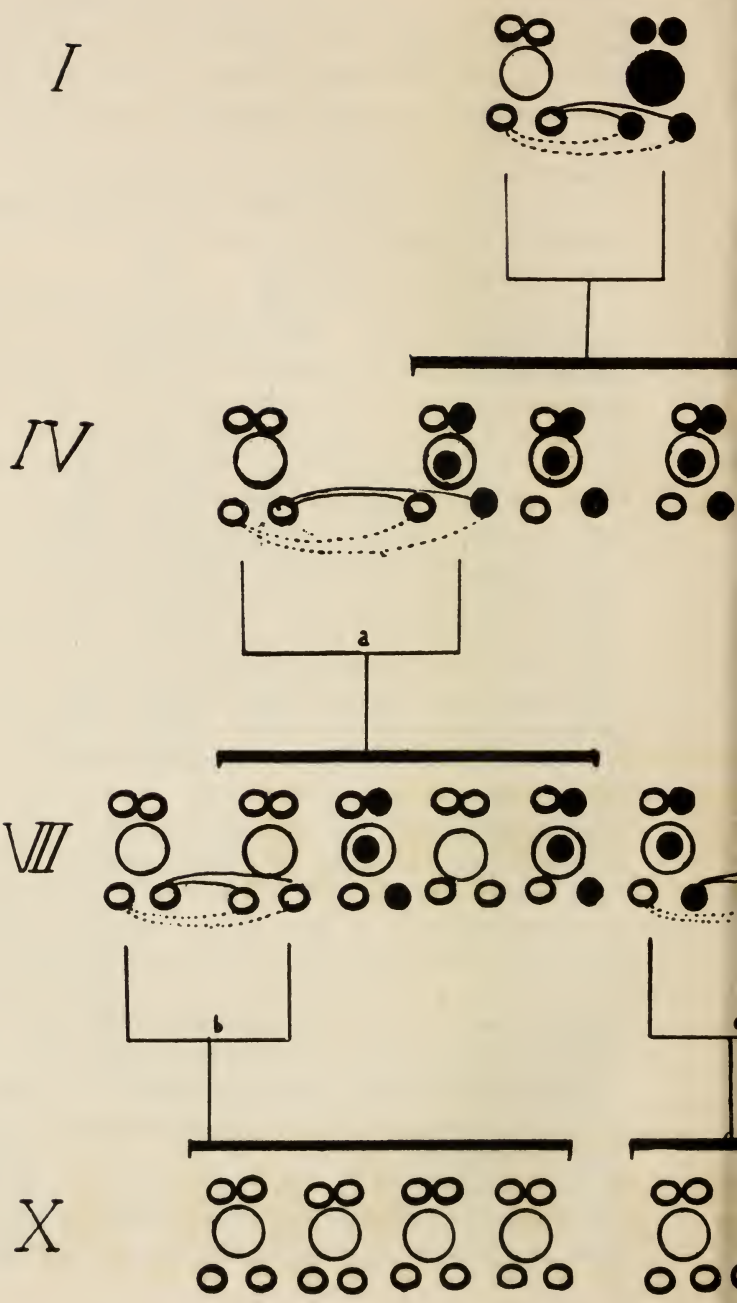
first not pure red or pure white or the prevalence of dominant characters, but the progeny shows a resulting mixture of red and white indicated by pink, but, as shown in Row IV, this progeny-colored pink is made up of heterozygous gameten, capable of producing both red and white. This is shown in the succeeding third generation, where we have one pure white individual, one pure red individual and two pink individuals. The white and red

are homozygous, while the two pink plants are heterozygous, and this explains the fact that while the law of prevalence is an important factor of Mendelian heredity, at the same time the most important fact is that the antagonistic characteristic factors do not produce any permanent combination, but always occur as a separate unchanged character in succeeding generations.

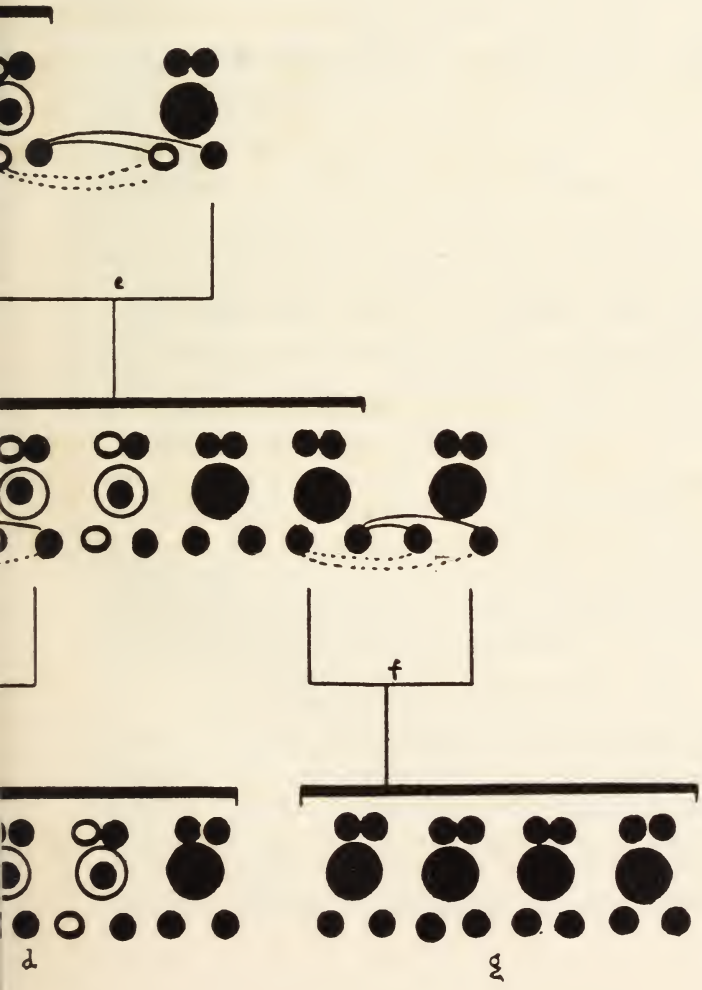
So far we have spoken of the inheritance of dominant characteristics, and this rule holds good when this dominant characteristic is an abnormality.

We are now to consider the laws of inheritance, where the abnormality is recessive. This is shown in Fig. IV. The recessive characteristics here are indicated by the black, hence are not to be confused with the other figures where the black indicates the dominant characters. Here there are six possibilities of a simple recessive Mendelian inheritance. In Row IV we see the result of the mating of a dominant homozygot with recessive heterozygot (Row I). All the individuals in Row IV are normal, notwithstanding the fact that they inherited a defect from one parent. This is illustrated by the white circle with the black dot. The abnormality is not apparent when, for example, the dominant normal mates with the dominant normal (in Row IV-a the dominant homozygot mates with a dominant heterozygot; Row VII-b, the dominant heterozygot mates with a dominant homozygot). There is a very important exception when the abnormality again comes to the surface, as seen in Row VII-c, where two dominant heterozygots mates with homozygots, in other words, two normal individuals with a duplex inheritance (normal and abnormal). Then we have one quarter of the progeny recessive homozygot, therefore abnormal in Row X-d. However, when recessive homozygot (abnormals) mates with dominant heterozygot (Row IV-e), then we have one-half of the progeny abnormal, and, finally, when two recessive abnormals, homozygots mate (Row VII-f) all the progeny will be abnormal (Row 10-g). When one of the parents is sick (abnormal), mated to a normal or normal mated to normal individuals, the progeny is normal children. They may also have abnormal children. Normal individuals from affected families will have normal progeny, the same as normal individuals from families without any inherited defect. We have these two phenomena, matings between normal individuals from

FIG. IV. (
"ANLAGEN" COMBINATION



○ Dominant characteristic, homozygot. Normal.
◉ Dominant characteristic, heterozygot. Normal but with latent tendency toward abnormal.
● Recessive characteristic, homozygot. Abnormal.
○ Dominant anlage.
• Recessive anlage.



families without hereditary taint will show all normal progeny. Where both parents are abnormal all the progeny will be abnormal.

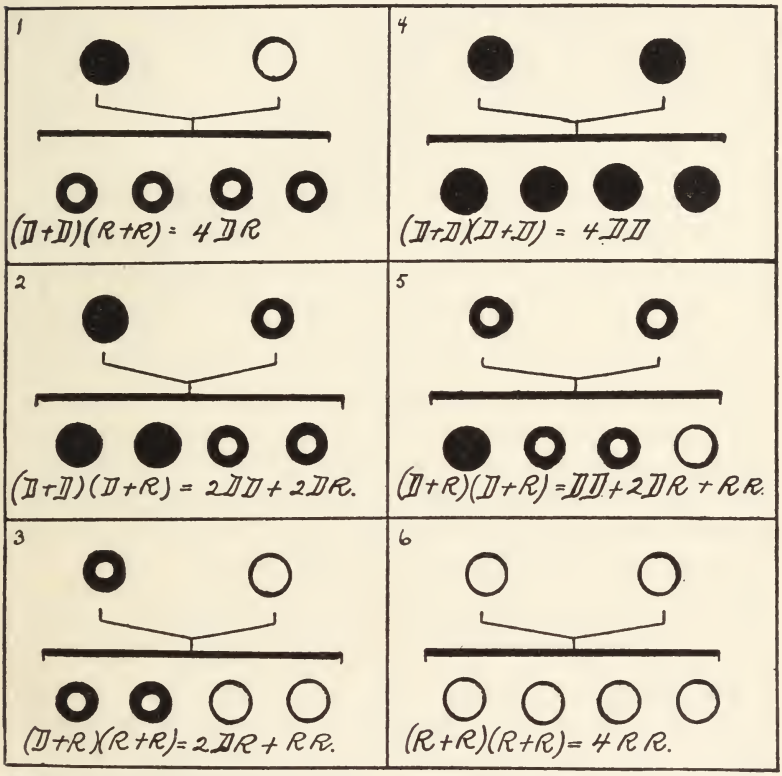
If we analyze the proportions in this chart, we will see that the apparent irregularity, with so-called exceptions, have a definite basis and follow definite rules. When two normal individuals have abnormal children, the proportion is one to four or two to eight, while parents who are normal and well, at the same time their germ plasm is not pure. Instead, the germ plasm is heterozygous. One or the other of the parents comes from ancestors, one of whom at a certain time was abnormal. When normal individuals have normal children, at least one parent has pure germ plasm, *i. e.,* homozygous, whose ancestors have never had any similar disease, or only through indirect ancestors, the grandparents or great-grandparents, or the collaterals, but never from the parents. In cases where an abnormal has normal progeny, the other parent must necessarily be homozygot (normal), and there is no tendency to abnormal. When an abnormal individual mates with a normal individual and have abnormal children, we find that the proportions of the abnormal to the normal will be as one to one, in other words, half of the children will be abnormal. In this case, however, the normal parent is heterozygot, normal.

Following Rüdin further in the discussion of this question on inheritance are given diagrammatic in Figs. V and VI, illustrating respectively the inheritance proportions, where abnormalities are dominant and where abnormalities are recessive. These diagrams give a better explanation of the proportions that are usually expressed by the formulas *DD, DR* and *RR,* which are given above.

Rüdin states that individuals with identical types of ancestors, which to all appearance, have identical characters, notwithstanding this, can have the combinations of gameten that are entirely different. Thus, in Fig. VI, in the square marked 5 and 6, the children have identical types of ancestors, but the children are not the same, and seven of the eight children who externally appear to be the same have entirely different combinations of germ plasms. The opposite also holds true that individuals can have identical gameten combinations and at the same time have quite different types of ancestors. In Fig. VI, square 2 and 3, we find quite a difference in inheritance where abnormality is dominant or recessive. In families where the abnormality is dominant there will

be a great many abnormal progeny, as shown in Fig. V, with 17 abnormal individuals, while in Fig. VI there are only seven abnormal individuals in the progeny, and particularly in each and every generation and in each and every family where one parent

FIG. V. (After Rüdin.)
Inheritance Proportions in Dominant Abnormal.



● Abnormal, dominant homozygot. ◎ Abnormal, dominant heterozygot.
○ Normal, recessive, homozygot.

is abnormal; on the other hand, in families with the recessive Mendelian abnormality in much fewer individuals, and not in each and every generation and each and every family. This latter fact is of extreme importance, for an abnormality can skip two or even three or more generations.

Contrary to the rule in the dominant type of inheritance of an abnormality, we see in families, where abnormalities are of the recessive type, that external normal individuals are not always at the same time produced by normal germ plasm, for the external appearance of normality may cause errors to be made, and this rule is important when the question of marriage of relatives, cousins, etc. While they may appear absolutely normal, at the same time the tendency to abnormal characteristics may be present in the germ plasm of each individual, consequently, the children of such mating are much more liable to be defective through the inheritance of these latent abnormalities in the parents. Each and every normal individual from a family with a dominant abnormality is also of normal germ plasm. In normal individuals, from a family of recessive abnormalities, the same can also occur, but it is not absolutely necessary Fig. VI, square 3). Through different associations of matings and pairing the resulting proportions through the experiments in animal and plant life, the homozygous and heterozygous elements have been produced and accurately settled. Each and every abnormal individual of a family with a recessive abnormality is consequently of abnormal germ plasm. On the other hand, abnormal individuals have a dominant abnormality when one of the parents is normal, possesses also an anlage to normal. In cases of dominant abnormalities there is the danger only for the progeny of the abnormals, but for the normal progeny there is no further danger.

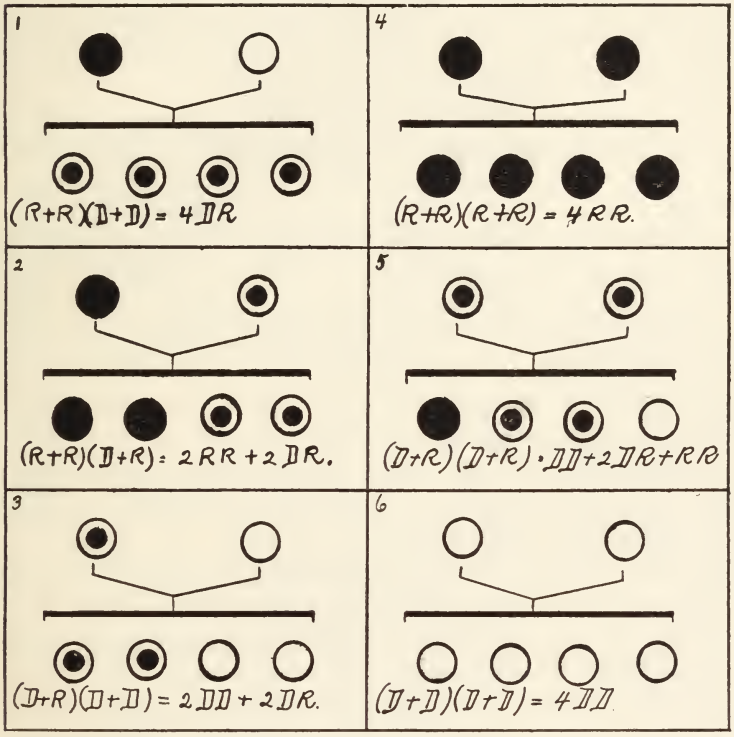
Rüdin gives us two main points from the standpoint of prophylaxis. 1. For families with inheritance of dominant abnormality, matings should be made only by normal members of a new stock, or with members of the same family. 2. In families with recessive abnormalities, matings can be made by all the individuals, but for the best results, certainly only the normal individuals, not only with new stock, but with the members of a normal new stock. These points that we have just spoken of can be said to be closely allied to the " mutation theory " of H. De Vries.

Rüdin discusses at length the Galton theory of inheritance of characteristics from ancestors, which views are utilized by Pearson, Darbishire and others, known as the Biometric School. Galton's law, concerning inheritance, is that a given person inherits one-half from the father and mother, from the grandparents one-

quarter, etc. That is, the given person is related one-half to the father and mother, and one-quarter to the grandparents, and one-sixteenth to the great grandparents. We have the following formula of the proportions of inheritance from the ancestors:

$$\tfrac{1}{2}+\tfrac{1}{4}+\tfrac{1}{8}+1/16+\ldots=1.$$

FIG. VI. (After Rüdin.)
Inheritance Proportion in Recessive Abnormal.



● Abnormal, recessive homozygot. ⊙ Normal, heterozygot.
○ Normal, dominant homozygot.

Rüdin opposes this law of Galton, which has had such an effect upon the English school. He maintains that a given person does not inherit half from the father and mother, or one-quarter from the grandparents, but inherits distinct characteristics of the fathers or mothers or grandparents. In other words, from the Mendelian point of view, a person may inherit characteristics which make

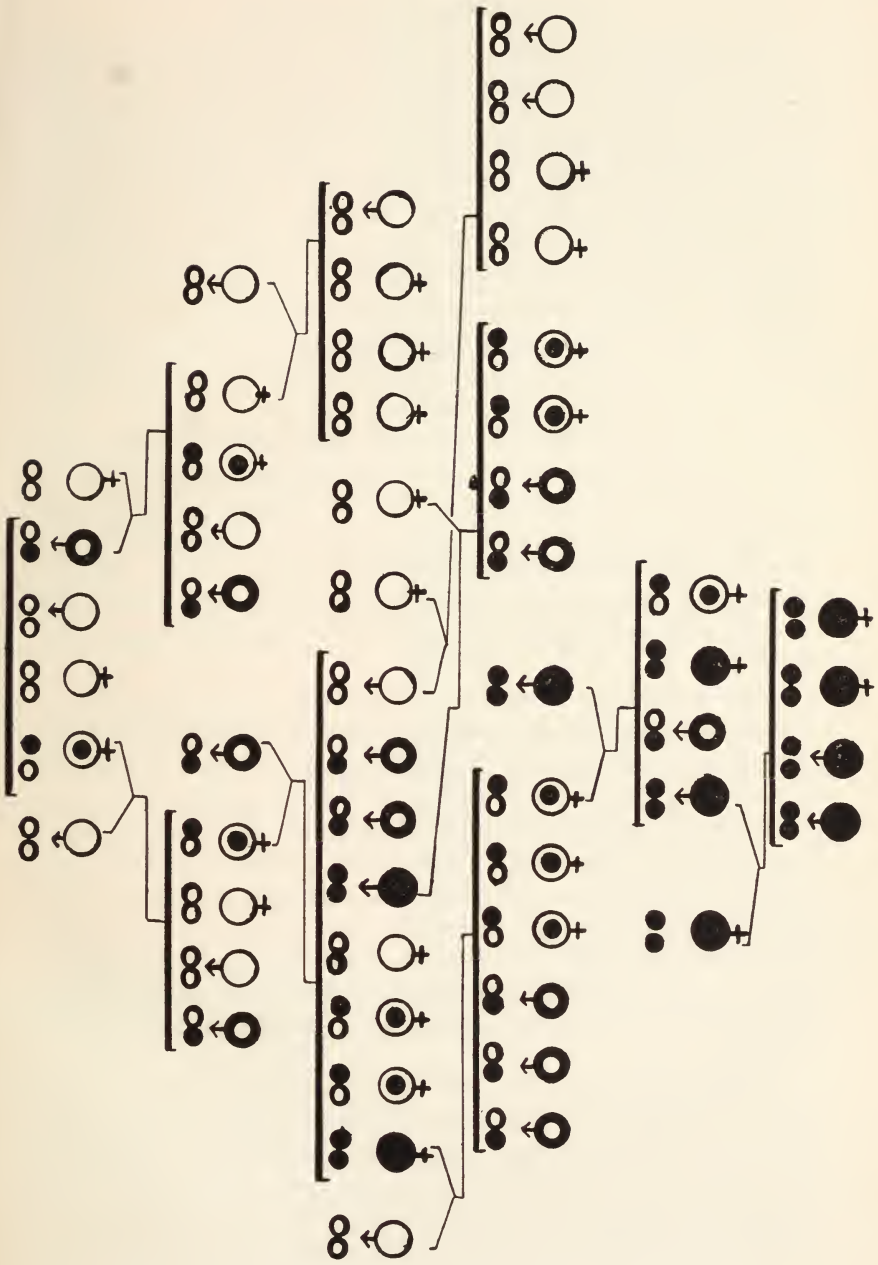
him directly related to any one of his ancestors, or there may be no points of resemblance. Rüdin further discusses the complication which arises when one and the same abnormality is dominant for male members of a family, on the other hand, recessive for female members of the same family.

In Fig. VII is shown the well-known chart of Bateson, exhibiting the inheritance of color blindness. This peculiar form of inheritance occurs in other diseases, especially in hæmophilia. This form of inheritance was looked on as an exception to the Mendelian rule. By such charts analyzed closely, it is seen that they follow a fixed rule.

Rüdin gives, further, a large number of characteristics and traits in both the botanical and zoological fields. But of interest to us here are the characteristics often dominant in human individuals.

The Hapsburg lower lip in the male sex is almost exclusively dominant over the normal lips.

Single births dominant over twins and triplets.
Huntingdon's chorea dominant over normal.
Familial periodic paralysis over normal.
Porokeratosis over normal.
Night blindness over normal.
Progressive muscular atrophy over normal.
Hæmeturia over normal.
Ptosis familias over normal.
Normal dominant over amaurotic idiocy.
Many familial muscular diseases over normal.
Familial psoriasis and cerebella hereditary ataxia over normal.
Many forms of ichthosis palmaris over normal.
Normal dominates over albuminurea.
Brachydactyle or hyperphlange over normal.
A great many skin diseases dominant over normal.
Diabetes incipidis ever normal.
Some forms of glaucoma over normal.
Normal over retinis pigmentosa.
Color blindness in males over normal.
Hæmophilia in males over normal.
Pseudo-hypertrophic muscular paralysis (Gower's) over normal.



THE INHERITANCE OF COLOR BLINDNESS.

The Problems in Psychiatry.

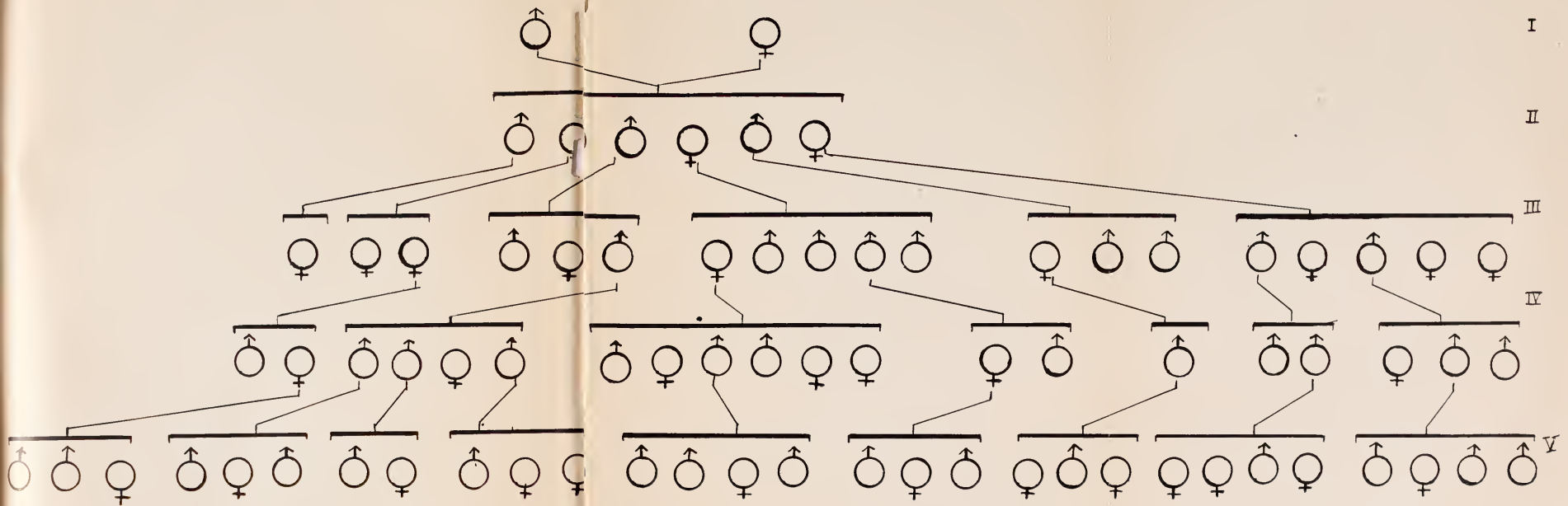
The problems relating to psychiatry will, therefore, be much more complex from our standpoint, for in a great many types external factors play a much more important rôle in the production of psychoses than the hereditary features. Especially is this true of the types which are due to the direct effect of toxins and poisons. Such is the case, for instance, in general paralysis, where we know to a certainty that the disease is not dependent upon heredity, but upon the effects of previous infection of syphilis. Delirious conditions, exhaustion psychoses, and, to some extent, manic-depressive insanity, might be produced by external factors which largely outweigh any effect of defective inheritance.

Among the problems met with by psychiatrists will be: 1st, study of direct inheritance of certain types. 2d, the effects upon the succeeding generations of the neuropathic constitutions as expressed by eccentricities, peculiarities, alcoholism, etc., in the parents. 3d, to what extent these factors are responsible for the occurrence of various types of psychoses in the progeny. 4th, the effect of the occurrence of certain types of psychoses in the ancestors upon the production of either similar or dissimilar types in the progeny.

Part of the work of the State Hospital, at Trenton, has been to collect as much accurate data as possible of the families and ancestors of patients coming under our care. It is a tremendous task to analyze this data, and often it is impossible to come to conclusions regarding the types of psychoses occurring in ancestors where we have only the description handed down from generation to generation. One has to be extremely careful not to be biased and make the cases fit certain laws. None the less, the value of such work is apparent when we consider the marked difference in the number of individuals about whom we now obtain information to the work previously done without the assistance of field workers.

While field workers are not trained psychiatrists, at the same time, we endeavor to have them attend the staff meetings whenever they are not in the field, to get some general idea of symptoms and diagnoses. In this way they become familiar with some of the important symptoms to be looked for, the age of the onset and whether individuals recover from their disease or become chronic,

FIG. VIII. (After Rüdin.)
Inheritance Chart.



and other points of importance. It is well that they do not know too much about diagnoses, as they might easily fall into the error of making certain diseases fit certain diagnoses, whereas, now their principal part is to collect *facts,* and then these facts are worked over and analyzed. When insufficient data is present no diagnosis is made and the case is considered as unclassified. We have been able to secure valuable data in a large number of cases, and will continue to make systematic investigations in all psychoses before giving any definite conclusions.

Rüdin states further that in the realm of psychiatry not enough work has been done to say with certainty whether these diseases follow the Mendelian laws or not, and he thinks it will be quite a long time before this question can be fully settled. He disagrees with Herron, that mental diseases do not follow any Mendelian laws, that they are neither dominant or recessive. He is inclined to think that the inheritance of dementia præcox follows recessive type, but the question cannot be settled at the present time. He is also inclined to think that manic-depressive insanity is a dominant type of inheritance. Although he thinks he has evidence which would substantiate these views, at the same time he is not willing to say that these diseases represent two separate types of inheritance. Many psychopathic states and defective conditions, he thinks, follow the same rule as in manic-depressive insanity. From the material at hand, he is inclined to think that intermediate types of inheritance, where there is a mixture in the offspring of the two distinct psychopathic conditions in the parents, is very seldom found. On the other hand, we see a certain similarity between psychopathic diseases in both parents and their offspring.

Rüdin enters into a lengthy discussion of the question of correlation, but for the present we will not get into a discussion of this principle.

He goes into a detailed discussion of the ways and means of systematic investigation of family histories of patients, and in Fig. VIII is reproduced his heredity chart. As will be seen, this method of charting differs somewhat from the method shown in Plate I. It is a question whether this is a simpler method of charting heredity than the former one. Instead of representing the matings by a line between two parents, Rüdin's method, as can be seen by his chart, is, to my mind, a better one, principally

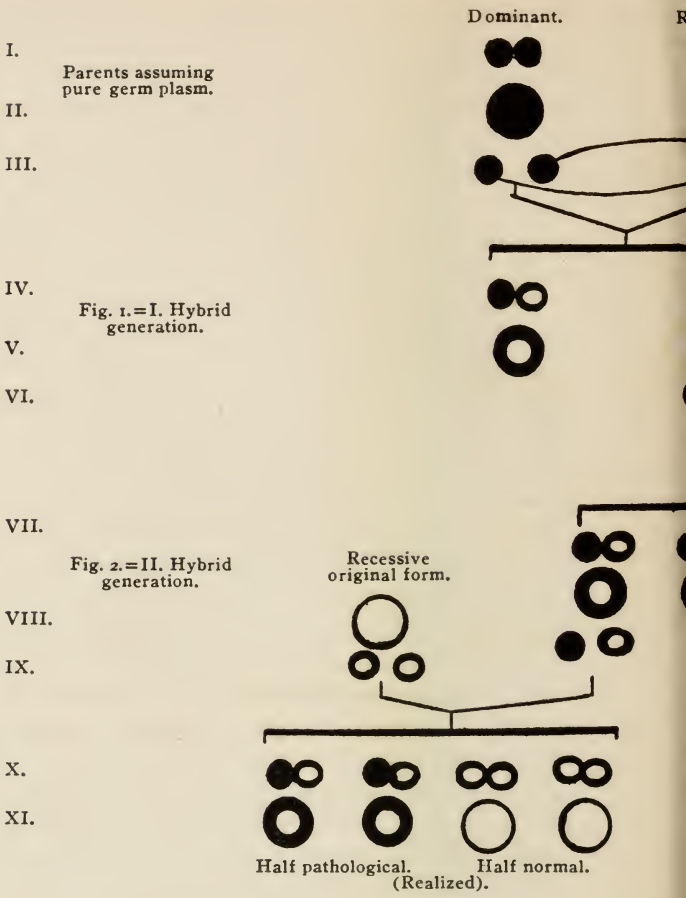
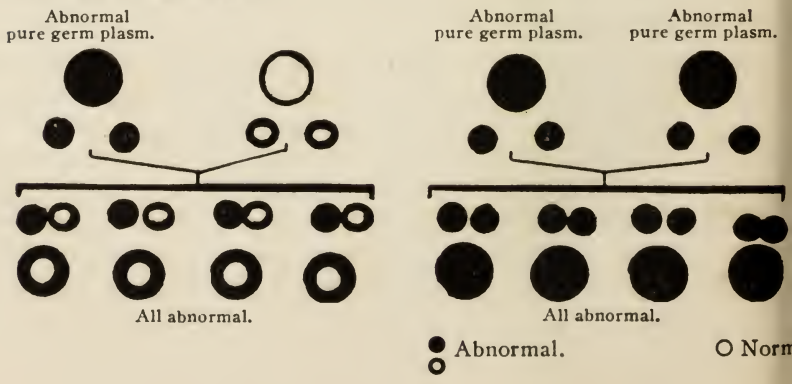


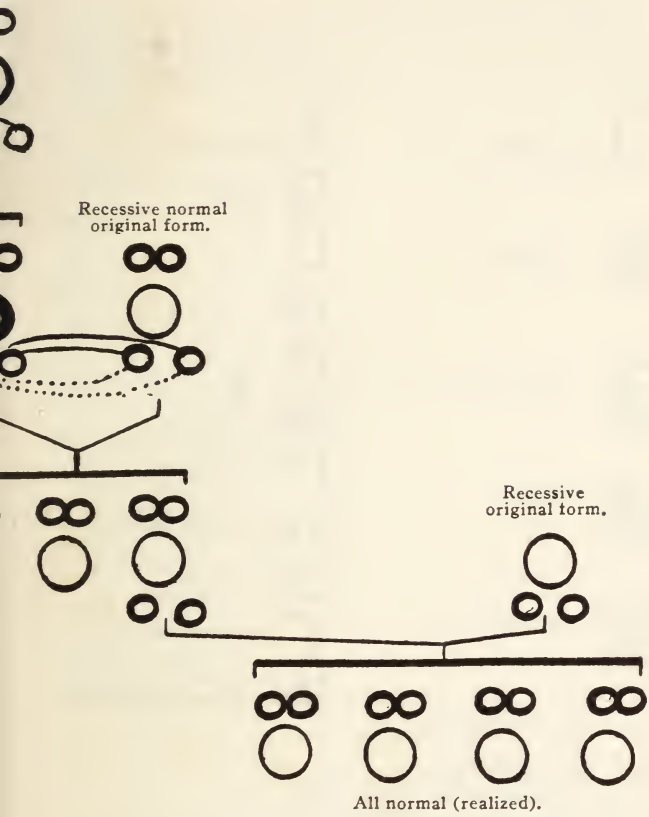
Fig. 1.=I. Hybrid generation.

Fig. 2.=II. Hybrid generation.

The following combinations are not realized in the reality, because the s
fails too in the actuality.



ve.



osition of the pure germ plasm of the abnormal and the mated abnormals

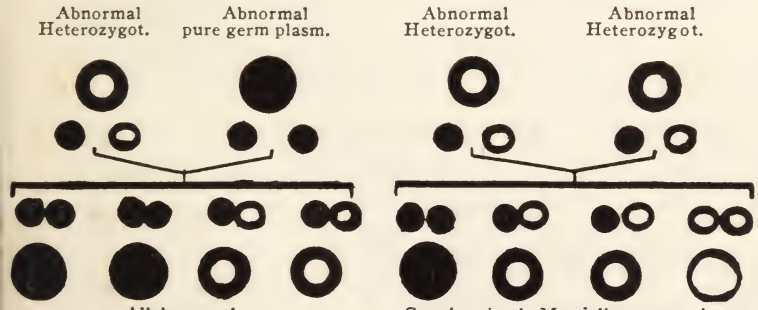
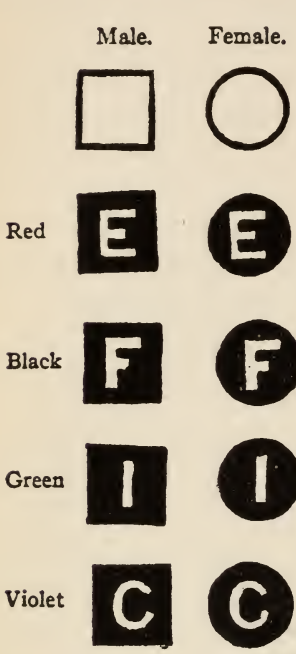


PLATE I.

KEY TO HEREDITY CHART, TAKEN FROM EUGENICS RECORD OFFICE, BULLETIN NO. 2.



Other letters used in or around the squares or circles are:

A Alcoholic.
B Blind.
D Deaf.
M Migraneous.
N Normal.
Ne. Neurotic.
P Paralytic.
Sx. Sexually immoral.
S Syphilitic.
T Tubercular.
W Wanderer or confirmed runaway.

FIGURES.

Above the line—Order in the line of birth.
Above the square or circle—Individual reference number.
Below the square or circle—Age at time of death or date of birth or death.
In squares or circles—Number of individuals of that sex.

SMALL LETTERS.

b—Born.
† or (d) Died or dead.
† (d) inf.—Died in infancy.
m—Married.

LINES.

Solid—Connects married individuals and fraternities.
Dotted—Not married or illegitimate.
For display charts: Green—Paternal side; Red—Maternal side } of individual under study.
Violet—Connects related charts or individuals on more than one chart.

SYMBOLS.

-- Shows patient at institution reporting.

Miscarriage or stillbirth.

Institutional care (place under symbol).

because it allows the brothers and sisters of the family to be placed directly in series, while, on the other chart, it is found necessary to separate these brothers and sisters in order to get in all of the matings. Even when matings are represented by drop-

PLATE II.

ABBREVIATIONS FOR CHARTS FROM EUGENICS RECORD OFFICE, BULL. No. 2

To be used with full face symbols.

a, alcoholic insanity.	p, paranoia.
d, dementia precox.	s, senile dementia.
g, general paralysis of the insane.	
m, manic depressive insanity.	t, traumatic insanity.

To be written on chart.

bd Bright's disease.	*la* locomotor ataxia.
ca cancer.	*md* manic depressive insanity.
cb childbirth.	*np* neuropathic condition.
ch chorea.	*obs* obesity.
cr cripple.	*pa* paranoia.
df deformed.	*pn* pneumonia.
dp dementia precox.	*sh* shiftlessness.
dt delirium tremens.	*sm* simple meningitis.
dy dropsy.	*sb* softening of the brain.
ec excentricity.	*sco* scoliosis.
en encephalitis.	*sd* senile dementia.
go goitre.	*su* suicide.
gp general paralysis of the insane.	*va* varices, varicose veins.
hy hysteria.	*ve* vertigo.
id ill-defined organic disease.	*x* unknown.
kd kidney disease.	*?* implies doubt.

ping one parent below the line, as is done at the Epileptic Village and the Training School, at Vineland, there is more or less confusion. In Rüdin's method one can see at a glance the number of brothers and sisters, and the proportion of those affected to the normals. The biological symbols used by Rüdin are not as clear as the symbols now in use, and shown in Plate I.

We have adopted at this hospital a combination between these two methods, in which the only change is the method of mating individuals. All other symbols are the same as the original method shown in Plate I.

Rüdin further gives some very complete printed blanks to be filled out by members of the family of the patient, or by a field worker. These are very complicated, and go much into detail, and for our use are rather too bulky.

The work of Rüdin is especially valuable to those working in this field, as many valuable suggestions are given, methods outlined and the essential data to be obtained to make the work valuable. Many of the problems are discussed and methods of attack suggested.

The Work of the Trenton State Hospital.

It will not be out of place here to describe the methods used at this hospital, where we combine "field work" for the study of heredity with "after-care" work.

We have had for a year two trained field workers, supplied through the courtesy of Dr. Charles B. Davenport, to collect data in regard to hereditary factors in the family history of patients. We have not limited them to any certain line, but have insisted that all possible information in regard to relatives should be obtained. In one instance one field worker obtained information in regard to 3300 members of a family group. This family group was located in one of the northern counties of the state, and had intermarried to such an extent that only five distinct families were represented. Of this number, 76 were insane, 22 were patients in the State Hospital, at Trenton, 14 in other hospitals and 40 not committed. Following is a list of the various abnormal individuals:

Sexual offenders	50
Epileptics	5
Alcohol	46
Feebleminded	13
Cancer	19
Sarcoma	1
Blind	2
Congenital defective	1

In practically every case investigated it is possible to obtain some information in not less than 200 members of a family, and sometimes a great many more. The field workers have found no difficulty in obtaining this information, and, without exception, they have received courteous treatment from the individuals whom they have visited. We find that the families are much interested in the work and will give all the information possible. The field worker becomes acquainted with the patient, and she talks with the patient before going to the family, and carries messages back and forth, and in this way establishes friendly relations. They spend on an average of fifteen days a month in the field. The rest of the time is spent at the hospital, writing up histories and making out charts. They do not attempt to make diagnoses, but take down all that is given by the relatives. Whenever the family physicians know anything about the family, they are visited, and their opinions also noted. Where relatives have been in the hospital, reference is made to this and a diagnosis made from the records when possible. When relatives have been in other hospitals, either in this state or in any other state, we have endeavored to obtain a copy of the records of these institutions.

The patients in the hospital are catalogued according to communities, towns, cities, etc., and when the field worker goes into a certain district she has the names of the discharged patients who are living in that community. A visit is made to these discharged patients to learn something as to their condition, and often the environment is such that it is necessary to report this to the hospital, and then advice can be given to the family as to the right method to pursue to prevent the recurrence of an attack.

This "after-care" work is a very important part of our field work, and has resulted in much good to discharged patients. Several times during the year the field workers devoted all their time to looking up discharged patients. Besides looking up the heredity in families, they inquire into the habits, domestic relations, occupation, and any other factors which are wanted by the physicians. In certain cases, where the statements of the family were questioned, the field workers had to go personally into the community, and they were able to prove or disprove these statements.

We have now collected a large number of pedigrees, averaging 200 or more to a family. It is not my purpose to go into any close

analysis of these charts, but a few are given merely to show the progress of the work.

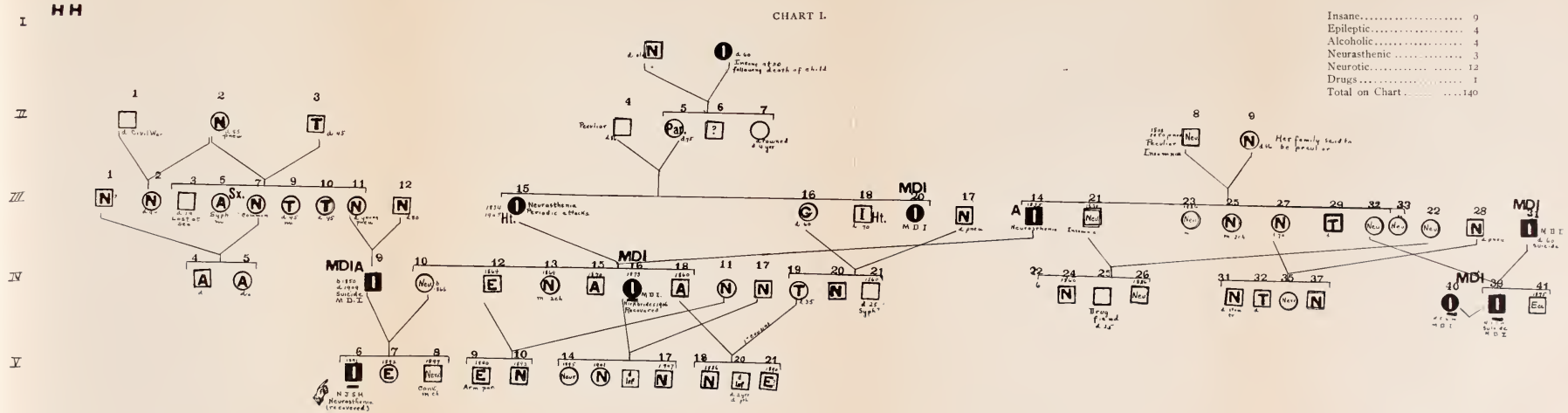
In Chart I we have a pedigree of a case of neurasthenia. The generations are given on the left-hand side of the chart. Each individual is numbered according to that generation. A transcript of the notes is given in this case to show the method of the work.

To summarize, we have a patient, a neurasthenic, one of three children, a sister of whom was epileptic and a brother nervous. The father was a manic-depressive case, who committed suicide, and the mother was neurotic. Father's family is apparently of good stock. The mother's family, however, shows marked defects. Maternal grandfather was a neurasthenic. Maternal grandmother was also a neurasthenic. One maternal uncle epileptic, and another alcoholic. Maternal aunt suffered from manic-depressive insanity, but recovered. An epileptic aunt has one epileptic boy. One of patient's great-great grandmothers, on the mother's side, was insane for 30 years, died at the age of 60, following the death of child, from which she did not recover. In the maternal grandparents' line there is a good deal of nervousness and neurasthenia. IV-39 was a patient in this hospital, manic-depressive insanity, recovered, and married a former patient. His wife had another attack, and recently the man committed suicide. His father was also a manic-depressive case, and committed suicide.

Following are the notes made by the field worker in this case:

H. H., neurasthenia (sexual) on constitutional basis. Admitted July 24, 1911. Age 20.

H. H., born in 1891, oldest child of John G. H. and Harriet S. H. He has always been extremely nervous since early childhood, had never been like other children and has always been a source of constant worry to his mother. He has always read "deep" books and stayed indoors to read them. Many of these books were quack medical books. He is the oldest of three children. The next child, a girl, Helen, is nervous and has suffered from convulsions after eating something which did not agree with her. It is perhaps epilepsy, as a brother of her mother suffers from epilepsy. The youngest child, a boy, is very nervous. There is a strong neuropathic tendency throughout the family, past as well as present generations. There is no insanity in the father's family, though there is a tendency toward sex perversion on the paternal grandmother's side. The patient's father himself committed suicide following two years drinking heavily after business reverses. The mother's family is all neuropathic, very few normal individuals to be found in the entire history. A great many of the people not committed were in a much more dangerous condition than the patient him-


HH
CHART I.
Insane..... 9
Epileptic..... 4
Alcoholic..... 4
Neurasthenic..... 3
Neurotic..... 12
Drugs..... 1
Total on Chart.....140
I
II
III
IV
V
MDI
MDIA
Sx.
Ht.
Neurasthenia
Periodic attacks
Neurasthenia
Recovered
Her family said to be peculiar
Suicide
Suicide MDI
Arm pain
Drug Habit
Insane

self. The maternal grandparents and great grandparents were eccentric, a great-great grandmother was insane, a maternal aunt was insane and recovered, a maternal uncle epileptic, a maternal great aunt and uncle insane, though never committed. A second cousin insane and, in this institution, recovered. Many others neurotic.

I. Nothing known of this generation of H.

II-2. E. P., married first a man by the name of H. He was killed in the Civil War. She died at 85 of pneumonia, and was normal always. She had one daughter by this first marriage, Eliza.

III-2. E. H., married C. F. (III-I), of W., N. J. She died at the age of 94 years. They had no children, but brought up J. H., the patient's father.

II-3. J. B., E. P.'s second husband died at 45, T. B. He was normal mentally. They had six children, James, Lydia, Angeline, Harris, Jane, Martha.

III-3. James, lost at sea, age 19.

III-5. Lydia B, who is still living. She is a *sex offender,* is alcoholic, has had syphilis and is described as a hard, bad woman. She married E. F. (II-4), a politician, of N., who is dead. Cause unknown. They had one child, Lizzie.

IV-2. Lizzie, who of T. B. She married and had one child, that died at birth.

III-7. A. B., still living, married and had two children. She is described as being "common." Her children were Edward and Harriet.

IV-4. Edward, very alcoholic, died of asthma. Married.

IV-5. Harriet, very alcoholic, died of dropsy while still young. Married. No children.

III-9. H. B., died at 45 of T. B. She married E. J., of N. They had one son.

IV-7. M. J., born 1865, married, and had four children, Raymond (LV-2-5), Perry, Anna May, Mildred, all normal children.

III-10. Jane, died at 45 of T. B.

III-11. M. B., died very young of pneumonia. She married.

III-12. E. H., of N. He died of old age at 80. He married again and had four children. Nothing known of them. They had one son, J. H., the patient's father.

IV-9. J. H., born 1850. He was a newspaper man, and, following business reverses, took to drinking heavily for two years. He quit drinking, but felt the disgrace so keenly he *committed suicide.* He must have been temporarily insane, for he had made all his plans for the next day—died 1899—suicide. He married Harriet S.

IV-15. Charles, born 1870, works on railroad, is normal, but drinks quite heavily.

IV-16. Louise S., born 1873. She was always nervous, even as a child. She was finally committed to *Hospital,* 1896. She remained there three or four months, but it was a year before she fully recovered. *Diagnosis, melancholia.* She married S. C., a school principal of W. They have had four children, Mildred, Frances, Cecil, and a baby that died at 11 days.

V-14. Mildred, born 1895, very *nervous.*

V-15. Frances, born 1901.

V-16. Boy, died at 11 days.

V-17. Cecil, born 1907, normal.

IV-18. David S., born 1860. Periodically alcoholic. He married Ella K., a first cousin, who died of T. B. at 35 years. They had three children, Clarence, Tuttle, Lester.

V-18. Clarence, born 1886, a government life saver. He is delicate, but normal.

V-19. He married G. T. They have one daughter.

VI. Ruth, born 1909.

V-20. Tuttle, died of diphtheria at 2 years.

V-21. Lester, born 1890, a farmer at F. He had *severe convulsions* up to the time he was 14 years. None since.

I-1. S. L., died very old. Married H. T.

I-2. H. T., *insane.* She went insane following the death by drowning of her 4-year-old daughter. She was kept at home and was mildly demented. Died at 60. They had three children, Margaret, Charles and the girl who was drowned.

II-5. Margaret L., died at 75 of paralysis. She married (II-4) F. C., a miller of F. He was of rather weak character and *was peculiar.* Died at 85 of old age. They had four children, Hannah, Mary, John, Anna.

III-15. Hannah (patient's grandmother), married C. S.

III-16. Mary, died at 60 of *cancer* of stomach. Married W. K. (III-17), who died of pneumonia. They had three children, Ella, Frank, Edward.

IV-9. E. K., married her first cousin, D. S. (IV-18). She died at 35 of T. B.

IV-20. Frank, living, normal.

IV-21. Edward, died at 25 in 1885, after 3 or 4 years sickness. He lost use of limbs, flesh rotted away, thinks it may have been syphilis.

III-18. John, died at 70 of heart failure. *He told lies, did not seem to know they were lies.* Married H. B., who is still living. No children.

III-20. Anna C. *She is subject to depressed spells. Has always posed as a martyr,* knowing she will be rewarded hereafter. Tells malicious lies about the neighbors and friends, seemingly believing them to be true.

II-8. D. S., born in 1808, died in 1850 of pneumonia. He was always nervous and peculiar. Suffered from *insomnia* always. He married (II-9) H. H., who died of old age at 86. She was normal, though her family is said to have been queer. They had fourteen children, Charles, Kate, Elizabeth, Jacob, Anna, Harriet, William, Helen, and six that died in infancy.

III-14. Charles (patient's grandfather).

III-21. Jacob, born 1832, still living. He is a newspaper man, an editor at times. He has been *neurotic* for years. Has suffered from *insomnia* and is *childish now.* He married Martha J. M., who is very nervous. Still living. They had nine children, Robert, Albert, Warren, and six that died in infancy.

IV. H. S., born in H. in 1866, a daughter of C., and H. S. She is of a highly *nervous temperament.* Worries over H., over the other children, etc. She recognizes plainly the neurotic tendency in the family. They have three children, Hamilton, Helen and Jack.

V-6. H. H., born 1891. *Admitted to N. J. S. H.* in July, 1911, diagnosed *neurasthenia* (sexual) *on constitutional basis.*

V-7. H. H., born 1892. Nervous temperament. She has had severe convulsions since infancy whenever anything she eats disagrees with her. Had one severe convulsion, summer, 1911. *Epilepsy.*

V-8. J. H., born 1897. Is extremely *nervous.* Had convulsions while teething.

III-14. C. S., born 1835. Lived in H., a mason by trade. He has always been ugly and *irritable, "devilish"* at times. Seemed to try to torment his family, drove his wife nearly insane, with his tormenting. Has been a steady drinker all his adult life. He married (III-15).

III-15. H. C., of F. She was one of four children. She was born in 1834, died in 1905 of dropsy and heart trouble. She was very nervous, almost, if not, insane, at times, when she became so excited that she had to be restrained. She and C. S. had six children, Harriet, Walter, Jennie, Charles, Louise, David.

IV-11. Walter, born in 1864. He is an electrician in N. Has suffered from *epilepsy* all his life. Is rather irritable and hard to get along with. He married L. H., who is strong and well. They have two sons, George and Ben.

V-9. George, born 1890, had severe convulsions as a child, one arm has been paralyzed since he was 6 or 7 years of age. *Epilepsy (?).*

V-10. Bert, born 1892, normal.

IV-13. Jennie S., born 1889. She is normal in every way. Married Richard N., of F., N. J., painter and decorator. Three children, Joseph, Ethel, Richard.

V-11. Joseph, born 1894.

V-12. Ethel, born 1897.

V-13. Richard, born 1902.

IV-24. Robert, born 1860. A musician in P. He married J. V., of L. No children.

IV-25. Albert, died at 25. He was a dentist, and became a morphine fiend, which drug probably caused his death.

IV-26. Warren, born 1886, a farmer. Very nervous. Married. No children.

III-23. Elizabeth, born 1826. *Very nervous.* Married E. T., who died of Bright's disease. No children.

III-25, Kate, still living. Married R. O., of H., N. J. They had several children, only two lived to grow up, Harry and Richard.

IV-31. Harry, a traveling man, married M. S., who died of stomach trouble. They had two children, a girl and a boy.

IV-33. R. O., died when a young man, of Bright's disease. He was a doctor.

III-27. A. S., normal. Died at 70 years of old age. She married T. T., who died of pneumonia. They had four children, Melville, Kate, Frank, Edward.

IV-31. Melville T, died of stomach trouble. He married M. C. They had two children, Melville and Anna.

IV-33. Kate, very nervous, married C. F., who died of appendicitis. They had two children, Norman and Charles.

IV-37. Frank, normal, married Olive —. They have 5 children.

III-29. William, died of T. B., while still a young man.

III-32. Harriet S., died of uræmic poisoning. She was always *neurotic,* always doctoring for her nerves. If she had not been so well taken care of would probably have been a neurasthenic case. She married W. S., a school principal, who *committed suicide* at 60 years. They had two children, Forrest and Lyle.

IV-39. F. S., admitted to N. J. S. H. in 1907. Discharged in 1908. A case of manic-depressive insanity, manic attack. Died March, 1912, suicide. He married E. D. C., whom he met while in the hospital. She was admitted in 1906, discharged in 1908, manic-depressive insanity, depressed type. Readmitted Febuary, 1912, M. D. I., manic type.

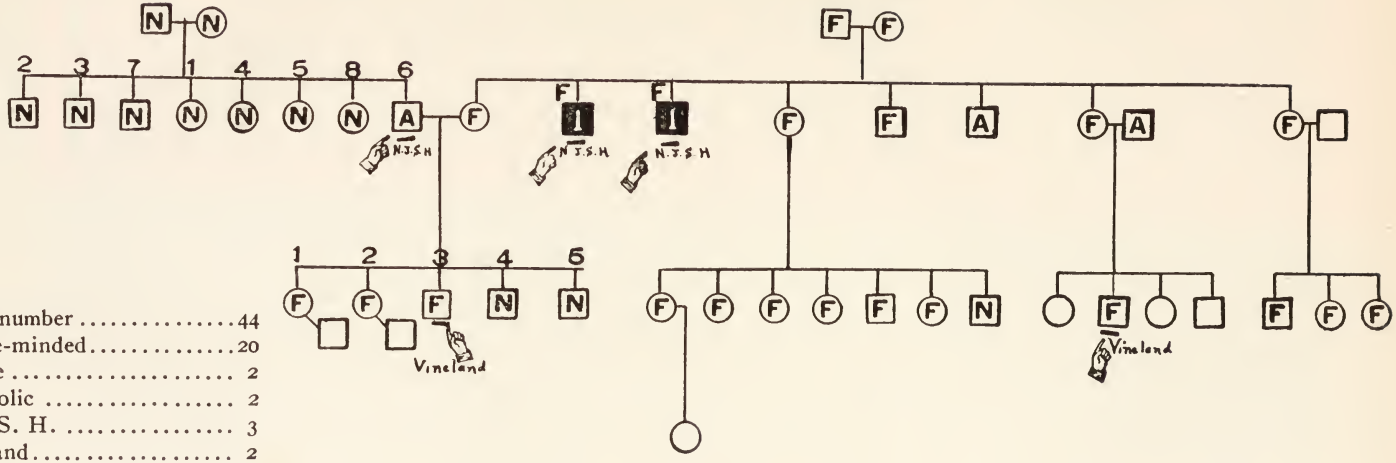
IV-41. L. S., born in 1875. He is a civil engineer, and is rather *eccentric.*

III-33. H. S., born in 1850. She is *very nervous.* Unmarried.

Chart II is an illustration of the method charting that is used by the Eugenics Record Office.

CHART II

BB

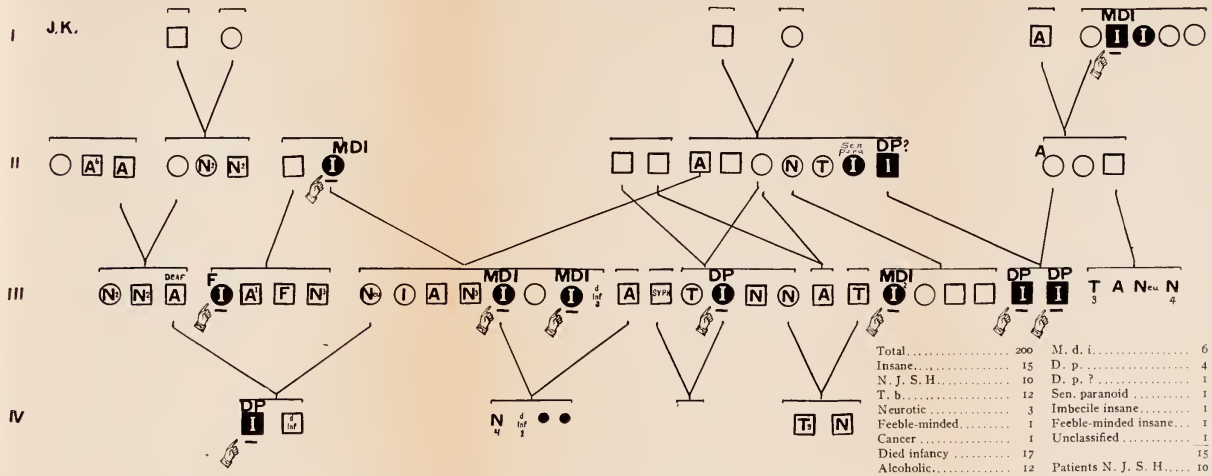


Total number 44
Feeble-minded 20
Insane 2
Alcoholic 2
N. J. S. H. 3
Vineland 2

There are 42 individuals in this chart, whereas, Chart I has over 70. This shows the inheritance through marriage of two feebleminded individuals. Nine children were born to this family, all of which are feebleminded, one alcoholic. Two of these children are inmates of this hospital at present. One girl of this feebleminded pair married a man, who was an alcoholic, but his family was normal. As the result of this union three are feebleminded and two normal. One feebleminded child is in Vineland. Another girl married a man, who was an alcoholic, and has one feebleminded child at Vineland. Four members of this group are now cared for by state institutions. There are altogether 22 feebleminded progeny from the original mating.

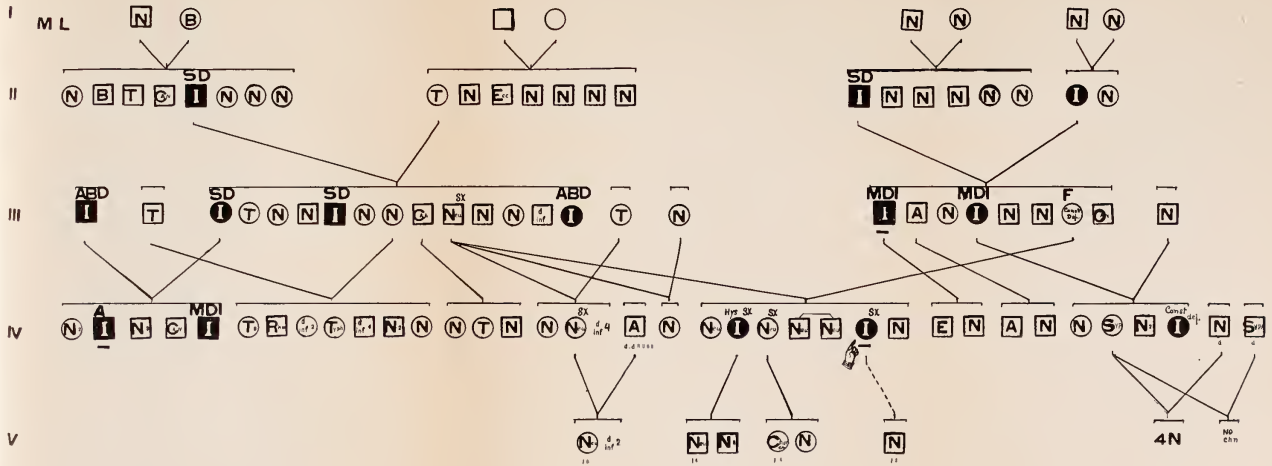
Chart III is a summary which represents 200 individuals, 15 of which were insane. Ten were in the Trenton State Hospital, and 12 were tubercular, three neurotic, one feebleminded. Seventeen died in infancy. Nineteen were alcoholic. Of the psychoses, we have six manic depressives, four dementia præcox, one questionable dementia præcox, one senile paranoid condition, one imbecility, one feebleminded and one unclassified. The paternal line is fairly good, with the exception of alcoholism. The maternal line, on the other hand, is very much affected. The mother is neurotic. One sister was a border-line case. There are two sisters manic-depressive. The mother had nine living children. Three died in infancy, making a family of 12. The mother was insane, had manic-depressive insanity, from which she recovered, and is now living at the age of 83. The father was alcoholic, had a sister who was a senile paranoid condition, and a brother dementia præcox. This brother had two children, both of which are dementia præcox, and inmates of this hospital. He married a woman put down as peculiar. A maternal cousin is a case of dementia præcox in this hospital. In this family, out of five individuals who were insane in the grandparents or great grandparents, only two were in institutions, while all the cases that were insane in the parents' children were committed to institutions. This fact will be found to run through all our charts, and one can conclude that in the preceding generations the percentage of cases who were insane and who were committed to an institution was much smaller than the percentage of the same class in present generation.

CHART III.



Total	200	M. d. i.	6
Insane	15	D. p.	4
N. J. S. H.	10	D. p. ?	1
T. b.	12	Sen. paranoid	1
Neurotic	3	Imbecile insane	1
Feeble-minded	1	Feeble-minded insane	1
Cancer	1	Unclassified	1
Died infancy	17		15
Alcoholic	12	Patients N. J. S. H.	10

CHART IV.



