REMARKS

ON

THE PATHOLOGY OF TABES IN RELATION TO
GENERAL PARALYSIS OF THE INSANE.*Made at the Opening of the Adjourned Discussion at the
Pathological Society of London.*

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MR. PRESIDENT AND GENTLEMEN,—I must first add my tribute to the recognition of the interest of the demonstration and the ability of the exposition we received from Dr. Mott at the last meeting, and of the proof it affords of the wisdom of the step taken by the London County Council, and of the appointment by which they completed it. I have little to criticise in the statements made by Dr. Mott, and much to endorse in the remarks of Dr. Buzzard. The question of the preponderant relation of tabes to syphilis has, I think, long passed into the region of admitted fact. I have published more extensive statistics on the point, but, for the sake of the discussion this evening, I have been through my notes of the last 100 consecutive cases of tabes seen in private, which constitute an unselected series, with this result: In 68 cases there was unquestionable evidence of past syphilis. In 12 per cent. the disease was probable; there was evidence of a chance of uncertain nature, making 80 per cent. of certain or probable syphilis. In the remaining 20 per cent. the disease was possible, that is, there had been exposure to the ordinary risks of infection, emphasised in most cases by the occurrence of gonorrhoea, often several times. In not one of the 100 cases could the disease be excluded by the absence of any exposure to risk of its contraction in the ordinary way. We all know by experience that it is only by this means we can actually exclude it, for all have met with such cases as those quoted by Dr. Mott from Dr. Radcliffe Crocker, in which the constitutional disease has occurred without any history of a primary sore. Two of the cases in this series would have gone into the "possible" class but for the facts that the wife of one suffered from syphilitic choroiditis, and a child of the other from the interstitial keratitis. All these cases were males. In the case of women the facts are obviously more difficult to obtain, but evidence of syphilis is, I think, clear in at least half the cases, and the remainder are for the most part married women in whom the disease notoriously often eludes recognition. It is unjustifiable to inflict on the husbands the distress of direct questions.

In juvenile tabes we seldom have doubt. The evidence of inherited syphilis is usually clear. It was so in about 6 cases I have seen, and I have met with one instance in which both father and son suffered from tabes, the former from acquired, the latter from inherited syphilis. Optic nerve atrophy is also sometimes seen in these cases of juvenile tabes. So also is reflex iridoplegia—the Argyll-Robertson symptom. With regard to this, Dr. Mott remarks that apart from tabes, it is only met with in cases of syphilitic brain disease, but I would remind him that, ten years ago, in some lectures on Syphilis and the Nervous System, I pointed out that this isolated loss of the light reflex is not rare in cases of constitutional syphilis apart from any other characteristic nervous symptoms. It is so in men, women, and children, and I would emphasise it, because my observations since have confirmed me in the opinion that it is a diagnostic symptom of great weight and often very opportune.

Few of us are in a position to criticise the facts which Dr. Mott has given us regarding general paralysis and its relation to tabes. My own experience emphatically confirms the statement by Dr. Buzzard that when the symptoms of the two diseases are associated, those of tabes precede those of general paralysis. I have seen a large number of cases in which the latter supervened on the former; I do not recall any case in which the paralytic symptoms led the way and the knee-jerks afterwards became lost. The common rule in cases of this class is for the legs to remain normal, or more commonly for an excessive knee-jerk and foot-clonus to accompany progressive loss of power. In the tabetic cases a history of syphilis

is, I think, as common, and its presumption as constant, as in the pure cases of tabes; but I do not think that this statement can be made of the paralytic class, although the facts are not easy to ascertain. Of the juvenile cases of general paralysis described by Dr. Mott I have had no experience. The only cases of the kind I remember have been cases of arrest and irregular impairment of cerebral function, not progressive, associated with the inherited disease, but most readily explained by actual specific disease of the brain.

