and even hallucinations that are really unimportant and do not betoken insanity.

3. The Manageable Causes of Insanity.

4. Alcoholism and Insanity.

5. The Care and Treatment of an Insane Patient in a State Hospital from Admission to Discharge. Safeguards against illegal commitment. Supervision of hospitals by state boards of insanity. The large number of discharges (bearing on the popular belief that many are sent to insane asylums, but few come out). Diet of the patients. Systematic training of the nurses. Oversight of nurses and precautions against neglect, abuse and escape of patients. Medical attention to patients and study of the disease in hospitals. Harm done by the usual newspaper accounts of insane patients and hospital management. The contrast between care at home and hospital care. Condition of patients when brought to hospitals and best means of transportation of difficult cases, etc.

With such a list of subjects to draw from, it would seem easy to inaugurate a suitable course of lectures which could hardly fail, if under the right auspices, to enlighten and ease the public mind.

But the manner in which the subject shall be presented to the audience is quite as important as the subject itself. This has been effectively urged in editorials on the subject of popular medical lectures in the BOSTON MEDICAL AND SURGICAL JOURNAL. A tendency has been noticeable in the lectures thus far given to paint in too somber colors and to make too much of the dangers of incurring this and that dread disease for lack of proper precaution, and for want of care in analyzing statistics, magnifying the prevalence or increase of the disorders. Now while the possibilities of harm should not be understated, neither should they be exaggerated to the extent of creating unnecessary and unhealthy alarm. The utmost care should be exercised to prevent misconceptions on the part of our hearers, theories should be discarded and only actual ascertained facts dealt with which have a definite, practical bearing. The difficulties are in one way far greater than in lecturing to a class of medical students and the temptation greater to speak with less accuracy to a popular audience. It goes without saying that what is said needs to be simply, clearly and graphically put. For the popular medical lecture to be most effective requires in a medical man, according to President Eliot (who originated the course at the Harvard Medical School), the power of interesting description with telling exposition and moving exhortation. To apply these several cautions to psychiatry: the terrors of insanity and its treatment have held the boards far too long, and the more we are able to say with truth that will tend to lessen its horrors in the minds of the community, the better shall we serve the purposes of true psychiatry; and the more widely and generally the nature of our work is known, the more the dignity and importance of our calling is enhanced.

HEREDITY IN DISEASES OF THE NERVOUS SYSTEM WITH ESPECIAL REFERENCE TO HEREDITY IN EPILEPSY.*

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A BELIEF in the heredity of many diseases, especially those of the nervous system, is one of the accepted tenets of medicine,—a tenet to which every one is tempted to subscribe, whether he notes the invasion of Friedreich's ataxia, Huntington's chorea, or muscular dystrophy, in member after member of some doomed family, or studies the social degradation of the Jukes¹ family, or the degeneration and extinction of the Spanish Hapsburgs.² It is a belief, too, which has been held even more strongly by the laity, from the days of the Hebrew prophets until the present time. With the development of the doctrine of the neuropathic or psychopathic predisposition by Griesinger the possibilities of morbid heredity in diseases of the nervous system became multiplied, and the studies of the French writers in particular, Dejerine,³ Féré,⁴ Le Gendre⁵ and Paul Raymond,⁶ have sought to establish heredity as the dominating etiological factor in the vast majority of nervous and mental diseases.

