An Address

ON THE

DIAGNOSIS OF CERTAIN FORMS OF RENAL DISEASE.

DELIVERED BEFORE THE RICHMOND DIVISION OF THE METROPOLITAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

By J. ROSE BRADFORD, M.D., F.R.C.P.,

PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL.

MR. PRESIDENT AND GENTLEMEN,—In selecting a subject on which to address you this evening I have chosen one dealing with common affections, as the problems connected with these are really of more importance than questions concerning rare diseases, and, as it has been well said, anomalous forms of common diseases are really much more common than rare diseases. During recent years our views have changed considerably, both as regards the nature of various renal diseases, and more especially as regards the treatment of the more common forms of the inflammatory diseases of the kidneys.

I propose to address you to-night on the diagnosis of certain forms of renal disease, and also on the principles of treatment of Bright's disease.

The diagnosis of renal disease depends very largely, as is well known, on the examination of the urine, and only too often the mistake is made of arriving at a diagnosis merely on the result of the examination of the urine. Nothing is more common than to diagnose nephritis, both in its acute and chronic form, merely on the strength of the presence of blood or of albumen in the urine, and at the very outset of the subject stress should be laid on the importance of never diagnosing the presence of serious renal disease merely on the strength of urinary examination. This should be always controlled by a clinical examination of the patient, with especial reference to the state of the cardio-vascular system and the general nutrition. Not only is this true, but the converse is also true, that renal disease should not be excluded on the strength of a single examination of the urine showing the absence of albumen. In some of the gravest forms of renal disease albumen may be absent from the urine, at any rate temporarily.

FUNCTIONAL ALBUMINURIA.

The importance of observing these rules is well illustrated by the study of the condition known as functional, cyclical, or physiological albuminuria. This is an affection of great interest and of considerable importance to the practitioner, and one about which our views are still much less definite than is desirable. Physiological albuminuria is one of the forms of albuminuria that may be present in the apparently healthy, but it is well to bear in mind that albuminuria due to serious renal disease may also present itself in persons who are apparently healthy, so that this characteristic alone is not sufficient for purposes of definition, and some other must be sought. Various authorities have described this albuminuria as resulting not only from exercise, but also from diet, from cold bathing, etc.

A more extended study of the condition has shown that the most constant factor in the production of the albuminuria is the effect of posture, and hence of recent years the term "postural" or "orthostatic albuminuria" has been applied to the condition. It has been shown that the influence of diet is often extremely small, and that not uncommonly the most rigid diet fails to produce any diminution in the albumen. On the other hand, posture produces the most marked effects, and so long as the patient remains in the recumbent position the urine remains free from albumen. Thus the urine secreted in the night, and passed the first thing in the morning on rising, is free from albumen, but even after a few minutes of assuming the erect posture the urine becomes albuminous, and remains so throughout the day until the late afternoon, when not commonly it may diminish or disappear. The assumption of the recumbent posture during the day leads to the speedy disappearance

of the albumen, and this remains absent so long as the patient is kept in bed.

One of the most interesting points with reference to this condition is the recent observation that the occurrence of such a febrile disease as scarlet fever does not necessarily lead to albuminuria when the disease affects individuals suffering from postural albuminuria. So long as they are confined to bed, notwithstanding the presence of pyrexia, no albuminuria results. Modern investigation has shown, then, that posture, or the assumption of the erect attitude, has more to do with the development of physiological albuminuria than diet or bathing. Exercise, however, undoubtedly increases albuminuria, and it has been shown that albumen may appear in the urine as the result of severe exercise in a large number of individuals—for example, soldiers who are not only healthy but who do not suffer from postural or functional albuminuria, so that both posture and severe exercise must be looked on as possible causes of albuminuria in the apparently healthy.

Variations in the amount of albumen dependent on posture may, however, occur in renal disease as well as in the so-called functional albuminuria, and one of the most important discoveries of recent years is unquestionably the fact that in the granular kidney the albuminuria may be of the postural variety. This is a point of great clinical importance inasmuch as the postural characteristic—namely, the absence of albumen in the morning urine—has been looked upon by many as an absolute criterion of functional disease.