The pathological questions I must, with Dr. Buzzard, leave to those with whom remains the opportunity for elaborate investigation. Dr. Alexander Bruce does not, perhaps, remember how strenuous, 25 years ago, was the advocacy of the dependence of the changes in the posterior columns on the meningitis over them, and on the interstitial and perivascular alterations, although not precisely in the minute form which he suggests. The theory disappeared for a reason which I think still holds good. These extra-neural alterations are inconstant, especially in degree, and they can be equally explained by the secondary neuroglial and vascular alterations which occur whenever there is a primary degeneration of the nerve elements; for example, in that which is secondary to a traumatic process or focal myelitis. On the other hand, the theory failed entirely to account for the most remarkable fact of tabes, the restriction of the symptoms to function, the manner in which, in the slighter cases, without cutaneous sensory disturbance, there was only degeneration of the fibres of the posterior median columns, which we have learned to regard as the upward path from the afferent muscle nerves, and the frequent, almost usual, loss of sensation to pain, it may be almost complete throughout the legs, with no impairment to sensibility to touch. There may be, though this is indeed a rare condition, loss of sensation to touch only, limited to the soles of the feet. We cannot explain such a functional limitation by a process essentially random in incidence. All the teaching of our pathological knowledge since has been that such restricted impairment is the result of the action of a toxic blood state on the nerve elements themselves. The difference in their function involves variations in their susceptibility, by which slight differences in the nature of the toxic agent, perhaps inconceivably minute, involve quite different effects in the symptoms produced.

We must remember, moreover, that in the case of other toxic agents the chief effects are produced on the extremities of the nerves, lessening in degree as we approach the centres. I think, therefore, that we need not be surprised at the slowness of the changes that can be perceived in the cells of the posterior ganglia. If the fibres which conduct from the ganglia are degenerated more completely—may not this be in accordance with a law which we cannot yet understand? We must expect to meet with facts we cannot yet explain.

The question of the identity of tabes and general paralysis as one disease, due to the same cause, acting on cerebral and spinal neurons, seems to me very much a question of words. Does even common causation create identity of diseases which differ so widely in symptoms as the cerebral form of general paralysis and the pure form of tabes? Does the fact of blending forms, and cases with common symptoms, create identity when there is a complete contrast between the extreme forms? If so, we ought to regard a scrofulous tumour of the brain and pulmonary phthisis as one disease, or even erythema nodosum and the acute articular rheumatism which may coincide with it. But would this correspond with our common use of the term "disease," which is connected with a definite aggregation of symptoms rather than with their discerned causation? This statement may, however, be regarded by Dr. Mott as handing to him the point at issue. It does, I think, so far as concerns the cases of tabetic general paralysis, but the cases of the paralytic form seem open to more question, which I must leave to Dr. Savage to solve. I would point out how imperfect is our present nomenclature by which we have to speak of tabetic and paralytic general paralysis. The German term, "paralytic dementia," has never obtained a foothold here, the word "dementia" having different associations. I do not know that any other word, such as "parementia," would be appreciably better.

The final question put by Dr. Mott raises a problem of great importance on which I have long held a decided opinion.

Dr. Mott asks whether the toxic agent "does not lower the durability of the nerve elements, so that some other factor" induces a premature decay. I have long maintained that this is an essential secret of the mysterious incidence of these degenerative diseases. An average man inherits an equal vitality in all his structures. He goes to pieces at last like the famous "one-hoss shay," all at once, in mature, equable, senile decay. At the other extreme are those individuals who possess structural elements (it may be in some part of the nervous or the muscular system) so deficient in vital endurance that they decay as soon as, or soon after, complete development is achieved.

Such are some cases of idiopathic muscular atrophy, of congenital atrophy of the optic nerves, in which sight fails in a whole family soon after puberty, or of "hereditary ataxy,"—"Friedreich's disease"—in which certain structures of the nervous system decay soon after adult age is reached. In other cases the defect in vital endurance is only sufficient to render some structures less resistant than others, to cause, for instance, the motor nerve structures to decay before the others under the influence of grief, or the nutrition of the cerebral structures to give way under the strain of work or anxiety. This disposition to decay, sometimes inherent, strongly or slightly, seems to be induced artificially by toxic and perhaps other influences. Their effect is specific in many cases, probably in more than we can discern. They lead to failure of nutrition, decay, degeneration. It may seem spontaneous, or be evidently excited. It may occur soon, a few years after its cause has seemed active, or the effect may be only manifest so long afterwards that the connection may be only perceptible by these considerations. Thus tabes may be a post-syphilitic disorder in middle life, or only, yet with the same relation, when the general failure of approaching age obscures the significance of the cause. We shall understand many things more clearly if we perceive that acquired influences may have the effects of congenital dispositions, that either may cause effects unaided, and may predispose to the results of influences that would otherwise seem to act alone. These considerations are, I fear, somewhat beyond the question before us, but they are not, I think, beyond their indications.

ON "ALBUMINURIA IN THE APPARENTLY HEALTHY."

