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."

By HERBERT P. HAWKINS, M.D., F.R.C.P.,
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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

eggs; or (2) by the taking of food of any kind. The first of these classes undoubtedly exists, but, as regards the second class, where the mere taking of a meal apparently produces albuminuria, more doubt exists, because the meal at which observations have been chiefly made is breakfast, and here the influence of position of body and rising from bed has not always been eliminated. The conjunction of albuminuria with chronic dyspepsia (and often oxaluria) hardly comes under our definition of albuminuria in the apparently healthy. These four classes of albuminuria are easily recognised with a little care. In each of them there is some one definite cause, which is clearly removable, and I doubt if they need be looked upon as more than trivial departures from a healthy condition.

"POSTURAL ALBUMINURIA."

Finally we come to the class to which I believe most instances of albuminuria in the apparently healthy belong. It is sometimes spoken of as "postural" on the ground that it is largely influenced by the position of the body. It is also called "cyclic" from the fact that the daily appearance of albumen shows a certain definite rhythm or periodicity. This class probably also includes the majority of cases which have been termed "albuminuria of adolescents." I think it is the most common form. I have watched 6 such cases over varying periods up to seven years, and during the same period I have come across no case that I could reasonably classify under any of the other heads, excepting, of course, exertion albuminuria. It is of this class that the greatest doubt as to prognosis exists.

We have a certain number of facts concerning this form of albuminuria. It is most commonly met with in young people, but it may certainly be found up to the age of 50, though at such an age great care must be exercised in distinguishing it from an early stage of granular kidney. But the age at which it commences and the absolute frequency of its occurrence are unknown, because the condition is nearly always discovered accidentally, either as a result of examination for insurance, or by examination of the urine as a matter of routine in some illness. For the same reason our information as regards sex is imperfect. It is apparently far more common in males, but females seldom present themselves for insurance, and there is generally more difficulty in obtaining a specimen of their urine for examination. The time of day at which the albumen appears in the urine is constant. It is not present in the urine passed immediately on rising from bed in the morning. It is present in the after-breakfast specimen. It may not be found again in the course of the day, but in many cases it is present after the midday meal. It is nearly always absent in the evening, when the day's work is done. The albumen in these cases is always small in amount, being seldom a measurable quantity, sometimes, however, being one-tenth or one-eighth, according to the usual rough clinical method of estimation. In all other respects the urine is healthy, as regards quantity, colour, specific gravity, and amount of urea. In some cases hyaline casts are found, especially after centrifugalising. The general health is commonly perfect, and there is no evidence of any affection of heart or arteries, and there is no increase in the arterial tension as estimated in the radial artery. As regards the family history of these cases no constant factor can be discovered, though, by some, gout is supposed to play a part. A very important matter is the fact that this condition may be found to exist in more than one member of a family. In one recorded case three out of five children in one family always showed albuminuria for three hours after breakfast, and these children were carefully watched for seven years without the appearance of any renal or other disease, and without any sign of ill-health.

Before discussing the prognosis of such cases it is necessary to agree upon some explanation of this albuminuria. The central fact on which an explanation is based is the constancy in the time of day at which the albumen appears. It is a morning albuminuria. This constancy might conceivably be explained either by the rising from bed and assumption of the erect position, or by the cessation of sleep, or by the taking of breakfast after some hours of fasting, or by the taking of the matutinal cold bath. Of these possible factors the evidence points to the first, and I think it can be shown that this morning albuminuria is dependent on the rising up, the standing

