sents an arrest of growth of the whole of the small intestine. The preparation was obtained by Mr. Abernethy from a boy aged 12 years, in whom the small bowel was only 2 ft. in length; the colon measured 4 ft. Mr. H. S. Clogg¹⁵ has recorded two very instructive cases of occlusion of the ileum; in one the ileum at Meckel's point was reduced to a thread; in the other the small intestine, which lay within an umbilical hernia, showed a complete absence of the segment of the bowel in the neighbourhood of Meckel's point. Dr. D. Waterston¹⁶ has also reported a case in which the ileum near Meckel's point was absent, the corresponding part of the mesentery showing a V-shaped deficiency.

Occlusion at the Ileo-caecal Junction.

Although cases of congenital occlusion at the ileo-caecal junction have been recorded, there are no representatives of the lesion in the metropolitan museums with the exception of one in the college collection. Seeing that the musculature at the termination of the ileum is sphincteric in action, one may expect constrictions to occur at the ileo-caecal orifice similar to those which occur at the pylorus, but the only specimen I have seen which may represent such a lesion is No. 2522A in the college museum. The preparation was obtained from a woman, aged 62, who suffered from habitual constipation. The constriction at the ileo-caecal sphincter is supposed to result from syphilis, but there is no evidence that this is the case. In the specimen of congenital ileo-caecal occlusion the lumen of the ileum is closed $\frac{1}{2}$ in. above the ileo-caecal orifice.

Congenital Occlusion of the Colon.

This lesion is represented by only four preparations in the medical museums of London. In one the great intestine is absent beyond the hepatic flexure; in two the splenic flexure is represented merely by a thread which united the transverse and descending colons; in the fourth a septum occurs at the junction of the pelvic colon and rectum. Congenital atresia of the colon is thus of uncommon occurrence; apparently the splenic flexure is the part most frequently affected. Atresia at the termination of the rectum is by far the most common point at which occlusion of the alimentary canal occurs. The specimens illustrating this lesion have been already described.¹⁹

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At its last meeting the Paris Académie des Sciences elected Sir Patrick Manson a corresponding member.

THE International American Congress of Medicine and Hygiene will be held on May 25th in Buenos Aires in commemoration of the first centenary of the May revolution of 1810. The President of the Congress will be Dr. Eliseo Canton, who, in conjunction with the Minister of the Argentine Republic at Washington, has appointed a Committee of Propaganda, of which Dr. Charles H. Frazier of Philadelphia is Chairman, and Dr. Alfred Reginald Allen of Philadelphia, Secretary. The work of the congress will be distributed among eight sections: Biology; medicine; surgery; public hygiene; pharmacy and chemistry; sanitary technology; veterinary police; and dental pathology. There will also be an exhibition of hygiene. The official languages of the congress will be Spanish and English.

A Lecture

ON

SOME ASPECTS OF POLIOMYELITIS.

DELIVERED AT THE NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC, DECEMBER 14TH, 1909.

BY

SIR WILLIAM R. GOWERS, M.D., F.R.S.

GENTLEMEN,—If you were asked the question, "What disease causes the most numerous mistakes in diagnosis?" your answer would probably depend on the character of your experience; but I think if you said "poliomyelitis" you would not be far from the truth. The errors to which it gives rise do not imply ignorance. They are due to the fact that the symptoms of the paralysis are so often preceded by others that resemble some general malady, and this is first supposed to exist, and to it the paralysis, when recognized, is often supposed to be secondary.

This danger does not always arise. In many of the cases, especially in those which arise in the early period of life, the paralysis comes on at the onset or at the second day of any general symptoms. There is then no room for error. But when the onset is after the infantile period, as the first three years of life may be termed, the general initial symptoms are much more marked in character and more prolonged in duration. These misleading cases have seemed to be more frequent during recent years, and are certainly of sufficient frequency to justify a word of warning. They occur at any period up to the completion of development, and sometimes even later. These patients are naturally able to describe their symptoms in greater detail, and so sometimes to add to the delusiveness which special symptoms confer upon them. I shall have some cases to show you which illustrate this fact.