Total..... 371	Insane..... 14	Syph..... 2	Deaf..... 2	Alc. ins..... 1	Con. def..... 1
Abnormal..... 73	Alcoholic..... 6	Blind..... 2	Cripple..... 2	Sen. dem..... 2	Ar. scl..... 3
Normal..... 298	T. b..... 16	Cancer..... 5	Con. def..... 2	Sen. psyc..... 3	M. d. i..... 2
D. Infancy..... 16	Sx..... 11	Epil..... 1	Neur..... 10	Traum. psyc..... 1	Psychas..... 1
	47	10	16	7	7

Patient is psychasthenic.
Father is neurotic and sx., mother constitutional defective.
Paternal line: 3 senile dementia, 2 arteriosclerotic, 1 m. d. i., 1 alc. ins.
Maternal line: Grandfather senile dementia, grandmother insane, 2 m. d. i., 1 f. insane, 1 epileptic.
Fraternal line: 7 neurotic, 4 sx., 2 insane.

This has an important bearing on the apparent increase in the number of insane in institutions at present, for I think I can be definitely shown that a large proportion of those who were insane in the community in previous generations were kept at home.

Chart IV represents a family of 371 members, in which 73 were abnormal in the following proportions: Insane, 14. Alcoholic, six. Sexual offenders, 11. Syphilitic, two. Blind, two. Cancer, five. Epileptic, one. Deaf mutes, two. Congenital, two. Constitutional defects, two. Neurotics, 10.

Diagnoses of the insane are as follows:

Alcoholic insanity	1
Senile dementia	2
Senile Psychoses	3
Senile trauma	1
Senile defective	1
Arteriosclerotic brain disease	3
Manic-depressive insanity	2
Psychasthenia	1

The patient represented by three asterisks was psychasthenic. She has six brothers and sisters. Two brothers, twins, and neurotic. Two sisters are neurotic, and one sister insane, with diagnosis of hysteria. We find that the mother was a constitutional defective. There were two brothers and sisters, each manic-depressive insanity, and the maternal grandparents were both senile psychoses. The father was neurotic or psychopathic sexual individual. He was married three times. He had two sisters. One was senile and the other arteriosclerotic. One brother suffered from senile psychosis, due to head trauma, at the age of 50. There were 13 brothers and sisters in this family. Three could be classed with the senile psychoses, and we find that the father of this family died at the age of 60, of senile dementia, while the mother came apparently from normal stock.

This chart illustrates a very important point, that is, the hereditary features of senile psychoses. Here the diagnosis is not made merely on old age, because there are in this family thirteen normal individuals living at the ages of 70, 93, 60 and 85. This tendency to senility in this family seems to be in the proportion of three to twelve, 13 children dying in infancy.

Another significant fact is the tendency in succeeding generations to develop manic-depressive insanity and psychopathic states.

Chart V illustrates inheritance in a case of dementia præcox. The patient was one of six children, three of which died in infancy, one was neurotic and one normal. The father was a case of manic-depressive insanity, recovered, living at the age of 62. He is one of six children. One brother has arteriosclerotic brain disease, three normal, one sister neurotic. Patient's maternal grandparents, the grandmother's line, is apparently normal. His paternal grandfather was a constitutional defective, died at 66, of arteriosclerosis. The grandfather has 12 brothers and sisters. One brother is put down as melancholy. One died at the age of 42, had sunstroke, and died insane. One was a constitutional defective, died at the age of 19. There were five affected individuals in this group of 13. Seven could be put down as normal, one neurotic. The mother of the patient was neurotic. She was one of fifteen children, seven of which died in infancy. Three were normal and one had harelip. The maternal grandmother was put down as insane. The maternal grandfather apparently normal line. We have a summing up, then, of a total of two hundred ninety-two. Insane, fifteen. Epileptics, four. Feebleminded, one. Neurotic, five. Harelip, one. Syphilis, two. Sexual offenders, two.

Diagnosis of those insane are as follows:

Constitutional defective	2
Melancholia	1
Dementia præcox	1
Psychoses following sunstroke	1
Hysteria	1
Manic-depressive insanity	3
Arteriosclerosis	2
Depression	1
Feebleminded	1
Unknown	2

Conclusions.

We have not attempted to analyze these charts carefully, but they are given merely to illustrate the progress of the work, and also to illustrate what a difficult task the analysis of these charts means. Frequently, when a point in question is necessary, the field worker visits the family again to clear up these disputed points.

In this paper no attempt has been made to give any definite conclusions regarding the hereditary factors in the various psychoses. We have reviewed some of the most important work done so far, and outlined the methods to be pursued to obtain the best results in future work.

We have also spoken of some of the difficulties to be met with, especially when analyzing the material as it comes from the field workers. It is again well to emphasize the necessity of maintaining an open mind regarding these problems, and not to be too biased in attempting to make the facts fit the Mendelian laws. At the same time, we recognize that a comprehensive knowledge of the laws will assist us materially in analyzing our data and in arriving at practical conclusions. We also feel that much valuable material will be obtained which will aid us in solving the problems of prophylaxis and prevention of mental diseases. We hope that other hospitals and institutions will adopt this method of studying these important questions.

I wish to express my thanks to Miss Florence I. Orr and Miss Elizabeth P. Moore (field workers at this hospital) for their valuable assistance in making charts and furnishing valuable data for this paper.

LITERATURE (from Rüdin).

BIOLOGY.

Archiv für Rassen- und Gesellschaftsbiologie, einschliesslich Rassen- und Gesellschaftshygiene. Jahrgänge 1904, 1905, 1906, 1907, 1908, 1909, 1910, 1911. Originalien und Referate über samtliche Fragen der Vererbung, Variabilitat, Entartung, Degeneration, Mendel'sche Lehre. usw. Verlag von B. G. Teubner, Leipzig.

Bateson, W.: Mendel's Principles of Heredity. Cambridge, 1909. At the University Press.

——— The progress of genetics since the rediscovery of Mendel's papers. Progressus Rei Botanicae, 1, 1906. Verlag von Gustav Fischer, Jena.

——— An address of Mendelian heredity and its application to man. Brain, 29, 157, 1906, und Brit. med. Jour., 1906.

——— Mendelian heredity. Brain, No. 114, 1906.

——— and Saunders, E. R.: Reports to the Evolution Committee of the Royal Society, 1, 1902.

——— Methoden und Ziele der Vererbungslehre. Biol. Centralbl., 29, 1909.

Baur, E.: Einige Ergebnisse der experimentellen Vererbungslehre. Med. Klin. (Beiheft), 1908.

——— Einführung in die experimentelle Vererbungslehre. Berlin, 1911. Gebr. Bornträger.

Beard, Erblichkeit: Brit. med. Journ., No. 2285, 1904.
Beitzke, Vererbung: Berl. Klin. Wochenschr., Nr. 36, 1905.
Bevan-Lewis, W.: The presidential address on the biological factor in heredity, delivered at the sixty-eighth annual meeting of the Medico-psychological Association, held at Wakefield on July 22 and 23, 1909. Journ. of Mental Science, 55, 591-630, 1909.
Biffen, R. H.: Studies in the inheritance of disease-resistance. Journ. Agric. Science, 2, 1907.
Biometrica, A.: Journal for the statistical study of biological problems. Cambridge, at the University Press.
Castle, W. E.: Heredity of coat characters in guinea pigs and rabbits. Publ. Carnegie Inst., Wash., 1905.
——— The heredity of Angora coat in mammals. Sc. N. S., 18, 1903.
——— The laws of heredity of Galton and Mendel and some laws governing race-improvement by selection. Proc. Amer. Acad. Arts and Sc., 39, 223, 1903.
——— and Allen, Gl. M.: The heredity of Albinism. Contrib. from the zool. Labor. of the Mus. of comp. zool, at Harvard Coll., Cambridge, Mass., 1903. Proc. Amer. Ac. 38, 1903.
Chantemesse, Pr.: Hérédité. Progrès médical. October, 1900, No. 40-42.
Chapeaurouge, A. de: Einiges über Inzucht und ihre Leistungen auf verschiedenen Zuchtgebieten. Hamburg, 1909.
Conklin, E. G.: The mechanism of heredity. Science, 27, 89, 1908.
Correns, C.: Gregor Mendels Versuche über Pflanzenhybriden. Bot. Ztg., 58, 230, 1900.
——— Experimentelle Untersuchungen über die entstehung der Arten auf botanischem Gebiet. Archiv f. Rass. u. ges. Biol., 1, 27, 1904.
——— Ueber Vererbungsgesetze. Berlin, 1905, bei Gebr. Borntträger.
——— Gregor Mendel's Briefe an Carl Nageli 1866-1873. Abt. Math.-Phys. Kgl. Sachs. Ges. d. Wiss., 19, 1905.
——— Bastarde zwischen Maisrasse. Biol. Bot. H., 53, 1901.
——— Ueber Bastarde zwischen Rassen von Zea Mays usw. Berl. D. Bot. Ges., 19, 1901.
——— Die Ergebnisse der neuesten Bastardforschungen für die Vererbungslehre. Berl. D. Bot. Ges., 19, 1901.
——— Der Uebergang aus dem homozygotischen in einen heterozygotischen Zustand usw. Berl. D. Bot. Ges., 28, 1910.
Crzellitzer, A.: Sippschafttafeln, ein neues Hilfsmittel zur Erblichkeitsforschung. Med. Reform, Nr. 48, 50 u. 52, 1908.
——— Methoden der Familienforschung. Zeitschr. f. Ethmol, 182, 1909.
Cuénot, L.: Rapport sur l'hérédité. Ass. Franc. Adv. Sc. Congrès de Lyon, 1906.
——— L'hérédité de la Pigmentation chez lez souris. Arch. zool. expér. et gén. (4), 2, 1904.
——— L'hérédité de la pigmentation chez les souris. 5 Note, Arch. zool. expér. et gén. (4), 6, 1907.

Damaye, L'hérédité collatérale, sa valeur et son importance en pathologie. Revue scientifique, 1, 5 série, 1904.

Darbishire, A. D.: On the result of crossing Japanese waltzing with albino mice. Biometrica, 3, 1904.

——— On the result of crossing round with wrinkled peas, etc. Proc. Roy. Soc., 80, B., 1908.

——— Notes on the results of crossing Japanese waltzing mice, etc. Biometrica, 2, 1902.

——— On the supposed antagonism of Mendelian to biometric theories of heredity. Mem. and Proc., Manchester Lit. and Phil. Soc., 49, 1905.

——— On the difference between physiological and statistical laws of heredity. Mem. and Proc., Manchester Lit. and Phil. Soc., 50, 1906.

——— On the experimental estimation of the theory of ancestral contributions in heredity. Proc. Roy. Soc., B. 81, 1909.

Davenport, C. B., u. G.: Heredity of skin pigment in man. Amer. Naturalist, 44, 641, 1910.

——— Heredity of hairform in man. Amer. Naturalist, 42, 341-349, 1908.

——— Heredity of eye-color in man. Science, 26, 589-592, 1907.

Davenport, C. B.: Inheritance in poultry. Carnegie Publication 52, Washington, 1906.

——— Degeneration, albinism, and inbreeding. Science N. S., 28, 44, 1908.

——— The imperfection of dominance, etc. Amer. Naturalist, 44, 1910.

——— The origin of black sheep in the flock. Science N. S., 22, 1905.

Delamare, Hérédité morbide. Journ. de l'anat. et de la phys., No. 6, 1903.

Demoll, Zur Lokalisation der Erbanlagen. Zool. Jahrb., 30, 133, 1910. Abt. f. zool. Phys.

Devrient, Ernst: Familienforschung in Aus Natur und Geisteswelt, 350. Leipzig, 1911, B. G. Teubner.

Dietrich, O.: Die Bedeutung der Vererbung für die Pathologie. Tübingen, 1902.

Duerst, N. J.: Selektion und Pathologie. Hannover, 1911 (Arb. d. D. G. f. Züchtungskunde, 12).

Farabee, W. G.: Inheritance of digital malformation in man. Papers of the Peabody Museum of American Archaeology and Ethnology, Harvard University, Vol. 3, No. 3, Cambridge, Mass., 1905.

Fehlinger, H.: Die Gültigkeit der Mendelschen Vererbungsregeln für den Menschen. Polit. Anthrop., Revue, 9, 374, 1910.

Fick, R. V.: Vererbungsfragen, Reduktions- und Chromosomen-Hypothesen, Bastardregeln. Ergebn. d. Anat. u. Entwicklungsgesch., 19, 1906.

Fischer, E.: Weitere Untersuchungen über die Vererbung erworbener Eigenschaften. Allgem. Zeitschr. f. Entomologie, 1902.

Fischer-Planer: Die Vererbung Geistiger Fähigkeiten. Archiv f. system. Philosophie, N. F., 13, 1907.

Fruwith, C.: Untersuchungen über den Erfolg und die zweckmässige Art der Durchführung von Veredlungs-Auslese-Züchtung bei Pflanzen mit Selbstbefruchtung. Archiv f. Rass.- u. Ges. Biol., 4, 145, 1907.

——— Die Zuchtung landwirtschaftlicher Kulturpflanzen. Berlin, 1905.

Galton, F.: The average contribution of each several ancestor to the total heritage of the offspring. Proc. Roy. Soc., 61, 401, 1897.

——— Natural inheritance. London, 1889.

——— Englishmen of science. London, 1874.

——— Hereditary genius. London, 1892 (Deutsch von Neurath, Leipzig, 1910).

——— Entwürfe zu einer Fortpflanzungshygiene. Archiv f. Rass.- u. Ges.-Biol., S. 812, 1905.

——— Correlations and their measurements. Proc. Roy. Soc., 45, 136, 1888.

Garrod, A. F.: The incidence of alkaptonuria; a study in chemical individuality. Lancet, 13, Dez., 1902.

Goldschmidt, R., Einführung in die Vererbungswissenschaft. Leipzig, 1911, Engelmann.

Grob, J.: Ueber einige Beziehungen zwischen Vererbung und Variation. Biol. Centralbl., 26, 1906.

Gruber, M. v.: Organisation der Forschung und Sammlung von Materialien über die Entartungsfrage. Concordia, 1910.

v. Gruber u. Rüdin: Fortpflanzung, Vererbung, Rassenhygiene. München, 1911. J. F. Lehmanns Verlag. Zweite, erganzte und verbesserte Auflage.

Hacker, Valentin, Allgemeine Vererbungslehre. Braunschweig, 1911, Friedrich Viehweg & Sohn.

——— Wandtafeln zur Allg. Biologie. Leipzig, 1907 u. 1909, Quelle & Meyer.

——— Ueber Axolotl-Kreuzungen. II. Mitt. Verh. d. D. Zool. Ges., 1908.

——— Ueber die Neueren Ergebnisse der Bastardlehre, ihre zellengeschichtliche Begründung und ihre Bedeutung für die praktische Tierzucht. Archiv f. Rass.- u. Ges.-Biol., 1, 321, 1904.

——— Bastardierung und Geschlechtszellenbildung. Zool. Jahrb. Suppl., 7, 1904.

Hammer-Stuttgart: Die Bedeutung der Vererbung für die Haut und ihre Erkrankungen. Verhandl. d. Deutsch. Dermatol. Gesellsch., 10. Kongr. Frankfurt a. M., 8-10 Juni 1908, S. 71-93, Berlin 1908, Julius Springer.

Hansemann, D. v.: Deszendenz und Pathologie. Berlin, 1909.

Hardy, G. H.: Statistical results of Mendelian heredity. Science N. S., 28, 49.

Heidenreich, E.: Familiengeschichtliche Quellenkunde. Leipzig, 1909, H. A. Ludwig Degener.

Herbst, H.: Vererbungsstudien. Archiv f. Entwickl-Mechanik, 21, 22, 24, 27, 1906-1909.

Hertwig, O.: Der Kampf um Kernfragen in der Entwicklungs- und Vererbungslehre, Jena, 1908.

Hurst, C. C.: Experimental studies on heredity in rabbits. Journ. Lin. Soc., 29, 1905.

——— Inheritance of eye-colour in man. Proc. Roy. Soc., London, Biometrica, 80, 85, 1908.

Johannsen, W.: Elemente der exakten Erblichkeitslehre. Jena, 1909, Gustav Fischer.

——— Ueber Erblichkeit in Populationen und reinen Linien. Jena, 1903.

Kammerer, P.: Vererbung erzwungener Fortpflanzungsanpassungen. Archiv f. Entwickl.-Mechanik, 25, 7, 1907.

——— Beweise für die Vererbung erworbener Eigenschaften. Berlin, 1910 (12. Flugschr. d. D. Ges. f. Züchtungskunde).

Kekule v. Stradonitz: Streifzüge durch die medizinisch-genealogische Literatur. Mitteil. d. Zentralstelle f. deusche Personen- u. Familiengesch., 3, H. Leipzig, 1908.

——— Angabe einer Ahnenbezifferungsmethode. Vierteljahrsschr. f. Wappen-Siegel u. Familienkunde, 26. Jahrg., S. 64 ff.

Kierman, J. W.: Heredity. Medical News, 15. Febr., 1902.

Lang, A.: Ueber Vererbungsversuche. Verh. d. D. Zool. Ges., 1909.

——— Ueber die Mendelschen Gesetze, Art und Varietätenbildung, Mutation und Variation, insbesondere bei unsern Hain und Gartenschnecken. Schweiz, Naturf.-Ges., Luzern, 1905.

——— Ueber die Bastarde von Helix hartensis Müller und Helix nemoralis L., eine Untersuchung zur experimentellen Vererbungslehre. Jena, 1908.

——— Ueber alternative Vererbung bei Hunden. Z. f. indukt. Abstammungslehre, 3, 1, 1910.

——— Die Erblichkeitsverhältnisse der Ohrenlange usw. Z. f. indukt. Abstammungslehre, 4, 1910.

Lendenfeld, Robert: Karl Pearson's Untersuchungen über verwandtshaftliche Ähnlichkeit und Vererbung geistiger Eigenschaften. Archiv f. Rass.- u. Ges. Biologie, 1, 78, 1904.

Lenz, Fr.: Ueber die Verbreitung der Lues, speziell in Berlin und ihre Bedeutung als Faktor des Rassentodes. Archiv f. Rass.- u. Ges.-Biol., 7, 306, 1910.

Lock, Robert: Recent progress in the study of variation, heredity and evolution. London, 1907.

Lorenz, Ottokar: Lehrbuch der gesamten wissenschaftlichen Genealogie. Berlin, 1898.

Lütgendorf-Leinburg, Familiengeschichte, Stammbaum- und Ahnenprobe. Frankfurt a. M.

Lutz, A.: Ueber einige Stammbäume und die Anwendung der Mendelschen Regel auf die Ophthalmologie (Graefes Archiv, 1911).

Martius, F.: Krankheitsanlage und Vererbung. Leipzig u. Wien, 1905.

——— Referat über Krankheitsanlage und Vererbung mit besonderer Berücksichtigung der Tuberkulose. Neurol. Centralbl., 1905, S. 489.

——— Das Vererbungsproblem in der Pathologie. Berl. klin. Wochenschr., 1901.

——— Das pathologische Vererbungsproblem. Leipzig u. Wien, 1909.

——— Die Bedeutung der Vererbung für Krankheitsentstehung und Rassenerhaltung. Archiv f. Rass.- u. Ges.-Biol., 7, 470, 1910.

Mendel, Gregor J.: Versuche über Pflanzenhybriden. Verh. Natur.-Verein, Brünn, 10, 1865 (Ostwalds Klassiker der exakten Wissenschaften).

Morgan, Th. H.: Experimentelle Zoologie (Deutsch von Rhumbler), Leipzig u. Berlin, 1909.

Naegeli-Ackerblom: Die Geminität und ihre Erblichen Beziehungen. Virchows Archiv f. pathol., Anat. u. Physiol., 170, 1902.

Nettleship: Seven new pedigrees of hereditary cataract. Ophthalmol. Soc. Trans., 29, 1909.

Orschansky, J.: Die Vererbung im gesunden und Krankhaften Zustande und die Entstehung des Geschlechts beim Menschen. Stuttgart, 1903. S. 347.

Orth, J.: Die Bedeutung der Erblichkeit für die Pathologie. Berl. Klin. Wochenschr., 1906.

——— Ererbte und angeborene Krankheiten und die Krankheitsanlagen. In Krankheiten und Ehe (Senator und Kaminer), 1, 26.

Park: Heredity. Scott. Med. and Surg. Journ., 9, No. 5, 1901.

Pearson, K.: Note on inheritance in man. Biometrica, 6, 1908.

——— The theory of ancestral contributions of heredity.

——— On the ancestral gametic correlations of a Mendelian population mating at random. Proc. Roy. Soc., 81, N. 547, B. S., 219 u. 225. London, 1909.

——— The law of ancestral inheritance. Biometrica, 2, Part 2, 1903.

——— On the laws of inheritance in Man. 1. Inheritance of physical character. Biometrica, 2, 1903. 2. On the inheritance of the mental and moral characters, etc. Biometrica, 3, 1904.

Pearson, K.: Ueber den Zweck und die Bedeutung einer nationalen Rassenhygiene (National Eugenik) für den Staat. Archiv f. Rass.- u. Ges.-Biol., S. 67, 1908.

Pick, F.: Ueber Krankheitsvererbung. In der Deutschen med. Wochenschr., 1911.

Plate, L.: Ueber Vererbung und die Notwendigkeit der Gründung einer Versuchsanstalt für Vererbung- und Züchtungskunde. Archiv f. Rass.- u. Ges.-Biol., 3, 777, 1906.

——— Vererbungslehre und Deszendenztheorie. Antrittsvorlesung zur Uebernahme der Zoologischen Professur in Jena. Festschr., z. 60. Geburtstag Richard Hertwigs, 2, 1910. G. Fischer, Jena.

——— Ein Versuch zur Erklärung der gynephoren Vererbung Menschlicher Erkrankungen. Archiv f. Rass.- und Ges.-Biol., S. 164, 1911.

——— Ueber die Bedeutung des Darwinschen Selektionsprinzips und Probleme der Artbildung, 2 A. Leipzig, 1903.

Ploetz, A.: Ziele und Aufgaben der Rassenhygiene. Deutsche Vierteljahrsschr. f. öffentl. Gesundh., 43, H. I., S. 164.

——— Die Begriffe Rasse und Gesellschaft und einige damit zusammenhängende Probleme. Schriften der Deutschen Gesellsch. f. Soziologie, 1, Serie, 1. Bd. Tübingen, J. C. B. Mohr.

Poll, H.: Mischlingsstudien. System und Kreuzung. Sitzungsber. d. Ges. d. Naturf.-Fr. Berlin, 1907.

Prinzing, Fr.: Die Vererbung pathologischer Eigenschaften. Monatsschr. f. Kriminalpsychol. u. Strafrechtsreform, 5, 1908.
Punnett, R. C.: Mendelism in relation to disease. Proc. Roy. Soc., 1908.
——— Mendelismus. Verlag. Carl Winiker, Brunn, 1910.
Raymond, P.: L'hérédité morbide. Paris, 1905.
Ribbert, H.: Die Vererbung der Krankheiten. Pol. Anthropol. Revue, Mai, 1904.
——— Ueber Vererbung. Vortrag. Marburg, Elwert, 32 S., 1902.
Schuster, E., and Elderton, E. M.: The inheritance of ability. London, 1907.
Schwalbe, E.: Das Problem der Vererbung in der Pathologie. Münch. med. Wochenschr. Nr. 37, S. 1579; Nr. 38, S. 1460; 1903.
——— Missbildung und Variationslehre. Jena, 1910.
Semon, R.: Der Stand der Frage nach der Vererbung erworbener Eigenschaften. Fortschr. d. naturw. Forschung, 2, 1, 1910.
Sommer, Max: Die Brown-Séquardsche Meerschweinchenepilepsie und ihre Uebertragung auf die Nachkommen. Inaug.-Diss., Jena, 1900, u. Zieglers Beitr. z. path. Anat. u. z. allg. Pathol., 27, 1900.
——— Zur Frage der erblichen Uebertragbarkeit der Brown-Séquardschen Meerschweinchenepilepsie. Neurol. Centralbl., 1901, S. 152.
Sommer, R.: Goethes Wetzlarer Verwandtschaft. Leipzig, 1908.
——— Goethe im Lichte der Vererbungslehre. Leipzig, 1908.
Standfub, M.: Zur Frage der Gestaltung und Vererbung auf Grund 28 jähriger Experimente. Leipzig, 1902.
——— Die Resultate 30 jähriger Experimente. Verh. d. Schweiz. Naturf.-Ges., Luzern, 1905.
Steiger, A.: Studien über die erblichen Verhältnisse der Hornhautkrümmung. Zeitschr. f. Augenheilk., 16, 229, 1906.
Stockmann: Heredity. Glasgow Med. Journ., 65, No. 3.
Strasburger, E.: Die Stofflichen Grundlagen der organischen Vererbung, Jena, 1905.
The Treasury of human inheritance (thesaurus hereditatis). University of London. Francis Galton Laboratory for National Eugenics. Mit Abbildungen und Stammbäumen. London, Dulau & Co.
Thomson, J. A.: Heredity. London, 1908.
Tschermak, E. v.: Der Moderne Stand des Vererbungsproblems. Archiv f. Rass.- u. Ges.-Biol., 5, 305, 1908.
——— Die Mendelsche Lehre und die Galtonsche Theorie vom Ahnenerbe. Archiv f. Rass.- u. Ges.-Biol., 2, 663, 1905.
Vries, H. de: Die Mutationstheorie. Leipzig, 1901.
Weinberg, W.: Pathologische Vererbung und Genealogische Statistik. Archiv f. klin. Med., 78, 1903.
——— Ueber den Nachweis der Vererbung beim Menschen. Jahreshefte d. wurtt. Vereins f. vaterl. Naturkunde, 1908.
——— Ueber Vererbungsgesetze beim Menschen. Zeitschr. f. induktive Abstammungs- u. Vererbungs-lehre, 1, 2, 1909.
——— Aufgabe und Methode der Familienstatistik bei medizinisch-Biologischen Problemen. Zeitschr. f. soz. Med., 3, 1907.

Weismann, A.: Semon's "Mneme" und die Vererbung erworbener Eigenschaften. Archiv f. Rass.- u. Ges.-Biol., 3, 1, 1906.

Weldon, W. F. R.: Inheritance in animals and plants. Lectures on the method of science. Oxford, 1906.

——— Albinism in Sicily and Mendel's law. Biometrica, 3, 107, 1904.

Zeitschrift für induktive Abstammungs- und Vererbungs-lehre.

Ziegler, H. E.: Vererbungslehre in der Biologie. Jena, 1905.

Ziermer, M.: Genealogische Studien über die Vererbung geistiger Eigenschaften (Konstanz der Familiencharaktere im Mannesstamme), nachgewiesen an einem Material von 1334 Waldauer Haushaltungen. Archiv f. Rass.- u. Ges.-Biol., 5, 178, 327, 1908.

PSYCHIATRY.

Alzheimer, A.: Ist die Einrichtung einer Psychiatrischen Abteilung im Reichsgesundheitsamt erstrebenswert? Zeitsch. f. d. ges. Neurol. u. Psych. Orig., 6, 242, 1911.

Ameline, De: L'hérédité, et en particulier de l'hérédité similaire dans la paralysie générale. Annales Med.-Psychol., Mai u. Juni, 1900.

Anton, G.: Alkoholismus und Erblichkeit. Psych. Wochenschr., Nr. 14, 1901.

Apert, E.: Maladies Familiales et maladies congenitales. Paris, 364 S., 1907.

Arnaud, F. S.: Les Maladies mentales familiales. Revue de Psychiatrie, n. s., III, Paris, 1900.

Benedikt, Degenerationskrankheiten des Nervensystems. Deutsche med. Wochenschr., Nr. 35, 1906.

Berze, J.: Die Hereditaren Beziehungen der dementia præcox. Leipzig-Wien, 1910.

——— Die Manisch-depressive Familie H. Beitrag zur Hereditatslehre. Montasschr f. Psych., 26, 270, 1909.

Bing. R.: Die Heredofamiliaren Degenerationen des Nervensystems in erblichkeitstheoretischer allgemeinpathologischer, rassen-biologischer Beziehung. Med. Klin., 1906.

——— Heredofamiliare Nervenkrankheiten. Deutsches Archiv f. klin. Medizin, 83, H. 3 u. 4, 1905.

Bischoff, E.: Ueber Familiäre Geisteskrankheiten. Jahrb. f. Psych., S. 109, 1905.

Blin, Contribution à l'étude de l'hérédité dans les états mentaux de l'enfant. Congr. internat. de med. C. r. Paris 1900, sect. de psych., 522.

Bolton, Josef Shaw: Evidence as to the existence of heredity of insanity and of a parental and family degeneracy in the subjects of dementia paralytica. Journ. of mental Science, 54, 26, 1908.

Bourneville: Note Statistique sur le rôle de la consanguinité dans l'étiologie de l'épilepsie, de l'hystérie, de l'idiote et de l'imbécillité. Progr. Med., 4, Mai, 1901.

Brachet, A.: Pathologie mentale des rois de France, Louis XI et ses ascendants. Paris, 1903.

Bratz, Ueber Vererbung. Vortrag in Psychiatrischen Verein zu Berlin, Sitzung vom 18. Dezember, 1909. Mit Diskussion. Neurol. Centralbl., Nr. 2, 1910.

Burnet, Anne: A study of heredity among the women in the State Hospital at Clarinda, Iowa. Womans M. J., Toledo, 9, 375-380, 1900.

Buscham G.: Bedeutung der Verwandtschaftsheiraten für die Nachkommenschaft. Neuland des Wissens, 1, 1910.

Cannon and Rosanoff: Preliminary report of a study of heredity in insanity in the light of the Mendelian laws. Journ. of Nervous and Mental Diseases, 38, 272, 1911.

Cox, W. H.: Degeneratie, eene copulativogene correlatiestoornis. Psychiatrische en Neurologische Bladen, 1907, No. 1, 77 S., 8°, 6 tafeln. Ref. im. Archiv f. Rass.- u. Ges.-Biol., 1908, S. 125.

Dana, Charles F.: The modern views of heredity with the study of a frequently inherited psychosis. Medical Record, 77, 245. 1910.

Diem, Otto: Die Psycho-neurotische erbliche Belastung der Geistesgesunden und der Geisteskranken. Archiv f. Rass.- u. Ges.-Biol., 2. 215-252 u. 336-368. 1905.

Doran: A consideration of the hereditary factors in epilepsy. Amer. Journ. of Insanity, 60, 1903.

Elmiger: Beiträge zum Irresein bei Zwillingen. Psych.-Neurol. Wochenschr., Nr. 8, S. 78; Nr. 9, S. 85; 1910-1911.

Ermakow, J.: Observations Personelles sur les Psychoses alcooliques et l'hérédité alcoolique chez les battants de la guerre russo-japonnaise. Arch. intern. de neurol., 1909; ref. Neurol. Centralbl., 1910, S. 1357.

Etchepare, B.: Folie Familiale, délire d'interprétation antilogique communiqué entre sept personnes. Annales méd.-psychol., 68, 5, 1910.

Fauser, Endogene Symptomenkomplexe bei exogenen Krankheitformen. Allgem. Zeitschr. f. Psych., 62, 165.

Fitschen, Eleanore: Die Beziehung der Heredität periodischen Irresein. Monatssche. f. Psych., 7, 127, 224. Berlin, 1900.

Fitzgerald, F. G.: A study of phases of family psychosis. Bull. of the Ontario Hospitals for the Insane, 2, 23, 1908.

Foerster, Ueber die Klinischen Formen der Psychosen bei direkter Erblichkeit. Mit Diskussion. Allgem. Zeitschr. f. Psych., 64, H. 1, S. 176, 1907.

Fornaca: Sull influenza dell-ereditarietà morbosa nella paralisi Progressiva. Rivista sperim. di freniatria, 33, Fascic II, III, 1907.

Foque, P.: Maladies mentales familiales. Paris, 1900.

Frankhauser, K.: Geschwisterpsychosen. Zeitschr. f. d. ges. Neurol. u. Psych., Orig. 5, 52, 1911.

Fuller, L. O.: Alcoholism, crime and insanity. Journ. of Mental Science, 55, 692, 1909. Aetiology (heredity), S. 700.

Galippe, V. L.: L'hérédité des stigmates de dégénérescence et les familles souveraines. Paris, 1905, Masson et Cie. et Gaz. des hôp., S. 903, 1905.

——— Hérédité similaire. Revue de méd., No. 11 et 12, 1901.

Goddard: Heredity of feeble-mindedness. Amer. Breeders Magazine, 1, Nr. 3, S. 165-178, 1910, Washington, D. C.

Goring, C.: On the inheritance of the diathesis of phthisis and insanity; a statistical study, based upon the family history of 1500 criminals. Drapers Company Research memoirs, Department of Applied Mathematics. University College, London; published by Dulau & Co., 3 shillings, 1909.

Gottgetreu: Die Bedeutung der Vererbung bei der Dementia paralytica. Inaug.-Diss. Jena, 1902.

Gowers: Heredity in diseases of the nervous system. Brit. med. Journ., 1908.

Graeter: Dementia præcox mit alcoholismus chronicus. Atiologie, S. 178 u. f. Leipzig, 1909.

Hahnle, E.: Der Heutige Stand der Erblichkeitsfrage in der Neuropsychopathologie. Neurol. Centralbl., Nr. 18, S. 843, 583, 892, 1904.

Hartmann: Ueber die hereditären Verhältnisse bei Verbrechern. Monatsschr. f. Kriminalpsychol., I. Jahrg., S. 493, 1904.

Hartzell, W. H.: The burden of hereditary mental diseases. Lehigh Valley, M. Mag., Easton, Pa., 12, 21-27, 1901.

Hermann: Paralytikerkinder. Münch. med. Wochenschr., Nr. 20, S. 1025, bis 1027, 1909.

Heron, David: A first study of the statistics of insanity and the inheritance of the insane diathesis. No. 2. der Eugenics Laboratory Memoirs, of Francis Galton Laboratory for National Eugenics. London, 1907. Dulau & Co., 33 S.

Hirschl, Jos.: Adolph, Zur Aetiologie der Dementia præcox. Vortrag, gehalten im Verein f. Psych. u. Neurol. in Wien am 12. März, 1907. Jahrb. f. Psych.

Hoche, A.: Zur Frage der erblichen Belastung bei Geisteskrankheiten. Med. Klin., Nr. 18, S. 427, 1905.

——— Kultur und Geisteskrankheit. Freiburg i. Br., 1910.

Jakowenko, W.: Erblichkeit bei Geisteskrankheit. 1904, H. ½ des Journ. neuropathol. i. psych (Organ der Neuropathol. Gesellsch zu Moskau). 1905, S. 683. Ref. in Centralbl. f. Neurol. u. Psych., S. 683, 1905.

Jeffrey, G. R.: The significance of heredity and the neuro-insane constitution as important factors in the production of mental disease. Journ. of ment. Sc., Vol. 56, 273-196, April, 1910.

Jendrassik, Ernst: Beiträge zur Kenntnis der hereditaren Krankheiten. Deutsche Zeitschr. f. Nervenheilk, 22, H. 5. u. 6, 1902.

Die Hereditären Krankheiten. Lewandowskis Handb. d. Neurol. 2, 321. Berlin, 1911.

Joerger, J.: Die Familie Zero. Archiv f. Rass.- u. Ges.-Biol., 2, 494, 1905.

Jones, W. H. S.: Ross R. und Ellett, G. G.: Malaria, a neglected fact in the history of Greece and Rome. Cambridge, 1907.

Kalmus, Untersuchungen über erbliche Belastung. 6. Jahresversamml. Nord-deutsch. Psychiater u. Neurologen. Centralbl. f. Nervenheilk. u. Psych., S. 75, 1905.

Karpas und Morris, J.: Contribution to our knowledge of the aetiology of Dementia præcox. New York Med. Journ., 5 u. 12, Dez., 1908.

Karplus, J. P.: Zur Kenntnis der Variabilität und Vererbung am Zentralnervensystem des Menschen und einger Säugetiere. Leipzig u. Wien 1907.

Kekule v. Stradonitz: Die Untersuchung von Vererbungfragen und die Degeneration der spanischen Habsburger. Archiv f. Psych., 35, H. 3, 1902.

Kirchhoff: Fragen aus dem Gebiete der Erblichkeit. Allgem. Zeitschr. f. Psych., 56, 6, S. 871.

Knapp, Philip Coombs: Heredity in diseases of the nervous system with especial reference to heredity in epilepsy. Read at the meeting of the Amer. Neurol. Assoc. at Washington, 8. May, 1907.

Kneidl, C. (Prag): Beiträge zur Statistik und Aetiologie der Epilepsie (Casopis ces lék Prag, 1904). Ref. Neurol. Centralbl., S. 220, 1905.

Koch, J. L. A.: Die Erbliche Belastung bei den Psychopathien. Kinderfehler, 8. Jahrg., 1903, H. I.

König, W.: The problem of heredity from the psychiatrical aspect. Brit. med. Journ., Oct. 15, No. 2285.

Kraepelin, Emil: Zur Entartungsfrage Centralbl. f. Nervenheilk. u. Psych., 2. Oktoberheft, 1908.

Krauss, W. C.: Heredity with study of the statistics of the New York State hospitals. Buffalo, M. J., n. s. 41, 892-899, 1901-1902.

Krauss, Kenneburg: Ueber Vererbung von Geisteskrankheiten. 23 Versamml. d. sudwestdeutsch. Irrenarzte in Stuttgart am 1. u. 2. Sept., 1902. Allgem. Zeitschr. f. Psych., S. 224, 1903.

Kreichgauger, Rosa: Zur Frage der Vererbung von Geisteskrankheiten. Inaug.-Dis., Freiburg i. Br., 1909, u. Centralbl. f. Neurol. u. Psych., S. 877-885, 1909.

Lagriffe, L.: Recherches sur l'hérédité dans les maladies mentales. Archives d'anthropolog. criminelle et méd. légale, 1910.

Lange Fe, Middelfart, Dänemark: Degeneration in Families: Observations in a lunatic asylum. Engl. Uebersetzung, London, 1907. 8°, 3s. 6d. net.

——— Slaegter, Jagttagelser fra en Sindsygeanstalt. Kopenhagen, 1904. 146 S., 3 Kr.

Levi-Branchini, Marco: Sull' età di comparsa e sull' influenza dell' ereditarietà nella patogenesi della demenza primitiva o precoce. Rivista sperim. di freniatria 29, Facs. 3.

Lewis, W.: Alcoholism, crime and insanity. Journ. of Mental Science, 52, 203, 1906. Heredity, S. 212.

Liepmann: Psychose der Mutter und Psychose der Tochter, mit Krankenvorstellung. Sitzung d. Psychiatr. Verein zu Berlin vom 24. Juni, 1905. Allgem. Zeitschr. f. Psych., S. 841, 1905.

Lomer, Weibliche Trinker und ihre Nachkommenschaft: Pol. Anthr. Rev., 1907.

Lui, A.: Eredità ed Alcoolismo. Annali di Nervrologia, 18. Napoli, 1900.

Lundborg, Hermann: Ueber Geschlechterforschungs- und Erblichkeitshygiene. Allgem. Zeitschr. f. Psych., S. 175, 1906.

——— Om släktforskning och ärftlighets hygien. Upsala Läkaref förhandl., 1905. Ref. in Centralbl. f. Nervenheilk. u. Psych., 5, 660, 1906.

McGugan, A.: The importance of heredity as a cause of insanity. Journ. of the Amer. Med. Assoc., Chicago, 38, 371-376, 1902.

——— Heredity and the investigation of insanity. Amer. Med. Philad., 2, 166, 1901.

MacDonald, P. W.: Medico-Psychological Association of Great Britain and Ireland, Presidential Address delivered July 25, 1907, by MacDonald. Journ. of Mental Science, 1907, S. 667. Heredity, S. 695.

MacDonald, C. F.: The etiological potency of heredity in mental diseases. Philad., M. J., 8, 739-741, 1901.

Marandon de Montyel: Prédisposition et causes directes en étiologie mentale. Revue d. Psych., S. 115, 1905.

Marc: Ueber Familiäres Auftreten der progressiven Paralyse. Allgem. Zeitschr. f. Psych., 61, 5, 660, 1904.

Marioni: Contributions à l'etude l'hérédité chez les paralytiques généraux. Thèse, 1900.

Mendel, G.: Geisteskrankheiten und Ehe. In: Krankh. u. Ehe (Senator u. Kraminer), München, S. 642, 1904.

Merzbacher, L.: Gesetzmässigkeiten in der Vererbung und Verbreitung verschiedner hereditar-familiarer Erkrankungen (mit 19 Fig. u. 2 farb. Tafeln). Archiv f. Rass.- u. Ges.-Biol., S. 172-198, 1909.

Meyer: Die Ursachen der Geisteskrankheiten. Jena, 1907.

Moebius, P. J.: Ueber Entartung. Wiesbaden, 1909, Bergmann.

Mondio, G.: Eredita e dengenerazione nello sviluppo della demenza consecutiva ed in quello delle stereotipie riscontrate in essa. Merita la demenza consecutiva un capitolo a sé in nosografia mentale? Rivista mensile di Neuropat. e. Psych., No. 4-5, 1900.

Mönkemöller: Eine Vagabundenfamilie. Arch. f. Kriminalanthropol. 1907.

Mott, F. W.: The hereditary aspects of nervous and mental diseases (Huxley lecture). The Lancet, S. 1057-1064. October 8, 1910.

Muller, Herm.: Einige Beziehungen des Alkoholismus zur Aetiologie der Epilepsie. Monatsschr. f. Psych. u. Neurol., 28, Erg. Heft, S. 1, 1910.

Munson, J. F.: The rôle of heredity and other factors in the production of traumatic epilepsy. Epilepsia, Vol. 2, 343.

Naecke, P.: Erblichkeit und Prädisposition resp. Degeneration bei der progressiven Paralyse der Irren. Archiv. f. Psych., 41, 295-366, 1906.

——— Die Rolle der erblichen Belastung bei der progressiven Paralyse der Irren. Neurol. Centralbl., Nr. 16, 1900.

——— Das prozentual augedrückte Heiratsrisiko bez. Ausbruchs und Vererbung von Geistes- und Nevenkrankheiten. Allgem. Zeitschr. f. Psych., 63, 3. u. 4. H., S. 482-505, 1906.

Naegeli-Ackerblom: Medikohistorischer Beitrag zur Frage der erblichen Belastung. Therap. Monatsschr., H. 6, 1907.

Neisser, Clem.: Individualität und Psychose. Vortrag bei der Naturforscherversammlung, 1905.

Nina-Rodrigues: Atavisme psychique et paranoia. Arch. d'anthrop. crim., Lyon et Paris, 17, 325-355, 1902.

Obersteiner, Heinr: Zur Frage der hereditären Uebertragbarkeit zierterrierter pathologischer Zustände. Neurol. Centralbl., Nr. 11, S. 498, 1900.

Pearson, Karl: On the inheritance of the mental and moral characters in man, and its comparison with the inheritance of the physical characters. Journal of the Anthropological Institute of Great Britain, 33, 179-237, 1903.

——— Inheritance of want of mental balance. Brit. Med. Journ., 1905 (1).

Peipers, Consanguinität in der Ehe und deren Folgen für die Deszendenz. Allgem. Zeitschr. f. Psych., 58, H. 5, S. 793-862, 1901.

Pelletin, M.: L'hérédité Biologique et l'hérédité psychologique. La Médecine moderne, 1905.

Petró, F.: Della pazzia analogo o gemellare. Annali di freniatria e sc. affini, 20, 142, 1910.

Pieraccini: Ulteriore contributo allo studio delle leggi che regolano la ereditarietà psicopatica. Rivista sperim. di freniatria, 28, 326-330. Reggio-Emilia, 1902.

Pilcz, Alexander: Beiträge zur Lehre der Heredität. Festschr. z. Feier. d. 25 jahr. Bestandes d. Neurol. Institutes a. d. Wiener Universitat, herausg. v. Dr. Otto Marburg, 1. Teil, 1, 282-309. Leipzig u. Wien, 1907.

Plaut und Göring: Untersuchungen an Kinder und Ehegatten von Paralytikern. Münch. med. Wochenschr., S. 1959, 1911.

Rehm, P.: Die Ergebnisse der Untersuchung von Kindern manisch-depressiver Kranker. Zeitschr. f. d. Erforsch. u. Behandl. d. jugendl. Schwachsinns, 3, 1909.

Reid, C. A.: Alkoholism, a study in heredity. London, 1901.

——— Principles of heredity with some applications. London, 1905.

Reiss, Eduard: Konstitutionelle Verstimmung und manisch-depressives Irresein. Berlin, J. Springer, 1910. Vererbung, S. 255-261.

Ris, F.: Vorstellung von Familiengruppen Geisteskranker. Korrespondenzbl. f. Schweiz Arzte, 32, 447-449. Basel, 1902.

Römer, L. S. A. M.: Beiträge zur Erkenntnis des Uranismus. Heft 1: Die uranische Familie, Untersuchungen über die Aszendenz der Uranier. Leipzig, Amsterdam, 107 S., 1906.

——— Die erbliche Belastung des Centralnervensystems bei Uranier, geistiggesunden Menschen und Geisteskranken. Jahrb. f. sexuelle Zwischenstufen, 7. Jahrg., 1, 67. Leipzig, 1905.

Roemer, Hans: Zur Symptomatologie und Genealogie der psychischen Epilepsie in der epileptischen Anlage. Allgem. Zeitschr. f. Psych., 67, 588-627, 1910.

——— Eine Stammliste aller amtlich bekannt werdenden Fälle von Geisteskrankheit. Psychiatr. Neurol. Wochenschr., 13. Jahrg., 1911, H. 10, S. 94.

Rogue de Fursac, J.: L'hérédité dans l'avarice. Journ. de Psychol. norm. et Pathol., Mai-Juni, 1909.

Rohde: Das Vererbungsproblem in der Neuro- und Psychopathologie. 79. Versaml. Deutsch. Naturf. u. Ärzte in Dresden vom 15-21 Sept., 1907. Neurol. Centralbl., S. 972, 1907.

Roubinowitsch et Phulpin: Contribution à l'étiologie du syndrome de la démence précoce. Annales méd-psychol., Juni, 1903.

Roubinowitsch, J., et Levaditi: Le rôle de la syphilis dans l'étiologie de la démence précoce. Gazette des Hôp., Juni, 1909.

Rüdin, Ergänzende Bemerkungen zu Otto Diems Artikel: Der Psychoneurotische, erbliche Belastung der Geistesgesunden und der Geisteskranken. Archiv f. Rass.-Ges.-Biol., 2, 470, 1905.

Rybakow, Th.: Alkoholismus und Erblichkeit. Monatsschr. f. Psych. u. Neurol. 20, Erh.-Heft, S. 221-234, 1906.

Sandy, William C.: Studies in heredity with examples. Amer. Journ. of Insanity, 66, 587-589, 1910.

Sanger-Brown: Heredity and epilepsy. The Alienist and Neurologist, July 1901.

Schloss: Zur Kenntnis der Aetiologie der Angebornen und frühzeitig erworbenen psychischen Defektzustände. Psych.-Neurol. Wochenschr., Nr. 48-50, 1907.

Schlub, H. O.: Ueber Geisteskrankheit bei Geschwistern. Allgem. Zeitschr. f. Psych., 66, 3. u. 4. H., S. 514-541, 1909.

Schüle: Ueber die Frage des Heiratens von früher Geisteskranken. Leipzig, 1904, S. Hirzel.

——— Ueber die Frage des Heiratens von früher Geisteskranken II. (Geisteskrankheit und Ehe). Anlegung der statistischen Tabellen über Erblichkeit. Klinische und biologische Fragestellungen. Genealogische Stammbäume von 20 Zyklischen Geisteskranken (mit Tabelle). Vorschläge zur Prophylaxe. Mit 11 Tafeln. Berlin, 1905. G. Reimer, 46 S.

——— Nochmals das Heiraten von früher Geisteskranken. Ref. Psych.-Neurol. Wochenschr., Nr. 100, 1904.

——— Vorlage eines Bogens für Festellung der Erblichkeit. Jahresversamml. d. Deutsch. Vereins f. Psychiatrie in Dresden am 28. u. 29. April, 1905. Neurol. Centralbl., S. 484, 1905.

Schwarzwald, Bernard: De la démence précoce familiale. Thèse de Doctorat. Lausanne. Imprimerie Cosmopolite. Pré-du-Marché 45, 1907.

Semelaigne, René: Family mental diseases. Journ. of Mental Science, S. 150, 1901.

Sichel, Max: Ueber die Geistesstörungen bei den Juden. Neurol. Centralbl., Nr. 8, S. 351, 1908.

——— Der Alkohol als Ursache der Belastung. Neurol. Centralbl., 29, 738-748, 1910.

Sklarek und van Vleuten: Gleichzeitige geistige Erkrankung bei 3 Geschwistern. Allgem. Zeitschr. f. Psych., 61, 5.

Sommer, R.: Psychiatrische Untersuchung eines Falles von Mord und Selbstmord mit Studien über Familiengeschichte und Erblichkeit. Sommers Klinik f. psych. u. nervöse Krankh., 1, 1, S. 55.

——— Die Beziehungen zwischen Psychologie, Psychopathologie und Kriminal-psychologie vom Standpunkte der Vererbungslehre. Vortrag, gehalten in der Gesellsch. f. soz. Medizin usw. am 21, Februar., 1909, in Berlin, nebst Diskussion. Bibl. f. soz. Medizin usw., H. 3, Berlin, 1910. Allgem. Verl.-Anstalt., 30 S.

——— Zur Theorie der Verwandtenehen und des Ahnenverlustes bei Menschen und Tieren. 5, 4. H. Halle a. S., 1910.

——— Familienforschung und Vererbungslehre. Leipzig, 1907.

——— Bermerkungen zu einem Fall von ererbter Sechsfingerigkeit. 5, 4. H. Halle a. S., 1910.

——— Psychiatrie und Familienforschung. Allgem. Zeitschr. f. Psych., H. 2-3, S. 463, 1907.

Stansfield, T. E. K.: Heredity and Insanity. Journ. of Ment. Sc., January, 1911.

Stelzner, H. F.: Die psychopathische Konstitution und ihre soziologische Bedeutung. Berlin, 1911. S. Karger. Aetiologische Momente, S. 13-74.

Strohmayer, W.: Ueber die Bedeutung der Indiviualstatistik bei der Erblichkeitsfrage in der Neuro- und Psychopathologie. Münch. med. Wochenschr., 48, 1786, 1842. 1901.

——— Die Ahnentafel der Konige Ludwig II. und Otto I. von Bayern. Ein genealogisch-psychiatrischer Deutungsversuch. Archiv f. Rass.- u. Ges.-Biol., S. 65, 1910.

——— Ueber den Wert Genealogischer Betrachtungsweise in der psychiatrischen Erblichkeitlehre. Monatsschr. f. Psych. u. Neurol., 22, Erg-Heft, S. 115, 1907 (Festschrift für Otto Binswanger).

——— Zur Kritik der Feststellung und der Bewertung psychoneurotischer erblicher Belastung. Archiv f. Rass.- u. Ges.-Biol., 5, 478, 1908.

Sutherland, J. F.: Recidivism. Part II. Journ. of Mental Science, 1907, S. 568. Heredity, S. 584.

——— Recidivism. Part III. Journ. of Mental Science, 1908, S. 68. Biometrics and National Eugenics, S. 71, Degeneracy, S. 72. Causation of Rec., S. 73.

Thompson, W. N.: Heredity and the transmission of the insane diathesis. Vermont M. Month. Burlington, 325-337, 1900. Also Journ. Med. n. Sc. Portland, 7, 337-342, 1900-1901.

Tigges, W.: Die Abnormitäten der Aszendenz in Beziehung zur Deszendenz. Allgem. Zeitschr. f. Psych., S. 891, 1907.

——— Die Gefährdung der Nachkommenschaft durch Psychosen, Neurosen und verwandte Zustände der Aszendenz. Allg. Zeitschr. f. Psych., 63, 448-481, 1906.

——— Untersuchungen über die erblich belasteten Geisteskranken. Allgem. Zeitschr. f. Psych., 64, H. 1, S. 47 S., 1907.

Tiling, Th.: Individuelle Geistesartung und Geistesstörung. Wiesbaden, 1904.

——— Zur Aetiologie der Geistesstörungen. Centralbl. f. Nervenheilk. u. Psych., Sept., S. 561, 1903.

Tredgold, A. F.: Remarks on the subsequent history of children born whilst that mother was insane. Lanset, 1, 1380-1385, London, 1902.

Trenel: Maladies mentales familiales. Annales méd-Psychol., Paris, 8 S., 11, 96-107, 1900.

Urquhart: The Morison lectures. On insanity, with special reference to heredity and prognosis. Journ. of Mental Science, April, Nr. 221, S. 233, 1907.

Vogt, R.: Om Arvelighed. Separataftryk af Tidsskrift for Nordisk Retsmedicin og Psykiatri.

Vorster: Ueber die Vererbung endogener Psychosen. Monatsschr. f. Psychoatrie, 9, 1901.

——— Zur Erblichen Uebertragung der Geisteskrankheiten. Diskussion. Allgem. Zeitschr. f. Psych., S. 493, 1901.

Voss: Ueber die Bedeutung der Entartung un der Psychiatrie und Neurologie. Verhandl. d. Med. Vereins zu Greifwald, Jahrg., 1909, S. 44, Leipzig, Georg Thieme, 1910. Also, Deutsche med. Wochenschr., 1909-1910.

Wagner v. Jauregg: Ueber Erbliche Belastung. Antrittsvorlesung. Wiener Klin. Wochenschr., Nr. 44, 1902.

——— Einiges über erbliche Belastung. Wiener klin. Wochenschr., Nr. 1, 1906.

Weinberg, W.: Verwandtenehe und Geisteskrankheit. Archiv. f. Rass.- u. Ges.-Biol., S. 471-475, 1907.

——— Statistik und Vererbung in der Psychiatrie. Klinik. f. psych. u. nervose Krankh., 5, H. 1, 1910.

Wiglesworth, J.: The Presidential Address delivered at the 61st annual meeting of the Medico-Psychological Association, held in Liverpool, on July 24, 1902. Heredity and Insanity. Journ. of Mental Science, S. 611-645, 1902.

Wille: Ueber Erbliche Uebertragung von Geisteskrankheiten. Korrespondensbl. f. Schweiz. Arzte, S. 603, 1902.

Wlassak, R.: Das Problem der Degeneration. Vortrag, gehalten in Akadem. Abstinensverein Wien Aus: Internat. Monatschr. z. Erforsch. d. Alkoholism n. Bekampf. f. Trinksitten. 20. Jahrg., H. 3, März, 1910.

Wolfsohn, Ryssia: Heredität bei Dementia Præcox. Allgem. Zeitschr. f. Psych., 64, 347 ff., 1907.

Woods, Frederick Adams: Mental and moral heredity in royalty. A statistical study of history and sociology. New York, 1906. 12, VIII, 312 S. ill.

Word, J. M. S., und Urquhart, A. R.: A family tree illustrative of insanity and suicide. Journ. of Mental Science, Oktober, S. 764, 1901.

Cannon, L. Gertrude, A. M., and Rosanoff, J. A.: Preliminary report of a study in insanity in the light of the Mendelian laws. Eugenics Record Office. Bull. No. 3. (Reprint from Journ. Nervous and Mental Disease, Vol. 38, No. 5, 1911.)

Davenport, B., Chas. Laughlin, H. H. Weeks, F. David, Johnstone, E. R., Goddard H. Henry: The Study of Human Heredity. Eugenics Record Office. Bull. No. 2.

Davenport, C. B.: The Trait Book. Eugenics Record Office. Bull. No. 6.

Davenport, C. B., and Weeks, F. David, M. D.: A first study of inheritance in epilepsy. Eugenics Record Office. Bull. No. 4. (Reprint from Journ. Nervous and Mental Disease, Vol. 38, No. 11, 1911.)

Goddard, H. Henry, Ph. D.: Eugenics Record Office, Heredity of Feeblemindedness. Bull No. 1.

Mott, F. W.: Inborn Factors of Nervous and Mental Diseases. Brain, Parts II and III, Vol. XXXIV, Nov., 1911.

Rosanoff, A. J., M. D., and Orr, I. Florence, B. S.: A study of heredity in insanity, in the light of the Mendelian theory. American Journ. of Ins., Vol. LXVIII, No. 2, Oct., 1911. Eugenics Record Office, Bull. No. 5.

Rüdin, E.: Wege und Ziele Familienforschung, mit Rücksicht auf die Psychiatrie. Zeitschrift für die Gesammte Neurologie und Psychiatrie, 7 Band, 5 Heft, Nov. 18, 1911.

COMPARATIVE PSYCHOLOGICAL STUDIES OF THE MENTAL CAPACITY IN CASES OF DEMENTIA PRÆCOX AND ALCOHOLIC INSANITY

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[Reprint from "Studies in Psychiatry," Vol. I,
New York Psychiatrical Society.]

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Scope and Object of the Investigation

The work of the investigators in the field of experimental psychology in recent years has shown remarkable progress, so that new light has been shed upon a great many obscure and complicated problems connected with our mental life.

Mere speculation as to the mechanism of fundamental psychical processes has given place to some extent to more accurate knowledge of these problems, so that today many obscure and complicated reactions can be graphically demonstrated and these same processes spoken of in terms of mechanical values and equivalents. To reduce these complicated processes to figures and curves has been a stupendous task in the field of normal psychology, and much remains to be accomplished before we can have an accurate idea of all psychical phenomena.

That the experiments with normal individuals have not been entirely successful is due partly to a lack of methods, and partly to defects in some of the methods devised. If this be true of the field of normal psychology, then we cannot wonder that investigators in the field of abnormal psychology have experienced great difficulties in representing by graphic methods the complicated abnormal psychical processes in the insane.

The effects of drugs and poisons upon the psychical processes have been graphically shown by Kraepelin and his pupils, and their work has been of immense importance in paving the way for a better understanding of abnormal psychological phenomena.

But experiments with normal individuals do not present such obstacles as we find when we attempt to analyze abnormal psychical processes of the insane by similar methods. The normal processes with and without the effect of the poisons can be carefully analyzed and compared in the same individual, whereas, in the experiments in abnormal psychology, we must compare similar experiments upon normal individuals with the results in abnormal individuals or the insane.

Methods that are readily adapted to the normal individual become useless when applied to investigations of abnormal mental conditions as found in the insane. Either the patients absolutely refuse to perform the experiments, or only partially fulfill the conditions, and the figures and results of such experiments are not comparable with similar experiments with the normal. And such figures seldom give an accurate and clear picture of the pathological mental condition.

We have studied the various clinical symptoms of the insane, and we know that certain symptoms refer to disturbances of certain psychical fields. The problem of experimental psychology is to measure these abnormal reactions and compare the results of such measurements with those obtained in normal psychical processes.

For many years Kraepelin has searched for simple methods which were suitable for abnormal as well as normal persons, and he and his pupils have been successful in finding methods that on the one hand were suitable for studying the psychical processes of normal persons, while on the other hand, they were adaptable to abnormal psychical processes as well.

His method of continuous addition of single numbers was first used in experiments with normal individuals, and occupied two hours a day continuously for its completion. By this simple method he succeeded in graphically representing some very important psychical processes, and also the relation of various processes to each other, i. e., fatigue—the effect of the rest or pause during addition, in which recuperation takes place; the effect of stimulation; the impulse and intensity of the will, and variations in the will; and the increase of actual work done through practice and familiarity with the work. Kraepelin has

shown this in his monograph on the "Arbeitscurve,"[1] and in one chart all these various processes are plotted, and can be graphically compared. Kraepelin admits that it took ten years of experimenting with normal individuals before he could interpret all the facts shown by the "Arbeitscurve." Gradually Kraepelin shortened the time from two hours to one hour, then half an hour, until finally the experiment was reduced to ten minutes. It was soon apparent that two hours continuous addition was very fatiguing to a normal individual and wholly unsuited to abnormal ones.

Through a series of experiments extending over these ten years it was found that the length of time in the various experiments had little to do with the relation of the various phenomena; that certain laws were uniform, whether the experiment was for two hours or ten minutes, and that the relation of these phenomena was shown to be constant.

The method used in investigations by the author of this paper is practically the original method reduced to ten minutes work and simplified. And in spite of the simplicity and crudeness of the method, it has been used with success to show at least some of the abnormal psychical processes of the insane.

The object of this work was to investigate two forms of psychoses by this method, i. e., dementia præcox and alcoholic psychoses, and to compare the results obtained, with the results of the experiments on normal individuals of the same station in life and grade of intelligence. The method is simply a continuous addition of single numbers. These numbers are placed in long columns, and the task of the patient is to add consecutive numbers together and place the result opposite these numbers. This continues for ten minutes, and at the end of each minute, by a signal, the patient makes a line to indicate the same. The experiment lasts ten days. On alternate days a five minutes' pause or rest is given, and on other days the patient adds for ten minutes without any pause or rest. Then the number of additions during each minute is taken as units of the curve. Experience has shown that the amount of time lost in writing the result is very small, and extends uniformly over the entire experiment, and need not be

[1] Die Arbeitscurve, Emil Kraepelin, Leipzig, 1902.

taken into account, also, that mistakes made need not be considered, as the task is so simple that any one who has a rudimentary education in arithmetic can undertake the experiment.

This investigation was begun in the Psychological Laboratory of the Royal Psychiatric Clinic in Munich in the spring of 1906. The patients for investigation were partly from the Psychiatric Clinic in Munich, and partly from the District Insane Hospital at Egelfing, in the vicinity of Munich. The experiment with the patients is a comparatively simple matter, but calculating the results and interpreting the same is extremely difficult, and to Professor Kraepelin should be given the credit for a large share of the work, for without his assistance in interpreting the results the experiment would have been worthless.

