THE GULSTONIAN LECTURES ON EPILEPSY.

Delivered before the Royal College of Physicians of London.

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LECTURE I.

MR. PRESIDENT AND GENTLEMEN,—The founder of the lectures, the delivery of which for the present year I have the honour of commencing to-day, left certain directions regarding their method and scope, doubtless well known to you. The progress of medical science has, however, rendered strict adherence to those directions scarcely practicable. Two or more diseases were to be discussed, and a body if possible dissected in illustration of them. But the time is long past since the demonstration of a chance "subject" could afford instruction to the Fellows and Members of this College; while our study of disease has become so elaborate that, in the space of three hours, it is impossible to consider more than a few points regarding any one disease. May we not read "between the lines" of the founder's direction the anticipation that the progress of pathological anatomy would speedily reveal the organic basis of all diseases? Such an anticipation may, when the words were written, have been not unreasonable. Yet, vast as is the progress of pathology—aided as the scalpel is by sciences which, two and a half centuries ago, were undreamed of—the changes which underlie the so-called functional diseases seem almost as far as ever from discovery. The anatomist may search in vain for any evidence of their existence. Even the microscope reveals but traces of their effects, and can detect no indication of their nature. Our knowledge of them has still to be gained chiefly by inductions based on clinical study. In this, as in anatomy, modern science has given us the assistance of fresh methods, for which, if far feeble than those supplied by the microscope or the retort, we may still be grateful. More precise methods of observation and of record, more exact modes of reasoning, have added much to the power and scope of clinical observation. Foremost among these methods is to be placed that of numerical analysis.

These remarks are strictly true of the functional diseases of the nervous system, and of none more so than of those characterised by convulsion, to epilepsy and its congeners. Pathological anatomy and experiment have taught us something of the coarser pathology of convulsion, but for most of our knowledge we are still dependent almost wholly upon clinical observation. How successfully numerical analysis may be applied to the study of convulsive diseases we have learned from Dr. Reynolds, Dr. Sieveking, and others; and we have all learned, and are glad to be still learning, from Dr. Hughlings Jackson, how much a minute analysis of individual cases may teach. I trust, then, that I shall not be departing from the spirit of the founder's directions in taking this class of diseases as the subject of these lectures, and applying to them the methods of numerical analysis on a somewhat larger scale than has been hitherto attempted, and under conditions which may be considered as, at least approximately, those essential for the successful use of this method. I propose to ask your attention to some of the facts presented by a series of cases which have been under my care during the last ten years, mainly at the National Hospital for the Paralysed and Epileptic. The cases have been taken consecutively, without selection; the facts compared have, with few exceptions, been ascertained on an uniform system, and they are, I hope, sufficiently numerous to yield trustworthy results. On some points, the conclusions to be mentioned are the same as have been reached by previous investigators from small numbers of cases; on other points, they are somewhat different. It is scarcely possible for me to refer, except in a few instances, to the results obtained by other workers in the same field. My excuse for the omission of such reference must be the brief time at my disposal, and the fact that the conclusions I have now to offer are the result of an entirely independent investigation.

The cases, then, which constitute the subject of these lectures are those of which the symptoms consisted in chronic convulsive attacks, not due to organic brain-disease. All cases have been excluded from analysis in which there was any reason to suspect tumour, chronic

meningitis, coarse syphilitic or other organic disease, and all cases of convulsions succeeding hemiplegia. All cases of simple hysterical attacks, unattended by distinct loss of consciousness, have also been excluded. The series includes, then, epilepsy proper, epileptiform seizures in which there is no evidence of organic brain-disease, and cases such as it has been of late the custom to designate hystero-epilepsy, which present, with loss of consciousness, convulsive movements consisting of more or less co-ordinated spasm—spasmodic movements of quasi-purposive character, opisthotonos, emprostotonos, etc. These latter cases form a series from simple hysteria to true epilepsy. The co-ordinated spasm may exist alone, or may be combined with epileptoid phenomena, or with true epileptic symptoms. Hence it is neither practicable nor wise to attempt to separate these cases entirely from pure epilepsy; and I am satisfied that, in almost all published statistics of epilepsy, many of them have been included. But I shall endeavour to ascertain how far each conclusion, with regard to the whole series, applies also to each of the two classes taken separately—the epileptic, and those presenting hystero-epileptic or co-ordinated phenomena, and subsequently to consider what is the relation between the two. We may at the outset ask, What is the proportion between the cases presenting these two classes of symptoms? Of 1,000 cases, in which an opinion reasonably exact could be formed of the nature of the attacks, they were purely epileptic in 815, while hystero-epileptic symptoms were present in 185, or 18½ per cent.

In the present lecture, I propose to deal solely with the subject of the causation of these diseases: the influences to which they can be ascribed, and the proportions of cases in which the action of those causes can be traced.