Not only may the albuminuria of the granular kidney be affected by posture but the same characteristic may be seen during the convalescence from nephritis. Under these circumstances the albuminuria during the height of the nephritis is doubtless abundant and persistent; but as convalescence sets in and the nephritis subsides, the albuminuria becomes first of all less abundant, and later on may be only present when the patient assumes the erect posture. Finally, it disappears altogether. The fact that albuminuria may be of the postural variety when dependent on such gross organic disease as the granular kidney or during a subsiding nephritis led, of course, to the view that in many cases of functional albuminuria the albuminuria was really due to a former attack of nephritis, which was either subsiding or else had left a slight lesion of the renal structures. This explanation is, however, probably untenable, inasmuch as in many cases of functional albuminuria no history of any infection may be obtained.

We see from these considerations that, however characteristic of functional albuminuria the effects of posture may be, these results are also seen in certain organic diseases, but that, on the other hand, there is no evidence to support the statement that all cases of functional albuminuria are due to some slight non-progressive renal lesion the result of some former infection.

After all, the main interest of functional albuminuria, apart from a discussion of its etiology, lies in the errors of diagnosis which are liable to be made. As we have already seen, the postural characteristic may be present in cases of subsiding nephritis and in some cases of granular kidney, and it is well to remember that granular kidney, even of a severe type, may be sometimes seen in quite young patients.

Functional albuminuria may, however, not only be confounded with subsiding or slight nephritis and with granular kidney, but there are at least two other affections that may be confused with it. The diagnosis of functional albuminuria, strange as it may seem, has been made more than once where the real underlying condition was a renal calculus, and where the patient has either not suffered much pain or the history of pain has not been elicited. This is a mistake in diagnosis that would scarcely seem possible, and probably would not be made if a very thorough examination of the urine were performed. It is not unusual for a trace of blood or of pus to be overlooked, and only the albumen recognized that is really dependent on the presence of the blood or pus. At any rate, explain it as we may, it is not very uncommon for such an error of diagnosis to be made.

The other condition with which functional albuminuria may be confounded is movable kidney, or, to put the matter in another way, some cases of movable kidney may present themselves as cases of functional

albuminuria. In fact some writers have supposed that movable kidney is one of the most frequent causes of this form of albuminuria, and that the characteristic effect of posture is to be explained by dislocation of the organ with kinking of the renal vein produced by the erect posture. A number of observations have been made tending to show that the activities of the kidney are more marked during rest and in the recumbent posture, and this has been attributed to the greater freedom of the circulation through the kidney in this position. Where the organ is movable it has been supposed that the upright posture may lead to slight kinking of the renal vein, and thus cause the albuminuria. Whether movable kidney is to be regarded as a cause of most cases of functional albuminuria is much open to question, and personally I think this view is quite untenable, but, on the other hand, it is very probable that some cases may be so produced. This condition affords a ready explanation of the subsidence of the albuminuria with rest. At any rate, the association is of sufficient importance for the condition to be searched for where functional albuminuria is present. There is really at the present time no completely satisfactory explanation of functional albuminuria, and there can be little doubt that different cases are due to different causes. Some may result, as already pointed out, from the presence of a slight renal lesion; others may perhaps be due to undue mobility of the organ; a very considerable number are dependent on the urine containing other elements, such as pus cells, and especially spermatozoa—and it is probable that more cases are dependent on this last condition than is sometimes thought; but a certain number remain over which cannot be explained in any of these ways, and we are driven to suppose that this must be dependent on some vasomotor disturbance of the renal circulation. In some of the cases other vasomotor disturbances, such as cold extremities and tendency to faintness, may be present, and this faintness may very probably also be of vasomotorial origin, and dependent on lack of control of the nervous system over the vessels of the abdominal area.

NEPHRITIS.