The whole doctrine of heredity, however, has been modified by the theories of Weismann,⁷ which have been accepted by many biologists and a number of physicians. According to Weismann, as is well known, acquired characteristics are not transmitted to the descendant, but any influence which, developing in the lifetime of the individual from some external cause, injuriously attacks the germ plasma, may give rise to some defect or modification in the descendant. Weismann holds that there is a continuous transmission of the germ plasma which contains all the elements of the future being. The ovum, in dividing, transmits an integral part of itself to the new cells, so that the ovum of one generation comes directly from the ovum of the preceding generation, and the personal influence of the generator will have no effect upon the quality of the germ plasma, unless the germ plasma be injured by some external influence, such as trauma or poison. Binswanger⁸ affirms that we can speak positively of an inherited morbid predisposition coming from the ancestry, only when germ plasma, pathologically altered in one or both individuals at the moment of procreation, has served to form the new individual. This pathological change in the germ plasma in the generators may be directly inherited from their parents or acquired during their own lives from various injurious causes, such as chronic intoxication (alcohol, morphine, lead), infection (syphilis, tuberculosis), constitutional disease (anemia, leukemia, chlorosis, diabetes, gout, arthritis deformans), or local diseases of the generative tract. This pathological alteration of the plasma leads to a general constitutional weakness of the individual, in whom later

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certain special factors may determine that the nervous system shall first or chiefly give evidence of that weakness.

Although Weismann's theories are not yet universally accepted, Le Gendre and Hähnle,⁹ among others admitting that acquired characteristics may be inherited, and although Weismann himself has modified them somewhat, so as to admit certain possibilities of the transmission of seemingly acquired characteristics, or that acquired traits due to an external cause may act on a predisposition, the doctrine, as stated above, is of much importance in our conception of the part played by heredity in the etiology of disease.

Another feature in the manifestation of heredity has been pointed out by Galton,¹⁰ and has been further emphasized by the teachings of Bouchard.¹¹ In various studies Galton has shown that, although certain characteristics are so common in certain families as to suggest hereditary influence, these peculiarities have a tendency to disappear in a few generations. He has formulated the law, too, that each parent contributes about one half to the make-up of the individual, each grandparent about one fourth, and each great-grandparent an eighth or less. Thus the sound parent may do something to correct the taint coming from the diseased parent.

Galton's ratios, however, are not always constant. Occasionally some ancestor seems to show a remarkable power of impressing his characteristics upon successive generations, as Ernest the Iron of Carinthia impressed the Hapsburg lip and the prognathous jaw upon several hundred descendants through eighteen generations,¹² or as Justin Morgan impressed the peculiar characteristics of the Morgan horse upon an uncounted progeny. The comparative study¹³ of the Jukes and the Edwards family points to a similar power. Although some of this remarkable transmission may be accounted for by in-and-in breeding, yet there is much left unaccounted for which opposes Galton's ratios.

The theories of Mendel,¹⁴ long forgotten and recently revived by De Vries,¹⁵ Bateson¹⁶ and others, explain this condition by the dominance of certain characters in heterozygous or impure individuals. According to Mendel, when two individuals possessing opposite characteristics come together, we may regard the germ cells as units. Each cell may contain one or the other of the two characteristics, but not both. These germ cells come together in pairs to constitute the body cells, and these body cells may be pure or homozygous, when both germ cells contain the same characteristic; or impure, or heterozygous, when each germ cell contains a different characteristic. If a given characteristic be dominant in the impure cell, this cell, although composed of two different germs, may resemble the pure cell composed of two similar germ cells. Mendel's theory, moreover, teaches that these impure individuals may give rise to pure types having the dominant characteristic, and that these pure types subsequently give rise to other pure types without variation, — in other words, that the

special morbid characteristic of a race has a tendency to become fixed or to disappear, and in either event be reproduced subsequently without a change. According to his teachings, if an individual escapes the morbid defect of his family, he is likely to produce offspring without this defect, and thus Mendel finds little room for atavism.

The application of Mendel's theory to human heredity has hardly begun, and the complexity of the problem is so great that it is a question whether its practical bearings can be determined for many years to come. At all events, as Bateson has shown, the facts of heredity must be collected with far greater accuracy than has thus far been done. Bateson points out that data must be presented for each family separately, that the normal members must also be regarded, and that we must be assured that the cases are similar, that is, dependent on one pathological state. It requires but a glance at any collected body of statistics of heredity of nervous disease to show that these requisites have almost invariably been disregarded.

