HARLEY
ON
ALBUMINURIA.

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Nitric acid redissolves phosphates & [illegible] acid. the former without the latter [illegible] effervescence.
Acetic Acid throws down Cystin - where it exists - If uric acid is in great excess it will precipitate that.
One kind of albumen is the normal constituent of urine - this form is not ppt by Nitric Acid or heat.
In alkaline urine, a small quantity of Albumen is not ppt by heat. it should be made acid.
Alcohol - Chloroform - Tannic Acid. alum Pho. A Bichlor. Mercury & other mineral salts ppt albumen they are not reliable.
Quantitative analysis.

ALBUMINURIA.

J. V. Kerry

ALBUMINURIA,

WITH AND WITHOUT DROPSY:

ITS DIFFERENT FORMS,

PATHOLOGY, AND TREATMENT.

BY

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PREFACE.

THIS article on Albuminuria, with and without Dropsy, in connection with the different forms of local as well as constitutional disease, giving rise to a coagulable state of the urine, forms part of the course of original lectures on the urine and diseases of the urinary organs which, with slight modifications, I have annually delivered to medical practitioners since the year 1856.

This article first appeared in the "Medical Times and Gazette," at the close of last year (1865), and it is on account of the original views which it contains, on pathology and treatment, that I have reprinted it, like the one on Diabetes, in a separate form.

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ALBUMINURIA.

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Chemistry.

As urine containing albumen may be either acid, neutral, or alkaline, the most common, and, at the same time, the best, method for detecting this substance is to employ heat and nitric acid, at first separately, afterwards combined. In doing so, a drachm of urine is put into a test-tube, and boiled. Should the liquid thereby become turbid, albumen is most probably present, but not necessarily so. To decide this point, therefore, a few drops of nitric acid are added; and should the turbidity caused by boiling be increased rather than diminished on the addition of the acid, it is due to the coagulation of albumen. If, on the other hand, the turbidity either wholly or in part disappears, it has, in all probability, been due to the precipitation of some earthy phosphates or carbonates,—two inorganic substances which are often mistaken for albumen, in consequence of their being readily precipitated from neutral or semi-alkaline urine by heat. The nitric acid re-dissolves both of these precipitates, the phosphatic without, the carbonatic with, effervescence. Although nitric acid precipitates albumen from urine without the aid of

heat, it must never be used alone; for when employed *per se*, it is a most fallacious test, in consequence of its giving an amorphous precipitate of uric acid in urine containing a great excess of urates, and a crystalline one of the nitrate of urea in urine loaded with that substance. The uric acid precipitate is frequently, the nitrate of urea precipitate rarely, mistaken for albumen. The application of heat in either case removes the difficulty by dissolving the precipitate, and enabling the substance to reassume its characteristic crystalline form during the cooling process.

In consequence of the above-mentioned source of fallacy in testing for albumen with nitric acid, some authors recommend the substitution of acetic acid; but it, too, has its drawbacks, for acetic acid throws down a precipitate of cystin when that substance exists in quantity in human urine; and if uric acid be in great excess, acetic acid is even sufficient to precipitate it. The microscopic examination of the deposit in all cases of doubt allows of its true nature being ascertained.

In testing human urine for albumen there is yet, unfortunately, another source of error—namely, the possibility of overlooking its existence when it is actually present. This may arise from two causes; one being the nature of the albumen, the other the condition of the urine.

In afterwards speaking of its physiology, it will be shown that one particular kind of albumen is a normal constituent of healthy urine, but as that variety of the substance is neither coagulable by heat, nor precipitable by nitric acid, we have nothing to do with it at present. What I now allude to, is the fact, that in the urine of disease albumen may exist, and yet escape detection, unless certain precautions be employed in order to ensure success.

In the first place, in alkaline urine a *small quantity* of albumen is not coagulable by heat; hence the necessity of always adding acid in order to avoid this source of error.

In the second place, some varieties of abnormal albumen are not coagulated by heat when only a little nitric acid is added, the addition of an excess being requisite to ensure the precipitation. On the other hand, another less common kind is occasionally met with where a great excess of acid redissolves the coagulum.

If, then, we wish to avoid these sources of error, when a discrepancy seems likely to arise, we must test repeated portions of the urine with different quantities of acid—say, with five, fifteen, and thirty drops of nitric acid to the drachm of urine. Thus, and thus only, are mistakes likely to be avoided.

There are still other reagents the employment of which in cases of doubt is attended with advantage. Such, for example, as absolute alcohol, chloroform, tannic acid, alum, acetate of lead, bichloride of mercury, and a variety of other solutions of the mineral salts—almost all of which precipitate albumen. None of these are, however, in experienced hands, at all to be compared, in point of accuracy, with heat and nitric acid.

The effect of the presence of sugar in causing albuminous urine to give a fine mauve colour with the sulphate of copper and potash has already been pointed out.

Specific Gravity.—It is generally stated that the specific gravity of albuminous urine is very low. But this is by no means invariably the case. In fact, it only holds good with one form of albuminuria, namely, that consequent upon structural change in the kidneys. A low specific gravity, therefore, aids in distinguishing that from the numerous other forms of albuminuria, in some of which I have met with it as high as 1032, or even 1035. These, however, were quite exceptional cases—the former being one of intermittent hæmaturia, the latter due to the co-existence of diabetes. In what is commonly called Bright's disease, the specific gravity ranges between 1005 and 1014. The usual average being 1010—12. In the other forms of albuminuria the specific

gravity of the urine has no limit, it may be as low as 1005, or, as just said, as high as 1035.

The reason why the urine in certain cases of kidney disease has such a low specific gravity, is on account of the disorganised renal tubes being incapable of eliminating the urea and other urinary crystalloids. The lower, therefore, the specific gravity in these affections, the more dangerous is the case.

Quantitative Analysis.—In all cases of albuminuria a quantitative analysis is an invaluable aid to prognosis and treatment. It is best done by measuring off 50 c.c. (about two ounces) from the twenty-four, or, better still, from the forty-eight hours' urine of the patient, and adding it drop by drop to a couple of ounces of boiling distilled water acidulated with acetic acid. The capsule should be placed over the spirit-lamp, and its contents kept boiling vigorously the whole time the urine is being added. A drop or two of acetic acid should occasionally, during the process, be allowed to fall into the boiling mixture, in order to ensure its being always faintly acid. When the coagulation of the albumen is completed, the capsule is to be put aside, to allow of the coagula falling to the bottom of the vessel, and thereby facilitating the next stage of the process, which is the collection of the precipitate on a filter and thoroughly wash it with distilled water. After this, while still moist, the albumen is to be transferred to a watch-glass, dried, and weighed. The weight of the dried albumen represents, of course, the quantity in 50 c.c. (about two ounces) of the patient's urine.

The calculation for the twenty-four hours' urine is then simple enough. Suppose, for example, the quantity of urine passed be 1500 c.c., and the weight of dried albumen 0·2 grammes, then

$$\frac{0{\cdot}2 \times 1500}{50} = 6 \text{ grammes of albumen in the twenty-four hours.}$$

The quantity of albumen passed varies considerably in diffe-

rent cases, at different times, and in different forms of the disease. It may be from 1 to 2 grammes (15 to 30 grains) only, or it may amount to from 20 to 30 grammes (300 to 500 grains) in the twenty-four hours. In average cases the amount leaves about 10 grammes (150 grains) daily.

To follow the above method time and apparatus are alike requisite. By those, therefore, who have neither the one nor the other at their disposal, the following mode of procedure may be adopted; it may be called the "rough and ready method," and, although far inferior to the other, is yet much better than none at all:—Take the twenty-four hours' urine of the patient and dilute it till it measures 3000 c.c. (100 ounces).(a) Then, to two drachms of this diluted urine, in a test-tube, add ten drops of nitric acid, either after or before coagulation by boiling. When this is accomplished, place the tube aside until the precipitate is all deposited. The amount of coagulum yielded daily gives a rough relative approximation of the quantity of albumen passed, and by preserving the test-tubes, and comparing their contents from day to day, a tolerably fair idea of the progress of the case may be obtained.