Having considered some of the points in relation to functional albuminuria, I will now pass on to the consideration of certain facts in connexion with nephritis. The classification of nephritis is well known to be difficult, and many diverging views are held, but at any rate two marked forms of acute nephritis may be recognized, in one of which dropsy is well marked and in the other dropsy is absent, although the urinary changes are well marked, and it is not uncommon even for the changes in the urine to be more severe in the latter type of case than in the former.

Much confusion is produced owing to the fact that the same causes, or at any rate similar causes, may give rise to both varieties of nephritis. Thus in scarlet fever, for instance, the onset of nephritis may reveal itself by the suppression in the flow of urine or the secretion of a scanty, highly blood-stained urine without the development of any dropsy. Or, on the other hand, dropsy may occur with the secretion of a highly albuminous urine containing little or no blood. The dropsy cannot be correlated with the mere severity of the urinary changes, as certainly some of the most severe forms of nephritis due to scarlet fever and other cases are not necessarily accompanied by dropsy.

The recognition of these two forms of nephritis is important from the point of view not only of diagnosis but also of prognosis, since, speaking generally, there can be little doubt that the outlook, or at any rate the ultimate outlook, is more grave in cases accompanied by dropsy than where this is absent. From the point of view of diagnosis, it must be borne in mind that a scanty blood-stained urine may arise from other causes than the onset of acute nephritis; thus, for example, it may be due to extreme passive congestion of the kidneys, as in some cases of heart disease, and it may also arise suddenly as a result of infarction, and under these circumstances is peculiarly liable to be erroneously diagnosed as acute nephritis. In many cases of infarction no haematuria is present, the only urinary change being the development of sudden and copious albuminuria, and this may also lead to the suspicion that nephritis is present when such is not the case.

Infarction is characterized not only by the sudden onset of haematuria and albuminuria, but also by its equally sudden subsidence, and in this way the diagnosis can often be made. Pain is by no means so frequently present as was at one time thought, and in the diagnosis of infarction the sudden onset of the renal changes without the development of dropsy, and their equally sudden subsidence, are really the main points of importance.

The dropsy of acute renal disease is not only characterized by its well-known distribution and its tendency to involve the looser subcutaneous tissue, but also by its great persistence, and not uncommonly its severity. It is uncommon for the patient with cardiac disease to be so dropsical as to become unrecognizable, but this frequently occurs in renal disease. Further, the dropsy of renal disease is very difficult to treat and is much more persistent and refractory than the dropsy of heart disease.

Apart from these considerations, there is another characteristic of renal dropsy that is of very great practical importance. Renal dropsy never involves the subcutaneous tissues to a high degree without at the same time affecting the serous cavities, and especially the solid organs. It is, perhaps, more difficult to detect traces of dropsy in the serous cavities and solid organs than in the subcutaneous tissue, and hence these internal dropsies are no doubt frequently overlooked.

Pulmonary oedema is especially of importance, owing to the frequency of its occurrence and owing to its danger. Renal dropsy is more likely to lead to pulmonary oedema than to oedema of any other solid organ, although oedema of the brain and of the larynx sometimes occurs. Pulmonary oedema not only occurs in association with renal dropsy, but also in cases of renal disease where uraemia is present and where dropsy may be absent. In fact, the frequency of the involvement of the lungs is most characteristic of renal disease. The oedema usually affects the lungs, generally from base to apex, but it is not uncommon for the condition to be more marked at the apex than at the base, and cases may be seen where the degree of the involvement of the apex is such as to produce actual dullness on percussion as well as the well-known rales and crepitations associated with this condition.

Pulmonary oedema when at all well marked or extensive reveals its presence not only by signs but also by symptoms, and a peculiar hissing respiration is often present. In higher degrees of the affection the dyspnoea may, of course, be extremely urgent, and this is especially the case where the oedema involves the larynx as well as the pulmonary tissue.