Recalling the various theories of Weismann and Mendel, if we turn to the recorded data of morbid heredity we find these data absolutely unsatisfactory, if not utterly worthless. The whole teaching as to the etiological importance of heredity rests upon a mass of inaccurate, undigested data which often have no bearing at all upon the real etiology of the disease under consideration.

In the ordinary discussions of nervous heredity we speak of similar and dissimilar heredity, — the latter manifesting itself as the neuro-psychopathic predisposition. Similar heredity is far less frequent than neuropathic heredity, and is seen most strikingly in certain rare diseases, — Friedreich's ataxia, Huntington's chorea, muscular dystrophy and the like. Occasionally it is noted in some of the more common diseases, especially in migraine, as is shown by a recent case under my observation, where migraine existed in eight out of sixteen members of a family in three generations. The fact that these rare diseases occur with remarkable frequency in the members of one family seems a striking demonstration of their hereditary character, especially when the occurrence of the disease is noted in generation after generation, with fatal regularity.

Nevertheless, this is not absolute proof that the disease is due to heredity. On the day that I write these words I have chanced to see two cases of Friedreich's ataxia. In one, a son of intelligent parents, no other case was known in the family. In the other no case was known in the antecedents, although two other cases had occurred in the same generation, in a sister and a cousin. The old theory of atavism, that a morbid tendency may be latent for several generations and then reappear, was formerly invoked to explain these cases where no traits of the disease could be found in parents, grandparents or collaterals of either generation, and this explanation could usually not be contradicted, because few of us know anything definite about our great-grandparents and remoter

ancestry. Even in the case of Queen Victoria nothing whatever is known of at least one of her great-grandparents.¹⁷ This explanation, always somewhat hypothetical, can hardly be maintained to-day in face of the teaching of Mendel, that the impure types become unvarying after a generation or two. It occasionally happens, moreover, that we have the opportunity to note one of these rare diseases, apparently of sporadic origin, where no previous case can be discovered in a family, and later we observe its reappearance in a subsequent generation. I had under my care, some twenty years ago, a boy with muscular dystrophy, where no case could be discovered in the antecedents and no definite cause could be made out. Years later, another case of the disease manifested itself in his sister's child.

As I have just said, the occurrence in one family of a dozen or fifteen cases of a disease so rare that only a few hundred are recorded in medical literature, would seem conclusive proof of a hereditary influence at the least, and so much may be admitted. When once the disease is established, it is easy to conceive that the pathological state of the germ plasma may persist in individual after individual among the descendants. Yet these things are little help in understanding the genesis of the affection in individuals where no previous case has occurred in the antecedents. In these cases, too, we find nothing more than ordinary occurrences which might conceivably injure the germ plasma, occurrences which affect the majority of the race at some period or other, trivial accidents, common infections and the like. Even if we assume that such occurrences lead to pathological changes in the germ plasma which are transmitted to the descendants, giving rise to some abiotrophy, why is it that this banal accident so affects the germ plasma as always to cause an abiotrophy of a special set of nerve fibers, — certain ascending tracts in the cord, certain cortical cells, or some of the fibres of certain special groups of muscles?

De Vries' theory that the normal way in which new varieties are produced is by sudden mutation is perhaps a more satisfactory method of explaining the sporadic manifestation of these rare affections. De Vries has found that new varieties appear suddenly and are often constant from the first. He furthermore admits the possibility of a reproduction of the same mutation at different times and in different localities. Yet even if we assume that what is demonstrated as occurring in plant life is equally applicable to man, we find certain effects in the incidence of these diseases which are still inexplicable, such as the predominant occurrence in one sex and transmission by the other, or the disappearance of the disease in certain branches, and its persistence in others, although the tainted stock be crossed with new blood to the same extent in either case.

Although the origin of these rare diseases is still obscure, nevertheless it is possible that they may arise by sudden mutation and persist in the affected family by true heredity, owing to the change set up by this mutation in the germ plasma.