In these cases, in consequence of the dilution of the urine, the coagulum seldom looks to be much. Having now not only ascertained that the urine of the patient is albuminous, but even the amount of coagulable matter passed, we are still but on the threshold of our diagnosis. The source and cause of the presence of the protein substance has yet to be discovered.

The coagulable matter found in the urine may have come directly from the serum of the blood, as in Bright's disease; it may be the result of a lesion of the kidney, as in renal calculus; it may be the product of inflammatory action, as in

(a) The amount of dilution is immaterial so long as it is always the same. Few patients pass more than 100 ounces of urine in the twenty-four hours; hence, I have made it the standard of comparison.

cystitis, or the secondary result of a variety of affections quite independent of renal disease. Many things have therefore to be taken into account ere we can arrive at a correct diagnosis even in a case of albuminuria. Before touching upon them, however, I must first say a few words regarding the physiology of our subject.

Physiology.

In studying albuminuria, it is well to bear in mind that the presence of albumen in the blood is a normal condition, while its existence in the urine is always abnormal. When I say the existence of albumen in the urine is abnormal, I mean of a kind and in a quantity sufficient to be detectable by heat and nitric acid. There is always albumen in healthy urine, but like the albumen found in the stomach, after being acted upon by the gastric juice, it is neither coagulable by heat, nor precipitable by nitric acid. I believe it, in fact, to be the effete albumen of our blood, excreted like urea or any of the other products of tissue metamorphosis.

Gigon was the first to call attention to the presence in healthy human urine of a substance coagulable by chloroform. He thought it was ordinary albumen, and that it had been previously overlooked in consequence of the tests not being sufficiently delicate for its detection. This, however, can scarcely be the case, seeing that we pass on an average 2·6 grammes (40·3 grains), in the twenty-four hours of this coagulable matter. Becquerel (a) says the substance described by Gigon(b) is simply mucus; but this cannot be the case, as Gigon finds that chloroform gives a precipitate when shaken with urine from which all the mucus has been removed. He has even shown that the same thing occurs in urine taken direct

(a) *Comptes Rendus*, Nov. 21, 1857.
(b) *Union Med. de Paris*, No. 12, 1858.

from the pelvis of the kidney, in which there is no mucus. Moreover, if the chloroform precipitate be collected, dried, and redissolved in acetic acid, it gives with ferrocyanide of potassium the reactions of albumen. In experimenting on this subject I found that absolute alcohol is even a better agent than chloroform, by which to extract this albumen from the urine, and by its means I was enabled to confirm Gigon's statement regarding the presence of albumen in urine devoid of mucus. The conclusion, however, which I arrived at was, as before said, that this albumen is not like that met with in the urine of disease, but like the albumen which after having undergone the modifying action of the gastric juice, is neither coagulable by heat nor nitric acid.

Experiments on animals have shown that unless the albumen of our food has been properly modified during the digestive process, it cannot be assimilated by the tissues, but acts in the blood like a foreign material, and is as such eliminated by the kidneys. Thus, for example, it has been found that when the albumen of the hen's egg is injected into the blood of dogs, it does not become incorporated with the tissues, but is rapidly thrown off along with the urine; whereas if the same albumen be put into the animal's stomach, and digested ere it enters the circulation, it is assimilated, and, consequently, does not appear in the urine in the shape of albumen coagulable by heat and nitric acid. This physiological fact gives us a clue to the well-known circumstance that temporary albuminuria often follows upon disordered stomachal digestion. Stomachal digestion being not only a dissolving, but a transforming process, we can readily understand how, when this transforming process is interrupted, either in consequence of the unsuitable quality of the food, or of some derangement of the digestive system, albumen may be absorbed into the circulation, not sufficiently changed to enable it to become incorporated with the frame,

and therefore, as in the case of the dog, appear in the urine.

There can be no doubt that the albumen found in albuminuria is not always in the same form. The mere fact, already alluded to when on the chemistry of the subject, of its occasional capricious behaviour towards heat and nitric acid is sufficient to prove this. There can be no doubt that even the urine itself has a powerful effect in modifying the reactions of albumen. Mr. Alfred H. Smee (a) has shown that while a current of oxygen gas passed through ordinary albumen, or albumen from the fluid of a spina bifida, at the temperature of the human body (98° F.) transforms a certain amount of it into fibrin, the same gas fails to produce any such result when passed through albuminous urine, even when the albumen is in so large a quantity as almost to solidify the entire urine when boiled.

Albuminuria may be produced by the physiologist artificially in animals by a variety of operative procedures. Section of the renal nerves in rabbits produces albuminuria (Michel). Division of the cerebral peduncles (Schiff) as well as section of the fifth pair of nerves within the cranium, is followed b a like result. Section of the spinal cord in the dorsal region of dogs I have found to be followed in the course of a few hours by albuminuria; and all experimental physiologists are aware that puncturing the calamus scriptorius of rabbits, often times causes the urine to become highly albuminous. It has frequently happened to me also to bring on an albuminuria in animals, into whose portal circulation I had injected stimulants, for the purpose of rendering their urine saccharine.

In all these cases the albuminuria is most probably the result of the congestion of the kidneys, which almost invariably, to a greater or less degree, follows upon the above mentioned operative procedures.

(a) *Proc.* Royal Society, June 16, 1864.

Pathology.

Since the introduction of the improved methods of chemical and microscopical investigation, the subject of albuminuria has undergone an entire revolution, and the time may now be said to have arrived when a philosophical arrangement of the different pathological conditions giving rise to it is not only desirable, but absolutely necessary. To grapple with preconceived notions, trample upon cherished theories, re-arrange data, and promulgate new principles, is, no doubt, a task not to be entered upon without serious consideration. Nevertheless, as we all know, if Medicine is ever to take her place among the exact sciences, some one of her votaries must gird on his armour, and boldly run the risk of obloquy in attacking old dogmas. While waiting the advent of a more able exponent, I shall, trusting to the support of the emancipated among my brethren, attempt to handle the subject of albuminuria in a manner similar to that in which I have elsewhere treated jaundice.

Unfortunately, however, at the very outset, there is an important difficulty to be overcome as regards the meaning which should be attached to a well-known title of Kidney disease. The united labours of Basham, Johnson, Christison, Virchow, Parkes, Bennett, Goodfellow, Begbie, Aitken, Quain, Stewart, Dickinson, Wilks, and others have shown that the term "Bright's disease" is at present indiscriminately applied to widely-differing pathological conditions, such as nephritis, cirrhosis, fatty and waxy degeneration of the kidneys, etc.,—renal affections which have no clinical connexion beyond what they derive from the associated symptoms of albuminuria and dropsy. If we desire to keep pace with the progressive advance of science, it is absolutely necessary for us either to limit the term "Bright's disease" to one particular form of renal affection, accompanied

with albuminuria and dropsy, or to abandon it altogether. The latter alternative I am in no wise inclined to adopt, for the name of the man who first discovered the connexion of dropsy, albuminuria, and renal disease is, I opine, well deserving of being handed down to all time. I therefore would venture to propose that the name of Bright be perpetuated by associating it with that form of disease which is not only the one most frequently met with, but also that with which Dr. Bright was most familiar, namely, the albuminuria of nephritis. By so doing, we shall not only be enabled to make a philosophical arrangement of the different morbid states upon which albuminuria depends, but at the same time render justice to an honoured and deserving memory.