We have investigated altogether fourteen cases of alcoholic psychoses, among which were several cases of delirium tremens, alcoholic hallucinosis, and chronic alcoholic insanity. Also twelve patients with dementia præcox who were in the first stage of the disease, mostly the catatonic form. The figures for the *normal* were taken from the work of Drs. Plaut and Rehm of the Psychiatric Clinic in Munich, who investigated these normal cases in conjunction with similar work with manic depressive insanity and psychasthenia.

I am indebted to these men for furnishing me with figures of normal people, and for their assistance in preparing this work. I wish also to express my thanks for the courtesy of the assistants in the Clinic of Munich, as well as the assistants in the District Insane Hospital at Egelfing, who rendered much aid in providing suitable patients for these studies.

The Method in Detail

As we have mentioned before, the method consists in adding single figures together and placing the sum opposite the printed numbers. Thus, a page of printed figures is before the patient, arranged in long columns, about 50 figures to a column.

These experiments are performed every day for 10 days. On alternate days a rest of five minutes is allowed, so that the rest is indicated by a dotted line between the fifth and sixth minute (see Fig. 1).

Thus—

8	
9	17
3	12
4	7
6	10
2	8
5	7

the left hand figures being the printed figures, and the right hand figures the additions of consecutive numbers. A stop watch is held by the investigator, and at the end of every minute the patient is told to "mark," which he does, and goes on adding until the 10 minutes are finished.

The curves shown in Fig. 1, are made up as follows: The abscissa represents the minutes of work from 1 to 10 and the ordinate represents the total number of additions performed in each minute. The curve on the left "without pause," is made from the work on alternate days, first, third, fifth, etc., days, and the curve on the right "with pause," is made up from the work on the second, fourth, sixth, etc., days. These curves represent the average of each five days' work. To illustrate, one example of the work done on normal persons by Wilhelm Specht. Below are given the numbers for each minute in the ten days' investigation:

1		3		5		7		9	
63	54	75	66	73	69	75	73	78	75
56	52	69	67	71	66	70	71	73	71
58	56	64	68	69	67	70	72	75	66
53	52	68	66	70	68	72	74	73	66
54	52	68	62	73	68	73	70	71	65
284	266	344	329	356	338	360	360	370	343

2		4		6		8		10	
48	61	72	69	73	76	77	80	74	76
44	55	64	66	67	72	74	72	74	75
43	51	57	66	70	70	74	67	75	74
44	47	63	64	67	69	70	70	73	72
44	50	59	64	68	69	70	70	72	74
223	264	315	329	345	356	365	359	368	371

In the above table the vertical figures represent the number of additions in minutes, the first five minutes on the left of the vertical line, and the second five minutes on the right, and the successive days, as explained above. The average of these daily figures is shown in the two curves—in Fig. 1 we see in curve *b* (with pause) that there is a decided downward course of the curve, showing a drop from 42.5 to 38.7 at the end of the fifth minute. The cause of this sinking of the curve is due principally

to fatigue (mental) as the mental work progresses. But there is another factor to be considered, and that is practice. For practice without fatigue would show an increase in the amount of work done, and the curve would rise instead of falling. But the fatigue overbalances the practice and consequently the curve sinks.

After a five minutes' pause, it will be noticed that the work done in the sixth minute is not only much greater in amount than

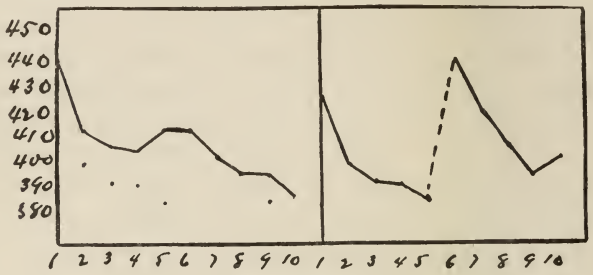

FIG. 1.

at the fifth minute (directly before the pause), but is also larger than the amount done at the beginning of the experiment, and consequently we find the curve beginning almost at 44 instead of 42 the first minute.

This increase in the amount of work done the sixth minute is explained by two facts which have taken place during the five minutes' rest or pause. In the first place the fatigue has been overcome by recuperation, and the person starts out freshened and ready to work. At the same time the effect of the practice of the first five minutes has some value in the amount of work done, being more than in the first minute. It is true that during the pause or rest, the practice has to a small degree lost its effect, but the disappearance of fatigue also must be considered in explaining the larger value of the work in the sixth minute. Especially is this seen in comparing the work of the sixth minute in curve *a*. Here the value is 41 against 44 for the same minute after a pause of five minutes. The work of the entire five minutes after the rest is much greater than the work of the first five minutes. Hence, we must ascribe the increase largely to the effect of recuperation during the rest, and the loss of fatigue.

From Table I we get the following values: The numbers on the left side of the line are the values of the fifth minute before the pause for five days. On the right side of the line are the values for the sixth minute following the pause for five days. The sum of these separate columns gives the total amount of work done in the fifth and sixth minute, with an intervening pause.

44	61
59	69
68	76
70	80
72	76
313	362 = 11.5 per cent. increase after the rest.

Here is shown distinctly the effect of the pause or rest in the 11.5 per cent. increase in work done in the sixth minute over that done in the fifth minute before the pause. When we compare the work done in the fifth and sixth minutes on the days with the pause with the same minutes on the days without the pause, we see in curve *a* that the fifth and sixth minutes are practically the same, that no increase can be noticed as compared with curve *b*. For the fatigue has been compensated in the latter, but not in the former instance. The curve shows in general a descending tendency due, as we stated above, to the overbalancing of the fatigue over the practice, but the fifth and sixth minutes are at the same level. The slight rise of the curve at the fifth minute is not due to a balance between the fatigue and practice, but to the effect of the tension, or straining of the will, which overcomes the fatigue. And the straining of the will frequently shows itself in the curves and is considered an impulse of the will to overcome the fatigue. This impulse is effective in keeping the amount done in the fifth and sixth minute at the same level. Without this straining of the will in the course of the work, both values of the fifth and sixth minutes would be much smaller and the curve would descend at this point as a result of fatigue. So that 11.5 per cent. increase after the pause stands for the direct effect of the pause, when taken with the percentage of increase on the days without the pause, thus:

11.5 per cent. increase after pause.
0.0 per cent. increase without pause.

11.5 per cent. increase after pause, or direct effect of pause.

We must also consider the total amount of work done before the pause and after the pause or the total work of the first five minutes must be compared with the total work of the five minutes after the pause, and also compare these percentages with those obtained in the same manner by comparing the same values of the days without a pause.

From the table we get the following figures, the left hand column representing the total work of the first five minutes for five days, and on the right side, the total work of the last five minutes after the pause.

223	264
315	329
345	356
365	359
368	371
1,616	1,679 = 3.3 per cent. increase of total work after the pause.

This increase after the pause is explained by the effect of the practice, a portion of which remains even during the rest, and therefore more work is accomplished in the last five minutes, although the fatigue also plays a part as shown by the descending curve. When we assume that during the pause the fatigue has been entirely recuperated by the rest, then the increase in work after the pause represents the practice coefficient of the individual.

Under what conditions, and in what manner the fatigue is compensated during the pause, at present we are unable to state.

Through the increase of the work after the pause, although a certain residual of fatigue is present, we must conclude that the practice has overcome to some extent the fatigue. The residual can be great, and at the same time hidden by the effect of practice when the latter is sufficiently great. And we can also conclude that possibly during the rest or pause, that the fatigue is entirely compensated, and no residual remains, and that the effect of practice is very small.

To come to a definite conclusion in regard to this question, we must first compare the work equivalents of the days when no rest was taken, and where consequently the effect of the rest does not come into play. From the table we again take the following

figures. The figures on the left represent the total work of the first five minutes for five days, and on the right of the column, the total work for the last five minutes for five days.

284	366
344	329
356	338
360	300
370	343
1,714	1,636 = 4.1 per cent. decrease in last five minutes over first five minutes.

In other words, without the good effect of the rest, the fatigue shows itself, and the work fell off 4.1 per cent. But from this coefficient alone we cannot compute the effect of fatigue. In the second five minutes work the fatigue overcomes everything else, but we cannot tell to what extent the total amount of work has been influenced by the simultaneous opposing effect of the practice.

It is possible that the decrease of the work in the second five minutes might be much less, if the individual was capable of improving by practice. We get an idea of the importance of the fatigue when we compare the differences in the total amount of work done after the pause and without the pause.

That the work of the second five minutes on the days without and with a pause or rest, approach each other as regards the effect of practice, can be shown. However, where recuperation of the fatigue during the pause has taken place, the comparison of days without any rest (where the second five minutes is under the influence of the fatigue) and the days with rest, the difference in the two series will give us the value of the fatigue. This difference is shown below.

3.3 per cent. increase in total work after pause.
4.1 per cent. increase in total work last five minutes.
7.4 per cent. coefficient of fatigue.

For computing the individual fatigue we make use of two groups of figures obtained by this method from the same individual. On one hand the relation of work values of the fifth and sixth minutes, and on the other hand, the difference in the total

amount of work done in the first and second five minutes on the days with and without pause. We will see later, when discussing fatigue in our three groups, how the coefficient of fatigue is vastly different in dementia præcox from normal individuals and alcoholics.

Comparison of Individual Work Curves

In Fig. 2 we have shown a typical curve from the groups investigated and compared with a normal curve.[2]

The curves on the left each represent the average of five days work for ten minutes per day uninterrupted. The curves on the right represent the average of five days work with a pause of five minutes after the fifth minute of work. This period is represented by the dotted line. The curve of the following five minutes represents work after the pause.

In the normal curve *a-I* it will be seen that there is a gradual tendency of the curve to descend, although in places it appears almost horizontal. However, the values do show that there is a decrease in amount of work done in successive minutes, but in tenths so that it cannot be accurately shown in the curve. There is a certain regularity to the curve, when compared to that of dementia præcox. Also in curve *a-II* (normal), the effect of the pause is distinctly shown, for after the pause, represented by the dotted line, the curve begins at a much higher point than the fifth minute, and a trifle higher than the beginning of the curve. This curve also shows the gradual decline in the amount of work done, both before the pause and after the pause, although the entire curve after the pause is distinctly higher than the curve of the first five minutes. As we said above, this is due to the loss of fatigue during the rest, and also to the residual practice, which has shown itself in the period following the rest.

Comparing this curve of a normal individual with curve *b*, that of a dementia præcox case, the difference between the two is at once apparent. In the first place one sees tremendous variations in the curve *b-I*. The curve sinks very low at the fifth minute,

[2] Curve taken from figures of a normal person given by Wilhelm Specht in Über klinische Ermüdungsmessungen, Archiv für die Gesamte Psychologie, Band III, Heft 3.

then rises again and ends much higher. This can only be accounted for by the variations in the intensity and impulse of the will, of which we will speak more in detail later. When we observe the curve *b-II,* representing the period with a rest, we

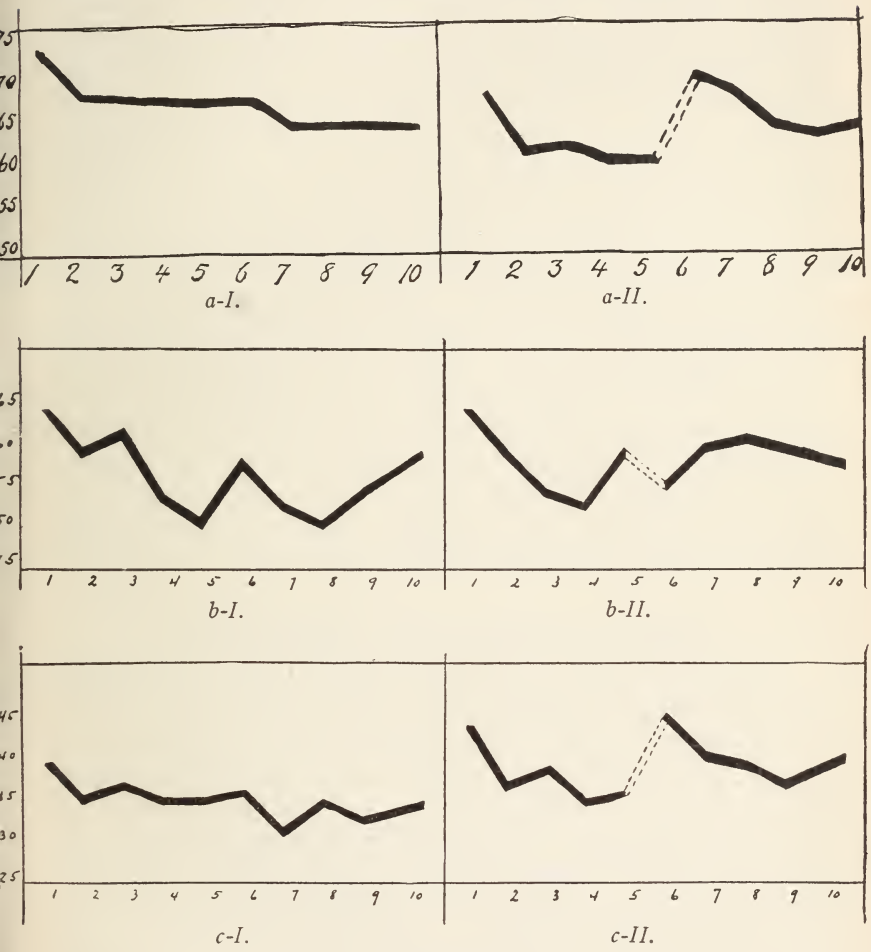

FIG. 2.

see that instead of the sixth minute rising distinctly above the fifth minute (before pause) the curve starts at a much lower level. This shows that the rest has not had a good effect upon the work of the patient, but has had a decidedly unfavorable

influence on the after work. It also shows that there was either no fatigue present or that if present, it was not compensated during the pause. And from what we know clinically of dementia præcox cases, especially the catatonic forms, the first supposition is the correct one. The sinking of the curve, then, is not due to fatigue, but to fluctuation and variations in the intensity of the will; although apparently working steadily along, the curve shows that the will is far from being under the control of the patient.

In curve *C-I* and *II*, that of a patient with alcoholic hallucinosis, we find very little deviation from the normal. The fatigue is shown by the gradual descent of the curve. And after the pause or rest, the curve begins at a much higher level, not only as compared with the fifth minute, but with the first minute as well. While these curves are shown as typical of the three groups under discussion, it must not be supposed that all of the cases in the three groups would show similar curves respectively in each group. There are wide variations in individuals of the three groups, but at the same time the average of the curves of each of the three groups will show distinct differences from each separate group. Especially will this be shown later when different factors are considered, such as fatigue, impulse of the will, daily increase in work, total amount of work done, etc.

Combined Average Work Curves (Fig. 3)

An effort was made to show the average curve of each group, but the result of averaging all the curves of one group was not a representative curve, as the variations of each individual curve would balance that of the others of the same group, and the result was not a representative curve. But this difficulty was overcome by Professor Kraepelin, when he suggested curves made up of the relation of each minute to the first minute, without any reference to actual values. So the combined work curves were constructed as shown in Fig. 3. Here the peculiarities of each group were represented graphically, and the result was startling. The work of the first minute in each group is the basis for computing the proportion of every other minute to the first. The standard value of the first minute is placed at 100. The value of the other minutes is computed by logarithms in terms of the first

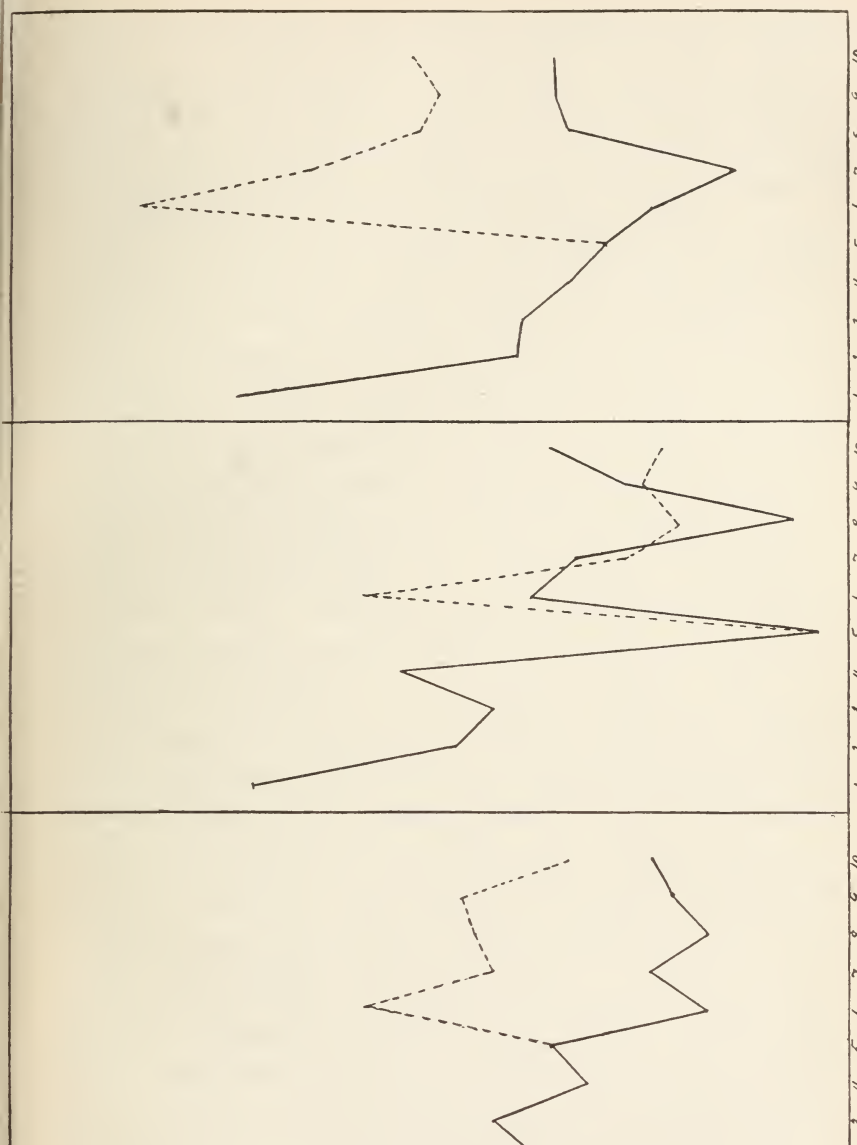

FIG. 3.—Combined average work curves of normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

minute, or 100. Thus, the normal curve, constructed from the average proportion of the 25 individuals, shows the second minute to be 92.4, in other words, the relation of the average of the first minute to the relation of the average of the second minute of all individuals, is as 100 to 92.4, and so on for the rest of the curve. And the same method was used for the other two groups. It will be noticed that at the fifth minute the curve divides, one curve continues on, and the other is represented by a dotted line. This division of the curve is to show the difference in the relations of the last five minutes without a pause (the uninterrupted line), and the last five minutes following the pause or rest, as shown by the dotted line. The first five minutes of each day is taken, as the work is uniform until the end of the fifth minute, when on one day a pause intervenes, and on alternate days no pause is allowed. In other words, the first five minutes of all curves of an individual group are obtained under similar conditions, while after the fifth minute, the pause makes a distinction. And the effect of the pause is graphically shown by the curve represented by the dotted line. In the normal it will be seen that the curve gradually descends, although there are some slight variations throughout the ten minutes work without a pause. (Note the abscissa indicates the minute of work, the ordinate the proportions of each minute to the first minute.) The sixth minute (dotted line) is separated from the sixth minute (straight line) by a considerable space. In the former the value is 97, in the latter 88. This difference represents the direct effect of the pause, and it will be noted that the course of the curve (dotted line) continues at a much higher level than the other curve. This corresponds with the typical normal curve in Fig. 1. Another peculiarity is that in the last two minutes of the lower curve there is a slight rise, showing the effect of the straining of the will by a distinct effort to overcome the fatigue. As the fatigue after the pause is less, we do not have this strain at the last, and the curve sinks during the last minute of work.

The difference between the values of the first and second minute represents the impulse of the will (Antrieb). As we all know, when one has a certain task to perform one goes at it with a strong impulse to accomplish the task, and this tension, as soon as the work is started, lets up, and the second minute shows a

considerable decrease in the amount of work done as compared with the first minute, or at the beginning of the task. And the difference between the sixth and seventh minute after the pause (dotted line) represents the impulse of the will after the pause. This is not so great as the impulse at the beginning, as through practice the task is known, and one does not strain the will to accomplish the work at hand to such an extent as at first.

When we observe the combined curve of the dementia præcox cases, we at once notice a marked contrast with the normal. The same variations and fluctuations in the curve are seen as were seen in the typical curve of dementia præcox in Fig. 1, *b*. But this is a composite curve of the twelve cases of dementia præcox, and the variations would be lessened rather than increased. One point of difference between this and the normal curve is the sudden sinking of the curve from the fourth to the fifth minute. Here is a drop from 96.5 to 84 and then a sudden rise to 93 at the sixth minute. As seen by the fluctuations, the question of fatigue does not come into consideration at all. First, because there are two ascents to the curve following this, and secondly, because the sixth minute after pause is comparatively very little above the sixth minute without any pause.

To find the explanation, then, we must consider the will which is seriously affected in dementia præcox. Here the defect is graphically demonstrated, and the sudden descent of the curve can be ascribed to the neglect of the will (Vernachlässigung des Willens) or deflection of the will.

The fluctuations of the curve in the last five minutes (without pause) is remarkable and is explained by the difficulty of keeping dementia præcox cases at a given task. The apathy shown clinically is here graphically demonstrated.

The course of the curve after the pause (dotted lines) also bears out the statement that there is little fatigue, and the curve, instead of remaining at a higher level after the pause, actually sinks lower than the continuous curve. So that from this curve three facts are demonstrated: (1) The absence of fatigue, (2) the irregularity of the tension of the will, (3) the absolute deflection of the will. The impulse of the will at the onset of the work is much less than in the normal and much less than that in alcoholics. The third curve represents the composite curve of the alcoholics.

This type of patients investigated was variable, and some were practically normal, especially those recovering from delirium tremens. The others belonged to the class of alcoholic hallucinosis and alcoholic paranoic conditions.

This curve does not differ materially from the normal, except in the height of the curve at the sixth minute after the pause. Here the curve starts at 103 or 3 points above the height at the beginning of the work and six points above the same value in the normal curve.

This increase in the amount of work done in the sixth minute in alcoholics over the normal is explained, not so much by the fatigue, but by the fact that during the pause the residual idea of the work is lost, and they start in with a marked tension of the will, assisted by the residual practice-effect (Übungs Nachwirkung). And they seem to have lost the impression of the work during the pause, so that it is in one sense a new task to them, while in the normal the memory impression is more lasting, and the normal does not start out with such a great impulse to accomplish the task.

Total Mental Capacity

(*Absolute Leistung*)

We obtain the values for the total capacity for mental work or the absolute ability for work in the various groups, by taking an average of the total number of additions performed in the first five minutes for the ten days. It is impossible to take the average of the whole ten minutes for on alternate days a rest or pause is allowed after the fifth minute, and consequently the last five minutes of work cannot be used in computing the amount of work performed by the individuals of the various groups.

By observing Chart I we will see the values for the total mental capacity represented in a graphic manner. The values are represented by columns for comparison, and it will be seen that there is very little difference in the amount of work done by the three groups. The values for the normal group vary from 263 to 104, and the average for the group is 185. In the dementia præcox group the variations are between wider limits, i. e., from 289 to 92, but the average is considerably lower than in the case of the normal group, viz., 165.

In the alcoholic group the variations are between much wider limits, i. e., from 323 to 96, and the average for the group is 188, a trifle higher than the normal average, while the average for the dementia præcox group is much below that of the normal and alcoholic groups. One is at first surprised that the difference is so slight. But when we consider the clinical features of dementia præcox, this finding is not so surprising, and the clinical and psychological facts are in harmony. We know that frequently in this class of cases the intellectual defects are slight, and often absent for many years, so that the patients afflicted with this disease retain to a considerable degree their intellectual faculties, although profound disturbances of other psychic fields (emotion, will and ideation) are present and prevent the patient from living a normal mental life.

We will demonstrate later that the disturbance in dementia præcox is to be found in other fields than the purely intellectual. Of course, in the end stages of the disease this field also suffers and we see cases with profound mental deterioration. The alcoholic group exhibited only slight deviations from the normal, and none of the cases were demented so that the intellectual capacity does not differ materially from the normal. So this fact is in absolute harmony with the clinical symptoms of the disease.

In connection with the total intellectual capacity we must consider the daily increase in the amount of work performed through practice (Übung). This is shown, first, in Chart I, now under consideration, and is represented by the lined columns at the base of the solid block columns. This is illustrated in this manner so that one can compare the daily increase with total capacity in individual cases of the three groups. And the figures given in the squares represent the values of the daily increase in each case. The values are shown graphically again in Chart 2 for a comparison of the three groups.

By comparing the values in Chart 1 it will be seen that there is considerable variation between the amount of work performed, and the daily increase through practice in different individuals. Thus, in the normal group the highest daily increase (14) occurs in two cases, in one where the total work value is 180, somewhat below the average, and in the other with a total work value of

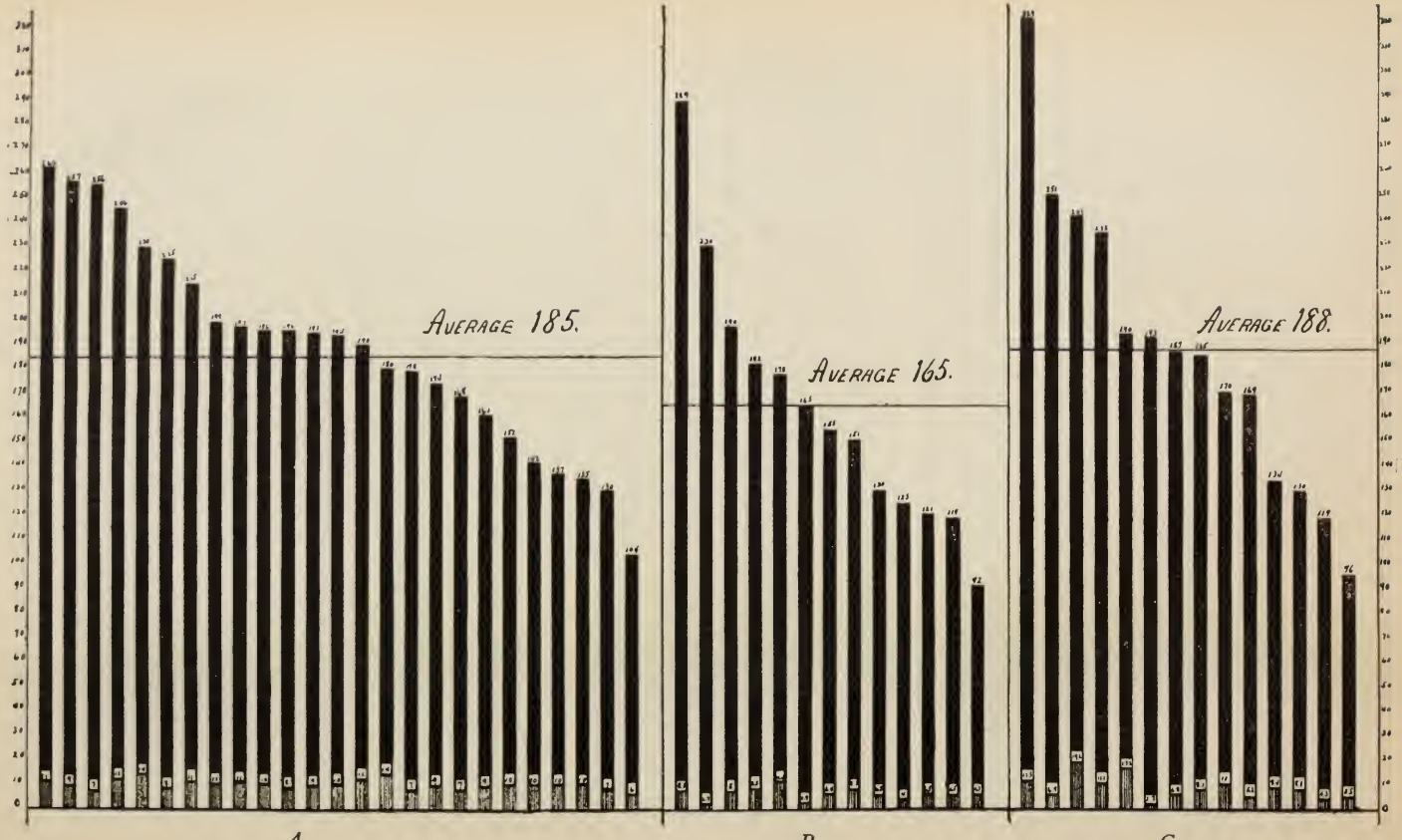

Chart 1.—Total mental capacity of normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

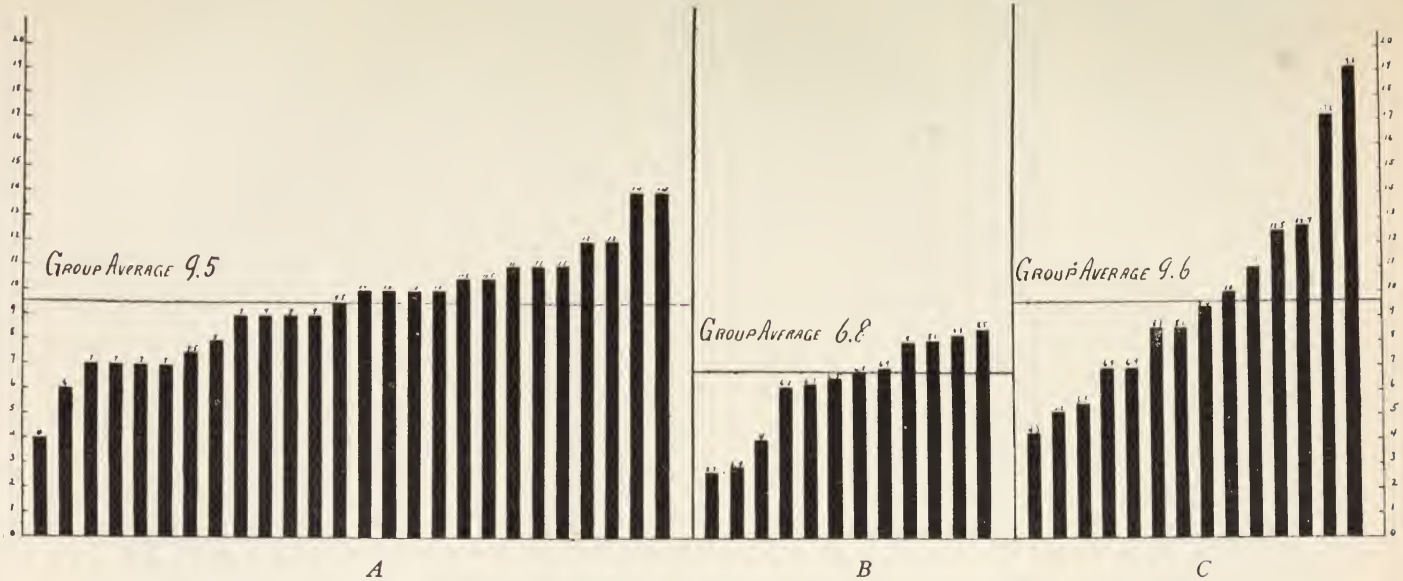

Chart 2.—Daily increase of work in normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

230, somewhat above the average of 185. The variations, however, are not so marked as in the dementia præcox and alcoholic groups. In the normal the average for the first 14 cases, with work values above the average for the group, is 10.2, while the average for the 11 cases below the group average (185) is 9, and by comparing Chart 2 the average daily increase is 9.5 for the normal group. Hence, in general we can state that in the normal group the daily increase is proportionate to the total work performed.

In the dementia præcox group we see more variation in the relations of these values. The lowest daily increase 2.7 is found in next the highest column, 230, and the highest daily increase, 12.4 in column 178, somewhat above the average 165. By comparing the daily increase in cases where the work values are above the average, we find almost the same condition as in the normal. The average of daily increase in five cases above the group average of work values is 8.5, which is 1.7 above the average daily increase for the group, while 6.4 represents the average of the seven columns below the group average. So that it can also be said for dementia præcox that the same conditions are demonstrated as in the normal regarding the proportion of the daily increase to that amount of work done.

In the alcoholic group the variations in the daily increase show much greater variations than either dementia præcox or normal, between 19.2 and 2.7, and the average 9.5. Again considering the cases which are above the group average, we find six cases with an average of 11.6, and these below the group average in work values, eight cases with an average of 7.4. Again the rule as applied to the normal holds good for alcoholics, except that the difference between these two averages is 4.2, which is greater than either the normal or dementia præcox groups.

By comparing the three groups, as shown in Chart 2, we see that the normal and alcoholic group are practically the same (9.5, 9.6), while dementia præcox falls far below, 6.8. And this is in harmony with the facts shown in Chart 1, in which the dementia præcox group shows total capacity for work much below the normal and alcoholic group.

Effect of Practice (Übung)

In close relation to the total intellectual capacity and average daily increase, is the effect of practice, in fact, the total amount of work accomplished and the average daily increase depend largely upon the effect of practice. The effect of practice is shown in average daily increase in the amount of work performed. In Fig. 4 we have plotted typical curves for each of the three groups. These curves are obtained by computing the amount of work performed during the first five minutes each day, and making a curve for these values for the ten consecutive days. Here, again, this second five minutes cannot be utilized because of the pause as previously explained.

And for the same reason that we could not use actual figures for constructing an average work for the various groups, we could not construct a curve representing the effect of practice. Therefore somewhat typical curves from each of the groups are selected. In these curves the abscissa represents the days, and the ordinate represents total amount of work done in the first five minutes on each day. The normal curve shows a steady rise with slight variations from 70 the first day to nearly 180 on the tenth day. In other words, this individual did two and one-half times the amount of work on the tenth day as he did at the beginning, or the increase was 250 per cent., due to practice and familiarity with the work. Of course, all normal curves would not show this tremendous increase, although they would approximate this curve to some extent.

The curve representing a case from the dementia præcox group, we see a decided difference. It will be noticed at first that the curve begins at a point where the normal ends, that is, the total amount of work done is much greater than in the normal case. This is shown on Chart I, where the column marked 230 is next to the highest column of this group. But the average daily increase is only 2.7, which is the lowest of the three groups. You will notice that there is a very rapid rise for the first four days (from 170 to 275 or 160 per cent. increase), then a sudden drop to 210, and from here to the last variation, until the curve ends at 245, or 140 per cent. increase over the work down in the first minute. This shows graphically the well known clinical symptom

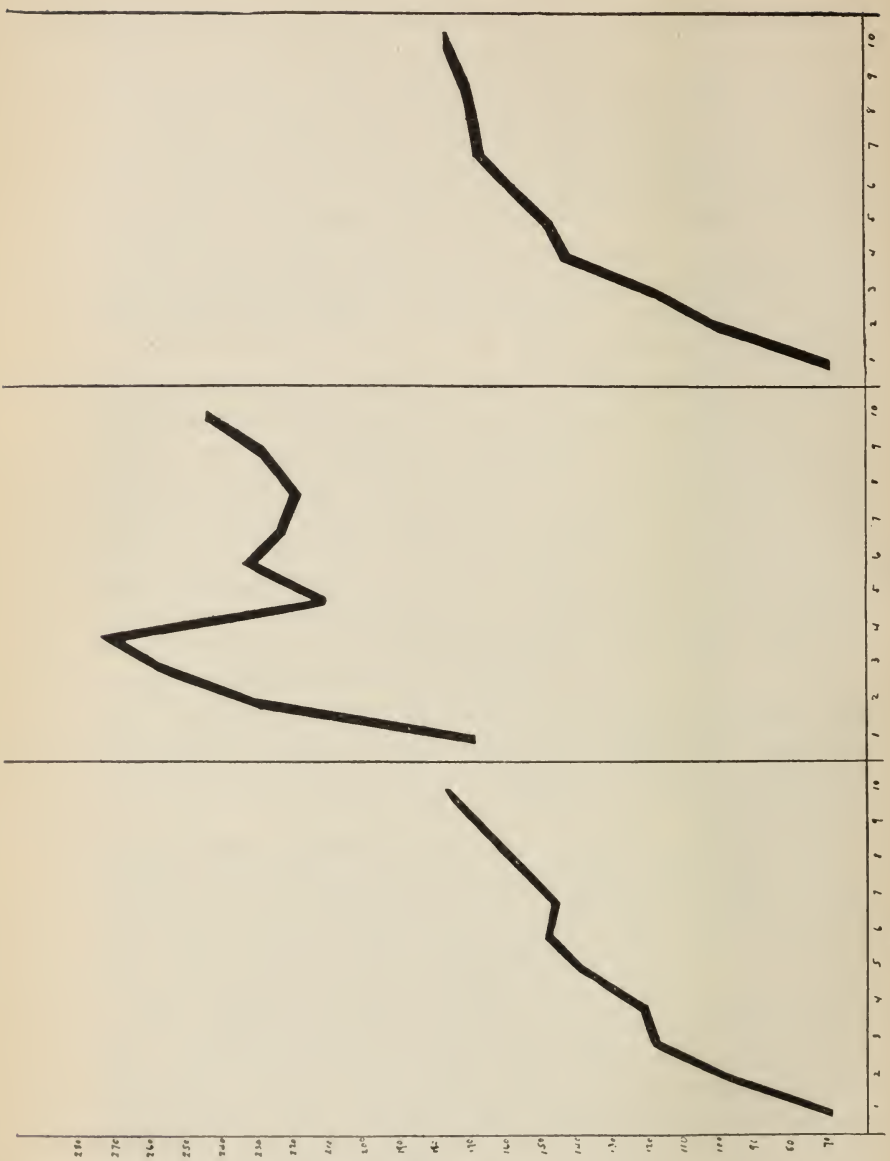

FIG. 4.—Effect of practice in the work of normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

of this form of mental disease, that is, a general emotional apathy, a loss of interest in surroundings, work, pleasure, etc. So then one is not surprised to find that this feature of the disease is shown so well graphically by this method. And this curve is not the most pronounced one of the group, but is fairly representative of the group. The curve of the alcoholic patient is nearly the same as the normal, and is also fairly representative of this group, although some of the cases show more variation.

Susceptibility to Fatigue (Chart 3)

(*Ermüdbarkeit*)

We obtain the figures for the coefficient of fatigue, showing susceptibility to fatigue by considering the general effect of the pause upon the individual's work. This method was explained in the first part of this paper, and will not be elucidated again.

By this method we obtain the values represented by the columns in Chart 3. In the normal group we see variations in the coefficients from 1 to 17, and the average for the group is 6. It will be seen that no negative values are present, as in the dementia præcox and the alcoholic group. In the normal group, without exception, the pause or rest has had a beneficial effect upon the after work, as we saw in Fig. 3. This is not so, however, in the dementia præcox group, for in the group 8 out of the 12 cases show a negative coefficient of fatigue, varying from 5.1 to 1, and are represented by columns extending below the line 0. The average for the group is only 1. The average for the 8 negative cases is —2.8. This result is rather striking, and corresponds to the facts explained in regard to curves shown in Fig. 3, only here the comparison between the groups can be more distinctly made. It can readily be seen that the effect of the rest was very unfavorable to the dementia præcox group, and that fatigue was not present in the large majority of cases.

In the alcoholic group the average 6.5 is just above the normal and there is very little difference in the coefficients. However, we have 3 negative cases out of 14, and these 3 cases were of the dull, stupid type so often seen among this group. In both groups (dementia præcox and alcoholics) the cases in which the fatigue

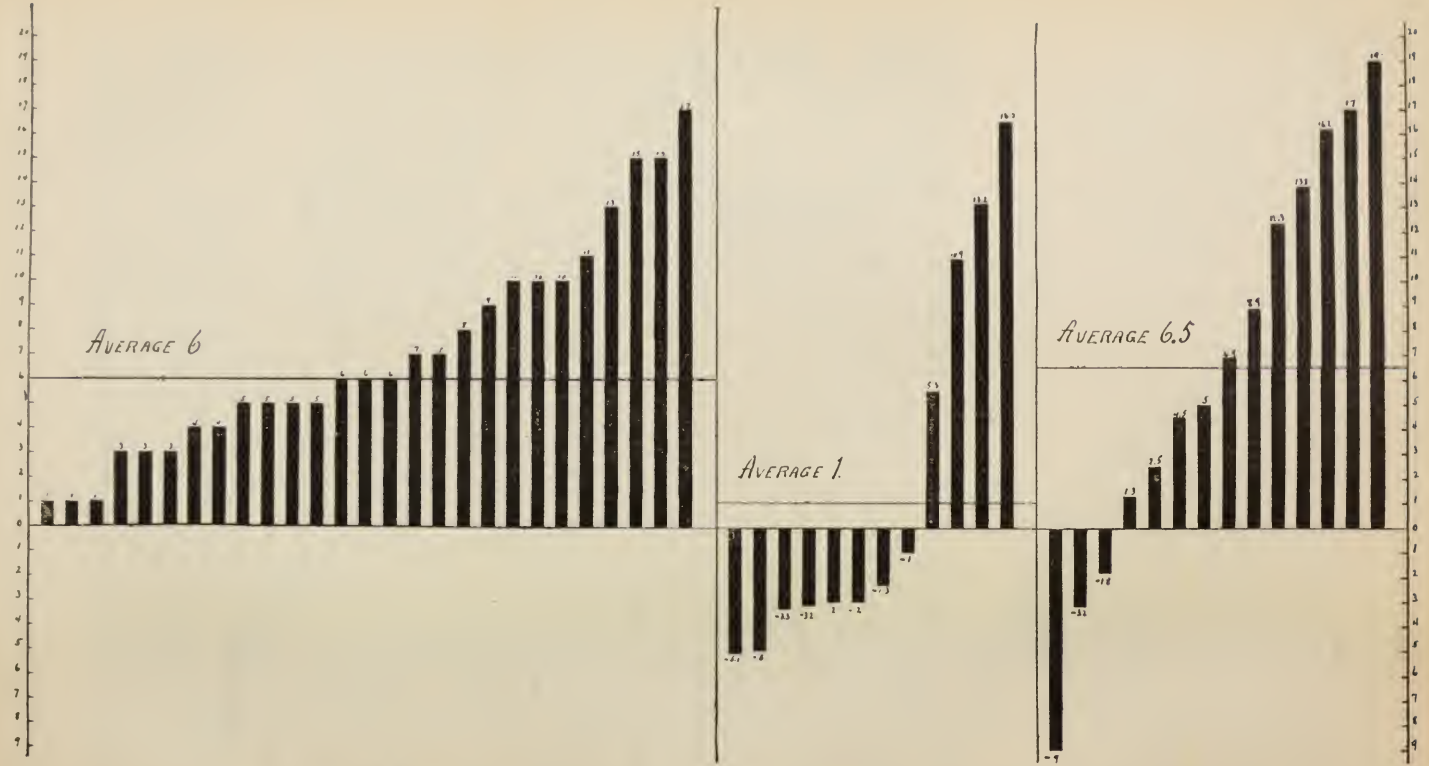

CHART 3.—Susceptibility to fatigue in normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

coefficient was negative, were also the cases with a smaller total capacity in comparison with other cases of the same groups.

In Chart 4 the values representing the direct effect of the pause

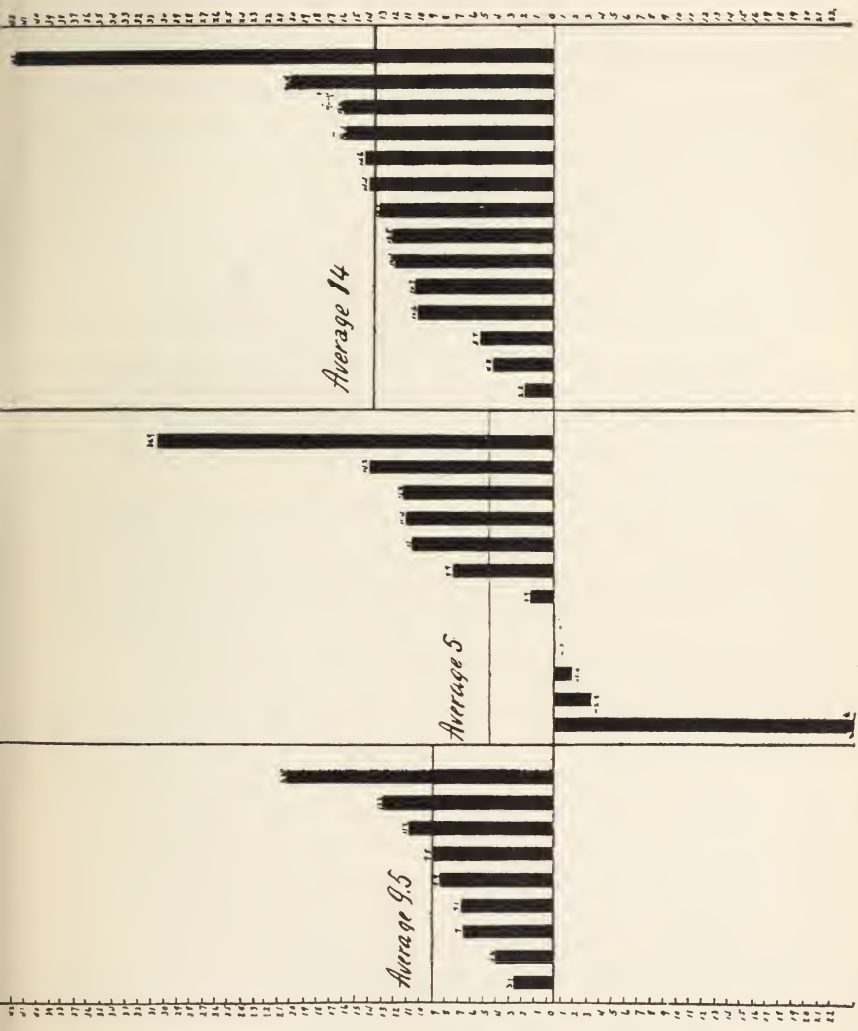

CHART 4. Direct effect of pause in work of normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

are shown. We have explained previously the method whereby these values were obtained. Unfortunately, I was unable to obtain in the normal group more than the values for 9 cases. But these

show no negative values. The average for the normal group is 9.5, which is considerably below the average for the alcoholic group, viz., 14. And this is also shown in Fig. 3 by the height of the dotted line after the pause where it reaches 103. And this has also been explained by the fact that in the alcoholic cases, although the fatigue values are not much above normal, yet the work after the pause shows an increase, probably because these patients after the pause have lost the effect of the work given before and start in as if they were beginning a new task. By comparing the values of the impulse after the pause, as shown in Chart 6, we find that in alcoholics the average for the group is lighter than the average for the normal, 4.5 and 1.5, respectively. And from these facts we can assume that the intensity of the will is not lost as soon as in the case of the normal group, and in consequence of this the alcoholics go to work after the pause with greater intenisty of the will, and the values of the work in the sixth minute after the pause are higher than in the normal group. Also the readiness for work is lost more quickly than in the normal person, as explained above.

In dementia præcox, however, during the pause, a different process has been in operation. We have seen that no great amount of fatigue is to be compensated or dissipated by the rest, and the sudden decrease in the intensity of the will counterbalances whatever recuperation from fatigue might have taken place. Thus, the work of the last minute before the pause, and the next minute following the pause (sixth minute) does not show such a great difference as is the case in the alcoholic and the normal groups. And here again we must take into account the absence of a readiness for work, the presence of which is shown in alcoholics and in normals. For through a certain indifference to the work before them they show no anxiety to go ahead and do their best. Hence in Chart 4, five cases out of 12 are represented by negative values, and the average for the group is only 5 compared with 9.5 and 14 for the normal and alcoholic groups respectively.

From these charts we can conclude, then, that the effect of the pause or rest in the alcoholic and normal groups exerted a favorable influence upon the later work, especially in the former group. But in the dementia præcox group a pause of the same length had

a very unfavorable effect upon their work. That fatigue is greater in alcoholics than in normal individuals has been shown by other methods and experiments, and the facts found here corroborate the views of other investigators.

Intensity of the Will (Impulse)

Chart 5

In normal individuals the intensity of the will, or the impulse of the will is influenced by four factors, as follows: (1) In the beginning of any work the feeling is that there is something to be overcome, or some task to be performed, and that causes a high tension of the will; (2) the intensity of the will is shown at the end of a task when one wants to do as much as possible before one finishes; (3) the entrance of fatigue causes a feeling of weariness, and in order to overcome this feeling one, so to speak, strains the will to greater activity; (4) after a disturbance in a certain period of work or task, one's attention having been distracted, one begins again to work, and the impulse of the will is again shown. This is shown in the experiments by the decrease in amount of work done in the second or third minute. Comparing this with the first minute before and after the pause, we must now endeavor to find how the normal relation is disturbed in our abnormal groups.

We see that the initial impulse at the start of the task is much greater in normal than in alcoholics or dementia præcox, thus, the values are 29, 9 and 11, respectively. And when we consider that the fatigue in the dementia præcox group is very small, the fact that no great intensity of the will is present, these two facts harmonize. By observing the combined work curve, Fig. 3, we will see this explained. And if we have no marked impulse or tension of the will the only way to explain this sudden decrease in the third or fourth minute is by a sudden failure of the tension of the will. The sudden sinking of the curve to 83 in dementia præcox is in great contrast to the gradual sinking of the curve in alcoholics and normal, respectively 89 and 91. These latter figures show distinctly the effect of fatigue which has overcome the tension of the will. And we have seen that the fatigue is very small in dementia præcox and cannot come into play in explanation of the sudden sinking of the curve.

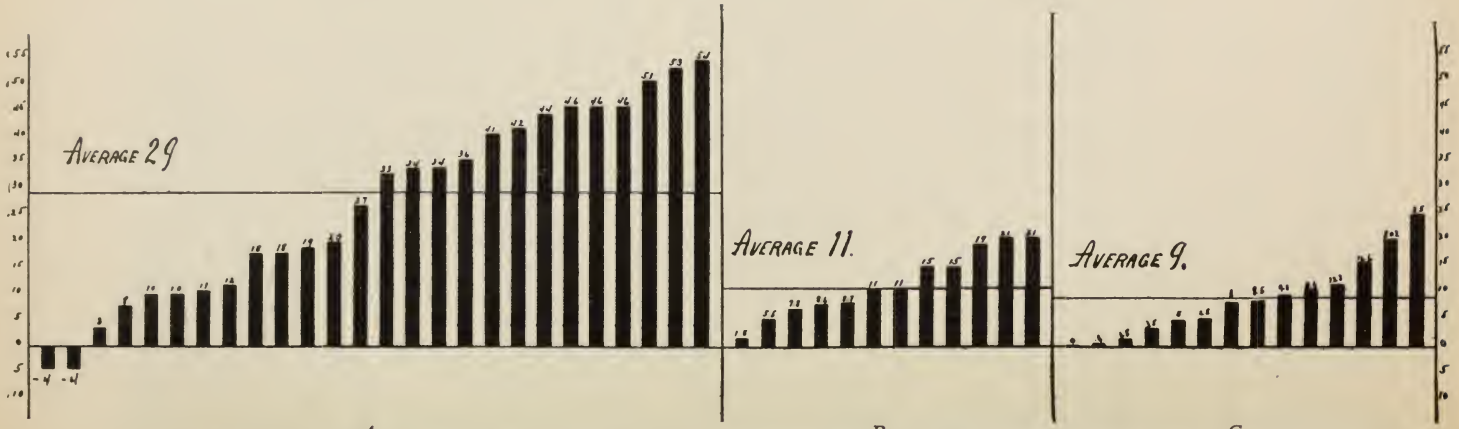

Chart 5. Intensity of the will in normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

The impulse after the pause shows two facts: First, that during the pause the fatigue has been compensated in the alcoholics, and in consequence we see a tremendous rise in the curve after the pause. The curve rises at 103, which is higher than in the start of the task, and the tension of the will is not as high as at the start. How is it in dementia præcox? We have seen that we have a lesser intensity of the will, and therefore less fatigue, also a sudden sinking of the curve in the fourth minute. And we have an apparent high point of the curve after the pause, but only to 95.6, which is very much below the alcoholics. And during the pause no recovery has taken place, and the work after the pause is less than the alcoholics. Then follows again the sudden failure of the tension of the will. The pause has been unfavorable, for the amount of work done after the pause falls below the amount of work done without a pause (see curve, Fig. 3). Again we must take into account the readiness for work.

We must conclude that in both groups a rapid disappearance of the readiness for work has taken place during the five minute pause, and in normal this is explained by the fact that at the start of the work the normal person shows a great deal of interest, but after a short pause of five minutes the tension of the will has decreased considerably compared with the tension before the pause. We know from experience (although such a fact has not been established experimentally) that normal persons hold the readiness and interest for work during a short pause. And when they again begin to work they have the feeling that the task is not so difficult because they know what they have to do. Therefore, they do not exert themselves, and the amount of work done by the normal after the pause is much less than that performed by the alcoholics. Also, at the end the curve sinks, while the curve without pause rises a little bit because of practice and familiarity. And we conclude (1) that the tension of the will before and after the pause, which in dementia præcox and alcoholics have very nearly the same value, is caused by the fact that the readiness for work is rapidly lost during the pause, and they begin to work after the pause just as if they had a new piece of work to do. While the great difference of the normal will tension before and after the pause is due to the fact that this readiness

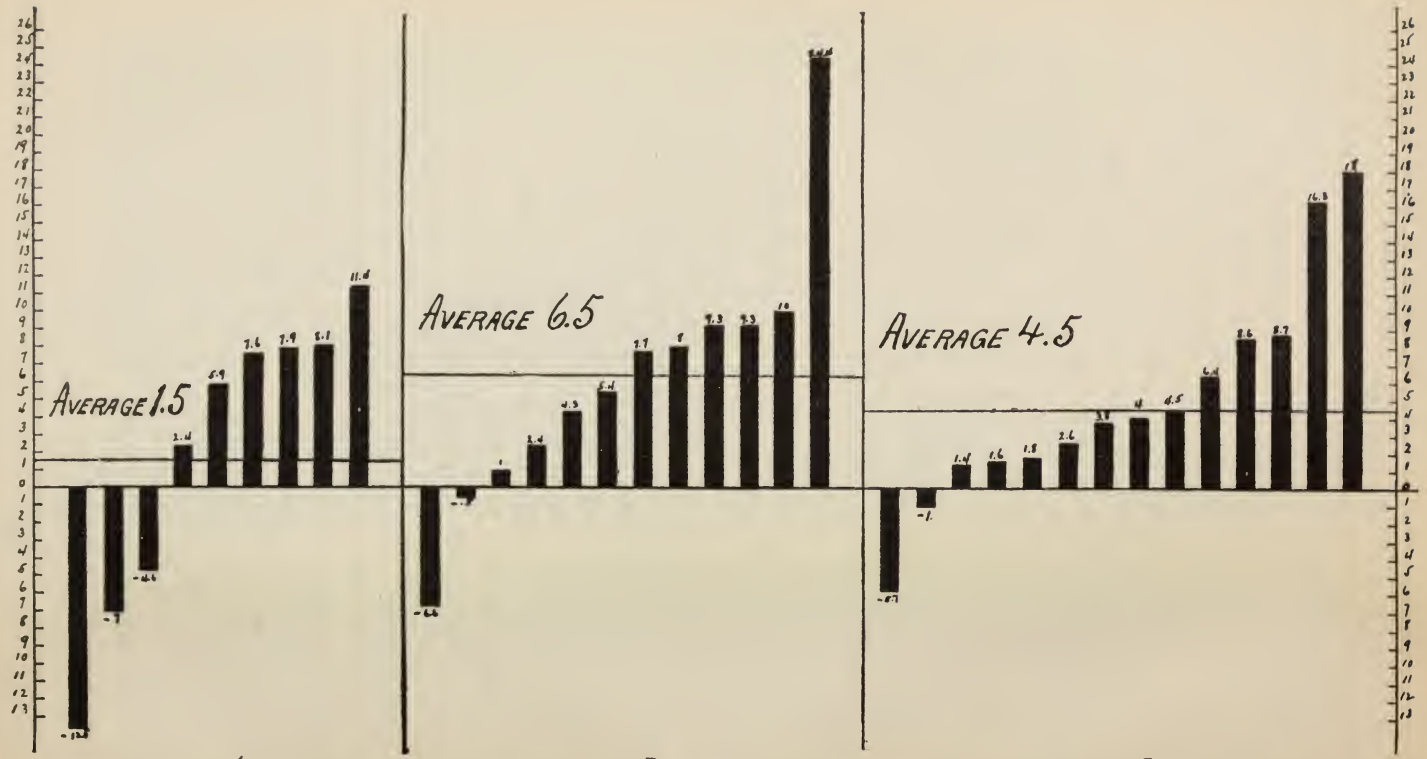

CHART 6. Values of impulse after pause in work of normal (*A*), dementia præcox (*B*) and alcoholic psychoses (*C*).

for work has not disappeared, they are prepared for the task at hand.

It can be demonstrated by experiment that after pauses or rest periods of different lengths that the readiness or preparedness for work in normal also is lost, and the tension of the will is gradually increased when the time of the pause is lengthened.

Second. That the increase in the work done after the pause in alcoholics is due to the dissipation[3] of fatigue during the pause, and also the higher tension of the will. In dementia præcox the total capacity is not so high as in alcoholics, but the impulse of the will is about the same as before and after the pause, because no fatigue has been counterbalanced, and the readiness for work has disappeared. The irregular course of the work curve, now high, now low, shows distinctly the fluctuation of the tension of the will.

In Chart 6 we have illustrated the impulse of the will after the pause. What we have said regarding the work curves in Fig. 3 is corroborated by the facts deduced from this chart. In the normal group we have 3 cases out of 29 showing negative values, and the average for the group is only 1.5. In the dementia præcox group there are only 2 negative values out of 12 patients examined, and in the alcoholic group only 2 negative values out of 14 patients examined. The average for the dementia præcox group is here shown to be higher than the other 2 groups, or 6.5 compared to 1.5 normal and 4.5 alcoholic. And this greater value for dementia præcox as compared with the normal and alcoholics is only apparent, or when we consider the space between the sixth minute dotted line and sixth minute full line in the 3 groups. In Fig. 3 this increase is explained. It is not that the cases of dementia præcox do so much more work after the pause, or that the impulse is greater, but the sinking of the curve before the pause due to the neglect of the will to act, accounts for this light average in dementia præcox.

Conclusions

By the simple experimental procedure outlined in this paper, we have shown:

1. That the disturbance of the will is the most important symptom of Dementia Præcox.

[3] Erhohlung.

2. Because of this defect of the will, the rest from work ,so beneficial to normal individuals, and the alcoholic cases, has an unfavorable influence in Dementia Præcox.

3. The effect of practice in Dementia Præcox is of much less values than in normal persons and alcoholic cases, which conforms to the general apathy shown in Dementia Præcox cases.

4. Absolute deflection of the will, a prominent symptom in Dementia Præcox, is shown by this method.

5. Fatigue is absent in Dementia Præcox, and greater in alcoholics than in normal persons.

6. Absolute mental capacity, as shown by the amount of work performed, varies but little in the three groups, being less in Dementia Præcox than in the other two.

7. That the daily average increase in the individual cases is proportionate to the total amount of work performed, is true of the three groups.

8. That the experimental results agree with the clinical symptoms of Dementia Præcox and Alcoholic Insanity.

PRACTICAL EUGENICS

By Henry A. Cotton, M.D.

Director of the New Jersey State Hospital, Trenton, N. J.

The question of the effect of hereditary influences upon the causation of mental diseases is one that has been receiving considerable attention of late. Up to within a few years our opinions of the effect of this influence in the causation of mental diseases was based upon rather insufficient and inadequate data. We spoke loosely about "heredity in the family," and were content to divide a certain number of cases into classes, those having heredity, and those showing no heredity.

The interest in heredity was, primarily, aroused by the work of Chas. B. Davenport, of the Eugenics Record Office at Cold Spring Harbor, L. I., N. Y. At this office, which is under the American Breeders' Association, remarkable data have been tabulated on the work in heredity in families. Not only heredity in mental diseases, but all sorts of traits, both mentally and physically. This pioneer work attracted the attention of the State Hospitals, and within two years Dr. Davenport had trained many expert field workers who were sent out to the State institutions to study this subject.

Practical work in Eugenics in this country was begun at the Training School for Feebleminded at Vineland, N. J.,

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under Prof. Johnstone and Dr. Henry H. Goddard. This was followed by the work at the Epileptic Village at Skillman, N. J., under Dr. David F. Weeks.

No one can deny the importance of thoroughly studying the question of heredity, and we have found the only practical way in which it can be studied is by having expert field workers go out into the community and gather information. We have now in the State Hospital at Trenton some 20,000 individuals charted, and the work of analyzing these data is enormous. The charts I shall show you this evening are selected charts, as it would be impossible to show all the cases we have on our records. We summarize important data for demonstration. We cannot say that we have any definite laws yet regarding mental diseases, based upon our researches. The careful analysis of this information on the heredity of mental disease has yet made only a good beginning. The subject is quite complicated, for insanity is a name that has covered a whole list of mental defects of most dissimilar characters and types. As far as possible, we eliminate the word "insanity" and try to make a diagnosis of the mental condition. Feeblemindedness, eccentricities, epilepsy and alcoholism are some of the conditions which seem to exert strong hereditary influences.