What, in the first place, is the influence of sex? In the total number of cases analysed—1,450—females preponderate over males, nearly in the proportion of 6 to 5. More exactly, the percentage of females is 53.4; of males, 46.6; that is, to every 100 males, there were 114 females. This is not due to accidental circumstances; for at the hospital in which most of these observations were made, in the cases which do not present convulsion, males preponderate. It may be thought that this excess of females is due to the inclusion of cases of hystero-epilepsy. But this is true only to a small extent. In the cases of pure epilepsy, the females are still in excess, amounting to 52 per cent., the males constituting 48 per cent. In the cases presenting hystero-epileptic symptoms, females preponderate to a much larger extent, and are to the males as 2 to 1, 66 per cent. being females, 33 per cent. males. In most statistics of epilepsy hitherto collected in this country, males have preponderated. This is probably due to the inclusion of cases of syphilitic brain-disease, which we have only lately learned to separate.

There are few diseases in which heredity is a more important etiological condition. The difficulties of the investigation of heredity among the poor are well known. Of all diseases, it is probably true that the results obtained fall short of the actual facts, but the discrepancy is probably less in these affections than in almost any others. The occurrence of fits or insanity in any member of a family is generally well known, and there is not the same tendency to conceal the existence of these diseases among the poor as among the rich. The facts ascertained in this inquiry relate to the occurrence, in ancestors or collaterals, of epilepsy, insanity, hysteria, chorea, and certain forms of paralysis—viz., paraplegia and infantile paralysis. Ordinary hemiplegia was not included, because primarily due to disease of vessels, etc., and not of the nerve-tissues. Habitual drunkenness, however distinctly allied in some cases to neuroses, is too frequent among the poor to make it possible to attribute any significance to its occurrence as an antecedent of epilepsy. The proportion of cases in which evidence of inheritance is to be traced was found by Echeverria in 300 cases to be 28 per cent.; by Dr. Reynolds, from a smaller number, to be 31 per cent. In my own cases, the proportion is rather higher. Of 1,250, in which the point was carefully investigated, 452, or 36 per cent., presented evidence of neurotic inheritance. There is no difference in the frequency of inheritance in the two varieties; in each it is nearly 36 per cent.—that is, roughly speaking, of every eight cases of epilepsy, or of hystero-epilepsy, on an average, three will present evidence of neurotic heredity, and five will not. Inheritance does not affect the two sexes equally. In the cases in which there was no inheritance, the males amounted to 48, and the females to 52, per cent.; but, among those in which the disease was inherited, the females constituted 57, and the males only 43, per cent. It seems, therefore, that when there is an inherited taint, the females of a family are more likely to suffer than the males. The same fact has been noted by other observers. In examining the mode of inheritance, we obtain some light on the origin of this excess. Of the 450 cases with inheritance, the disease came from the mother's side rather more frequently than from the father's, the difference amounting to 4 per cent.—from the father's side

in 35 per cent. (146 cases), and from the mother's side in 39 per cent. (175 cases), while it was from both sides in about 5 per cent. (25 cases). The side from which the disease is inherited is not unimportant, for it has a distinct influence on the occurrence of the affection in the two sexes. In the cases in which the disease was inherited from the father's side, males were in excess of the females by about 8 per cent. (76 males to 70 females), which is an excess of 14 per cent. above the average for the whole number (46 per cent. males). Whereas, in the cases in which the disease is inherited from the mother's side, females were in excess by about 14 per cent. (107 to 82). Thus the percentage of the males affected is 22 per cent. greater when the disease is inherited from the father's than when from the mother's side. The affection of brothers or sisters must be regarded as an indication of an inherited taint, although we may be unable to trace antecedent instances. In these cases, 89 in number, the females are in very large excess—53 females to 36 males; but the total number is probably too small to permit safe generalisation. We may now see, in part at least, why females preponderate so much more in the cases of inherited disease than when there is no inheritance. First, the inheritance is more frequently from the mother's side than from the father's; and, secondly, when the inheritance is from the mother's side, girls suffer in a much larger proportion than boys. Of the relatives who suffer, the mother is herself diseased in a much smaller proportion of cases than the father. The father himself was the subject of neurotic disease in 45 per cent. of the cases in which the inheritance was paternal; the mother, in only 35 per cent. of the cases in which the inheritance was maternal.

Of the neurotic diseases which can be traced in the antecedents of epileptics, epilepsy is incomparably the most frequent. A history of epilepsy was obtained in nearly three-quarters of the inherited cases. In rather more than half (240) it existed alone; in 54 it was combined with insanity; in 5 cases with chorea; and in 3 with both insanity and chorea. Insanity occurred in the relatives of about one-third (157) of the inherited cases, and of these it existed alone in two-thirds. Chorea occurred in other relatives in 35 cases, in 24 of which it existed alone; in 5 it was associated with epilepsy; and in 3 with insanity. The forms of paralysis, paraplegia, and infantile palsy, included in the investigation, were not more frequent in the families with other evidence of neurotic tendency than in those who presented no such history. The cases in which an antecedent family history of hysteria was obtained were very few. Although the mother was herself less frequently affected than the father, the two suffered from epilepsy with equal frequency, and the difference in the frequency of their affection depended upon the fact that the mother suffered from insanity much less frequently than the father. Of 394 cases of inherited epilepsy, the father was epileptic in 45, and the mother in 46 cases; the father was insane in 21 cases, the mother in only 11. In some cases, the family tendency, as evidenced by the number affected, was very strong. In one case, no less than fourteen members of the family suffered from epilepsy—the patient's mother and maternal grandmother; her mother's sister and mother's brother's son; four of her own sisters, and five of her sisters' children.