The natural tendency of acute nephritis is undoubtedly to subside and ultimately to clear up, although, of course, death occurs in a certain number of cases. It is exceptional for the condition to pass on to chronic nephritis or chronic Bright's disease, and there can be little doubt that in many instances where this is supposed to have been the case the more correct reading of the facts is that the disease has really been chronic from the beginning, and, owing to the sudden manifestation of symptoms, its onset has been thought to have been acute. It is now well recognized that the onset of acute symptoms is not always due to the onset of acute disease, but may be dependent on sudden manifestation of chronic disease. This is especially true of renal disease, and illustrations of this may be seen in such very chronic diseases as cystic degeneration of the kidneys, and also in the ordinary granular kidney. Death in acute renal disease may not only be caused by such well-known complications as uraemia and secondary inflammations of the pericardium, pleura, and lungs, but it may also be due to the development of cardiac degeneration and the production of great cardiac dilatation; in fact, cardiac dilatation from myocardial degeneration should really be regarded as one of the great dangers of acute renal disease, and the occurrence of this complication is the main reason why all tendencies to plethora of the circulation should be neutralized as far as possible by purgation, and to some extent by the limitation of the amount of fluid consumed by the patient.

Although the natural tendency of acute nephritis is to subside and clear up, yet it must be admitted that not uncommonly patients who have suffered from this disease are liable to recurrences, and not infrequently to more than one recurrence. After repeated recurrent attacks

the disease may undoubtedly become chronic, but, as already stated, it is not usual for the malady to pass from the acute stage straightway into the chronic.

Acute nephritis may not only leave behind it a liability to further attacks, but it is also possible that an acute nephritis of slight severity may perhaps lead to such changes in the kidney, that a chronic disorder like the granular kidney develops as a sequel after the lapse of many years. This is by no means proved, but the occurrence of granular kidney sometimes in young patients has suggested that it may possibly arise in this way.

One of the most important varieties of nephritis is that associated with syphilitic infection, and the relation of nephritis to syphilis is undoubtedly frequently overlooked. It is well known that nephritis is commonly produced as a result of some infective process. This is well recognized in the case of scarlet fever, typhoid fever, pneumonia, and influenza. But it is curious that in the case of renal affections the part played by cold in the production of disease is more insisted upon than in the case of most other diseases. Pneumonia, pleurisy, peritonitis, pericarditis, were all at one time attributed to the direct action of cold; it is now recognized that all these maladies, even when apparently due to cold, are really due to a specific cause. That cold plays some part in the etiology of many of these diseases, and especially in the case of pneumonia, cannot be gainsaid; but, although we all recognize that pneumonia may follow exposure to cold, no one looks upon the disease as directly and solely dependent on such exposure. The most that the cold does is in some way to affect the organism so that the virus of pneumonia, the pneumococcus, is able to produce its effects. Further, it might be pointed out that myelitis was also frequently attributed to exposure to cold, and numerous cases were recorded in which the disease followed such exposure, and especially, perhaps, to the combined action of cold and damp. We now know in the case of myelitis that a great many of such cases are really of syphilitic origin and dependent on the effects of endarteritis. Nevertheless, the influence of cold as a direct etiological factor in the production of renal disease is still very largely held. It is, of course, possible that there is some obscure relation between the skin and the kidneys of a nervous or chemical nature; and when we remember that such a gland as the pituitary secretes a material which is a potent diuretic, it is clear that the chemical and nervous interrelationships of different organs is very complex, and it may well be that there is some connexion between the skin and the kidneys, but it has not at present been discovered.

Putting these theoretical considerations on one side, there can be no question that nephritis of the type of parenchymatous or diffuse tubal nephritis is by no means uncommon in the first two years following syphilitic infection. Further, this nephritis often occurs at a time when the patient is well, or, at any rate, is not profoundly affected by the underlying specific disease, and hence the nephritis when it occurs is extremely liable to be looked upon as produced by exposure. The presence of syphilitic infection in many of these cases is discovered, one may say, accidentally; thus, in what seems an ordinary case of acute or subacute nephritis, a few spots of some characteristic secondary syphilitic eruption may be seen, or a node or a gumma may be found on examination. It is interesting to observe that nephritis occurs in the syphilitic much at the same period that myelitis or hemiplegia are liable to occur—that is to say, within one or two years of infection.