Mental defectives, classed as feebleminded, form only one group of the larger class of mental diseases, and the same can be said of epilepsy. The causes occurring in these two conditions have a certain uniformity which, however, is lacking in the larger group of insanity, or, as we prefer to say, mental diseases. But even here, all cases of feeblemindedness are not of the same type, and the cause producing these types may be quite different in individual cases. There can be no question but that feebleminded parents will produce feebleminded offspring, and that the hereditary transmission of such diseases as syphilis will produce idiots and other mental defectives. Hence, it appears that we should make a little closer examination of the types of feeblemindness, not so much from the degree of the defective, which has been done by Dr. Goddard, but we should also consider more thoroughly the cause producing feebleminded-

ness. Accidents at birth, arrested development through infectious diseases, such as scarlet fever, will frequently produce feeblemindedness, and also, as has been mentioned, direct transmission of syphilis, etc. The largest proportion, however, are caused by direct hereditary influence of the mental type of parents who are both feebleminded. In some cases where one of the parents is feebleminded, the offspring will undoubtedly show some feebleminded individuals.

The question of the inheritance of epilepsy is even more complicated than in the case of mental defectives, and here again, many factors are concerned in the causation of this disease. We have to consider accidents at birth, accidents in childhood, which result in head injuries, the inherited diseases, and then the direct inheritance of epilepsy, which has been shown by those engaged in this work.

When we come to consider the large group of mental diseases, of which each separate type must be treated by itself, it makes a very complicated problem. There is no question but that the majority of the cases of mental disease have a bad heredity to start with, but often this heredity is only one of several important causes. Thus, two sisters both inherit a taint from their parents. One of them marries and has a child. The effect of childbirth upon the mental condition is very pronounced, and the factors causing mental derangement may be considered both physical and mental, and frequently the woman develops mental disease following childbirth. Under this strain the married sister might become mentally disturbed, while the unmarried sister, with a similar tendency to mental weakness, without the exciting cause of childbirth, might escape, and live and die a rational human being.

Another type of cases which should receive consideration, is that known as dementia praecox. This is a chronic mental disease which occurs mainly in young people. In a great many cases they are unmarried. From the fact that they are unmarried, the progeny of that particular line or strain soon ceases, and as the families of these patients are peculiar, and many do not marry, the whole family will apparently end. Does this, then, lessen the number of dementia praecox in each succeeding generation? We are forced to say that it

does not. Hence, we must look to some other factors than heredity in producing this disease. There is no doubt but that we do have very marked hereditary influences in these cases, but notwithstanding the fact that these peculiar lines die out, we have an increase in this disease. It may be possible that hereditary influences are not limited to a tendency to mental derangement, but that other peculiar traits occurring in the ancestors are more exaggerated in the offspring, and finally produce this disease.

In another type of mental disease, such as general paralysis, or softening of the brain, we feel confident in assuming that heredity plays very little part in their causation. The main point for consideration in these cases is the fact that they have previously been infected with syphilis, probably ten or fifteen years prior to the onset of the mental disease. This type is purely an acquired condition, and the question of heredity may play no part whatever in the causation of the disease. It has been a much discussed question whether syphilis had any connection with general paralysis, but today every one is convinced that it stands in direct relation to general paralysis. Without syphilis there would be no general paralysis. Recently, Noguchi and Moore have found the germs of syphilis, the treponema pallidum, in the brains of persons dying of general paralysis. This at once settles the question as to the relation of syphilis to general paralysis.

The question of eugenics is not limited merely to the study of heredity. It should cover a much broader field. If we are to stop the increase of mental diseases we should endeavor to adopt measures which would secure normal offspring; and where the offspring would probably be abnormal, the State should take measures which would prevent such offspring.

The inheritance of feeblemindedness is so well proven, that in several states laws providing for the sterilization of such persons have been passed. We have such a law in New Jersey, which is now being tested for its constitutionality before the Supreme Court of the State. Recently, some States in the West have, through their Supreme Courts, decided that such a law was constitutional, and we hope that this will

have a tendency to influence the decision of the courts of this State.

A measure so new and far-reaching should, of course, be put in operation only upon the best of grounds. We feel that we have such grounds. Feeblemindedness is certainly hereditary. The law protects the rights of individuals in the most careful manner. Three commissioners—a surgeon, a neurologist, and the State Commissioner of Charities and Corrections—sit with the superintendent or medical director of the institution where the inmates are to be examined, as a committee of four to pass on each individual case. The patient is not only allowed counsel, but is provided with counsel by the State.

A considerable number of cases have been passed upon by the Commission, and they are now awaiting the decision of the Supreme Court, before the decree is carried out. If the decision should be favorable to the law, as we have every reason to hope that it will be, we shall be able to set free at least one hundred persons from the State Hospitals at Trenton alone, persons who are now confined merely because of the danger that they might transmit their mental defects to succeeding generations.

Perhaps I should explain that a rather large percentage of the inmates of our institution are what are called "moral imbeciles." They are not incapacitated from living in freedom and earning their living, but they simply lack moral power. They do not know what sexual morality is. At present we are forced to confine them in our crowded State Hospital, where we must keep the women throughout the period of their lives in which they may bear children. By submitting them to an operation, we can remove that danger, and they would then be much better off outside the institution.

It has been argued in objection, that by such a process we remove the penalty for immoral acts. The reply is simple: these people have no moral sense, and they are not subjects for punishment in any way. At present there are many such moral imbeciles at liberty, especially in the country towns of our State, and they have children who will be defective, and

in their turn pass their defects on to other generations. Prompt sterilization of as many as possible of these unfortunates is the best remedy that society has for such a condition.

One other measure of practical eugenics is now on the statute books of New Jersey. It is a dead letter, and necessarily so. It provides that no marriage shall be performed between two persons one of whom has been an inmate of a hospital for the insane.

That, of course, would work rank injustice in many cases. A considerable proportion of the cases that come into our care are discharged cured, under circumstances in which it would be perfectly proper for them to marry. I certainly think that there should be a prohibition of the marriage of certain persons who have shown the insanity taint, and I feel that a law should be passed providing that at the time of the discharge of a patient from a State Hospital he should be given by the authorities of the institution, a certificate of some kind, stating definitely whether his subsequent marriage would be advisable or unadvisable.

The preventable causes of mental diseases include vices, as alcoholism, and such diseases as syphilis. Twenty-eight per cent. of the male patients in the State Hospital come as the direct result of alcoholism; that is, these cases become mentally deranged and have to stay in the hospital for varying periods. This does not take into account a large number of cases in which alcohol plays an indirect part; and, according to some authorities, this indirect effect of alcohol is very large. Coupled with alcohol, is the question of syphilis, which is the direct cause of mental disease in 11 per cent. of all admissions. These two factors are the ones which it seems to me directly concern this Society, and it also appears that if there were more education along these lines suggested there would be, perhaps, fewer cases. Especially is this true in the matter of syphilis. I think that the parents of boys are, to a large extent, responsible for the fact that they do contract syphilis. Most boys have been taught the moral side of the question, but we have found that this is of very little help in deciding their course in these matters. If they should have the physical side of these diseases emphasized,

and were taught the consequences of immoral conduct, they would certainly pay more heed than they do at present. A large majority of the cases of syphilis are contracted through ignorance.

The important question of prostitution, its control, etc., will not be discussed here, although it is a very vital problem and is directly connected with this work.

The Value of Field Work in the Study of Heredity in Mental Diseases

HENRY A. COTTON, M.D.
TRENTON, N. J.

THE VALUE OF FIELD WORK IN THE STUDY OF HEREDITY IN MENTAL DISEASES *

HENRY A. COTTON, M.D.

TRENTON, N. J.

Since the discovery by Gregor Mendel, the German monk, in 1866, of the laws now so well known, much valuable data have been collected and many experiments in plant and animal life conducted. But not until within the last few years has any attempt been made to study the hereditary features of insanity in the same systematic manner; in fact, it can be said that only within the last two years has any systematic work been accomplished in regard to mental diseases, although it has been known to all engaged in the study of insanity for years that heredity plays a very important rôle in the etiology of the various psychoses.

For years statements have been made regarding heredity in insanity and opinions given by those connected with insane hospitals, but when one reviews the data on which these statements are based one is struck with the fact that these data are wofully incomplete, inadequate and often inaccurately taken. Not only does this apply to the older histories, where no attempt had been made to find the facts, where the statements of the committing physicians were taken as a basis for statistics collected in the hospital, but even in hospitals where modern methods can be said to be employed, and where an attempt is made to obtain accurate histories of the patients admitted, the same criticism of the facts regarding heredity can be made. For even with the most conscientious work of the physicians on the histories taken in the hospital at the time of admission of the patient it is often impossible to obtain information relating to more than brothers and sisters, parents and sometimes grandparents of the patient. Often the husband or wife of the patient is the only one to whom we have access in the hospital for information, and frequently they know very little about the family histories of each other, and we have been contented to consider the statement of insanity in the family as sufficient data on which to base opinions as to the character and degree of heredity. It is not unusual even to-day to see reports in insane hospitals, i. e., insanity in the family without regard to the character or type of the mental disease either in the patients or their ancesters. We must all plead guilty to this previous lack of interest in this matter.

It must be acknowledged that in this country at least the attention of the psychiatrist has been attracted by one outside the medical profession as well as outside the field of psychiatry. I feel that we owe Dr. Davenport a debt of gratitude for the tremendous interest he has aroused on this subject among the psychiatrists. It is needless for me to review his work here, but merely to state that two years ago he began to cooperate with

* Read in a Symposium on Mental Diseases at Hotel La Salle, Chicago, April 19, 1912. Read by Miss Florence Orr, field worker, New Jersey State Hospital at Trenton.

institutions, first in the feeble-minded and epileptics, and soon aroused the sympathy and support of those interested in the insane, and two years after his event in this field finds quite a few hospitals for the insane making use of the field workers trained by him at Cold Spring Harbor engaged in collecting statistics regarding heredity. It is also gratifying to note that New Jersey has taken the lead in this line of work, and the pioneer work of Professor Johnson and Dr. Goddard at Vineland and of Dr. Weeks of the Epileptic Village at Skillman is now a matter of record. We can gratefully bow to their leadership in this field and humbly follow in the way that they have so successfully blazed.

The epileptic institution at Munson, Mass., under the direction of Dr. Flood, must also be mentioned here as among the first to employ field workers to study the question of heredity among the patients. Although Kings Park State Hospital, of New York, was the first to employ field workers in the study of heredity in insanity, we believe that the New Jersey State Hospital at Trenton was the first to organize a permanent department in this field. The number of field workers has increased each year until last summer found over twenty taking the special course and these were later sent to various hospitals and institutions among which were many insane hospitals. The great activity exhibited in this field of research speaks well for our future knowledge of the subject, and before many years, no doubt, we will have a much better conception of the question of heredity in insanity and kindred diseases.

In only one other country, as far as I am aware, can it be said that any original field work or systematic study of heredity has been established, and as one would expect we find this attempt in Germany. As far as I am aware the work is confined entirely to one individual at the present time. Dr. Rudin, Oberarzt of the Psychiatric Clinic at Munich, is the man I refer to. It is all the more surprising that such work has not been carried on in Germany much before this, as Mendel was a German, and usually such opportunity does not slip by the German. However, if it had not been for the English and American investigators Mendelian laws would mean little to us to-day, so in one branch of science at least the Germans must yield the palm in originality to England and America, especially as regards work accomplished in this field. As stated before, aside from Rudin's work the field may be said to be barren. In the application of the Mendelian laws to the heredity in mental diseases his work is so unusual and important that I feel it is worth our while to look at it more in detail. Since 1909 he has been Oberarzt of the Clinic at Munich. During this period and for some time previous he has been collecting material for complex study of the question. So far one important contribution has come from the pen of Dr. Rudin, although in the amount of data collected I feel sure that he must have much more material than anyone else engaged in this work.[1]

The work of Rudin is more remarkable and noteworthy from the fact that he has done the field work personally and at his own expense. He

1. His article, "Einige Wege und Ziele der Familienforschung mit Rucksicht auf die Psychiatrie," appears in Vol. vii, Part 5, of the Zeitschrift für Gesamte Neurologie Psychiatrie.

gives up his position at the clinic so many months each year and goes into the field to collect his data. He employs several clerks and stenographers in his office also at his own expense. I consider his work more systematic and accurate than that of any other investigator in this field. Aside from seeing the individual personally as far as possible, which is a tremendous advantage, he has classified lists of all the inmates of hospitals in Bavaria and can get very good records and histories of all cases from these hospitals when necessary. This access to accurate records is a great advantage, especially in the cases committed years ago.

From the majority of records of the hospitals in this country, even within a few years, it is almost impossible to make any sort of a diagnosis, or even guess from what form of mental disease the patient in question suffered. Without financial encouragement and against difficulties that would appear insurmountable to us, he has achieved wonderful results. His course on entartung given at Munich this fall was extremely instructive and interesting and very important to those who were working in the same field. I had the privilege of hearing his lectures and also had several interesting conferences with him in regard to the work. I had taken over some of our charts, but felt that they were very meager compared with some he was able to show me, and that our work so far was to be considered insignificant in view of the tremendous amount of material at his disposal.

The publication that I have spoken of is extremely important to us all, as it is practically an introduction to the subjects and points of the various problems to be investigated, indicating the line where one's energies can be expended with the possibility of obtaining the best results. In his work of 100 pages he gives none of his findings or produces none of his many charts, and this certainly teaches us an important lesson, and that is to proceed cautiously and have plenty of facts and a great many family pedigrees before coming to a definite conclusion regarding the subject. Not only has he collected valuable data in the field, but he has summarized the best literature on the subject and gives the best digest on the subject that has been published as far as I am aware. I would commend this work to all interested in the subject.

The literature in regard to the question of heredity in various mental diseases is as yet very meager. The work of Professor Johnston and Dr. Goddard of Vineland and Dr. Weeks of Skillman is familiar to you all. With the exception of the work of Dr. Rosonoff at Kings Park nothing has appeared in the literature regarding insanity.

We are just on the threshold of important developments in the study of this complex problem, and it behooves us to proceed with caution. Accuracy is the one thing we must attain in our work. The problems of the feeble-minded and epileptic are much simpler than are those of the other insane. In the former conditions we are dealing largely with a defective anlage idiocy and external factors play only a subordinate part in producing the abnormalities; on the other hand, in mental diseases the external causative factors play a very important rôle in the etiology of

many forms, and these factors must be taken into consideration. So that it is absolutely necessary to consider forms of insanity either by themselves or only consider those which are closely related in the same group together. Each form will probably show peculiar features of heredity, and in some forms we may expect no more heredity that in normal individuals. As example we can cite defective childbirth or puerperal states in women, these conditions often precipitating not only delirious states, but often attacks of manic depressive insanity and even dementia præcox. Other sisters in the same family remaining single are not exposed to this precipitating factor, consequently they may not ever show any mental trouble. Many psychogenic factors present in one member of the family may be entirely absent in the sibs, and consequently latent predisposition to various mental affections may never be brought to light. So each individual type of insanity must be considered primarily alone.

The present rather unstable conditions of our classifications and the lack of definite pathology for many types whereby a clear differentiation can be made will necessarily cause us to proceed cautiously in our conclusions regarding hereditary features of many special groups. The large group of dementia præcox will probably offer more uniform types than will the manic depressive group. In the latter group, in view of recent anatomical findings, are many types which do not rightfully belong there. I might mention cardiac genetic psychoses, central neuritis, some forms of delirious stuporous states, which are often looked on as depression, and as we obtain more knowledge of the anatomico-pathologic changes in the cortex more cases will be taken from this group. So far it has been our inclination to make our cases fit into certain known laws, but if we have not the facts on which to explain the various hereditary conditions according to these known laws we should not assume too much; it would be better to admit our inability to correlate our cases according to the known laws than to force them into these laws without sufficient data. The tendencies to-day of families to have only two or three children at the most will often vitiate our results, or in families where a large part of the children die in infancy we are again being blocked from correlating the cases according to definite laws.

The conclusions reached by Dr. Rosanoff in the study of heredity in insanity in the light of the Mendelian theory are as follows:

The neuropathic constitution is transmitted from generation to generation in the manner of a trait which is, in the Mendelian sense, recessive to the normal condition. Rules of theoretical expectation are accordingly as follows:

1. Both parents being neuropathic, all children will be neuropathic.

2. One parent being normal, but with the neuropathic taint from one grandparent and the other parent being neuropathic, half the children will be neuropathic and half will be normal, but capable of transmitting the neuropathic make-up to the progeny.

3. One parent being normal and of pure normal ancestry and the other parent being neuropathic, all the children will be normal, but capable of transmitting the neuropathic make-up to their progeny.

4. Both parents being normal, but each with the neuropathic taint from one grandparent, one-fourth of the children will be normal and not capable of transmitting the neuropathic make-up to their progeny, one-half will be normal, but capable of transmitting the neuropathic make-up, and the remaining one-fourth will be neuropathic.

5. Both parents being normal, one of pure normal ancestry and the other with the neuropathic taint from one grandparent, all the children will be normal, half of them will be capable and half not capable of transmitting the neuropathic make-up to their progeny.

6. Both parents being normal and of pure normal ancestry, all the children will be normal and not capable of transmitting the neuropathic make-up to their progeny.

THE WORK OF THE TRENTON STATE HOSPITAL

It will not be out of place here to describe the methods used at this hospital, where we combine "field work" for the study of heredity with "after care" work.

We have had for a year two trained field workers, supplied through the courtesy of Dr. Charles B. Davenport, to collect data in regard to heredity factors in the family history of patients. We have not limited them to any certain line, but have insisted that all possible information in regard to relatives should be obtained. In one instance one field worker obtained information in regard to 3,300 members of a family group. This family group was located in one of the northern counties of the state and had intermarried to such an extent that only five distinct families were represented. Of this number, seventy-six were insane, twenty-two were patients in the State Hospital at Trenton, fourteen in other hospitals and forty not committed. Following is a list of the various abnormal individuals:

Sexual offenders	50
Epileptics	5
Alcohol	46
Feeble-minded	13
Cancer	19
Sarcoma	1
Blind	2
Congenital defective	1

In practically every case investigated it is possible to obtain some information in not less than 200 members of a family, and sometimes a great many more. The field workers have found no difficulty in obtaining this information, and without exception, they have received courteous treatment from the individuals whom they have visited. We find that the families are much interested in the work and will give all the information possible. The field worker becomes acquainted with the patient and usually talks with the patient before going to the family, and in this way carries messages back and forth and establishes friendly relations. They spend on an average fifteen days a month in the field.

The rest of the time is spent at the hospital, writing up histories and making out charts. They do not attempt to make diagnosis, but take down all facts given by the relatives. Wherever the family physicians know anything about the families, these are visited and their opinions also noted. Where relatives have been in the hospital reference is made to this, and a diagnosis made from the records when possible. When relatives have been in other hospitals, either in this state or in any other state, we have endeavored to obtain a copy of the records from these institutions.

The patients in the hospital are catalogued according to communities, towns, cities, etc. When the field worker goes to a certain district she has the names of the discharged patients who are living in that community. A visit is made to these discharged patients, and an endeavor made to learn something as to their condition. Often they find the environment such that it is necessary to report conditions to the hospital, and then advice can be given the family as to the right method to pursue to prevent a recurrence of the attack. This "after-care" work is a very important part of our field work and has resulted in much good to discharged patients. Several times during the year the field workers devote all their time to looking up discharged patients. Besides looking up the heredity in families, they inquire into the habits, domestic relations, occupation and any other factors which are wanted by the physicians. In certain cases where the statements of the family were questioned, the field worker went into the community and they were able to prove or disprove these statements. We have now collected a large number of pedigrees, averaging 200 or more to a family.

It is not my purpose to go into a close analysis of these charts, but these are given merely to show the progress of the work.

In Chart I (H. H.) we have the pedigree of a case of neurasthenia. Generations are given on the left-hand side of the chart. Each individual is numbered according to that generation. A transcript of the notes is given in this case to show the method of the work.

To summarize: We have a patient, a neurasthenic, one of three children, a sister of whom was an epileptic and a brother nervous. The father was a manic depressive case who committed suicide and the mother was neurotic. The father's family is apparently of good stock. The mother's family, however, shows marked defects. The maternal grandfather was a neurasthenic. The maternal grandmother was also a neurasthenic. One maternal uncle epileptic and another alcoholic. A maternal aunt suffered from manic depressive insanity, but recovered. The epileptic uncle has one epileptic boy. One of the patient's great-great grandmothers on the mother's side was insane at the age of 30, following the death of a child, from which she did not recover. In the maternal grandparents' line there is a good deal of nervousness and neurasthenia.

IV-39 was a patient at this hospital, manic depressive insanity, recovered and married a former patient. His wife had another attack and recently the man committed suicide.

HH

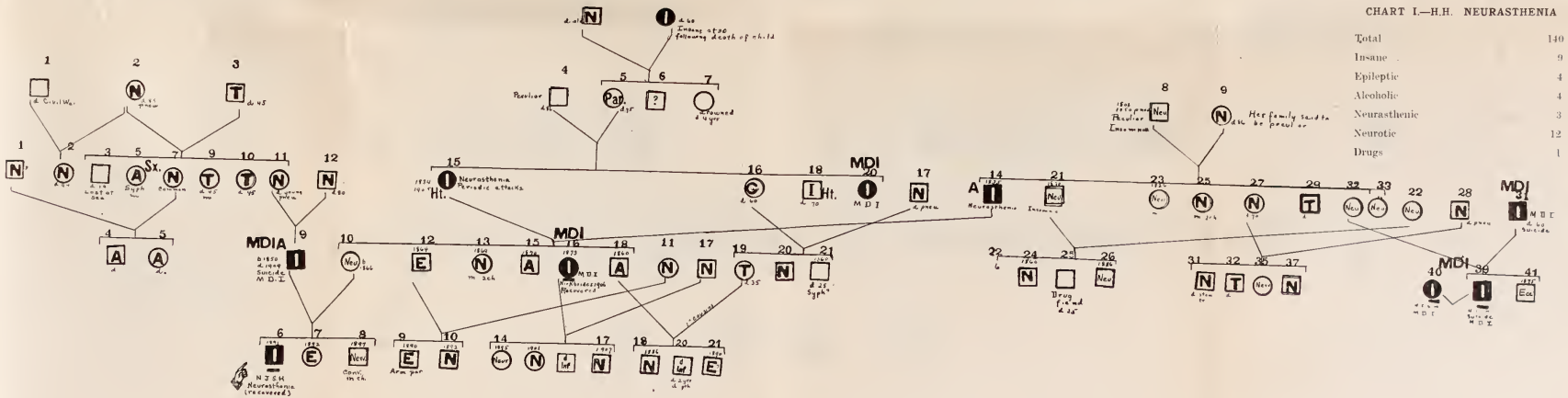

CHART I.—H.H. NEURASTHENIA

Total	140
Insane	9
Epileptic	4
Alcoholic	4
Neurasthenic	3
Neurotic	12
Drugs	1

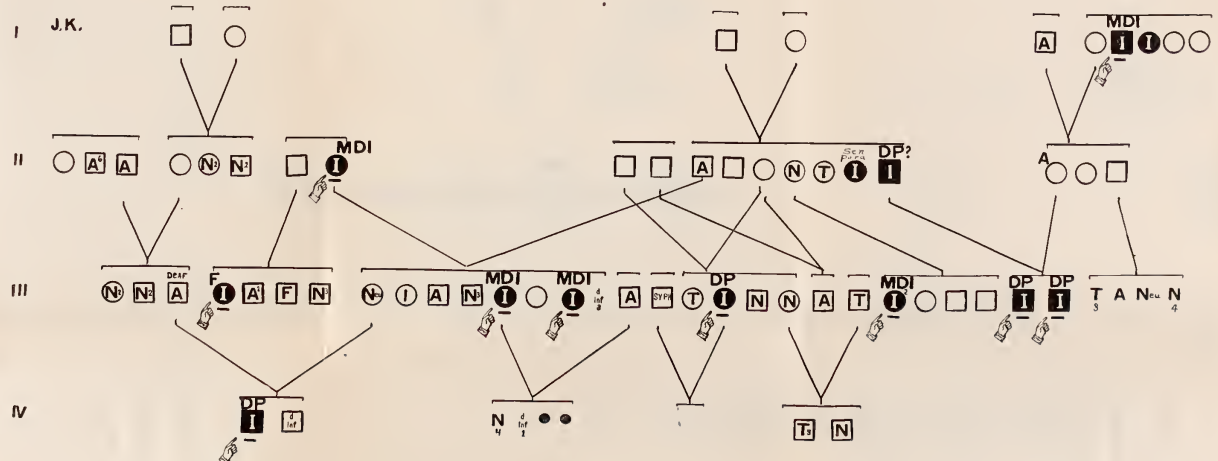

CHART II.—J.K. DEMENTIA PRAECOX

Total	200
Insane	15
In N.S.H.	10
Neurotic	3
Tubercular	12
Feebleminded	1
Alcoholic	12
Died infancy	17

Diagnoses of the Insane

Manic depressive insanity	6
Dementia praecox	4
Dementia praecox ?	1
Senile paranoid	1
Imbecile insane	1
Feebleminded insane	1
Unclassified	1

Sexual offenders	11
Syphilis	2
Epileptic	1
Constitutional defective	2
Neurotics	10
Alcoholic	6

Diagnoses of the Insane

Alcoholic insanity	1
Senile psychoses	3
Senile trauma	1
Constitutional defectives	1
Arteriosclerotic brain diseases	3
Manic depressive insanity	2
Psychasthenia	1

CHART IV.—P.H.W. DEMENTIA PRAECOX

Total on chart	292
Insane	15
Epileptic	1
Feebleminded	1
Neurotic	5
Syphilis	2
Sexual offenders	2

Diagnoses of the Insane

Constitutional defective	2
Melancholy	1
Dementia praecox	1
Psychosis following sunstroke	1

H. H., born in 1891, oldest child of J. G. H. and H. S. H. He has always been extremely nervous since early childhood, had never been like other children and has always been a source of constant worry to his mother. He has always read "deep" books and stayed indoors to read them. Many of these books were quack medical books. He is the oldest of three children. The next child, a girl, H., is nervous, and has suffered from convulsions after eating something which did not agree with her. It is perhaps epilepsy, as a brother of her mother suffers from epilepsy. The youngest child, a boy, is very nervous. There is a strong neuropathic tendency throughout the family, past as well as present generations. There is no insanity in the father's family, though there is a tendency toward sex perversion on the paternal grandmother's side. The patient's father himself committed suicide following two years drinking heavily after business reverses. The mother's family is all neuropathic, very few normal individuals to be found in the entire history. A great many of the people not committed were in a much more dangerous condition than the patient himself. The maternal grandparents and great grandparents were eccentric, a great-great grandmother was insane, a maternal aunt was insane and recovered, a maternal uncle was epileptic, a maternal great aunt and uncle insane, though never committed. A second cousin insane and in this institution, recovered. Many other neurotic.

Chart II (J. K.) is a summary which represents 200 individuals, fifteen of whom are insane, ten were in the Trenton State Hospital, twelve tubercular, three neurotic, one feeble-minded, seventeen died in infancy, nineteen were alcoholic. Of the psychoses, we have six manic depressive, four dementia præcox, one questionable dementia præcox, one senile paranoid condition, one imbecile, one feeble-minded, one unclassified. The paternal line is fairly good, with the exception of alcoholism. The maternal line, on the other hand, is very much affected. The mother is neurotic. One sister was a border-line case. Two sisters were manic depressive cases. The mother had nine living children, three died in infancy, making a family of twelve. The mother had manic depressive insanity from which she recovered and is now living at the age of 83. The father was alcoholic, had a sister who was in senile paranoid condition and a brother dementia præcox. This brother had two children, both of whom are dementia præcox and inmates of this hospital. He married a woman put down as peculiar. The maternal cousin is a case of dementia præcox in this hospital. As we see here, in the preceding generation the number of children exceed the large number of children of the present generation. In this family, out of five individuals who were insane in the grandparents and great grandparents, only two were in institutions, while all the cases that were insane in the parents' children were committed to institutions, and this fact will be found to run through all our charts. One can conclude that in the preceding generations the percentage of the cases who were insane and were committed to an institution were much smaller than the percentage of the same class to-day. This has an important bearing on the apparent increase in the number of insane institutions at present, for I think it can be definitely shown that a large proportion of those who were insane in the community in previous generations were kept at home.

Chart III (M. L.) represents a family of 371 members, in which seventy-three were abnormal in the following proportions:

Insane	14
Alcoholic	6
Sexual offenders	11
Syphilis	2
Blind	2
Cancer	5
Epileptic	1
Deaf mutes	2
Congenitals	2
Constitutional defectives	2
Neurotics	10

Diagnoses of the insane are as follows:

Alcoholic insanity	1
Senile psychoses	3
Senile trauma	1
Constitutional defective	1
Arteriosclerotic brain disease	3
Manic depressive insanity	2
Psychasthenia	1

The patient represented by three asterisks was a psychasthenic. She has six brothers and sisters; two brothers twins and neurotic, two sisters neurotic, one sister insane, with diagnosis of hysteria. We find the mother was a constitutional defective. There are two brothers and a sister manic depressive, and the maternal grandparents were both senile psychoses. The father was neurotic, and was a psychopathic sexual individual. He was married three times. He had two sisters, one was senile, and the other arteriosclerotic. One brother suffered from senile psychosis, due to head trauma, at the age of 50. There were thirteen brothers and sisters in this family. Three could be classed with the senile psychoses, and here we find the father of this family died at the age of 60 of senile dementia, while the mother apparently came from normal stock.

This chart illustrates a very important point, that is, hereditary features of senile psychoses. Here the diagnosis is not made merely on old age, because of this family there are thirteen normal individuals living at the ages of 70, 93, 60 and 85, and this tendency to senility seems to be in this family in the proportion of three to twelve, thirteen children dying in infancy. Another significant fact, is the tendency in succeeding generations to develop manic depressive insanity and psychopathic states.

Chart IV (P. H.) illustrates the inheritance in a case of dementia præcox. The patient was one of six children, three of whom died in infancy, one neurotic and one normal. The father was a case of manic depressive insanity, recovered, living at the age of 62. He is one of six children. A brother has arteriosclerotic brain disease, a sister neurotic. Paternal grandparents, the grandmother's line, is apparently normal. Paternal grandfather was a constitutional defective and died at the age of 66 of arteriosclerosis. Grandfather has twelve brothers and sisters. One brother is put down as melancholic, one died at the age of 42, had a sunstroke and was insane. One was a constitutional defective, died

at the age of 19. There were five affected individuals in this group of thirteen, seven could be put down as normal, one neurotic. The mother of the patient was neurotic. She was one of fifteen children, seven of whom died in infancy, three were normal and one had hare-lip. The maternal grandmother was put down as insane. The maternal grandfather apparently a normal line. We have, then, summing up, a total of 292.

Insane	15
Epileptic	4
Feeble-minded	1
Neurotic	5
Hare-lip	1
Syphilis	2
Sexual offenders	2

Diagnoses of those insane are as follows:

Constitutional defective	2
Melancholy	1
Dementia præcox	1
Psychosis following sunstroke	1
Hysteria	1
Arteriosclerosis	2
Depressed	1
Feeble-minded	2
Unknown	2

CONCLUSIONS

We have not attempted to analyze these charts carefully, but they are given merely to illustrate the progress of the work, and also to illustrate what a difficult task the analysis of these charts means. Frequently, when a point in question is necessary, the field worker visits the family again to clear up these disputed points.

In this paper no attempt has been made to give any definite conclusions regarding hereditary factors in the various psychoses. We have reviewed some of the most important work done so far and outlined the methods to be pursued to obtain the best results in future work.

We have also spoken of some of the difficulties to be met with, especially when analyzing the material when it comes from the field workers. It is again well to emphasize the necessity of maintaining an open mind regarding these problems and not to be too biased in attempting to make the facts fit the Mendelian laws. At the same time we recognize that a comprehensive knowledge of the laws will assist us materially in analyzing our data and in arriving at practical conclusions. We also feel that much valuable material will be obtained which will aid us in solving the problems of prophylaxis and prevention of mental diseases. We hope that other hospitals and institutions will adopt this method of studying these important questions.

THE PRESENT STATUS OF OUR KNOWLEDGE OF THE PATHOLOGICAL HISTOLOGY OF THE CORTEX IN THE PSYCHOSES.*

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The value of pathological anatomy of the cortex as an aid to the further development of clinical psychiatry is to-day viewed diversely by various authors.

Kurella, for example, in his work on Cesare Lombroso, gives voice to the opinion that this branch of research even in the hands of the most talented technicians has so far given nothing positive. Ziehen is also of the opinion that the pathological anatomical standpoint, in spite of all naive illusions, has been of value in only a very few psychoses, and probably never will offer much help. Others will admit that the histology of the cortex in psychiatry has been of some use, but very little, and question whether it will be of any further help in explaining or clearing up the question of the cause and nature of mental diseases.

Kraepelin, on the other hand, has, in the third volume of the eighth edition of his psychiatry, described the various anatomical findings in the various mental diseases, and it cannot be denied that many such diseases can be explained on an anatomical basis, and that our knowledge of clinical psychiatry will be advanced by investigations in this important field.

In the recent years much has been written in this field, and many opinions expressed regarding the worth and value of pathological anatomy in the field of psychiatry, and it would be of some value to review all of the work. *But to do this, other facts come into consideration.* In this field, as well as other branches of psychiatry, an extraordinary amount of work has been done, and it is impossible without a great amount of labor and patience to review all that has been published. Among many worthy investigations in this field

* Zeitschrift für die Gesamte Neurologie und Psychiatrie. Referate und Ergebnisse. Band V. Heft 8. Aug. 6, 1912.

are to be found many articles of little value, wherein the results arrived at are either influenced by technical deficiencies or the conclusions cannot be taken without serious objections.

In the literature well-established facts are compared with unknown and useless facts, and problems already decided are rendered unstable, or unsolved problems are represented as already solved, to the confusion of the whole subject. Many new investigations are directed toward uselessly solving problems that have already been solved, when there is so much that could be done in attacking the unsolved questions at hand.

So, in order to further advance in this field, it will be necessary to take questions over which there is some strife or difference of opinion, or those questions which at present are not clear.

Progressive Paralysis or General Paralysis.

Many investigators have appeared in the field of general paralysis. Very few have added anything to the condensive and exhaustive investigations of Nissl during the period of this review.

One can well say that in general, through the many subsequent investigations by various authors, the principal facts of Nissl's investigations have been fully substantiated, and that at present fundamental facts established by him have a secure place in the scientific work in this field. But it is also true that many minor factors as established by Nissl have not stood the test of time, and that new questions have been unearthed in many directions and many significant facts added to our knowledge of this disease.

The principal facts of these investigations can apparently be divided into two classes:

(1) Those establishing differential diagnostic factors, which help to differentiate general paralysis from other diseases.

(2) Those establishing the fact that in general paralysis, aside from the inflammatory process of the blood vessels, a corresponding and often independent degenerative process occurs in the nervous elements of the cortex.

In the first group, particularly, many new investigations have appeared. The infiltration elements of the lymph channel of the blood vessels have been studied by many.

The not unessential strife over the origin of the plasma cells, which is the basis of our knowledge of the process, is as old as the

description of these elements, and has, through the work of such pathological anatomists as Maxinow, Weidenreich, Schaffer and others, substantiated our views that they are modified lymphocytes.

Among other grounds substantiating this view is the fact that the plasma cells have been found in the blood in many different disease processes (a collection of this data has recently been furnished by Cerletti). A few authors, however, still defend the histogenesis of these cells. Rossi believes, because of the fact that in dogs injected with his neurotoxin serum, among numerous plasma cells he finds adventitial cells with strongly marked basophilic cell plasma, that there is a possibility of the derivation of plasma cells from advential cells. Papadia and Catola are decidedly of the opinion that plasma cells are derived from transformed fibroblasts. Nissl and his school challenges Papadia with the fact that plasma cells are interchangeable with basophilic mononuclear blood elements.

Papadia challenges Nissl and his pupils with the fact that they confuse plasma cells with basophilic mononuclear elements of the blood, and that their criterion for the differentiation of the plasma cells is so indefinite that false interpretation is permitted. It is a well-known fact that proliferating advential cells can take on an intensive colorable protoplasm, but we have had many opportunities of observing the fact that these cells do not become plasma cells. Plasma cells and basophilic adventitial cells are found lying next to each other, but that does not indicate that one is derived from the other. After all is said, Papadia has brought confusion where the relations were apparently definite and clear.

There is no difficulty in general paralysis to teach beginners in a few lessons the morphological and tinctorial peculiarities of the plasma cells, and on the basis of this knowledge to convince them of the identity of these cells, even over a wide range of variations of the cell, so that they seldom confuse them with other cells except when plasma cells show a severe grade of degeneration.

Anyone who reviews the literature would be convinced that the description of the plasma cells, as given by Nissl and his school, is substantiated by the most noted investigators.

The hemiatogenous origin of the plasma cells is to-day on as firm ground, if not firmer, than in the year 1904, and there are no grounds for doubting or questioning this origin.

On the other hand, Rheindorf, who claims that the plasma cells spring from endothelial cells, finds all varieties of transitional forms between plasma cells and lymphocytes, and that plasma cells are to be regarded as modified lymphocytes under certain conditions. Apparently the narrow spaces of the capillaries give the places where the best conditions for their derivation are found, while in the massive infiltration of larger blood vessels the plasma cells are found mostly isolated and in small numbers, while lymphocytes are found in large numbers.

Lhermitte and Perusini have investigated the degenerative forms of the plasma cells. The former describes them as "cellules muriformes," apparently those cells which formerly were designated as colloid degenerated plasma cells by me, and which Spielmeyer also noted in "sleeping sickness." Perusini, in an arbeit of extraordinary importance and extent, in which all cell forms are discussed which can be confused with plasma cells, describes especial deviating degenerative forms.

An arbeit of Cerletti, regarding the *blood vessel changes* in general paralysis, has much of interest. He has not been able, by thorough investigation, to find a case in which new blood vessels have been formed by sprouting from capillaries (as described by me), that could be proved beyond a doubt. The lumenless connections between blood vessels, as shown particularly by the Weigert's elastic stain, and designated by him as obliterated vessels, have atrophied and because of the destruction of the nervous tissue and shrinkage in size of the tissue of the cortex have become unnecessary. The truth of his findings must be acknowledged, and in very atrophic cortices, as found, for example, in the circumscribed foci in Lissauer's paralysis, the majority of these connections between capillaries can be explained by his views. Besides these forms, there is no doubt that a large part of the increase in blood vessels in the paralytic cortex, as Cerletti has shown, is not caused by new formed vessels, but by the collapse and shrinkage of old vessels in atrophic nervous tissue.

But in early cases of general paralysis (compare Spielmeyer), one can see the changes in the blood vessels as described by me, and it has not been proven in these cases that Cerletti's ideas are altogether correct. The active signs of proliferation that one finds in cells of the vessel walls whose nuclei are extraordinarily

rich in chromatin, and with pronounced color to the plasma, which are seen to grow out, or wander from vessel walls, do not harmonize either with Cerletti's view.

In any case it is to be hoped that the question of the vessel formation will be studied by the use of suitable material, *i. e.,* in very acute cases of general paralysis. It is probable that the new formation of small vessels in paralysis does not take place by bridge formation from the sides of proliferated endothelium as I formerly viewed the change, but occurs through the growing out of capillaries in the same direction and lying on the main stem of the vessel. Besides these changes there occur in general paralysis, particularly in marked atrophic cortex, although seldom in arterio-sclerosis, many other forms of vessel changes which have been already described.

Recent investigators have entirely neglected a very interesting field of the paralytic process, *i. e.,* the *proliferation of the cells of the vessel walls.* In general it can be said that a few cases of paresis show an extraordinary amount of proliferation of these cells, while other cases more numerous show very little, and it is even absent in some. The absence of such proliferation is usually found in extraordinary and often fulminating types of paralysis. One must almost think of a combined process in cases where a marked endoarteritis of the small vessels is found of general paralysis and cerebral syphilis. Such a hypothesis can be substantiated only by thorough and exhaustive investigation.

Regarding the *ganglion cells,* the principal interest has centered in the changes in the neurofibrils, since the method for demonstrating these fibrils has been used more and more during this period we are reviewing.

Among numerous investigators, only one, Dagonet, who used the method of Cajal, has found the fibrils unchanged or normal. The others, including Ballet and Laignel-Lavastin, Ballet and Pitulesecu, Marchand, Marinesco, Bielschowsky and Brodman, Jansky, Raecke, Schaffer, Ronkichi Mariyasu, Gierlich and Herxheimer, Sciuti (who used the method of Donaggio), Fuller, Schultz, Ansalone, all found the same results, *i. e.,* the protoplasmic processes of the ganglion cells are ruined or destroyed or remain only as a short truncated process. In single processes the fibrils are often preserved, while in the cell body they are frequently seen

only on the border of the cell, and in the center of the cell the fibrils are all in well-advanced stages of degeneration. They are at times thickened, either from swelling, or sticking together of several fibrils, or in short heavy broken pieces and fine granules disintegrated. There is no marked thinning of the intercellular fibril network, and the destruction of fibrils appears to be more marked in the external layers than in the internal layers, and the very finest fibers are affected. "It is noteworthy that in sections of the cortex where the process is most severe and in the most advanced forms of general paralysis that one can find cases in which the fiber elements are still present." (Bielschowsky and Brodman.)

The result of all these zealous investigations shows that no specific changes have been found in the fibrils of the ganglion cells in general paralysis.

Compared with the variety of cell changes, as shown by the Nissl method, the pictures of the fibril changes usually vary but little. With very few exceptions the same can be said of other degenerative brain diseases. The only changes in the fibrils of the ganglion cells in general paralysis which would differentiate this disease from many others, which is a severe destruction of the cells, are a peculiar rotting off of the process, and disintegration of the fibrils.

The findings by Alzheimer regarding the *fibrils of the neuroglia* have been substantiated by the investigations of Kollmer. The latter found, aside from the thickening of the glia superficial layer, a particular intensive proliferation of the glia in combination with a new formation of vessel in the transitional zone between grey and white substance of the cortex. Alzheimer had called attention to the fact that aside from the definite high grade proliferation of neuroglia fibrils (Stütz glia) in general paralysis, also that the scavenger glia cells (Abbau glia) play a very important role; while in severe acute cases we find mostly near the ganglion cells showing the severe alterations of Nissl, many amœboid glia cells in the grey and white substance that show a pronounced neurophagic activity, and that particularly in the cases of Lissauer's paralysis many gliogenic Körnchen cells are present, which are signs of a secondary degeneration in the grey and white substance of the cortex.

Recent investigations cause Alzheimer to modify his former opinion, that the " Stäbschen " cells found in general paralysis are mesodermal in origin, or are mesodermal elements.

First, Cerletti considered these cells as altered glia cells, from the fact that the relation of " Stäbschen " cells to ganglia cells was quite similar to that of the traband or satellite glia cells. And that the frequent occurrence of glia cells with more or less elongated nuclei resemble Stäbschen cells. Sträussler has shown that occasionally Stäbschen cells formed a center of glia fibril formation, and he believes that such cells must be regarded as glia cells.

Finally, Achúcarro found the formation of Stäbschen cells took place in the stratum radiatum as the result of rabies produced in rabbits, and he was of the opinion that because of the peculiar relations of the stratum radiatum these cells were adapted glia cells.

Perusini holds to the morphologic conception of the Stäbschen cells, and considers such cells described by him in presenile conditions as originating from glia cells. Bonfiglio believes that in lead encephalitis in dogs, Stäbschen cells occur which are of mesodermal and glial origin as well. Rondoni, Agostini, Rossi, Torata-Sano are of the opinion that the Stäbschen cells originate from glia cells. Lately, Cerletti, by a combination staining method of Resorcin-fuchsin and Toluidin blue has undoubtedly proven that many of the Stäbschen cells which are found in atrophic cortices of general paralysis are to be reported as adventitial or endothelial cells of obliterated vessels.

It was possible to show Stäbschen cells with glia fibril formation as described by Straüssler, also many which are of a *glial* nature. A very thorough investigation of the Stäbschen cells was undertaken by Ulrich. He is of the opinion that the long Stäbschen cells of the paralytic cortex are mesodermal elements, while the short Stäbschen cells are glial elements. In any case, the recent investigations have shown that cells with " Stäbschen " formed elongated nuclei are a great deal commoner than we formerly thought.

While the finding of the peculiar long forms of Stäbschen cells in large numbers is still characteristic for general paralysis, we find, however, fairly long forms occasionally in large numbers in multiple sclerosis, tubercular, luetic and endemic meningitis, and in luetic endarteritis. They are mostly short forms, and are found

in senile dementia and arteriosclerosis, and these can be found in all forms of atrophic processes of nervous tissue.

There occur many transitional forms between the round nucleated forms and those with extreme elongated nuclei. Glia fiber formations in these cells occur very rarely. But the facts which have developed regarding the extraordinary variety of glia cells having a distinct biological significance, makes it easier to understand such peculiar forms of glia cells without fibril formation. Certain relations of glia cells to the vessels, Alzheimer holds, would point to the mesodermal character or origin of many glia cells, as, for example, the observation that Stäbschen cells either lie in the adventitia or radiate from a vessel, and a similar relation of the glia cells of the vessels and glia cells with fibril formation can be observed.

In any case, many Stäbschen cells in the general paralytic process are of glio-genetic origin; and it has been proven that many are of mesodermal origin by the investigations of Cerletti. The method of Achúcarro also shows that Stäbschen cell nuclei are in the connective tissue bundles which grow from the adventia of the vessels into the ectodermal tissue. It is possible that included in the Stäbschen cells of the general paralytic process, that lie free in the tissue and are not connected or related to obliterated vessels, are many that are of mesodermal origin. To prove this beyond a doubt at present is impossible, because of lack of proper methods; Snessarew and Achúcarro have shown, with the help of the Bielschowsky method, which they have modified by introducing a new mordant (Beize), that in general paralysis bundles of connective tissue grow out from the adventitia into the surrounding nervous tissue and such connective tissue bundles build up a very fine and complicated network of fibrils. These facts are also of very great importance in general pathology, because they show that the border line between mesodermal and ectodermal element tissue is not alone passable for blood elements of inflammatory processes.

The relation of this growing of connective tissue into the nervous tissue to the membrana limitans gliæ perivascularis needs to be further investigated.

Recent investigations have shown that the *plasma cells* occur much more frequently in human brain disorders than was at first believed; and this fact has modified, to some extent, the importance

of this single finding as a differential diagnostic point. In luetic brain disorders, sleeping sickness, rabies, many forms of encephalitis, brain abscess, tubercular meningitis, and in the neighborhood of tumors, focal softenings in arteriosclerosis, and finally in multiple sclerosis (Behr, Oppenheim and Spielmeyer) and they also occur in infectious delirium (Kleist in unpublished work on scarlet fever, psychoses). In many cases of mental disease, in senile dementia, arteriosclerosis, dementia præcox, and also in cases without mental disease, Alzheimer has found single isolated plasma cells in the lymph channels of the adventitial sheaths of vessels of the cortex, but only when the patients have died of sepsis. And he considers that there is a complication in these cases that must be noted. Other diseases, in which in older reports, the presence of plasma cells was noted, *must be again excluded,* as in lead paralysis, Huntington's chorea and pellagra.

But when one excepts the luetic brain conditions, sleeping sickness and rabies (which we will later discuss again), the fact is patent that plasma cells only occur as isolated cells and in very circumscribed places in the cortex, and the process is not that of an extensive plasma cell infiltration of the whole cortex. A focal type of circumscribed plasma cell infiltration or the presence of single plasma cells has not been considered by us as characteristic for general paralysis. So Klippel and Lhermitte only fall against open doors when they declare that plasma cells can be found in sleeping sickness, tumors, abscess, traumatic or grip encephalitis, and are not characteristic for general paralysis.

When, however, plasma cells are found in extreme diffuse infiltration in " Confusion Mentale Primitive," there can be no doubt that we are dealing with general paralysis. Also in tubercular meningitis, the plasma cells are in visible relationship to the extent of the meningitis, and are especially found in the superficial layers of the cortex. (Ranke, Spielmeyer.)

Regarding the frequency and extent of the plasma cell infiltration in multiple sclerosis, we as yet have no thorough investigation of a large amount of material. But the differential diagnosis between multiple sclerosis and paralysis is not necessarily difficult, because of the focal character of the former, in spite of the fact that Spielmeyer has demonstrated that focal lesions resembling those of multiple sclerosis can occur in paralysis. The diffuse infil-

tration of the lymph spaces with plasma cells and lymphocytes is one of the easiest recognized signs of general paralysis, and therefore to-day the most practical and important diagnostic finding. This has been demonstrated by the majority of recent investigators (Jelgersma, E. Meyer, Ris, Elmiger, Spielmeyer, O. Fisher, Straüssler, Weiss, Behr, Rheindorf and others).

Janssens is the only one who seriously doubts this fact. De Albertis is of the opinion that the Stäbschen cells are of more importance than the plasma cells. The error is easily recognized when we consider the present status of the Stäbschen cells, as discussed above.

Naecke, who represents the view that syphilis is not a necessary antecedent to general paralysis, bases his opinion upon studies of the literature rather than upon his own histological investigations, and states that " the findings of Alzheimer cannot be considered as pathognomonic." When he uses as proof of his opinion, the fact that plasma cells occur in other organic brain diseases, he loses track of the fact that I admit the occurrence of plasma cells in those other conditions, and hold characteristic for paralysis the diffuse plasma cell infiltration in the cortex and other parts of the central nervous system.

It so happens that Naecke quotes investigators to contradict my findings who are entirely in accord with me. When Naecke claims that in order to make a positive diagnosis it is necessary to consider other things, such as: 1. Examination of the cerebrospinal fluid for pleocytosis. 2. Increase of albumin in fluid. 3. Wassermann reaction in blood and fluid. 4. Clinical picture. 5. Macroscopic findings of the brain. 6. Microscopic examinations of the brain and spinal cord, and that very few cases are so thoroughly investigated I can say in contradiction that in many of my cases all these methods have been used. And it has been found that 1 and 3 are of great value, as well as 2. In many cases 4 is uncertain, while 5 is often absent, and that 6 is the most constant of them all.

All examinations, taken together, show beyond a doubt that it is impossible to have general paralysis without antecedent syphilis.*

A careful investigator would not be satisfied unless he was able to demonstrate a diffuse plasma cell infiltration.

* The recent work of Noguchi and Moore, who found the spirochete of syphilis in the cortex of cases dying of general paralysis, absolutely substantiates Alzheimer's view.

Particularly severe changes in the ganglion cells, disturbance in position and layers of cells, numerous oblong Stäbschen cells, a rich production of glia fibers, infiltration of the meninges, involvement of other parts of the mental nervous system by the infiltration process, fiber tract degeneration in the cord, all make the diagnosis more accurate and are positive findings, but the diffuse infiltration is, without doubt, of the most significance. When one does not find an extensive infiltration of the lymph spaces of the cortical vessels one can exclude general paralysis (with the exception of stationary forms of paralysis, in which the infiltration is always present but usually very slight).

Other morbid processes, in which an infiltration of lymph spaces occur, either do not come into question, such as sleeping sickness, or are easily differentiated by other characteristic findings, as in multiple sclerosis, or the infiltration is not diffuse in character. Most of the authors agree with Jones, that the findings in general paralysis, particularly those changes in the blood vessels, are so characteristic that one can say with absolute certainty whether the case is one of general paralysis or not.

Important aids to the accurate diagnosis of paralysis are the examination of the cerebrospinal fluid (from lumbar puncture) and the Wassermann reaction, for antecedent syphilis, which in the last few years have been added to psychiatric methods. And these two methods are of special value because they can be used during life and often very early in the disease the diagnosis can be accurately established. But there are cases of paralysis in which these methods fail, and only by the histological findings can the diagnoses be made.

The histological examination has often substantiated the accuracy of the diagnoses in clinically doubtful cases where the Wassermann reaction was positive. The value of the law formulated by Plaut can also be proved by the histological findings, such as, cerebral syphilis, blood+cerebrospinal fluid−or weak+, general paralysis blood and cerebrospinal fluid both+, and therefore makes this method of value in the differential diagnosis of paralysis.

Alzheimer calls attention to the fact that while general paralysis can be diagnosed histologically from a very few sections of the cortex and with the use of a simple staining method, at the same time very little use is made of the fact in correcting or substantiat-

ing histologically the cases diagnosed as such clinically. It would make it easier to differentiate certain clinical cases which resemble general paralysis, but which are separate and distinct processes, if such studies were more frequently made. Only one important work of this kind has come to his attention where errors in diagnosis have been corrected by the histological findings. (Southard.) The lack of published records of such cases is to be deplored, although in some hospitals undoubtedly such methods have been followed.

A difficulty in histological differential diagnosis has arisen, in case the opinion of Buckholz is correct, through the processes caused by trauma, which are similar, clinically and histologically, to paralysis, but which are independent of the syphilitic paralytic process. Aside from the diffuse plasma cell infiltration, and proliferation of the glia, changes in ganglion cells of a diffuse character occur which are different from the general findings (also clouding of the pia over the occipital lobes, severe involvement of the occipital region, involvement of the anterior and lateral fiber tracts of the cord with the posterior columns intact. Marked marginal gliosis of the cord, mild grade of alteration in relation to the long duration of the disease process, collection of infiltration cells outside of the blood vessels). Whoever has examined a large amount of general paralysis material must concede the fact that all these findings, although they may be present only occasionally or in a small number of cases, at the same time occur in luetic syphilitic general paralysis.

On the other hand, numerous experiments on animals (Jacob) have shown that processes similar to the paralytic processes cannot be caused by trauma to the cranium or brain. But what is more important, every year one has the experience that traumatic cases give the typical Wassermann reaction of paralysis in blood and cerebrospinal fluid, where the clinical picture, that of a traumatic hysteria, persists for some time, even in the later stages of the process; in the cases where the paralysis was present before the trauma, and in a shorter or longer time after the trauma came to the surface.

So there is considerable doubt as to whether a traumatic process similar to paralysis coexists, or is combined with a syphilitic process.

In any case, one would only consider such cases as traumatic where the Wassermann reaction had been made and was found to be negative. With "lead paralysis," the relation is the same, and Alzheimer found that in very few cases examined by him did he find a true "lead process," which was easy to differentiate from paralysis, but in most of the cases he found a true syphilitic paralysis developed in lead workers who showed, beside paralytic symptoms, also symptoms of lead poisoning.

The second important point for establishment of the histology of paralysis, is that aside from the inflammatory changes in blood vessels, and independent of these changes, there occur degenerative processes in the nervous tissue, which fact has been confirmed by recent investigations. Sträussler found a disproportionate relation between a mild inflammatory process of the vessels and marked atrophy of the cerebellum would substantiate this theory. Spielmeyer also was able to demonstrate in a very early case of paralysis that the degenerative changes in the nervous tissue were much more extensive than the inflammatory process in the vessels, and that in many sections where the latter process was absent the degenerative process was more marked than in sections where the inflammatory process was more advanced. Fisher found in the foci of fiber degeneration, about which we will speak later, evidence that the disappearance of the parenchyma was not dependent upon the changes in the blood vessels and connective tissue. Alzheimer expresses the opinion that the extraordinary regular and equal destruction of the nervous elements in both hemispheres in the usual typical cases of paralysis, which fact Entres has noted, speaks for independence of the processes, and that, finally, the converse is true, that the focal character of paralytic cortex destruction is not dependent upon the blood vessel changes. He emphasizes the fact, however, that only in exceptional cases can one find destruction of the nervous elements without a corresponding affection of the blood vessels, and that it is not the rule. In general, the destruction of nervous elements which is independent of the blood vessel changes, is a type which belongs to the nature of paralysis, and indicate that, histologically, the process is metasyphilitic rather than syphilitic.

But this does not alter the fact that perhaps there exists in the body depots or foci where spirochetes are present and propagating.

On this ground, however, one cannot speak of process resembling paralysis, when plasma cells are found in the nervous tissue as Rossi, in his experiments with neurotoxic sera, has done.

Something essentially new for the histology of paralysis is found in the work of Borda, Fisher and Spielmeyer, whose investigations concern especially the focal character or circumscribed areas of fiber degeneration in the white substance. One finds in the older literature allusions to the focal character of the changes in paralysis. Siemerling, particularly, has demonstrated such changes. The insufficient and inadequate presentation of the finer histologic changes, however, made it impossible to decide whether one was dealing with a process produced by a combination of syphilis or arteriosclerotic changes or foci caused by a hemorrhage, softening or devastation or, perhaps, in some few cases with coincident paralysis and multiple sclerosis.

In this focal type of fiber degeneration in paralysis we have, however, the characteristic changes to guide us. Fisher has already, in his first arbeit, noted the similarity of these foci with those of multiple sclerosis, and Spielmeyer has described them in detail. The latter was able to show that such foci can occur in the cord as well as the cortex, and even reach the size of the placques in multiple sclerosis. Particular attention is called to the fact that placques appear to be a quite frequent finding in paralysis. Fisher found them in 65 per cent, Spielmeyer in 50 per cent of his cases, and Alzheimer found them not infrequently. This finding brings into the picture of paralysis a new unknown factor, and perhaps the various relations between paralysis and multiple sclerosis will gradually shed some light upon the nature of the latter disease. Finally, Alzheimer adds that from his recent investigations that in many cases with a diffuse extensive degeneration of the medullated fiber in paralysis medullated axis cylinders can be found.

Since the discovery of the spirochete pallida, many attempts have been made to find them in the central nervous system and other organs of the body. Occasional references to unsuccessful attempts are found in many investigations, and many have not considered it necessary to publish their negative findings. R. Stanziale has published his unsuccessful attempts to find the spirochete. Many others, from the fact that they are not found in paralysis, believe that the disease is a pure metasyphilitic process (O. Fisher),

others, from the fact that the Wassermann reaction is present, believe that the spirochetes are present in some portion of the body, and call attention to the fact that in many cases of tertiary syphilis the spirochete cannot be found (Plaut).*

Robertson has classed tabes as well as general paralysis as a process due to infection caued by a diphtheroid bacillus (paralyticans longus and brevis) which gains entrance to the nervous system through a weakened and susceptible mucous membrane of the respiratory and digestive apparatus, also through the bladder; such susceptibility of the mucous membrane being due to syphilis and alcohol. He claimed the paralysis bacillus could be grown on cultures, and by inoculation of animals symptoms similar to paralysis were caused. In Germany, where Robertson has predecessors in this work, every one holds his work as entirely false and worthless. (The same opinion is generally held in America.) Robertson has a follower in Langdon, and Flashman and Latham also believe in Robertson's theory, while H. Lind, in an investigation undertaken to prove or disprove Robertson's work, examined cultures from the blood, cerebrospinal fluid, mucous of the nose, nasopharynx, urethra and the urine and obtained monthly sterile cultures, and in some cases well-known staphylococci or streptococci.

Haempe investigated the extent and intensity of the paralytic process over the cerebrum. The investigation was macroscopic and hardened brains were used. The frontal, upper section of the central, and the parietal convolutions were the most atrophied.

Mignon and Marchand found in the brains of paralytics areas of amyloid degeneration. The process began in the transitional arteries and capillaries and progressed from the center outward. The endothelial coat withstood the process the longest. The nerve cells atrophied secondarily, but in some the amyloid degeneration was also present. The process was principally in the cortex, and localized especially in the pyramidal layer.

Witte found, in a case of paralysis, hyalin degenerated vessels, although they were limited to a small area in the cortex, and with

* The finding of the spirochete pallida by Nogouchi and Moore in the cortex of cases dying of general paralysis will settle the question as to the luetic character of paralysis and renders any discussion irrelevant at present.

this hyalin degeneration was found ruptured muscularis coat which was impregnated with amorphous masses. Probably without sufficient reason he considered this vessel change as luetic, and therefore classed the case as a combination of lues and paralysis.

Considerable investigation has been done on the *cerebellar changes in paralysis*. Jakasu has not brought forward anything not already described by earlier authors. Auglade and Latreille only substantiate the work of Sträussler, who completed, in three large and thorough arbeits regarding the cerebellar changes in paralysis following acquired syphilis, the meager information of other authors on the same subject, and supplemented the investigations of histology in paralysis in general by his work. According to him the infiltration of the meninges is the most constant finding, although there are wide variations in the intensity of the process. The destruction of the nervous tissue appears to be more marked on the summit of the convolutions and decrease toward the base. And this can involve the complete destruction of the Purkinje cells in a large area. The granular layer appears much lighter than normal. The large nuclear cells, Gogli cells, seem to possess a remarkable resistance to the process. The destruction of the medullated fibers is parallel to the destruction of the nerve cells.

The proliferation of the glia in the cerebellum often reaches a very high grade, more frequently than in the cerebrum are found in the cerebellum, focal lesions in connection with luetic vessel changes. The superficial convolutions are more affected than those lying deeper. The tonsilla seem particularly affected, which can be explained by the damage done by intercranial pressure.

Laignel-Lavastine and Pitullescu have worked with the method of Cajal and Bielschowsky on the changes of the neurofibrils in the cerebellum. According to their opinion the Purkinje cells are less involved than the giant pyramidal cells of the cortex. In the neighborhood of normal Purkinje cells one finds all varieties of transition types even to severe alterations. The Golgi cells are more affected than the Purkinje cells. The extra cellular fibrils are normal although occasionally isolated fragmented fibers are found. Another arbeit by these authors, is that regarding the "Déformation globeuse homogène," of the axis cylinders, which will be discussed under hereditary paralysis.

