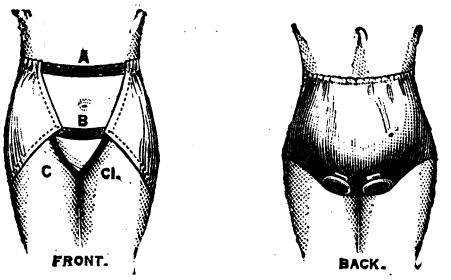
The arrangement permits of the use of an ordinary diaper. The rubber rings do not cause noticeable projection when worn under ordinary clothing, as they take up a position below the gluteal folds when the patient is standing.

When a good-fitting model has been obtained it is as well to have at least half a dozen made, as the pair of



rubber rings can be transferred from one garment to another in a minute, and thus a clean one can be used daily.

I have found decided relief from the severe pain of coccydynia has resulted in such cases as I have described, and the plan might be worth a trial by others who have greater opportunities for testing it than I have.

I have deposited my model with Messrs. Lindsey and Sons, instrument makers, Ludgate Hill.

ON THE CAUSATION OF PARENCHYMATOUS NEPHRITIS.

By GEORGE W. WATSON, M.D., M.R.C.P.,
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ALTHOUGH the etiology of acute nephritis is perhaps better understood than that of chronic nephritis, it cannot be said that very much is definitely known as to the actual causation of any variety of inflammation of the kidney. Owing to the association of acute nephritis with many of the infectious and febrile disorders, especially scarlatina, and its comparative rarity apart from these affections, the disease is considered with reason to be due to micro-organisms concerned in the production of the original fever or to their toxins.

In certain cases of nephritis occurring in infectious disorders, the specific micro-organisms have been demonstrated in the kidney or in the urine or in both. For instance, typhoid bacilli have been found in nephritis after typhoid fever, pneumococci in nephritis following pneumonia, etc. This does not necessarily prove the causation of the nephritis in these cases; but, on the other hand, acute inflammations of the kidney have been induced by Pernici and Scagliosi by the injection into the blood of certain micro-organisms, such as the bacillus of anthrax, the *Staphylococcus pyogenes aureus*, etc. It was found also that the toxins of these organisms when injected were productive of renal change, but were less harmful than the organisms themselves. Acute nephritis has been induced by the injection of the diphtheria toxin by Roux and Yersin, Spronk and others.

Beyond the association of acute nephritis with the acute fevers, very little is known regarding the causation. The disease has been attributed to the influence of cold, to pregnancy, to the ingestion of deleterious articles of diet, to drugs, to toxins elaborated in the body as the result of abnormal metabolism, and to many other things.

With regard to the causation of chronic nephritis our knowledge is even more vague. The relation of chronic nephritis to a previous acute nephritis is, I believe, far from being understood. And in the absence of more precise knowledge we include in the etiology of chronic nephritis such factors as chronic alcoholism, the continued action of damp and cold, various constitutional diseases leading to anaemia and cachexia, syphilis, and so on—factors which from observations we have learnt to associate with the appearance of the disease. But how these various circumstances operate we are still ignorant.

With the idea of obtaining some further insight into the etiology of renal inflammations, I have recently, in conjunction with experimental work, been engaged in the analysis of 100 cases of nephritis occurring in the wards of the General Infirmary at Leeds during the years 1900, 1901, 1902, 1908, and 1909. Save for the exclusion of cases of definitely interstitial nephritis or primary sclerotic kidney occurring beyond the middle period of life, there has been no selection in the series analysed.

Out of the total of 100 cases, there were 80 cases of undoubted chronic disease of the kidneys. Of the remaining 20, some were unquestionably examples of primary acute disease, whilst the others did not present a sufficiently clear combination of clinical facts to warrant a definite diagnosis. For convenience, both in description and deduction, I propose to consider first the chronic cases.

Before passing on to a consideration of the factors probably concerned in the production of chronic nephritis, it is necessary to mention briefly the different types of disease which are met with. Some cases exhibit marked albuminuria, with perhaps haematuria, scanty urine, dropsy, and anaemia, but no very pronounced toxic symptoms and no retinal changes as a rule. This clinical picture is associated with a distinct type of morbid kidney. The organ is large and pale, differentiation is good, and the capsule strips easily. Microscopically there is a widespread fatty degeneration of the tubules, and to a lesser extent of the glomeruli and the stroma, with also some amyloid change. Indications of interstitial inflammation may be slight, but are probably always present. This type of kidney is known as the "large white kidney."