It is incorrect, however, to regard heredity as the cause, and the tendency to recurrence of the disease in the affected family steadily diminishes by crossing with healthy individuals, as is shown by the teachings of Galton and Mendel. In this process of regeneration, however, although the affected stock may gradually be restored by cross-fertilization, a certain number of diseased individuals may be produced during one or more generations, until the morbid taint is obliterated, so that the old warning against breeding from such a stock is justified.

Turning now from these rare diseases in which the family occurrence is so great as to compel the belief that heredity plays a considerable part in their transmission, if not in their origin, we must next consider the part played by heredity in the production of the more common diseases of the nervous system. In these affections it is generally admitted that similar heredity is rare, and almost all writers agree in laying chief stress upon the neuropathic or psychopathic predisposition. In this predisposition are included all the different forms of insanity, all the functional and organic diseases of the nervous system, all peculiarities of mind and conduct, genius, the artistic temperament, criminality, alcoholism, pauperism, and even tuberculosis, cancer, diabetes and the different affections classed under arthritism.

The most available statistics for the study of the influence of this neuropathic heredity are those referring to the heredity of insanity; but these are useless for any study except that of the futility of human effort, since the vast amount of labor employed in their collection is wasted, on account of the habit of regarding insanity as one disease, and mixing the statistics of general paralysis, senile dementia, paranoia and all other forms of mental disease included in the various classifications together into one grand whole, and then drawing conclusions as to etiology, symptomatology, prognosis, prophylaxis and treatment, — a method as scientifically accurate as it would be to draw similar conclusions from a collection of cases of bronchitis, pleurisy, pneumonia, pulmonary tuberculosis, pulmonary cancer and embolic infarction of the lung, or, as Von Jauregg¹⁸ suggests, to draw similar conclusions in regard to skin diseases from lumping together scarlet fever, favus, eczema, herpes zoster and psoriasis. I have therefore studied the statistics of the heredity of epilepsy, often regarded as the most hereditary of all nervous diseases,¹⁹ in 320 patients of my own and in 1,200 patients at the Massachusetts Hospital for Epileptics, the data of which were put at my disposal by the kindness of Dr. Flood, and have compared them with the statistics cited by different authors. Even here the error which Bateson condemns, and all writers on insanity have fallen into, is not wholly avoided, since epilepsy must be regarded not as a single disease, but as a syndrome due to various affections of the brain. The error, however, is not quite so glaring as in the collections of statistics of "insanity."

The method which obtains in the collection of statistics as to neuropathic heredity in epilepsy and other nervous diseases seems to be as follows: a more or less careful inquiry is made as to the existence of any form of nervous or mental disease, and often the other diseases referred to above, not only in the direct ascendants, but also in the collaterals. Cousins are often included, as by Turner,²⁰ forgetful that we usually have there the introduction of a different stock. If any such disease is found, the case is regarded as manifesting neuropathic heredity, either similar or dissimilar; the total number of cases in the ascendants and collaterals is usually disregarded. If the heredity be dissimilar, as is usually the case, little or no attention is paid to the form or nature of the nervous affection occurring in the other members of the family. The percentage of cases showing neuropathic heredity thus ascertained is calculated. This percentage is apt to be larger than the percentage of any other one alleged cause, and the conclusion is drawn that neuropathic heredity is the chief determining factor in the production of epilepsy or any other form of nervous disease under consideration.

asylums of New York State vary from 14% to 76%. Hähle has collected equally discrepant statements concerning morbid heredity in special forms of mental disease, and Binswanger²² quotes like variations in the statistics as to the heredity of hysteria.

It is undoubtedly true that the more exhaustive the inquiry and the more intelligent the patient or his family, the more information will be elicited and the greater will be the percentage of morbid heredity discovered; but I must confess to a certain degree of doubt as to whether this is a sufficient explanation of the variation shown in these percentages. At the Massachusetts Hospital for Epileptics, for example, the inquiry I think has been more minute and careful than in my own cases, and yet the percentage is less than one half of the percentage I have found and less than one third of that found in New York.