Is epilepsy predisposed to by any other morbid heredity than that indicated by the occurrence of its own allies? Rheumatism and phthisis have both been supposed to favour the occurrence of epilepsy. Regarding rheumatism, apart from the heart-disease which it may cause, I have been able to obtain no evidence in support of the view. The effect of phthisis is very difficult to ascertain. It is certain that the family history of epileptics presents a large proportion of cases of phthisis; but in the opinion of some writers, as Nothnagel, the frequent coincidence is accounted for by the commonness of lung-disease. Others, as Echeverria, believe that the frequent association supports the view that there is a causal relation between the two diseases, and Dr. Savage has expressed a similar opinion with regard to insanity. That there is a relationship between the two is suggested strongly by individual cases, as, for instance, one of my patients, who, presenting no evidence of neurotic inheritance, had lost father, mother, and six brothers and sisters from phthisis, she herself, the sole survivor, being epileptic. The statistics which I can offer on this point are unfortunately not very extensive; as far as they go, they do not give support to the opinion that there is a relationship between the two diseases. Phthisis was inquired for in 300 cases, and a history of it in parents, grandparents, brothers or sisters, uncles or aunts, was obtained in 108, or 39 per cent. Considering the commonness of phthisis, this does not seem a larger proportion than might have been expected, although I am not aware that there are any statistics which can be taken as the standard of comparison. Moreover, if phthisis have any influence in causing epilepsy, we should expect to find it traceable more frequently in the cases without than in those with neurotic inheritance; but it is

not so. Of 214 cases without, and 86 cases with, neurotic inheritance, the proportion of phthisis is just the same—39 per cent. Further, if phthisis exerted any causal influence, we should expect to find the proportion in which a neurotic heredity is traceable smaller when there was a tendency to phthisis than when there was not. This also is not the case. The proportion of inherited cases was rather less in these 300 cases than in the whole 1,250, being 28 per cent. The proportion was about the same in the cases with and those without a family history of phthisis, 28 per cent. in the former and 29 per cent. in the latter. These facts suggest that the association of phthisis and epilepsy is wholly accidental. In 8 cases, the patients were the subjects of well marked inherited syphilis. In all of these cases, the attacks had the aspect of idiopathic epilepsy, cases in which there were symptoms suggestive of local brain-disease being excluded. In only two of these cases did the attacks begin in infancy. In all the others they commenced towards the end of or after childhood. Inquiries were made in a considerable number of cases for consanguinity of parents, but in no instance was it met with.

We may consider next the influence of age on the occurrence of epilepsy as evidenced by the date at which the first fit occurred in the series of 1,450 cases. If we take, first, decennial periods, we find that, under ten years of age, more than one quarter of the cases commenced, 29 per cent. Between ten and twenty, nearly one-half of the total number, 46 per cent., began. In the next decennial period, twenty to thirty, the number falls to a seventh, 15.7 per cent. Between thirty and forty, only 6 per cent. began; between forty and fifty, only 2 per cent.; between fifty and sixty, only 1 per cent.; and over sixty, only one-third per cent. of the total number. Thus, just three quarters (75 per cent.) of the cases commenced under twenty years.

The relation of the cases to age is shown in greater detail in the following table, which presents the annual number of cases commencing in each year:

Age.	Cases.	Age.	Cases.
Under 1	78	15	84
1	66	16	84
2	36	17	67
3	36	18	53
4	28	19	44
5	25	20	40
6	31	21	31
7	43	22-29	14-26
8	34	30-39	3-15
9	48	40-49	1-6
10	54	50-59	1-3
11	52	60	2
12	71	64	2
13	74	71	1
14	82		

A large number of cases, no less than 12½ per cent. of the whole, commenced during the first three years of life. In this group, no cases of simple infantile convulsions are included, only such as, beginning in infancy, continued as chronic epilepsy. There is a difficulty in ascertaining the exact date of commencement in all cases. Of about one-third, all that could be learned was, that they began in infancy. In the table, these have been distributed through the first three years of life in the same proportion as presented by the two-thirds in which the exact date of commencement could be ascertained. The number of cases is largest in the first year (5½ per cent. of the whole), and falls rapidly to three years of age, and then more slowly until five, when the minimum for the early period of life occurs, only 1.7 per cent. commencing. From this there is a considerable rise at seven, the commencement of the second dentition, then a fall at eight, and from this the numbers increase slowly at nine and ten, rapidly at twelve, until the maximum is reached at fifteen and sixteen, at each of which 84 cases commenced, 5¾ per cent. of the total number. From this period, we have a rapid fall to twenty-one, when only 2 per cent. of the whole commenced. During the next eight years, the number of cases commencing in each year varied from 26 to 14. Between thirty and forty years, the annual number varied between 15 and 3, and between forty and fifty it varied from 1 to 6. Between fifty and sixty, the annual number of cases was from 1 to 3. Over sixty, the cases were—2 at sixty-two, 2 at sixty-four, and 1 at seventy-one.