There are other characteristics, however, of syphilitic nephritis which reveal its nature. Thus, it is characterized by very intense albuminuria, the urine frequently becoming solid, and although the urine is loaded with albumen, the patient may not present very marked signs of illness. Thus, it is not uncommon for the condition to be discovered more or less accidentally, the symptoms being vague, and the urine perhaps examined as a matter of routine. In some instances dropsy undoubtedly occurs of the usual type, and this form of the disease is more severe in its course. The albumen is not only present in large amount, but it persists for a very long time—six or eight months or even longer; and it is important to recognize that, notwithstanding this long persistence, it may not uncommonly ultimately completely clear up and the patient may regain his usual health, neither the urine

nor the vascular system showing any sign indicative of the renal affection which has been present.

Nephritis occurring during the course of secondary syphilis and of a high degree of severity is undoubtedly much more common than is usually supposed, and this possible etiology should be borne in mind in cases where one might at first sight be inclined to attribute the disease to exposure. I do not mean to imply that all cases of renal disease not due to scarlet fever, alcohol, or lead are due to syphilis, but I would suggest that it is probable that all cases of renal disease are really due to toxic agents and not directly due to cold; and that in some of the cases imputed to cold syphilis is really the causative factor. It is well established that toxic agents are capable of producing nephritis precisely similar in its clinical course and morbid anatomy to the nephritis said to be produced by cold; thus, for example, in phthisis, Bright's disease of the ordinary type is not uncommon as a complication, and it would seem probable that it is produced here by the tubercle bacillus. In other words, that the specific organisms, like the bacillus of tubercle, may not only cause a definite tuberculous lesion of the kidney, but that it may also give rise to a nephritis which, although of tuberculous origin, is not characterized by the presence of definite tuberculous lesions. The bacillus of tubercle has been found in the kidney and in the urine of many cases of so-called chronic Bright's disease complicating phthisis. Of course it may be argued that their presence is accidental.

Another variety of chronic Bright's disease of great interest is that which may be called the "small white kidney." This form of the disease is of importance to the practitioner, inasmuch as it so frequently runs a latent course, and the patients only come under observation with the terminal symptoms, which are often of great severity and are of the uraemic type. These patients are usually young, or at any rate under 30 or 35 years of age, and not uncommonly under 25. They generally seek advice on account of the general disturbances of nutrition, loss of flesh, anaemia, or else on account of headache, with, possibly, some failure of sight. But, in a very considerable proportion of cases, advice is not sought until uraemic symptoms, often of great severity, have developed. It is very remarkable that sometimes these uraemic complications develop at a time when the patient is apparently not very ill. Thus, instances are on record where a patient has perhaps sought to have his life insured, and therefore presumably is not obviously suffering from ill health, and where not only the life has been declined, but where death has taken place within a few days or weeks of such examination. These patients with contracted white kidney pass an abundant urine containing a considerable quantity of albumen; they do not usually suffer from dropsy, but cardio-vascular changes are well developed, and albuminuric retinitis is very generally present. In many of these instances the disease is first recognized owing to the failure of sight. On the whole, perhaps, this form of chronic Bright's disease is the most important from a clinical standpoint, inasmuch as it is very fatal, and so suddenly fatal, and the symptoms may be extremely equivocal. Most curious errors of diagnosis may be made in such cases; thus, it may be confounded with cerebral tumour, with uraemia, with epilepsy, with chlorosis, and with Addison's disease. The last mistake may be made owing to the great impairment of strength that is sometimes present, and also owing to the fact that some degree of pigmentation may occur. A proper examination of the urine and of the fundus oculi should prevent such errors being made.