The etiological relation of syphilis to tabes has been established statistically by the discovery that whereas syphilis exists in about 10% of non-tabid patients, it exists in about 90% of tabid patients. This points to the fallacy of the conclusions drawn from the statistics as to morbid

	No. of Cases.	Percentage neuropathic heredity.	Percentage similar heredity.
Delasiauve (cited by Binswanger),	120	11	4
Osler (Principles and Practice of Medicine. First edition, p. 948),	435	14.5	1
Massachusetts Hospital for Epileptics,	1,200	14.5	4 2 in parents only
Herpin (cited by Binswanger),	380	21	2
Echeverria (Epilepsy),	306	25	
Hammond (Diseases of the Nervous System. Ninth edition, p. 678),	396	28	16
Reynolds (cited by Binswanger),	38	31	
Moreau (cited by Binswanger),	364	32	
Aronsohn (Abst. in Neurol. Centralbl., xiii, 631, 1894),	508	32	16
Berger (cited by Binswanger),	71	32	
Knapp,	320	35	13.5
Starr (Nervous Diseases, Organic and Functional, p. 741),	2,000	35	70.9 in parents only
Binswanger (Die Epilepsie. Nothnagel's Specielle Pathologie und Therapie, xii, 1. Abt. 1, p. 81),	121	36	
Gowers (Epilepsy. Second edition, p. 7),	2,400	40	30
Hamilton (Pepper's System of Medicine, v, 468),	980	50	
Turner (Epilepsy, p. 26),	676	51	37.2
Spratling (Epilepsy and its Treatment, p. 64),	1,070	56	16
Bourneville (cited by Binswanger),	350	70	
Kraepelin (Psychiatrie. Sixth edition, ii, 473),		87	25
Beau (cited by Binswanger),	232		9
Leuret (cited by Binswanger),	106		10

I have put down in the accompanying table the figures given by different writers and the percentage of neuropathic and similar heredity in epilepsy as found by them. This percentage varies from 11% in the old statistics of Delasiauve or 14½% in the statistics of the Massachusetts Hospital for Epileptics, to 87% claimed by Kraepelin; and the percentage of similar heredity varies from 1% as given by Osler to 37.2% as given by Turner. The discrepancies are so great as not to be readily explained, and they leave us in doubt as to the real prevalence of neuropathic heredity among epileptics. The same discrepancies occur in other statistics as to heredity in nervous disease. In Munich different men in the same asylum for the insane found in different years from 37% to 66% of morbid heredity. Krauss²¹ finds that the percentages in the different

heredity in epilepsy and other nervous diseases. The percentage of neuropathic heredity in the healthy has been absolutely ignored, and it is only within recent years, and in a single community, that any extensive inquiry as to such heredity has been made, — in Zurich by Thomann-Koller²³ in 1895, and by Diem²⁴ in 1905. Naecke²⁵ and Jost²⁶ have made researches on a much smaller number of individuals, which seem far less thorough. Thomann-Koller found neuropathic heredity in 59% of 370 healthy persons, and Diem found such heredity in 66.9% of 1,193 healthy persons. In 370 insane, Thomann-Koller found 77% of morbid heredity, and Diem found the same percentage in 3,515 insane. They both conclude that the difference between the morbid heredity of the sane and the insane is so slight as to be disregarded, but they have both found that,

on limiting the inquiry to the morbid heredity in parents alone, and to the prevalence of mental disease only, excluding many of the forms of organic nervous disease, such as apoplexy, there is a greater amount of morbid heredity among the insane.

Another fallacy of this method of estimating morbid heredity is that little attention is paid to the nature or etiology of the forms of disease existing in the patient's family. Dejerine gives a family tree which he regards as one of the most conclusive in its indication of nervous heredity, and he counts in, as showing the neuropathic taint, deformity, deaf-mutism, idiocy, and exophthalmic goiter, as well as various forms of mental and nervous disease. It is so obvious that a case of nervous or mental disease of traumatic, infectious or toxic origin should not be considered as showing a special neuropathic taint, that it would seem impertinent to mention it, were it not so invariably ignored. The existence of several cases of epilepsy in the children of one family, even, cannot be regarded as proof of an inherited tendency, unless we know that they are not due to injury at birth from too small a pelvis.