The proportion in which the two sexes suffer varies considerably in the different periods of life. We may compare, first, their affection in the several decennial periods. In the cases commencing under ten years, the females exceed the males by 6 per cent. In the second decennium, between ten and twenty, the excess of females amounts to 18 per cent. In the third, between twenty and thirty, there is a great fall; but the excess of females amounts to 12 per cent. Between thirty and forty, the numbers being still smaller, the relation is reversed; the males exceed the females by 16 per cent. Between forty and fifty, with a continued fall of numbers, the excess of males is still greater, amount-

ing to 36 per cent. Between fifty and sixty it reaches 40 per cent., and over sixty males only suffer.

If we examine the several periods of life more minutely, we find that in the cases which commence in the first year of life the females are almost twice as numerous as the males (51 and 27). In the second year, the number of females falls considerably, while that of males rises slightly, so that the difference between them is less. During the two following years the numbers fall, until a minimum of each is reached at five years, the females being still in slight excess. At six and seven years a rise occurs, and the number of cases in each sex is, in the latter, nearly the same. From seven to thirteen, the earlier relation is reversed; the males are in excess of or equal to the females, with the perhaps accidental exception of the cases commencing at eleven. At twelve—that is, in the thirteenth year of life—the maximum for males is reached, and from this until sixteen there is a slight fall. The maximum for females occurs later than for males. The numbers increase at twelve and thirteen, and then more slowly, until at sixteen the maximum for any year is attained (fifty cases), and they are in that year one-third more numerous than the males. During the next five years, the numbers in each sex fall rapidly, the females being still in excess, but the two are nearly equal at twenty-one. The females continue, however, in slight excess until twenty-nine, when the two sexes are affected equally. After this, the relation between them is reversed—in almost every year the males are more numerous. The females present no increase at the climacteric period, and they cease much sooner than do the males. The latest age at which epilepsy commenced in a woman was fifty-five, whereas in men cases commenced at fifty-eight, fifty-nine, sixty-two, sixty-four, and seventy-one. Thus at each of the maximum periods, at infancy and puberty, the excess of females is very great. During the later period of childhood, the numbers are nearly equal. The excess of females lessens after puberty, and ceases by middle life.

How far does an inherited tendency influence the age at which the disease commences? It is often stated that when there is heredity, the disease begins before twenty. We shall, however, see that, so far from this being true, the influence of heredity continues until a late period of life, and, although absolutely greatest in youth, it is relatively only a little less during adult than during early life. Particulars as to heredity and age at commencement were noted in 1,120 cases, of which heredity existed in 408. The distribution of these through life was nearly the same as that of the non-hereditary cases. About 5 per cent. began in the first year of life, and the annual number fell to a minimum in late childhood to rise to a maximum at puberty. This maximum, however, occurred at fourteen years of age—a year earlier than the maximum for non-hereditary cases, which was at fifteen and sixteen. The cases with heredity lessen gradually during life, but continue to occur to the last. The latest case of the entire series was a man who suffered from what was apparently idiopathic epilepsy commencing at the age of seventy-one years, and in him the disease was inherited, his father having suffered from fits, clearly epileptic, for many years.

The relative frequency of heredity is shown by the proportion which the heredity cases bear to the total number commencing at each period of life. The relative frequency, in periods of twenty years, is shown in the following table.

Age.	Total Cases.	Heredity.
Under 20	844	319, or 38 per cent.
20 to 39	235	80, or 34 ..
40 and over	34	9, or 27 ..

The average for the whole of life is 36 per cent. This is exceeded by 2 per cent. in the first twenty years; in the second twenty years, the percentage is only 2 below the average; while in the cases commencing over forty, it is only 7 per cent. below the average. During the first three years of life, the disease is inherited by a larger proportion of females than of males (females 41, males 33 per cent.). During the next three years, the proportion of heredity in females is nearly the same, that in males rises. The third triennial period, six to eight, presents a lower percentage of hereditary cases than any similar period under twenty, but the minimum proportion in males occurs in the next triennial period, nine to eleven. In females, however, there is in this period a rise, and the maximum proportion for this sex is attained in the period twelve to fourteen; it falls slightly in the next two periods. The proportion in males rises after the minimum in the period nine to eleven, but never quite attains the proportion presented by the second triennial period of life. In the first twenty years as a whole, the excess of heredity in females over males amounts to 7 per cent. (males 34, females 41 per cent.). Between twenty and forty, the proportion is reversed, the percentage heredity is greater in males than in females (males 36, females 31 per cent.). Over forty, of twenty-four males, there was heredity in six; of seven females, there was heredity in three.