I may mention another symptom which often causes error—the great pain in the limbs in the early stages, often accompanied by much tenderness. It may endure much longer, and is then attended with definite signs of inflammation of the nerves. In the early stage much fever accompanies the pain, and it is easy to think that the symptoms indicate rheumatic fever; to which it is easy to think that the spinal cord affection is secondary. This is probably always a mistake. I saw a case not long ago in whom the febrile disturbance of the disease lasted for a fortnight, and a diagnosis of typhoid fever was made to which the spinal affection was thought to be secondary. In such cases it is easier not only to say but to think that the original diagnosis was correct, and the spinal affection was really secondary to it. Many diagnostic errors are made in a kind of honesty over which the mind glides easily to a wrong conclusion.

Moreover, a mistake is often rendered easy by the severity of the initial symptoms; they naturally cause prostration. The area affected, by the paralysis is usually at first wide, sometimes almost universal, and only the return of power in some part shows that it cannot be ascribed to mere prostration. Those of you who have already had some experience of practice will realize how firmly rooted an idea may be about the nature of a child's illness in the maternal mind, especially if it was originally formed after a consultation with the mind of an older individual of the same sex. I remember once seeing a child with severe paralysis of this form whose mother had allowed it to go on for three months before she thought it worth while to see a doctor.

The disease was formerly known as infantile spinal paralysis because it chiefly occurred during the first three years of life, but cases later in life seem to have been more frequent in recent years, and during the last ten or fifteen years it has been observed to occur in apparent epidemics. In consequence, the pathology of the disease has attracted much attention, and our knowledge of it has been found to be most defective.

The epidemics of the disease have varied much in their extent, but all have occurred during or just after the hot season of the year. This is not a new feature; it has always been so with the sporadic form. As I expressed it many years ago, two thirds of the cases occur during the

hottest third of the year. Excluding the epidemics that is nearly the truth, and it is less than the truth if they are included. July, August, September, and October are the periods of most of the cases. Both sporadic cases and those in epidemics have been noted to occur after hot dry weather.

The most extensive epidemic occurred in the United States in 1907, and is believed to have included several thousand cases. Two have occurred in Australia—one in Queensland in 1903 and one at Melbourne in 1908 (of 137 cases). The second largest occurred in Sweden in 1905, which has been very thoroughly investigated by Wickman, who has published an admirable account of it, much more thorough than any other outbreak has received. It consisted of a great number of small local epidemics, each of which is separately described and illustrated by a small map. He is convinced that the extension of the disease in each locality can only be explained by personal contagion, although in every instance it seemed to begin without infection. Unfortunately he has thought it desirable to introduce a new name for the disease, and calls his book the *Heine-Medin Malady*, from two writers who described it, the latter in its epidemic form.¹

It is curious that extensive outbreaks have been preserved for the present time. But many smaller epidemics have been described—one in the United States as long ago as 1841. This country has, fortunately, been free from such as merit record. A curious series of cases in one family in London was recorded a few years ago by Dr. Pasteur, and a very instructive account of an outbreak last year in the south of Essex has been given by Dr. W. W. Treves, of the London Hospital, in the last number of *Brain*.² It comprehends only 8 cases which occurred in Upminster, July to September, 1908. It closely resembled the individual outbreaks in Sweden. No other epidemic is known to have occurred in this country last year, but I have 3 sporadic cases to show you, the onset of which occurred then. Two of them have a peculiar interest because they are two brothers, who lived together near Wisbech in Lincolnshire, and were taken ill on the same day, whilst the epidemic at Upminster was in progress. But no connexion between the two cases can be traced, and Wisbech is about 130 miles from Upminster.

It has been noted that during the epidemics cases have been frequent which transcend in age the common infantile period of incidence of the disease, and that they present features that are thought to be special to the epidemic form. It is probable that these features are chiefly connected with the older age of the patient. Dr. Berg,³ of New York, has specially insisted on the features of the epidemic form in the United States, but these features have been conspicuous in the cases that have occurred here in the last two years, including those I have to mention to day.

The first two cases I have to show you are young men, two brothers, healthy young farmers. They lived in the same house in the country, and were taken ill on the same day a year and a quarter ago. No other cases are known in the district, before or since, I am assured by the doctor who attended them. The younger, George C. is now 17, he was 16 when, on August 27th, 1908, he was suddenly taken ill with sore throat and some headache. He soon improved, but did not feel strong enough to resume his usual work. On September 5th the weakness seemed to increase, and he had a difficulty in standing up, and the following day he had greater weakness, especially in the left leg; he also again felt ill, and his temperature was considerably raised—to 104°F. The following day the right leg was also almost powerless; and afterwards he found that the arms were also weak. There was no loss of sensation or impairment of the sphincters. In ten days power began to return in the feet; he stayed in bed for a fortnight, and slowly improved, but it was three months before he was able to stand. No symptoms in the region of the cranial nerves were felt, but his power of coughing was much weakened.