Another type is that occurring in pale, thin, weakly-looking individuals. There is little or no dropsy, definite but less marked albuminuria, a normal or even an increased quantity of urine, pronounced toxic symptoms, and, usually, retinal changes. In this form the cardio-vascular changes are much more evident than in the preceding. The appearance of the kidney from a case of this kind is equally characteristic. The organ is small and contracted, differentiation is poor, the capsule strips with more or less difficulty, and the surface of the kidney is "pimply." Microscopically there is a great increase in the interstitial tissue, together with degenerative changes in the tubules and the glomeruli. This kidney has been termed the "small white" indurated kidney.

Clinically and pathologically both these types are definite enough when they occur, and present no difficulty in diagnosis. But by far the greater proportion of cases present features common to both varieties, and cannot strictly be classified as either. When we examine the kidneys of such we find a great variety of appearances. There are variations in size, in colour, in the ease with which the capsule can be stripped, and the character of the surface of the kidney. And microscopically these variations can be demonstrated to be due to differences in degree of the pathological changes in the individual tissues affected.

Much confusion is caused by the introduction into clinical medicine of such terms as "large white kidney," "small white kidney," "mixed nephritis," etc. It would lead to clearer conceptions if cases were recognized as cases of chronic parenchymatous nephritis, with little, some, or much induration, bearing in mind that the quantity of urine secreted and the intensity of the toxic symptoms tend to vary proportionately with the degree of induration, whilst the amount of oedema and degree of albuminuria usually vary inversely with the same factor.

Most authorities hold that some interstitial change occurs in the kidney in all cases of chronic parenchymatous nephritis, and that the degree of induration is largely a matter of time. That is to say, the longer a patient lives with chronic parenchymatous nephritis, even though at first the symptoms suggest that there is very little fibrosis of the kidney, the more does the clinical picture tend to approximate to the type which we have learnt to associate with much induration of that organ.

It is not my present object to discuss the question whether or not the same factor is responsible for the development of both the tubal disease and the interstitial fibrosis. I have therefore made no attempt to classify the cases according to type, but have considered them all, for my present purpose, simply as cases of chronic parenchymatous nephritis.

Chronic Nephritis May Exist Without Symptoms.

Undoubtedly we must admit that chronic parenchymatous disease of the kidney may exist for some length of time without giving rise to any symptoms of illness, its presence being therefore unsuspected. And when symptoms do appear we are often surprised by the degree of disease which we find to exist. A striking example is as follows:

Richard M., aged 33, a weaver, stated that he had always been strong and well up to two months before admission to hospital. Then he began to suffer from headache and morning vomiting. There was never any oedema. He died four weeks after admission, and at the autopsy advanced parenchymatous disease of the kidney, with extreme induration, was found. Both kidneys together only weighed 5 oz. This morbid change must have been going on for a considerable time, probably for years, but with an entire absence of symptoms.

Cases such as this—and I believe they are not very uncommon—can only be explained on the hypothesis that we begin life with a much larger amount of renal substance than we require for ordinary purposes, and that it is only under extraordinary circumstances that the surplus or reserve amount is called into use. Such extraordinary circumstances apparently did not occur in this man's life, and the slow inflammatory process went on unrecognized until the residue of kidney parenchyma was inadequate for even the small claims made upon it in his ordinary existence.

In twelve other cases of the series a diagnosis of acute disease was made on the development of symptoms, but on *post-mortem* examination chronic disease was found to exist—in some no doubt of several years' duration. In these no affection of the kidneys was suspected before the terminal illness.

How Symptoms May Manifest Themselves.

The onset of symptoms may be acute or gradual. In the former case the clinical picture is that of an acute nephritis with much oedema, scanty urine containing much albumen and perhaps blood, together with some toxic symptoms. In these instances a diagnosis of acute nephritis is usually made. Virtually the condition is an acute nephritis occurring in an organ already diseased, but it differs from a primary acute nephritis, not so much in the acute stage as in its course afterwards. In primary acute nephritis, unless death occurs, the blood disappears from the urine, the albumen entirely or almost entirely disappears, the dropsy subsides, and the toxic symptoms clear up after a variable but comparatively short period of time. But in the acute nephritis associated with the presence of chronic renal disease the improvement is slower and more unsatisfactory. The oedema persists, or varies in degree from time to time, or may disappear altogether only to reappear again. The blood disappears from the urine more or less quickly as a rule under the influence of rest and treatment, but the albumen, although it usually diminishes in amount, nevertheless persists in appreciable quantity. These are the cases where a diagnosis of "acute nephritis becoming chronic" is made, a sequence which I am of opinion seldom occurs.