The data at our disposal for determining the importance of heredity in the production of epilepsy, and of other forms of nervous and mental disease, are therefore inadequate, so far as they relate to a general neuropathic predisposition, since there is no proof that there are more neuropathic conditions in epileptics, for example, than in healthy persons, and the cases of nervous or mental disease that do occur may be due to causes affecting the individual alone, and not to morbid changes in the germ plasma. One thing alone points to the possibility of an hereditary influence in epilepsy. Turner²⁷ assumes that there are from one to three epileptics to every thousand persons. In the figures furnished me by Dr. Flood, in 1,200 cases of epilepsy there was a similar heredity in 29 parents. The exact number of parents is unknown, but estimating them at the maximum of 2,400, this would give over 12 cases of epilepsy in a thousand parents, which shows that there is a somewhat larger percentage of epilepsy than the average in the direct ascendants of the epileptic. Other statistics would, of course, increase this percentage. We see, therefore, that the data as to morbid heredity in the production of epilepsy are too misleading to warrant any definite conclusions, and the skepticism which has been expressed by various writers, from Delasiauve to Marie,²⁸ Sachs, Osler, and Von Jauregg has a reasonable foundation. We must not forget the old adage of Morgagni, — *Observationes perpendendæ non numerandæ sunt*. Each case must be considered on its own merits, and dissimilar affections must not be compared. We must be sure that a case is idiopathic epilepsy, and not epilepsy symptomatic of brain injury or cerebral infantile paralysis. We must study the whole life history of all the other members of the family previous to the generation of the patient, to determine the general physical health of the ascendants, and to eliminate the

various accidents of pregnancy and childbirth. If nervous disease occurs in the family, we must consider its nature and character, whether indicative of an intrinsic nervous defect, or the result of some extrinsic cause, such as infection or trauma. Furthermore, we must study the patient's own life history, and decide how great an influence post-natal causes play. A child of thoroughly neurotic parents may owe much of his nervous disorder to faulty example, lack of proper restraint, defective training, late hours and improper feeding, and little to his direct inheritance. Only by such thorough study can we come to any definite knowledge of the prenatal causes of disease. If we continue to note "cousin had chorea," "uncle apoplectic," "brother paralyzed" (fractured spine), "aunt insane" (puerperal), and then decide that the patient has hereditary disease because such antecedents are found in 40% of the cases, although they exist in 60% of healthy persons, our conclusions will remain wholly unworthy of consideration.

In studying the etiology of epilepsy, the following case will show the importance of certain prenatal influences. Three older brothers in a family were strong and well. The youngest, always a delicate child, became epileptic. The grandparents on both sides showed no special indications of disease. The parents were healthy until the close of their lives, but they died two years after the birth of this patient, the one of cancer and the other of tuberculosis. No history of nervous disease or epilepsy existed in the family, but is there any question that the diseases which affected the parents led to a defect in the germ plasma and the production of a weaker child in whom epilepsy might develop? Such I believe to be the true interpretation of much of the so-called neuropathic predisposition. Some morbid process, constitutional disease, chronic intoxication, infection or local injury to the generative tract causes pathological changes in the germ plasma, and these changes give rise to a general constitutional weakness in the progeny. The child is not simply a neuropath or a psychopath, — he is a weakling. The constitutional weakness shows itself, as we often see, in the various stigmata of defective development and in general physical weakness, — a weak digestion, a small thorax, a hypoplastic aorta, deficient circulation, poor teeth, slender frame, delicate muscles, undeveloped sexual organs, a small spinal cord and the like. Given this constitutional weakness as a consequence of pathological changes in the germ plasma, various forms of nervous weakness may develop from extrinsic causes which determine whether the weakness shall be manifested in the nervous system, and if so, in what particular way it shall appear. In this way, too, acute infections may temporarily injure the germ plasma, so that the offspring conceived or in utero during the acute infection may show marked neurotic phenomena, while offspring conceived before and after the period of infection may be healthy, and the parents themselves may be for the rest of their lives in perfect

health. This explains the alleged preponderance of nervous defects in the children of alcoholic subjects, or the French "children of the sieve." Unless, however, there be some general or local cause of injury to the germ plasma, it is doubtful whether any acquired mental or nervous disease of the ancestors can be the starting point of a pathologically tainted family, or even of sporadic cases of disease in the descendants (Binswanger, Morel²⁰).

