AN ADDRESS

ON

CHOREA.

Delivered before the Thames Valley Branch, at Richmond.

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GENTLEMEN,—I assume that the purpose of the discussion which I am requested to open this evening, is to assist in furthering the inquiry now being made respecting chorea. You are aware that the subcommittee which is collecting evidence about this disease has issued, in the first number of the *Collective Investigation Record*, a preliminary report, and this showed that the inquiry was likely to prove fruitful in due time. There should be little difficulty in gathering all the necessary facts in an inquiry of this sort. The disorder is common, readily recognised, and the evidence relating to it not usually far to seek.

Permit me, then, to state the points on which further knowledge is desirable.

First, it is required to have a much larger number of cases from which to deduce facts.

Secondly, the ages and sex of the patients are wanted in all cases, and every case in each reporter's practice should be taken note of, for without this, a fallacy may come in respecting the incidence of chorea on different classes of patients.

Thirdly, information is sought as to the general prevalence of the disease in different districts.

Fourthly, under the head of exciting causes, it is of the last importance to be exact as to the nature of these, and the interval between the exciting cause and the onset of choreic symptoms.

Fifthly, more complete information is desirable as to the condition of the heart at the beginning, in the progress of, and after the disorder has passed off. It is very important to secure records not only of the state of the heart a few years after attacks of chorea, but also of the subsequent general life-history of such patients. The latter, indeed, has equal significance with the antecedents of these cases.

Lastly, more knowledge is sought as to the common ailments to which choreic patients are liable. It ought to be possible to determine the average duration of choreic attacks. My colleague, Dr. Andrew, has gathered from a study of cases that this is somewhat over ten weeks, about ten weeks and three days. With gaps such as these in our stock of knowledge respecting chorea, we may rightly feel that there is still much for us to do. To-night, then, I direct your attention to these several points, and beg you to pursue them in your respective fields of observation.

Next, I would crave your attention to an expression of the views of this disease which best commend themselves to me. We are brought in face of several theories as to causation. From the time of Cullen, we have had opinions oscillating from a nervous to a humoral pathogeny, we have also had a mechanical and mechanico-humoral, and now once more a nervous pathogeny for this malady. Sydenham well described the disorder, but he ventured on no theory of it. The writers of a century ago mostly adopted the view of debility with some degree of irritation of the organic or ganglionic nerves. They described it as a morbid susceptibility of the nervous system, generally with diminished power, increased mobility, and irregular action of the muscular system.

This irritability was thought to be due, in children, to debility of the stomach and "collatitious" organs, as Mason Good put it; and, in those approaching the period of puberty, to menstrual nixus and development of the genitalia.

Forty years ago, the teaching of Elliotson was, that chorea was due to a morbid excitability of a certain portion of the nervous system (the medulla oblongata or spinal marrow), with which the nerves of voluntary motion are connected; but not a sufficient irritation to produce tetanus. The proximate cause, he thought, was seated in the head as well as in the spinal marrow, since the highest nerves were affected.

Marshall Hall regarded chorea as an affection of the true spinal system, illustrating the want of harmony between the cerebral and spinal acts. He thought it was at first a centripetal disease, and, subsequently, a centric one. This view was coloured by the prevalent

pathological ideas of the time, but is only partly held now. Elliotson believed chorea to be a constitutional disorder, but he did not know if it was hereditary.

Todd (in 1843) showed that irritation of the spinal cord was not the cause of chorea, because the hemiplegic characters of it indicated lesion above the decussation of the pyramids. Later on, the humoral view of the production of chorea came into prominence, more especially in connection with rheumatism, Bright, Scudamore, Copland, Todd, and James Begbie, having drawn attention to this. Begbie, in particular, observed the occurrence of the two diseases in different members of the same family, indicating, as he believed, their probable dependence on the same morbid diathesis. Bright conceived chorea to depend, in these cases, on irritation conveyed from the inflamed pericardium by the phrenic nerves to the spinal cord. Burrows thought the pneumo-gastric nerves might equally be the medium of irritant influences. Watson conjectured that a simultaneous spinal meningitis might arise, or that the cardiac disease exercised some influence on the afferent nerves of the cord. Todd preferred to associate the cardiac disease with the low nutrient state of the nerve-centres common in the rheumatic condition, believing that the defective nutrition which led to rheumatism was equally favourable to the development of chorea, and that thus the rheumatic constitution was one from which chorea might be evoked. Herein, he was well in advance of his time. The frequency of associated endocarditis led J. Begbie to the belief that the morbid state of the blood in rheumatism was the cause of chorea.

He had observed the presence of vegetations on the mitral valve, but regarded these as due to the effects of altered blood. Later on, J. W. Ogle and Barnes advanced this view. Addison, about 1849, at Guy's Hospital, first directed attention to the frequency of cardiac murmurs in chorea.

The state of opinion was now ripe for the discovery, at St. Bartholomew's Hospital, of arterial embolism by Kirkes, and his application of this theory to explain chorea by the presence of cerebral capillary plugging. This view diverted attention from the rheumatic element in cases of chorea, and, in accordance with the tendency of the pathology of that day, which somewhat limited the science to a narrow field of morbid histology (a part only, be it remembered, of this vast subject), reduced the terms of the question to the mere association of vegetative valvulitis with chorea.