In the series of 80 cases no fewer than 12 were diagnosed from the character of the onset as acute nephritis, but were proved at the *post-mortem* examination to be cases of chronic nephritis of some duration. And in many others in which a similar diagnosis was made the clinical course after the acute attack suggested very strongly the same state of affairs, although *post-mortem* evidence was wanting. An illustrative case is as follows:

Henry K., a miner, aged 34, stated that he had never been ill in his life up to the onset of the present symptoms. He commenced two months before admission to hospital with aching pains in the legs. A week later his body and legs began to swell, the urine was much diminished in amount, and contained blood and albumen. A diagnosis of acute nephritis was made, but whilst the blood disappeared from the urine the albumen persisted, and likewise the oedema. Death from uraemia occurred three months after the onset, and at the autopsy an enlarged kidney, with advanced parenchymatous change and a little induration (typical large white kidney), was found.

In other cases initial symptoms developed insidiously. These are usually of the nature of mild uraemic symptoms, such as headache, dimness of vision, nausea, shortness of breath. Or there may be a slow development of oedema, with or without uraemic symptoms. The nature of the onset has, as will be discussed later, some bearing on the prognosis.

Age Incidence.

Out of 80 cases, 4 were under the age of 10, being respectively 1, 3, 3, and 9 years old; 14 were between the ages of 10 and 20, 33 between 20 and 30, 19 between 30 and 40, 5 between 40 and 50, and 5 beyond the age of 50 years. From this it would appear that the period of life at which the onset of renal symptoms in chronic parenchymatous nephritis is commonest is between the ages of 20 and 30.

Relation of Infectious Fevers to Chronic Nephritis Developing After an Interval.

Only 6 of the cases in my series could refer to a past attack of scarlatina, and of these 2 only had any renal complication at the time. Twenty cases gave a history of some other infectious disease, or of some septic process. Five had had typhoid fever, 5 influenza, and 5 pneumonia. Others had a history of diphtheria, measles, quinsy, or "blood poisoning." Not one of these 20 cases had renal symptoms at the time of the acute illness.

A proportion of 6 cases of scarlatina out of a total of 80 is surprisingly small. And this, too, in persons suffering from a disorder of which scarlatina is accounted an important etiological factor is even more remarkable. Again, a proportion of 20 cases with a history of other infectious and septic disorders, out of a total of 74, is not more than one would expect in healthy members of the community.

Although the number of cases dealt with is small, the evidence they furnish supports the view which I hold strongly, namely, that acute diseases have very little, if anything, to do with the production of chronic renal disease developing after a reasonable interval. In chronic parenchymatous nephritis the change in the kidneys is progressive, and must therefore be due to an agent operating continuously or intermittingly over a period of time. There is in all the organs of the body a tendency to the repair of diseased tissues when the cause of that disease has been removed, and there is no reason to consider the kidney an exception to this rule. Therefore, I believe that inflammation of the kidney occurring as a complication of acute illness tends to recovery when the cause of that acute illness no longer exists. True, there may be some loss of kidney substance, owing to severe injury, and the functional capacity of the organ thereby lessened, but the kidney now is not the seat of a nephritis. Possibly an organ such as this is more prone to inflammation in the presence of a cause potent to reduce it.

Nothnägel, in his *System of Medicine*, states that it is not uncommon at an autopsy to find kidneys of normal or increased size, in which the remains of former inflammatory change can be detected by the microscope, and where there were no clinical manifestations of renal disease during life, even no albuminuria.

Lead as a Factor in the Production of Renal Disease.

In only three of my cases was lead a possible etiological factor. All were painters by occupation, and all were accustomed to mix their own paint. In two of them there was marked vascular thickening, and the symptoms were those of parenchymatous nephritis with much induration. The third was a doubtful case, as the man had not been in contact with lead for many years, and here there was very little arterio-sclerosis. It is well recognized, however, that chronic lead intoxication is a potent factor in the production of the indurated kidney. This is proved by reference to the records of any large hospital, and moreover, the lesion had been produced experimentally by Charcot, Prior, and others.

Alcohol.