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It has been known for some twenty-five years that albumen may be found in the urine of persons who are apparently in good health. During the last twenty years a large number of facts relating to this albuminuria have been accumulated. Theories as to its causation and meaning have been put forward. But very little advance has been made towards answering the important questions which must occur to anyone who has a case of this kind before him. We want to know: What is the subsequent history of the individual in whom this albuminuria is found? What is its effect on the duration of life? Does the albuminuria disappear? Does the patient develop true renal disease at a later period? Or is he a weakly individual who succumbs to some other ailment before his expectation of life has been realised? These are old questions, but a definite answer based on actual experience is still wanting.

DEFINITION.

Before discussing the matter it is necessary to have a clear idea as to the meaning of this term "albuminuria in the apparently healthy." There are two points to be noted. In the first place, in using this term we must of course rigidly exclude all cases of albuminuria in which there is the possibility of suspicion that any true organic change in the kidneys may be present. And in this respect care must be taken to eliminate more particularly the early stage of granular kidney, in which albuminuria is often slight and inconstant. We must also exclude the albuminuria which is associated with morbid states of the blood, such as anæmia, and with the puerperal condition. We have nothing to do with the

albuminuria which may be attributed to congestion of the kidney, such as results from cardiac disease or cardiac failure from any cause. And we have nothing to do with the toxic albuminuria which occurs in the specific or bacterial diseases. We are left to consider albuminuria in individuals who either are in perfect health, or at any rate present no sign of disease to which that albuminuria may reasonably be attributed. In the second place, it is necessary to understand in what sense the word "albuminuria" is used. It is used in a clinical sense, and not in a laboratory sense. There are some delicate tests for proteids, which give a reaction in nearly all specimens of urine, so that from the laboratory point of view albuminuria is the rule rather than the exception. Even the picric acid test, which reveals a percentage of albumen of 0.0005, may be said to err on the side of delicacy, and it is said to show a trace of proteid matter in some 30 per cent. of urines examined. For practical clinical purposes it is sufficient to employ the two common tests—namely, the boiling of acid urine, which shows a proteid percentage of 0.0003, and the addition of nitric acid, which shows 0.003 per cent. By albuminuria, then we mean the presence in the urine of such an amount of albumen as is revealed by these two tests.

CLASSIFICATION OF CASES.

The next step is to consider under what conditions this "albuminuria in the apparently healthy" is met with. Without necessarily covering the whole ground, there are some four or five such conditions, and a provisional classification may be based on them. But it is unlikely that such a classification will prove a true and permanent one, because it is by no means certain that all these conditions stand to the albuminuria as cause and effect, and it is not improbable that some one primary defect underlies them all.

"EXERTION ALBUMINURIA."

The first class, in which the albuminuria is clearly dependent on "muscular exertion," is easily studied, as it is of very common occurrence. This exertion albuminuria is found in the healthiest and strongest individuals. One of the earliest observations was that of Leube, who found albumen in 19 out of 119 soldiers after a long march, that is, in 16 per cent. He found no casts. The same phenomenon has been shown to occur in boys at school. Numerous other observations of the same kind might be quoted, especially those of Grainger Stewart. This exertion albuminuria is very common, and it may be that after sufficiently severe and prolonged exertion it is universal. I may mention an instance which confirms this. Dr. Jenner, Superintendent of the Clinical Laboratory of St. Thomas's Hospital, examined the urine of some of the members of the Hospital Rugby Football Team, namely, nine forwards and one half-back, immediately after the Final Cup Tie three years ago. Albumen was present in every one of them, and, what is an important fact, hyaline casts were abundant in the urine of all except one. All these men were apparently in perfect health and training. In another case he found albumen and numerous hyaline casts in the urine of a student after a long cross-country run. Under this heading it may be noted that the urine sometimes becomes albuminous after a long series of epileptic fits. It is also worth while noting that in one recorded case, where albuminuria always occurred after slight exercise, massage was followed by no such effect; and it may be shown that the chief factor in the production of this form of albuminuria is the prolonged muscular contraction and consequent rise of blood pressure.

NEUROTIC ALBUMINURIA.

Of the next class our knowledge is less precise. It has been termed "nervous" or "neurotic" albuminuria. There is some evidence to show that transient and slight albuminuria may follow prolonged overwork and mental strain or anxiety. It is possible that the transient albuminuria which has been noted in exophthalmic goitre may come under this head. A third class is formed by individuals who present transient albuminuria after a cold bath, but this class is small and unimportant.

"DIETETIC ALBUMINURIA."

A fourth class comprises cases in which a slight transient albuminuria is apparently induced either (1) by the ingestion of large quantities of proteid material, such as cheese and