For nearly thirty years, then, the battle has raged on this ground, and some of the best minds in medicine have contended for and against this embolic theory. It is right to state that, within this period, the pathology of chorea has been greatly advanced on all hands. The associated frequency of carditis is everywhere admitted, as is also that of rheumatism. Great merit is due to the Parisian school for maintaining the importance of the underlying rheumatic element in most cases of the disease; to M. Roger, in particular, who showed that heart-trouble might sometimes succeed, as well as precede, choreic attacks; and to M. Jules Simon, who affirmed that chorea was most commonly accompanied by cardiac disease, and seemed to be a consequence of the rheumatic diathesis. It was soon found that the incidence of the disease, mainly on children of the female sex, corresponded exactly with that of rheumatism, and also with the excessive occurrence in them of endocarditis; and, hence, it was no longer possible to ignore the association of rheumatism and of endocarditis with chorea. Clinical and *post mortem* evidence also afforded unequivocal evidence of the embolic theory in certain cases, but not in all.

The difficulty now was to explain the occurrence of the disorder in cases presenting no signs of rheumatism, past or present, and, in particular, in such instances as were fairly attributable to eccentric causes, such as pregnancy, or to mental emotion, such as fright. This is the place to record the masterly studies of chorea by Hughlings Jackson and Broadbent, which have done so much to throw light upon this malady. Jackson supported and elaborated the embolic theory of Kirkes, believing that vascular obstruction led to congestion and damage of the corpus striatum, or its neighbourhood, and entailed an unstable condition of these parts. He also thought that this instability might be the result of other than gross mechanical causes, and might represent a neurotic state. Broadbent endeavoured to show that chorea was a symptom rather than a morbid entity, and due to changes in certain definite parts of the encephalon. He drew attention to the many points of parallelism between chorea and hemiplegia, suggesting that these two affections did but represent two different conditions and degrees of damage of the same nerve-centres, and believed that Kirkes' embolic theory was fitted to explain many cases. Like others, however, he showed that this theory was mainly proved in fatal cases, and could hardly be invoked in simple cases where there

was no cardiac damage, and where recovery followed removal of eccentric irritation. He therefore recognised other causes for the choreic symptoms, such as impaired vigour and increased irritability of the great ganglia, resulting, possibly, from prolonged arterial spasm, capillary thrombosis, and hyperæmia.

Dickinson's researches demonstrated the presence of hyperæmic foci, spread very generally over the brain and cord, with dilated arterioles and small hemorrhages, especially in the basal ganglia.

Broadbent affirmed that multiple symptoms in chorea were significant of multiple lesions, and hinted that to limit the area of localisation to the basal ganglia, or their vicinity, might be fallacious, since motor areas are now recognised in the cerebral cortex.

At the present time, we have the problem of chorea reduced to the following terms, in accordance with the most recent knowledge. 1. That the disorder is associated with rheumatism in a large proportion of instances. (This is denied by Dr. Sturges and others.) 2. That, consequently, it is also associated with endocarditis, and also often co-exists with signs of heart-disease, even if this be not clearly of rheumatic origin. 3. That the latter conditions predispose to the occurrence of embolisms, and certainly give rise to these in some cases. 4. That the condition of the motorial centres is one of debility and instability, however induced, whether mechanically or diathetically. 5. That, given this peculiar unstable condition, eccentric or other irritants and excitants may readily evoke the irregular modes of motion recognised as chorea.

Now, as in many other cases, where disputants take up opposing sides warmly, there is probably much to be said for both parties, and each is often found to espouse portions of truth. So here, we may, I think, find the truth respecting the origin of chorea to lie completely in no one of the theories I have enumerated. Taking chorea to be always symptomatic, we find it as a result of varying conditions affecting certain portions of the encephalon. Foremost amongst these, I am of opinion that we must recognise the close connection with all that is now signified by the rheumatic habit of body. I believe that closer study of all cases will bring many more of them into this category. The remainder will, I conceive, be found in connection with an ill-nourished and feebly developed nervous system, subjected to unwholesome influences, exhaustion, and unwise nurture. The connection of chorea with other spasmodic neuroses appears, as Dr. Barlow thinks, to be very little marked; and I agree with him. As a neurosis, it is, however, probably capable of heredity and transformation, as are other like conditions.