His residual condition you now see. His arms are only a little weakened, but the thoracic muscles are very feeble, and he can exert no power with the abdominal muscles. The left erector spinae is much more wasted than the right, and considerable curvature has developed. The legs are still weak, but he can walk for a mile, and the knee-jerk is fair on the right side; on the left it is only just to be obtained.

The brother was 17½ when he was taken ill, in the same way, on the same day, August 27th, with sore throat, frontal headache, and general malaise. He was in bed two days and spent the third in the house, but felt so well on August 31st that he went back to his farm work and did an ordinary day's work. The following day was wet, and he got wet through, but came home and changed his clothes, feeling quite well. He slept well that night and got up in the morning feeling nothing amiss. But at 9 a.m. he felt giddy and could not walk straight. The condition came on quite suddenly, while he was churning. Headache came on at the same time, frontal and at each side; it was severe, the giddiness rapidly increased, and was accompanied by weakness of the legs, which were so feeble that at 11 o'clock he went to bed. He slept most of the time till the following morning, when he had lost all movement in the legs and the arms were distinctly weak. He was found to be feverish, his temperature being 104°F. For a fortnight he continued to feel ill, with much headache and sleeplessness. The arms began to gain strength after the first few days, and in six weeks seemed as strong as before, but the legs presented wasting, and even now there is only a very little movement in the toes.

Before I speak further of these two brothers I will mention the onset of the disease in the two other cases I have here. This boy was taken ill a year ago, in November, 1908, when he was a little more than 14 years old. For two days he had general pains, especially severe in the back. This is a common seat for the initial pains. On the third day weakness of the legs was found and he was more feverish, his temperature being 104°, and there was some delirium. The weakness of the legs continued with wasting, and even now very little recovery has taken place.

The fourth patient is younger, only 7. His illness dates from this year; it began in the middle of last September. It at first resembled a severe cold, and lasted for a week. He was kept in bed for three days, and seemed to get quite well and to be so for two days. (Compare this intermission with that of the two brothers.) Then, while writing a letter in a noisy room, he went to sleep. He was carried upstairs, undressed and put to bed, still asleep. He slept on through the rest of the day and night. He woke in the morning to find that he could not move his legs or the right arm. He had pains all over, and his neck was rigid. His temperature is said also to have been 104°. Power in the arm was soon regained, during the first week, and the stiffness of the neck disappeared, but his legs still remain paralysed.

These cases illustrate the common rule, but varieties constantly occur in the severity and duration of the early symptoms. Sometimes they are scarcely to be recognized, occasionally, as I have said, they continue for a couple of weeks. All the cases I have shown you are beyond the common infantile epoch, and, as a rule, the older the patient the more pronounced are these general manifestations, and the longer their duration. In the remarkable occasional prevalence of the disease during recent years, an unusually large proportion of the cases have occurred in patients who transcend in age that of cases usually observed, and in whom, therefore, these general symptoms have been unusually conspicuous.

I may mention to you another sporadic case of last year. A girl of 16½, ten days after getting wet through, was seized with a febrile illness on September 26th. Her temperature was 101° on the second day and 102° on the third, with paralysis of the legs and loss of all reflex action. Retention of urine existed from the first, but power of expulsion was regained in a month. Moderate recovery very slowly followed.

The following case presented the affection of the centre of the medulla, which has been thought to be a feature of the epidemic form. A boy, aged 10, at school played football after rain on the last Saturday in September, 1909, and sat down afterwards on the damp grass. On the Monday he had headache and a temperature of 104°. On Wednesday weakness of the right side of the face was noticed, and it was definitely paralysed on the next day. Then the right arm was found to be paralysed, and in a day or two both legs also, and at the end of a week there was strabismus, apparently due to paralysis of the sixth nerve. The temperature did not become normal until after the tenth day. The face recovered power in a week.

The strabismus ceased after a month; the arm can now be moved freely but is generally wasted, and the legs are still too weak to enable him to stand.