and moving about during the early part of the day, and that it is not influenced by the taking of food or of a cold bath. In confirmation of this, I may mention a well-marked case of this kind of albuminuria, where the condition had been present for two years. It was discovered as usual quite accidentally; as a matter of fact, the patient examined his own urine. I induced him to stay in bed for eighteen days, thinking that perhaps the long rest would lead to the total disappearance of the albuminuria. It had no such effect, and the albuminuria has now continued for another three years since then, making five years in all, without any impairment of health. But though the rest produced no permanent effect, it had considerable temporary influence. During the eighteen days which he spent in bed, at the very time at which the albuminuria is most constant, namely, about an hour after breakfast, on eight days no albumen appeared, and on ten days there was only a minute trace. It must be noted here that although the patient was kept in bed, yet a certain amount of movement of the body was entailed by the making of a toilet and the sitting up to take breakfast in bed, so that a condition of absolute rest was not strictly maintained. It was further found that the character of the breakfast and the taking of a cold bath had no obvious effect. The urine was examined also one hour after the midday meal, and although under the usual habits albumen was known to be commonly present at this hour, during these eighteen days spent in bed it was present on one occasion only, and then only as a minute trace. The effect of the rest in a recumbent position may be seen in this, but it is still more manifest in the evening record. Late in the evening the urine is almost always free from albumen, and on twelve of these eighteen days of rest no albumen was found. But on six evenings not taken consecutively, but at random, he was allowed up and about for two hours, and on every one of these occasions the urine passed at the expiration of this period of movement was found to contain albumen, and the amount of albumen was, moreover, greater than on any other occasion during this period. No doubt similar observations have been recorded. I think there is good evidence for believing that the one great factor which influences the albuminuria in the majority of these cases is the position and movement of the body.

It is possible to carry the explanation a step further. Our knowledge of the physiology of the secretion of urine, and the facts that we have at our disposal relative to the morbid conditions under which albumen appears in the urine, indicate that the escape of albumen occurs in the glomeruli. Further, we may believe that the process of secretion of urine in the glomeruli is not merely a matter of filtration, which would vary directly with the blood pressure in the glomerular capillaries, but that it is a process of filtration checked and controlled by the activity of the flat epithelium which covers the capillary loops. In other words, in order to understand the mechanism of albuminuria two elements must be considered—the pressure in the glomerular capillaries and the state of their epithelial covering. One of these two elements must be primarily at fault in this form of albuminuria. Now there is evidence to show that the activity and integrity of the epithelium is dependent on the maintenance of a certain normal blood pressure in the capillaries, and that if this pressure is disturbed, either raised or lowered, for even a very short period of time, the epithelium quickly responds by a failure to perform its selective function with efficiency and albumen appears in the urine. For example it can be shown experimentally that if the arterial supply to the glomeruli is cut off or diminished for a few hours, on restoring the blood current, and blood pressure to the normal, the first urine secreted is albuminous. On the other hand, we know that a rise of blood pressure in the glomeruli, such as is produced by venous stagnation, is also attended by the escape of albumen, and that this is not a mere matter of altered filtration under a higher pressure, but that damage inflicted upon the epithelium also plays a part is shown by the fact that the amount of urine under this condition is diminished and not increased.

Now, in this condition of albuminuria in the apparently healthy, in exertion albuminuria, in so-called neurotic albuminuria, in albuminuria dependent on cold bathing, all our evidence tends to indicate: (1) That the immediate cause of the escape of albumen is temporary impairment in the efficiency of the epithelium of the glomeruli; but (2) that this

impairment in epithelial efficiency is itself the result of temporary alterations in the blood pressure in the glomerular capillaries. In other words, the actual seat of the trouble appears to be in the vasomotor mechanism which regulates the blood flow into and out of the glomeruli, and not in the kidney structure itself. This is the view which seems to be pretty generally held at the present time. It is supported by the periodicity and transience of the albuminuria, by its continuance over many years without increase and without impairment of health, by its close connection with movement and exertion, and by its disappearance on resting. All these facts would be difficult to explain on any theory involving a primary defect in the epithelium.