I have no evidence to bring forward to indicate that alcohol is ever a cause of chronic parenchymatous nephritis. The cause of primary indurative nephritis does not come within the scope of my remarks, and it is with this form of disease that over-indulgence in alcohol is chiefly associated. Sixteen of my cases gave a history of alcoholism in excess, but there were other factors which made it impossible to estimate the share, if any, that this had in the causation of the disease. In three instances, however, acute symptoms developed apparently as a result of a "drinking bout." In two of the cases a diagnosis of acute nephritis following acute alcoholism was made; a diagnosis which was corrected later to one of "acute nephritis," developing in the course of a chronic nephritis.

In the third instance, the man was known to be the subject of nephritis, and the true relation of the alcoholism to the acute onset of symptoms was appreciated.

The only definite effect that alcohol produced in the cases reviewed appeared to be the determination of symptoms in an already existing latent chronic nephritis.

Fevers and Sepsis.

In seven cases symptoms appeared apparently as a result of some fever or septic process. This is not surprising, for if we accept the infectious fevers as a cause of acute nephritis, it is only to be expected that they should be potent to cause an acute exacerbation in the course of chronic disease.

The effect on the kidney of a long-continued chronic septic affection is not clear. In four cases symptoms of renal disease developed in the course of a chronic affection of the lungs.

Brief summaries of the important facts in these are as follows:

Ada D., aged 9, had suffered from a chronic cough for some years, but there was no history of any acute illness at any time. A week before admission to hospital oedema of the face and legs came on gradually. The urine was diminished in amount, and contained a moderate amount of albumen, with cellular and granular casts. An interesting fact also was that streptococci were found in the urine. Examination of the chest revealed fibrosis of the lung with bronchiectasis at the left base, and displacement of the heart to the left side. Whilst in hospital there were no indications of renal disease beyond the oedema and the urinary findings. The dropsy disappeared, and she was discharged at the end of three weeks. The urine still contained albumen in quantity, and a diagnosis of chronic parenchymatous nephritis was made.

Sam R., aged 38, had suffered from cough for five years. No other history of illness. One morning he noticed some swelling of the ankles. This gradually increased, and later there was general anasarca with ascites. The urine contained much albumen and a variety of casts. Death occurred three months after the onset, and at the *post-mortem* examination fibrosis of the lung with marked bronchiectasis (tuberculous in origin) was found. The kidneys were large and pale, and showed extensive chronic tubal and slight interstitial change. This was not a case of general lardaceous disease, the other organs of the body being healthy.

It is important to note on this case that with the exception of the cough the man felt perfectly well until the onset of the final illness. The development of renal symptoms without any obvious determining cause had, I think, some bearing on the unfavourable course which ensued. This point, however, will be referred to later.

Esther M., aged 31, had never been very strong, having suffered from bronchitis with much sputum for many years. There was never any clinical evidence of phthisis. She had three children. Her first confinement was uneventful, but after the second and third she suffered for a few weeks from swelling of the feet. The state of the urine was not recorded. After the third confinement she enjoyed her usual health for eighteen months, and then the oedema of the legs again slowly developed, and lasted until her death a year later. When under observation in hospital during the last few weeks, the urine was diminished in quantity and contained much albumen and many casts, but no blood. No *post-mortem* examination was made in this case. Note here that what were presumably renal symptoms were twice associated with labour, and these disappeared after a short interval. The symptoms of the final illness were not associated with any definite cause and never disappeared.

Doris W., aged 3, had pneumonia when 2 years old, and again six months later. She was never quite well afterwards. Two months after the second attack of pneumonia she began to be troubled with cough, and a little later oedema of the face and legs appeared. She died with symptoms of uraemia. The kidneys when examined were large and pale, and showed marked chronic tubal and a lesser degree of interstitial change. In addition there was a small abscess in the left lung.

Senator, basing his opinion on a number of cases specially investigated with regard to this point, believes that, in Berlin at least, of all chronic diseases, tuberculosis must be held to be the most common cause of chronic tubal nephritis not necessarily amyloid in character. I have made clinical and pathological observations on a large number of cases of pulmonary tuberculosis, but have not met with associated chronic nephritis sufficiently often to support this view. Still, a chronic septic process in the lung must not be lost sight of as a possible cause of progressive renal change.

Another possible source of infection is the throat. Several cases gave a history of uniform good health with the exception of frequently recurring "sore throats." The frequent association of acute nephritis with scarlatina and diphtheria is suggestive of a milder anginoid affection being a possible cause of the chronic disease. And the fact that diphtheria organisms may remain in the fauces for a considerable time and give no indication of their presence renders the view not unlikely that absorption of products from other organisms in the same situation may occur without any indication of infection.

Cold and Damp.