Let us inquire, next, what may be said respecting the evolution of chorea in members of a rheumatic family. The disorder may occur either during or after an attack of rheumatism; or, again, it may appear long antecedent to such an attack. Yet, again, it may occur in persons whose family only have had attacks of rheumatism. The seat of chorea is certainly in the nervous motor centres, and rheumatism is a disease particularly affecting motor structures—for example, the heart and the joints. The latter are believed to have their nutrient centres in the medulla oblongata, whence arise also the greater portion of the nerves innervating the heart. It is easy to suppose that a common kindred vulnerability, or susceptibility, in the great motor centres should predispose to one or other, or to both, of the disturbances known as chorea and rheumatism. This susceptibility, or instability, may therefore be a part of the diathetic proclivity of rheumatism; and, given this state, an overt rheumatic attack, or some other peripheral irritant, fright or emotion, intestinal or utero-ovarian irritation, may suffice to evoke the peculiar perverted action of the motor centres. I come now to declare my belief that it is in some such mode that chorea arises as a symptomatic disorder; that it is the outcome of a motorial neurosis. This is a speculative theory, I allow; but I think it is a helpful and suggestive view. It accords with much that is gaining acceptance in pathology at the present time, for the wheel has gone round again to neuro-pathology, and diseases of the blood are somewhat at a discount just now. It is perhaps not satisfactory in itself that opinions should thus oscillate, or that fashions should prevail in any science; but it is true, notwithstanding, that progress occurs with each turn of opinion; and, if we are really seeking what is true, no harm will accrue. The nervous theory of rheumatism itself is not new. Addison was wont to bring forward many facts in favour of its being primarily a disease of the nervous system, and my own belief is strongly set in this direction; for I accept the arguments in support of a basic arthritic diathesis, or potential state of certain nerve-centres, from which, under certain conditions, may be evolved either rheumatism or gout. But I none the less ac-

cept the associated humoral conditions, believing that these in themselves are, however, insufficient to explain all the phenomena of these states. I term these conditions, therefore, neuro-humoral. Let me ask you to discuss, as the outcome of this expression of my views, whether it is, or is not, probable that there is a peculiar basic or fundamental condition—a neurosis, if you will—which is capable of hereditary transmission or transformation, in the subjects of which may be set up at one time rheumatic, and at another choreic manifestations. This view will explain many cases of chorea, in which, even after skilful inquiry, no history of rheumatism, personal or family, can be obtained. The rheumatic evolution has not occurred; but the central motorial instability has given token of its presence in the form of chorea. The future life-history of some of these so-called non-rheumatic cases shows that rheumatic manifestations may supervene later, thus confirming the connection. If we accept this view, it is not difficult to conceive, further, that eccentric sources of irritation may excite chorea in subjects thus predisposed to motorial instability. Some light is also thrown by this theory on the value of arsenic in chorea. It is recognised as a most useful remedy in many cases of chronic rheumatism, promoting better nutrition and more stable condition of nerve-centres; and thus it counteracts the conditions on which these states mainly depend. Guided by this conception, you will not fail to understand the full value and importance of completing the inquiries already set forward on this subject by the Collective Investigation Committee of our Association, in whose behalf I appear before you on this occasion.

THE TREATMENT OF INTUSSUSCEPTION.

Read before the Medical Society of London.

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IN order to give precision to the data upon which the various modes of treatment adopted in intussusception are based, it is necessary to consider briefly certain points in connection with the anatomical and clinical forms of invagination, the morbid changes, the prospects of spontaneous cure, and the general mortality of the disease.

If all forms of intestinal obstruction—exclusive of those due to hernia and congenital malformation—be classed together, it will be found that the cases of intussusception will form 30 per cent. of the whole. The affection, therefore, may be considered to be a common one.

From an anatomical point of view, intussusceptions may be divided into four kinds—enteric, colic, ileo-cæcal, and ileo-colic. The relative frequency of these varieties may be represented respectively by the following proportions in every 100 cases of invagination—30, 18, 44, and 8. In the enteric form, the small intestine, and usually the lower jejunum, is alone involved, and the resulting tumours are generally small. In the colic, the colon is alone implicated, and most commonly that part to the left of the transverse colon. In the ileo-cæcal intussusception—the most frequent form of all—the terminal part of the ileum, carrying the ileo-cæcal valve at its apex, is turned into the cæcum; while, in the ileo-colic, the ileum is prolapsed through the valve, and following upon such prolapse is a secondary invagination of the cæcum into the colon. The colic form is, as a rule, chronic, while the remaining varieties usually assume an acute or subacute course.

From a clinical standpoint, intussusception may be separated into four classes: 1, the ultra-acute, in which death follows within twenty-four hours; 2, the acute, in which the disease runs its course in from two to seven days; 3, the subacute, in which the period is extended to between seven and thirty days; and, 4, the chronic, in which the duration of the malady has extended beyond thirty days. The first named variety is exceedingly rare, and would appear to be invariably fatal. Of the remaining varieties, out of every 100 cases about 48 will be acute, 34 subacute, and 18 chronic.

Intussusception is most commonly met with in children; and, indeed, no fewer than 50 per cent. of all cases occur under the age of ten years. It is among the young also that the acute forms are especially met with, while chronic cases are most frequent between the ages of 20 and 40.

With regard to the principal pathological changes in invagination, it may be pointed out that they tend to lead to two grave conditions—to obstruction of the bowel, and to strangulation of all that part involved in the intussusception. These two conditions need not coexist;

¹ Laycock held views much resembling these, and I imbibed them from his teaching. They have been elaborated and enlarged by others, notably and ably by Dr. T. J. MacLagan, and by Dr. Barlow.