In studying albuminuria, there are two things that must always be borne in mind: first, that there are certain forms of albuminuria with dropsy, and certain forms without it; and, secondly, that all cases of permanent albuminuria are, in the long run, always associated with an organic change of structure in the tissues of the kidneys, and that even in those cases in which the renal organs were not primarily at fault, as, for example, when the albuminuria springs originally from passive portal congestion induced by heart or liver disease, paraplegia or hemiplegia. Contrary to what some authors affirm, I assert that permanent albuminuria is in all cases the *result*, and not the *cause* of renal derangement, just in the same way as pus is the *result* not the *cause* of an abscess. And just as an abscess may be due to a great variety of causes, so may renal derangement originate in a number of different morbid conditions—some constitutional, some local. When Johnson, Walsh, Aitken, and other authorities, say that the textural changes in the structure of the kidneys are only the local expression of the constitutional disorder, as suppuration in a gland may be but the local expression of constitutional struma, I perfectly agree with them; but when they go so far as to

say that the excretion of albumen by the kidneys is due to a constitutional cause, and not to a local structural change, I beg to differ from them *in toto*. 'Tis true that in a limited number of cases of albuminous urine the healthy kidney is only eliminating a foreign material, for, as already shown in the physiological part, when mal-digested albumen enters the blood, the tissues of the body being unable to assimilate it, the renal organs are forced to excrete this form of albumen as they would any other noxious material; but these cases are not only few in number, but have no similarity whatever with the pathology of the ordinary forms of permanent albuminuria. So small, indeed, is the proportion of cases of permanent albuminuria in which the primary cause cannot be directly traced to the kidney itself, that they do not even, as a whole, amount to more than 20 per cent.

With these few remarks as a proviso, I may at once enter upon my task, and as, the great art in explaining a difficult subject is to use as few and as simple words as possible, I shall venture to put my views of the pathology of albuminuria, as I did those of jaundice, into a tabular form. It must be borne in mind, however, that as all diagrammatic classifications labour under the disadvantage of being more or less arbitrary, it will be necessary, in order that the views of the pathology of albuminuria here enunciated be thoroughly understood, that the chemistry and physiology of the subject be previously studied. Moreover, if the subjoined table is to be turned to practical as well as theoretical account, the explanations to be subsequently given of the different forms of albuminuria, as met with at the bedside, must be carefully considered.

ALBUMEN IN URINE.

- **Normal** (not detectable by heat and NO^5)
  - Always during health small quantity.
  - Detectable by absolute alcohol and chloroform.
- **Abnormal** (Detectable by heat and NO^5).
  - **Primary Albuminuria** (cause originating in kidney).
    - Temporary
      - Bright's disease.
        - Traumatic,
        - Idiopathic, and
        - Scarlatinal nephritis.
    - Permanent
      - Fatty,
      - Waxy,
      - Chronic hypertrophy.
      - Cirrhosis.
      - Cancerous,
      - Tuberculous, and
      - Scrofulous degeneration.
      - Suppuration
        - Simple abscess.
        - Renal calculus.
  - Digestive System
    - Improper food.
    - Imperfect digestion.
  - Nervous System
    - Hemiplegia.
    - Paraplegia.
    - Injury to brain.
    - Lesion of renal nerves.
    - Pregnancy—Reflex nerve irritation.

- **Secondary Albuminuria** (cause not originating in kidney).
  - **Organic Disease** (Passive renal congestion).
    - Cardiac.
    - Hepatic.
    - Pancreatic.
  - **General Affections** (Active renal congestion).
    - Febrile.
      - Small-pox,
      - Typhus,
      - Typhoid,
      - Scarlet,
      - Yellow,
      - Puerperal,
      - Rheumatic, and
      - Remittent fevers.
    - Non-febrile.
      - Pneumonia.
      - Pleurisy.
      - Bronchitis.
      - Phthisis.
      - Peritonitis.
      - Gout.
      - Scurvy.
      - Erysipelas.
      - Purpura.
      - Cholera.

With the albumen present in the urine of health we have now nothing to do, so I at once pass to the consideration of the first great class; namely—*Primary Albuminuria*, that form of the affection in which the cause of the albuminuria is to be found in the kidney.

As indicated in the diagram, primary albuminuria naturally divides itself into two well-marked varieties—a temporary and a permanent variety—the former being by far the most common, the latter the most dangerous. Each of these will now be specially and separately considered.

Bright's Disease.—The pathology of this temporary form of primary albuminuria is simple enough, and may be summed up in one word—namely, "nephritis;" for, be the exciting cause what it may, the immediate cause of the albumen in the urine is an engorged or an inflamed condition of the kidney. Thus, for example, it may be the result of direct injury to the loins, or arise from an attack of cold, either in a perfectly healthy individual or in one rendered abnormally susceptible to the affection by intemperance or other debilitating cause; and lastly it may follow upon scarlatina, in consequence of the kidneys having an excess of work precipitately thrown upon them by a sudden check being given to cutaneous exhalation. The diagnosis of the nephritic form of albuminuria, which is almost always acute and curable, is in general easy; for, besides the history of the case, we have a most important guide in the condition of the urine.

Should the nephritis be the result of injury, the case is clear enough, for pain in the loins, the albuminous urine, and the dropsy will be found to follow in its immediate wake. On the other hand, when the disease is the result of cold and wet, it is sometimes so insidious in its march as to give no indication of its presence until the existence of dropsy attracts the attention of the patient. In the most common of this class of cases, however, the general history is, that after exposure to

cold the patient suffers more or less malaise; is chilly and feverish, occasionally even shivering; has pain in the lumbar region, with scanty and high-coloured urine; next a puffiness is noticed under the eyes, the feet begin to swell, and the belly is observed to be enlarged. Should the attack be a severe one, not only do the limbs become decidedly œdematous, but the abdomen gets so filled with fluid that the patient is unable to wear his ordinary clothes. All this may occur in a very few days, and what is still more alarming to the patient and his friends is, that the effusion does not always stop here, but continues rapidly to extend until the whole trunk and upper extremities are included in one general anasarca. Before this time, however, Medical aid is generally sought, and the progress of the dropsy thereby arrested. In men, I have occasionally observed that one of the most distressing results of the effusion has been an excessive œdema of the genital organs, the prepuce becoming, in some cases, so enlarged and distorted as to obstruct micturition. At other times the liquid is chiefly diffused into the cellular tissue of the scrotum, giving to it the appearance of an immense scrotal hernia. In the spring of 1864 a little boy, aged 8 years, was brought to me at University College Hospital with the cellular tissue of his scrotum so distended with fluid that it was nearly as large as his head, the tense skin being at the same time so bright and glistening, and the effusion so transparent, that most of the students took it for an enormous hydrocele. As I pointed out to them, however, the diagnosis between these cases and hydrocele is very easy; for in the one case the fluid is in the subcutaneous cellular tissue only, and consequently the skin retains the impress of the finger; while in the other, the fluid contained within the fibrous tunica vaginalis, prevents the retention by the tissues of the impress of pressure, however firmly it may have been applied. It has been hinted that we frequently meet with cases of Bright's diseases with very little or no pain,

or even malaise. I may now add that we occasionally encounter others in which the œdema, especially at first, is so trifling that it would fail to awaken alarm in the patient, were it not accompanied with considerable discomfort, and scanty and high-coloured urine. The smoky, red, or bloody-looking urine at the same time generally yields a copious dark-coloured deposit on standing, which further aids in arousing the suspicions of the patient, which suspicions the Medical attendant is able, by the aid of a little nitric acid and heat, at once either to confirm or dispel. To diagnose the case properly, however, it is not alone sufficient to prove the existence of albuminuria; for that, as seen in the table, may occur under a great variety of circumstances, and even when the urine is so loaded with albumen as to allow of its being transformed into a solid mass—so solid that the tube may be inverted without danger of any falling out—we are still unable to certify to the existence of renal disease until we have called the microscope to our aid. If the case be one of Bright's disease, in the urinary sediment will be found blood corpuscles, epithelium scales, renal tube casts with their epithelial lining, amorphous urates, and, perhaps, even crystals of uric acid or oxalate of lime.