Sudden and severe chilling of the body has long been considered to be a cause of acute nephritis. In my series there were six cases in which symptoms of acute nephritis developed apparently as a direct effect of a severe chill. In many more the onset was attributed to cold, but in these the association was not so convincing.

Details of three out of the six cases are as follows:

Alexander S., aged 29, a miner, came under observation in 1909 with marked dropsy of the face, body, and legs. The urine was scanty and contained a large quantity of albumen and some blood. The onset had been rapid, and followed a severe chill, due to getting wet through. This case might have been diagnosed as one of primary acute nephritis, due to cold, had it not been that the man had been under observation in 1905 with an exactly similar condition. When discharged from hospital the first time the urine still contained some albumen, but the patient remained in perfectly good health until his second attack. When he left the hospital the second time the urine was not clear of albumen, but there were no symptoms.

Samuel S., aged 18, a bricklayer, gave a history of typhoid fever when 16. He was apparently perfectly well after this illness for two years, when he got wet through and caught a chill. The following day he was seized with severe and persistent vomiting. His legs began to swell, and the urine became scanty and dark in colour. When admitted to hospital there was marked oedema, and the urine contained a large amount both of albumen and blood. After a few weeks the blood had entirely disappeared, but the albumen persisted and was present in fair quantity on his discharge.

Alfred B., aged 60, a man with a strong alcoholic history, had enjoyed uniformly good health up to a month before his admission to hospital. Then he caught a severe chill during cold weather. Almost immediately the legs began to swell and later the body. The quantity of urine was much diminished, and contained much albumen and blood. The man died with uraemic symptoms, and at the autopsy advanced parenchymatous disease of the kidney, with much induration, was found.

In the last quoted case it would have been impossible to have made a correct diagnosis from clinical data. To all appearances the condition was one of primary acute nephritis due to cold, and the true state of affairs was only revealed after death.

As already mentioned in the first case, the true diagnosis of chronic parenchymatous nephritis with an acute manifestation could only be made with the help of the previous medical history, and such assistance is not always forthcoming.

The other three cases were very similar to the foregoing, and therefore details have not been given.

In my investigations I have not been able to find one single convincing instance of primary acute nephritis arising from cold. Most of the supposed cases occurred in adults, in whom I believe the primary acute disease rarely occurs. When acute symptoms have arisen apparently as a result of cold, I am inclined to regard the cold merely as a factor determining the onset of symptoms in an already existing chronic disease.

The effect of damp surroundings, frequent wettings, and insanitation, frequently in evidence in the etiology of nephritis, is probably one which merely contributes to the development of the disease by depressing the general health and resisting power of the individual, and thereby favouring the operation of the true cause.

Excessive Muscular Work.

The following case is of interest:

John R., aged 27, had never suffered from any definite illness, but was never very strong. He was employed as a tram conductor, and had never done any heavy manual work. Whilst changing his house he assisted in the removal of heavy articles of furniture, working very hard for half a day. The next day he did not feel very well, and could not go to work. On the following day his face and legs began to swell, and the urine contained albumen and blood. The oedema and albuminuria disappeared within a week under treatment, but the albumen persisted, though in lesser amount. Examination of the eyes revealed albuminuric retinitis.

Apparently this was a case of chronic parenchymatous nephritis running a latent course, in which symptoms were determined by unusual and severe muscular exertion. This is not surprising when we remember that albumen with casts is not uncommonly met with in the urine of

healthy athletes after hard muscular exertion such as football, rowing, running, and the like. Here it would appear that the functional power of the kidney is taxed to the uttermost for a short time. And that the functional limit of a diseased kidney should be the more easily reached is a fact which needs no pressing.

In the same way can be explained those cases in which symptoms arise as the result of labour. In 5 of my cases the onset of symptoms was definitely associated with delivery. In one instance, which has been recorded in the paragraph dealing with fever and sepsis, the woman had symptoms of nephritis in two successive deliveries with good health between, and also afterwards for a period of months. Symptoms appearing a third time caused her death, and the diagnosis of chronic disease was confirmed by *post-mortem* examination.

It is clear, from the facts set forth, that the factors associated with the development of clinical signs and symptoms of renal disease cannot be held to be the cause of that disease. With the exception of lead, which usually gives rise to the indurative form of nephritis, and long-continued septic absorption in chronic disease—the effect of which in the kidney is not precisely determined—I am strongly of opinion that none of the factors enumerated above do more than act as contributing causes of nephritis, or determine the development of symptoms when the kidney is diseased. I would also say that symptoms of nephritis occurring in an adult, no matter whether the clinical manifestations suggest an acute or chronic process, indicate in the vast majority of instances a chronic change in the kidney. Of the actual cause of this chronic change we know practically nothing. There are many cases of chronic Bright's disease in which there is no history of previous illness, and in which there is nothing to criticize in the occupation, habits, or home surroundings, and where we have not the slightest clue to the causation.