If, then, it is accepted that this albuminuria is due to a defect—either a sluggishness or an undue excitability (perhaps congenital) in the vasomotor regulation of the glomerular blood pressure, we are in a somewhat better position to discuss the question of prognosis. But most diverse opinions are still met with. Some are sanguine enough to dismiss it by attaching to the condition such terms as “physiological” or “functional.” Others, with some reason, have suspected that it tends to end in true organic change in the kidneys.

At present we have no certain knowledge of the subsequent history of these individuals, and consequently a patient, if he makes a round of doctors, as he frequently does, becomes the possessor of a number of different opinions. The answer can, I think, only come from those who are so placed in practice as to be able to watch such cases through a lifetime; it cannot come from hospital practice. We know that the condition continues for many years without increase and without impairment of health; on the other hand, it is doubtful if in any clear case of the kind it has been known to disappear. Though the vasomotor defect which probably lies at the bottom of it is a small matter in itself, one may be justified in feeling dubious as to its total result after a lapse of twenty years. And I believe that it is one's duty, while reassuring the patient, to warn him that he must lead in all respects the healthiest life he can compass, and he should have his urine examined three or four times a year. In insurance work I am inclined to accept such an individual only with a heavy addition, acting so rather for the protection of other policyholders than as expressing any conviction that he will not live out his natural term of years.

TREATMENT.

As regards the treatment of apparently healthy people whose urine is albuminous, there is little to be said. On coming across one of these cases in whom the albuminuria is unexpectedly and accidentally found, the first duty is to make certain that there is no actual disease of the kidney or elsewhere. In the next place, by observation under varying circumstances, it will be possible to determine to which of the conditions mentioned above the appearance of albumen in the urine may be attributed. In the cases of exertion albuminuria, of neurotic and dietetic albuminuria, and of the form which is dependent on cold bathing, the apparent cause can be removed. In the far more common case of morning albuminuria dependent on position of the body, there is no indication for special treatment beyond the removal of anxiety from the mind of the patient and the enjoining of a perfectly healthy life. There is no reason for believing that medicinal treatment is either necessary or useful.

ON LOSS OF THE STEREOGNOSTIC SENSE.

By R. T. WILLIAMSON, M.D.LOND., M.R.C.P.,

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CASES of cerebral disease are occasionally met with in which there is no loss of sensation to tactile impressions and yet the patient is unable to tell the nature of objects placed in the hand when the eyes are closed. A coin or other familiar object placed in the affected hand is felt, but (the eyes being closed) the patient fails to recognise what it is. This condition has been described as “touch paralysis” or loss of the stereognostic sense. Under the former description I recorded 2 cases the BRITISH MEDICAL JOURNAL in 1897; 2 other cases I

have recorded since,² and the following are the notes on a fifth case.

Left-sided Facial Paresis: Loss of the Stereognostic Sense and of the Sense of Pressure and Position in the Left Hand: Other Forms of Sensation Normal.—A. M., aged 44, came under my care at the Ancoats Hospital, October 24th, 1899, on account of inability to recognise objects (especially coins in his pocket) with the left hand.

History.

Three months previously he had suffered from severe pain in the head, chiefly on the right side, just behind and below the parietal eminence. It was worse at night, and kept the patient awake. At the end of two weeks the pain disappeared, but he began to be troubled with attacks of temporary failure of vision. The vision became dim or lost for five or ten minutes, but there was no loss of consciousness, no pain, no faintness, and no convulsions. He had about a dozen such attacks in four weeks' time. The attacks then ceased, and he first noticed inability to recognise the nature of objects with the left hand. He also experienced difficulty in buttoning his clothes, and in handling small objects with his left hand. Then he noticed slight clumsiness in the movements of the left leg, and afterwards, about a week before admission to the hospital, he experienced difficulty in spitting, and observed a difference in the appearances of the two sides of the face. History of gonorrhoea twelve years ago. No history of chancre or secondary syphilis. Vision greatly impaired in left eye owing to opacity of the lens and choroiditis, the result of injury in childhood.

Condition on Admission.