FIG. 1.

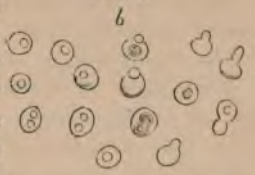
There are two points to which I must here direct special attention, otherwise difficulties which need not exist may come in the way of beginners. In the first place, it occasionally happens that a number of granular cells are found in

the field which look very like pus corpuscles (Fig. 2). They are bigger than the red blood cells, and smaller than the epithelium. These, in the majority of instances, are simply white blood corpuscles, and are most numerous after meals. In some few cases, when the nephritis is accompanied by vesical catarrh, mucus cells are likewise abundant. In others, again (as in that of a child aged 3 years, four weeks after scarlatina and fourteen days after the first appearance of the dropsy began) the white blood corpuscles are actually more numerous than the red. This, however, is rare, for during fifteen years special observation I have only once met with it. The increase in the number of white corpuscles after food is common enough, and is readily accounted for on physiological grounds, it being now an admitted fact that after every meal a large excess of white corpuscles are poured into the circulation.

Fig. 2.

The second source of difficulty to the beginner consists in the fact that in some instances the blood corpuscles appear nucleated. A remarkable case of this kind fell under my notice in June, 1859, the patient, a man aged 32, being at the time under the care of my colleague Professor Hare. The albumen in the urine had begun to diminish, the tube casts and free epithelium to disappear, when the red corpuscles were noticed to have the appearance presented at Fig. 3.

Fig. 3.



Some of the corpuscles possessed one, others two perfectly distinct maculæ. By slightly altering the focus of the lens a halo became apparent round each macula, which then assumed the characters of a black spot in the centre of a bright ring. It was further observed that the spot was not in the interior

of the corpuscle, but only imbedded in its cell-wall, the appearance of its being in the centre of the corpuscle arising simply from the position in which the cells happened to be viewed. In many of the discs the spot was seen, as in the figure, to project beyond the cell-wall, thereby carrying, as it were, the latter with it. This irregularity in the form of the blood corpuscles I have seen still better marked in a case of hæmaturia. Some looked exactly like the developing nucleated blood corpuscles of the human embryo.

The peculiar appearances above described, which may prove so puzzling to the beginner, are due to the action of the saline urine upon perfectly normal blood cells. In 1860 Dr. Addison showed that sherry wine in certain proportions induced projections from the walls of blood discs, and in 1863 Dr. Roberts threw additional light upon the subject, by discovering that solutions of tannic acid and magenta bring into view a vesicle in the cell-wall of nucleated, as well as non-nucleated blood corpuscles. The inner vesicle being analogous, he thinks, to the primordial utricle of vegetable cells.(a)

Till now I have spoken of Bright's disease as it affects both kidneys simultaneously, but it occasionally, though much more rarely, happens that it only attacks one, and in this case there is albuminuria, and all the microscopic appearances already described, *but no dropsy*. Anasarca, in fact, only makes its appearance when not alone both, but the whole of both kidneys are so much affected as to be unable to excrete the urinary products. So long as one kidney remains healthy, these manage to escape from the circulation, and dropsy does not appear. This, indeed, is, I believe, the true explanation of the occurrence of those cases of Bright's disease without dropsy which have hitherto been considered so anomalous. Having diagnosed the case properly, the next thing is to watch its course, and try and guide it to a successful termination.

(a) *Proceedings* of the Royal Society, 1863.

The progress of the disease is best learned from the condition of the urine.

The first sign of improvement is a diminution in the number of the blood corpuscles; the second, an increased elimination of urine, which is soon followed by a decrease of the dropsy. The quantity of urine daily voided after the first stage of the attack has passed is sometimes very large, being far beyond the normal standard. Occasionally, the twenty-four hours' urine of a patient in the second stage of Bright's disease will amount to something above 3000 c.c. (more than 100 ounces). And, what is still more important, is the fact that with this excessive elimination of water, there is often a corresponding excessive elimination of the organic and inorganic salts. Luckily for the patient it is so, it being this accumulation in the blood of these excrementitious products which in general is the immediate cause of a fatal termination. In one case, three weeks after the commencement of the attack, or, I should rather say, of the onset of the dropsy—for it was the first thing the patient observed—Mr. Alexander Bruce, my former assistant, found that the patient—a young woman, aged 29,—passed the following quantity and quality of urine:—

Water . . .	2525 c.c.	= (81½ ounces.)
Specific gravity .	1012	
Urea . . .	25·25 grammes	= 391·37 grains.
Uric acid . . .	0·233 ,,	= 3·6 ,,
Phosphoric acid .	8·837 ,,	= 137·0 ,,

And this, too, be it remembered, when the patient was taking very little food. As a case goes on improving the albumen diminishes, the specific gravity of the urine rises, and the tube-casts disappear.

Should, unfortunately, the affection resist the action of remedies, and run on to a fatal termination, which is rather the exception than the rule, the kidneys will be found enlarged, congested, sometimes softened, and always readily denuded of

their capsules; but the capsule often has portions of the cortical substance adhering to it. They may weigh in very bad cases as much as twice the normal amount (which is about 10 ounces for the two). When examined microscopically, small irregular punctiform extravasations will be found scattered among the urine tubes. As regards the urine tubes themselves, some will be found entirely denuded of their epithelial lining, others distended and blocked up with detached epithelial cells, blood corpuscles, and amorphous granular matter, giving to them a well-marked dark outline, readily recognisable both in longitudinal and transverse sections.

The treatment of cases of Bright's disease will be considered after the other varieties of albuminuria have been explained. Meanwhile, it may only be remarked that the form of Bright's disease here described is one of the most amenable to treatment of all renal affections.

We now pass to the consideration of the *permanent* variety of *primary albuminuria*, which is, as shown in the table, met with in the following morbid conditions of the kidney:—Chronic hypertrophy, fatty, waxy, cirrhosis, tuberculous, scrofulous, and cancerous degeneration.

The chronically enlarged kidney, which is by far the most common accompaniment of permanent albuminuria, is in general the result of repeated attacks of inflammatory action (Bright's disease). It is likewise the ultimate product of prolonged passive renal action, such as arises from cardiac and hepatic disease. Besides this, however, all constitutional as well as local affections (fevers, tumours, calculi, etc.) which produce congestion of the kidneys may be said to pre-dispose to their permanent hypertrophy.

The simply hypertrophied kidney is exceedingly difficult to diagnose during life from the circumstance that so long as the enlarged organ remains in a quiescent state—that is to say, in an uncongested condition, or is not the seat of any

acute inflammatory attack—there are no symptoms sufficiently characteristic to admit of its ready detection, for the urinary secretion may be copious, normal in colour and specific gravity, as well as entirely free from any deposit; so that unless some accidental circumstance directs the attention of the Physician to the kidneys, the existence of albuminuria may be readily overlooked. Even the œdema of the ankles is often in such cases so trifling as to escape the notice of both patient and Medical attendant, until some slight attack of renal congestion specially calls attention to the state of the kidneys and their secretion.

Regarding the morbid anatomical condition presented by the hypertrophied kidney, little need be said, for, except being much beyond the usual size, it looks to the naked eye exactly like a normal kidney. At least, this is the condition met with when the patient is suddenly cut off by some other disease. In other cases, again, the hypertrophied kidney is found engorged with blood, softened, and friable, in consequence of its being, or having recently been, the seat of an acute inflammatory attack. It is, indeed, surprising how prone the enlarged kidney appears to become congested, and even excessively so, without any very apparent cause.