The next point which demands consideration is, how far age and sex influence the occurrence of the hystero-epileptic or co-ordinated forms of convulsion. We have seen that, of 1,000 cases in which the form of convulsion could be ascertained, it was co-ordinated in 185, or 18½ per cent. At every age the proportion of females is greater than of males. Under ten, these cases constitute 15 per cent. of the male and 18 per cent. of the female cases. Between ten and twenty the difference is, as might be expected, much greater. They constitute 14 per cent. of the male, and no less than 26 per cent. of the female cases. Between twenty and thirty, the percentage of the males remains the same (14), and that of the females falls slightly (to 21). Between thirty and forty the male cases with hystero-epileptic symptoms fall to 12 per cent. while the females present a slight rise, and constitute 24 per cent. In the male cases commencing over forty, none of this character occurred; and in women, although 29 cases of the series commenced over forty, only 1 presented these symptoms. Thus cases with hystero-epileptic symptoms commence almost exclusively under forty. In males, they bear at each age nearly the same proportion to the cases of pure epilepsy, decreasing very slightly from a maximum in childhood to a minimum in middle life. In women, their minimum is in the first decade; their maximum is in the second; and the proportion after adolescence remains nearly double that of males during the period in which they occur. I think, however, that some surprise will be felt at the fact shown by these figures—which my actual observation of attack fully confirms—that, even up to the fourth decade of life, one-third of the cases presenting hystero-epileptic phenomena occur in males.

I pass next to consider the information which this series of cases affords as to the exciting causes of epilepsy. The first point which demands attention is the causation of the large number of cases which begin in the first three years of life. Of these, 180 in number, no information was forthcoming in about one-third. In 24 cases, although information was obtained, no cause could be ascertained. In 97 cases, however, a cause for the first fit was assigned. In 2, it was exposure to the sun; in 1, ascariasis; in 7, an acute disease; in 8, a fall; and in 8, the first fit occurred soon after birth, the labour in several of these cases having been difficult, and in some the forceps employed. In the remaining 72 cases, 33 males and 39 females, the first fits occurred during dentition, and were attributed to teething. The percentage of neurotic heredity in these cases was nearly the same as for the whole of life—34 per cent.

Thus nearly two-thirds of the cases beginning in infancy, the conditions of origin of which could be ascertained, arose from the so-called dentition convulsions. It seems legitimate to ascribe to the same cause a similar proportion of the cases respecting which no information was forthcoming. If so, we have a total number of cases due to this cause which constitutes 7 per cent. of all the cases investigated. Few persons who have attended closely to the diseases of children can fail to be convinced of the truth of the opinion long ago urged by Sir William Jenner, and now widely accepted, that almost all convulsions associated with dentition are really due to the constitutional condition of retarded development, which we call rickets, to the irritability of the nervous system which accompanies this condition. The further details of the cases I am considering fully confirm this, for in a large number there was a history of late teething, late walking, and in many of crooked limbs. Even these facts probably do not represent fully the influence of rickets in the production of epilepsy. In many of the cases just described, the course of the attacks was continuous from infancy to adult life. In others, however, there were intermissions, especially during the period of childhood. In several, for instance, the fits ceased at seven, to recommence at ten or eleven years. Now, in 27 other cases which commenced after the period of childhood was over, repeated convulsions had occurred during the first dentition, accompanied in many with other signs of rickets. It seems reasonable to ascribe to these convulsions of infancy a share in predisposing to the convulsions of later life, and the addition of these cases raises the number, in which a causal influence may with probability be ascribed to rickets, to nearly 10 per cent. of the whole. Whether rickets is or is not entirely preventable, there can be little doubt that in the degree in which it leads to convulsions it is always preventable by proper attention to the diet and hygiene of infancy. These facts, therefore, suggest that a considerable proportion of the cases of epilepsy are really within the range of preventable diseases. They suggest, also, that inherited taint exerts the same influence here as at other periods of life predisposing to the occurrence of convulsions in backward children, and to the persistence of the fits, and that the management of children with such inherited taint should be a subject of extreme care.

The statements made by patients as to the causes of their fits have of necessity to be received with caution, and the facts carefully investigated. An occurrence so striking as a fit is naturally

assumed to be due to some cause at least discoverable, and so is often attributed to a remote and improbable antecedent. Of 1,150 cases in which this point was noted, a reasonable cause for the first fit was given in 428, or 37 per cent., and, in the remainder, no probable cause could be ascertained. From this series, the cases distinctly originating in dentition convulsions are excluded. An exciting cause is met with in males much more frequently than in females: in 44 per cent. of men (235 out of 537 cases), and in only 31 per cent. of women (193 out of 616 cases)—a difference between the sexes of 13 per cent. This can scarcely be accounted for by greater exposure to exciting causes, since some of these act more on the female sex; it seems to indicate a greater predisposition on the part of women. If we examine the proportion of cases in which an exciting cause can be traced in the different periods of life, we find that, under ten, it is nearly the same for both sexes (males 41, females 40, per cent.). From ten to twenty, there is a great difference between the sexes, excitants being traceable in 19 per cent. more males than females (males 47, females 32, per cent.). In the next two decades, 20 to 40, the percentage in men continues the same (at 42 per cent.); but in women, it rises (to 35) in the first, and falls (to 32) in the second. In the cases which occur over forty years of age, the percentage in each sex is about equal (40); but in the male cases commencing over fifty, an exciting cause could be traced in as high a proportion as two-thirds. Thus the most striking peculiarity is the large proportion of cases which commence in women between ten and forty years of age without any discoverable exciting cause to which they can be ascribed.