There was paresis of left side of the face. The nasolabial fold was less distinct on the left side than the right. This difference was increased when the patient was asked to show his teeth; it was then observed that the angle of the mouth was not retracted so much, and the upper lip was not elevated so much on the left side as on the right. The patient could not close the left eye so tightly as the right. The tongue was protruded straight; there was no paralysis of the ocular muscles (there was an old internal strabismus of the left eye dating from childhood). The right pupil was a little larger than the left; both reacted to light and accommodation. The optic disc and retina were normal on the right side; the right field of vision was also normal. The fifth and eighth cranial nerves were not affected. There was no motor aphasia, no word blindness, no word deafness. The patient could perform all the movements of the arms and legs, and was able to walk quite well. The left knee-jerk was more active than the right; there was no ankle clonus; the plantar reflexes were present. He complained only of clumsiness in the movements of the fingers of the left hand and of difficulty in recognising objects with the left hand without the aid of vision. There was no wasting of the muscles of the left hand or arm; all the movements of the fingers and arm could be performed. The grasp was unimpaired, and he could lift a heavy bucket of coals with the left hand when once he had obtained a good grasp of the bucket handle, but he had great difficulty in buttoning his clothes with his left hand. Also he showed a little unsteadiness in the movements of his left hand when he attempted to touch the tip of his nose with the index finger, or when he grasped any small object.

Sensation to tactile impressions, pain, and temperature, and the power of localising tactile impressions were normal on the left hand (as well as on other parts of the body). Though objects placed in the left hand (when the eyes were closed) were felt, their nature was not recognised; whilst they were readily recognised in the right hand. The following objects were employed in testing the sensation: Coins, keys, a watch, wood cube, ball, domino, small box, etc. Usually he had no notion of the shape or hardness of these objects when placed in the left hand (the eyes being closed) even after careful handling. Occasionally he had some knowledge of the shape and hardness, but was unable to say what the object was. Many objects, however, were recognised readily when simply placed in contact with the outstretched fingers of the right hand.

He was unable to tell the nature of passive movements and the position of the fingers and of the hand on the left side, the eye being closed; but he could tell the nature of passive movements at the left elbow and shoulder quite well, and also the nature of passive movements of the fingers, hand, and arm on the right side, and of the legs on both sides.

The sense of pressure, as tested by a pill box containing varying quantities of lead pellets, was completely lost in the left hand and arm, but it was normal on the right arm and on both legs, and both sides of the chest and face.

The apex of the heart was just outside the nipple line in the fifth space; the second sound was accentuated, the first sound was abrupt. The pulse tension was a little increased. The urine had a specific gravity of 1.020; albumen and sugar were absent.

On November 6th the patient became suddenly blind in the right eye, and ophthalmoscopic examination revealed the appearances which are seen in sudden obstruction or embolism of the central artery of the retina.

The following is a brief summary of the symptoms in four cases which I have previously recorded:

CASE II.—A. B., female, aged 26; syphilitic history, headache, double optic neuritis. Convulsions commencing with numbness and twitching of the left thumb. No paralysis of arms or legs; slight temporary left-sided facial paresis. Gradual recovery, but the patient became blind, owing to secondary optic atrophy, and the stereognostic sense was lost in the left hand. She was able to feel and localise tactile impression on the left hand correctly; she could feel painful impressions and distinguish between warm and cold objects quite well; she was able to tell the direction of passive movements of the fingers, hand, and arm on the left side, and was able to touch tip of the nose with the left index finger without inco-ordination when the eyes were closed. Sense of pressure not affected. Tenderness on deep percussion of the skull at one spot near the parietal eminence on the right side.

CASE III.—Male, aged 25; sudden onset of paresis and “stiff sensation” in the right arm and leg. Gradual improvement. Two years and a-half later all the coarse movements of arms and legs could be performed quite well. Grasp of right hand equal to that of left, but difficulty in writing and in buttoning his clothes with the right hand. Slight inco-ordination