When in a congested state it pours out large quantities of albumen, blood corpuscles, and epithelial tube casts. The urine at the same time becomes scanty and high coloured, the feet and legs rapidly increase in diameter, and the patient presents all the appearances of a well-marked case of Bright's disease.

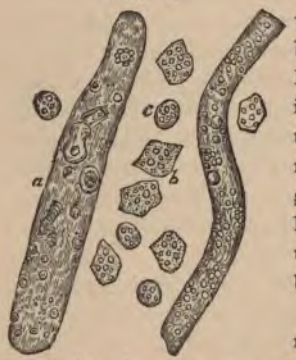
Moreover, the hypertrophied kidney is frequently the precursor of both the fatty and the waxy forms of degeneration, and the almost invariable forerunner, I believe, of the cirrhosed state of the organ.

Fatty Degeneration.—Although the fatty form of kidney is frequently the result of chronic inflammation, it may occur quite independently of it, just as we find fatty degeneration arising from constitutional causes in other organs of the body.

Hence we often find that fatty kidney is the accompaniment of phthisis, of heart disease, of fatty liver, etc.

Its most characteristic symptoms are permanent albuminuria and dropsy, but as in the early stage of the attack it is impossible to say whether the albuminuria is to be persistent or not, these symptoms of themselves teach us but little. It is to the microscopic condition of the urine that we have to look for further information, and, fortunately, we need not look in vain, as a peculiar form of sediment not only reveals to us the nature of the pathological lesion, but even indicates the stage of the renal disorganisation. In the first place, the presence of oil globules in the tube-casts, and in the free epithelium, tells us that the albuminuria is the product of a kidney in a state of fatty degeneration; while, secondly, the amount of the oil globules and the quantity of albumen, together with the special character of the tube-casts present in the fluid, indicate the exact stage of the affection. Lastly, the supervention of an acute inflammatory attack upon a fatty kidney is at once made manifest by the existence of blood corpuscles in the urine, in addition to the presence of oily tube-casts and epithelium.

Fig. 4.



The accompanying drawing, which was kindly made for me by my former pupil, Mr. Ed. De Morgan, is a very faithful illustration of the condition of the tube-casts and epithelium met with in cases of fatty degeneration of the kidneys. The history of the case from which this drawing was taken is briefly as follows:—

A labourer, aged 36, was admitted into University College Hospital, under my care, on October 4, 1864. He acknow-

ledged to have been a very intemperate man, but stated that he had, nevertheless, generally enjoyed good health. He could give no very definite account regarding the commencement of his illness, except that he noticed his legs first begin to swell about three months before his admission, from which time his health had gradually failed. There was no account of his having at any time had an acute attack of nephritis, nor of his present illness having been due to exposure to cold or wet. On admission, the legs and abdominal walls were exceedingly œdematous, but the face was only slightly puffy, and there was no anæmia to speak of. The urine was, however, highly albuminous, becoming almost solid on boiling. It was acid, contained oily tube-casts, and epithelium, as represented in the drawing; but there were also a few blood corpuscles and granular casts scattered among them. The case was, therefore, at once diagnosed fatty degeneration of the kidney, with the supervention of an acute inflammatory attack.

The patient continued much in the same state till November 9, when erysipelas, excited by a sore on the face, set in, and from then till December 5 he lingered on, sometimes better, sometimes worse, until he was suddenly seized with rigors and vomiting, followed by a worse outbreak of the erysipelas. He died on the 12th. The quantity of urine passed by this man varied from 70 to 116 ounces, and the specific gravity from 1010 to 1015.

On post-mortem examination made by one of my colleagues, the cortical substance of the kidneys was said to have appeared much increased in width, the line of demarcation between it and the pyramids being almost lost; while under the microscope the substance of the kidney was described as being in a state of granular and fatty degeneration, much free fat being scattered in globules over the field.

Where only one kidney is involved, fatty degeneration may run on to almost any extent, till at length the organ assumes

almost the appearance of a fatty tumour. One such specimen has at least been brought under my observation. The case occurred in the practice of Dr. Hullett Browne. The patient was a boy, aged 11, who had for a considerable time suffered from renal calculus. At the post-mortem examination, the left kidney was found to be $6\frac{1}{2}$ by $3\frac{3}{4}$ inches in diameter, and so completely transformed into adipose tissue that only a mere trace of renal structure could be detected in the centre of the mass, faintly indicating, as it were, the outline of the original kidney. The right kidney was much enlarged, congested, soft, and friable. This was not, of course, an ordinary case of fatty renal disease; but I quote it merely to show to what extent fatty degeneration of the kidney may proceed. If both kidneys are equally affected, death would, of course, inevitably ensue long before the degeneration could extend to anything like the stage here indicated. (a)

Between this and the healthy kidney there is every possible intervening grade of fatty degeneration. In the ordinary run of cases which terminate fatally, the kidney is found somewhat increased in size, greasy to the touch, and pale in colour, the cortical and medullary substances having generally much of the same appearance, and are so blended together that it is often extremely difficult to point out where the one begins and the other ends. A scraping of either part placed in a drop of water and viewed with the microscope presents, in an exaggerated degree, the appearances delineated in the figure —that is to say, the epithelium cells both in and out of the tubes are found loaded with oil globules.

Waxy Kidney.—Although this form of renal degeneration is not so common as the fatty variety, it is tolerably often observed, and is usually associated with a similar degeneration in some of the other organs, more especially of the liver

a) The case is reported in the Path. Soc. *Trans.*, vol. xiii., p. 131. The specimen is in University College Museum; No. 4720 of the catalogue.

and spleen. The waxy kidney is darker in colour than the fatty, and is easily distinguished by its smooth texture and glistening waxy aspect. To the touch it is firm, being sometimes indeed so hard as to take the impression of the nail without tearing. On section, the medullary and cortical substances are found to be blended together, and the malpighian bodies in the latter distinctly marked as translucent spots. The capsule is easily detached, and the surface beneath is smooth, pale, and slightly mottled with small rosettes of red vessels. The diagnosis of this form of renal disease during life is not so easy as that of the fatty variety; still, however, it can be accomplished by carefully examining the urine from day to day, when the detection of what are termed hyaline casts serve to indicate the pathological condition. The casts are often absent for two or three days at a time; then for the next two or three days they may be present. The annexed figure, which was also made for me by Mr. De Morgan, indicates the appearance presented in the ordinary forms of hyaline casts.

Fig. 5.

The patient who passed the casts from which the above drawing was made was a woman, aged 30, with a well-marked syphilitic history (secondary eruption, sore throat, loss of hair, tibial nodes, etc.). She attributed her urinary symptoms to an attack of cold, which induced headache, thirst, shivering, pain in loins, scanty and high-coloured urine, with dropsy. This occurred several months before her admission into the Hospital. At the time the above represented casts were found there was still albumen in the urine, but comparatively little dropsy, the lower limbs only being œdematous, the face having quite a natural appearance. As the waxy is sometimes associated with a partially fatty condition of kidney, it occasionally happens that both hyaline and oily casts are met with in the same patient's urine. This is always an unfavourable complication. Hyaline casts alone, though generally indicating an advanced stage of disease, are by no means invariably indicative of immediate danger. The waxy form of degeneration is sometimes almost entirely limited to one kidney, or portion of a kidney, and however severely affected the part may be, it causes little disturbance so long as the other kidney or portion of kidney, is healthy. With fatty kidney, on the other hand, it is usually the reverse. When once one part becomes affected the disease rapidly spreads; and this seems to be especially the case when the tissues of the organs have already become weakened by the pre-existence of other disease.