The *ependymal granulations* have been investigated by H. Baird. He finds them in 90 per cent of all cases of paralysis, and also in hydrocephalus, in senile dementia and arteriosclerotic brain disease. He comes to the conclusion that they are due to a primary proliferation of the glia, and not a proliferation caused by the irritation of the pathological cerebrospinal fluid, and that ependymal granulations secrete the pathological fluid. They are formed somewhat like glandular tissue, but Alzheimer does not agree with this theory, that they secrete the fluid, and thinks another explanation can be offered.

Regarding the spinal cord in paralysis, many new investigations have been made, notably, the work of Knichi Nacka, Vigouroux et Laignel-Lavastine, Boumann and Oppenheim, and also E. Meyer. Knichi Nacka found the paralytic posterior column degeneration of the cord did not always correspond in localization to the tabetic degeneration. The involvement of the posterior roots is irregular. An early degeneration of the endogenous fibers such as the comma tract, dorsomedial bundle, which is frequently found in the paralytic cord degeneration, is not found in tabes. The posterior root degeneration, which reaches through the grey substance to the lateral tract, which has been described in a case of paralysis, is foreign to tabes. In most of the cases, however, the tabetic and paralytic posterior cord degeneration are identical. In regard to the opinion of Reichardt, that the stiff pupil in paralysis is caused by the degeneration of the pupil fibers in the cervical cord, Alzheimer claims that all the changes in the cord which have been described, and their connection to the pupillary disturbances, have nothing to do with these hypothetical pupil fibers.

Boumann claims that the affection of the endogenous posterior tracts in the cord and the combination with lateral column degeneration of the paralytic posterior cord degeneration on one side, and the lateral column degeneration in tabes, on the other side, speaks for the difference between the two processes, but the similarity of the finer histological changes speaks for the similarity of the tabetic and paralytic process. Joffroy et Mignot in their book on general paralysis declare that the tabetic and paralytic process in posterior cord degeneration are not similar, and give as their reason the facts of different localization as described above. Alzheimer is of the opinion the processes are similar and identical, but

only differ as to localization. Vigouroux et Laignel-Lavastine call attention to the different causes of the posterior column degeneration of the cord in general paralysis (encephalitic, meningo-radicular, meningo-myelitic affection of the spinal ganglion and systematic degeneration).

E. Meyer and Oppenheim have worked extensively on the plasma cell infiltrations of the cord. Meyer found them in five out of six certain cases but in very small numbers, much less than in the cortex. Oppenheim claims that they are found constantly in the cord, but usually in small numbers. He also finds then in cords where no fiber degeneration is present. One can assume, therefore, that the process in the cord does not differ materially from that of the cortex and other parts of the central nervous system.

The *peripheral nerves* in paralysis have been studied by E. Stransky. He comes to the conclusion that parenchymatous alterations in the peripheral nerves ceteris paribus are to be found more frequently in paralysis and the changes are much greater than is the case with other psychoses due to marasmus and physical disease, and claims, therefore, that this is a substantiation of his opinion that progressive paralysis is a general disease of the entire organism.

Through the investigation of *other organs* of the body, an endeavor has been made to further substantiate this opinion. De Albertis and Masini found the thyroid gland affected in 75 per cent of the cases with a type of diffuse or insular sclerosis. They claim that there is a direct relation to the apoplectiform attacks and the affection of the thyroid gland. But Alzheimer claims that the disease of the thyroid gland cannot be considered as a cause of the paralysis, but it is to be regarded as a coincident and parallel affection. Schmeirgeld has investigated the glands concerned with internal secretion, thyroid, hypophysis, spleen, liver and ovaries. He found severe changes which could not be regarded as terminal exhaustion affections, and at the same time they could not be regarded as the cause of paralysis.

Lukacs also believes that the paralytic changes are not limited to the central nervous system, but were also found in the atrophic degenerative changes in the heart, the parenchymatous organs and the intestines. Catola claims even that one can make a probable diagnosis of paralysis, when infiltration of plasma cells

and lymphocytes is found distributed in liver or kidney, and when one can exclude focal lesions or infections, as tumors, parasites or abscess, etc., and other easily diagnosed conditions, such as tuberculosis, lues, cirrhosis, etc. Alzheimer formerly held this view. But the more thorough investigation of the organs for such infiltration has shown cases of paralysis with very slight infiltration in these organs, and cases not paralysis with very rich infiltration. In many extremely fresh or early cases of paralysis it was surprising to frequently find plasma cell and lymphocyte infiltration and in the majority of the cases the infiltration was very marked. But the finding of plasma cells in other organs of the body is not of such importance apparently as their presence in the central nervous system, and as Catola has shown, they cannot be considered as significant. It would be of much interest if more investigations along these lines were carried on with a large amount of material used for comparison.

K. Krajka has found in 92 cases of paralysis, changes in the heart and blood vessels 79 times, and 70 times in the aorta. Probably both of these changes are due to lues, and not coincidence. Daniels and Arendt found in 75.5 per cent changes in the aorta or valves, and Ladamme gummous mesarteritis in some cases of dementia paralytica.

Quite a few commentaries regarding the *focal localized type* (Lissauer's paralysis) have appeared (Buder, Hoch, Poetzl and Schüller, Pick and O. Fisher). The latter has described the pathological relations of these cases, and claims that the majority of such focal lesions are not caused directly by the paralytic process, although in some cases there is a high grade of simple paralytic atrophy present, which is the cause of the focal lesions. This characteristic process is known as the "spongy cortex devastation," which is found also in senile dementia, tabes, in presenile states, and also described by Probst as occurring in a twenty-four year old patient.

Alzheimer's own extensive investigations of cases of atypical paralysis leads him to conclude that the "spongy cortical devastation" of Fisher is not a characteristic or peculiar finding, but merely the expression of a particularly rapid type of degeneration or destruction of a great amount of nervous tissue. Various processes, such as paralysis, lues, arteriosclerosis can cause this

picture. We also always see, however, in this spongy cortical devastation of paralysis the typical changes of paralysis in the atrophic focus. He has seen cases of spongy cortical devastation in the frontal regions in typical paralysis, also many cases of Lissauer's paralysis without this devastation.

In cases of atypical paralysis of long duration and repeated apoplectiform convulsions to which they finally succumbed, one finds all the nervous elements of the circumscribed focal lesion in a process of disintegration. The deep layers of the cortex are full of gliogenic fatty Körnchen cells, while the fibers of the glia cells encircle the vessels with a massive coating and build connective strands from vessel to glia, and one finds also that the tissue is bathed in lymph. When this disintegrated nervous tissue is cleared away there remains the spongy scar tissue in its place. It is in any case noteworthy that at one time the "spongy cortical devastation" follows focal lesions of paralysis caused by a common but high grade of paralytic atrophy, at another time it is the result of an area of softening. Alzheimer also disagrees with O. Fisher, and thinks that one should speak of a destruction of cell layers in cases of atypical paralysis only with the greatest reserve. The third cell layer is very frequently the first to disappear, and this is explained by the fact that the cells of the second layer, although markedly affected, are more resistant to the process of dissolution, but show more of a sclerotic process which remains, and they last much longer. The end result of the paralytic atrophy is a complete destruction of the cell element of the whole cortex. Also, O. Fisher's opinion, that the common or usual form of paralytic atrophy begins with degeneration of the medullated fibers, is disputed by Alzheimer as unproven. For example, in the most rapid cases of paralysis, one finds the severest alterations of the ganglion cells, and even with the finest methods it is impossible to find any destruction of the medullated fibers. Martini, in a typical case of Lissauer's paralysis, found no evidence of a particular localized process. Alzheimer found in all cases of a localized process a high grade of atrophy. Ciaffini demonstrated in paralysis post tabem, quite different arrangements and localization of the paralytic process. A particular involvement of the cerebellum or the occipital lobe was not observed.

Another variety of progressive paralysis has been distinguished by the investigation under the auspices of the Bavarian Psychiatric Society, under whose direction cases were chosen in which the diagnosis of paralysis had been made, and later the process did not advance, or only very slowly in the course of many years. Alzheimer was intrusted with the examination of the material of these cases, and did the histological work. He found one case that presented histologically the typical picture of paralysis, in which the disease had lasted for thirty-two years, showing that a typical case can live for an extraordinarily long time.

In another group, that is now not so small, he found a somewhat different picture. In looking over the sections of the cortex, one saw in every part of the cortex single plasma cells and lymphocytes and " mast " cells, but never in great numbers or large collections. In many regions one could find only ten in a section of a convolution. In other cases they were more frequent; often the cells were very small.

At the same time one found very few infiltration cells in the highly thickened fibrous pia. Generally the smaller vessels of the cortex showed evidences of endoarteritis, proliferation, and in one case this was very marked. The limited inflammatory process in the vessels was accompanied by a still smaller affection of the nervous elements. The cell architecture was not particularly disturbed, the disappearance of ganglion cells was very slight, and many cells showed a chronic or sclerotic process. Preparations of the medullated fibers showed only a light diffuse disappearance of fibers. The glia was proliferated and showed fiber formation, but only to a small degree. Stäbschen cells were only found in the short forms. The evidence of the relationship of these cases to paralysis and the identity of the process is further substantiated by the presence of true paralytic changes, the involvement of the lateral and posterior columns of the cord, which, however, were present only in a small degree. So we can distinguish another variety of paralysis, which can be called *stationary paralysis,* and characterized not by an unusual localization of the process, but by a peculiar lessened intensity of the process. Further, Alzheimer speaks of many cases diagnosed as stationary paralysis which are not paralysis at all but forms of cerebral lues.

Watson and Mott have studied and reported the histological investigations of cases of *Juvenile General Paralysis.* The investigations of Sträussler regarding the occurrence of paralysis in persons with hereditary syphilis are of much importance. In the older works one finds the view generally expressed, that the anatomical picture of the juvenile paralysis does not vary from that of paralysis due to acquired syphilis. Formerly Alzheimer held the opinion that mast cells were present in these cases, but the material was limited in amount, and he now is willing to agree with Sträussler that such findings are not constant or characteristic. We are indebted to Sträussler for the peculiar and characteristic changes in the cerebellum of juvenile paralytics. These consist of, *first,* the presence of double or triple nucleated Purkinje cells. *Second,* the presence of characteristic spindle formed bodies, which lie either in the molecular layer or the granular layer in the center of a stalk that stands in relation to the Purkinje cells; and their structure in the molecular layer appears to be *granular* and in the granular layer, *homogeneous.*

The finding of double nucleated Purkinje cells has been proven by Ranke, H. Vogt, Trapet, Rondoni, Hough, Fischer, Achúcarro, Lafora, and Alzheimer found them in five out of six cases. Sträussler considers these cells as evidence of a developmental anomaly which occurs in the intrauterine period, and he regards them as an indication of hereditary syphilis. Trapet and Rondoni are of the same opinion, and Ranke believes that there is some question as to whether they indicate fœtal lues. In fifteen cases of congenital syphilis these cells were present only in one case but were also found in a case who died of delirium tremens, and whose father was a general paralytic. Statistics which show that juvenile paralytics in the majority of cases have parents who suffered with metasyphilitic disease, he claims, show, aside from these developmental changes, hereditary predisposition to paralysis. We have found no double nucleated Purkinje cells in three cases of hereditary lues without paralysis.

Recently E. Schroeder has reported the finding of double nucleated Purkinje cells in two cases of dementia præcox and one case of constitutional mania, and is of the opinion that such cells are not an indication of hereditary lues but an expression of a degenerative "Veranlagung." Rondoni described double and triple nucleated ganglion cells from the cortex of a case of hereditary lues.

Recently, since my attention has been called to this subject, I have found double nucleated Purkinje's cells isolated in a case of dementia senilis (2 cells in six sections), in tubercular meningitis of the cerebellum of an adult (4 cells in 4 sections), but was unable to find them in six cases of epilepsy, eight cases of dementia præcox, six cases of manic-depressive insanity, and in six cases of idiocy of various types, one case of amaurotic idiocy of Warren-Tay and Vogt-Spielmeyer form, one case of tuberosa sclerosis, one of hereditary syphilis and two of arrested development. It is apparent, then, that double nucleated Purkinje cells are rather uncommon. Only by thoroughly investigating a great number of normal brains and brains of those affected with mental trouble, can one decide whether or not these cells are of pathological significance, and where the border line between normal and pathological is to be drawn.

In hereditary paralysis the subject is apparently somewhat difficult. One sees in these cases surprisingly frequent double nucleated Purkinje cells and less frequently triple nucleated cells. We see that the nucleus shows quite a striking difference from those of various other forms of double nucleated cells seen in other conditions, and that the relation between the plasma and nucleus is much disturbed, often to the extent of damaging the former. In the neighborhood of ganglion cells with normal form and branching of the dendrites, we find cells with many processes with remarkable striking forms of branching, which differ so materially from the branching of dendrites in the normal cells and lie in unusual positions, that one hesitates to say that these forms can be explained by atrophy of the nervous tissue or especially of the Purkinje cells. So, when one on account of a lack of experience must question the findings of Schroeder, at the same time it must be admitted that the findings in the cerebellum in hereditary paralysis are highly significant and important. One case of Alzheimer, however, convinced him that the explanation is not always so simple. When Sträussler, and also Lafora, claim the presence of many double nucleated cells indicate paralysis on the basis of hereditary lues, even when the disease does not show itself until the fortieth year, so Alzheimer reports a case of paralysis in a physician who gave an exact and accurate account of a syphilitic

infection in the third decennium, which was also corroborated by the attending physician, in which were found the same double nucleated Purkinje cells as found in hereditary lues. Without one was able to concede that this patient had hereditary syphilis before he acquired his disease, which is highly improbable, even then one could not say with certainty that such a combination indicated that the double nucleated Purkinje cells of the cerebellum made it possible to diagnose " paralysis hereditaria tarda." Even the entire question of late paralysis on the basis of hereditary syphilis, as stated by Lafora, loses its heaviest support.

Alzheimer also reports another case of paralysis in an adult, wherein it was highly possible that he suffered from acquired lues, in which he could demonstrate cerebellar atrophy with many double nucleated and abnormal formed Purkinje cells.

The question of the origin and signficance of these double nucleated cells must therefore at present be considered as unsolved and unclear, and further investigation is absolutely imperative if we hope to solve the question. Particularly, one should observe and study a great number of cases of paralysis in persons of middle age, especially those in which cerebellar atrophy is well marked, and endeavor to find whether double nucleated cells are present or not. The case cited above, in case no other explanation is made, may lend color to the view that double nucleated cells may develop later in life in paralytics.

Sträussler's investigations have opened up a question of wide importance. Even in his first work he described the swelling of the dendrites of the *Purkinje cells* and the ballooning of the cell which change had been found by Schaffer and others in the Warren-Tay-Sachs form and Spielmeyer-Vogt form of amaurotic idiocy, and further the same change was seen by Schaffer in a case of congenital cerebellar atrophy. He noted, further, in his case of juvenile general paralysis the hypoplasia of the cerebellum and the Clark's columns of the cord, a congenital smallness of the medulla oblongata, and saw in all these facts a congenital arrest of development of the spino-cerebellar system; in short, a combination of paralysis with hereditary cerebellar ataxia and in the same manner a connection with the juvenile form of amaurotic idiocy.

Mertzbacher has busied himself with the work of Sträussler, and was able to fill up a gap, which the latter had for a long time

wanted filled, and made it possible to bring his aplasia axialis extracorticalis congenita (Pelizzaeus-Mertzbacher disease) in the great field of hereditary degenerative conditions.

If we turn our attention again to the swelling of the dendrites of the Purkinje cells, we must recognize two forms; those in the molecular layer appear finely granular, while those in the granular layer appear homogeneous, according to Sträussler. In the cerebellum of many cases of paralysis with undoubted acquired syphilis, we find the latter form as frequent as in juvenile paralysis, and in cerebellæ of adults with focal lesions and diffuse atrophy of various kinds. These changes are not infrequently seen. And E. Schroeder described them in an unmistakable manner in his case of dementia præcox, with double nucleated Purkinje cells. In the two forms of amaurotic idiocy they are infrequently seen. From the fact that these cells were free from lipoid deposits, they could be differentiated from other forms of swelling of the dendrites of the cell in amaurotic idiocy. The thickening of the axis cylinder of the Purkinje cells which Marinesco has recently described in his new contribution to the regeneration of the fibers in the central nervous system, and which he has illustrated, belong apparently to this class. By more exact microscopic observation (which is demonstrated easily with the Alzheimer modification of the Mann method) one sees a solid homogeneous picture, frequently vaculated, in any case only seldom with slight lipoid deposits.

Laignel-Lavastine and Pitulescu, who have studied these changes, speak of a déformation globouse homogène. Nagéatte believes that the neurites of the Purkinje cells are damaged before the branching of collaterals, while Laignel-Lavastine and P. Pitulescu disagree with this view, because of the fact that the swelling is observed running in various directions and can be observed in the molecular layer. In the cerebellum they apparently are not to be seen. In any case, this form of axis cylinder change is widely scattered over the cerebellum and cannot be identified with the swelling of the axis cylinder which occurs in other parts of the cortex and nervous system in amaurotic idiocy.

The other forms of swelling of the dendrites which was described by Sträussler, Alzheimer was unable to find in several cases of hereditary juvenile general paralysis with severe cerebellar atrophy. In another case many cells were present with dendrite resembling

the horns of a reindeer, and in some cases with slightly vaculated or ballooning of the dendrite. When one compares this with the changes in amaurotic idiocy (Warren-Tay-Sachs) one finds in the latter the dendrite are extraordinarily expanded and much larger. In the small amount of material at the disposal of Alzheimer of the Vogt-Spielmeyer form, and which he studied, he found in the cerebellum no expanding of the dendrites of the Purkinje cells. Schob recently reported a case with such dendrites unusually numerous. While, however, in both forms of amaurotic idiocy in the cell bodies one finds easily recognized changes in the neuro-fibrils and deposits of lipoid matter, in juvenile paralysis no such deposits or changes in the fibrils can be observed. The changes, aside from those described above, are a high grade cerebellar atrophy similar to that which occurs in paralysis of later years. Only once did Alzheimer observe what he believed were red globules or granules in the expanded portion of the dendrite of Purkinje cells of juvenile paralysis, in a section stained with Scharlach. The expanding or ballooning of the dendrites of the ganglion cells of the central nervous system is not an exclusive peculiarity of the cell changes in amaurotic idiocy. One finds them occasionally in senile dementia (Sinchowicz) and Alzheimer found them in a case of arteriosclerosis, and once in the cerebellum of a case of Huntington's chorea (in another case of the same kind they were absent) so it is not without some foundation when one assumes that from the changes of the Purkinje cells in hereditary paralysis, some connection between this disease and amaurotic idiocy probably exists. And it is Alzheimer's opinion that the weight of Straüssler's argument is not sufficient to prove the connection between juvenile paralysis and " herediataxia cérébélleuse." In a case of paralysis with previous acquired syphilis, in which there was a particularly severe cerebellar atrophy, it was noticed that the pons was exceptionally small. Indeed there was in this small extensive atrophy of the nuclei of the pons and reduction of the cerebellar pons arm, a rather severe inflammatory process in the region of the pons. It was difficult to say whether this was due to a secondary atrophy or to a primary paralytic process, or due to a combination of both processes. Similarly, in two cases of juvenile paralysis, in which the atrophy of the pons was particularly noticeable, there was a

pure paralytic infiltration process of the cerebellum, the brain stem, the pons and medulla which was quite marked.

In another case, in which no particular atrophy of the cerebellum was present, the pons was not especially reduced. The cerebellum is frequently atrophied in hereditary paralysis. Also in the mid-brain the paralytic process is marked and much greater in the average than in the adult paralysis. Alzheimer is of the opinion that there is some connection between these findings and certain disturbance of motion which are seen in the juvenile cases. To these primary infiltrations are added later the secondary changes.

A striking smallness of the spinal cord so often found in idiots, which apparently has nothing whatever to do with hérédoataxic cérébélleuse, for example, in a case of arrested development in the cerebrum, with heterotopia in an idiot affected with an old luetic meningitis over the hemispheres. Almost all cases of Mongolian idiocy which Alzheimer has seen show under development of the pons, medulla and spinal cord, which is very striking, and has been noted by other observers as well. So it appears to Alzheimer that the changes which Straüssler interprets as those of hérédoataxia cérébélleuse, are probably the result of the paralytic process or are connected with fœtal developmental disturbances in the cases of juvenile paralysis.

Also other portions of the cortical nervous system in juvenile paralytics, are the seat of developmental disturbance. Sträussler described such disturbance in the central canal of the spinal cord, and Rondoni found heterotopia of the spinal cord in juvenile paralysis. In Rondoni's cases, the paralysis was grafted upon idiocy, and one expects to find such disturbances in these cases. According to Rondoni, in the hemispheres, one finds very few differentiated cell forms but incomplete neuroblastic forms of the ganglion cells and ganglion cells which after wandering in the cortex remained in the white substance, a condition in which the cortex could not be differentiated and all trace of the six layers of the cortex was lost (thick circumscribed granular layer in the motor area of the cortex) narrow pyramidal layer and very few of giant pyramidal cells. In the Bielschowsky preparations only occasionally fibril formation could be seen, which could be interpreted as an incomplete formation of the cells. The fibers of the white matter and the intercellular fibrils in the superficial cortical

layer were also absent, which indicated an arrest of development. Anyone who has studied the subject will be convinced of the fact that the fibrillization of the ganglion cells takes place at an early time in embryonic state, and would have some doubt as to the interpretation of ganglion cells without fibrils, especially as the Bielschowsky method, in spite of all the care given of the preparations, often demonstrates in a defective manner the neurofibrils, and also because the fibrils are much disturbed by the paralytic process. From the fact that it is characteristic feature of the paralytic process to affect the medullated fibers and intracellular fibrils of the superficial layer of the cortex, it is not possible to connect the change with a disturbance of development and the same can be said in regard to the observation of the small number of pyramidal ganglion cells. Trapet described a case of paralysis in a case of an idiot, affected from birth with severe alteration of the hemispheres, which he interpreted as disturbance of development. The hemispheres showed both in the position and the form of the ganglion cells marked deviations from the normal, as well as the form of the different layers. Directly under the zonal layer was a band of cells, placed very close together, resembling neuroblasts. In these cells of an embryonic type one could see distinct evidences of cell division in the various stages of development. The internal granular layer was of an infantile character. Unfortunately, the description was not accompanied by illustrations. As Alzheimer and others have not found such cell division in persons over one year of age in a large number of cases of idiocy and juvenile paralysis studied, he deplores the fact that no illustrations were given, and is suspicious that the cells have been confused with glia elements.

Hough has described calcification of the vessels of the cerebellum in a seventeen year old juvenile paralytic. Klieneberger has described even more severe disturbance of development in the brain of a juvenile paralytic. The corpus callosum was absent and on the inner surface of the hemisphere, as is usually the case where the corpus callosum is absent, an unusually distinct radial arrangement of the sulci and convolutions and in place of the corpus callosum was found a markedly developed frontal-occipital bundle.

In any case there is much that is interesting to learn in the field of developmental changes in the cortex of the hemispheres

in the juvenile form of paralysis. However, one must be cautious not to fall into the error of calling undeveloped cells the pathological and degenerated cells in places where the cellular architecture is disturbed. A ganglion cell resembling a neuroblast is not easy to differentiate or to define. Alzheimer emphasizes that fact, that it is extremely difficult to decide whether these cells are undeveloped or degenerative types, and is in doubt as to whether one can decide the question at the present time.

Notably, Naecke has attempted to show that there is evidence in the sulci and convolutions of the hemispheres of the brains of paretics, to substantiate his theory, and also the opinion of others, that these cases have a congenital abnormal " anlage " and has an illustrated atlas to show this. But the great variation in the form of the convolutions of the human brain would lead one to doubt if such findings will be of value, not at least until a great deal of material is worked over and normal brains studied.

In an unusually capable article, Sibelius has demonstrated the anomalies of the cord in paralytics and he has contributed much to the question of the evidence of intrinsic degeneration and to congenital predisposition to adult paralysis. In studying the brains of idiots one meets many signs which indicate degenerative changes, and for the question of psychical degeneration it is possible that such studies will be of the utmost importance if we also consider that in the extraordinarily complicated brains we do not find these peculiarities which are the cause of the defective mental activity. Sibelius investigated twenty-four paralytics and fifteen normal cords where most all of the segments were studied. He described the abnormal findings with those in which degenerative changes were shown in the cell elements, and the changes shown in the architecture, the former were rare and the latter more frequent.

Regarding the disturbance in the cell elements he found three times double nucleated ganglion cells, in one case in two " spinal ganglion colonies of ganglion cells," and a spinal ganglion with double nucleated cells. He regarded these findings as those of arrest of development. The architectural abnormalities were shown partly in the central canal (Gliotic anlage). Twice central gliosis was found with vacual formation, and twice in connection with hydromyelia. They were regarded as developmental anomalies which had later undergone further changes. Other changes in the forma-

tion of the various columns of the cord and the commissures which Alzheimer regards as better evidence of congenital anomalies than the formation of tongues in the substantia gelatinosa Rolandi. Frequently there were absent the anterior columns of the pyramidal tact, or very strongly developed, markedly short, plump posterior horns and sulci formation in the dorsolateral part of the periphery of the lateral column. Every normal cord had 0.93, and every paralytic cord 2.71 anomalies, and Sibelius is of the opinion that his findings possibly speak for an endogenous predisposition for the development of paralysis.

Conclusions.

When we review the principal facts of the newer histological investigations of the paralysis process, we can conclude that this process is so well differentiated that it can be separated from all other diseases of the central nervous system. Our knowledge of the pathological anatomy is of extreme value for the clinical symptomatology of paralysis, as it allows us to diagnose cases of an atypical and extraordinary character.

Further, the newer findings indicate that the process cannot be entirely accounted for by the inflammatory process in the blood vessels. More often independent degenerative changes are seen in the nervous tissue which go hand in hand with the inflammatory changes.

The histological investigations of the other organs of the body make it more probable that the paralytic process is not only a disease of the central nervous system, but a disease of the entire body. But more investigations are needed to prove this point.

Focal or circumscribed degeneration of the medullated sheath of the axis cylinders, particularly in the cortex, which can approach even the focal lesions of multiple sclerosis, is a frequent accompanying change in the paralytic process of the central nervous system.

It is possible to differentiate a third type of paralysis which can be added to the typical cases and atypical localized forms (Lissauer's paralysis) the stationary paralysis which is characterized by a particularly mild process in the histological and clinical picture.

The paralysis on the ground of hereditary lues is associated frequently with characteristic changes in the cerebellum, particularly the abnormal form and multiple nuclei of the Purkinje cells. This

characteristic finding of the Purkinje cells is so frequently found, and in so many cases, that it is impossible to class them with isolated double nucleated cells found in the cerebellum of cases of other mental diseases. It is not entirely clear from whence these cells originate. From the fact that they are occasionally found in paralysis in middle life, and after late acquired syphilis in relation to a severe cerebellar atrophy, and are similar to the cells in juvenile paralysis, at present one cannot conclude that their presence indicates paralysis hereditaria tarda. Thorough examination of particularly atrophic cerebellum in adult paralysis is necessary before we can arrive at any conclusion.

The investigations of Sibelius indicate, possibly, that in the central nervous system of paralytics, who have acquired syphilis, that frequently developmental anomalies are present. And possibly this means that there is a specific predisposition towards a later paralysis when accompanied with syphilis.

STATISTICAL STUDIES IN SYPHILIS WITH THE WASSERMANN REACTION, WITH REMARKS ON GENERAL PARALYSIS.

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INTRODUCTION.

The subject of syphilis has been approached from all sides, studied from all standpoints, and investigated in detail wherever medical research has been conducted.

Especially since the introduction of the various biological tests and the discovery of the treponema have etiological relationship between syphilis and a multitude of morbid processes been sought, and the Wassermann reaction in particular been applied to large series of cases of diseased conditions affecting the various organs and systems of the body with results that are known to all.

In this the field of psychiatry has not been neglected, and there are already on record a number of limited investigations conducted wholly among the insane. The results of these investigations, however, have been as varied as the number of their observers, and aside from any possible connection between syphilis and mental disorders, were one merely to ask what is the percentage of syphilitic infection among the insane as a class, the answers from various quarters could not be other than confusing, and in no case would this conflict of figures be explained. This then is one of the purposes of the present investigation. By a series of examinations sufficiently extended and comprehensive to rule out all possibility of accidental and artificial influences, to determine not only with just what frequency syphilis could be found in an institution for the insane, or to what, if any, type of psychosis, other than paresis, it might stand in definite etiological relationship, but more in particular to likewise determine by a full statistical study whether or not syphilis under these circumstances differed in any particular

[1] Assisted in part collection of clinical data by William C. Sandy, M. D., Assistant Physician, State Hospital, Trenton, N. J.

of its frequency or manner of occurrence from that found in general communities, and also with the end in view of accounting for the marked discrepancies noted in previous similiar observations.

Still further it was argued, that if the results among the insane were found not to differ importantly from those which might be expected among normal individuals the figures obtained would not only be indicative of conditions among the mentally affected, but would be equally valuable as statistical records of the corresponding general communities of which these individuals were representatives.

The difficulty of obtaining even approximate statistics on venereal disease is well recognized, and the desirability from many standpoints of really accurate figures for any general population where reports are not to be had is obvious.

In fact, then, this is in part a statistical investigation of the prevalence of lues in the state of New Jersey by laboratory methods.

As a still further incentive for the investigation, it was the intention of discovering for observation purposes all cases of strictly latent lues, and by following them to autopsy to be able to finally add something to the as yet unsolved question as to the true clinical and pathological significance of a positive serum reaction in the face of a persistently normal physical existence.

Finally the general paralysis material available during the investigation has been used in an effort to assign by correlation of certain of the various reactions found in this disease, the relationship which they bear to one another and to the pathological process of the condition.

Material, Method of Investigation and Technique.

The material of the investigation consists of 1583 individuals confined as patients in the Trenton Hospital. This number includes the entire insane population of the hospital from May, 1911, to June, 1912, irrespective of age, physical health, occupational activity, private or indigent class, nationality, color or mental diagnosis.

No attempt whatever was made to include, or exclude, certain types of individuals, or to reduce or increase the numbers in certain varieties of psychoses. On the contrary, every effort was exerted to obtain statistical data which would represent with the greatest

accuracy possible the entire general population without the disturbing influence of the personal equation in selecting material. With this end in view the institution was canvassed by wards, and each and every patient subjected to examination.

Both the Wassermann and Noguchi methods, either alone or combined, were employed. It was the original intention to adhere to the original Wassermann method throughout, but in such an investigation as the present, where it was desirable to complete a large number of reactions in the shortest time, and with the minimum expenditure of effort and material, the Noguchi modification undoubtedly offers great advantages over the Wassermann and, when properly checked and controlled, as later described, the accuracy of results need suffer no reduction.

The technique employed for the Wassermann method corresponded to that originally described and included a full set of controls for every set of reactions; the quality of natural antisheep amboceptor present in each serum was noted.

The Noguchi tests were made according to the technique laid down by the originator.

The only departure from the original technique of both methods was in the antigen employed. In both procedures alcoholic extract of syphilitic fetal liver was used instead of the Wassermann watery extract and the Noguchi acetone insoluble lipoids. Only inactivated sera were tested and 2 units of hemolytic amboceptor were used in both procedures.

It cannot be denied that by using alcoholic extract in the place of the watery emulsion the number of positively reacting sera is reduced, as is shown by comparing our per cent of positive general paralysis reactions with the 98 to 100 per cent claimed by Plaut and others who used the original Wassermann antigen. However, since this error is upon the conservative side it is relatively unobjectionable, and furthermore, as will be shown, it is possible to correct this deficiency when computing the general results of a statistical study such as the present.

Concerning the accuracy of the two methods as demonstrated in this series, it is perhaps worth while to speak briefly and to record in what manner the most reliable readings were obtained.

In all, 1909 reactions were performed, including both blood and cerebro-spinal fluid; 1769 being blood and 140 fluid. This

number (1909) includes 186 repeated serum reactions and 24 repeated fluid reactions performed on different dates for the purpose of clearing up obscure readings and the elimination of any possible confusion of samples.

All of the cerebro-spinal fluid tests were made by the Wassermann method only since it was found in testing spinal fluid by Noguchi that the alcoholic liver extract brought about rapid hemolysis of the human cells.[2]

Considering, then, the serum reactions alone, there are 1769 examinations which were performed as follows:

By Noguchi Only.	By Wassermann Only.	By Wassermann & Noguchi.
1047	503	219

These reactions include those of 70 cases of general paralysis, and gave as results of a first test 104 positive, 42 doubtful, and 1613 negative.

Of the 104 positive sera, 61 were from cases of general paralysis, and 43 were patients in whom no parasyphilitic manifestations were present.

All of these 104 sera reacted positively by Noguchi and 97 reacted similarly by Wassermann, thus giving seven more positive reactions in favor of the Noguchi technique.

It is on the final analysis of the reactions of these seven cases that the conclusions reached in this study as to the value of the two methods is based, but before taking up this question it is necessary to indicate what readings are here considered respectively positive and negative.

In the Wassermann reactions two quantities of patient's serum (.2 and .1 cc.) were tested with full doses of antigen, and the final readings made 12 to 18 hours later according to Citron's scale, in which as low as a partial inhibition in the .2 cc. tube with complete hemolysis in the .1 cc. tube is counted as positive and given a value of $\frac{1}{+}$; the more complete reactions being designated 2, 3 and +. No reaction by the Wassermann system was finally called negative until after one or more repeats, if, at the first trial, even a trace of inhibition in either tube was found after 12 hours.

[2] Since this never occurred when testing blood it must be supposed that human serum contains a substance which is protective for the red cells against hemolytic antigen and which is absent in the spinal fluid.

For the Noguchi tests .08 cc. of inactivated patient's serum was employed and a positive reaction recorded only if complete inhibition of hemolysis occurred. If a trace of hemolysis was noted after 12 hours the reaction was called "doubtful positive," and final decision reserved for repeated tests. Partial inhibitions and partial hemolyses were looked upon as diagnostically valueless, but were also referred to future re-examinations. With regard to these latter types of reactions, which are particularly liable to occur with the Noguchi technique, of the 42 sera so reacting in this series 36 were later proven to be definitely negative by both methods. The remaining six, which were at first called doubtful, either because of a trace of hemolysis in the Noguchi system or because of trouble with the controls in the Wassermann, were found on retesting to be positive. As will be seen later in the tables of results no "doubtful" column appears, since, aside from obscuring conclusions, there is really no necessity of so recording reactions if opportunity exists for making repeated re-examinations at will.

Of the seven sera giving contradictory reactions by the two methods (positive by Noguchi and negative by Wassermann) five were from cases of undoubted clinical general paralysis with spinal pleocytosis and globulin excess, and two were from individuals entirely without anamnestic or clinical evidence of lues or parasyphilitic disorders. All contained sufficient natural antisheep amboceptor to bring about complete hemolysis of the sheep cells in the Wassermann test at the time of final reading, and three sufficient to completely hemolize in two hours.

On further investigaton of these sera by repeated reactions conducted simultaneously by both methods the five paralysis sera continued definitely positive by Noguchi and negative by Wassermann as was found on the first trial, thus giving the Noguchi a 5 per cent advantage over the Wassermann in positive findings.

Two of the seven, however, those of the clinically lues-free cases, now were found to be negative by Noguchi as well as by Wassermann, and so continued in any number of reactions with four different antigens.

That these two sera did in reality give a *bona fide* positive reaction on their first trial and were not so recorded through technical error is proven by the fact that all positively reacting sera were checked by retesting the same specimen on the day following

and not recorded as positive unless the reactions on the two days agreed. In no instance did the Wassermann system fail to give repeated corresponding reactions if, with controls in agreement, the first trial proved positive.

Various explanations have been advanced for the occurrence of these non-specific reactions with the anti-human system. Noguchi himself states that they are likely to occur if active sera are tested with alcoholic organ extracts and also cautions against collecting blood at a short interval following a meal when the serum is likely to contain lipoid food stuffs; Kaplan[3] has found that certain "yellow" or "orange" colored sera are likely to give such reactions, while others have emphasized the possibility of non-specific complement fixation when free hemoglobin is present in serum or complement. Whatever the explanation the occurrence of such reactions is the most undesirable error which can happen in serodiagnoses.

Since its first inception the principal objections raised against the Noguchi system was the occurrence of these very non-specific reactions and this instance seems to be a case in point, although, it must be remembered that here alcoholic liver extract, and not the acetone insoluble lipoids recommended by Noguchi, was used. On the other hand, in the United States Army Medical Service[4] as well as elsewhere the Noguchi modification with alcoholic syphilitic liver extract as antigen is the standard method of choice, and implicit reliance is placed in the readings so obtained.

But while from a critical standpoint of the methods it would be as unjust to condemn the Noguchi method for its non-specificity without fractionated lipoids as antigen, as it would be to criticise the under sensitiveness of the Wassermann when aqueous liver extract is not employed; and while it is clearly understood that criticisms of a test are not well founded unless the conditions of the procedure are rigidly adhered to, still the reactions in this series were carried out as are by far the great majority in this country, and as clearly shown in this series, both methods, if used alone, are liable to considerable error when so conducted. The Noguchi

[3] Kaplan: American Journal of the Medical Sciences, January, 1910.
[4] Craig: Journal of Experimental Medicine, 1911, Vol. XIII, No. 4, Journal of Infectious Diseases, November, 1911.

method, if it may be so expressed, is too sensitive, and the Wassermann not sensitive enough.

As found in this series there were two too many positive reactions by Noguchi and five too few positive reactions by Wassermann.

Taking 102 positive reactions (104 less the two non-specific Noguchi reactions) to represent 100 per cent accuracy in this series, the error of deficiency by the Wassermann is 5 per cent and the error of excess by the Noguchi is 2 per cent.

In view of these findings it becomes evident that neither one of these reactions by itself, when performed with the kind of antigen commonly used, will lead to results upon which reliance can be placed. What is equally true, however, is that the two reactions performed simultaneously on the same serum check one another in a manner better calculated to prevent error than any number of controls used in either test individually; and, basing our conclusions on what we have found in this series, we believe that the only reliable method of sero-diagnosis, when alcoholic extract antigen is used, consists in simultaneously testing each serum by both the Wassermann and Noguchi systems.

Such a procedure was adopted in the present investigation and in the following manner: Since the above conclusion, that the Noguchi was "too sensitive," and that the Wassermann was missing, was tentatively reached in the early part of the series it was determined to use the more sensitive Noguchi as a scout by which to first detect all sera which showed any tendency to give a positive reaction, to perform the entire series by this method, and then to follow with a detailed investigation by both methods of all those individuals whose serum had been found positive, suspicious, or doubtful by the Noguchi. This led to the rather troublesome performance of 186 separate repeat reactions, all performed on different dates, but which resulted, as already described, in correcting a 5 per cent error in the Wassermann, the detection of a much more serious 2 per cent excess error in the Noguchi and the complete elimination of all so called "doubtful" results.

Consideration of the Findings.

As already mentioned, the field covered in the investigation includes the entire hospital population during an interval of a year.

This material is statistically arranged with regard to age, sex, color, civil condition, nationality, stay in hospital and the type of psychosis of each individual examined.

With regard to the mental diagnoses used in the tables, this was determined as accurately as circumstances permitted. The case record of each patient was examined, and in many instances a new status was taken. In the cases of "functional" psychoses which had been in the hospital over long periods of time, diagnostic errors were doubtless made, but on the other hand, in a large series, such errors are very likely to compensate one another, and we believe that for the statistical purposes required there are no discrepancies sufficient to influence the general results.

In the case of individuals giving a positive reaction, and in the organic field, where because of the relatively limited number of cases a margin of error of even one or two cases might lead to erroneous conclusions, particular care was exercised in accurately determining the diagnosis. Lumbar puncture was resorted to and repeated in every case giving a positive serum reaction and in all cases where there were any suspiciously suggestive clinical features.

Since it was one of the primary objects of the investigation to accurately ascertain the frequency of purely latent lues in a mixed population such as this one, special heed was paid to general paralysis in order that the figures for this condition might be kept strictly to themselves and not unknowingly included with those of the other cases.

That these precautions were not unnecessary was strikingly proven by the fact that among 46 cases reacting positively to the serum test, and which were supposedly all instances of purely latent lues suffering from an intercurrent psychosis, five were found by one or more lumbar punctures and closer clinical observation to be in reality cases of general paralysis clinically overlooked and wrongly diagnosed.

While five cases in error among a total of over 1500 is a small number, still when these are added to or deducted from the number of actually positive latent lues cases (41) it constitutes an error of more than 12 per cent, and it is our firm conviction that failure to detect and eliminate such cases is at least partially responsible for the strikingly high figures given in some of the previous investigations of like nature.

Since it is the intention to keep separate to a certain extent the general paralysis figures, it seems well to briefly record and discuss them at this place together with some of the other findings in this condition.

General Paralysis.

Seventy cases come under this category and include all the ordinary and many of the unusual types of the disease in all its stages from incipiency to termination, and as it occurs at all ages from juvenile to senile. Foudroyant, progressive, stationary, remissive, masked, juvenile, tabetic, focal, ordinary and complicated types are all represented and no attempt has been made to influence results or to obtain a high per cent of positive reactions by selection of cases.

The serum of all cases was examined by both the Wassermann and Noguchi methods, and all of the spinal fluids by at least two methods; the Fuchs-Rosenthal cell count and Noguchi globulin reaction. The majority of the fluids were also examined by the Alzheimer differential cell method and by the Wassermann reaction.

Placed in the form of a table the complement fixation results are as follows:

TABLE I.—GENERAL PARALYSIS.

Complement Fixation Tests of Blood Serum and Cerebro-spinal Fluid.

	No. cases examined.	Positive.	Negative.	% Positive.
Serum	70	61	9	87.14
Fluid	57	46	11	80.7
Individual Cases.				
Serum only examined	13	11	2	
Blood and fluid examined	57			

Of these 57 cases in which the blood and fluid were both examined the corresponding results showed:

43 cases had a positive reaction in both blood and fluid...75.2% (of the 57)
7 cases had a positive blood and a negative fluid.....12.3%
3[5] cases had a negative blood and a positive fluid.....5.35%
4 cases had a negative blood and a negative fluid..... 7.0%
Positive reactions considering both blood and fluid in individual cases 93%

[5] The significance of such reactions will be discussed in another article.

As can readily be seen these figures do not differ materially from those obtained by other workers with a fairly extensive unselected material, and may be fairly said to represent the average results obtained, although individual figures from different quarters offer in many instances striking disagreements.

Thus Marie, Levaditi and Yamanouchi [6] found only 59 per cent of positive serum reactions in 30 paretics, while in the spinal fluid 93 per cent were positive.

Plaut,[7] and also Boas, each obtained 100 per cent of positive blood serum reactions in 180 and 42 cases respectively, although later Plaut admitted in his more extended series that he did not always get a positive result.

In this country, Kaplan's [8] and Kaliski's [8] findings in one each of their series showed respectively 65 per cent and 66 per cent positive blood reactions, while Rosanoff and Wiseman [9] obtained only 49.3 per cent positive results in the serum with 76 per cent of positives in the fluid. Nonne's [10] results were 90 per cent positive in both blood and fluid.

From the tables compiled by Noguchi,[11] comprising the work of 26 individual observers working independently, the averaged results of 660 serum examinations and 481 spinal-fluid examinations in general paralysis show 84.5 per cent positive reactions in the blood and 91 per cent positive reactions in the fluid.

As to the greater or lesser frequency of positive findings in the blood as compared with those of the fluid there has been considerable disagreement, the French investigators in particular inclining to the belief that a positive result in the fluid is more frequent, while Plaut and his co-workers in Germany are of decidedly the contrary opinion. This latter view is the one held more largely at present in this country and has been found to be true in the present series.

[6] Marie, Levaditi and Yamanouchi: Cont. Rend. Soc. de Biologie, 1908, LXIV, pp. 169.

[7] Plaut: Centralbl. fur Nervenheilk. und Psych., 1909, pp. 659.

[8] Quoted by Noguchi.

[9] Rosanoff and Wiseman: American Journal of Insanity, Vol. LXVI, Jan., 1910.

[10] Nonne: Syphilis und Nervensystem, S. Karger, Berlin, 1909.

[11] Noguchi: The Sero-Diagnosis of Syphilis. Lippincott Co., Philadelphia, Pa.

If it is really the case that the cerebro-spinal fluid is less frequently supplied with antibodies than is the blood serum, it would not be unreasonable to believe that the former was supplied with reacting substance from the latter, and that in the cases reacting positively in the serum and negatively in the fluid this transfer had failed to occur. Such a theory would imply that no antibodies were formed locally within the cerebro-spinal cavity, but such we know must occur at least in those cases which, contrary to the usual findings, give a positive result in the fluid and negative result in the blood.

Although thought to be associated with the globulin, with what bio-chemical body the reacting substance is definitely identified has not been determined, and among Plaut's cases, and also in the present series (which see), there are found individual instances where a positive reaction was obtained in the fluid in the face of a negative globulin test and a cell count well within the normal. Nevertheless, when the entire series is considered and the results of the cell counts and globulin determinations compared with the different combination types of serum and fluid reactions in the same individuals, it becomes apparent that there must be at least a very intimate relationship between the active inflammatory process (as shown by cells and globulin) taking place in the central nervous system and the behavior of the body fluids toward the complement fixation test. This relationship is shown in the following table where the different reaction groups are compared with the cyto-chemical fluid findings.

TABLE II.—GENERAL PARALYSIS.

The Relationship Between Complement Fixation and Cyto-Chemical Fluid Findings.

Complement Fixation.	No. cases.	Cells per cmm. average.	% Globulin +.
Negative blood and positive fluid.....	3	196	100.
Positive blood and positive fluid.....	43	74	95.3
Positive blood and negative fluid.....	7	35	85.8
Negative blood and negative fluid.....	4	20	75.
	57	81	91

In these figures there is seen a very striking corresponding gradation of those fluid changes (cell and globulin excess) which

may safely be regarded as the indicators of the severity of inflammatory phenomena, and that this gradation corresponds with surprising accuracy to the antibody content of both blood and fluid as shown by the fixation tests. Thus in the three cases having a positive Wassermann reaction in the fluid alone, and in which the antibodies must be supposed to be wholly derived locally from the seat of the pathological process, the cell content is more than double the average for the whole series and the positive globulin reactions reach 100 per cent. (The individual counts were 125, 293, and 171.)

On the contrary in the cases where no antibody could be detected in either blood or fluid the cells and globulin are at a minimum, and indeed in three of the four cases so reacting to the complement fixation tests there were only 2-8 and 11 cells respectively, an average of seven (only two above the normal limit), while the globulin reactions were just barely sufficient to be called positive. The fourth case showed 58 cells and gave a good globulin response, but was so decidedly atypical clinically as to arouse suspicion as to the corrections of the diagnosis (which see).

Still further it is seen that in the cases having a positive blood alone and no antibodies in the fluid, the cell counts and positive globulin reactions are still low and much below the series average; and indeed, in this group there are four cases with a cell content of only six and below (two fluids each with 6 cells and two more with 4 and 5 cells respectively).

In the " typically " reacting group, by far the larger, where both fluid and blood are positive, the cyto-chemical fluid findings of an average of 74 cells and of 95 per cent positive globulin tests, most closely approximate the average results of the series in its entirety, and are in very close agreement with the findings generally obtained in general paralysis. In this group there are only three cases in which the cell count was found to be relatively low; respectively, 9, 10, and 12.

Of course, it is not to be forgotten in the present series that the number of cases in three of the reaction type groups is small, and that perhaps more extended observations might fail to show a similar correlation between the fixation tests and the cell-globulin content of the fluid, but it must be admitted that the comparative results are sufficiently striking to suggest that in general paralysis

the presence of antibodies is largely dependent upon the activity of the inflammatory part of the pathological process in the brain itself; nor can we, when we consider the fundamental differences in anatomical relations and the radically different bio-chemical natures of the blood and fluid, allow even the more frequent finding of positive reactions in the blood than in the fluid to entirely negative this assumption.[12]

Whether or not in general paralysis the process of antibody productions and pleocytoses per se are identical or necessarily associated is another question.

Plaut,[13] because he found in cases of secondary and tertiary syphilis clinically without cerebral lues or indications of general paralysis a spinal lymphocytosis associated with a negative result of the fluid Wassermann, states as his definite conclusion that lymphocytosis and the biological fluid reaction do not go parallel, and that the mechanism of the processes are entirely dissimilar.

In lues without general paralysis this is perhaps true, although in the 41 cases of latent lues with no clinical evidence of parasyphilitic disease of the central nervous system found in this series, and which will be discussed in another section, there was not a single instance of cellular increase in the fluid on repeated examinations. But even were such the case, it is obvious that the relationship or non-relationship between spinal pleocytosis and the Wassermann reaction in paralysis cannot be argued upon the findings in cases of plain lues in either its secondary or tertiary stages.

Of more significance in this connection are the cases of paresis itself in which there is a disagreement of biological and cytological reactions. Two types of such discrepancy are possible: pleocytosis with negative Wassermann, and normal cell count with positive Wassermann. First as to cases which present a spinal pleocytosis without complement fixation.

Plaut makes mention of eight cases considered general paralysis, but on which in another section of his monograph he is very careful to throw doubt as to the diagnosis because the fluid reacted

[12] Particularly is this true in view of the fact that by using larger quantities of fluid in the test the percentage of positive reactions is very appreciably increased.

[13] Plaut: The Wassermann Sero-Diagnosis of Syphilis in its Application to Psychiatry. Nervous and Mental Disease Monograph Series, No. 5, 1911.

negatively, and of which he makes clear that he considers them as much cases of cerebral syphilis as of proven general paralysis, which showed a distinct pleocytosis and at the same time gave a negative response to the biological test.

It has already been shown in the table of comparison between cytology and Wassermann reactions in this series that in the group of cases in which the biological test failed in both the blood and fluid, the average results of the cell counts is remarkably low as compared to those reacting positively (an average of only seven if the case quoted below is excluded), and that in only one of these cases was the count significantly above normal. This case is so distinctly atypical clinically as to either throw strong suspicion on the accuracy of the diagnosis or to form an instance of correlation between cases of unusual clinical course and atypical laboratory findings.

Henry G., a traveling salesman, age forty-seven years, was admitted January 10, 1911. The father died insane in this hospital. Patient admits penile sore in the twenties; wife has had two miscarriages.

Eight years before admission while on a business trip the onset of the psychosis began with a "spell." He went to bed one night as usual, but when he awoke "his mind was a complete blank." He wandered about, wrote foolish letters and did not know where he was. This passed and after two weeks he resumed his occupation, but was very forgetful, took an undue interest in trivial things and could not retain a grasp of his duties. He improved temporarily, however, and was able to hold his position for a year. He was then discharged for incompetency. He was neither euphoric nor depressed, but lacked interest. Later he was again able to work, but again there was a short interval of complete disorientation and amnesia without convulsions or coma. He was committed because of assault on his wife.

On admission he was oriented, spoke quietly and said he had been sick. Physically the knee-jerks were a little exaggerated; the pupil light reflex was present, but diminished in range. A slight tremor of speech was noted, but writing was without defect. Sensory phenomena were absent and motility of the various muscle groups was unimpaired. There was a slight tremor when the fingers were extended.

The mental status showed principally an emotional apathy without trends. At this time he was perfectly oriented on all points and was able to give an accurate account of his past. No date discrepancies could be elicited. Memory and retention tests all gave good responses and he appeared well informed on current events. Occasional mistakes in calculation were noted, but as a whole these tests were also well given.

Although he expressed interest in the examination there was a manifest indifference to the situation. He seldom volunteered a statement, made no

persistent requests and seemed equally content to go or remain. He admitted that he had been sick and that at times his memory was bad, but saw no occasion for his commitment. Euphoria, delusions, hallucinations and trends all conspicuously absent. Lumbar puncture showed 58 cells per cmm., positive globulin response and plasma cells by Alzheimer's method. Both blood and fluid were negative to the complement fixation test.

After admission there was no considerable change save that the slight tremor and speech defect became scarcely noticeable. Memory tests continued to be always well given. A staff meeting note at discharge states "deterioration cannot be demonstrated."

Eight months after admission on request of the wife the patient was discharged. On returning home he obtained work and up to the present time (10 years after onset) continues to hold his position as a mill worker, although considerably aged in appearance and physically feeble.

In the hospital this case was diagnosed "general paralysis," largely upon the history of a probable previous syphilitic infection, the peculiar attacks and the spinal pleocytosis. The case notes, however, fail to show that cerebral syphilis was at any time considered or eliminated in the differential diagnosis. Of course it may well be that the case is in reality one of atypical general paralysis with prolonged more or less stationary course and remissions, but the preponderance of the clinical evidence is certainly more indicative of Kraepelin's syphilitic pseudo-paresis with endarteritis than of genuine general paralysis.

As before intimated, if this case is withdrawn, there are no cases of paresis in this series showing negative Wassermann reactions in both blood and fluid in which pleocytosis occurs.

With regard to discrepancies in the other direction between cytology and fixation tests, Plaut has also described eight cases considered as general paralysis, of which five were in the early stages, one tabo-paralysis and two in which the diagnosis lay between paresis and cerebral syphilis, in which the cell count was 10 or under, but which gave a positive biological fluid reaction. Three such cases were found in the present investigation and certain features of their findings warrant emphasis. Clinically all three are classical general paralysis and display the typical physical and mental signs of the condition. One has already died and come to autopsy where the diagnosis was fully confirmed.

The first patient, Annetta J., a sixty-five year old colored woman, was at once diagnosed as paresis on admission, but although typical enough clinically, the determined diagnosis was withheld because on lumbar punc-

ture but 1 cell per cmm. could be counted and the globulin test was negative. Shortly after a blood Wassermann test gave a ++++ reaction and paresis seemed still more probable. Lumbar puncture was again performed and again the count was normal (4 cells) and the globulin not in excess. The fluid Wassermann, however, was positive.

In view of these findings, here, one would say, was a case of paresis with no pleocytosis together with a positive biological fluid reaction. But a third lumbar puncture made only two months later showed 25 cells per cmm. and a good globulin response and still further lumbar punctures (Nos. 4 and 5) at farther intervals of one and five months showed respectively cell counts of 7 and 44.

The second case is that of a forty-five year old woman (Lena Y.) who, on admission, showed all the classical signs of paresis and was so diagnosed. Lumbar puncture gave 3 cells per cmm. and no excess globulin. The blood serum gave a ++++ reaction. At this time the fluid reaction was not tested. One year later the fluid showed 9 cells, positive globulin response and reacted positively to the complement fixation test. A third spinal puncture twelve months after the second showed 42 cells and no change in the biological reactions.

The third case is similar to the other two and need not be further described.

The point to be taken from these findings is: When are we justified in saying that a given case of general paralysis shows no pleocytosis? In none of Plaut's cases is more than one spinal puncture in each case mentioned. If we are content with the findings of one puncture alone we will doubtless not infrequently say that a case of paresis has no pleocytosis, but at the same time does give a positive biological reaction in the fluid, and that therefore the mechanisms of the two are entirely different. But if on the other hand we find by repeated punctures that in these cases apparently without pleocytosis there is only a temporary remission of cell excess wherein we are unable to demonstrate the continued presence of the process by our relatively crude method of mere mechanical enumeration, while with the more delicate biological test antibodies are still detected, we are not in a position to say that the underlying mechanisms responsible for both conditions are not the same.[14]

[14] The findings by Alzheimer's method of fluid examinations is a point still further in favor of this argument for it is possible without exception to find plasma cells even in those cases with a cell count diagnostically entirely negative and well within the normal.

This question obviously needs further study, but the findings in this series indicates that there is as much for one side of the question as for the other, and that it cannot be decided offhand on the evidence of one lumbar puncture in a limited number of cases.

General Results and Statistical Tables.

In perusing the literature dealing with investigations on the frequency of syphilis in various institutional populations, the most striking feature encountered is the enormous discrepancies in the results obtained. In practically none is there any semblance of agreement.

From the findings in some instances one would conclude that, as revealed by sero-diagnosis, in syphilis we have the basis for a preponderance of all sorts of varieties of psychic disorders, mental deficiencies and congenital defects. From others on the contrary one would be led to believe that not only was syphilis in no way at the bottom of the conditions, but that such types of population were unusually free from infection.

Thus, Raviart, Breton and Petit [15] in France found 28.7 per cent of positive reactions among 323 insane, very largely idiots, epileptics and organic dementias, but not including paralysis; and Roubinowitch and Levaditi [16] found 25 per cent of positive reactions in dementia præcox alone. Raviart, Breton and Petit frankly state that they selected their material. Lippmann [17] in Germany obtained 13 per cent positive results with the reaction among idiots, but was able to demonstrate evidence of infection in 40 per cent of the same material by using both serum reaction and physical examination. On the other hand, Brown [18] in this country found only 1 to 1.5 per cent of hereditary syphilis in his idiot material, and Wachsmuth [19] saw not a single case among 185 idiots in the Institution at Merxhausen!

The most extensive and complete previous statistical study with the Wassermann reaction is that reported by Thomsen, Boas et al, in Denmark.[20] These investigators applied the reaction to 2061

[15] Raviart, Breton and Petit: Cont. Rend. Soc. de Biologie, 1908, p. 358.
[16] Roubinowitch and Levaditi: Cont. Rend. Soc. de Biologie, 1909, p. 880.
[17] Lippmann: Munch. Med. Woch., 1909, p. 2417.
[18] Brown: Neurolog. Centralbl., 1887, p. 453.
[19] Wachsmuth: Arch. für Psych., 1901, p. 34.
[20] Boas, Thomsen, Hjort and Leschly: Berlin Klin. Woch., 1911, p. 891.

individuals which included the entire population, both rural and urban, of feeble minded, blind, deaf and dumb and epileptic of all ages confined in the institutions of Denmark. Among the entire 2061 only 31, or 1.5 per cent, were found to give a positive response, and among the epileptic group (259) the per cent positive was only .39. These authors very justly conclude that, in Denmark at least, there is no connection between feeble mindedness and syphilis.

In the United States there have been no extensive systematic investigations.

Rosanoff and Wiseman [21] examined 406 cases of various mental disorders in the King's Park State Hospital, and out of 333 cases not general paralysis or cerebral lues found 15.6 per cent positive, although their positive findings in paresis were only 49 per cent. Their material came entirely from large cities and was doubtless selected since the number examined constituted only a relatively small proportion of the institutional population.

Ensor [22] reported 22 per cent positive reactions among 262 male inmates (including paretics) of the Mount Hope Retreat (Baltimore), and Atwood [23] obtained 14.7 per cent positive results in 204 idiots in New York City institutions.

As can be seen these investigations were either directed toward some particular type of disorder, or were, as in Rosanoff's and Wiseman's, where all types of insanity were included, very limited in numbers. Furthermore all of the investigations referred to in this country have been conducted with material wholly or largely derived from cities, and similar figures for rural districts by contrast are lacking. That this factor is largely responsible for the failure of agreement of results in general is shown by the findings of the present investigation which, when compared with others, sharply emphasizes the great importance of the rôle played by the type of population as an influence on the frequency of syphilitic infection as found in institutions.

The type of population here considered is very largely distinctly rural. The territory of the 10 counties from which the hospital inmates are derived is very largely given up to agricultural pur-

[21] *Loc. cit.* American Journal of Insanity.
[22] Ensor: Jour. of the Amer. Med. Assoc., 1910, p. 216.
[23] Atwood: Jour. of the Amer. Med. Assoc., 1910, p. 464.

suits, and the population is either entirely scattered or divided among small towns and villages. In but two of the counties are there cities of any considerable size (90,000).

As in most state hospitals the average social status of the inmates would be designated as the "lower middle" and "laboring" classes, the majority being laborers, small farmers, clerks, domestics, mechanics and factory workers, and for the most part of sober and industrious habits. Twenty-six per cent are of foreign birth and 5½ per cent are negroes. The ages range from 9 to 93 years, the majority being in the 4th, 5th, and 6th decades.

As before stated every inmate of the institution was examined and the figures obtained may safely be regarded as truly representative of the frequency of latent lues in communities of the type described.

For statistical purposes we have classified the findings with regard to the psychosis, age, sex, nationality, color, civil condition and geographical distribution.

In order to determine what, if any, influence the time factor would have on the presence of the reaction under the present circumstances we have also compiled a table with reference to the number of years' residence within the hospital.

Table III shows the general results in the total population.

TABLE III.—GENERAL RESULTS OF THE SERA-REACTION IN THE HOSPITAL POPULATION.

	Positive.	Negative.	Total.	% +.
Total, including cases of general paralysis.	102	1481	1583	6.3
Not including cases of general paralysis	41	1472	1513	2.7
Cases of general paralysis	61	9	70	87.15
Total syphilis				7.00

It is at once noticed that both the aggregate per cent positive figures and the per cent positive without paresis are strikingly low and at wide variance with the reported findings of others.

In partial explanation of their high figures (15.6 per cent in cases of insanity not paresis) Rosanoff and Wiseman suggested that there were factors in certain forms of insanity which of themselves might predispose toward acquiring luetic infection, and

Blaschko[24] stated that syphilitics represented a two and one-half times larger proportion of the mentally diseased than corresponded to their percentage of the total population.

The present investigation fails to support any such claim, either that individuals who are of a make-up upon which insanity is likely to develop are prone to be more exposed to syphilitic infection, or that syphilis is more frequent among the insane than in any other community. On the contrary, we believe that the proportion of syphilis among the insane is exactly proportionate to that of the type of general community in which they have had their original environment.