Of all the immediate causes of epilepsy, the most potent are psychical fright, excitement, anxiety. To these were ascribed 157 cases, more than one-third of those in which a cause was given, and one-seventh of the whole series. Of the three forms of emotion, fright takes the first place with 119 cases, 10 per cent. of the whole. The relation of this cause to age is, however, very distinct. It is effective chiefly in early life, when emotion is the most ready, and is most powerful at the transition from childhood to adult life, while after middle life it is almost inactive. Of the 119 cases, only 3 commenced after thirty years of age, and 100 commenced under twenty. Of these, the majority, 70, commenced between ten and twenty, only 30 cases under ten. The female sex is notoriously the more emotional; and accordingly the disease, as Dr. Sieveking and others have pointed out, resulted from fright in a larger proportion of females than of males, although the difference is, perhaps, less than might be expected—71 females and 48 males, 60 and 40 per cent. respectively. It is notorious, also, that the difference between the sexes in emotionality increases as life advances. In childhood, one sex is as emotional as the other; but, with puberty, men become far less emotional than women. The influence of fright as a cause of epilepsy is in strict harmony with this fact. Under ten years of age, the sexes suffer equally. Between ten and twenty, the males are to the females as 3 to 4; between twenty and thirty, as 3 to 13; and over thirty, the only cases due to this cause occurred in women. After seventeen, only 3 cases occurred in men, while 20 occurred in women.

The influence of this cause is accurately recognised in popular phraseology, and is easily comprehensible when we consider how powerfully fright affects the nerve-centres. The stimulation of the motor centres shows itself in the sudden muscular actions, such as the start of alarm, which, no doubt, originates in the neck for immediate action, which danger often entails. The tremor, which often persists, indicates the enduring disturbance of the nerve-centres. In the organic nerves, the vaso-motor centre is first stimulated and then inhibited, the sphincter centres are released from control, the sweat-nerves are excited, and the heart's action is accelerated, perhaps, by inhibition of the vagus, and the last effect is the most constant, the most ready. The exact form of fright varied, of course, in different cases, but the list teaches many lessons. In no less than 8 cases, the cause was some stupid practical joke—as a pretended ghost. In 2 cases, children were shut up in dark cupboards. In 5 cases, the fright was from an alarm of fire; in 6, from burglars. In 6 cases, the patient had watched other persons in fits. Three resulted from alarm during severe thunderstorms. One case was that of a soldier who had his first fit a few hours after being terrified, while on sentry duty, by the unexpected appearance of some goats. Of 76 cases in which the interval between the fright and the fit was noted, the fit occurred immediately in 28, or one-third; within a few hours, in 16. The interval was between a day and a week in 19, and it was more than a week in 13. As might be anticipated, in a considerable number of cases the fits were of the hystero-epileptic type. Of 90 cases, in 61, or two-thirds, they were purely epileptic; in 29, or about one-third, they were hystero-epileptic. The difference between the two sexes in the frequency with which hystero-epilepsy succeeds fright is not great. Of 51 cases in females, 18, or 35 per cent., were

hystero-epileptic. Of 39 cases in males, 11, or 28 per cent., were of this type. A relation can be traced between the interval that elapses before the fit and the character of the attack. The longer the interval, the smaller is the proportion of hystero-epileptics. In the cases in which the interval was more than a week, only 18 per cent. were of this character; in the cases in which the interval was less than a week, but more than a day, the percentage of this form was 21. In the cases in which the interval was only a few hours, the percentage was 25; while in those in which the fit occurred immediately, no less than 57 per cent. were hystero-epileptic.

Other forms of sudden emotional excitement caused the first fit in 19 cases—9 males and 10 females; and these cases also occurred during the emotional period of life, under thirty years of age. Anxiety was the only traceable cause in 29 cases, and in these males preponderated, 16 males to 13 females; and 7 occurred over thirty years, 6 of these being men. Next, in numerical importance, among the exciting causes of epilepsy, are traumatic influences, blows and falls on the head. To these, after the elimination of doubtful instances, 65 cases were due. A third (21 cases) occurred under ten years; rather more than a third (25 cases) between ten and twenty. Males are affected by this cause more than females, the numbers being 39 to 26. This is clearly due to the risks of occupation; for, in the cases commencing before ten years, the females were in excess. Between ten and forty, the male cases in each decennium were rather more than twice the number of the females. Over forty, the only cases were in men. Hystero-epilepsy resulted from a traumatic cause in only a small number of cases—6 out of 44; and, in estimating the significance of these, it must be remembered that traumatic causes often involve intense emotional disturbance. In two-thirds of the cases, the injury was a fall on the head; in one-third, it was a blow on the head. In most of the cases, the patient was stunned for a time; but in only a fifth (8 cases) did the first fit occur immediately; in the rest, an interval elapsed. In one-third of the cases, the interval between the injury and the fit was more than a day and less than a week; in another fifth, the interval was between a week and a month; and in about the same proportion the interval was more than a month.