The confusion with epilepsy is liable to arise for several reasons, first and foremost because the uraemic seizure may take the form of a series of epileptiform convulsions, occurring perhaps at a time when the patient thinks himself in good health. Thus I have known of a case where a man, returning in the early spring from fishing in Scotland, and therefore presumably feeling well, was seized with uraemia of this type on the night of his arrival in London. Examination of the urine and of the fundus oculi revealed the true nature of the case. But it must be remembered that there are apparently rare forms of true epilepsy with the recurrence of severe fits, often at long intervals, and where for some days subsequently to the fit the patient passes a highly albuminous urine.

Traces of albumen in the urine are, of course, common after epileptic seizures; but I have met with two instances where epileptic seizures occurring at an interval of many years have been of great severity, and where the state of the urine for many days after the fit was such as to lead to the suspicion that serious renal disease was present, but the albuminuria subsequently cleared, and the patients regained their usual health. In one of these cases there could be no doubt as to the disease being primarily epilepsy, inasmuch as the fits had followed an injury of the head received in childhood.

The confusion with asthma is very liable to be made, owing to the fact that in most forms of uraemia, and especially in acute uraemia, dyspnoea is a leading feature. As already mentioned, the error of diagnosis can generally be avoided by careful examination of the urine and of the fundus oculi.

The true granular kidney is also a lesion of great importance to the practitioner, as here, also, we have to deal with the insidious disease. The usual type of case with marked cardio-vascular changes, high tension and abundant urine with a trace of albumen, is easily recognized, and the main point of clinical interest with reference to these cases lies in the recognition of the fact that this malady may exist in a very advanced form and yet the patient present a spurious appearance of health, being apparently vigorous, rosy in colour, and with nutrition apparently well maintained. This type of case is often difficult to treat, inasmuch as the patient refuses to recognize the real gravity of the underlying condition. There are several other types of granular kidney which are of clinical interest. Thus, it is important to recognize that albumen is not constantly present in the urine in all cases. Further, that the albuminuria, when present, may sometimes be of the postural type, and therefore absent in the morning specimen of urine. Granular kidney should never be excluded on the strength of a single examination of the urine. It is more important to pay attention to the quantity of the urine, the specific gravity, and the state of the retinal arteries as revealed by the ophthalmoscope. The ophthalmoscope should be used in these cases not so much from the point of view of detecting albuminuric retinitis as for the purpose of detecting the earlier changes in the retinal vessels.

Again, in some instances of granular kidney the patient presents marked neurasthenic symptoms, and the very serious error may be made of looking upon all the symptoms as entirely due to such a functional cause when arterial sclerosis and granular kidney are really present.

There are two other types of granular kidney which are of interest clinically, although they are both of them rare. There is one where an extreme form of atrophic granular kidney may be present and where the patient presents no cardio-vascular changes, or at any rate no changes which can be recognized with certainty clinically. It is obvious that grave errors are liable to be made in the diagnosis of these cases, inasmuch as the leading symptoms of high tension, arterial thickening, and cardiac hypertrophy are absent. Such patients present vague symptoms of ill health generally for a prolonged period, accompanied with much wasting, and finally die, probably of uraemia or of some secondary inflammation such as pericarditis or pneumonia, and in many of these cases the nature of the disease is only recognized in the *post-mortem* room.

Lastly, cases are sometimes seen of granular kidney where there is the most intense anaemia, rivalling that of pernicious anaemia, and where the symptoms are entirely referred to this anaemia and disorder of nutrition, the obviously renal symptoms, such as headache and vomiting not being well marked. These cases are also difficult to diagnose, owing to the prominence of the anaemia and the slight changes in the urine.

THE late Mrs. Jane Harriet Oakley, of Hove, whose will has now been proved, made the Sussex County Hospital her residuary legatee. The amount to which the institution will benefit is believed to be some £15,000.

UNDER the will of the late Mr. Charles Henry Bailey, of Newport, Monmouthshire, the Newport and Monmouthshire Hospital receives a sum of £700 for a child's cot, and an endowment of £52 10s. a year for the maintenance of a bed in the men's ward.

A Clinical Lecture ON ALBUMINURIC RETINITIS.