The percentage of positive results in this table among the total number of all psychoses not paresis is so strikingly low (2.7 per cent) that not only in the aggregate is there obviously no connection between lues and "insanity" as cause and effect, but that here the insane as a class certainly appear as not necessarily more predisposed by peculiarities of moral and ethical behavior to acquire syphilitic infection.

This low figure is in strong contrast with some of the results of other investigations already quoted (e. g., Rosanoff and Wiseman, 15.6 per cent; Lippmann, 31 to 40 per cent; Raviart, Breton and Petit, 28.7 per cent), and since the only discernible difference in the material investigated in different instances lies in the fact that on one hand it was entirely urban and on the other strictly rural, it must be regarded that this factor, viz., the type of population, has more to do with the incidence of syphilis than any individual peculiarity of make-up or of physical or mental trend.

Blaschko[25] found that 20 per cent of the total male population of Berlin is infected with syphilis, and Cabot estimated that the figures were at least as high for cities in this country; yet if we include all of the cases of general paralysis, who are certainly syphilitic whether reacting positively or not, the total incidence of syphilis in the male population in this investigation is only 9.8 per cent (82 syphilitics out of 802 individuals), or just one-half that found in urban communities.

That this factor of the type of population is the real reason for the discrepancies found in different investigations is still further

[24] Blaschko: Verhandlungen der Berliner. Med. Gesellschaft, 1908, p. 102.
[25] *Loc. cit.*

emphasized by the figures for the individual counties which furnish the institutional population, as shown in the following table, where the male cases (not including general paralysis) are considered alone.

TABLE IV.—THE RELATION OF THE SERO-REACTION TO THE TYPE OF COMMUNITY.

(Not Including Cases of Paresis.)

County Residence before Admission.	Positive.	Negative.	Total.	% +.
Camden (City of Camden)	4	46	50	8.00
Mercer (City of Trenton)	10	197	207	4.83
Monmouth (Rural)	3	99	102	2.94
Hunterdon (Rural)	1	33	34	2.94
Middlesex (Rural)	4	149	153	2.61
Somerset (Rural)	1	43	44	2.2
Ocean (Rural)	0	39	39	0.0
Other Counties (Rural)	2	91	93	2.15
	25	697	722	3.6
Counties with cities	14	243	257	5.4
Counties without cities	11	454	465	2.3

For an understanding of the above figures it is necessary to explain that the two counties of Camden and Mercer contain the cities of Camden and Trenton respectively, each with a population of 90,000, and the only two cities of size in the territory of the institution. It may be added that the city of Camden is only separrated from Philadelphia by ferry. The other counties tabulated are all of rural type and have no cities of importance.

It is at once seen that the relationship between the amount of syphilis in cities and country before noted in comparing the total syphilis (including paresis) among males in the institution (9.8 per cent) and the Berlin figures (20 per cent), is still exactly the same, viz., twice the number of cases in city communities (5.4 per cent) as compared to rural communities (2.3 per cent), and that again in this respect there is no difference between syphilis in general and syphilis among the insane.

As to the relation of the sexes the results are also in agreement with what is generally known to be true in general—that lues is considerably more frequent in men than in women.

TABLE V.—SEX.

(Not Including Paresis.)

	Positive.	Negative.	Total.	%+.
Male	29	748	778	3.87
Female	12	723	735	1.65

According to Blaschko [26] the proportion in general communities (not insane) is 4 to 1. It will be seen from the above figures that there is no essential difference in this respect in the findings in a general community and among the insane, and in such agreement we may see an additional argument that insanity per se has no influence on the manner of occurrence of syphilis.

Still further is this emphasized in the figures with reference to the civil condition and nationality.

TABLE VI.—CIVIL CONDITION.

(Not Including Paresis.)

Total—Male and Female:	Positive.	Total.	%+.
Married	18	703	2.56
Single	21	808	2.59
Male:			
Married	10	284	3.40
Single	19	494	4.00
Female:			
Married	8	419	1.93
Single	2 [27]	314	.63

From this it appears that while taken in the aggregate there is no difference between the frequency of lues among the married and unmarried, when the sexes are separately considered with regard to this point it is seen that among the insane as among the normal it is the single male who is the chief offender (4 per cent), that the single female has maintained her virtue (0 per cent), and that the married woman has paid the price of her husband's lapses (1.93 per cent).

Still further, in Table VII of nationality, do we see that the luetic taint of the insane follows the same pathways as among the sane.

[26] Quoted by Plaut.

[27] Both cases imbeciles with hereditary lues confined in the hospital since childhood.

TABLE VII.—NATIONALITY.
(Not Including Paresis.)

	Positive.	Total.	%+.
Foreign born	6	409	1.46
Native born	30	1020	2.94
Negro	5	79	5.95
Average			2.70

For here is shown the relative freedom from lues of the agricultural European peasant class, the average figure for the native-born and the effects of the well-known immorality among the negroes.

From all of the foregoing tables it cannot fail to be realized that syphilis among the insane differs in no way in frequency or manner of occurrence from that among the sane, and that for this reason the figures obtained in an insane community are a reliable guide to similar conditions existing in the general communities wherein the individuals examined have resided.

Furthermore, as before stated, and also in view of these facts, it is clearly shown that insanity of itself exerts no influence whatever on the frequency of lues, but that the latter is entirely dependent upon the type of the general community, whether urban or rural, in which any given investigation is carried out.

Of course, the mere fact that syphilis is more frequent in cities is in general a well recognized fact, and for obvious reasons, but very exceptionally are conclusions in this respect reached by accurate statistical studies, and the figures are as a rule only approximate.

It is thought that the present investigation not only serves to point out the real reason for the widely varying results previously reported, but in addition gives statistical data based on exact methods which will form a basis for future comparison and furnish definite knowledge as to what proportion of certain types of population, regardless of insanity, is suffering from latent lues.

Findings in the Various Psychoses.

As already shown by the figures of the general results, there can be traced no relationship between syphilis and insanity (using the term in its incorrect general sense) as cause and effect in either direction.

It remains to be seen whether in this series there is anything of significance in the findings in individual psychoses.

Tables VIII and IX show the material grouped according to the determined mental diagnosis.

TABLE VIII.—THE SERO-REACTION IN THE VARIOUS PSYCHOSES.

	Positive.	Negative.	Total.	%+.
General Paralysis	61	9	70	87.15
Organic Neurologic Conditions[28]	1	7	8	12.5
Arteriosclerotic Brain Disease	5	52	57	8.76
Alcoholic Psychoses and Drug Addictions	6	104	110	5.45
Paranoid Conditions	2	49	51	3.92
Imbecility and Idiocy	3[29]	94	97	3.09
Dementia Præcox	18	742	760	2.36
Constitutional Inferiority	1	49	50	2.00
Senile Dementia	2	99	101	1.98
Manic Depressive Insanity	3[30]	201	204	1.47
Epilepsy	0[29]	51	51	0
Toxic Exhaustive Psychoses	0	10	10	0
Psycho-Neuroses	0	14	14	0
Total	102	1481	1583	6.3
Without cases of General Paralysis	41	1472	1513	2.7

TABLE IX.—PSYCHOSES BY SEX.

	Male.			Female.		
	Total.	Positive.	%+	Total.	Positive.	%+.
Organic Neurologie	6	1	16.6	2	0	0
Arteriosclerosis	31	5	16.13	26	0	0
Alcohol and Drugs	94	6	6.4	16	0	0
Paranoid Conditions	17	1	5.87	34	1	2.94
Imbecility and Idiocy	51	1	1.96	46	2	4.34
Dementia Præcox	401	12	2.96	359	6	1.66
Constitutional Inferiority	29	0	0	21	1	4.76
Senile Dementia	36	1	2.77	65	1	1.53
Manic Depressive Insanity	71	2	2.81	133	1	.75
Epilepsy	30	0	0	21	0	0
Toxic Exhaustive Psychosis	3	0	0	7	0	0
Psycho-Neuroses	7	0	0	7	0	0
Total	778	29	3.87	735	12	1.65

[28] Including 2 cases of cerebellar disease, 1 of postero-spinal sclerosis, 1 of spastic paraplegia (syphilitic), 1 of Freidreich's ataxia, 2 of Huntington's chorea and 1 of insular sclerosis (autopsy).

[29] One positive case is a low grade congenital imbecile who has epileptic convulsions.

[30] One of these is an unclassified depression.

As a contrast to the figures in this table the results obtained in some of the previous investigations already mentioned may be quoted more in detail.

Raviart, Breton and Petit[31] reported 30.8 per cent. positive results in cases of idiocy, "semi-idiocy," and imbecility with and without epilepsy (40 per cent in "semi-idiocy" and imbecility with epilepsy), 16 per cent in epilepsy, 26 per cent in dementia præcox, 30 per cent in "organic dementia" (arteriosclerosis ?) and 60 per cent in senile dementia.

Rosanoff and Wiseman[32] found 9 per cent positive reactions in involutional melancholia, 17 per cent each in epilepsy and dementia præcox, 19 per cent in manic depressive insanity, 30 per cent in alcoholic psychoses and none in arteriosclerotic brain disease and senile dementia.

The enormous discrepancies in the results of all these investigations are obvious.

The principal cause of this disagreement has already been pointed out as due to the type of population investigated, but it is also to be noted that in both of these previous reports the material was selected and very limited as to numbers. Raviart examined only 19 cases of dementia præcox and only 11 cases of "organic dementia."

Rosanoff's table shows only 21 cases of manic-depressive insanity, 10 cases of alcoholism, 9 cases of arteriosclerotic brain disease, etc.

In the table of the present investigation where each of the important psychoses is represented by figures sufficiently large to give fairly representative results, we see that in all of the so-called functional disorders, with the exception of alcoholic and drug addictions, the distribution of positive reactions is strikingly uniform, and that there is no significant differences in the percentages ranging from 1.47 per cent for manic depressive insanity, the lowest, to 3.92 per cent for paranoid conditions, the highest; certainly no differences to which any significance could be given.[33]

[31] *Loc. cit.*

[32] *Loc. cit.*

[33] We have investigated the positive cases of paranoid condition with Kraepelin's syphilitic paranoid psychoses in mind, but can find no relationship between lues and the mental disorder.

That the highest percentage of positive results among the functional psychoses should be found in alcoholics and drug habitués (5.45 per cent) is a finding that certainly might well be expected, aside from any question of insanity, from what is known of the habits of these individuals. In this instance there is still further reason for the relatively high figures in the fact that these individuals are not really representative of the type of general population investigated. For actual investigation of the histories of the cases of this type which reacted positively showed that one-half of these individuals were not state residents at all, but were commitments from outside the state, and that in each instance the individual in question had been a chronic wanderer from city to city.

The highest figure, aside from general paralysis, is that for the organic neurologic conditions, but the number of these cases examined is here too small to permit discussion.

Of all the true psychoses (other than paresis) the most striking results are found in the figures for arteriosclerotic brain disease, and in view of the notably and uniformly low proportion of positive reactions obtained in all other types of mental disorder, they must be accorded some significance even as mere statistical data.

That there is a definite clinical significance as well is shown by investigations of the case records. All five of the positively reacting cases belong to the focal type of cerebral vascular disease and all present various forms of focal paralysis (for the most part either total or partial hemiphlegia). The age of onset in three is under 50 years (47), one at 51 and one at 67; in each instance the initial symptom was cerebral insult. Aside from the positive serum reaction there are no definite signs of lues, but the age of onset of the vascular disease alone is sufficient to clinically indicate the etiology. In none is there spinal pleocytosis or globulin excess (repeated punctures).

These cases are clearly differentiated from the diffuse type of non-syphilitic arteriosclerotic brain disease, in particular from the focal brain disorders of senility, with or without senile dementia, and from true cerebral syphilis. They are pure forms of focal cerebral lesion dependent upon vascular degeneration, the result of long standing "latent" lues.

The frequency of this type of case in a group of all types of arteriosclerotic brain disease in both sexes in this series is shown

to be 8.76 per cent, in which the sero-reaction alone is depended on to indicate the etiology. It is undoubtedly more frequent since by no means all cases with focal cerebral lesions and a past history of syphilis react positively. Furthermore, in this series there are included under the general head of "arteriosclerosis" 26 cases among senile women which are tentatively called "diffuse arteriosclerosis," but which in reality may very well be, and probably are, all cases of simple senile dementia.

If the sexes are considered separately (Table IX) it is seen that in the female column there is not a single positive result in "arteriosclerosis," but that all five positive reactions occurred among 31 male patients, which number includes all types of arteriosclerosis, focal, diffuse, senile. On this more nearly correct basis the number of positive reactions in these cases reaches 16.13 per cent, more than double that for any other psychosis and almost eight times the figure for senile dementia.

It is unnecessary to state that it is realized that the relation between syphilis and the focal brain disorders of middle life is well recognized, although some of the similar previous investigations have failed to show it, but the object here has been to show definitely by means of actual figures just how frequently such cases may be looked for in a sufficiently extended series of individuals of the population type investigated.

That the percentage of positive reactions in arteriosclerotic cases is not only conspicuously larger than that in any other psychoses, but in fact is almost as high as for all other psychoses combined is clearly indicative of the fact that in these cases we have, aside from general paralysis (and cerebral lues), the only type of psychosis to which syphilis stands closely and definitely related as real cause and effect.

Obviously the cases of failure in mental development from congenital conditions—idiocy and imbecility—come under a separate category and here, of course, we deal with hereditary lues. There can be no doubt from the large proportion of hereditary syphilis among individuals of this class found by many observers that lues plays the etiological rôle in at least a portion of these conditions. The careful work of Lippmann, in particular, who used both clinical observation and the serum reaction and found 40 per cent of his idiot material in Germany to be congenitally syphilitic, shows

that there is substantial basis for this view. That this is not constant, however, but subject to wide variation under varying circumstances is shown by the already quoted even more comprehensive work of Boas and Thomsen, who in Denmark obtained only 1.5 per cent positive reactions in a material of 2061 feeble-minded individuals.

We believe that what we have shown to be true of the frequency of syphilitic infections in general holds true for the proportion of hereditary lues in idiocy and imbecility. That this factor is very largely dependent upon the type of population investigated. If this is such that luetic infections are frequent and abundant, then there is a proportionate number of hereditarily acquired infections found in the idiots and imbeciles, and if in the general community syphilis is relatively infrequent the same is the case with the congenital defectives, and other causes must be assigned for their occurrence. (Lippmann's rural material gave only 9.3 per cent positive serum reactions while his city material gave 13.2 per cent; Boas' material was largely rural.)

Still another factor in causing discrepancies in results in determining the proportion of hereditary syphilis in idiots and imbeciles by means of the serum reaction alone is the question of the age of the individuals at the time the examination is performed.

Lippmann called attention to the fact that in its behavior toward the serum reaction hereditary syphilis behaved like acquired syphilis, and that the older the infection the less likely was a positive result to be obtained. In his own work he was very careful that all of his patients should be under 20 years of age.

Boas' material included all ages from under 5 to over 40 years, and although in none of the decades were his results other than low, still in his table, which shows 3.8 per cent positive reactions under 10 years, 1.4 to 1.7 per cent from 10 to 30 years, and only .9 per cent over 40 years, there is excellent evidence to show that this factor exerts a strong influence upon the results.

The idiot and imbecile material in the present investigation is almost entirely adult. Only eight are under 20 years of age, while 60 per cent are between 20 and 40 years, and 25 per cent over 50 years. Of the eight cases under 20, one reacted positively, thus giving 12.5 per cent positive for this group which is almost identical with Lippmann's figures obtained by using the reaction alone (13.2 per cent).

In view of the unquestionable importance of this age factor and the fact that our material is practically entirely adult, we do not feel that definite conclusions as to the frequency of syphilis in the idiots and imbeciles of this material are warranted. It may be pointed out, however, that the positive results are low (3.09 per cent); are in accord with the average frequency of syphilis for the series, and are probably relatively indicative of the frequency of syphilis in this class of patients coming from an environment such as described.

The Material from the Standpoint of Age and Number of Years in Hospital.

It was thought that perhaps in a fairly comprehensive series such as the present, where all data were available, it would not be without interest to see what influence, if any, would be observed as a result of the different decades of life in which the patients were examined.

Ordinarily we would, of course, expect to see the greatest frequency of syphilitic infection in individuals from 20 to 40 years; that is, the greatest amount of recent infection.

A hospital population, however, differs from a general community, in the fact that the average age of the population is greater and that the time of the infection bears no relation to the time of examination; *i. e.*, the infections are of more or less long standing and are latent. From this we would expect that the maximum of syphilis would be advanced in the decades and occur at a somewhat later period. That this is actually true is shown in Table X, where the reaction results are given by decades and where for comparison the number of cases of general paralysis found in the institution during the investigation are given in a separate column.

TABLE X.—AGE BY DECADES.

Age.	Positive.	Negative.	Total.	% +.	No. of cases of paresis.
20- 30	5	234	239	2.00	2
31- 40	11	310	321	3.42	16
41- 50	14	321	335	4.17	26
51- 60	4	302	306	1.30	21
61- 70	7	192	199	3.64	4
71- 80	0	85	85	0	1
81- 90	0	26	26	0	
91-100	0	2	2	0	
Total	41	1472	1513	2.70	

TABLE XI.—AGE BY SEX.

Age.	Men.			Women.		
	Total.	Positive.	%+.	Total	Positive.	%+.
Under 20	16	0	0	8	1	12.5
21- 30	125	3	2.4	90	1	1.11
31- 40	187	9	4.81	134	2	1.49
41- 50	179	8	4.46	156	6	3.84
51- 60	140	3	2.14	166	1	.60
61- 70	83	6	7.22	116	1	.86
71- 80	38	0	0	47	0	0
81- 90	10	0	0	16	0	0
91-100	0	0	0	2	0	0
Total	778	29	3.87	735	12	1.65

Here it is seen that beginning in the third decade with 2.0 per cent positive reactions (the second having too few cases to be of use), the positive findings increase in the fourth to 3.42 per cent, and in the fifth decade reach the maximum of 4.17 per cent. Following this there is a drop to 1.3 per cent in the sixth decade and again an increase to 3.64 per cent, and among males even to 7.22 per cent, in the seventh decade.

It will be noticed how closely this rise and fall of positive results follows the general paralysis figures, and how the maximum figure for each is reached in the fifth decade. From this comparison one might suspect that these positively reacting latent lues cases had something in common with paresis in so far that the greatest number of each were to be found in an institution for the insane at the same period of life, and that some of these so-called latent cases might after all owe their mental condition to syphilis. It so happens that among the 14 individuals reacting positively in this decade there are three cases which, although otherwise diagnosed and giving persistently negative spinal fluid findings, are still such that clinically one cannot rid one's mind of the suspicion that they are related to the syphilitic mental disorders. But since such signs as pupil fixity may occur and persist in wholly latent lues cases not mentally affected, and that about 20 per cent of cerebral syphilis cases give negative lumbar puncture, this point cannot be determined till autopsy.

The striking decrease in positive results in the sixth decade and the equally marked rise in the seventh is accounted for by what

has been demonstrated as to the age mortality among syphilitics and by the fact that here is felt the influence of the special kind of population (insane) under consideration.

Blaschko[34] is authority for the statement, basing his conclusions on an extensive study of life insurance figures, that the mortality of syphilitics between the thirty-sixth and fiftieth year is more than double that for the entire population including the syphilitics themselves.

In view of this it is not remarkable that the number of positive Wassermann reactions found in any population should show a sudden drop following the fiftieth year. That there should be such a striking rise in the later seventh decade, however, must be otherwise explained. A hint as to the reason for this is seen in comparing the figures for the two sexes in Table XI, where in the female column the positive per cent shows a consistent decrease after the fifth decade, but where the male findings rise from a consistent decrease to 2.14 per cent in the sixth decade to a discrepant 7.22 per cent in the seventh.

It will be remembered that all the cases of focal brain disease with a positive reaction occurred among men, and investigation of their present ages shows that it is these very cases (four of them) which causes the unduly high figure. These cases represent individuals who at the appointed time (the late forties and early fifties) were visited with the frequently terminal scene of luetic sequellæ (stroke), but who did not die, and passed on to a late admission to an insane hospital.

With regard to the positive cases themselves in general, is there anything peculiar to them? As already shown in the tables they occur at all ages in all psychoses, and with the exception of those with focal brain disease originating in middle life there is certainly no definite traceable connection between their syphilis and their psychic disorders. On the other hand, it is not without interest to see if they present anything of note simply as cases of syphilis. It is to be understood that they are all of the latent type, that is, they present no diagnosticable clinical evidence of lues, and the greater number (about 75 per cent) have reached the tertiary period. An idea of the approximate duration of the infection is

[34] *Loc. cit.*: Verhand. der Ber. Klin. Geselsch.

gained by examination of Table XII, which shows the occurrence of the positive cases with regard to the number of years' residence in the institution.

TABLE XII.—HOSPITAL POPULATION BY YEARS OF RESIDENCE.

Years in Hospital.	Positive.	Negative.	Total.	% +.
1- 3	13	563	576	2.2
4- 6	11	209	220	5.0
7-10	4	159	163	2.45
11-13	4	124	128	3.1
13-15	5	100	105	4.76
16-18	2	70	72	2.77
19-21	1	59	60	1.66
22-24	1	48	49	2.0
Over 25	0	120	120	0
Total	41	1472	1513	2.7

As is well known, only about 40 to 50 per cent of inactive (latent) tertiary cases react positively to the Wassermann test, and in well treated cases still fewer. These individuals here then represent the persistently positive reacting type of case who carry their positive Wassermann with them to the grave. In other words they are chronic spirochæte carriers (?).

Were such not the case we would expect to see in the Table some connection between the period of hospital residence (approximate duration of infection) and per cent of positive findings. That is, the longer the stay in the hospital, and hence the further from the time of initial infection, the fewer would become the percentage of positive reactions.

That this is not the case is plainly evidenced from the scatteringly irregular distribution of the positive cases among the population of various periods of residence, and it is apparent that the per cent positive is practically just as high at all periods up to twenty-five years.

What becomes of the chronic spirochæte carrier is an interesting question, particularly as to those who safely pass the usual general paralysis-tabes-cerebral hemorrhage age.

Matthes (quoted by Plaut[35]) followed for 20 years the fate of

[35] Plaut: Allgemeine Zeitsch. für Psych. und Psychisch-Gerichtliche Med., 1909, p. 340.

syphilitics who, during a definite time had come under observation at the Jenens Clinic, and found that only 1 or 2 per cent developed general paralysis. Blaschko [36] estimated that about one-third of all luetics died as a result of their syphilis. But even this leaves a wide margin and we know that there are healthy, sound individuals who, with a positive Wassermann reaction in their blood, become the parents of healthy children, live to a ripe old age, die of purely intercurrent diseases, and even at autopsy fail to show pathological processes to which an etiology of syphilis can with assurance be assigned.[37]

In general medicine, where matters are urgent and must be dealt with in a practical way, it is these very cases of long standing purely latent lues which present the greatest difficulty in interpreting a positive Wassermann reaction. From a *practical,* as well as scientific, standpoint, just what does a positive serum reaction mean in the face of a twenty- or thirty-year-old infection and no symptoms? Does it always mean that some insidious process due to lues is playing out its rôle; that although inactive, the spirochætæ are still present; that these individuals are still syphilitic; or may it at times simply signify that at some time the patient has had syphilis? We do not know. How can such patients be advised? Should they take treatment, and then if the reaction is still positive, still more treatment, and then yet more? Must they be forbidden to marry even after they have been saturated and resaturated with treatment simply because a non-specific biologic test is against them? Will they develop gumma, tabes, paralysis or aneurysm, or will they die peacefully at eighty of broncho-pneumonia? In short, what is the real prognosis of such cases, what is their ultimate outcome and what process or processes can be found at the base of such a conflict between demonstrated clinical facts and scientific deductions? It is to the end of contributing something on these points in the future that the latent lues cases now under observation have been collected.

It can hardly be doubted that all cases of paresis are, before the actual development of the disease, included in this class of so-called chronic spirochæte carriers; but why one group of these individ-

[36] *Loc. cit.*

[37] We have already had such an autopsy on one of our positively reacting cases which will be reported in another place.

uals should become paretics while the remainder escape is another question which has not as yet been solved. In his exhaustive article on "Die Lues-Paralyse-Frage,"[38] Plaut has fully discussed this problem and among other hypotheses mentions that of Hirschl —that an inflammatory invasion of the pia during the active stages of syphilis is responsible for the later development of paralysis, and argues support to this theory by the cerebro-spinal fluid findings in syphilis without actual involvement of the brain and cord.

Nonne[39] states that 40 per cent of all cases of lues show spinal lymphocytosis, and Plaut himself found that the cells were either distinctly (14 to 77) or slightly (6 to 8) increased in 48 per cent of syphilis in all its stages, and even as high as 25 per cent in the latent tertiary period. Plaut's cases together numbered 27. In this series we have had the opportunity of examining the fluid in 40 latent lues cases, all with possibly few exceptions, in the latent tertiary stage. All have had two or more lumbar punctures. In two there was a very weak or doubtful Noguchi globulin reaction, but in one only was a pleocytosis discovered; 77 cells on the first puncture, 1 or 2 only on two subsequent trials. This case came to autopsy and showed a spot of softening which had broken into the ventricle, thus fully accounting for the temporary lymphocytosis. In none of the other 39 cases was any cell increase (over 5 cmm.) observed.

Why in Plaut's 12 cases of latent tertiary lues there should be a cell increase in 25 per cent, while in the present series of 39 there are none, can only be conjectured. It is possible that none of our 39 cases is to develop paresis, but the real explanation probably lies in the difference in material. Although not so stated, Plaut probably derived his material from the clinics of general hospitals where the cases were of comparatively short standing, whereas in the present investigation the majority were of very long standing, many over 15 and some over 20 years, so that they are least likely to show cytological evidence of active pial invasion.

Whatever the case, we hope by future observation of these individuals, and in particular by the autopsy findings with reference to the presence or absence of syphilitic processes and to the

[38] Plaut: Die Lues-Paralyse-Frage. Allgemeine Zeit. f. Psych. und Psych.-Gericht. Med., 1909.

[39] Nonne: Syphilis und Nervensystem, 1909.

detection of spirochætæ, to be able to add something to our knowledge of the behavior and outcome of latent lues, and especially of the significance of the Wassermann reaction in such cases.

Summary and Conclusions.

The entire population of the New Jersey State Hospital at Trenton, which is representative of the rural type of general population of the state, has been examined by means of the Wassermann syphilis reaction, in all 1583 individuals being tested. Seventy of these were cases of general paralysis, and 1513 were cases otherwise diagnosed. Of the cases not general paralysis, 1472 reacted negatively and 41 positively, on which basis there is a percentage of 2.7 latent lues in the total hospital population not paresis.

Including cases of general paralysis about 7 per cent of all individuals of both sexes examined, and about 10 per cent of all males were found to be infected with syphilis as judged by the serum reaction, and this we take as accurately representative of the entire general adult population of the state of New Jersey.

These relatively low figures, as compared with those of some previous more limited investigations in institutions, we believe are solely and wholly accounted for by the type of general population and rural environment from which the individuals examined originally came, and further, that in the differences of environment is found the explanation for the discrepancies in results among these previous investigations themselves.

We believe, furthermore, that this investigation, both in itself and by comparison with some of the more reliable previous figures, shows that the relative prevalence of syphilis in city and country is in general exactly two to one.

A careful analysis of all data including age, sex, color, nationality, civil condition, and type of psychosis leads to the conclusion that syphilis among the insane is no more frequent in occurrence and differs in no particular of its distribution from that found in any general community; that in fact investigations as to the frequency of syphilis among the insane offers the most convenient method of accurately determining the figures for any corresponding general population, and it is for this reason we state that the

prevalence of lues in the entire general adult population of the state of New Jersey is 7 per cent.

Only one type of psychosis other than general paralysis (and cerebral lues) has been found to be definitely related to syphilis as cause and effect in either direction. This is the type of focal brain disorder which occurs in middle life as a result of vascular sclerosis. This group was found to constitute 16 per cent of all the arteriosclerotic mental disorders among men and which, although not symptomatically differentiated from other similar focal brain diseases, has an entirely different etiology which may be demonstrated by biological methods.

In the field of general paralysis comparison of biological, cytological and chemical findings has pointed out that not only are the processes thus indicated usually found in association, but that there is at least as much evidence for the belief that all are dependent upon one and the same mechanism, viz., the inflammatory process of the central organ, as for the conclusion reached by others that each is separate and independent.

Our experience in this series with the original Wassermann technique and that of the Noguchi modification has shown that with alcoholic extract antigen the Wassermann gave a 5 per cent error of deficiency, and the Noguchi a 2 per cent error of excess, and that reliable results are only to be obtained by the use of both methods in combination.

Finally, it is believed that with a perfectly controlled material of some 35 cases of purely latent lues with positive serum reaction, it will be possible by future observation and by final autopsy findings to assist in acquiring a more definite knowledge of the significance of the Wassermann reaction in such cases than has as yet been attained.

ELEMENTARY CONSIDERATIONS OF APHASIA.*

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A consideration of the subject of aphasia as here presented is in a sense open to criticism from two standpoints. To the special workers in the field it can offer nothing new; while to those who are not brought into frequent contact with such cases the entire question is prone to be regarded as one of principally theoretical interest, of but little practical import and of limited application.

In view, however, of the fact that it is largely through the investigations of aphasic disorders that much of what is known of cerebral function and localization has been learned, it must be conceded that in this respect alone the subject is of more than limited scientific interest.

On the other hand the protracted discussion of many disputed points has perhaps lead to some confusion as to what in reality are the accepted teachings of the various authorities, and this together with the rather radical revision of the entire subject as recently proposed by Marie would seem to justify a limited review, in which both the classical teachings of the earlier school and the more recent conceptions of Marie are summarized and stated in as concise a manner as the subject permits.

In such a discussion there is obviously no claim to originality and the statements and subject matter presented are taken from the works of the various authors to whom reference is made.

General Considerations of Aphasia.

The conceptions of aphasia as taught by the earlier school of Broca, Bastian, Lichtheim, Dejerine, v. Monakow and Wernicke are based on the assumption (or proof?) of certain definite centers of cerebral activity, each of which, though intimately connected with the others, possesses a distinct and separate function in the realm of speech; and the area of the cerebrum embracing these

*Read by title at the Sixty-Seventh Annual Meeting of the American Medico-Psychological Association, Denver, Colo., June 19, 1911.

centers has been termed the "zone of language"; the anterior portion in general being motor, and the posterior portion, sensory in function.

On the nature and location of at least two of the special centers there is (or was) practically general agreement; the center for the storage of "kinesthetic memory pictures of speech," first demonstrated by Broca, in the foot of the left third frontal convolution; and termed the motor speech center; and the center for "sound memory pictures" located in the first (and second) left temporal convolution, originally largely determined by Wernicke, and termed the sensory speech center. Thus, aphasic disorders in their simplest forms would arise from circumscribed lesions of these two centers and would be displayed purely as disturbances of emissive and receptive speech respectively.

The entire aphasic syndrome is rendered complex, however, by reason of the intimate relation which one center bears to the other, the disputed question of the existence of a third center, and the conceptions of Wernicke concerning the relation between the more abstract intellectual activities and the clinical types and manifestations of aphasic disturbances.

Since the relations and associations which exist between the emissive and reception centers of speech (largely through the island of Reil and Claustrum) are necessarily of the most intimate character, it is obvious that in the main, a disorder though primarily affecting one of the centers, must assuredly lead to a disturbance in function of the other.

Thus, Broca's aphasia, in which motor phenomena predominate, is constantly accompanied by certain readily demonstrable defects of comprehension; and conversely in aphasias of essential sensory type, when the sensory center itself is affected, emissive speech becomes disordered and uncertain.

The importance of the sensory speech center in the latter connection is obvious when it is recollected that in learning to speak, this faculty in the majority of instances at least, is probably acquired by the repetition of word sounds which are first comprehended and stored in the receptive center of language. Furthermore, since as claimed by Wernicke, the process of *thinking* is accomplished by means of *sound* pictures, disorders of the sensory center will likewise result in profound disturbance of the process

of "internal language," that is, of the process of *word conception,* and consequently of the ability to read and to write (verbal alexia and agraphia). But, according to Wernicke the sensory speech center in itself is in reality but the termination of the auditory nerve where merely the *sounds* of spoken words are perceived and understood as such. It can therefore have nothing to do with the understanding of the word *sense,* but serves as a transmitting station for properly perceived word sounds to a higher center where the concrete meaning of such sounds are appreciated; "a center for the understanding of the word sense." A typical example of the operation of the lower sensory speech center without the necessary interaction of a center of word conception is offered when the word sounds of a foreign language are heard before a knowledge of their meaning is acquired. Similarly, for the correct appreciation of the nature of *objects* analogous center of "concrete conception" (Ziehen) is indicated, and in this must converge the various sensory projection fields by which are conveyed the knowledge of the physical properties of objects. For example, the conception of a rose would be the sum of an optical memory picture, an olfactory memory picture and a tactile memory picture all so intimately connected in the various cortical fields, together with the accompanying *word* conception, that the stimulation of even one sense by the object is sufficient to call to mind all of the other properties of the object together with its proper word symbol. Thus the definition of a concrete conception is "the definite grouping of associated memory pictures with each other," and the relation of the centers of conception to *mind blindness* (so-called apraxia) becomes of obvious significance.

It is on the (probable ?) existence of such higher centers of "word conception" and of "concrete conception" that Wernicke conceived the types of aphasic disorders which he designated as "transcortical"; * and which arise from an interruption of the association tracts which connect the motor and sensory speech centers with the so-called region of conception.

The location of such a region is obviously theoretic and comprises the realm of "intellect," hence the general cerebral cortex;

* Transcortical being used in the sense that the lesion is situated beyond the nearest center the activities of which are interfered with in the given syndrome.

and in the transcortical forms of aphasia there are disturbances of "internal language" similar in general to those observed in disorders of the motor and sensory speech centers themselves. The entity of the transcortical aphasias is questioned but frequent cases are observed in which such relations are said to offer the only satisfactory explanation of the clinical facts.

In contrast to the cortical and transcortical aphasias, in which either of the motor and sensory speech centers themselves are involved, or in which their associations with the centers of "intellect," are disturbed, and in which both emissive and receptive speech and internal language are disordered, are the forms of aphasic disorder which result when the projection fields below the centers are alone divided.

Since in such case both the cortical centers and their higher conception connections are intact, intellect will be unimpaired, the power to read and to write will suffer no reduction and the entire process of internal speech will remain intact. The manifestations in such cases are thus confined solely to a central mechanical inability of speech sounds to leave or to reach the centers in which they are normally elaborated or perceived.

From the location of the lesion in such forms of aphasia, they are designated "subcortical" aphasias and represent the purest forms of speech disorders; pure word mutism and pure word deafness. They are of uncommon occurrence and Marie contends that pure word deafness does not exist though Wernicke and Liepmann claim undoubted cases of this nature.

It is, however, in the disturbances of written language that the greatest disagreement among authorities exists as to the centers and mechanism concerned. It is claimed by Dejerine, and supported by Bastian and Pick, that the functions of written language are dependent upon the integrity of a definite center for the storage of "optical memory pictures of words"; that this center is unilateral; located in the cortex of the left angular gyrus, and connected by association paths with the primary optic sensory field in the calcarine area of the mesial and basial surface of the occipital lobes. This conception obviously narrowly constricts the function of written language and tends to regard reading and writing as faculties, in a sense, more or less independent of spoken language and speech conceptions. Dejerine says: "a

unilateral focus deeply seated in the medullary structure of the parietal lobe produces a combination of right-sided hemianopsia and isolated writing blindness or alexia; if the affection is disseminated and reaches the cortex of this portion of the brain, agraphia is added to alexia." The accuracy of this statement in general is not disputed by Wernicke and v. Monakow. The latter observers, however, deny the existence of any so-called "optical word center" and claim that the cortex of the gyrus angularis is not at all concerned in alexia and agraphia, but that these disorders result from division of certain association tracts lying beneath the cortex in this region.

Wernicke persistently claims that reading and writing are not independent functions, but are "transcortical subordinated activities from the centers of *spoken language,*" and that hence, "the faculty of writing depends upon spoken language, and is lost as soon as the word conception or internal speech is damaged (cortical and transcortical aphasia) and is retained as long as word conception and internal speech remain uninjured" (subcortical aphasia).

In the general anatomical considerations of aphasia the unilateral position of the two principal centers of speech in the left hemisphere, as well as the exception and modifications to which this is subject, is of primary importance in the sense of cerebral localization.

Although the fact that the left hemisphere is the seat of the "zone of language" is well supported by anatomical observation, and that their unilateral location is perhaps adequately explained by the principle of the conservation of energy there is no good explanation of the fact that the conspicuous right handedness of most persons causes the development of the speech centers in the left half of the brain, and that in left handed individuals a similar function is assumed by the right hemisphere.

That the localization of the speech centers is largely the functional acquirement of each individual is clearly demonstrated by certain observations of aphasic disorders in left handed individuals and of cerebral lesions in childhood. Thus, Oppenheim reported a case in which marked sensory aphasia due to a tumor appeared simultaneously with a left hemiplegia; the history revealed the fact that the patient was not left handed from

birth, but that owing to an injury to the right hand at the seventeenth year she had subsequently become left handed. Autopsy revealed the suspected tumor in the left hemisphere. This observation leads to two conclusions; first, that the right hemisphere may assume the function of speech even in adult life in place of the left hemisphere whose function it is normally; second, that the left hemisphere may completely lose the previously acquired function of speech if the person becomes left handed.

In childhood, disturbances of speech caused by disease of the left hemisphere are rapidly compensated for, even when subsequent findings reveal the complete destruction of the left sided speech centers. Apparently in childhood the preexisting left sided cerebral function is readily transferred to the right hemisphere; under these circumstances left handedness is not necessarily produced and in the later years fresh lesions in the right sided centers may cause aphasia in persons who are not really left handed.

In the fully developed brain how far such a substitution for destroyed speech centers occurs is much discussed. It is generally conceded that slowly growing pathologic foci, which act largely by displacement (brain tumor and abscess), may produce no symptoms, not even of speech disturbance, although autopsy findings may show the center or tracts under consideration to be severely affected. Oppenheim's case is contrary to this, since substitution from the right hemisphere to the previously active left sided centers did not occur.

The reason advanced by Wernicke as to why, in this and similar instances, substitution of centers does not take place is, that while in some cases the lesion in one hemisphere may have no affect upon the integrity of function of the opposite side, in others the presence of a one-sided lesion may so damage the general cerebral functions that the opposite hemisphere is prevented from assuming the function of the other.

The principal points in the general considerations of aphasia, up to the time of the revision of the subject by Marie, then are:

(1) Two well recognized forms of aphasic disorders; simple motor and sensory aphasia, arising from destruction of the two definitely established cortical centers of motor and sensory speech upon which all observers are agreed; both forms displaying a mixed picture and disturbances of internal language. (2) The

less definite and rarer forms of pure emissive and receptive speech disorders due to affection of the projection fields below the centers

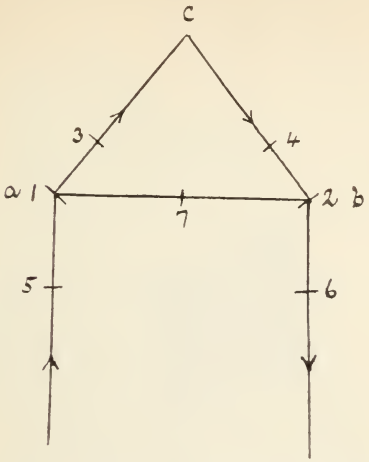

FIG. 1 (modified by Wernicke after Lichtheim).—The diagrammatic representation of the relations between the various centers concerned in spoken language together with their association tracts and the theoretical location of the lesions in the different clinical types of aphasia. *a*, sensory speech center; *b*, motor speech center; *c*, higher center of concrete conceptions and conception of words. 1 and 2, sensory and motor cortical aphasia; 3 and 4, transcortical sensory and motor aphasia; 5 and 6, subcortical sensory and motor aphasia; 7, conduction aphasia.

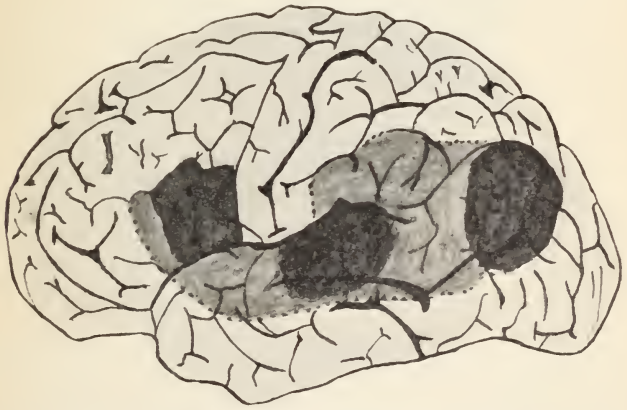

FIG. 2 (after Dejerine).—The "zone of language" as outlined by Dejerine. The lighter-shaded area includes the entire zone, while the darker areas represent the location of the three definite centers believed to exist by Dejerine, Bastian and others. With the exception of the posterior center in the gyrus angularis this was also accepted by v. Monakow and Wernicke. *a*, motor speech area; *b*, sensory word center; *c*, optical word center.

and in which internal language remains intact. (3) The partially demonstrated but disputed transcortical aphasias of Wernicke

in which the speech centers are severed from the realm of conception and intellect. (4) The disputed question of whether disturbances of written language are dependent upon a disorder

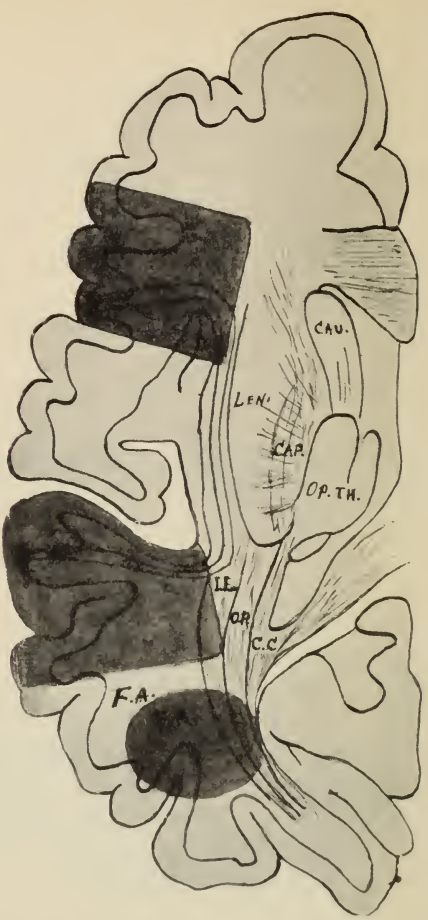

FIG. 3 (after v. Monakow).—The location and relative extent of the lesions of motor and sensory aphasia and alexia and agraphia in a horizontal section of the left hemisphere. In the posterior half of the section is shown the three deep fiber tracts of the inferior parietal lobe cut by the lesion of alexia (*I. L.*, inferior longitudinal bundle; *O. R.*, optic radiation; *C. C.*, fibers of the corpus callosum). Between the inferior longitudinal bundle, and the cortex, in the true medullary substance of the gyrus angularis pass the fibers of the fasciculus arcuatus (*F. A.*) said by Wernicke to be the association tract concerned in agraphia.

of a definite unilateral cortical center for optical memory pictures or are the secondary result of disturbances of the centers of association tracts concerned in spoken language.

The various types of aphasia with their clinical syndromes and anatomical bases as demonstrated and taught up to the time of Marie's revision are included in the following. As before indicated, a number of the types, notably the transcortical and conduction aphasias and sensory subcortical aphasia, were subject to some dispute, and on the subject of certain principles involved in the disturbances of written language there was practically no agreement among the earlier observers themselves, even before Marie assailed the entire structure of the previous conception of aphasia in 1906.

In Figs. 1, 2 and 3 show the extent of the zone of language and the location of special centers; the diagrammatic conception of the speech mechanism as affected in aphasia; and the anatomical locations of the lesion in the more common forms of aphasic disorders as taught and accepted by the majority of the earlier observers.

The Clinical Syndromes and Anatomical Bases of the Classical Aphasias.

simple motor aphasia. (broca's aphasia; cortical motor aphasia.)

Clinical Syndrome.

The power of articulate speech is lost, both spontaneously and by repetition: The patient is practically mute so far as true articulate expression is concerned. If any power of speech is retained it consists in a senseless repetition of the same few inarticulate sounds or syllables. Sometimes profane or emotional expressions are uttered. These expressions, as well as the few retained syllables, are not voluntary or intended to express what is meant (as in transcortical motor aphasia), but are the invariable reactions to all demands on the speech centers. "The mechanism of speech is forgotten." Words and phrases which are sometimes uttered in sleep are impossible of repetition when awake.

The power of understanding spoken language is in the main retained: Simple orders are carried out, objects are produced on demand and questions understood. Although the receptive apparatus is *relatively* clear, the finer differentiations of speech are always disturbed by reason of the intimate association of the

two centers; the reactions to complicated constructions, involved commands and abstract conceptions are invariably impaired.

The power to write voluntarily and to dictation is lost: The motor aphasic is able to form a limited number of individual letters, but the writing of words is impossible. The ability to *copy* is preserved; the motor aphasic copies print in *script.* (A *verbal* agraphia as distinguished from a *literal* agraphia).

"Internal speech" is impaired: The patient cannot read silently with understanding. "The motor aphasic cannot talk to himself and cannot read to himself." The Proust-Lichtheim test* is negative (contrast with subcortical motor aphasia and subcortical sensory aphasia).

Anatomical Basis.

According to the teaching of Broca, v. Monakow, Wernicke and others of the earlier school the lesion constitutes a destruction of Broca's area; the foot of the left third frontal convolution.

SUBCORTICAL MOTOR APHASIA (PURE WORD MUTISM).

Clinical Syndrome.

The simple abolishment of the power of translating the intact (internal) word conceptions into word sounds due to the division of the motor projection fibers leading from the motor speech center: Although totally unable to pronounce a single word aloud the internal mechanism of speech is unimpaired. The internal word conceptions are intact as shown by the rhythm of the articulate sounds produced in efforts to speak; therefore the patient responds positively to the Proust-Lichtheim test; "he can talk to himself, but not aloud."

The comprehension of spoken words is absolutely intact: The ability to write spontaneously, to dictation and to copy is entirely unimpaired. Silent reading is readily performed with perfect comprehension.

The patient with subcortical motor aphasia hears and thinks in word symbols; he is intellectually unimpaired; "silent language" is perfect; he is incapable only of articulate speech.

* The power to correctly indicate by signs or gestures the correct number of syllables in a given word even though the power of articulation be lost.

Anatomical Basis.

As taught by the earlier school the lesion is one involving the medullary fibers of Broca's convolution, either in the convolution itself (beneath the cortex), or one involving the fibers as they join the centrum ovale. v. Monakow states that a partial destruction of the lower part of the convolution (toward the Sylvian fissure) also produces pure word mutism.

Marie claims that the lesion is one involving the lenticular zone alone; he does not recognize pure word mutism as aphasia, but designates it as "aphemia" (anarthria).

TRANSCORTICAL MOTOR APHASIA (AMNESIC APHASIA: AMNESIA VERBALIS).

Clinical Syndrome.

The arrest or very decided impairment of spontaneous speech due to the division of the association tract between the centers of "concrete conception" and the motor speech center: While at best the patient can utter but occasional single words or short phrases spontaneously, repetition is fluent and perfect and is only limited by the power of retention. The command of language is unlimited and automatic. Long sentences, verses or speeches learned by rote (Lord's Prayer, multiplication tables, etc.), are smoothly recited, either at command or by repetition; articulation is absolutely faultless. But the simplest communication, conversation or difference of opinion is impossible through spontaneous speech; once in a while a single short answer is obtained; questions are usually answered by gestures. The speech still at command is not made up on a few senselessly repeated words or syllables as in cortical motor aphasia, but there are occasional expressions of discontent, helplessness and anger, therefore true emotional reactions.

Spoken language is well understood: Words are correctly heard and comprehended and directions and commands correctly carried out. There is an impairment of the power to write spontaneously corresponding to the spontaneous speech defect present. Writing to dictation is correct or accompanied by paraphasic distortions. The patient reads silently with full understanding; reading aloud is performed with paraphasic admixtures.

In the lighter forms of transcortical motor aphasia the disturbance consists in a difficulty in "word finding"; there is an inability to name objects seen (mind blindness); a difficulty in finding substantives in conversation. When the desired word, or part of the word, is suggested it is recognized and eagerly accepted. This type of aphasia (partial transcortical motor aphasia) is designated *amnesic aphasia* (Pitres), or *amnesia verbalis,* and is best exemplified in mental disorders such as senile psychoses with cerebral atrophy.

It is illustrated in normal individuals by occasional inabilities to recall certain words of limited importance on certain occasions; and similarly in persons with a limited command of a foreign language who are unable to express themselves in words which they understand correctly when heard. When such phenomena are observed in connection with a native language *amnesic aphasia* is demonstrated.

The condition is usually not associated with focal lesions, but is commonly encountered in *mental disease* and the transcortical motor and sensory aphasias in senile dementia may often be recognized without traces of focal disorders (mental blindness demonstrates the condition).

Focal symptoms, if found in amnesic aphasia will be of secondary importance to the actual difficulty in word finding and will merely indicate the area which is the seat of general decrease of cerebral activity; as for instance, in cases of post apoplectic dementia.

Transcortical aphasia (both motor and sensory), since they involve the higher centers of conception and intellect are the syndromes constituting the border line pictures between true focal organic disturbances of cerebral activity (on which are based the studies of cerebral localization), on the one hand and the essentially functional psychic disorders of pure mental diseases on the other. It has even been suggested that the peculiar mutisms observed in mental disease (*e. g.,* catatonia) are due to some obscure form of transcortical aphasic disorders.

Anatomical Basis.

The lesion of transcortical motor (as well as sensory) aphasia is said to be one which cuts the fasciculus arcuatus (in the neighborhood of the left first temporal at the Sylvian fossa) thus sever-

ing the connection between the temporal sensory speech center and Broca's motor speech area.

In amnesic aphasia (amnesia verbalis) the alterations consist in an atrophic reduction of the general cerebral cortex (the higher conception center) as is seen in senile and alcoholic brain atrophies.

It should be understood that a part at least of the conceptions of the transcortical aphasias as introduced by Wernicke are based largely on theoretical considerations, and although clinically such cases are not infrequently encountered, the anatomical basis is admitted to be incomplete.

THE SO-CALLED CONDUCTION APHASIA.

This form of aphasia, introduced by Wernicke, is based largely on theoretical grounds and certain clinical observations. Much of the clinical data is contradictory and the anatomical basis has not been satisfactorily demonstrated. The entity of conduction aphasia is disputed. Theoretically conduction aphasia is the interruption of the *directly* communicating tract over which in childhood the word sounds perceived by the sensory center are conveyed to the emissive part of the speech apparatus.

In conduction aphasia neither of the centers themselves or the higher fields of conception are disturbed.

The clinical syndrome of such an interruption should include:

An undisturbed faculty of spontaneous speech.

A full comprehension of spoken words.

An inability to repeat meaningless words or phrases, such as a foreign language.

A paraphasia with ability to correct errors in repeating words and phrases whose meaning is understood.

SIMPLE SENSORY APHASIA (CORTICAL SENSORY APHASIA; WERNICKE'S APHASIA).

A defect in the comprehension of the sense of spoken words arising from a defective comprehension of word sounds due to the division of the medullary "acoustic word fibers" entering the sensory speech center. "An arrest of the understanding of speech with retention of the power of hearing": In cortical sensory aphasia the *word sounds* themselves are not appreciated and hence meaning can not enter into consciousness, while in

the *transcortical* type the *sounds* are plainly perceived but their significance is not grasped.

The power of articulate speech is retained: Sensory aphasia is the aphasia of comparative speechfulness as compared with the speechlessness of the motor type; "the sensory aphasic is a babbler." Until by progressive destruction of the sensory center the vocabulary is diminished the sensory aphasic often displays loquacity; his conversation, however, showing many mistakes in forms of expression; incorrect and distorted (paraphasic) words and sentences are unnoticed and the confusion of words in spontaneous speech may so increase as to render the meaning entirely incomprehensible (jargon aphasia). Sometimes under excitement, however, whole sentences may be correctly spoken.

The patient cannot imitate word sounds and hence cannot repeat what is said to him (contrast with the transcortical type in which repetition is fluent).

The function of "internal language" is much disturbed: The power to think in word symbols, to read understandingly and to write are all profoundly reduced.

There is a marked disturbance in spontaneous writing ability (verbal agraphia) corresponding with the reduction of auditory word perception. Spontaneous writing may be preserved to a certain extent, but the output is senseless and disordered. Both writing and reading are most profoundly affected when there is an accompanying word blindness.

The patient cannot write to dictation, but is able to copy: Copying is done *servilely,* the patient copying print in print, in reality *drawing* the letters; he does not copy print in script as does the motor aphasic.

A difficulty in finding names for objects shown: Objects may be incorrectly named, and distorted words are frequently used in naming them.

In Broca's aphasia the trouble is predominantly of articulation and writing (emission); in sensory aphasia all the elements of language are disordered.

Anatomical Basis.

A lesion involving the sensory cortical speech center in the posterior portion of the left first (and second) temporal convolutions.

SUBCORTICAL SENSORY APHASIA (PURE WORD DEAFNESS).

Clinical Syndrome.

A loss of the understanding of word sounds of what has been spoken, an adequate power of hearing being retained. The entire internal speech apparatus is undisturbed. The word conception remains clear; the patient can think in word symbols and can "talk to himself."

Spontaneous speech is absolutely free and clear, but the patient cannot repeat what is spoken to him. There are no paraphasic admixtures as in cortical sensory aphasia.

Ability to write spontaneously and to copy is unimpaired; writing to dictation is impossible.

There is no impairment of ability to read silently and aloud with full understanding.

Since in subcortical sensory aphasia only those fibers which convey *word sounds* to the auditory center are affected, various other sounds, including the *voice* sounds, are heard; the comprehension of *word* sounds alone is lacking. The lack of understanding is due to actual inability to hear word sounds.

The appearance of such patients is significant. They are quiet and observant; their glance shows suspicion or fear and their demeanor is one of restlessness. Their altered manner, the inability to repeat what is said to them and the marked diminution of spontaneous speech often cause them to be looked upon as dements.

Anatomical Basis.

A lesion of the medullar structures at the foot of the left first temporal convolution at a point where the fibers unite with the island of Reil.

TRANSCORTICAL SENSORY APHASIA.

Clinical Syndrome.

An abolition of the understanding of the word sense with a retained understanding of the word sound and an intact power of hearing, due to the division of the association tracts between the centers of "concrete conception" and the sensory speech center: The speech *sounds* as such are perfectly understood as shown by the *ability to repeat spoken words;* but the accompanying *concep-*

tion of their meaning is not awakened by reason of the division of the connecting pathways between the (lower) *speech* center and the (higher) realm of *conception.* The patient hears the word sounds as such, but they convey no meaning to him as is exactly the case in an unfamiliar foreign language.

The power of speech is actually retained; but is slightly impaired, in that the accuracy of the words spoken by the patient cannot be tested by his own disturbed power of understanding spoken words; hence, speech is frequently to some degree *paraphasic,* and there may be suggestions of admixtures of incorrect and distorted words of syllables.

The patient is able to read aloud: Reading aloud may be fluent, perfect and without effort, but there is no comprehension of what is read since the corresponding conceptions are not awakened. Reading may be compared to the reading of a foreign language which is not understood, but is composed of *similar word sounds.* In some cases reading is paraphasic.

Spontaneous writing shows to an increased extent the paraphasic disturbances of active speech and paraphasic distortions frequently render it incomprehensible.

Anatomical Basis.

As in transcortical motor aphasia the lesion is one severing the connections between the lower (sensory) speech center and the higher centers of conception and probably divides the fasciculus arcuatus at a point near the sensory speech center where the projection fibers are concentrated in a converging bundle.

DISTURBANCE OF WRITTEN LANGUAGE (ALEXIA AND AGRAPHIA).

In the domain of written language, as before indicated, the subject of alexia and agraphia formed for the earlier observers a point of entire disagreement; one school maintaining the action of a special optical word center and the other denying its existence.

In the main, however, both alexia and agraphia are of two well differentiated types, essentially different in both clinical phenomena and anatomical basis; these types are *verbal* and *literal.*

Verbal alexia and agraphia are the terms designating those forms of disturbances of written language which accompany all

forms of both motor and sensory aphasia in which the function of "internal language" is disordered. They are thus a part of the syndromes of the motor and sensory, cortical and transcortical aphasias, while in the subcortical types (pure word mutism and pure word deafness) they are absent.

As distinguished from the literal forms, verbal alexia and agraphia arise from disordered *word conception,* the conceptions of letters remaining more or less intact. Thus patients with verbal alexia or agraphia are able to recognize and to form individual letters and figures, but the meaning of their combinations into word symbols is faulty and distorted. Since the verbal forms of alexia and agraphia constitute a part of the aphasic complexes which arise from disorders of emissive and receptive speech, the lesions from which they result will be identical with those of motor (Broca's) and sensory (Wernicke's) aphasia.

Literal alexia and agraphia on the other hand are considered as separate entities and arise from a loss of conception for *letters,* and since, as claimed by Wernicke, reading and writing are accomplished by spelling, there is likewise a resulting inability in the emission and reception of written words.

In their *pure* forms literal alexia (word blindness) and agraphia are the rarest types of aphasic disturbances. But two authentic cases (those of Rieger and Sommer) of the coincidence of pure literal agraphia and alexia without essential disturbance of speech are on record. In these the patients had lost all conception of certain small and capital letters of the alphabet while the others were well retained and there was no essential speech disorder demonstrable.

Wernicke cites the case of a patient, who, after writing a well connected, and in the main, correct letter to a relative was unable to read a single word he had written (word blindness). Such phenomena are designated by some as "subcortical visual aphasia" and are usually accompanied by right sided hemianopsia; there is word blindness, well preserved spontaneous speech and good comprehension; the patient is able to write voluntarily and to dictation, but cannot read what is written either by himself or others.

The occurrence of *pure isolated literal agraphia* is disputed and in the main is not accepted as a distinct entity. Marie denies it

absolutely; Wernicke himself, who observed a case offering probably the nearest approach to the condition, denied the existence of a pure literal agraphia after prolonged observation of his patient which showed that what at first appeared to be a purely isolated writing defect was in reality accompanied by disturbance of internal speech. The same observer states the rule that if in agraphia there is shown any ability to form letters, no matter how bad the writing, the agraphia is *verbal* and not literal; hence not an isolated affection, but one belonging to a speech disorder.

Theoretically pure agraphia should consist in a more or less total loss of the conception of writing movements with inability to form written letters, figures and words, while, the power of emissive speech and the comprehension of spoken and written language remains practically intact.

Such a combination of preserved speech faculties with an isolated defect of written language is held by some to indicate the existence of special centers for reading and writing. Thus, Exner and Charcot believed that a special motor center for writing movements is localized in the base of the left second frontal convolution, and Dejerine, Bastian and Pick claim that the cortex of the gyrus angularis of the left lower parietal area is a center for the storing of "optical word pictures." Exner's contention of a special writing center is held to be untenable and is disproved by the fact that writing movements can be performed by the leg and even the tongue, when obviously the center of writing movements are localized in entirely different areas. The existence of a unilateral center for optical word memory pictures is denied by Wernicke and v. Monakow.

Wernicke positively states that if a center for the storage of optical memory pictures exists it is one merely for *letters* and not for words because the idea that all words seen can be visually stored is impossible and absurd; and furthermore that it is bilateral. He denies that the cortex of the gyrus angularis possesses any such function and doubts that the proper conceptions of even letters can be awakened unless their corresponding sound conceptions are intact; that is, that the appreciation of the individual symbols of the components of the alphabet, and hence the faculty of written language, is in reality dependent upon the function of the sensory speech center in the first temporal convolution.

Hence agraphia (according to Wernicke) is always due to a disturbance of *internal speech;* the result of disordered word conception, and hence transcortical in nature.

Without attempting to definitely decide the nature and localities of the possible centers concerned in disturbances of written language, Wernicke applies to these forms of emissive and receptive speech the same principles embodied in the consideration of the various forms of motor and sensory aphasia. Thus the theoretical centers concerned in alexia and agraphia are: the lower centers for the optical memory pictures of letters and conception of writing movements respectively, and a higher center of word conceptions. Alexia and agraphia result when the tract connecting the center for optic alphabet memories and the center of word conceptions is divided; pure literal agraphia would arise from a similar division of the tract directly connecting the center of optic memory pictures with the center of conception for writing movements.

On these theoretic considerations as well as on the part played by the cortex of the angular gyrus there is no agreement. The location of the lesion causing essential alexia and agraphia however, is definitely established as one lying in the lower region of the left parietal lobe; that is, the angular and supramarginal convolutions. The operation of the lesion as maintained by Dejerine is through the destruction of the cortical center for the optical memory pictures in the gyrus angularis or the fibers leading therefrom. Wernicke denies this and as proof of this contention calls attention to the fact that if such a center actually existed the occurrence of pure isolated alexia and agraphia would be frequent, whereas in reality they are extremely rare.

Wernicke describes the essential lesion of alexia and agraphia as a focus situated deeply in the medullary layers of the gyrus angularis, but which has no immediate or essential connection with the cortex of this area.

In alexia the lesion involves the deep sagital medullary layers of the gyrus angularis and destroys the inferior longitudinal bundle, optic radiation of Gratiolet and tapetum tracts, thus severing the connection between the speech centers and both occipital lobes with resulting word blindness and right hemianopsia.