Relation of the Character of the Onset to the Prognosis.

I have spoken above chiefly of cases in which the onset of symptoms is associated with some definite cause. But there are a great number of instances in which symptoms appear apparently spontaneously and without reason. This was the mode of onset in 37 of my cases. In most of these the development of clinical manifestations was not acute but gradual, usually the slow appearance of oedema. And where indications of renal disease have developed in this way, the after progress has been more unsatisfactory than in those in which a definite factor was responsible for the onset.

A reasonable explanation of this would be that the disease is progressing slowly, but that up to a certain point the kidney is able to perform its function under the ordinary circumstances of life, and no symptoms arise. But when that point is reached, the kidney is inadequate to meet even moderate claims made upon it, and symptoms appear. Rest in bed with appropriate treatment will relieve the kidney of a proportion of work, and so ameliorate or remove symptoms. Any return to activity, however, brings back all the conditions that existed before, with a return of the signs of renal inadequacy. If, however, the onset of symptoms is determined by some unusual circumstance, this being removed, the effect of treatment is more permanent.

As a general rule, it may be said that when symptoms of chronic renal disease owe their appearance to some definite cause, the prognosis is better than when the symptoms arise insidiously and apparently spontaneously.

I know of no way of diagnosing primary acute nephritis with certainty during the acute stage. The presence in the urine of a marked quantity of nucleo-albumen is held to denote an active destruction of renal epithelium, but this occurs in other conditions besides primary acute nephritis. The association of acute renal symptoms with an acute fever would reasonably lead one to suspect a primary acute nephritis, if the individual had previously had no indications of renal trouble. That the diagnosis can never be certain has been shown already. One other example, however, will not be out of place here, and will illustrate the difficulty:

A woman, aged 32, had a severe attack of influenza, and during her convalescence suddenly developed extensive dropsy with albuminuria and haematuria. Her home surroundings were satisfactory and her previous health had been uniformly good. When she came under observation she had marked oedema and distinct urinary changes, but no toxic symptoms of any moment. In little over a week the oedema and the haematuria had disappeared. The heart, however, showed some enlargement, and the second aortic sound was distinctly accentuated. The albumen did not disappear, but was present in fair amount when she left the hospital four weeks later. The case was diagnosed at the time as one of primary acute nephritis becoming chronic, but I have no doubt at all that it was one of chronic nephritis, with an acute manifestation initiated by the attack of influenza.

If we consider the cases which exhibit renal signs and symptoms occurring in association with or immediately following some acute fever, and which for this reason may be suspected of being cases of acute nephritis, we cannot fail to be impressed by the fact that, although albumen may be present in large amount in the urine, and may be accompanied by blood and a variety of renal casts, yet in the great majority of instances all these abnormal constituents disappear, and the urine becomes normal in a comparatively short time (a few weeks) under the influence of rest and treatment.

I would not go so far as to suggest that all cases of acute nephritis which do not die recover absolutely, for there must be cases in which the kidneys are so extensively damaged in the acute inflammation as to be beyond all hope of repair, but I am firmly of opinion that in the very great majority of cases complete recovery is the rule.

In analysing my series of cases, I have been inclined to consider those in which albumen and casts disappeared from the urine permanently after a comparatively short time, with cessation of symptoms, as probably genuine examples of acute nephritis. On this assumption, out of the series of 100 cases, there were 18 of acute inflammation of the kidneys. Of these one died, and the diagnosis was confirmed at the autopsy. The remaining 17 apparently recovered completely.

Two other cases occurred in association with infectious fevers, and presented clinically all the features of acute nephritis. In the absence of any evidence of previous disease of the kidney these were also regarded as cases of acute nephritis. In both the urinary changes were very marked, and the symptoms were very severe. In both, also, although recovery was apparently complete, there was a slight persisting albuminuria. These cases are as follows:

Annie J., aged 21, had worked in a cloth mill for four years, the work being heavy and the hours long. She had had whooping cough and measles as an infant, but no other illness. She began to be anaemic some years before she came under observation in 1909. She then stated that she had had "influenza" a fortnight previously. This was followed in a few days by marked diminution in the quantity of urine passed, together with dropsy of the face and limbs. When admitted to hospital the urine contained much blood and albumen, with epithelial and granular casts. She was discharged three weeks later, when the oedema had disappeared, the urine was normal, with the exception of a faint trace of albumen, and she felt perfectly well.