It is apparent, therefore, that the old data upon which the present doctrine of neuropathic heredity is based are misleading, and a much more thorough investigation of the whole question is required before we can draw trustworthy conclusions. Each individual case must be studied by itself, with due regard to the frequency of disease in the ascendants, especially in the direct line. Such disease in the collaterals seems to be of slight importance. The nature of each case must be carefully considered, especially with reference to possible injury to the germ plasma. The frequency of disease in the ascendants needs to be compared with the frequency of disease in the ascendants of healthy persons, and finally all possible causes of disease in the individual himself must be considered, and special attention must be paid to the whole life history of the patient, to determine how far a defective organization is at fault, and how far other influences, such as accidents of parturition, faulty training, bad examples, injuries, infections and intoxications have led to the development of the disease. Only by such study can we determine accurately the influence that heredity exerts.

At the present day there is an increasing dread among the laity of heredity in general, and neuropathic heredity in particular. This dread exerts a considerable influence upon marriage and the procreation of children, and threatens to give rise to ill-considered legislation. The present doctrines are certainly too pessimistic. Galton has pointed out the tendency to regeneration, or to return to the normal average in a generation or two, and further study may throw light upon the question raised by Mendel, as to whether morbid traits may be dominant or recessive. Weismann's teachings show us the importance of guarding against all injuries which may affect the germ plasma, so that it may be possible to guard against the procreation of weaklings, either with or without a history of long continued disease in the ancestry. Furthermore, investigation will often permit us to reassure the individual, when some case of nervous or mental disease develops in another member of the family, or even in himself, and to explain to him that it may indicate no disposition to similar disease in himself or his descendants.

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HENRIK KELLGREN AND HIS METHODS OF MANUAL TREATMENT.

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ALTHOUGH it may truly be said that the name of Henrik Kellgren is known almost throughout the civilized world, yet it must be conceded that few persons know anything definite about the man or the methods of treatment he employs. The following brief sketch of his life and work is intended to supply the particulars hitherto lacking.

Henrik Kellgren, born in 1837, at Alingsås in South Sweden, matriculated in 1855, and in 1858 became an officer in the Swedish army. From 1863 to 1865, he went through the course of training at the Royal Gymnastic Central Institute in Stockholm, and in the latter year received the diploma, whereupon he accepted a post as teacher of pedagogical gymnastics in Lidköping. It was in the latter place that he obtained his first case for treatment by medical gymnastics, and already at this early period he realized how important it is to give not merely local, but also general, treatment (of this more hereafter), but owing to his preoccupation with military duties, he was not yet led to develop any great interest in the subject of medical gymnastics as a whole.

At this time a series of troubles fell upon him, and the death of his wife and only son changed the course of his life. Leaving his native land, he went to Germany with the intention of proceeding to Algiers, where the French were then in conflict with the insurgent Arabs. At Bremen, he became acquainted with a medical gymnast, Dr. Ulrich, with whom he stayed for some weeks. This Dr. Ulrich was the first to recognize in young Kellgren, the great natural aptitude for medical gymnastics, which afterwards developed so remarkably; and he urged him to devote himself to this new career. In the end, Kellgren was prevailed upon, and in the year 1869, he settled in Gotha in Thuringia as medical gymnast.

He naturally began to treat on the lines laid down in the Royal Gymnastic Central Institute, but his real talent, aided by instinct, soon led him to remodel several of the exercises on his own