In agraphia the lesion occupies the same general situation, but lies nearer the cortex in the true medullary substance (in front, above and external to the lesion of alexia) and divides the fasciculus arcuatus and fibers of the corpus callosum, in which lie the association tracts between the motor cerebral regions and the occipital lobes and lower parietal lobes of the same side, and between the sensory speech center and the lower parietal and occipital lobes of the same and opposite sides.

Marie claims that pure word blindness does not exist and that pure alexia does not occur clinically and anyhow it cannot be considered as a syndrome of aphasia at all, but an extrinsic complex resulting from damage to the posterior cerebral artery. These questions are not decided.

MARIE'S REVISION OF THE SUBJECT OF APHASIA.

The papers of Pierre Marie which in 1906 assailed the entire conception of aphasia as previously taught, have as their most radical departures: The absolute denial of the existence of a motor speech center in Broca's convolution; the splitting up of the previously designated "motor aphasia" into two distinct components; the demonstration of the importance and nature of the "lenticular zone"; the elimination of the angular gyrus as a center for optical memory pictures and of pure word deafness and pure alexia from the aphasic complex; the contention that the so called sensory speech center is in reality not a *sensory* center for the auditory image of words, but an intellectual center; and that lesions of the "zone of Wernicke" alone give rise to true aphasic disorders.

Figs. 4 and 5 show the areas of the cerebrum concerned in aphasic disorders as advanced by Marie: the lenticular zone and zone of Wernicke.

Marie maintains that the third frontal convolution of the left side does not play any special role in the function of language; that which is called motor aphasia or Broca's aphasia in reality is anarthria plus aphasia; that the aphasia of Broca is not a disease, not a clinical entity, but a syndrome, a superimposition of aphasia upon anarthria, or better, a simple combination of two distinct troubles, anarthria and aphasia. As to aphasia itself, Marie holds that there is only one aphasia, which he proposes to call Wernicke's

aphasia, and only one speech center diffusely localized in the left temperoparietal lobe, and that this center is a region of intelligence specialized for language, not a center of sensory images. The clinical splitting up of the aphasia of Broca into two elements, anarthra and aphasia, Marie maintains is verified by autopsy. One finds constantly lesion of the lenticular zone associated with lesion of the zone of Wernicke.

Marie describes the lenticular zone as an area included between two parallel lines drawn inward from the anterior and posterior fissures respectively of the island of Reil to the lateral ventricle. Its territory includes the caudate nucleus, the lenticular nucleus,

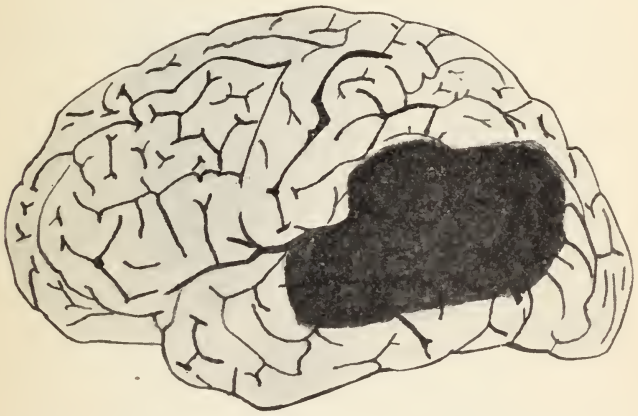

FIG. 4 (after description of Marie).—The shaded area denotes the "intellectual center specialized for language" and called by Marie the zone of Wernicke. It is from lesions in this area alone, according to Marie, that all true aphasic disorders originate. It is to be noted that with the exception of the third left frontal area, which is excluded, Marie's intellectual area is similar to the zone of language of the earlier writers.

the external capsule, the claustrum, the cortex of the island of Reil and the internal capsule. It is in this territory very distinctly separated from the third frontal convolution that the lesion that determines anarthria is especially situated.

Anarthria (the old aphemia of Borca) is characterized clinically by the loss of speech with preservation of the understanding of words, of reading and writing. It is produced by a lesion in the lenticular zone, interfering with the co-ordination of movements required for the formation and articulation of words, without inducing true muscular paralysis. Broca's aphasia is produced by a combination—the proportion varying with the case—of the

lesion of anarthria with a lesion of Wernicke's zone or lesion of the fibers coming from this zone.

The third left frontal convolution plays no part in the function

FIG. 5 (after description of Marie).—Marie's "lenticular zone" (shaded) in which is located the essential lesion of Marie's anarthria, and which when combined with a lesion of the zone of Wernicke gives rise to Broca's aphasia. The upper and lower limits of the lenticular zone have not been defined.

of speech. The true speech center is the zone of Wernicke; which must not be considered as a *sensory* center, but as an intellectual center. Wernicke's zone consists of the supramarginal and angular gyri and the feet of the first two temporal convolutions.

Lesion in this center determines in proportion its extent, and in addition to the disturbances of speech, deficient understanding of words, inability to read and write, as well as the disappearance of certain concepts of a didactic character. The foot of the first temporal convolution cannot be said to constitute a sensory center for the auditory image of words. Pure word deafness does not exist. Pure word blindness (pure alexia) does not occur clinically. The lesion producing it is a lesion of the posterior cerebral artery, not of the Sylvian artery as in the other aphasias. It is useless and inaccurate to drag in the angular gyrus, which cannot be recognized as the center of visual word images.

Marie maintains that there is no reason to preserve the classification of aphasia into the cortical and subcortical forms. As a matter of fact, aphasia due to focal lesions is never exclusively cortical. It is, moreover, advisable at present not to refer to the cerebral cortex the entire pathologic physiology of aphasia, since the subjacent white matter seems to play a part of perhaps greater clinical importance than the gray matter. If one insists upon classifying aphasia, the varieties of which are connected by a scale of innumerable transition forms, the best division would be into (1) *intrinsic* aphasia, in which Wernicke's zone or the fibers coming from it is directly and considerably affected by the lesion. (Broca's aphasia, Wernicke's aphasia), and (2) *extrinsic* aphasia, in which Wernicke's zone with its fibers is not directly involved.

For Marie there is only one aphasia, the aphasia of Wernicke. The term sensory aphasia, he thinks, should disappear. The aphasia of Wernicke has for its substratum lesion of the zone known by his name. Intrinsic aphasia is accompanied by trouble of the internal language and intellectual deficit. The other alteration of language, anarthria and pure alexia, are extrinsic syndromes. Pure alexia is dependent upon lesion of quite another vascular territory than aphasia. Aphasia is dependent upon lesions of the Sylvian artery, and alexia upon obliteration of the posterior cerebral arteries. He maintains that in autopsies upon patients with Broca's aphasia there is a double lesion. One causes the anarthria and the other the trouble of internal language, of reading, and of writing. The terms Broca's aphasia, ataxic aphasia, total aphasia, designate progressive degrees of the same syndrome.

The three important factors in the discussion are: (1) is Broca's area, the foot of the third frontal convolution, the center in which is stored the memories of phonetic speech, articulatory, kinesthetic memories? In other words, is motor aphasia or Broca's aphasia real aphasia at all? Is there a well defined, though not sharply marked syndrome to which the name of Broca's aphasia is given, upon what is it dependent anatomically? (2) Do lesions of the so-called lenticular zone give rise to a simple complex, parallel to that of so-called subcortical motor aphasia, the anarthria or aphemia of Marie? (3) Is the area of language the area in which memories and words seen and heard are stored, and from which alone they can be evoked by peripheral stimuli, or is the storage of such memories a function of the anterior pole of the brain (the so-called psychic sphere) or of the whole brain itself?

It must be granted that there is no adequate clinical or anatomical evidences for considerating Broca's center to be the seat of memories of articulation. There does not exist in the literature a case of Broca's aphasia clinically in which the lesion was confined narrowly to the foot of the third frontal convolution, nor does the brain upon which Broca based his original thesis show it. It is probable that Marie is correct in assuming that the third frontal convolution is not an area in which are stored memories of articulation and it may be stated that our present conceptions of the zone of language must be modified in so far as denying the existence of the storage of articulatory kinesthetic memories in the anterior poles, the foot of the third frontal convolution.

It is admitted by everyone that there is an aphasia, which is clinically distinctly characterized, to which the name motor aphasia or Broca's aphasia is given. Is it sensory or Wernicke's aphasia plus anarthria or aphemia? If it is true that the foot of the third convolution is not the seat of phonetic memories, then Marie's explanation is probably the correct one. Marie admits that Broca's and Wernicke's aphasia are clinically two distinct varieties. The distinction between the two is not always very sharply drawn, but, as a rule, certain characters permit the distinction to be made. Those who have contended that the symptomatology of the two forms of aphasia are very unlike have pointed out that it is a mode of copying that distinguished motor aphasia from sensory

aphasia. It is admitted by everyone that the motor aphasic has some word deafness and some word blindness, but it is maintained that he copies print in script while the sensory aphasic copies servilely. There has been recent important testimony to show that this is not true.

In regard to the mental defect of aphasic patients, whether they have sensory or motor aphasia, which Marie has emphasized, this has received due consideration in every treatise on aphasia, and there can be no doubt of its existence; considering the lesion of the brain in the majority of cases of aphasia, the wonder is that the mental defect is not more pronounced.

That lesion of the so-called lenticular zone gives rise to the symptom complex parallel to that of so-called subcortical motor aphasia there can be little doubt. The preservation of internal speech is the distinct feature in each of them. Whether the loss of speech capacity which results is called anarthria or aphemia is not of prime importance. But the term aphasia should be reserved for those cases in which internal speech is disordered. Those cases in which the internal speech is not disordered and in which there is inability to speak (pure motor aphasia of Dejerine; pure word mutism of Wernicke) should be classified as cases of anarthria or aphemia. This anarthria may exist from the onset of the patient's illness, that is, there may never be any true aphasia associated with it.

The answer to the third question in regard to the storage of memories of words seen and heard cannot be given positively at this time. We do not know how memories are localized, or indeed that they are localized at all. We assume that cells undergo modification from each impression that reaches them, and that the cell reacts in a different way to different impressions at different times.

The position taken by Marie is not so revolutionary as has been commonly supposed. In the first place, it has never been seriously denied that there is not a certain amount of mental defect in practically all cases of true aphasia. This mental defect varies in different cases, but it has been generally recognized. In the second place, the anarthria or aphemia of Marie is in reality the same thing as the subcortical motor aphasia of Wernicke, and the pure motor aphasia of Dejerine. The important contribution that Marie has made is that the foot of the third frontal convolution

(Broca's convolution) is not the seat of articulatory kinesthetic memories, and is not an integral part of the zone of language, and that, therefore, destruction of it does not cause aphasia. It is not enclosed in the lenticular zone of Marie.

The service done by Marie with regard to the subject of aphasia has been great, in spite of the perhaps just critism that he has torn down without building up.

In the last analysis, it still remains to be seen as to which teaching, that of the old or the new schools, will in the main prove the correct one.

HEREDITARY ATAXIA.

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Hereditary diseases of the familial type are interesting especially in the light of the increased activity in the study of heredity. In these cases there seems to be something more definite and tangible in the inherited element and there are not the accessory or accidental causes to be eliminated. In the study of such nervous diseases, it is also important to consider the relationship between the different types, if any, and whether they are simply various manifestations or different stages of the same disease process.

In 1863, Friedreich first described the symptoms which afterwards were called by his name. Thirty years later, Marie grouped together a number of cases reported by several individuals and named the disease "hereditary cerebellar ataxia." Since then, it has always been a question whether Marie's hereditary cerebellar ataxia was a distinct disease entity or whether it was closely related to Friedreich's hereditary ataxia, being perhaps a different stage or type of the latter disease.

The prominent features of the two classes of cases are not markedly different, yet there are clinical distinctions which may or may not justify separate classification.

In Friedreich's disease, there are the family type, onset before the sixteenth year, unsteady ataxic gait, a contraction of the posterior tibial muscles causing a talipes equinus, awkward oscillating movements of head and body, arms less affected than legs. There is nystagmus. Pupillary reflex undisturbed. Speech is slow, difficult and indistinct. Patellar reflex lost or greatly diminished. There are rarely any disturbances of sensation, and bladder and rectum are under control. The patients may grow up to die of some intercurrent disease in adult life. According to Starr, pathologically, without going into detail, there is a defective development of all the fibres of the spinal cord most marked in the posterior half, with some atrophy of cells in both horns.

In the cases described as Marie's hereditary cerebellar ataxia, there is the familial type of heredity, the onset is between twenty and thirty, a gait similar to Friedreich's cases, the same awkward, oscillating movements of head and body, speech slow, indistinct and explosive. The arms as well as the legs are affected. The reflexes are exaggerated and the pupillary reaction is commonly imperfect. There is no deformity of the feet. The supposed distinctive pathologic change was atrophy of the cerebellum.

Since the description of Marie's cases, a number of cases have been reported with various opinions as to classification. Noteworthy among these are Sanger Brown's twenty-one cases. He concluded that these cases should really belong with Friedreich's disease, although the onset was late in life and they showed such unusual symptoms as exaggeration of the patellar reflexes. He thought the disease picture should be modified so as to include such cases.

Ormerod of London and Bernhardt of Berlin, however, in discussing Sanger Brown's paper, both thought that the symptoms being so different from Friedreich's disease they should make up a different type of hereditary ataxia.

On the other hand, supporting the contention that Marie's cases are a type or a different stage of Friedreich's disease are the facts to which Patrick called attention.

He showed that the age of onset in Friedreich's disease varies. Some otherwise typical cases begin later in life. Where there are several members of the family affected, the cases of more recent onset may have typical absent knee jerks, while those which have been going on for some time longer may have the reflexes present or even exaggerated. He also cited some instances where the case began with knee jerks present but later on becoming diminished or absent.

While there may seem clinically to be a real difference in these two classes of cases, yet necropsy often fails to confirm such a distinction. Adolph Meyer examined one of Sanger Brown's cases and found no circumscribed cerebellar lesions. Parts of the spinal cord and medulla having relations with the cerebellum were affected. The characteristics of the lesion were not identical with that of Friedreich's disease, but Meyer explained this by the difference in the age of onset and the extent of the pathological

process. Meyer concluded that the separation as a clinical type of Marie's cases was anatomically justified to a less extent than Marie expected.

Nonne, however, reports the autopsy in which the only anomaly was diminution of the size of those parts of the brain developing from the fore and mid brain and exceptional diminution in size of the cerebellum, the medulla and spinal cord being absolutely normal.

Patrick's case reported in 1902, under the title Hereditary Cerebellar Ataxia, was probably due to hereditary syphilis. In this paper, Patrick had expressed doubts about there being such a disease as hereditary cerebellar ataxia and pointed out the considerable resemblance his case bore to paresis. A case of Klippel and Durante, one of those originally reported by Marie, showed a normal cerebellum but spinal cord changes found in Friedreich's disease.

Bearing in mind the above uncertainties in classification, the following cases are described as presenting the symptoms grouped under Marie's hereditary cerebellar ataxia.

The M. family in which these cases occurred is of German origin, the grandparents of the patient first described not having come to this country.

Case I.—F. M., white, male, age 36, laborer, married.

Family History.—Several members of his family similarly affected. Described later on.

Personal History.—Early life negative except showing a defective basis, not making good progress at school or advancing above a common laborer. He has been temperate. Married about 17 years, getting along all right with his wife except he had an abnormally strong sexual appetite. Has had three children.

Onset.—Gradual about eight years ago or at the age of 28. Ataxia began in the legs and has become worse until he has been unable to keep a position, as his employers would consider him intoxicated. He was unable to pick up things or work well with his hands. Naturally quick tempered, he has become more irritable, especially towards wife when she refused to gratify his excessive sexual desires.

On Admission.—He was well oriented and able to walk but with a staggering awkward gait.

Physical Examination.—A fairly well nourished, large boned, muscular white man, height 5 feet 9¼ inches, weight 160 lbs. Some general bodily deformity, chest egg shaped, sternum bent forward at nipple line. Unnatural deep curve of spine at lumbar region with protrusion of abdo-

men. No signs of syphilis. No subjective complaints. Up and down oscillating of lids when gazing at an object intently. Lateral nystagmus. Pupillary reaction normal. Triceps, biceps and patellar reflexes exaggerated. Ankle clonus present. Motor functions considerably impaired. Mastication segment not good. Unable to move tongue without great effort. Head not moved well on body. Inability to shrug shoulders well. Balancing power not as good as normal. Gait staggering and ataxic. Unable to manage feet well. In standing there are awkward oscillating movements of head and body, protrusion of abdomen and sinking down appearance of chest. Marked speech defect. Pronounces electricity as "electristic." Truly rural as "trury lury." Speech indistinct and explosive. No sensory disturbances. Bladder and rectum under control.

Mentally, he was quiet, orderly and agreeable, inclined to talk and associate with other patients. Answered questions relevantly. At times showed a tendency to be pert or trifling in reply to questions. No paranoid trends, hallucinations or delusions elicited. Well oriented as to time, person and place. Gave a fair account of his past life. Memory for immediate past good. Retention good. School knowledge and calculation probably in keeping with education. Showed some insight, saying he was nervous. Judgment defective in that he thought he was able to work as well as ever.

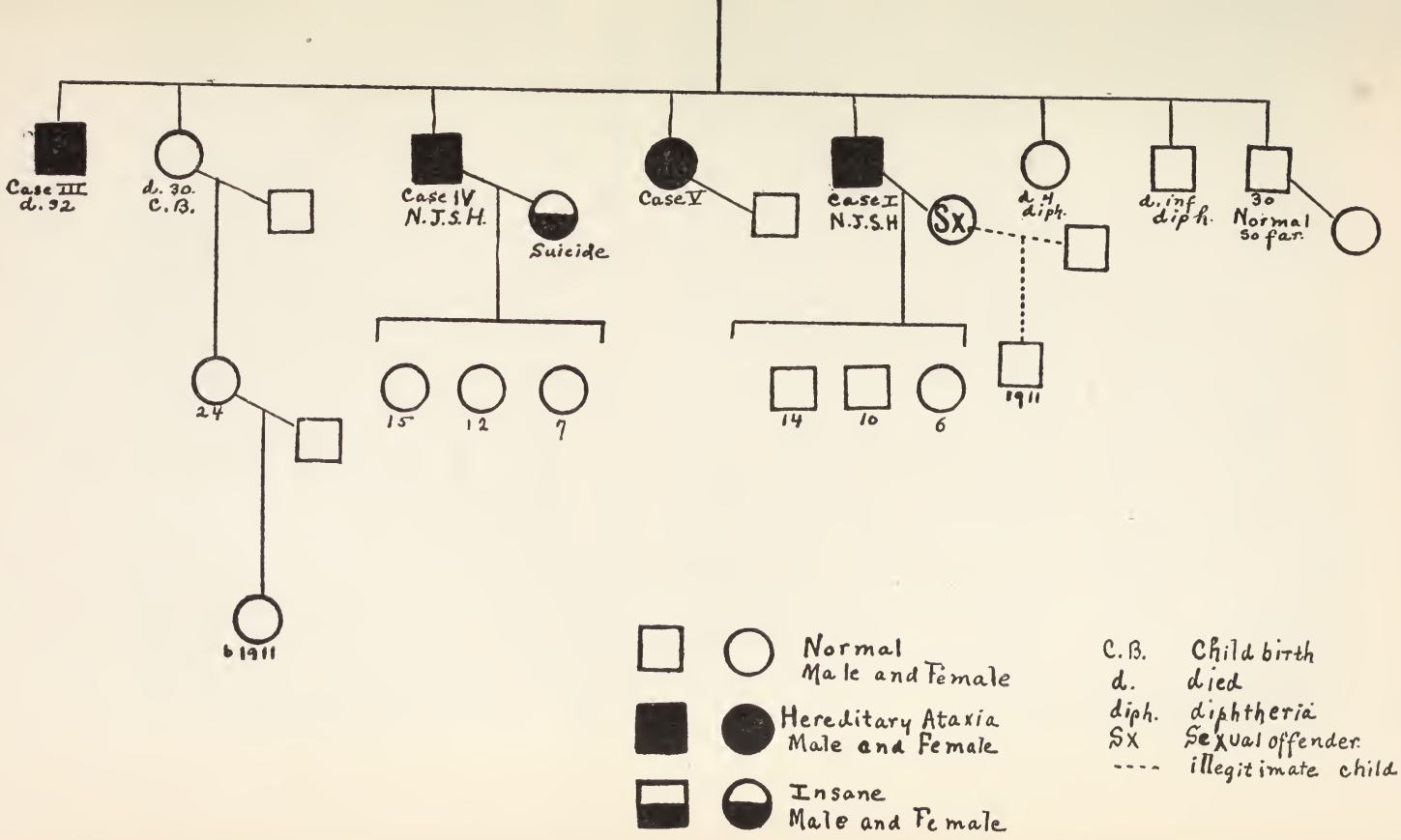
Since admission patient has gradually become more ataxic and now has to be assisted a little in standing up or walking about. Deep reflexes continue to be much exaggerated with ankle clonus on both sides. Pupils are rather contracted but equal. Reaction to light somewhat sluggish. Nystagmus not constant. There are the same awkward movements of hands, head and body. When commanded to do so, he can stop these movements for a few moments, but they begin again when he tries to change his position or do anything else. He has control of his organic reflexes. Shows some apparent deterioration in not recalling length of time in the hospital and in not being able to give month, day and year. Recalls physician's name, although he has not seen him for some time.

Ophthalmoscopic Examination.—Negative.

Wassermann Blood-Serum Reaction.—Negative.

Case II.—Patient's mother is said by reliable informants to have been similarly afflicted. The onset in her case was at about the age of 34. At this time one of her children was nearly burned to death and she seemed "to go to pieces" from the shock. She became "shaky all over." Her face and hands were never still and for twelve years she was never able to walk but sat in a wheel chair. The doctor called it a disease of the spinal cord.

Case III.—H. M., brother of Case I and son of Case II, was alcoholic. Very little information obtained in regard to this case. He died at the age of 32 in a general hospital. He evidently presented peculiar and unusual symptoms of an organic brain condition, as the authorities were anxious to obtain permission for a post-mortem examination, but the relatives would not allow it. The hospital records have been destroyed, but the certificate of death gave the cause as "brain tumor and meningitis."


Case III
d. 92
d. 30.
C. B.
Case IV
N. J. S. H.
Suicide
Case V
Case I
N. J. S. H
Sx
d. H
diph.
d. inf
diph.
30
Normal
so far.
24
b 1911
15
12
7
14
10
6
1911
Normal
Male and Female
Hereditary Ataxia
Male and Female
Insane
Male and Female
C. B. Child birth
d. died
diph. diphtheria
Sx Sexual offender.
---- illegitimate child

Case IV.—Record not very satisfactory, as thorough examination was not made at that time.

J. H. M., brother of Case I and son of Case II, married, age 35, farmer. Admitted to N. J. S. H. December 8, 1905. Symptoms began eight years before admission at the age of 27. When admitted was excited but soon became quiet and showed no delusions or other mental abnormalities. He could walk with difficulty, had awkward movements of head and body, difficult explosive speech. Patellar reflexes were exaggerated. He developed pulmonary tuberculosis and died January 10, 1907. No post-mortem examination allowed.

Case V.—A. W., sister of Case I and daughter of Case II, age 40, widow. Onset of symptoms said to have been about three years ago at the age of 37, although she has been "nervous" for about seven years since her husband's death making the real onset probably at the age of 33. At present has the characteristic speech defect, awkward movements of hands, body and head, difficulty in walking.

As will be seen by the chart, Cases I and IV have several children, none of whom has yet reached the age of onset, the oldest being fifteen. The three children of Case IV have been described as "nervous" but otherwise normal so far as can be judged. These children, however, have had the additional handicap of an insane mother.

The second daughter of Case II died at the age of 30 of childbirth, normal so far as known. Her daughter is still normal at the age of 24.

The youngest child of Case II, a son, is 29 years old and as yet unaffected.

There are, therefore, five members of the M. family, the mother and four children, who have been affected by nervous disease. Three of these cases, I, IV and V, are known to have presented similar symptoms. According to reliable informants the mother was undoubtedly affected in the same way and from the meagre records there is a strong probability that Case III was one of ataxia. The age of onset varied from 27 to 34, with speech defect, awkward movements of hands, body and head, difficulty in walking. In two cases only are the reflexes reported, in both being exaggerated. There has been no loss of organic reflex control and no marked mental affect. The cases would seem to come under the class described by Marie as hereditary cerebellar ataxia.

As have been stated before, however, the age of onset, the reflex findings and other differential points are not constant.

Pathological examination of cases regarded as typical have been disappointing, many times showing little or no cerebellar change. Under the circumstances it would seem best to regard Marie's cases and Friedreich's disease as types of the same condition, showing variations due perhaps to age of onset, duration or stage of the disease.

In conclusion, I wish to express my obligations to Miss Florence I. Orr for her assistance in investigating the M. family.

BIBLIOGRAPHY.

Patrick: Jour. Nervous and Mental Disease, Vol. 29, p. 129, Mar., 1902.
Patrick: Jour. Nervous and Mental Disease, Vol. 33, p. 459, 1906.
Meyer, Adolph: Brain, 1897, p. 276.
Brown, Sanger: Brain, 1892, p. 250.
Starr: Jour. Nervous and Mental Disease, 1898, p. 175.
Spiller: Brain, 1896, p. 588.
M. Nonne: Abstract in Jour. Nervous and Mental Disease, Vol. 33, p. 49, 1906.
Raymond: Abstract in Jour. Nervous and Mental Disease, Vol. 32, 1905.
Church and Peterson: Nervous and Mental Disease, p. 454.
Oppenheim: Text-Book Nervous Disease, Vol. I, p. 197.
Reference Handbook of Medical Science, Vol. VII, p. 347.
Neurolog. Soc. of United Kingdom, Brain, 1906, Vol. 29.
Starr: Text-Book of Nervous Disease.

POLYNEURITIC DELIRIUM—KORSAKOFF'S PSYCHOSIS.*

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The polyneuritic or Korsakoff's psychosis has been recognized for many years. Although much has been written in regard to this interesting condition there are a number of disputed points and differences of opinion especially as to etiology prognosis and even as to the existence of such a disease entity. Moreover, when the frequent resemblance to severe organic states is considered the importance of a careful study of this psychosis must be realized.

It is not the purpose at this time to enter into a detailed discussion of Korsakoff's disease in all its aspects. Following a description of the symptom-complex a few of the more undecided points will be considered, especially emphasizing the results of an examination of a series of cases at the New Jersey State Hospital at Trenton.

In general, the symptoms may briefly be stated as delirium with confusion, disorientation, memory disturbances (Merkfahigkeit), with fabrications and pseudo-reminiscences, amnesic periods, hallucinations and delusions.

The mode of onset is somewhat varied. According to some authorities there is apt to be a prodromal period of irritability, nervousness and either insomnia or drowsiness, the latter in many cases being a profound stupor. Following sooner or later the patient enters into a period of active delirium varying in degree from a mild confusion and disorientation with hallucinations to a severe condition resembling the typhoid state.

Again, the case may begin with severe neuritic symptoms obscuring the mental phenomena which appear later. Still a third method of onset according to Bonhoeffer is a slowly developing increasing memory weakness.

* Read at the sixty-eighth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., May 28-31, 1012.

A most usual mode of onset is that of an acute delirium resembling a delirium tremens in many ways. Because of this, and the continuance of the symptoms, some writers have suggested calling the condition "chronic delirium tremens." The patient becomes confused, disoriented, irritable and apprehensive, fearing he is to be harmed. He has horrible hallucinations of sight and is unable to sleep. He talks constantly to himself. Unlike delirium tremens, as Lambert has pointed out, the critical sleep does not appear. As soon as the acute stage is over the characteristic mental symptoms ensue. The demeanor of the patient may not be far from normal to a casual observer, but a few questions will disclose the peculiar state of mind. There is practically complete disorientation. The patient does not realize his surroundings. Although he may have been in the hospital in bed for some weeks, he thinks he is at home or at his place of business. He has no idea of time, the present date or the period during which he has been sick. On the other hand, he may recognize his friends, but in severe cases even recognition may also be disturbed.

The memory disturbances form the most peculiar and distinguishing feature of the disease, the memory for the immediate past being markedly defective. The patient is unable to retain the most recent occurrences. He cannot recall what has just been said to him (Merkfahigkeit). He does not know what has taken place the moment before, to whom he has been talking or what has been done. He may ask repeatedly the same questions and no amount of emphasis will enable him to remember what he is told.

The memory for earlier events is usually retained up to the onset of the sickness, although the amnesia may be retrograde and involve some time before the beginning of the trouble. In other cases the memory for past events may be quite perfect, although the sequence may become disordered, the patient for instance naming his different occupations or places of residence in the wrong order. Again, there may be only partial memory, unaccountable lacunæ or gaps occurring in his recollection.

Related to the amnesia and probably resulting from it and being perhaps an effort to cover up or fill in the gaps are the pseudo-reminiscences, memory fabrications or falsifications. While the patient may have been confined to his bed for weeks, yet he describes journeys he has taken, people he has met, business deals,

details of work, conversations and various other things that have transpired which are entirely imaginary. These circumstances he will relate in detail, readily adding to the story at the suggestion of the physician.

The physical symptoms of neuritis may or may not be marked. Cases presenting the mental phenomena may show none of the characteristic signs of neuritis, or the symptoms may be overlooked, or may have passed away before the onset of the psychosis. The symptoms vary from only slight sensory disturbances to a severe state with paralysis and contractures. It is common to find pain and tenderness over nerve trunks and muscles upon pressure, especially over calves and biceps. The deep reflexes are apt to be diminished, absent or variable with wrist or ankle drop or both in the severest cases. There may be incoordination in bringing finger tips together or touching nose with eyes closed. The wasting of muscles is usually quite noticeable.

Pupillary disturbances are frequently present. Irregular, unequal pupils which may react sluggishly to light or not at all are found. Speech defects varying from simple thickness to an actual slurring are not uncommon.

The neuritic symptoms are usually most marked during the severe delirium and are disappearing or absent when the fabrications become well developed. Frequently the anamnesis has to be depended upon for the evidence as to a neuritis having been present.

In regard to etiology, alcohol was at first and for a long time deemed the most important if not sole factor. From time to time, however, cases would appear presenting typical symptoms of polyneuritic delirium in which there was absolutely no alcoholic history. Cases may be divided into three classes as to etiology. In the first place, those with alcoholic alone as the etiological factor are by far the most numerous and typical. As will be noted in the series under examination, alcohol was given as the cause in 75 per cent of the cases.

Again, alcohol may be associated with some chronic wasting disease, an infection or a traumatism.

Lastly, cases are recorded where alcohol has played no part in producing the disease. There seems to be an underlying basis of toxæmia due to other causes. Morphine, creosote, arsenic and

other poisons, tuberculosis, typhoid fever, puerperal fever, diabetes, gastroenteritis, malignant growths, etc., have all been given as the sole cause for polyneuritic delirium. It is a question whether or not all these cases should be called polyneuritic delirium. Where there are no physical signs of a polyneuritis and really the clinical picture of a delirium as in a wasting disease, with fabrications, why should this delirium be specially designated apart from that which could be explained by the basic disease?

In a chronic disease, such as tuberculosis with the appearance of a neuritis, care must be exercised not to mistake the cause of this condition. This was clearly shown by Salomonson who described "two cases of toxic polyneuritis in phthisis," and concluded that the polyneuritis was due to the creosote given and not to the tuberculosis *per se*.

The case described so fully by Hayman seems to have been a true example of polyneuritic delirium on a morphine basis, there being no alcoholic history. The physical signs included absent knee and achillis reflex, sluggish pupillary reaction, speech and writing defects, and mentally there were present the characteristic memory defects with confabulations.

Some investigators have found that Korsakoff's psychosis more commonly occurs in women than in men. Long-Landry in the "Clinic" of Paris states that "women are attacked in the proportion of seven out of ten cases." Stanley, in a recent paper, makes a similar assertion. Paton, however, reaches the opposite conclusion, and this seems to be borne out by the present series. When the fact that alcohol is the most common etiological factor is considered, it would seem reasonable to find the disease more often in men.

Pathologically considered, the characteristic findings expected in a case of multiple neuritis are seen together with certain other changes. F. Robertson Sims, in the cases he examined, found "acute degeneration of many peripheral nerves, axonal reaction in anterior horn cells, in cells of Clarks column and in some cranial nerve nuclei and various non-systemic degeneration of intra-spinal fibers, together with acute or axonal alterations of various cells of the cortex. In one case proliferating glia cells were found in the neighborhood of altered Betz cells of the motor cortex."

The following case is reported in abstract, as a post-mortem examination was made:

C. K. Female. Born in United States. Age 30. Divorced.

Family History.—Negative.

Personal History.—Early life not unusual. Made good progress in school and took up music, remaining at home until her marriage at the age of 21. The marriage was regarded as satisfactory, but later on it was discovered her husband drank to excess. Since her marriage she has had four abortions induced with the knowledge and consent of her husband and they both have had gonorrhœa. She acquired the habit of drinking, and two years ago her husband parted from her, the grounds being her excessive drinking. Four months before admission he got a divorce and was married the following day. She (the patient) drank to excess following this, as much as a quart of whiskey a day.

Onset.—About four months ago began to be rambling in her talk, indifferent to her work. Twenty-five days before admission began to have hallucinations of sight, seeing monkeys in the trees, negroes coming in through the windows, etc. She did not seem frightened or tremulous. This lasted about a day and her physician gave her bromides after which she talked in a wandering way and was untidy. Was in a general hospital ten days before admission.

Admitted February 4, 1911.—Carried in on a stretcher, was stuporous and could not be aroused.

Physical Examination.—Height about 5 feet four inches, weight about 125 pounds. Patient semiconscious and delirious. Much atrophy of muscles of arms and legs. Excessive pain upon pressure over calves and biceps. There is double foot drop. Patellar and achillis tendon reflexes absent. Marked incoordination and tremor of extended fingers. Inability to stand. Speech slurring. Loss of control of bladder and rectum. Pulse weak, intermittent, rate 120. Pupils active. Tongue coated, breath foul. Urinalysis, 1030; acid. Heavy white ppt.; reddish amber. A trace of albumin. A yellowish offensive vaginal discharge.

Mentally patient in delirium, apathetic and indifferent to surroundings unless disturbed, when she cries out with pain. Answers to questions mostly incoherent and irrelevant. Fabrications quite marked. Hallucinations of sight and hearing. Complete disorientation. Marked memory disturbance. Lack of insight and judgment. Lumbar puncture findings were negative and general paralysis ruled out. Patient failed rapidly and died February 10, 1911.

I am indebted to Dr. F. S. Hammond, pathologist to the New Jersey State Hospital at Trenton, for the autopsy report and microscopical findings hereafter recorded.

The anatomical diagnosis was as follows:

A young white woman showing well marked muscular atrophy without evident significant loss of fatty tissue.

Dura and pia microscopically negative. Beginning basilar arteriosclerosis as shown by small patches of grayish white color in the major trunks.

Brain of small size with reduced consistency without detectable atrophy or other gross changes—1120.

Spinal cord removed. Negative to naked eye.

Subacute bilateral plastic and fibrous pleurisy. Rather well marked bilateral posterior hypostasis and œdema with areas of collapsed tissue and beginning broncho-pneumonia.

Slight mitral and aortic endocarditis of chronic type. Heart muscle markedly grayish and turbid. Left ventricle only partially contracted. Much subepicardial fat.

Incipient aortic atheroma. A few small scattered patches only. Coronaries clear.

Moderate splenic engorgement.

Rather well marked chronic interstitial hepatitis. Old adhesions about the upper and under surface of liver. Perhaps some cloudy swelling.

Stomach and intestines negative except for slight or moderate injection of mucous membrane.

Kidney usual size. Capsule firmly adherent. Substance of kidneys dark turbid red. Cortex bulges beneath capsule when cut.

Pancreas negative.

Adrenals show apparently some engorgement of the medulla and some central softening. Cause of death polyneuritic delirium (chronic diffuse nephritis with acute cloudy swelling).

MICROSCOPIC EXAMINATION.

Brain.—Nissl: Aside from the special alterations present in the large cells of the paracentral lobules and to a less degree elsewhere, on the whole there is quite good preservation of nervous elements. Pigmentation is not unduly conspicuous and with the exception of an occasional cell displaying the effects of granular disintegration there is little of significance in either the general run of cells glia or vessels.

The Betz cells, and to a considerably less degree the large cells in the calcarine areas, are the seat of a quite distinct axonal reaction. The principal features of this condition are that by no means all the large cells are affected and those in which the process is observed do not display the intense granular breaking up of the Nissl bodies and peculiar glassy appearance of the cell body usually seen in central neuritis. Although the nucleus is displaced and the chromatin altered, the process lacks the severity usually observed.

By Marchi, the paracentrals show very distinct fiber degeneration corresponding in severity to the axonal reaction in the cells. In the frontal and temporal areas the osmic spotting is quite strikingly small as compared to the paracentrals.

Spinal Cord.—Nissl: A well marked axonal reaction in the anterior horn cells most conspicuous in the dorsal and cervical regions. Pal: A

quite distinct thinning out of the columns of Goll in the dorsal and cervical segments not present in the lumbar segments. Marchi: Well marked peppering of the white matter throughout the upper levels, but distinctly more pronounced in the posterior columns and lateral marginal zones in the dorsal and cervical and noticeably less so in the lumbar.

Lumbar Ganglia.—Negative.

Gasserian Ganglia.—Negative.

Medulla.—Nissl: Moderate amount of nerve cell pigmentation. Marchi: Rather small amount of reaction noticeable; very much less than in the cord.

Peripheral nerves.—(Right and left—great sciatic, ulnar, median, posterior tibial, bracheal plexus.) Nissl: Sections negative.

Marchi: In the nerves of the lower extremity a quite faint (or even doubtful?) reaction. A rather limited number of fine black pepperings. In those of the upper extremity, the medians particularly, a very evident reaction as shown by many plainly detectable black dots, clump and linear markings.

Spleen.—Chiefly characterized by marked engorgement of the pulp.

Lungs.—Mild grade broncho-pneumonia.

Pancreas.—Negative.

Thyroid.—Negative.

Adrenals.—Negative.

Heart.—Negative by eosin and thionin.

Kidneys.—Well marked chronic interstitial nephritis with high grade acute cloudy swelling.

Liver.—High grade periportal cirrhosis and fatty infiltration.

Pituitary Body.—Negative.

Small Intestine.—No changes of significance.

Left and Right Biceps, Left and Right Gastrocnemius.—Negative.

Before a further general consideration of Korsakoff's psychosis it may be well to analyze briefly the series of cases which have offered the excuse for this discussion.

There were 24 cases, 17 men, seven women. The ages of the men ranged from 30 to 68, those of the women from 30 to 48. Alcohol seemed the most important etiological factor in 14 of the men, and four of the women. In two male cases alcohol and trauma and alcohol and infection were associated. In one male case bromides, and in a female chloral and bromides, seemed to be the active causes. In one female the trouble started following an induced abortion with probable infection and exhaustion. In another, worry, grief and ill health.

The onset in 13 cases was acute, many times resembling delirium tremens at first, which became chronic. To be exact, the

onset in these was three weeks or less. In six cases the symptoms developed in from four to seven weeks, and in five cases they were several months in making their appearance.

In 62 per cent there were decided pupillary changes and in 87 per cent there was reflex alteration.

The neuritis was so marked in nine of the males and five of the females that the patients could not walk or help themselves in any way. Seven of the male and one female case did not have hallucinations. All but one (a female) showed fabrications, this one case having passed beyond that stage. All but this same case showed disorientation, retention defects and amnesia.

Twenty or 83 per cent recovered, 15 males and five females. One male only died, the cause being lobar pneumonia. Two females died, one of these having a severe chronic nephritis, the other being the case already reported under pathology.

Seven cases, two being women, were lumbar punctured, the spinal fluid findings being in each negative.

The importance of a careful differential diagnosis cannot be emphasized too strongly. Even with a fairly accurate anamnesis, one cannot always feel certain that the case is one of Korsakoff's psychosis, and without a good anamnesis the difficulties are multiplied. When it is seen that in the writer's cases 62 per cent showed pupillary changes, *e. g.,* either irregular, unequal or sluggish in reaction, 87 per cent reflex alteration besides tremors, writing and speech defects and incoordination, and frequently these physical signs are accompanied by expansive ideas, the possibility of confounding Korsakoff's psychosis with general paralysis is apparent.

In this connection it is interesting briefly to note two cases of the series. The following is not included among the 24 already mentioned.

Psychosis in Z. T. Colored, male. Age 49. Widower. Laborer.

Family History.—Negative so far as known.

Personal History.—Early life not unusual. Received a public school education and possessed ordinary intelligence. Has been drinking more or less all his life, for ten years to excess. Never arrested for disorderly conduct but frequently warned. Has been employed as a laborer, waiter, porter, etc. Agreeable disposition. Provided for his family.

Onset.—About four weeks before admission. Had been drinking to excess and had a convulsion about that time and another September 25,

1910. Was confined to bed and attended by a physician. A few days later was restless, violent and restrained. Gradually became more quiet and able to be on his feet, but weak and not right mentally.

Admitted to the New Jersey State Hospital October 20, 1910. Was disoriented.

Physical Examination.—Thin in flesh, poor muscularity. Sways when walking without assistance. Romberg symptom present. Speech defect. Tremor of extended fingers and toes. Patellar reflexes exaggerated. Tenderness over nerve trunks and muscles of upper and lower extremities.

Mental Status.—He was usually quiet, at times restless, wanting his clothes to go to work, etc. Talks spontaneously and answers questions relevantly. His mood was agreeable; at times he would become emotional, readily shedding tears. There were marked fabrications. Complete disorientation. No insight.

The diagnosis at first favored was Korsakoff's psychosis.

The fabrications continued but were not so marked.

His physical condition seemed to be improved. On January 25, 1911, he had a convulsion which was at first attributed to uræmia. The urinalysis, however, did not confirm this. He recovered to his former condition and lumbar puncture was performed. The spinal fluid proved positive for general paralysis, there being 293 cells to the cmm. The patient finally died and autopsy confirmed this diagnosis.

About the same time the following case was presented at staff meeting making a striking contrast to the above:

N. A. Psychosis in white man. Age 56. Married. A motorman.

Family History.—Negative except for the fact that his wife had three miscarriages.

Personal History.—Childhood and early life negative. Has worked at several occupations, farmer, engineer, etc. For the past 15 years has been employed as a trolley motorman. Married at the age of 24. Has never used alcohol. A steady and industrious workman. Always got along well with his wife. For the past 15 years has had a sore on lower leg which would break down about once a year. Had a bad attack of pneumonia last winter.

Onset.—Gradual. About ten weeks before admission began to talk and act a little queerly. Was obliged to stop work at that time because he was ill and nervous. Was quite restless. Must be moving about all the time. Had been given considerable bromide by physician's prescription, the same being renewed several times. About three weeks before admission became profane, talked of having large sums of money, owning a hotel at Atlantic City. Was at times emotional. Hallucinations of sight were present. Feared his imaginary money would be stolen. Showed fabrication.

Admitted to the New Jersey State Hospital January 24, 1911. Disoriented.

Physical Examination.—A well developed white man. Numerous white pitted scars over back and chest and arms. Ventral hernia at site of old abdominal operation (12 years ago for intestinal obstruction). No scars on genitals. Smell defective. Left pupil slightly larger than right. Reacts more sluggishly. Neither pupil gives a prompt reaction. Taste defective. Sense of position poor. No tenderness over nerve trunks or muscles. Patellar reflexes absent. Other reflexes are diminished. Balancing power very poor. Unable to walk in a straight line with eyes closed. Tremor of tongue and fingers. Writing and speech defects. Speech of scanning type. Heart sounds muffled and indistinct. Pulse slow and irregular. Urine contains albumin and casts.

Mental Status.—Restless, difficult to keep in bed, headstrong and obstinate. Disoriented. Memory defects. Emotionally changeable. At times exhilarated and often tearful. Expansive, ideas of possessing wealth. Fabrications, speaking of having been at various places, seeing various people and having different imaginary experiences.

From the marked physical signs and expansive ideas general paralysis was favored at the first presentation, but lumbar puncture was negative. The negative spinal fluid findings made the diagnosis Korsakoff's psychosis necessary. This patient finally made a complete recovery.

Thus, very graphically, the difficulties at times experienced in differentiating polyneuritic delirium from general paralysis and the important laboratory aid to this solution are shown.

A number of cases of Korsakoff's psychosis were lumbar punctured and pleocytosis was never discovered. Certain writers, I. M. Dupain and G. Lerat, claim to have found lymphocytosis in polyneuritic cases, but they have not recorded any differential cell count which would be important in such circumstances.

The possibility of Korsakoff's psychosis occurring in a general paralytic has been demonstrated.

The case Z. T., already cited, might be considered one of this kind. Kræplin showed the association of these two conditions.

Henderson of the New York Psychiatric Institute reported two such cases, one in 1909, another in 1910. One of these was a hack driver, 54 years old. He had a double wrist and foot drop, tenderness over calf muscles, marked exaggeration of the tendon reflexes. Pupils reacted promptly to light and accommodation. There were memory disturbances. The polyneuritic symptoms disappeared in a week's time.

The lumbar puncture showed a pleocytosis, positive globulin reaction and positive Wassermann of the blood serum and cerebrospinal fluid.

Cases of this sort are the exception, however, and it is generally conceded in this country at least that lumbar puncture in polyneuritics shows no pleocytosis, thus making this diagnostic aid of vital importance.

Senile dements may fabricate and present amnesia and memory disturbances similar to those found in polyneuritic delirium. In senile cases, however, the gradual onset, age of the patient, continued progressive deterioration, absence of neuritic symptoms, serve to differentiate this condition.

Certain traumatic psychoses, according to A. Meyer, present features of the Korsakoff's symptom complex, especially the fabrications and memory disturbances.

The following case very aptly illustrates the complication of a trauma:

E. W. White. 48. Widower.

Family History.—According to patient, father and brother alcoholic.

Personal History.—Patient picked up June 2 along trolley track with a fractured skull, punctured wound. Taken to Mercer Hospital where skull was trephined, left frontal prominence, and a small piece of bone removed. Patient became violent, was disoriented, evidently fabricated.

Onset.—June 2, so far as known.

Admitted to the New Jersey State Hospital June 19, 1910. He was talkative, good humored. Disoriented. Fabricated.

Physical Examination.—A fairly well nourished white man, height 5 feet 8 inches, weight 147 pounds. A history of venereal scars probably chancroidal in nature. Irregular scar about six inches long forming a flap over left eye, surrounding an irregular depression in skull, left frontal prominence, following trephining. Pupils unequal, left a little larger, irregular in outline, do not react to light. Unable to test well for accommodation. Slight external strabismus. Tactile, temperature and pain sense acute except in region of scar, left side of forehead. Considerable pain and tenderness upon pressure on calves. Reflexes less active on the right. Station unsteady. Swaying to left in Romberg position. Gait unsteady and staggering. Muscular power of legs seems diminished, does not keep them flexed well. Some difficulty in pronouncing "Peter Piper." Does not sleep well. Heart sounds weak and indistinct. Pulse 68, small, compressible, rather weak. A history of hemorrhoids. Liver dullness extends to umbilicus. Urine: light straw, turbid, 1010, acid: *microscopically,* bacteria present.

Mental Status.—A little restless and confused. Disoriented, fabricating freely according to suggestion until about July 4, when he became oriented for time and place. Mood, rather happy. Amnesic period for accidents and events following same. Confused in his accounts of his previous life.

Memory poor but improving. Retention fair. School knowledge fairly good. Insight and judgment defective.

It was learned from his employer that he drank to excess.

From the history of alcoholism, neuritic physical signs above enumerated which seemed to be clearing up, confusion, disorientation, marked fabrications, defective insight and judgment, alcoholic polyneuritic delirium, Korsakoff's psychosis was favored.

General paralysis would have to be considered but was excluded by recovery and discharge August 15.

The influence of the trauma was thought to be incidental or as the exciting cause. In the light of A. Meyer's teaching, however, it may be questioned whether or not the trauma was the cause of the peculiar delirium.

One case (McG.), age 65, on account of a history of several periods of unconsciousness, apprehension, irritability and partial disorientation with defective insight, arterio-sclerotic brain disease was considered. Later on, however, fabrications developed, the symptoms finally clearing up in the course of two months, and the patient was discharged, the diagnosis being Korsakoff's psychosis.

A mistake was probably made in the case in the first place in considering arterio-sclerotic brain disease without sufficient evidence of shock or with no residual focal symptoms of the same.

A brief recital of some of the opinions as to prognosis discloses a decided difference among the various writers. The leaning seems to be toward a bad prognosis and practically all have been unanimously guarded in their expressions.

Paton states that "certain writers hold that complete recovery sometimes takes place, an affirmation which I am at present unprepared either to accept or reject."

Hurd reported five cases with only two recoveries. In his monograph Hurd says he thinks "with persistent care and intelligent treatment, the prognosis may be made better than usually considered."

Stanley says in a recent paper "the prognosis is unfavorable. The disease rarely if ever terminates in complete recovery and may prove fatal in its early or later stages. The course is apt to be prolonged, resulting in the most favorable cases in more or less impairment of memory and emotional deterioration."

Kraeplin states "some patients recover after several months. Sometimes an incurable mental weakness is developed."

Other clinicians go so far as to say they have never seen a case of Korsakoff's psychosis make a complete recovery.

In the present series of cases it has been stated that 20 out of the 24 recovered. Most of them left the hospital and resumed their former occupations, to all appearances being in as good condition as before the psychosis. Of course, "if the persistence of some slight psychic defect" should make it necessary to call the case not cured, as Paton suggests, probably a majority of alcoholics would be so considered, as doubtless all have suffered some deterioration. One case remaining in the hospital is that of an old colored man with no friends to take care of him outside, but who is now in a normal mental condition. One of the 24 is still in a more or less delirious condition. Two of the three deaths could not be laid to the polyneuritic condition altogether, as one was caused by lobar pneumonia, the other by severe chronic nephritis.

In view of spinal fluid findings one may perhaps account for some cases of reported recovery of general paralysis by the possible fact that those cases were Korsakoff's psychosis in which lumbar puncture was not performed. And vice versa, some cases of Korsakoff's disease which failed to recover may have been general paralysis in which also lumbar puncture was neglected.

It is the writer's opinion, therefore, that the prognosis in polyneuritic delirium is generally better than has been stated in the books. It must be remembered the patients may die "during the delirium from some intercurrent complication" such as pneumonia or nephritis.

Active and careful nursing and general treatment will do much to improve the prognosis. Rest in bed is imperative and the prompt withdrawal of all alcohol indicated. A diet of milk and broths, plenty of water internally, remedies to stimulate the excretory organs, hot packs or warm baths if necessary for sedative and eliminative purposes, purgatives all have their routine uses. For profound toxæmia enteroclysis of normal salt solution is effective. The usual sedative remedies, *e. g.*, the bromides, chloral, etc., are to be used as indicated, as also the hypnotics trional, sulphonal and veronal. As the delirium passes away and

POLYNEURITIC DELIRIUM-KORSAKOFF'S

Case.	Sex.	Age.	Occupation.	Etiology.	Pupils.	Reflexes.
T. C.....	M.	43	Farmer.....	Alcohol.....	Unequal.....	Much dim.....
J. C.....	M.	35	Bartender....	Alcohol.....	Normal.....	Much dim.....
M. McG...	M.	65	Miner.....	Alcohol.....	Sluggish.....	Fair reaction...
P. L.....	M.	68	Laborer.....	Alcohol.....	Contr.,unequal.	Unequal.....
J. D.....	M.	55	Plasterer.....	Alcohol.....	Reaction limited.	Dim.....
M. F.....	M.	60	Hotel keeper.	Alcohol.....	Reaction limited.	Increased.....
P. J. (col).	M.	56	Coachman....	Alcohol.....	Sluggish.....	Increased.....
E. W.....	M.	48	Candy maker.	Alcohol and trauma.	Unequal, sluggish.	Rt. dim.....
C. R.....	M.	54	Butcher.....	Alcohol and infection.	Unequal, sluggish.	Active.....
F. P.....	M.	32	Clerk.....	Alcohol.....	Sluggish.....	Active.....
Z. T. (col).	M.	49	Laborer.....	Alcohol.....	Active.....	Increased.....
N. A.....	M.	56	Motorman....	Bromide (?)....	Unequal, sluggish.	Absent.....
J. F. (col).	M.	33	Waiter.....	Alcohol.....	Active.....	Dim.....
L. T.....	M.	63	Carpenter....	Alcohol.....	Active.....	Increased.....
J. S.....	M.	49	Barber.....	Alcohol.....	Contracted.....	Almost absent..
J. G.....	M.	42	Machinist.....	Alcohol.....	Active.....	Absent.....
G. W.....	M.	30	Grocer.....	Alcohol.....	Active.....	Absent.....
J. M.....	M.	54	Laborer.....	Alcohol.....	Sluggish.....	Increased.....
E. F.....	F.	43	Housekeeper.	Alc. gastritis...	Active.....	Increased.....
E. L.....	F.	36	Housekeeper.	Alcohol.....	Active.....	Increased.....
C. K.....	F.	30	None.....	Alcohol.....	Active.....	Absent.....
S. P.....	F.	36	Housewife...	Following curretage.	Active.....	Absent.....
A. M.....	.	39	Housewife...	Grief, ill health.	No reaction to light.	Increased.....
M. W.....	F.	45	Waitress.....	Alcohol.....	Sluggish.....	Increased.....
E. D.....	F.	48	None.....	Chloral and bromide.	Sluggish.....	Increased.....

SIS. W. C. SANDY, M. D., TRENTON, N. J.

Hallucinations.	Fabrications.	Disorientation.	Retention defect.	Amnesia.	Onset.	L. P.	Duration.	Results.
Yes.	Yes.	Yes.	Yes.	Yes.	3 weeks.....	.....	2 months.....	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	1 week.....	.....	1 month	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	2 weeks.....	.....	8 months	Rec.
No..	Yes.	Yes.	Yes.	Yes.	6 or 7 weeks....	Negative	6 months	Rec.
No..	Yes.	Yes.	Yes.	Yes.	10 days	.....	.....	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	3 weeks.....	Negative	3½ months	Rec.
No..	Yes.	Yes.	Yes.	Yes.	3 weeks.....	.....	11 months	Rec.
No..	Yes.	Yes.	Yes.	Yes.	2 weeks.....	.....	2 months	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	Sev. months (?)	.....	2½ months	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	7 weeks.....	.....	2½ months	Rec.
.....	Yes.	Yes.	Yes.	Yes.	4 weeks.....	Positive (G. P.)	.....	Died.
Yes.	Yes.	Yes.	Yes.	Yes.	10 weeks.....	Negative	6 months	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	3 weeks.....	.....	1½ months	Rec.
No..	Yes.	Yes.	Yes.	Yes.	4 weeks.....	.....	5 months	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	3 months (?) ...	.....	4 months	Died. Lobar pneumonia.
(?)	Yes.	Yes.	Yes.	Yes.	2 weeks.....	.....	Still in hosp..	
Yes.	Yes.	Yes.	Yes.	Yes.	3 weeks.....	Negative	2 months	Rec.
No..	Yes.	Yes.	Yes.	Yes.	3 weeks.....	.....	3 months	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	5 weeks.....	.....	3 months	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	3 weeks.....	.....	3 months	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	4 months.....	Negative	6 months	Died.
Yes.	Yes.	Yes.	Yes.	Yes.	10 weeks.....	Negative	4 months	Died. Chronic nephritis.
Yes.	Yes.	Yes.	Yes.	Yes	3 months.....	.....	3 months	Rec.
No..	No..	Yes.	Yes.	Yes.	4 weeks.....	.....	3 months	Rec.
Yes.	Yes.	Yes.	Yes.	Yes.	5 weeks.....	.....	2 months	Rec.

if there is much heart weakness during the active delirium the stimulative effect of strychnine and digitalis is beneficial.

During convalescence, massage and passive motion are necessary to overcome the effects of the neuritis. Daily suggestion and correction of the patient's false ideas are useful in hastening the restoration of his mental equilibrium.

In conclusion, there is no doubt in the writer's mind that Korsakoff's psychosis is a distinct entity, at least from an alcoholic standpoint.

It must be admitted, however, that the symptom complex, *i. e.* memory disturbances and fabrications, is seen in other conditions, but an analysis of the cases should usually serve to differentiate such conditions. Again, it has been shown that Korsakoff's psychosis may be present in combination with some other more chronic and lasting disease.

The prognosis would seem to be more favorable than usually stated.

A CRITICAL ESSAY ON MENTAL TESTS IN THEIR RELATION TO EPILEPSY

BY

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Sonderabdruck aus der Zeitschrift „Epilepsia". Vol. IV.

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BY

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Psychopathologist at State Village for Epileptics, clinical assistant Postgraduate Medical School New York. Read before the National association for study of Epilepsy in Vineland June 1912.

(From the Psychopathological Laboratory of the New Jersey State Village for Epileptics at Skillman.)

Psychology is a discipline of undue hopes and uncritical scepticism. Just at the present time the optimistic point of view is gaining the upper hand. It has been a hard battle, which in forty years time has elevated psychology from a cinderella science domiciled in one room at the Leipzig University to palace-like institutions such as for instance the Harvard Psychological Institute, and what more, has caused other branches of science previously either indifferent or opposed to psychology, to become

desirous of allying thenselves with the latter, — chiefly the pedagogic, medical, and the legal sciences.

It is in reality wonderful to trace the development of psychology. In this short period of time, not only has theoretical psychology arrived at a high standard of development, cultivating the knowledge of fundamental psychological experiences and forwarding the analysis of complex psychological processes by resolving them to their elements, but it is now preparing ground for the differentiation of applied branches of psychology. The psychology of children, pedagogical and criminal, religious and medical psychology have now begun to earn their own living.

Medical Psychology has even begun to split itself into two sub branches: psychopathology and pathopsychology.[1]) Although psychopathology is a broader term and although as a rule, a pathopsychologist must be primarily a psychopathologist, the ultimate purpose is different. While the first one tries to utilise the psychological experience in medicine, especially in psychiatry, the latter tries to illuminate normal processes by comparing the normal with abnormal phenomena, thus getting a better knowledge of the former. It is like importing raw psychological material into the Medical Realm and exporting in exchange the same substance as a wrought product into Psychology.

Unfortunately, the unexpected and unequaled development of psychology has intoxicated many up to now critical, cautious minds of skillful psychologists, has made them impatient to get "results" and draw practical conclusions. What is worse however, psychology is obliged to pay a severe penalty for once being a domain open to everbody. While no one in physics for instance or che-

[1]) The proof of interest which normal psychology is taking in abnormal psychology, is shown by the necessity for a special magazine for psychopathology, "Die Zeitschrift für Pathopsychologie", which has just made its appearance. All eminent german psychologists are contributors; France is represented by Janet and Bergson, american scientists by Muensterberg.

mistry would dare to enter into serious research work unless armed with a thorough knowledge of theory and practice, psychology is open to everyone. No licence is required, or rather a short course at some laboratory, a few books, suffice to qualify as an expert in psychology. It is however still worse. While a chemist trained in organic and anorganic chemistry would never dare to enter into research work in biological chemistry without making himself perfectly acquainted with biology, a psychologist seems justified to investigate the pathological phenomena of psychology without becoming acquainted with pathology. I do not need to add that even the one year psychologists aspire to utilize the fragments of their psychological knowledge in different other branches. The result of such a procedure is disastrous, as I pointed out in one of my previous papers with reference to Wundts criticism on Meumans work in psychological pedagogics[1]), which he described as pernicious to the "entente cordiale" of psychology with other branches of science. No doubt, that at the present time, enthusiasm about psychology is great, but even nowadays the more sober scientist, especially in medicine, is beginning to retain a cool reserve. There are enough enthusiasts in psychology; I am selecting the more ungrateful part of the pessimist, who will try to put a scientific brake of critical consideration even there, where success seems to be beyond doubt. I am doing this however in the spirit of the highest optimism, that there are at the present time points of true cognition, which will be able to stand any possible epistomological criticism and that, helping to correct available mistakes, the union of psychology with medicine will be an ultimate one.

One of the most sensational psychological attainments is the so called *Binet Simon* test chiefly introduced and modi-

[1]) The Detection of a case of simulation of insanity. Journal of abnormal Psychology, 1911, Vol. 6. Also compare Wundts Psychologische Studien, 5, 1910.

fied for use in America by *Goddard.*[1]) I must assume that the reader is acquainted at least with the general outlines of this test and knows about the excellent research work made by *Goddard* in normal children. I shall nevertheless in order to make clear the genesis of the test, sketch an outline of its original purpose. The french psychologists *Binet* and *Simon* prepared this test primarily for the use of normal children, in order to gain a more exact and uniform basis for placing children in the corresponding school grades. The necessary assumption for the arrangement was the uniformity of conditions, under which a child of a given age was supposed to be. A child for instance, of the age of nine, should have according to the arrangement of french schools such and such knowledge; the tests were made a posteriori with a selection of the highest percentaged questions, answered by children of a given age. Having a practical point of view in mind, this test has been of the greatest value in France and could be easily applied as a routine examination of children in any country, with the neccesary modifications. Although native ability plays an important role in such a test, the training is an essential condition of the child's success. If a child failed, and was retarded for instance two years the *Binet* test would diagnose the case as "retarded", without giving the cause for retardation. Such a retardation might be due to mental dullness — an inborn condition — or to lack of previous educational experiences, to sickness, adenoids, psychopathic timidity and nervousness or other more accidental causes. It is even possible that one should attribute to a child which is only one year behind his norm according to the B. S. test an especially

[1]) *A. Binet* and *Simon.* Les developpements de l'intelligence chez les enfants. L'Année Psychologique Nr. 14, 1908.