Rebecca P., aged 8 years, was admitted to hospital in 1909 with pronounced oedema, albuminuria, and haematuria. She had never had any serious illness up to four weeks before admission. Then she suffered from a bad sore throat. Three weeks later oedema of the face and ankles appeared, with frequent vomiting. She was in the hospital three weeks, at the end of which time she was discharged, feeling quite well. The oedema had disappeared, but there was still a trace of albumen in the urine. This slight albuminuria was present two months later.

From what has been stated previously, the fact that there were no indications of renal trouble in these cases prior to the onset of definite symptoms does not prove that the kidneys were healthy, for it has been shown that disease of the kidneys may exist for some length of time without any symptoms whatever. The significance of this slight persisting albuminuria therefore is not clear. It may indicate a previous slight affection of the kidneys, or it may mean a continuance of the pathological process initiated at the acute attack. But in either case I am inclined to regard the presence of albumen in the urine as an indication of some persisting irritation of renal cells.

Age Incidence in Acute Nephritis.

Of the 20 cases of acute nephritis 11 were under the age of 15 years, the youngest being 9 weeks old; 14 were under 20 years, and 18 under 30 years. The oldest case was that of a man aged 37 years. This bears out the commonly accepted view that acute nephritis is a disease of childhood and adolescence.

Causation

Previous History.	Exciting Cause.	Number of Cases.
No history of illness	Scarlatina	3
No history of illness	Other infectious fevers	8
History of previous fever. (No nephritis)	Influenza	2
History of previous fever. (No nephritis)	Sore throat	3
History of previous fever. (No nephritis)	Cold and exposure	1
No previous illness (child of 9 weeks)	Acute intussusception	1
No previous illness...	—	2

I am aware that the foregoing table is not very helpful in arriving at a conclusion as to the most frequent causes of acute nephritis. On one point, however, I am convinced, and that is, that the history of previous infectious disease is of very little importance. It is the exciting cause which is the important one.

How many of the population escape measles, whooping-cough, and influenza in the course of their lives? Only a small proportion, and this proportion is not less in those who suffer an attack of acute nephritis. The exciting cause is to be sought for in the exanthems, the catarrhs, and, to a less degree, in the septic affections. And as these become more effectually controlled, so will acute nephritis become a rarer affection.

Conclusions.

To sum up, I would put forward the following propositions:

1. Acute nephritis is not a very common disorder.
2. Acute nephritis is usually due to the direct effect of some infectious fever, catarrh, or septic process.
3. The great majority of cases recover completely, therefore the prognosis is good.
4. The diagnosis of acute nephritis (primary) can seldom be made with certainty during the acute attack, and therefore a prognosis cannot safely be given until after an interval of a few weeks at least.

PYONEPHROSIS: OPERATION: RECOVERY.

BY

EVAN OWEN BOWEN, M.B., CH.B.EDIN.,

CARDIGAN, SOUTH WALES.

THE condition found in the following case of pyonephrosis was probably due to some congenital malformation.

The following is the history of the patient, a boy aged 14, as obtained from his mother, who states that she herself, the boy's father, and other nine members of the family are perfectly healthy.

The boy's illness can be traced back to the time when he was three months old, when the mother noticed that the child was puny, thin, sickly, irritable, and continually crying. A doctor was called in and the baby was given some medicine, but the doctor did not then express any opinion as to what the trouble was. The child improved a great deal after that, and it was nearly three months afterwards before the mother found that there was a thick yellowish discharge on the napkins which she thought came from his bowels. She again consulted her doctor, and he informed her that the matter came from the bladder, and that there was some inflammation in that organ. It was then noticed that every time the child made water little lumps of matter passed through the urethra. The child cried before and after the act, and the colour of the urine, as far as the mother can remember, was invariably light. He was not what could be termed "ill" at that period, but from that time forward she noticed that the boy had to pass water far more frequently than any of her other children. Sometimes he would have pains for days, then these would suddenly cease, and the patient would be free perhaps for a week or more, when they would again come on quite suddenly and without any apparent cause. This intermittence has continued throughout all the years. Sometimes the pains were mild, but at other times they were so acute and severe that the boy was confined to bed for days. In the intervals of freedom from pain he was in perfectly good health and able to attend school. There was no sickness or vomiting when the paroxysms of pain came on, and the bowels had always been regular.