The waxy, like the fatty, kidney is prone to take on inflammatory action, and although during the quiescent stage of the disease no blood corpuscles are to be found in the urine, yet every slight attack of temporary renal congestion is apt to induce their appearance.

When examining the urine in a case of waxy kidney, a few epithelial cells are occasionally to be found scattered over the field of the microscope; but as all epithelial cells met with in the urine do not necessarily come from the diseased kidney,

care must always be taken not to fall into an error of diagnosis, by mistaking urethral, bladder, or even vaginal epithelium, for renal. Renal epithelium, like the blood corpuscles, only appears in the urine of the waxy kidney, when an acute inflammatory attack or temporary congestion has disturbed its circulation. Grainger Stewart thinks that blood from the waxy kidney generally contains an excess of white corpuscles, but this, I imagine, can only occur when the spleen is also affected.

Cirrhosed Kidney.—The kidney, like the liver, after repeated attacks of inflammatory action is apt to become cirrhosed and atrophied. All the three varieties of enlarged kidney—the chronically enlarged, the fatty, and the waxy—are liable after a time to shrink and assume a granular appearance, but this is more especially the case with the former. The capsule at the same time becomes thickened, and so adherent to the renal tissue, that when it is being detached it brings away with it portions of the cortical substance, thereby giving to the surface of the organ a very roughened appearance. In acute nephritis, as before said, little patches of kidney substance often adhere to the capsule when it is torn off, but in that case the kidney is larger and softer, while in this case it is smaller and harder than natural. Besides, it has not the same granular appearance on section. The cirrhosed kidney sometimes even looks fibrous, in consequence of the obliteration of many of its tubes and blood-vessels. As the cirrhosed and atrophied kidney is also liable to attacks of inflammation, the appearances here described are not always those found after death, for the case often ends fatally during one of the inflammatory attacks, which of course considerably modifies the anatomical conditions.

The process which leads to atrophy of the kidney is thought to be very slow; some think it may extend over several years, but on this point I have not sufficient data at my disposal to

admit of my offering an opinion. The cirrhosed and atrophied kidney is by no means easily diagnosed during life, for sometimes there is neither albumen nor tube-casts present in the urine during several days at a time, and then it is only the peculiar look, the pale, hydræmic face, the bleached lips, and puffy eyelids, with diminished flow of urine, which lead us to suspect the existence of renal disease. As a rule, the urine is acid and scanty, both as regards the frequency of micturition and the amount passed on each occasion. When tube-casts are detected they are usually small in size and somewhat granular in appearance. Their semi-granular nature has, indeed, led me to the conclusion that their presence in the urine in such cases, is perhaps not so much due to the atrophy or cirrhosis of the organ, as to the existence of trifling temporary congestions, which though insignificant in themselves, are nevertheless sufficient to disturb the balance of the circulation in an abnormally small organ, which even at the best of times has more work thrown upon it than it can well perform. The nature of the congestive granular tube-cast will be afterwards pointed out, when albuminuria arising from portal congestion is considered. We must now pass on to albuminuria arising from tuberculous, scrofulous, and cancerous degeneration of the kidney, regarding which I have only a few words to say at present, as their diagnosis and treatment come much better under the head of bloody and purulent urine.

In *tuberculous*, *scrofulous*, and *cancerous degenerations* of the kidney, the urine is coagulable by heat and nitric acid, not so much on account of the presence of ordinary albumen as in the preceding examples of disease, as from the existence in the urine of organised albumenoid elements—such as tubercle and pus cells, or blood and cancer corpuscles. In fact, in these diseases of the kidney, although the urine is both coagulable by heat and nitric acid, it is only when the other kinds of renal

disease, previously described, supervene in the course of tuberculous, scrofulous, and cancerous degeneration, that the coagulability of the urine dare be said to depend upon the existence of albuminuria, or that the affection ought to be classified in the category now under consideration.

Mr. Thomas Ballard has recorded(a) a very interesting case of encephaloid cancer of the kidney, in which there was the complication just described—namely, permanently albuminous urine of low specific gravity, associated with malignant disease of the kidney. These cases must, however, be rare. Before death, the lady, who lived to the age of 70, had frequent attacks of hæmaturia.

It ought never to be forgotten that ordinary renal inflammation may, and does under certain circumstances, run on to suppuration; but then, if the Medical attendant has carefully watched the progress of his patient by a diurnal microscopical and chemical examination of the urine, the case is easily enough distinguished from one of tuberculous or scrofulous degeneration,—for in the former case the appearance of pus corpuscles in the urine has been preceded by blood corpuscles renal tube-casts, and exudation cells; while in the latter the pus corpuscles have slowly and insidiously made their appearance independently of any inflammatory action. According to Basham, who is a good authority on such subjects, another of the peculiarities of nephritis ending in suppuration is the absence of dropsy. The existence of gravel or stone in an inflamed kidney is always a powerful provocative of suppuration but here, also, we have the previous symptoms of the case to guide us to a correct diagnosis.

Before quitting this subject, I should like to remark that the exact converse of what we have been describing occasionally occurs—namely, that a kidney, the seat of tuberculous,

(a) *Path. Soc. Trans.*, vol. x., p. 188.

scrofulous, or cancerous degeneration, may become suddenly the seat of nephritis, the supervention of which would be at once diagnosed by a change taking place in the urinary sediment, for, in addition to the elements of tubercle, cancer, or pus, the characteristic *débris* of Bright's disease, as already described, would become apparent.

Secondary Albuminuria (Cause not Primarily in the Kidney).

Albuminuria from Imperfect Digestion.—Every one who has had much experience with diseases of the urinary system is aware that cases of temporary albuminuria are occasionally met with in persons whose only symptoms are referable to disordered digestion. It may be that they are usually dyspeptic, or, what is still more frequently the case, they have been indulging in food that has disagreed with them. Thus, cheese has been known to cause albuminuria in children, and lobster or crab in grown-up people. The explanation of this is simple enough, as has already been explained in the physiological part of our subject, and which, therefore, need only be again alluded to here. In these cases there is nothing essentially wrong with the kidneys, and the albuminuria is simply due to the endosmotic equivalent of the albumen absorbed into the circulation being different from what it ought to be. Certain kinds of albumen, as before said, will not stay in the blood unless they have previously undergone modification by the digestive process. Hence, if the necessary modification be interrupted, either on account of the digestive organs being out of order, or by reason of the indigestible nature of the food itself, a temporary attack of albuminuria is the immediate consequence.

The effect of food on albuminurias of all kinds is much more important than most persons imagine; for, as I shall afterwards show when on the subject of treatment, by diet alone we can at pleasure augment or diminish the amount of albumen eliminated with the urine. Nothing further need therefore at present be said on the subject.

Albuminuria from Nerve Lesion or Reflex Irritation.—This, like the last form of the affection, is characterised by the absence of organic change in the structure of the kidneys. But the analogy does not proceed further, for although the albuminuria is due to no structural change, it can, nevertheless, be clearly traced to a disturbance of the renal circulation. It may be remembered that while on the physiology of the subject I mentioned that in the lower animals albuminuria can be produced at will by a variety of operative procedures—such, for example, as section of the renal nerves, division of the spinal cord, lesion of the cerebral peduncles, etc. So we cannot be in the least surprised at the detection of albumen in the urine of patients labouring under similar nerve lesions, the result of disease. As the cause of the albuminuria is apparent enough in the lower animals—namely, renal congestion—there need be, I think, but little doubt that in man the pathology of the morbid condition is precisely similar. Cases of albuminuria in connexion with spinal paraplegia, hemiplegia, apoplexy of the base of the brain, etc., have been so frequently reported, that nothing further need be said upon the subject, but we may at once pass on to the consideration of that form which is least understood—viz., the albuminuria of pregnancy.