L'intelligence des imbéciles, ibid 15, 1909.

H. H. Goddard.— A measuring scale for intelligence; The Training School, 6, 1910. The *Binet Simon* tests of intellectual capacity, ibid 5, 1908.

good native mentality, as he in spite of some cause (epilepsy for instance, as we shall later have the opportunity to see)[1]) has fallen no more than a year behind.

We thus see, that difficulties arise even in dealing with normal children and that even in public schools, this test would cause teachers who lack a psychological or more important even, a medical knowledge, to commit graver mistakes with the *Binet Simon* test, than they otherwise would. I can however say, that every teacher — provided he is of the same sex as the child —, would be able, when properly instructed, to apply the test under one condition, that the answers should be recorded verbatim, scored with the aid of a stop watch. The real problem, however should begin at this point, and every child having the record of failure, that is, every child having the same opportunity as the others, which would fail, should be turned over to a psychopathologist (with medical knowledge) who would have the task to investigate the cause. Thus applied, the *Binet Simon* test even in it's present form would be of great value, as it would lead to the individualisation of pedagogical attention and would result in proper segregation. Such a procedure however, would under the present arrangement of the test, although pedagogically interesting and valuable, be of little use to the scientific phase of the problem.

The apparent success of this test with normal children led the french psychologists to apply it as primarily divised for pedagogical purposes and for the use of normal children, to abnormal ones. They did not even stop at the using of this test upon children, but thought is possible to apply it to adults as well.[2]) The necessary hypothesis which led them to such an application is a psychogenetic point of view, that imbeciles reach a certain maximum of deve-

[1]) Cmp. case 7 on page 32.

[2]) *A. Binet* and *T. Simon*. Langage et Pensée. L'année Psychologique 14, 1908.

lopment and then stop for ever and that therefore, an imbecile of twenty five years may be compared with a child of five, if such an individual fails in the test devised for a child of six. *Kuelpe*[1]), justly questions this point of view, claiming that one has no more right to compare imbeciles with normal children of a certain age, than to claim that dwarfs are physically children who did not develop above a certain age. This point of view is indeed not new. *Wildermuth*[2]) tried to adapt this point of view to idiots, but failed to convert others to his mode of thinking. Let us consider the case of an imbecile of eighteen, whose mental age according to the *Binet Simon* scale is six years. As a rule such a low grade imbecile is far below a normal child of that age regarding adaptability to new surroundings, or ability for learning or being trained. He may exeed on the other hand a normal child in the knowledge of money, counting etc., which knowledge he has acquired during the twelve years of additional life. Finally, the sexual maturity will disclose a new life of inner psychic experiences unknown to the child. The same dissimilarity exists between a child and a dwarf, who is rather a caricature of an adult's body; in like manner the imbecile's mind is a caricature of a normal adult's mind.

This objection to a grading like the *Binet Simon* could be overcome, if instead of years, a system of grading independent of age would be substituted. For instance, a given complex of tests would indicate a certain grade of development. The same complex with graded difficulty would indicate a higher degree of mentality. Such a grading would necessarily be a quantitative one in most

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[1]) *Kuelpe*, Psychologie und Medizin, Zeitschrift für Pathopsychologie; 1. Band, 2. und 3. Heft. Compare also an article of Revault D'Allonnes, in Journal de Psychologie normal et pathologique, 1911, in which article he strongly opposes this parallelism.

[2]) Compare *Kraepelin* Psychiatrie Vol. 2, paragraph on idiocy.

of the tests. The *Binet* test has some of it's tests arranged in this way, such as for instance the impressibility test for words and digits, unfortunately without a systematic arrangement. In such an arrangement the grading would also be artificial, but it would do away with the confusion, especially among laymen, that imbeciles are children with a stunted development.

Another strong objection to the *Binet Simon* test is the haphazard arrangement of the questions. We miss an appeal to specified mental abilities, like memory, perceptibility, linguistic expression etc. for each age. The child of six years for instance should repeat sixteen syllables. This test does not appear again before the age of twelve, in which the child is supposed to repeat twenty six syllables. Is it of no vital interest, how quickly this ability developes in children between the age of six and twelve? If it is true that a child of eight is able to reproduce only two facts from a story once read and a child a years older six facts, is it of no interest to know, whether this great improvement is due to the rapid development of the childs memory, to a better understanding, or to an ability for linguistic expression. Why again is this test discontinued for the following ages? Is there no age limit, in which a child not only could but should repeat this story correctly and exactly? It is a so called "Aussage"[1]) test, which has gained lately great interest for itself in psychopathology. These examples are but a few out of many ones.

All these objections are of psychological character. There are others of more formalistic character, to the effect, that some questions are too easy, others too difficult for the pertaining age. As I myself have not worked with normal children and as besides, all workers along this line have

[1]) A separate article regarding the evidence test as comparative study of normal and abnormal, gained by the help of a moving picture device, will soon follow.

recognized this fact and made systematic objections and suggestions, there is no need of dwelling on this subject.[1])

Hence, there are many objections to the *Binet Simon* test even regarding its application to normal children, those arising in its application to abnormal children must necessarily be increased.

Imbecility is a collective diagnosis of many conditions not only dissimilar regarding etiology but also in its manifestations. It is often difficult to differentiate where the imbecility ends and the normal dulness begins. Psychiatry calls imbecility an abnormal state of mind with a manifestation of inferior intellect, a state which is either of congenital origin or which had its origin in some pathological conditions occuring in earliest childhood.

The latter conditions are strictly taken cases of dementia, it is however impossible to distinguish, especially from an anamnesis taken later in life, such a condition from infantile imbecility if it existed, as the impressions gained before the age of four are as a rule entirely forgotten. If for instance, a child becomes blind before the age of four, there is no difference between his blindness in later life and that of a person with congenital blindness. Both are unable to have any visual sensation or representation. If a person however becomes blind after the age of four, he his able to have visual images, be it only an idea of darkness. It is in like manner with imbecility. It is a defect, a minus, which descriptively could well be explained by the word amentia[2]), in opposition to a process of a diseased

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[1]) Compare *O. Decroly* and *J. Degand.* La Mésure de l'intelligence chez des enfants normaux d'après les tests de Mm. *Binet* et *Simon;* nouvelles contributions critiques. Archives de Psychologie, 9, 1910.

Also *G. M. Whipple,* Manual of Mental and Physical Tests. Page 514 and following.

[2]) The term Amentia which indicates the absence of mind, as the opposite to Dementia indicating the process of elongation from (once a normal) mind, is a term for a long time in use in the english

mind, dementia. A priori we must say, that there will be a vast difference in those conditions, and we can perceive that a conventional test like the *Binet Simon* test would not be able to make any differentiation, as it is theoretically feasible that an imbecile, reaches a certain degree of development, whereas the dement deteriorates apparently to the same point, both resulting in the mental age let us say of six.

The one diagnostically important point in imbecility is the impossibility of any marked improvement and the lack of the capacity for gaining knowledge by experience. The memory and an ability for gaining even an extraordinary mechanical knowledge of facts can however be intact. I remember seeing in Rome N. Y., at the Custodial Institution, an imbecile of a very low grade, who was able to recite the capitals of all the states and knew a great many historical data. These facts he repeated however, in a parrot like manner without inner understanding[1]). To make a diagnostical point of calling imbecile all those who are three years retarded in the Binet Simon test, means to create a new diagnosis, only valuable in its application to the Binet Simon test. There is no doubt, that there is a possibility of training an imbecile along some lines, where mechanical knowledge or memory is concerned, so that an individual whose grading was dependent on mechanical

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terminology; unfortunately Meynert usurped this term for a diagnosis of a disease hitherto known as Acute Confusional Insanity.

As Amentia in its specialized term became acclimatized in the last few years in the american psychiatrical nomenclature and is in use in some of the newest handbooks on psychiatry (for instance in Paton's book) it is better in order to avoid confusion tó abstain from the usage of this term in its original meaning.

[1]) Another still lower grade imbecile had an extraordinary musical talent being able to play correctly any piece on the piano which he had once heard. In our own institution we have a boy of ten whose mentality is not higher than a high grade idiot and who also shows a great musical ability. Another patient has a marvelous memory for data and names.

appliance and diagnostication, after a certain training would test perhaps no more than a year or two behind his age after a week training thus reaching the degree of backwardness or dulness. As on the other hand, a dementing process may set in slowly in a child (juvenile paralysis, dementia praecox, impossible to perceive in their initial stages, without mentioning epilepsy) a previously normal child might be classified as an imbecile. As we previously mentioned, imbecility is a well defined although not always an easy diagnosis and the Binet test is not adaptable to the making of a diagnosis of imbecility. Pedagogical psychologists too easily lose track of the fact, that imbecility is a term of pathology and not simply a gradation of mental ability. Such a diagnosis should be left entirely to a medical psychopathologist[1]) We do not make a diagnosis based on the tests alone but on the whole clinical picture which must correspond to the finding of the test. If we find sugar in urine, this fact is not sufficient to make the diagnosis of diabetes. We can however speak of a condition of glycosuria; it may be accidental, due to an unusual amount of sugar consumed or to some transitory pathological cause. The pathologist making the test would simply state the fact without making a diagnosis. Again, the finding of diphtheria bacilli in the throat does not necessitate the person having the disease. A diagnosis is a logical process of elimination, of narrowing the premises to only one possible conclusion. Therefor, not only this test, but any test what so ever, should not solely be taken into consideration, but a person's reaction to the necessities of life, his behaviour under unusual circumstances, and finally one should

[1]) In other words, the treatment of a subnormal child could be well left in the hands of pedagogical experts, the abnormal one belongs to the care of physicians. On the other hand however even in the treatment of subnormal children, the teacher would do well to consult a mental expert and vice versa, a physician could not be sucessful in the development of abnormal children, without the cooperation of pedagogues.

study the surroundings among which he was brought up in other words make a careful study of his life. It is not right to make a diagnosis of imbecility from the comparison of for instance a child of lower race with a higher one. It may be inferior as to race, but be up to the mark, for its own racial standard. This caution is especially imperious in America, where children of so many races and nationalities are brought into consideration.

After this critical synopsis, we may now turn to our own investigation. The Binet Simon test has been used on all patients at the New Jersey State Village for Epileptics. It was applied in about three hundred cases by Dr. Wallin who recently published the result of his investigations in the „Transactions for the National study of Epilepsy", and it has been revised and supplemented by me. Three hundred and ninety eight cases were taken into consideration, three hundred being adults, and ninety eight children under sixteen[1]).

Wallin found, that 5. 7 % of all epileptics were idiots, 27. 3 % were imbeciles and 61. 5 % were morons, (only slightly feebleminded) in other words, not taking into consideration the grades of feeblemindedness 5. 7 % were idiots end 88. 8 % were imbeciles, and hardly 5 ½ % were either

[1]) Dr. Wallin made the tests especially valuable for a statistical investigation because of the fact that he not only registered the answers to the questions devised for the given age but he would apply all tests to each patient. I followed his example.

In making statistics, he took as a differential age between „child" and „adult", the legal distinction: the age of 21. This distinction does not seem to me justifiable. From a psychological point of view there is no real difference between a boy of 21 and 22 years. No doubt that there is not a sharp line drawn where the mentality of the child stops and that of the adult begins. As puberty however causes a descided change in a persons physical and mental appearance, the age of sixteen, in which all children of both sexes normally in our climate gain sexual maturity, thus becoming „men and women", was selected as the dividing line, inasmuch as the Binet Simon test does not test over the age of 12.

retarded or normal, the actual test being only applicable up to the age of twelve, as the test originally devised for the thirteenth year and later transferred to the fifteenth, is not reliable and much too difficult. Judging from his conclusion, *Wallin* realized, that there is an essential difference between epileptic and „amented" imbeciles.[1]) By comparison with *Goddard's* results he thought to ascertain that, the mind of epileptics is higher than that of simple imbeciles, as *Goddard's* list of distribution shows that in almost the same number of Vineland patients 19.2 were idiots, 54.0 were straight imbeciles and 26.0 were morons. The majority of patients were imbeciles while the majority of epileptics at *Skillman* were morons. My reasons for not subscribing to this classification, are evident from the above. Epileptics are not imbeciles but demented. It is a problem in itself to find out how many epileptics are congenitally feebleminded and how many are demented, having previously a normal mind. The best proof against such a classification is the fact, that, while in the series of tested imbeciles, who remained in the institution, the diagnosis would probably be constant, the same epileptic material would show year by year a lower grading, that is if the diagnosis were based only upon the *Binet*

[1]) „It is apparent that there is a striking difference between epileptic degenerates and feebleminded retardives in the matter of intelligence. The intellectual superiority of the epileptic defective is conspicuous." Transactions of the National Association for the Study of Epilepsy. 1911, P. 36.

If *Wallin* unterstands by the superiority of epileptics over imbeciles their congenital potential ability, I should agree with him. It is however selfunderstood that if the inborn defect in some epileptics is due to epilepsy, in other words if those epileptics who are also congenitally mentally defective would only be taken into comparison their superiority would be at least very problematic. As the phychological comparison of these, with those otherwise imbecile is only permissable as I will have the opportunity to sustain in the latter part of the paper, *Wallin's* assertion can only be interpreted in the sense that epileptics are in the greatest majority not congenitally feebleminded.

Simon test and were made an arithmetical procedure of summing up the tests correctly passed and the failures. A striking example of this will be shown later.[1]) The diagnosis therefore would be variable. However, in order to compare to some degree the mentality of dements it would be permissable to make a diagnosis allied to idiots, to imbeciles and to morons. The analogy however would be but very superficial as between the mind of a demented and amented person there are just those differences, which make it worth while to insert the psychological probe as deep as possible, in order to study these essential differences. *Wallin's* assertion, that the mind of epileptics is different, is not sufficient and the confusion of welldefined termina unjustifiable.

To statistically study the percentage of failure in a given test, is of promising interest regarding the finding out of the direction either of the defect or the deterioration. Let us first review the mental characteristics of chronic epileptics as they have been clinically observed, in order to ascertain, whether the clinical observation is parallel with our findings. We can disregard the mental manifestations due directly to seizures and only review those symptoms which are apparent in the periods free from seizures.

The consciousness at these times is clear, the patient is able to recognize his surroundings and react sensibly. The process of thinking, however is greatly prolonged, as association experiments can prove, their interest is steadily narrowing, finally confining itself to their own person, making them egotistic, peremptory in their desires and hypochondriacal. It is therefore hard to gain their interest in anything outside of their own bodily wellfare. Their conversation and mode of expression is stereotype. The same jokes are made day by day, the same arguments put forward; the are unable to give a straight answer, but lose themselves in their narratives, in circumstantial details. No new

[1]) See case 6 on page 31.

experiences are gained, or only in great limitation. However an epileptic, in opposition to other forms of deterioration is able to utilize his previously acquired knowledge, although in narrowed limits.

This short description is not necessarily true of all epileptics. Many of them suffer but little from their affliction, at least in their intellectual sphere, others again show a residual permanent impairment of memory and reach a degree of dementia, which is equivalent to the lowest degree of idiocy; this latter is often the case, when epilepsy starts in childhood.

We at once, from the above given clinical picture are able to perceive the difficulties in the application of the *Binet* test to epileptics. A steady, tense, attention and interest is an essential condition even in normals. To gain it in children is an art in itself, "the child must always be won", ". . ." at all events get down to the level of the child", according to the instructions of *Goddard*. To do this an experience with children and a knowledge of child psychology is a conditio sine qua non. The same is true of epileptic or other psychic abnormalities. A knowledge of their psychology, the knowledge of different psychopathic manifestations, bearing no reference to intelligence, is essential to the understanding and the applying of the test. The general psychomotor retardation which in many cases increases to mental inhibition, a priori excludes the consideration of time limit, which is assigned for normal people as applicable to epileptics. We know for instance, that the retardation of thought process, and even an inhibition is not necessarily due to defective intellect, and while this symptom is most pronounced in Manic Depressive Insanity, patients suffering from this disease do not undergo any loss of intelligence. This difference is very essential. While there are many conditions of the mind which resemble each other the ones are temporary and capable of recovery, the others such as dementia implie a permanent damage to the mind.

This factor therefore calls for the modificatian of the *Binet* test application, as not the time required, but the modus of work must be taken into consideration. Another obstacle in epileptics is their lack of previous educational advantages. Most of the children committed to the Village can neither read nor write, thus making it impossible to apply many tests, which are based on that elementary knowledge. As a rule epileptics with severe convulsive manifestations are not admitted to public schools and seldom get a home education: They are, for this reason, entirely unprepared to solve any mental problem. Finally, some of the failures are due to the lack of interest of analphabets on the whole, and institutionised patients in particular. To these failures belong questions of age and date. In taking histories, in my capacity as physician at the Danvers State Hospital for the Insane, I had the opportunity to observe that for instance the illitterate Irish or Jewish informers were utterly unable to give either their own exact age or that of their families. If they knew their age within the limit of five to ten years, it was the best they could do. If therefore, patients especially adult women are unable to tell their age, it should only be recorded as a failure, when an absurd answer is given. For instance, when a person of thirty would give his age as sixteen or fifty etc. The same remark is applied to the knowledge of the date. We are well aware, how easy it is to lose track of the exact date even in or own cases, just as soon as we cease reading newspapers, writing letters or otherwise fail to do something which habitually recalls to us the date. If therefore a patient has an idea of the date, it should suffice for the judging of his intellectual ability, whether or not be is capable of grasping the meaning of the same.

Let us direct our attention to table.[1]) The *Binet Simon* test as devised in 1908 is printed there with the percentages of failures, for each of the individual tests. For the tests 54, 55 and 56, the percentage of correct

[1]) page 41.

answers are recorded, as these tests are the ones most contested and criticised as much too difficult being either too specialized or too much dependent on previous schooling. The writing tests Nr. 23 and 33, were not taken into consideration at all, as too many patients were illitterate. The other tests which implied a knowledge of reading and writing, were only checked as failures, when the given individual failed in spite of his knowledge of writing and realing. Our list includes 300 adults (above 16 years of age) and 98 children up to the age of 16 inclusive. The age limit of 16 was taken for the reason that the 16 year old individuals could still be classified under the circumstances as retarded to the age limit of 12, comprised in the *Binet Simon* test.[1]) The number 98 decreased with the higher age, as some of the children were under the age of a given test. For the age 6 therefore the percentage was calculated from the number 95, age 7 from the number 92, age 8 from 90, age 9 from 87, age 16 from 84, age 11 from 83, age 12 from 70, and for the last three tests the percentages were reckoned out of 57 individuals under 17, while the number of 300 adults was constant.

The percentage of failures for the test 1, — 24, is between 4.5 % as a minimum and 30.6% as a maximum. However, the test Nr. 15, calling for a repetition of 16 syllables which is passed normally by children of six years, shows a percentage of failures as high as 65.3%; the repetition of 5 numerals (test 25) has another high percentage of failures, 43.3%, only $^2/_3$ as high in comparison with the previous one. Beginning with test Nr. 43 the percentage of failures is over 50% with the maximum of 82.4% for the repetition of a sentence with 26 syllables, followed by a close second of the test 50, wich is a test of the repetition of seven digits. Other tests which show a percentage of failure of 80 % is the test for free associations (Nr. 49—80.ü %) and the test for inference (test 53, — 80.3%).

By comparing the percentage of failures in children, we

[1]) Compare former foot note, page 11.

shall see that with the exception of test Nr. 15, the percentage of failures in children is higher than in adults, in the tests 1, to 37. From the test 38 onward, the proportion is reversed, as the adults failed oftener, that is with the exception of test 41 and 42. In the tests 26, 35, 37 and 44, the difference is less than 3% in favor of adults, while in test 15, it is in favor of the children.

More than 50% of our patients failed in the test for impressibility; regarding the relative percentage of children and adults, the latter show a somewhat higher percentage even in the test Nr. 15. intended for 6 year old children. In the test Nr. 52, the difference is considerable, being 11% higher. In the repeating of 5 numerals the adults have a somewhat better record, while in the repitition of 7 digits, 16½ % more adults failed to respond. In remembering six facts from a story once read, there is also a higher rate of failures by 8% to the disfavor of adults.

The clinical observation of epileptics regarding memory defects is thus confirmed by the *Binet Simon* test. On the other hand, our statistics show, that this defect is acquired and not inborn. Children under 17, although many of them of very low mentality undoubtedly of congenital character show a better record regarding impressibility, even in a test designed for children not older than six years, while in a somewhat harder test the difference is striking. This indicates that the memory ability in Epilepsy deteriorates with age. The impressibility for digits, if there are no more than five is somewhat easier for adults; this is easily understood as adults are more familiar with numbers and as on the whole, this test is easier than the repetition of long sentences.

The most interesting percentage of failure, is the correct arrangement of weights in their order of difference of 3 grams. No more than 21 out of a hundred adults were able to do this test correctly, whereas 45 out of 100 children succeeded. This test is quite easy, and a normal person can do it without difficulty. Among those patients

who failed, were men and women, who otherwise responded to most of the other tests. As there is a very large difference in the ability to perform this test between children and adults to the disfavor of the latter, we must hypothetically assume therefore, that some condition developes in epileptics with age, which interferes with the ability for recognising smaller differences of weight. It is not a matter of intelligence, as we observed those patients to fail, whom we might call practically normal. On the other hand, much more defective children were shown to succeed. As the recognition of weight is due to inner tactile sensations of the joints and muscles, our hypothesis would be that a degeneration of that sensibility sets in. We could speculate, that the state of tension and convulsive movements, clonic and tonic contractions due to seizures, interfere with the normal functions of the joints and muscles. We reserve for a later period an exhaustive study of the sensibility of epileptics in that direction.

The retardation of thought process, found to be customary in epileptics is supported by the high pereentage of failure in test 47, and its preponderance in adult patients is shown as well. The deterioration in this regard is progressive with the duration of the disease. In the testing of more than 150 patients, I remarked, that the number of repetitions is very large. This perseveration is however otherwise noticeable. While giving to patients sentences to repeat, if the interval between sentences was not long enough, the patient, especially of a lower grade, was apt to repeat either partially, or entirely, the preceding sentence given him. The same is applied to numbers. This fact, which will also be an object of special investigation, confirms the clinical traits mentioned above.[1])

[1]) A casual observation made by one of the assistant physicians at our institution (Dr. *Gisela von Poswik*) may possibly throw an interesting light on the etiology of perseveration as being possibly a retardation of transmission of stimuli to the sensoric brain centers.

A boy of thirteen extremely dull and probably congenitally defec-

The power of combination and concentration is in epileptics also a subject of deterioration. Here also our observations correspond to those made in clinics. It is an effort for epileptics to concentrate their minds; in order to solve test 45 or 53, an interest in the test is necessary. An epileptic dement, becomes more and more passive and slow in thinking, which again explains a better record in children, than in adults.

The worse record of adults in some other tests is more of external character. For instance, the higher percentage of children, in test 46, may readily be explained by the fact, that whereas the latter, who are admitted to the State Village receive school education at our own special school, the former adult patients, who never or for only a very short time, have attended school, naturally have greater difficulty in the above named test. The success is therefore chiefly a matter of training.[1])

In the easy tests devised for smaller children, the adults show by far a smaller percentage. As we have seen that the more difficult the tests relatively were, the better solved they were by children, it proves, first, that the response

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tive with severe serial epileptic seizures, and whose mental examination revealed perseveration in associations to a high degree, was tested regarding his olfactory sense. When the valerian solution was given to him to smell, he was unable to tell what it smelled like. When menthol was given, he first said „I don't know". After the withdrawal of the bottle he said, „it was like white candies". The essence of cloves was then given, to which he answered „it smells like white peppermints". Finally when Asa Foetida was demonstrated he promptly said," it smells like red roses; (the essence of cloves the stimulus given him before Asa Foetida, has really a slight resemblance to the smell of red roses). A few seconds after the withdrawal of the bottle with the disagreable ingredients, the latter came to his consciousness which was markedly evident from his facial expression which showed disgust. The same patient showed an identical peculiarity regarding gustatory sensations.

[1]) This proves that it is justifiable to maintain schools in epileptic insitutions.

of many individuals, is dependent on training received in later life, and second, that persons if feebleminded do develope within certain limits during later life, as undoubtedly, among our patients there are quite a number of those who besides being epileptic are also congenitally feebleminded, and who even in spite of epilepsy are able to develope along certain lines involving knowledge, while deteriorating along other lines regarding e. g. memory. Thus it is natural, that the ability to perform elementary arithmetic is acquired in later life and that they learn to differentiate money in the outside world, while children, who perhaps were brought into custodial care when four or five years of age, have never had even a chance to see a quarter or half a dollar. The days of the week and the months of the year is a knowledge mechanically acquired by most adults. That it is only mechanical one can easily satisfy oneself, by asking a feebleminded person, who has promptly and faultlessly recited the months, how many months there are in a year. If the imbecile is of quite low grade, he will not be able to answer at all, or will promptly give an irrelevant and mechanical answer such as I had an opportunity to record as answer of a sixteen year old imbecile girl; „every month has 30 days with the exception of February, which has 31." The middle grade imbecile will answer this satisfactorily, but will surely fail to answer the following problem: if somebody in November asks you to return three months later when would it be? As previously stated an imbecile will fail upon this question, in the greatest majority of cases. In order to eliminate a simple computation, it is well to choose an example, in which the question would imply some month after December, as many imbeciles who might be able to add three months to May for instance are unable too proceed when they reach the last month of the year. *Decroly* and *Degand*, in their criticism of the *Binet Simon* test, raised the objection that these tests were too mechanical, but could be modified. It seems to me, that the proposed suggestion is quite an

adequate one. If the child is unable to repeat the months, this ignorance should not be recorded against it. As the proof of intelligence is the ability to utilise the acquired knowledge, it seems to me that the proposed modification meets this problem.

The statistics which I gathered, can also be exploited in a pathopsychological direction, as the statistic of the responses of epileptic dements, may readily be a helping guide to eliminate inappropriate tests. The proposed change of the months and days of the week, should also be utilised for normal children. Generally speaking, the individual application should be carefully given and in each doubtful case where there is a disproportion of answers in a certain direction, the question should be raised, whether the case is not an incipient one of some process of deterioration. In this way, many cases of brain disease, could be recognised and cared for in their early stages, as for instance, in hereditary syphilis, which process could be checked in positive cases, after de presence of lues had been ascertained by the *Wasserman* test, by the application of the proper treatment.

After applying the *Binet Simon* test to a few hundred patients, I have become convinced that the idea of the test is a most ingenious one, and is a great step forward in the determination of mental conditions. As a physician in an insane hospital, I was often confronted, as well as my colleagues, with unsurmountable difficulties, in the defining of a patient's mental condition. Again and again, the range of information[1]) was proven worthless and a good history of previous life was not always at hand. The necessity for such tests is especially urgent for the use of forensic psychiatry.

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[1]) How little actual knowledge has to do with native ability, was shown in the statistical study made among normal laborers in Berlin. The same was observed from statistics gathered in the British Army.

The *Binet Simon* test is a first step. To be of scientific use it needs long experimenting with; before its perfection, it should not be let out of the hands of specialists and should be confined to laboratories. I personally owe great enlightenment to this test and faulty as it is, it has helped to detect many conditions in epileptics. My criticism, therefore was a benevolent one and because I am anxious to see it in general use, I am also desirous of having it reach a perfected state, as far as it is possible. It is a long way to satisfy scientific criticism. It is from the psychologist working with normal people, that we expect the improvement to come, and we, who are engaged in psychopathological research, will gladly support this purpose by the adapting their experiments to our own human material.

We have previously said however, that the *Binet Simon* test will not help to decide in individual cases, whether or not the case is demented or imbecile. Other tests, have been devised and originated mostly in America. A very simple but very adequate one is the *Goddard's* Form Board test. It consists of placing ten blocks of various shapes into corresponding holes. The points of interest, are the time required and the method of procedure. Referring to the *Binet Simon* test I spoke of the impossibility for drawing absolute conclusions from the time curve. In normal individuals, it is permissable to draw inferences from a time curve; as applied to the abnormal, the objection from a methodological standpoint is that one cannot work with two variable unknown quantities. This is true in arithmatic, it is true of any investigation. The one norm must be therefore, the assumption of uniformity of one condition. Besides, as Healy says: „it would seem quite apparent that for estimating mental ability the method pursued in this task is of much greater value than the actual time. Probably all would acknowledge that a trial method, where the subject proceeds intelligently from one apparent possibility to another, even though a relatively long time is

consumed, will not necessarily indicate lack of native ability."[1])

All our patients were tested with the Form Board test. Dr. *Wallin* made a summary of this test regarding time, finding a parallelism between de mental age ond the durati on of procedure. The relation was inversely proportional; the lower the mental age, the longer the time. The curve, which he worked out shows a rapid fall from the age of three to five, a somewhat slower fall between the ages of five and nine and a very gradual descendance from this point on. The average time required for the ages three to seven (or imbeciles according to his classification) is 100.7 seconds; for the ages 8 to 12, (morons) only 19.5 seconds; and for those who passed the *Binet Simon* test devised for the age 13 was 14.18 seconds.

I do not know, what were *Wallin's* conclusions regarding these facts as he has not published a paper relating to this subject. To me it indicates only the confirmation of clinical observation about psychomotor retardation. As this symptom increases with the duration of the disease and with the degree of deterioration, it is obvious that the length of time would increase with the lower mental age. An abnormally prolonged time will therefore always indicate mental inferiority, but not native ability.

Of much greater importance is the method of procedure. This test is easy and only low grade imbeciles completely fail in it, while the medium grades although they make errors, finally succeed after many trials.

A few examples will illustrate this point:

1. A normal, very bright boy of three years succeeds in 46 seconds with one mistake, placing a similar but smaller block into a larger hole. He rectified his mistake, and is able to perform the test correctly when sufficiently attentive. His

[1]) Tests for Practical Mental Classification, by *William Healy* and *Grace Fernald*. The Psychological Monographs Vol. 13, Nr. 2.

method of procedure was to select a block and then by comparison with the shapes of the holes, find the corresponding one. He compared only by visual comparison, not placing the block into the hole, unless subjectively positive that it was the correct one.

2. An epileptic, 36 years old, male, very poor schooling. Has been in the institution since Mai 1911, and is one of the lowest grade patients. He answers questions half absentmindedly, and it is hard to obtain any information from him. He grades about 4, in the Binet scale. His record in the form board test is a follows. Time, 1 minute 38 seconds. Places correctly every block in the corresponding hole, in a manner similar to the above cited child.

3. An epileptic girl of 15. Father, alcoholic and feebleminded, mother epileptic. Has three out of four brothers who are epileptics; mother and 2 brothers also patients in the institution. B. S. age $6^1/_2$. Never attended school, but took care of household which was naturally in a state of terrible neglect. Is attending the Kindergarten class in our schools, where very slight progress if any is noticed. She is however anxious to learn to write in order to be able to compose letters. Has severe convulsions, but with free intervals of months. Has been in the institution since October 1911.

First trial: gave up after a minute attempt. Proceeds in the following manner: takes a block and tries to fit it an hole which looks the most like it. Tries impossible moves: tries to force a square block into a round hole. Persistsing doing so, turning the block from all sides. Is unable to evercome a slight obstacle. She tries for instance, to insert a half moon shaped block into the right hole, but does it upside down. Instead of turning it over, she gives up attempting altogether. It is very charakteristic, that after trying the test many times, on different days, she is unable to succeed at the first trial. Even when the process is demontrated to her, she is unable to grasp the idea.

4. An eight year old, brother of former patient. B. S. age 5.5. In school, attentive, making a slow but steady progress. He fails in the first trial, tries one impossible move (insertion of a star into a cross hole). He succeeds on the second trial whith the aid of close comparisons. On the third trial, he has entire success. Several days later he is able to perform the test slowly and correctly.

5. A 6 year old, younger brother von Nr. 3 and 4. B. S.

mental age 5.5. Makes good progress in school. Performs the test correctly on the first trial in 36 seconds[1]).

These examples are very instructive. A normal child as young as three years, works intelligently and succeeds in his attempts. An entirely demented patient who hardly grades four years, is able to proceed intelligently though slowly. On the other hand a girl of 15, whose whole life points towards a deep congenital defect, is utterly unable to perform the test. She does not learn by experience, and her way of proceeding is characteristic of her school record. Her brother, who grades a little higher is able to succeed with difficulties; the younger brother, but slightly retarded succeeds well. In spite of the fact, that all these children show about a uniform mental age, the grade of their mentality is very different. This test corresponds in its results, to their school standard, while according to the *Binet* scale, one would expect them to be of similar ability. From this test, I anticipated the results of their progress in school, which anticipations were justified. On the other hand, a man of the mental age of four, is slow but succesful, which makes the assumption justifiable, that he is demented, having once however possessed a mentality of a much higher order than that shown by the *Binet Simon* test. He therefore would not be classified as an imbecile bordering on idiocy.

We thus see, that the Board test is very helpful in differentiating the grade of feeblemindedness, where the B. S. test would fail, and also in cases where the dementia is very marked, it is of great value in differentiating the

[1]) It is perhaps interesting to notice the peculiar improve ment of native ability with the order of births in this family. The brother who is one year younger than Nr. 5, is at home, not epileptic and is reputed to be of especially bright intellect. He even graded somewhat over his age in the Binet Simon test made on him by our Field Worker. The youngest child however, although only two years old has epileptic seizures.

latter diagnosis from a congenital low grade imbecility or idiocy.

The two next tests, which we began to apply in our examinations of patients, were the construction puzzles A. and B., as described by Healy in his Pamphlet recently issued. I must refer the reader to it, as the idea of the test is only entirely comprehensable with the help of a photograph of the puzzle boxed. Healy outlines the idea of the test as follows: "this test brings out perception of relationships of form and also the individual's method of mental procedure for the given task- particulary his ability to profit by the experience of repeated trials, in contra-distinction to the peculiar repetition of impossibilities characteristic of the subnormal and feebleminded. groups." Test A., is readily and serviceably made of scroll-saw wood. The inside measurements of the empty rectangle are 4 by 3 inches. The subject is offered the test, with the seperate pieces irregularly disposed and is told that the space can be exactly filled up, if they are put in correctly . . . In the test B. it is absolutely necessary that the parts be exactly made so that they are interchangeable throughout . . . The test is presented to the subject, with the pieces well mixed up. He is told that if the pieces are put in correctly they will exactly fill all the spaces, and he is to see how quickly he can put them in their proper places. In this test, again, it seems to us that a trial and success method, can not be regarded as at all derogatory to native ability, but it does seem clear that in such a procedure, the constantly getting of one's self back into old impossible situations is, on the contrary, evidence of poor ability. The shrewdest method pursued is to eliminate the small pieces which can only fill up certain definite spaces. Some of our subjects, deliberately do this. . . . Time, again, in this test is hardly to be considered so important as estimation of the method pursued. . . . We find that most of our 12 years old children do puzzle A in time ranging from 12 seconds to 2 mi-

nutes. . . . Most of our 12 year old children, are successful in puzzle B in from one to three minutes."[1])

These tests have been tried on about 50 patients and belong to the routine mental examination of all new admissions, as they proved to be of extreme value in the differentiating of native ability from deterioration. The Puzzle B is not an easy one, but that it is not too difficult is proven by the fact, that the three years old boy previously referred to succeded without help after some 6 minutes of experimenting. It was very interesting to watch this child at work. He was not yet able to use the rational method of elimination or reasoning that for instance, the square and block is not meant for the round and shaped hole; he learned however by experience and did not repeat this move again. At no time did he try the inserting of a square shaped block into a round shaped hole, a move again and again repeatedly tried by imbeciles, children, and adults. Another advantage of these tests is their game like appearance; one can gain the attention of abnormal children, who are otherwise so distractable that a test composed of questions would not meet with success. Sometimes an abnormal individual will fail to complete this test; one must however encourage him and occasionally it is well to demonstrate to him the whole method of procedure. Imbeciles will fail in spite of a demonstration.

It will be of interest to try this test on a few hundred normal children using the scoring devised by Dr. *Mary S.*[2]) Heyes and also apply it to our patients. It is possible that we shall be able to find out, wether there is a certain standard of procedure in work at certain ages. The results will be easily obtainable and comparable for the reason, that success is almost entirely independant from schooling, which might be of help as a matter of economy

[1]) Ibid page 14 to 17 incl.
[2]) Ibid page 17.

of time due to routine practice, but would never be of help in concealing obvious defects. From the Form Board test to Puzzle B, all of which are constructed an the same principle, there is a grading of difficulties and whereas a middle grade imbecile could succeed finally with the first two, he would be sure to fail in the third test. Those patients however whose mental ability had deteriorated from epilepsy, even though their mental age was below the norm, were succesful with the test and their procedure was intelligent. In our further investigations we shall apply most of the tests mentioned by *Healy* in his pamphlet, as we are fully in sympathy with the idea and purpose of these tests which are to eliminate the matter of training and education to the smallest degree thus making them applicable to educated and uneducated people alike, as well as to the deaf and dumb.[1])

All the tests hitherto discussed, were tests, wich either like the *Binet* test disregard the analysis of intelligence into specialized abilities or where the analysis had to be made by the examiner from complicated psychological data. *Bechterew* and *Wladyczko* suggested a series of tests which are intended to reveal special mental traits and their abnormalities. *Bechterew* is an advocate of, as he calls it, the objective method of investigating the psychic or rather neuropsychic sphere. His idea, is to introduce a number of tests, which being easily adapted both to normal and abnormal individuals have quantitative objective measurements.[2])

The new tests referred to in his article are methods

[1]) I succeded well with two deaf and dumb patients who are in our institition.

[2]) *Bechterew,* Die objektive Untersuchung von Geisteskranken, in Zeitschrift für Psychotherapie und medizinische Psychologie (also in Traite Internationale de Psychologie Pathologique).

The description of the tests which we used with illustrations are in *Bechterews* and *Wladyczko's* article "Beiträge zur Methodik der objektiven Untersuchung von Geisteskranken", Zeitschrift für Psychotherapie, 1911, Vol. 3, Page 87 to 109.

relating to the quantitative determination of external objects thus testing the ability for concentration, which is essential for success in the given test; also the investigation of the capacity for observation, the ability to note differences of detail, the ability for reproduction, which is in this incident, the recognising of familiar objects from pictured illustrations and finally the ability for visual synthesis.

In order to test the first ability, a test of 6 pictures was arranged representing colorless circles and numbering 9, 16, 22, 32, 45 and 63 circles consecutively in each picture. The object of the test is to make a mental count of the circles. In this case the time required for accomplishing the test is of as much value as the error. A similar test is one in which pictured objects are substituted for the circles. This test however seems to me to be somewhat dependant upon previous training and should therefore only be used after the determination of the native ability, as it is more or less a test for determining the deterioration. Whe should not expect an imbecile to meet with any success, for as a rule, the latter is unable to acquire an ability for counting over certain limits which knowledge he can only mechanically utilise.

The test for observation and comparison is much better suited for our purposes. A simple picture with a few details is shown and exposed to the subject for a certain length of time. A series of pictures follow each composed of a slight added detail. In a similar manner, for the reversed procedure, another series of pictures is used with a decreasing number of details. It appears that in this latter procedure, from a psychological point of view, it is much more difficult to perceive the disappearance of small details.

The test for visual imagination consists of a series of pictures with the outlines of a familiar object. The distinctness of outline is gradually increased from a vague hazy representation to a definite outline in the 6th picture.

The last test is for the revealing of the ability for visual

synthesis, and it consists of an illustration of the scattered parts of an object, a lamp for instance, comprising the chimney, wick, stand etc. This picture is shown to the subject who is expected to state the name of the object which the combined details would represent.

Regarding the first test, the mental counting of objects, *Bechterew* found that the time of counting was not only prolonged in all forms of mental disturbances, but that some patients made errors even in the first picture. (Especially se in cases of Dementia Praecox and General Paralysis.) No data regarding epileptics were given by the author nor have I made any investigation regarding this point.[1])

The test for observation and construction was tried on about 40 of our patients. The very demented patients were only able to notice a difference in pictures Nr. 1, and 5, or 6. Low grade imbeciles were entirely unable to observe any difference whatsoever. A few epileptic dements tested by *Bechterew* showed a prolonged time of consideration, a fact also observed by me; his observations otherwise were also in agreement with my own. In *Bechterews* differential table, a worse record was shown only among General Paralytics. This experiment again confirms the results of clinical observation.

Both of the above named tests will be rather of more service in differentiating dementia epileptica from other forms of deterioration. The tests for visual reconstruction, imagination and synthesis however I found of great value for the determination of congenital defects. Many of the patients, who tested in the *Binet Simon* test much below their norm succeeded as well or better than the average normal individual. Some of the patients examined showed

[1]) We have been unable as yet to obtain original pictures but hope to get them soon from *Bechterews* clinic in St. Petersburg. We therefore used the illustrations printed in the above named article. As the illustrations to the first test were too small and indistinct we could not apply this test in our experiments.

a low record in the test of imagination for instance, at the same time showing an unusually good one in the test for visual reconstruction. On the whole, all these tests showed very a promising aspect for the differential diagnosis of epileptic deterioration from congenital mental inferiority, as well as from other forms of deterioration.

I must refer the reader for details of curves to *Bechterews* original article; to illustrate my process of reasoning and the special points of interest I shall cite a few cases which I tested with all the methods mentioned in the article.

6. Boy of 13, admitted to the Village early in 1910. On admission made almost a normal impression; was until recently one of the brightest children. Made good progress in school and in the band. He tested age 11 with the Binet Simon test, when 12 years old, thus he was hardly one year behind and could only be classified as backward, as he failed in both of the memory tests for the age of 12 (test 50 and 52 and in the tests 47 and 48). On the other hand he succeeded in the test N. 54. The Form Board test was performed without error in 21″. In the summer of 1911 he went home for a vacation. Since his return to the Village he has shown an enormous change in mentality and increased number of seizures. In the short time of a few months he demented an almost normal boy to an absolute dement. He apparently forgot whatever he had learned at school or in the band. He now sits most of the day in a chair, with a grinning expression on his face; does not even remember the names of his former acquaintances.

The Binet Simon test grades him at the present time as hardly 7, and this was only to tests of counting or other mechanical ones as he failed in other tests underthis age. A questions were answered slowly. The time of associations was between 4 to 9 seconds. He is able to count correctly but can not add over 18 counts of two digits in a minute, although formerly a very good arithmetician. The Form Board test is done correctly in 1′ 34″. Healys Puzzle A is done in 9 minutes with 21 moves. He had to be urged to proceed, did not take any special interest in the experiment, but not once did he attempt an impossible move.

He could not finish the Puzzle B, but was on the right way

and no doubt would have met with success with some amount of attention and interest.

In Bechterews test for comparison he detected the increase of details in the 4th picture; in the decreasing series, in the 3d picture. A picture of a cow he recognised as such in the 2nd picture, that of a wagon in the first one. The time needed for recognition was between 10 to 18 seconds. He recognises tho outlines of a lamp in the 3d, the outlines of a rooster in the 4th, the outlines of a knife in the second and finally the scattered parts as belonging to a lamp after 34″.

Even without previous knowledge of the case history, we should be able, from the tests at hand, to say that this boy must have once been a fairly smart individual, but that he is greatly deteriorated and that a diagnosis of epileptic imbecility would be unjust and misleading besides necessitating a change of the *Binet Simon* diagnosis, made in the space of a years time, from retarded to low grade imbecile.

7. A boy of 15, admitted December 1911. Father epileptic. Patient had two seizures before the age of 4, then began to have convulsions about the age of. 7. He went as far as the third grade, but his poor memory prevented him from making progress. Has remained at home since then.

According to the Binet Simon grading he tests about 9 and $^1/_2$. He fails in all tests relating to memory, assigned for children over the age of 5. The common sense tests are answered however very satisfactorily and the test for the age of 10 is passed correctly. This examination shows, that his memory for remote and recent events is very poor, in like manner his impressibility, and that therefore, it is no wonder that he makes no progress in school. He is not regarded by his teachers as incapable; they assign his failure to poor memory. The Form Board test was made correctly in 17 seconds. In Healy's puzzle test, prompt success was met with after one demonstration. No impossible moves were attempted. The tests for observation showed very poor results as he was unable to detect any difference in the pictures with increasing and decreasing details. His record for reconstruction was much better, recognising the back part of a cow as such in the second serial picture, and even recognising the anatomy of an animal in the first. The wagon was recognised in the first picture. His imagination was shown to be

excellent. The outlines of a lamp knife and a very difficult one of a rooster were recognised in the first picture; a better record than any normal person whom I tested.

My inference is that apparently defective and probably slightly feebleminded, he represents a condition, which is the result of epileptic deterioration, congenital feeblemindedness and lack of proper education. He shows traits which are not to be found in a congenital imbecile, to whom he bears a strong superficial resemblance.

8. A boy of 12. Father, dead, was in fair circumstances, and apparently normal, but with cases of epilepsy in his fraternity. Mother, a very pecular woman, neurotic, hypersexual, quarrelsome and insisting on her rights. She owns a store, but in spite of good opportunities, her business since her husbands death is unsuccesful, as all her customers are afraid of her. A typical querulant, it is an apparent pleasure for her to sue people, and at the present time she has two or three suits on her hands, mostly for imaginay damages. Her characters is well known, and lawyers af good standard refuse to take up her cases.

The boy has ad seizures since his earliest childhood. Was admitted to the Village in 1912. He was considered bright but rather lazy; had a schooling adequate to his age. Was described as having an uncontrolable temper. Since admission, makes good progress in school and the band, has occasional fits of temper; if angered very vulgar and quarrelsome, and is apt to fight at the slightest provocation. Shows strong likes and dislikes for people, insists upon his rights, and considers himself a judge, as to whether he is treated justly or not by others, especially his teachers. His unwillingness to study, he explains on the ground of personal resentment to some of his teachers, and his belief in their inability to teach in the public schools.

He is the only child, which answers all questions in the Binet Simon test correctly. Some of his answers show an excellent ability for logical definition, which is rarely found even among normal adults. Bechterews tests were answered better than the average normal person would do. Puzzle A test was apparently a difficult problem for him, as is took him over four minutes. Once however used to this kind of work, he performed the puzzle B test with only two moves

above the minimum, in a surprisingly short time of 21″, considering that the test is really not an easy one.

This boy is a interesting study. In the first place, from a hereditary point of view he is an epileptic due to an epileptic strain in the paternal ancestry; he is also however a psychopathic personality due to inheritance from his mothers side. All his mothers mental peculiarities are revived and easily traceable in his own mental makeup. I therefore claim that he is an epileptic whose intellect is as yet unattacked by the disease[1]), and that his character is independant from the disease and easily traced to his bad heredity on the mothers side.

From a psychological point of view, we come across a case which would probably be a puzzle to a teacher. Rather exceptionally smart, he still would not make the expected progress in school, as his emotional and volitional sphere is decidedly pathological, and as the whole he is defective.

From a purely psychological point of view the length of time required for solving *Healys* puzzle A is of interest, proving how little one can depend on the time expended, for judging a person's ability.

This case is well adapted for the introduction of a controversy which is at the present time actual. Some epileptologists, basing their opinion on research work done on heredity, claim that the feeblemindedness in epileptics, even if congenital is not feeblemimdedness in the true sense of the word but a symptom of the disease, the causation of convulsive symptoms ad the mental inferiority being identical. This view is strongly opposed by experts in feeblemindedness, who take the point of view, that

[1]) I wish to emphasise the fact that the disease did not attack the intellect as I believe it has an influence on his emotional life developing and exaggerating the already present psychopathic traits of character. It is perhaps interesting to know that the boy was never treated with bromides.

feeblemindedness is an independant factor. From a psychopathological point of view I am a disinterested party in this controversy. I wish to again take up the example of the congenitally blind person, and those who become blind before the age of 4. Their mental status is equal in any case, whether the blindness is due to accident, syphilis, gonorrhoea or atrophy. What is the causation of feeblemindedness, in most cases we are unable to say. It is quite probable, that in many cases the epilepsy is the etiological factor. It does not change the psychological aspect. Our standpoint is that there is an essential difference between those persons who are born defective, or become so in their earliest childhood and those deteriorated in later life. A great number of epileptics are born defective or become so in the first four years of their life. Between these and other cases of congenital defectives on the one side, and demented epileptics on the other side, there is a visable and by special tests demonstrable disparity.

Some observers[1]) claim that a feeblemindedness due to epilepsy, even though acquired at the earliest age, will show points of dissimilarity from imbecility which has been otherwise acquired. This is quite possible, but it needs however further study. Psychology, united with the study of heredity, could also make a special study of those, who being descended from epileptic parents are feebleminded but without epileptic seizures whatever. Should it be true, that epilepsy is the causation and that imbecility with epileptic causation has a different symptomatology, they would be expected to show characteristics of epileptic mentality.[2]) Thus we can readily perceive how

[1]) Echeverria for instance, that advocate of the exogenic causation of epilepsy.

[2]) The term of epileptic imbecility could be used as a diagnostic terminology for these patients. It would emphasise in the term "Imbecility" the congenital character of their mental defect, and the adjective "epileptic" would indicate the etiology of the defect. For

psychology, remaining on its own ground, that is, the investigation and analysis of the mental status presens of patients, and leaving the explanation of causes to other branches of science, con be inspired by the latter to new and unexplored fields and be of help to them in exchange.

Before closing the discussion on this point, I should like to add the remark, that if psychopathology and psychiatry in general is to be helped by the study of heredity, it should have a clear field for investigation without a pledge to any special theory. I do not doubt, that in many cases of epileptics all symptoms present are due to the disease, but that it will soon be the universal conviction that we are justified in making a diagnosis of epilepsy even though no other epileptic symptoms were present, than let us say a only specific feeblemindedness. But, specific must it be. At one time, the Italian school went so far as to call every outburst of temper and violence, epilepsy. We have justly freed ourselves from this conception. The guide for our present classification will be psychopathology and the heredity charts. With the help of both we shall be able to classify the cases justly. I refer the reader for instance to the above cited case Nr. 8. Psychological analysis disclosed in this boy certain mental characterists. He is an epileptic. We state that these characteristics are independant from the disease. This conclusion is justified by the fact, that while we can trace epilepsy on the paternal side, the patients mental make up is directly derived from his mother, giving a disastrous combination in an individual. This inference is perfectly in accordance even with the Mendelian law, as epilepsy and neuropathic tendencies may be and surely are two independant units and thus can be independantly transmitted. Thus the science of Eugenics can also be benefited by psychology.

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all other cases the term dementia or for the indication of milder forms, the term deterioration is the only proper and descriptive term for diagnosis.

There are many other problems of great interest. Our laboratory in Skillmann, being the first one erected in this country or anywhere for the purpose of special study of the psychopathology of Epilepsy, is of very recent date. We have thus far have made good progress, and hope, that soon, we shall be able to contribute valuable data to the understanding of clinical symptomatology, the effect of convulsions, the influence of bromides; the simple but ingenious reckoning experiment introduced by Kraepelin, has not failed to disclose valuable points toward the understanding of the pathological reaction of the epileptic working mind[1]).

I hope that this brief paper justifies the expectations in psychopathology, of those who have put trust in such a research work, and has helped to disperse the scepticism of those, who have often asked: what is the use of psychology in medicine? Psychology in its applied field of psychopathology, has not been able to accomplish much in this short space of time. However, when we look back over the progress of general pathology and the "facit" of this science, which for years has been at work in costly laboratories all over the world, I feel that psychology has no need of looking askance at its sister branch psychopathology.

Conclusions.

1. The extraordinary development of psychology and its methods in recents years, has brought the psychological science into prominence and has directed its appliance to practical purposes. Medicine has come into close contact with psychology and a new science, psycho pathology has sprung into existence.
2. Psychology is one of the most difficult fields of study and therefore the research work if valuable must be

[1]) Compare *Katzen-Ellenbogen* "The mental efficiency in Epileptics", Epilepsia, Vol. III.

preceded by the studious preparation of years in laboratories of experimental psychology.

3. In order to qualify as a research worker of psychopathology, a thorough knowledge of psychology on the one side, and a most intimate familiarity with the projections of the morbid mind on the other side, which can only be acquired by clinical observation and some years of experience in institutions for the insane and defectives, is absolutely essential.
4. Neither psychology itself nor medical experience alone suffices to accomplish scientifically valuable work.
5. The popularisation of results which are as yet not beyond a stage of experimentation, is harmful to the science which originated such results, at it leads to miscredit and failures.
6. Psychology has often committed such mistakes and work which should only be confined to laboratories and conducted by specialists, is being applied by those who are not able to grasp the significance of certain signs, thus causing them to draw hasty and faulty conclusions. They wreck their own field of work and cause unfavorable comments on psychology because failure is unavoidable and a reaction against such procedings of necessity follows.
7. Pedagogic is the science which is most at fault in this regard in its relations to psychology and to itself.
8. The greatest abuse and misuse is practised with the *Binet Simon* test.
9. In its present form although not scientifically free from objection the *Binet Simon* test may render valuable service for practical pedagogics making feasible a quick orientation about an abnormal pupils mentality.
10. It is not possible to introduce tests and methods of mental examination into psychopathology without modification. The *Binet Simon* test in its present form therefore cannot be used for the testing of mental defectives accurately.

11. Epilepsy is a disease which is not only commonly associated with a mental defect, but it leads more or less to a pronounced deterioration of the mind.
12. It is, therefore, useless to apply the *Binet Simon* test mechanically to epileptics, in its present form and to speculate about the patients corresponding age.
13. The statistical investigation of the *Binet Simon* test has shown conclusively that the authors did not succeed in eliminating the necessity of a school training, thus failing to form a test for the testing of pure native ability.
14. The overwhelming preponderence of failures in epileptics is due to the deterioration of memory, psychomotor retardation, lack of training due to the absence of school education and lack of the common experiences of life caused by the necessity of early confinement.
15. The comparison of the relative percentage of failures in children and adults was in this regard most convincing. Children showed a higher percentage of failures in questions relating to knowledge which must necessarily be acquired by contact in a community, for instance elementary arithmetic and the knowledge of money. Adults failed more frequently than children in those questions requiring a display of concentration and memory, thus proving the progressive deterioration of these mental faculties.
16. The surprisingly frequent failures of epileptics in the estimation of small differences of weight is surely not due to defects of intelligence, but finds its explanation in an abnormality of inner tactile sensation of the muscles and joints. The progressiveness of this abnormality is demonstrated by the higher percentage of failure in adults.
17. All findings are in accordance with the clinical observations of epileptics.
18. The experience gained with the *Binet Simon* test on epileptics can be exploited in a pathopsychological way,

by helping to eliminate objectionable questions from the tests.

19. For the test 41 and 36, the proposed modification could easily substitute the mechanical recitation.
20. The *Binet Simon* test is the most ingenious first step towards the determination of individual native ability, and it is of the most vital interest for applied psychology. None but skillfull psychologistes should apply these tests to normal individuals, and none but skillfull psychopathologists to abnormal individuals.
21. All results justifying the suspicions of an abnormal mentality, should be referred to a psychopathologist who alone would be justified to make a differential diagnosis between a congenital mental defect and an incipient psychosis, in other words, discover the mental cause for the existing retardation.
22. The psychopathology of epilepsy is a different problem than that of imbecility.
23. Imbecility is a congenital defect, for this reason the terminology "Epileptic Imbecility" is not the proper one.
24. The proper terminology of diagnostication for epileptics would be epilepsy with a congenital mental deficiency of imbecility, idiocy or moronity. In other cases, epileptic deterioration or dementia superposed in epileptics with etc.
25. Such a method of diagnosis emphasises the graver factor of the affliction and would meet with the approval of those whose theory claims that the congenital defect in epileptics is in itself one of the symptoms of latent epilepsy due to the same cause and different from other forms of congenital mental inferiorities.
26. The writer is not prepared at the present moment to take part in the controversy regarding the etiology of congenital deficiency in epileptics. Psychological researches must be directed in order to analyse the disparity of epileptic mental deficiency from others.

27. The Form Board test, Healy's puzzle test and the Bechterew test are most succesful in the elimination of the element of training, thus being of value for the analysis of epileptic mentality, regarding the amount of mental native ability and the damage due to deterioration. The preliminary investigation was very promising in its results. In all tests, not the duration, but the quality of work signifies the intelligence.
28. Psychopathology proves to be of the greatest value in the study of epilepsy together with careful clinical observations, general pathology and pathological physiology.

BINET-SIMON TESTS FOR INTELLECTUAL DEVELOPMENT.

	Total Number	Children	Adults
	Age 3.	% of failure	
1. Points to eyes, nose, mouth etc.	4.5	5.0	4.3
2. Repeats six syllables	11.0	13.1	10.3
3. Repeats two numerals	6.5	13.1	4.3
4. Enumerates familiar objects in picture	6.8	8.0	6.3
5. Gives family name	4.3	8.0	3.0
	Age 4.		
6. Knows own sex	6.3	9.0	5.3
7. Recognises key, penny, knife etc.	7.3	10.3	6.3
8. Repeats three numerals in order	10.8	17.1	8.7
9. Recognises a longer line by a cm	11.0	18.1	8.7
	Age 5.		
10. Discriminates weights of 3 and 12, 6 and 15 g	12.5	21.1	9.7
11. Draws square from a copy . .	15.8	24.1	13.0
12. Rearranges into a rectangular part triangular pieces	27.5	31.1	26.3
13. Counts four pennies	9.8	17.1	7.3

	Total Number	Children	Adults
Age 6.			
14. Shows left ear, right hand . .	12.6	24.4	9.0
15. Repeats sixteen syllables . . .	*65.3*	56.5	*68.0*
16. Distinguishes in pictures, pretty from ugly faces	20.0	29.8	17.0
17. Defines in terms of use the words, table, chair, horse etc.	19.2	28.8	15.7
18. Performs three commisions given simultaneously	29.1	35.8	27.0
19. Knows on age	29.8	37.8	27.3
20. Knows whether it is forenoon or afternoon	16.4	28.8	12.7
Age 7.			
21. Notes from portraits omissions of eyes, nose etc.	25.4	37.8	21.3
22. States number of fingers on right, left and both hands	16.9	28.8	13.3
23. Copies written sentence . . .	—	Omitted	—
24. Draws diamond shaped figure from copy	30.6	38.8	27.3
25. Repeats five numerals	43.3	49.4	41.3
26. Describes actions and scenes in test five	37.9	37.8	38.0
27. Counts aloud thirteen pennies in row	15.9	29.8	11.7
28. Recognises penny, nickel, dime, and quarter	17.4	28.8	14.0
Age 8.			
29. Reproduces correctly two facts after once reading a story . .	30.3	35.8	28.7
30. Counts three one, and three two cent stamps	34.9	41.3	33.0
31. Names red, green, blue, yellow	19.7	27.1	17.3
32. Counts from twenty backwards	37.2	42.4	35.7

	Total Number	Children	Adults
33. Writes easy dictation	—	Omitted	—
34. State differences between paper and cloth, wood and glass etc.	30.1	38.9	28.7
Age 9.			
35. Gives the date of the day . .	35.4	37.9	34.7
36. Names the days of the week in order	19.6	28.7	17.0
37. Gives correct change from a quarter, if nine cents expendend. .	*53.8*	54.0	53.7
38. Defines by description or classification the words of tests 17 .	*70.0*	62.0	*72.0*
39. Reproduces six facts, as in test 29	*6.84*	64.3	*69.7*
40. Arranges in order of weight boxes weighing 6, 9, 12, 15 and 18 g	*73.8*	55.1	*79.3*
Age 10.			
41. Names the months in order . .	32.5	42.8	29.7
42. Recognises money as in test 28, plus half a $, 2, 5, and 10 $ bills	21.8	28.9	20.0
43. Uses three given words in a tence	*56.5*	48.8	*58.7*
44. Replies to problem question: "what should you do?"[1]) . . .	*52.1*	52.4	52.0

[1]) 1. When you miss a train?
2. When struck by some one on intentionally?
3. When you break an object belonging to some one else?
4. When you are late for school?
5. Before you take part in an important affair?
6. When asked for an oppinion of some one you do not know well?
7. Why should you forgive a wrong act committed in anger, more quickly than one not committed when angry?
8. Why should you judge a person by what he does rather than by what he says?

	Total Number	Children	Adults
Age 11.			
45. Detects nonsense in three out of five statements	*69.5*	60.1	*72.0*
46. Uses three given words in one sentence	*67.6*	53.0	*71.7*
47. Utters at least 60 words in three minutes, exclusive of duplicates	*81.2*	62.6	*86.3*
48. Defines Charity, Justice, Kindness	*71.7*	66.2	*73.3*
49. Rearranges shuffled words in sentences	*80.9*	63.8	*85.7*
Age 12.			
50. Repeats seven numerals in order	*81.6*	68.8	*85.0*
51. Names three words that rhyme, f. i. day, spring, mill	*62.4*	51.6	*65.0*
52. Repeats twenty six syllables .	*82.4*	67.1	*86.0*
53. Infers facts from given circumstances which indicate the facts	*80.3*	65.6	*83.7*
Age 13.	% of success		
54. Images and draws triangle cut from sight of quarto folded paper	26.3	17.6	28.0
55. Images and draws new form produced by joining transpose pieces of diagonally divided rectangular card	11.8	5.2	13.0
56. Distinguishes abstract terms of similar sounds or meaning (pleasure and happiness, event and advent, evolution and revolution, pride and pretention, poverty and misery)	3.9	1.7	4.3

Druck von Radelli & Hille in Leipzig.