DELIVERED AT ST. BARTHOLOMEW'S HOSPITAL.

By WALTER H. JESSOP, M.B., F.R.C.S.,

SENIOR OPHTHALMIC SURGEON AND LECTURER ON OPHTHALMIC MEDICINE AND SURGERY AT ST. BARTHOLOMEW'S HOSPITAL.

GENTLEMEN,—We have had lately in the wards three cases exhibiting the classical signs of the retinitis associated with Bright's disease. I cannot do better than to refer first to the short notes taken of these cases, as they serve to summarize the clinical conditions of the disease. Before doing so let me remind you that the existence of eye symptoms was noted in the first observations of Dr. Bright, who described loss of vision, partial and complete, in some cases. Heymann gave the earliest description of the retinal changes which he and Coccia observed by the ophthalmoscope; whilst Liebreich in 1859 first illustrated the ophthalmoscopic appearances in his admirable drawings.

The first case described is typical of albuminuric retinitis as found in parenchymatous nephritis. The second case exemplifies the affection in chronic interstitial nephritis, and the third case illustrates the albuminuric retinitis of acute nephritis.

Parenchymatous Nephritis.

A horse-collar maker, aged 26, was admitted under Dr. Tooth. The past history of the case was that in October, 1905, he had severe frontal headache for twenty-four hours, since when he has had polyuria; four days after this attack he had headache and vomiting for twelve hours and had six fits the night after. He returned to work in a week and remained apparently well till November, when he had another attack of vomiting, lasting twelve hours, and since then has had continual headache with vomiting two or three times a day independent of food. He has lost 10 lb. in weight, and has had muscular twitchings for one month.

On admission the patient's face was pale and puffy; he looked anaemic, and his tongue was furred; temperature normal; pulse 76, with moderate tension. His eyes were slightly unsteady in conjugate deviation. The eyelids were rather puffy, the pupils were equal, and reacted normally. Ophthalmoscopic examination showed oedema of retina in the right eye; the optic disc edges were fairly clear, of muddy aspect, but with no swelling; the yellow spot at nasal side showed half a star of bright white small radiating patchy spots. The retinal arteries were convoluted and rather small. The retinal veins were dilated. Scattered about the retina, chiefly within the equator and near the vessels, were several flame-shaped superficial haemorrhages. Numerous white and yellowish-white patches and spots were near the optic disc and along the retinal vessels; no cholesterol spots present.

The left eye showed the same conditions except that there was no marked star at the yellow spot, but the Mullerian fibres were seen very distinctly as radiating lines; there was one large linear haemorrhage between the optic disc and the yellow spot. There was no detachment of the retina and there were no black retinal pigment spots.

The heart and lungs were normal but the pulse tension was increased. The abdomen was rather pigmented, the reflexes were brisk, and the spleen not enlarged; the knee-jerks were exaggerated; urine 1016, pale in colour; there was about 1½ albumen; there was no sugar. The past history of the patient was that he had scarlet fever at 11 years of age and typhoid soon afterwards. He had drunk a great deal of beer up till some months before admission.

On the eighth day after admission to the hospital the patient vomited several times, and then continued vomiting at times with slight delirium till the sixteenth day when he showed signs of uraemia, and died from uraemic convulsions on the seventeenth day.

The *post-mortem* appearances showed the large white kidney of chronic parenchymatous nephritis.

Chronic Interstitial Nephritis.

A man, aged 48, who consulted me in 1898, complaining of dimness of vision in the right eye, aching of the eyes, and headache. His vision, right and left eye, was 8. Ophthalmoscopically both eyes showed retinal arteries that were rather tortuous and with marked "copper-wire" look; the retinal veins were dilated, and in places were distended where crossed by the arteries. The urine was examined three times, and was of low specific gravity but did not contain albumen. His radial arteries were rather thickened, and there was increased tension of the pulse.

I saw him again in 1900, and found he still complained of headache and aching of the eyes; the vision was dim at times, and he had lately passed more urine than usual, and had