Apart from measles when he was 7 years old he had had no other illness.

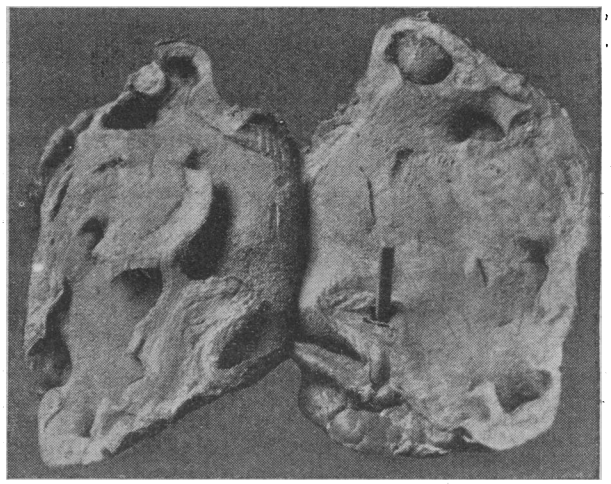
In the early part of 1911, a swelling commenced to show in the back over the region of the left kidney. This gradually grew bigger until it was lanced a few weeks afterwards by the medical attendant. A large amount of matter escaped at that time, and the wound did not close.

I saw the boy for the first time towards the end of August, 1911. He had been confined to bed for three days with an unusually severe attack of pain. He was looking languid, pale, and emaciated; his features were thin and drawn, his eyes were heavy and sunken, his tongue dry and furred, and thirst troubled him a great deal. There were occasional rigors. The pulse that evening was 84, and the temperature 99° F. Posteriorly on the left side there was a sinus, the opening of which was 2½ in. below the last rib, and 3½ in. from the middle line of the back. The dressings that had been placed by the mother over the sinus were saturated with a thin, greenish-yellow, muco-purulent pus. The patient complained of a dull aching pain in the left loin, and of frequency of micturition and great tenesmus. On palpation under an anaesthetic the left kidney was found to be markedly enlarged and very tender, and on pressure being gently exercised over it, pus was seen to exude freely from the sinus posteriorly. The urine was dark red, and contained a large amount of deposit. It was alkaline in reaction, with a specific gravity of 1004, and yielded positive results for albumen and blood. The microscopical examination of the centrifugalized deposit revealed the fact that the deposit consisted almost entirely of pus, with red blood corpuscles, and the normal urinary epithelial cells and mucin. Tube casts, renal cells, and crystals were also detected. Specially stained films failed to demonstrate the presence of the tubercle bacillus, though numerous micrococci were observed.

On August 24th he was admitted into my home, and kept under observation until August 29th. During that time his temperature was very irregular, and ranged between 97.2° and 100.4° F. His pulse-rate kept between 68 and 102. On August 30th I resolved to remove the left kidney.

Operation.

I chose the abdominal route, and the operation was performed on the lines advocated by Treves for abdominal nephrectomy.



The right kidney on examination appeared perfectly normal, but it was distinctly enlarged.

A great deal of difficulty was experienced in freeing the posterior surface of the diseased kidney owing to the existence of strong and dense adhesions. I was able, fortunately, to clamp the sinus before incising it in the course of dissection, thus preventing any escape of pus into the coffer dam which I had established at the commencement of the operation. Little difficulty was experienced with the pedicle. At the end of the operation the opening of the sinus was enlarged posteriorly, and a drainage tube inserted.

The patient bore the operation remarkably well, and we had little or no trouble with him during the whole time he was in the home. He was discharged on November 7th apparently cured, and is now playing about with his companions and attending school regularly.

The condition of the diseased kidney can be seen from the accompanying photograph. It was 5 in. long, 4 in. broad, and 1¾ in. in thickness. On section a large amount of greenish-yellow pus escaped, and the organ presented a picture of degenerated parenchyma with a number of abscess cavities, the majority of which were filled with soft, irregular concretions, which easily crumbled between the fingers. A direct communication could be demonstrated between most of the cavities and the pelvis. The pelvis was enlarged and much thickened. A piece of glass rod is inserted into the abscess cavity in the kidney that communicated with the perinephritic abscess.

From a consideration of the morbid anatomy of the diseased organ, it would appear that the condition must have been due, in the first instance, to some congenital malformation of the ureter or its pelvis. The lower portion of the pelvis is much more dilated than any other