Exposure to the sun was given as the cause of the first fit in a considerable number of instances, and in 27 it seemed probable that the statement was correct. From this cause, as may be expected, males, from their greater exposure, suffer most; 20 were males, and only 7 females. The cases were pretty equally distributed through life; but the cases in females were relatively most numerous in childhood, and decreased in adult life, to cease at thirty. The form of convulsion was almost always pure epilepsy; only one case presented hystero-epileptic symptoms. In 10 of the cases, there was a distinct attack of sunstroke, occurring in 6 in the tropics. In 10, the fit occurred immediately; in several others, in which an interval elapsed, this was marked by some form of nerve-disturbance, such as tremor. An acute disease was the apparent cause of the first fit in 37 cases (14 males and 13 females), and in more than half (19 cases) the disease was scarlet fever; in a quarter of the cases (9), it was measles; in 5 cases, it was continued fever, called typhus, typhoid, or low fever; in one case, it was rheumatic fever; and in one a severe attack of diarrhoea. The remarkable influence of scarlet fever in the causation of epilepsy demands further consideration. The cases due to this all commenced during early life, under seventeen. It may be thought that the first fits in these cases were due to uræmic poisoning; but this is not supported by the particulars ascertained regarding 14 cases. It is true that, in 3 of these, all that could be learned was that the first fit occurred during scarlet fever accompanied with dropsy; and in 2 others, that the fits occurred after an attack of scarlatinal dropsy was over. But in two other cases, in which the scarlet fever was succeeded by dropsy, the first fit occurred after the fever, before the dropsy. In 6 cases, there was no dropsy; in 2 of these, the first fit occurred after the fever was over; in 1, during convalescence; and in 1 a month later. In 3 cases, the first fit occurred during the height of the fever; and in 1, convulsions ushered in the disease, persisted throughout the attack, and continued afterwards as permanent epilepsy. In only 1 case did ear-disease also result from the fever. The urine was examined in most cases, but no albumen was found. These facts suggest that the first fits were due to some peculiar effect of the scarlet-fever poison upon the nervous system. It may be remembered that the scarlet-fever poison has a very wide-spread action in the system; and that, after scarlet fever, optic neuritis, leading to subsequent atrophy, may occur, without renal or coarse cerebral disease. Intestinal worms rank low as a cause of persistent epilepsy, but were noted in 6 cases, the convulsions continuing long after the worms had been expelled. In 5 cases, the first attack was attributed to digestive derangement. In 1 case asphyxia, and in 9 immersion in water, caused the first fit. Certain toxæmic influ

ences were the apparent cause of the disease in several cases. Of these, chronic alcoholism was the cause in a comparatively small number—13 cases only. In some, the first fit occurred during intoxication; and in a few the attacks recurred after each alcoholic excess.

Six patients were the subjects of chronic lead-poisoning. In two of these there was also chronic renal disease. In one of the others, the epilepsy had existed from infancy, but became much more severe when the plumbism was developed. In the remaining three, the lead-poisoning was the apparent cause of the attacks, which resembled, in character and course, those of idiopathic epilepsy. It is well known that acute lead-poisoning may be accompanied by convulsions; that chronic poisoning may cause chronic convulsions precisely like those of ordinary epilepsy, has been pointed out by others, but is not, I think, generally known. The list of nervous disturbances which may result from lead is a long one, and includes, as cases I have seen testify, acute and chronic mental derangement, neuralgia, brachial spasm, as well as palsy, and muscular wasting and optic neuritis. It is known that lead is to be found after death in the brain in considerable quantity. I have mentioned that in two of the cases of lead-poisoning there was also consecutive renal disease. In two other cases of chronic renal disease, convulsive attacks, resembling perfectly idiopathic epilepsy, brought the patients under treatment. In one, the absence of any coarse changes in the brain was proved *post mortem*. It appears, therefore, that such convulsive attacks may occur and recur for months without other sign of uramic mischief. Several cases in which chronic mental derangement resulted from Bright's disease have also come under my notice. I have met with no case which suggests that tobacco-smoking exerts any influence in the production of epilepsy; but in one case the fits were apparently due to the patient, a lad, having shortly before commenced working in a tobacco-factory, where his occupation caused frequent nausea. In one case, the first fit occurred after the administration of chloroform; and, in one, attacks, which had ceased for many years, recurred after the inhalation of nitrous oxide.