Another feature of the epidemic form is the frequency with which cases terminate fatally and also recover completely. The mortality has been estimated at about 12 per cent., and consists especially of cases in which the grey matter of the medulla has been extensively involved.

Numerous and strenuous have been the attempts to isolate and cultivate the material agent of the disease. But none can have been said to have been successful. In a large number of cases the results have been simply negative. Organisms have been sought in the spinal cord of early fatal cases, and also in the fluid obtained by lumbar puncture. Those that have been found in small quantity, bacterial or micrococcal, have failed to grow. The only success in reproducing the disease has been obtained in monkeys, which seem specially prone to the malady. Landsteiner and Popper⁴ failed to find any organisms in the spinal cord of a boy, aged 6, who died within twenty-four hours of the onset of symptoms in Vienna, nor could they succeed in attempts to cultivate. The substance of the cord was injected without result into rabbits, guinea-pigs, and mice, but of two apes injected, one developed typical paralysis after seventeen days and died in two days. The other monkey was taken ill the day after inoculation, and died two days later. There was general inflammation of the spinal cord, but not the typical condition of the disease. The spinal cord of the first was injected into two other apes, but without result. Recently greater success has been obtained by Flexner and Paul⁵ in the United States at the Rockefeller Institute, New York. They succeeded in producing the disease in six successive monkeys, using for the first the inflamed spinal cord of a fatal case of poliomyelitis, and for the next the spinal cord of the monkey affected. They employed intracerebral and intraperitoneal injection, but at first failed with subcutaneous injection, which, however, was at last successful. But they failed to find or to cultivate any organisms. They confirmed the supposition that their size is too small for observation, by pressing the material through a Berkefeldt filter, and after this found that it would still give rise to the disease.

This result seems to raise a question whether the poison which affects the spinal cord may not be a simple chemical poison like that which has been shown to cause the paralysis after diphtheria, or which causes the symptoms of tetanus. The prolonged manifestations of the early disturbance in many cases are often in harmony with a secondary production of the poison. It is so, for instance, in the cases of the first two patients and the fourth one I have shown you, in which the early general symptoms were separated by approximate or complete recovery from the onset of the symptoms of the affection of the nervous system. But in the third case the nervous symptoms seemed part of the early disturbance, and this is often the case.

Unfortunately, moreover, this discovery of the reproduction of the disease by the material from the seat of the specific inflammation affords no prospect of means of treatment. It leaves us therapeutically where we were before. Could a bacterial materies morbi be obtained, we might hope that its cultivation would afford the prospect of obtaining a means of counteracting the action on the spinal cord, but any discoverable organism seems further off than ever, and there is no promise of any prospective means of neutralizing the paralyzing agent. We can only hope that the power of recognizing the initial symptoms may grow, and that they may induce more care in the management of those patients who present them, even if they have the semblance of a simple catarrh. At least we may hope that this may be common when there are any signs of local prevalence of the disease. Any pronounced or special symptom of a catarrh should then cause more than usual caution, especially if pains in the limbs or back are conspicuous. When any loss of power presents itself it should be remembered that it may be the same at first whether the paralysis is going to be transient or enduring, and a posture should be adopted which will prevent congestion of the grey matter related to the part. Especially if the symptoms indicate that the spinal cord is affected, the patient should not lie on the back. If a position on

one or the other side is impracticable, pillows against the back will raise the spine from the lowest position and save the inevitable hyperaemia from being augmented by the effect of gravitation. We can never know the effect of the measures we adopt in a case in which the degree of change varies so much, but it is more than probable that this measure will often save from an extreme degree of damage which otherwise would easily occur. Any symptoms indicating an affection of the nerve centres should obviously prevent any functional excitement of the part, even so slight as that involved in the least locomotion. The medicine given at first may be that for an acute catarrhal state which the general character of the symptoms resembles. Strychnine should not be given until after the first two or three weeks. It is not wise to stimulate structures that are inflamed. Electricity should be deferred a little longer. Massage, or rather rubbing, may be commenced sooner, but with great gentleness. Upward rubbing should be employed, with such pressure as causes no pain. Kneading of the muscles is better avoided. The upward motion has a valuable influence on the circulation, vascular and extravascular; it tends to move the fluids towards the heart. The affected limbs are cold, and there is a tendency to slight oedema of the subcutaneous tissues which the effect of upward rubbing counteracts.