Albuminuria of Pregnancy.—Many years ago, when House-Surgeon to the Edinburgh Royal Maternity Hospital, I was led, at the suggestion of Professor Simpson, to examine the urine of every patient that entered the charity during three months. The urine of those who came in after the pains of labour had already begun was immediately drawn off by the

catheter, so that even in their cases all risk of error, from the accidental admixture of vaginal secretions, was avoided, and I was surprised to find how much less frequent the presence of albumen in th eurine of pregnancy was than I had been led to expect from the opinions expressed by previous observers. It did not amount to 4 per cent., which is actually less than a third the percentage of the cases of temporary albuminuria found to exist among female patients admitted into a general Hospital.(a) As regards the frequency of puerperal convulsions, an almost similar conclusion was arrived at—namely, that they are not so exceedingly common as generally supposed, for although I have since seen them both in private and public practice, not one case of the kind occurred among the 450 women who were at that time delivered under my superintendence, and at least ten of whom were passing albumen in the urine. Moreover, even in those cases where the albuminuria is most severe, accompanied by dropsy, and apparently entirely due to the pregnancy, there may still be no convulsions, as the following case will show:—On May 30, 1851, a servant-girl, aged 20, was sent into the Edinburgh Infirmary by a Medical man who thought her case one of Bright's disease. Her urine was highly albuminous, her face puffy, her legs swollen, and her abdomen greatly distended. On passing my hand over the abdomen I imagined that I felt an enlarged uterus, but on suggesting pregnancy, the girl indignantly denied its possibility. It was true that she had seen nothing for the last nine or ten months, but to that she attached no importance, as she had often before been irregular. She had never had morning sickness, never felt any movement, and besides, it was

(a) Among the women admitted into the Medical wards of University College Hospital, Parkes found 12·03 per cent. had temporary albuminuria (*Medical Times and Gazette*, January 1, 1859). Drs. Elliott and Van Arsdale found only two cases of albuminuria in 112 pregnant women that they examined (*New York Journal of Medicine*, 1856).

impossible she could be pregnant. Facts, however, are stubborn things, and fœtal hearts being not usually heard in cases of simple ascites, I quietly, but firmly, told her my opinion that the albuminuria was associated with, if not the direct result of, pregnancy; at the same time adding that she might remain in the Hospital a few days while we tried to relieve the dropsy. In four days she was taken in labour, and within an hour after I was called to her bedside she was delivered of a full-grown healthy child, without a single convulsion or any head symptom whatever. From this time the albumen began to diminish in the urine, the dropsy to disappear, and on June 16, twelve days after delivery, not a trace of matter coagulable by heat and nitric acid was to be found in the urine. On July 14 she was dismissed in apparently perfect health.

I have now to relate two other cases of what is called the albuminuria of pregnancy, but which, as shall afterwards be explained, have an entirely different pathology. The following is an abstract of a case which was published in Germany in 1854.(a) The analyses were made conjointly by the late Dr. Gegenbaur and myself; the clinical history was furnished us by Scanzoni:—

The woman, aged 36, began to menstruate in her fifteenth year, and conceived for the first time in her twenty-fourth year, which conception, pregnancy, and delivery were all perfectly normal. Menstruation recommenced eight weeks after delivery, and continued at regular intervals till February 27, 1853, when she again became pregnant. The patient continued well till July 24, 1853, when she was attacked with ascites and anasarca of the upper and lower extremities, the outer genitals, coats of the abdomen, and of the face. Being

(a) Harley and Gegenbaur: Researches on the Blood and Urine of Albuminuria of Pregnancy. Beit. zu Geburtskunde v. Scanzoni, Hf. II. 1854.

unable to work, she went to the Hospital in Schweinfurt, from whence she was discharged as cured in the course of six weeks. The patient attributed her illness to cold, which she said she had caught from being too lightly clad in damp, cold weather.

On September 18 she entered the Wurzburg Royal Lying-in Hospital, there to await her delivery. At the time of her entrance she was perfectly well, with the exception of an œdematous swelling of the lower extremities, which she ascribed to the long distance she had walked. After remaining fourteen days in the Institution, the patient again became dropsical, and the dropsy continued to increase so that on October 13 she presented the following appearance :—

Her pale face appeared swollen and puffy, particularly around the eyes; the upper extremities, especially the forearm and the hand on the left side, were very œdematous; the abdomen contained much fluid, and respiration was difficult, on account of the compression of the lungs; the heart's action was normal; the skin was extremely dry; the bowels torpid; and the scanty urine loaded with albumen. The fœtal heart was distinct. The patient was ordered a diuretic electuary, to remain quiet in bed, to take moderate nourishment, acidulated drinks, and the swollen extremities to be bound with a linen compress. During the next nine days both the œdema and ascites diminished, while the urine and perspirations increased. The amount of albumen passed was, however, much the same. From October 22, although the patient had ceased taking the medicine, the improvement still continued, and on November 5 2 ozs. of blood were withdrawn from the patient's arm, and the twenty-four hours' urine at the same time collected. The analyses of these fluids, as well as those subsequently made during the patient's illness, are given in the following table, in which, for the sake of more ready comparison, the constituents have been reckoned at so much per thousand :—

Blood.

In 1000 parts of blood were—

	1st analysis on Nov. 5.	2nd analysis on Nov. 17.	3rd analysis on Dec. 7.	4th analysis on Dec. 15.
Water	825·96	829·404	789·220	768·782
Solid matter	174·04	170·596	210·780	231·218
Fibrin	2·30	2·855	2·489	2·858
Albumen	54·16	60·693	65·967	73·431
Blood corpuscles	103·51	95·902	131·219	141·751
Extractive matter	1·22	0·821	2·005	4·884
Inorganic salts	12·78	10·315	9·100	8·287
In 1000 parts of serum were—				
Water	927·32	917·614	911·091	906·000
Solid matter	72·68	82·386	88·909	94·000
Albumen	60·69	67·269	76·154	86·538
Extractive matter	2·91	3·752	4·505	2·462
Inorganic salts	9·08	11·365	8·249	5·000

Urine.

In 1000 parts of urine were—

Water	963·1	971·1	967·3
Solid matter	36·9	28·9	32·7
Inorganic salts	22·9	15·9	17·5
Organic substances	12·88	12·8	15·3
Albumen	9·5	2·9	1·6
Urea	3·39	5·43	10·2

The first analysis was made during the thirty-third week of pregnancy, at which time, although there was not much ascites, there was considerable œdema of the lower extremities. The qualitative analysis of the blood showed that it contained, besides the above-named ingredients, a large quantity of sugar and traces of carbonate of ammonia. The urine collected at the same time had a strongly alkaline reaction and nauseous

smell—facts which led to the conclusion that the transformation of some of the urea into carbonate of ammonia was taking place in the circulation. On microscopical examination, the urine was found to contain a number of pale tube-casts, as well as numerous epithelium cells.