Of sexual processes, retarded or absent menstruation coincided with the first fits in a large number of the cases which commenced in girls between fourteen and seventeen; but the difficulty in determining the exact causal relationship between the two conditions is very great. Epilepsy once set up in such cases, the subsequent establishment of regular menstruation appears to exercise very little influence upon the fits. In boys who are epileptic, masturbation is unquestionably common; but here again it is most difficult to decide to what degree it can be regarded as a cause of the disease, and it was distinctly the cause in only a small number of cases. The first fit occurred in seven cases during pregnancy, without other obvious cause, and in five cases the disease commenced after parturition. No facts which have come under my notice suggest that acquired syphilis is a cause of idiopathic epilepsy. There remain for consideration two conditions, occasionally associated with epilepsy, in some cases causally, in others probably only by coincidence, or as the effects of a common cause—these are heart-disease and chorea. An abnormal condition of the heart was noted in 93 cases; but this does not represent the proportion of the whole in which abnormality existed, since in many its condition was not noted. I have, however, rarely failed to examine the heart in epileptics, and I think that the proportion of cases of valvular disease is not far below the mark. In 8 cases, there was extreme frequency of action; in 9, there was irregularity; in 13, all young persons, there was reduplication of the first sound. In 30 cases there was valvular disease; mitral regurgitation in 20; mitral constriction in 7; aortic regurgitation in 3; and aortic constriction in 1. There was obvious dilatation without valve-disease in 20, and considerable hypertrophy without renal disease in 2. The cases of irregularity and reduplication were all observed within a comparatively short space of time, and I am sure that, had my attention been earlier directed to their frequency, I should have found them in a larger number of cases. In a few cases, it seemed that the heart-disease and epilepsy were in accidental association. In some cases, the cardiac condition, especially simple dilatation and reduplication of the first sound, may have been a consequence of the repeated strain to which the heart had been subjected during the attacks. But in other cases, especially of valvular disease, there was reason to believe that the heart-disease existed before the epilepsy, and in many there was no other etiological condition to which it could be ascribed.

In 20 cases of epilepsy, the patients had also suffered from chorea. The relation between the two diseases was as follows. In 8 cases, epilepsy existed before the chorea. In 12, the chorea occurred first; in 4 of these, the fits began at the time of the chorea, and persisted afterwards. In only 2 of these was there heart-disease. In 5 cases the fits occurred long after the chorea, the interval being four, six, twenty-seven, thirty, and forty years respectively. In one case, peculiar choreic convulsions occurred at the height of the chorea, but passed away

as the disease lessened, and did not recur. It seems probable, in the cases in which the fits immediately succeeded the chorea, and possible, in the cases in which an interval elapsed, that the impaired nutrition of the motor centres during the chorea may have left a predisposition to further disturbance under the action of some other exciting cause.

In the next lecture, I propose to consider the illustrations presented by the cases of some of the symptoms of the attacks.

ABSTRACT OF LECTURES ON CERTAIN PRACTICAL POINTS IN CONNECTION WITH THE SURGERY OF CHILDHOOD.

Delivered before the Harveian Society of London.

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LECTURE III.

Eczema at Umbilicus.—Having already spoken of umbilical hernia in connection with the subject of the development of the abdominal cavity, I will now allude to a strange condition of the umbilicus of which I have seen a good many examples. Close around the cicatrix is a red and irritated or eczematous patch of skin, whilst from the depths of the umbilical fossa oozes a thin purulent fluid. For the cure of this affection, lotions and all other kinds of dressings avail nothing; for, at the bottom of the depression, and hidden by overhanging folds of skin, there is a small fleshy polypus which has sprung up from the scar of the fallen umbilical cord. By the ligature of one of these growths, I have seen a boy at once cured of a condition which had troubled him and his mother from the first month of his existence.

Imperforate Rectum.—In the ordinary course of development, the blind end of the intestinal canal descends into the pelvis, but remains separated by a considerable thickness of tissue from the surface of the perineum; subsequently a depression, which appears at the site of the future anus, traverses the horizontal partition, and so the intestinal outlet is completed; but, if the involution fail to perforate the partition, a perfectly formed anus will exist with complete intestinal obstruction. Such a case came under notice last year: no wonder that full and repeated doses of castor-oil had failed to relieve the constipation. (Digital examination should invariably be made when an infant passes nothing *per anum*.) After a slight operation, the child did well; but such cases are prone to relapse, on account of contraction of the remaining annular constriction.

The rectum may end high up in the pelvis, being represented in the lower strata by a fibrous cord which is connected with the top of the anal *cul-de-sac*. What should be the treatment adopted when no trace of the descending rectum or of an anal *cul-de-sac* can be detected? Exploratory puncture into anatomical darkness will probably accelerate the fatal peritonitis. First, the infant having been placed in the lithotomy position, and being under the influence of an anæsthetic, a careful dissection should be made in the middle line of the pelvic outlet, the finger working with the scalpel step by step, close up in front of the coccyx and sacrum, and a sound should be held in the vagina or empty bladder; and, if the search prove futile, the colon must at once be opened. Many parents would prefer to have their child sacrificed decently—that is, under the operative interference of a surgeon—rather than spared by his skill, if in the latter case the subject were to submit to the lifelong infliction of an artificial anus; but no sentiment must be allowed to come between the surgeon and his duty to his patient. The extraperitoneal (Amussat's) operation for artificial anus is not adapted for the newly born, for in these subjects the sigmoid flexure is so thoroughly invested in a meso-colon, that it is enabled to wander in bold flourishes across the aorta and even into the right flank. He who aims at the performance of the extraperitoneal operation in an infant, may consider himself fortunate if he even succeed in finding a piece of large intestine at all, and I doubt if the gut have been as often opened without damaging the peritoneum as the operation and search for the intestine have been abandoned; but Littré's operation is simple enough. After the ineffectual search has been made in the perineum, the patient is laid supine, and an incision is made in the left iliac region, as if the external iliac artery were to be ligatured; but the peritoneum is to be freely