The inflammation of the grey matter, if severe, destroys the motor nerve cells and entails degeneration of the motor nerve fibres, and in consequence the excitability of the muscles to faradism fails, but the muscle tissue retains its excitability to slowly interrupted voltaism. This is generally increased, the action occurring to a weaker current than in health, and more readily to the positive than to the negative pole; in other words, the anodal closure contraction occurs more readily than the kathodal closure, which normally occurs with the weaker current. This voltaic reaction of the muscles is very important, because, if employed, it preserves their power of action to the normal stimulus if the nerves regenerate, as they may do if the cells are not completely destroyed.

We do not always find the reaction changed as I have described. Sometimes the inflammation is slight, and only a few of the cells are gravely damaged. The muscle may then react to faradism through the unaffected nerves, although those that have degenerated cause some of the fibres to react to the anode of degeneration. On the other hand, the action to voltaic electricity may quickly disappear, as well as that to faradism. This is a sign of a very irritative form of nerve degeneration, which passes from the ends of the motor nerves to the muscular fibres, and entails their structural degeneration, so that they also perish and cannot be renewed. It is thus the gravest possible sign.

The two conditions may be reproduced experimentally. If the nerve is tied by a simple thread, a simple nerve degeneration is the result, with the persistent voltaic irritability in the muscle. But if the thread is made irritative by some acid and is passed through the nerve, the descending nerve degeneration is rendered irritant; it is passed on to the muscular fibres, and they speedily lose all reaction, and undergo rapidly the destructive degeneration.

Such cases are but rarely met with. Ordinarily, there is not complete loss of excitability, and after a few months improved reaction can be perceived and steadily increases. Indeed the progressive improvement goes on for much longer than would be thought probable and the degree of power ultimately gained transcends anticipation. After a month from the onset, strychnine should be given regularly. After a time, say six months, when the legs are affected, their power may be made more effective by artificial support carefully contrived to supplement the weakness. The deformities which are apt to develop from the contraction of the less paralysed muscles, in consequence of the loss of the force of those that should oppose them, must be carefully watched for from an early period. As far as possible they must be prevented by careful attention to posture; especially the contraction of the calf muscles must be obviated by support to the feet, and curvature of the spine when a child begins to sit up, if there is weakness of the spinal muscles which is greater on one side, may be counteracted by constant attention to posture. Often an appliance is needed to give the support that is lacking on one side and prevent the displacement

of the spine towards the less affected side, which, in these cases, often tends to occur.

The epidemic in Scandinavia has left the conviction that the disease is contagious. This is the conclusion of Wickman, who has described the whole epidemic most carefully. Cases of poliomyelitis are now carefully isolated in Denmark and Sweden, and are taken to the hospitals for contagious diseases, whether the cases are sporadic or the disease is epidemic. In this country, with the exception of the small epidemic I have mentioned, the disease has only occurred in its sporadic form. No second case has ever been known to occur from contact. This is true of the cases I have shown you to-day; even if we include the so-called abortive cases, no instances are to be heard of. Isolation is never thought of, and it is always neglected with perfect impunity. But when we read the account of the separate outbreaks described by Wickman, it is easy to understand the belief that it is contagious, and yet there are difficulties in applying the belief. Many escaped infection who were of the age that involves an apparent disposition to suffer. Others suffered in the same district who lived so far away and were affected so long afterwards that it was necessary to assume an indirect contagion by some unknown means. But this can only happen in the case of a malady that is intensely infectious, of which, in this malady, there is no evidence.

The only other conceivable explanation of these outbreaks is that the cause of the attacks is of local origin—that the so-called epidemics are really endemics. The Upminster outbreak is of significance in this respect. Although it was so small in extent, it presents all the features relevant to the question of its nature. The first case was a resident child, $3\frac{1}{2}$ years old, who was taken ill on July 26th. All the other cases were over 6. Two sisters were taken ill on August 24th who had been staying at a distance and returned to Upminster on August 18th; one proved an abortive case, but the other developed some paralysis. A boy was also attacked on the 24th. Two sisters who had been away until September 3rd were attacked on the 11th and 12th; a younger child was unaffected. The last case was a boy taken ill on September 16th, who had no constitutional symptoms and only transient palsy of one leg. No opportunities for personal infection are mentioned.

The cases which returned to Upminster from a distance suggest five or six days as the period of incubation of the disease. Attempts to determine it were made by Wickman without success. They varied from one to thirty days. It is certain that if the disease is endemic in origin and not due to contagion many cases must occur having the semblance of a contagious origin.

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An Address

ON

HEREDITY AND DISEASE.

GIVEN BEFORE THE SWANSEA DIVISION OF THE
SOUTH WALES AND MONMOUTHSHIRE BRANCH OF THE
BRITISH MEDICAL ASSOCIATION.

BY

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WITHIN the limits of this address I shall not attempt to deal with details, but shall confine myself to a statement of certain fundamental principles which must always be clearly kept in mind if we wish to get any real grip of the problems associated with the inheritance of disease.

As with many other concepts of science, heredity has passed through a stage in which it was looked upon as something mystical, or at any rate metaphysical—something akin to the vital essences or the humours of the old philosophers. But those old foes to mysticism, observa-

tion and experiment, enable us to conceive of heredity and inheritance as something entirely physical, to be expressed in terms of molecules and chemical combinations, with an actual physical basis of particles of protoplasm. Of course, as regards the eternal "why" of the whole process, we are no whit nearer than was Hippocrates—"Ignoramus, Ignorabimus"—but we have attained to a working hypothesis as to "how" heredity is manufactured in the laboratory of the reproductive organs.

Heredity, then, is not an abstract conception, or a principle, or an essence, but simply a short term for "the genetic relation between successive generations of organisms," and inheritance may best be defined as being "all that any organism is, or has, to start with by reason of its hereditary relations" (Thomson).

And what is the physical basis of inheritance? It may be taken, I think, no longer as a physiological theory, but as a fact, that this physical basis resides in those deeply stainable particles in the intranuclear network of the fertilized ovum known as the "chromosomes" or "idants," and one of the most interesting discoveries of modern times is that all the cells—all the cells of the different tissues—in the body of any particular plant or animal always contain the same number of chromosomes, and in all forms which reproduce sexually the number is even. The number varies greatly—as low as two in some of the threadworms, as many as 168 in some of the crustacea. The human species has twenty-four chromosomes, and we share this number with such differing fellow-beings as the mouse, the trout, the salamander, and the lily. It is known that the mature sperm cell and the mature ovum just prior to fertilization each contains only half the number of chromosomes characteristic of the body cells of the species—during the maturation of the germ cells, that is, there has been a reduction of the chromosomes by one-half. When the nuclei of the germ cells fuse to form the single nucleus of the fertilized ovum it is found that all the paternal and maternal chromosomes are present, there has been no fusion of the chromosomes, and the fertilized ovum now again contains the number characteristic of the species—twenty-four in man—half of which have been derived from the father and half from the mother. If we look upon the chromosomes as containing within themselves the physical bases of all the independently heritable and independently variable qualities which in their totality are the future offspring, this reduction to one-half the number during maturation followed by the close union of the two sets of chromosomes with their different ancestral histories both explains how it is that an offspring tends to resemble its parents and at the same time tends to vary from them—for in this mingling of the chromosomes there must be ample scope for new permutations and combinations of the smaller particulate constituents from which the chromosomes are built up.

Each cell of the future offspring will have 12 paternal chromosomes and 12 maternal. The mature sperm cell of the father has 12 chromosomes, having lost half its original number during maturation, but it does not of necessity follow—and this is the point of cardinal importance—that 6 of them were derived from the grandfather and 6 from the grandmother. The reduction of the paternal chromosomes from 24 to 12, which occurred during maturation, may take place more or less at hazard. The final number 12 is always attained, but it may be that 7, 8, or 9 of them come from the grandfather, and only 5, 4, or 3 from the grandmother. If the mature sperm cell in a given case had 9 grandpaternal and only 3 grandmaternal chromosomes, then obviously, so far as paternal inheritance goes, the offspring would tend to resemble its grandfather and his ancestors much more than its grandmother and hers. In view of the facts that certain qualities such as stature, or certain diseases such as haemophilia, show a definite relationship to the sex of the inheritor, it is probable that the question of sex causation is connected in some way with this irregularity in the reduction of the chromosomes. Similarly the chromosomes of the four grandparents are made up of contributions from eight great-grandparents, and if the reduction processes were always quite regular, the 24 chromosomes in the fertilized human ovum should contain in the 12 derived from the father 6 from the grandfather and 6 from the grandmother, and these latter groups should each