By the time the second analyses were made—twelve days later—the condition of the patient had improved, her complexion was more natural, and the dropsical swelling had considerably diminished. The blood no longer contained carbonate of ammonia, and only a small quantity of sugar was present. The urine had now become almost normal. The quantity had increased from 1169 cubic centimetres (38 oz.) to 1668 cubic centimetres (54 oz.), and the specific gravity had at the same time fallen from 1043 to 1025. Besides this, there were no longer any tube-casts to be detected. The improvement in the condition of the patient went steadily on until December 1, when she was safely delivered of a full-grown healthy child, without the occurrence of any nervous complication. On December 7—that is, seven days after delivery—the condition of the blood and urine, as shown by the third series of analyses, was still more favourable. The total quantity of urine then passed was 1792 cubic centimetres (58 oz.), the specific gravity 1015, and the reaction decidedly acid. Ten days after the confinement, the health of the mother was considered so good that she was sent as wet nurse to a resident family. Five days later, at the request of Scanzoni, she returned to the lying-in Hospital, and had over 2 oz. of blood abstracted from her arm, the result of which is given in the fourth table. Now, if we compare the first analysis of the blood with the figures given by Scherer of normal blood, the increase of the watery, together with the decrease of the solid constituents is very remarkable. The albumen is decidedly under the average of that found in non-pregnant females. The quantity of blood corpuscles is likewise less,

whereas the fibrin and salts are more abundant than in normal blood. The quantity of solid matter in the serum is also much less, but the difference is here caused by the smaller quantity of albumen present, since the amount of salts approaches that in normal blood. The result of the second analysis corresponds with the improvement of the patient: there being *a decided increase of the albumen*, and decrease of the salts in the blood. The third analysis of the blood, seven days after delivery, shows, in comparison with the two former analyses, a considerable diminution of the water, with a corresponding increase of solid matters; the fibrin is, however, less than in the second analysis. The great increase of albumen and blood corpuscles exactly correspond to the decrease of the albumen in the urine. In the fourth analysis, the water of the blood is seen to be still further diminished, and the solids at the same time as notably increased. The quantity of the salts is again less than in the previous analyses, so that all the analyses show a continuous decrease in these constituents.

The general conclusion to be drawn from the foregoing investigations is:—

1st. That in the albuminuria of Bright's disease there is less than the normal amount of albumen in the blood.

2nd. As the amount of the albumen in the urine increases, the quantity in the circulation proportionately diminishes; and as the albuminuria decreases, the amount in the circulation gradually reapproaches the normal standard.

3rdly. The quantity of albumen and urea in the urine are in inverse proportion to each other—that is to say, where there is much albumen there is little urea, and where there is much urea there is but little albumen.

Lastly. The condition of the urine affords us a pretty correct idea of the probable condition of that of the blood.

I have now to point out an error into which people are very liable to fall—namely, to look upon the albuminuria of preg-

nancy, and pregnancy with albuminuria, as one and the same thing, while, in reality, they are perfectly distinct. When we speak of the albuminuria of pregnancy, we mean that the pregnant state induced the albuminuria, as was the case in the example first cited; whereas when we speak of pregnancy with albuminuria we simply mean that a woman during the period of her pregnancy has been attacked with kidney disease, as in the case last cited.

I have now to call attention to a third form of albuminuria and pregnancy—namely, that in which a patient already the subject of kidney disease becomes pregnant. This is not only a graver complication than the "albuminuria of pregnancy," but, as a rule, even more dangerous, both to the life of mother and child, than a case of pregnancy complicated with kidney disease. The following case, of which I can only give a very brief abstract, will illustrate this remark :—

The subject was a lady, aged 30, who I saw in consultation with Dr. Magrath, of Teignmouth, and to whom I am indebted for the patient's history. Her mother died of diseased kidneys at the age of 45. The patient herself was healthy until she suffered from frequent attacks of intermittent fever whilst abroad. During her first pregnancy her legs were noticed to be œdematous, but, as albuminuria was not suspected, the urine remained unexamined. Her delivery was followed by a convulsion, for which she was bled to sixteen ounces. During the second pregnancy and confinement, nothing remarkable was observed, but after the third delivery she was again seized with convulsions, which were on this occasion arrested with chloroform. In the early part of 1863 she again conceived, and almost immediately before or afterwards, was, while recovering from a catarrhal fever, seized with hemiplegia. In the following month she was seen by Dr. Brown-Séquard, and, under the free administration of tonics, with change of air, she gradually regained the power of her arm

and leg. In August of the same year, shortly before I saw her, her face and hands were for the first time observed to be œdematous. The urine was of a specific gravity of 1020, and was not noticed to contain albumen. About this time she suffered from headache, vomiting, with stertorous breathing, and muscular twitchings during sleep. At the end of August, when I examined the patient, the specific gravity of the urine was 1014, it contained a large quantity of albumen, coagulating to one-third of its volume, and with the microscope both blood corpuscles and renal tube-casts were detected. As the symptoms of uræmic poisoning continued to increase until the breath had a urinous odour, the induction of premature labour was had recourse to, during which she had a convulsion, which was arrested with chloroform. After this the patient made a slow recovery until October 24, when she was considered convalescent. The urine at this time was abundant, of a specific gravity of 1014, contained very little albumen, and only a few hyaline tube-casts.

On November 25 the quantity of urine was forty ounces, and the specific gravity 1021. Waxy tube-casts were still to be detected.(a)

In this case, as in the preceding, the union of pregnancy and kidney disease must be looked upon as accidental. In the former the albuminuria supervened during the course of the pregnancy; in the latter, the pregnancy in the course of the kidney disease,(b) each being quite independent of the other. The distinction to be drawn between these cases and

(a) In a letter I received on October 10, 1865, regarding another patient, Dr. Magrath incidentally alludes to the above case, and says Mrs. —— "keeps very well; the condition of the kidneys seems stationary."

(b) Although there is no data by which the exact period when the kidney disease commenced in this case can be determined, yet when we consider the history as a whole, it appears not improbable that the renal affection was first induced in the predisposed constitution by the repeated attacks of intermittent fever, from which the lady suffered at the commencement of her married life.

that first cited—namely, the albuminuria of pregnancy—is of the utmost importance both as regards prognosis and treatment; for while in the albuminuria of pregnancy it is to the pregnancy, and not to the kidneys, that we must look for relief, in the case of the kidney disease associated with pregnancy, as well as in that of the pregnancy associated with kidney disease, it is to the condition of the kidneys that we must specially direct our attention. The reason of this is easily understood when we remember that in the albuminuria of pregnancy the albumen rapidly disappears from the urine after delivery, whereas in the others the mere circumstance of delivery influences the albuminuria only in a secondary degree. The patient must be as carefully treated for diseased kidneys after the expulsion of the child as during its sojourn in the uterus.

As regards puerperal convulsions in cases of albuminuria, as far as my own experience goes—and I have nothing else to guide me—they are far more frequently the result of diseased kidneys than of the simple albuminuria of pregnancy. Convulsions, as we well know, frequently occur without pregnancy, and we cannot be surprised at their occurrence when pregnancy is superadded to the albuminuria. In the latter case the convulsive attack may be delayed until the time of delivery, or it may occur at any period of the pregnancy. There can be no doubt that albuminuria, from whatever cause, predisposes to epileptic convulsions, just as any other disease which impoverishes the blood, and thereby leads to malnutrition of the nervous system; for malnutrition of the nervous system is of all things the most fruitful source of epilepsy. In the second case cited we had ample proof of the impoverishing influence exerted upon the blood by albuminuria; and the absence of convulsions in it might probably be attributed to the circumstance that the blood had almost entirely regained its normal condition at the time of delivery.

That epileptic convulsions may occur in uncomplicated albuminuria in the predisposed I see no reason to doubt; and a well-marked case of this kind, which is briefly as follows, fell under my notice in 1851:—

A dark-complexioned woman, of moderate development, who had been for nine years a strumpet,—she was then 28—was admitted on April 28 into one of the wards under the charge of Dr. Halliday Douglas. She had a dull, languid, and waxy appearance. Her urine was albuminous, of a specific gravity of 1008, and for several days after admission averaged 100 ounces per diem. She said her illness began with dyspeptic symptoms eight months before, and that shortly afterwards she had a fit, which had since recurred about once in every three or four weeks. On May 11—that is to say, thirteen days after her admission—she had a fit which lasted an hour, and from then until she died—seven days later—she had one, two, or more every day. She died immediately after coming out of one. From the 11th to the 18th she was in a state of stupor, and was not easily roused; but when awake she answered questions correctly. The urine fell from 100 to 50 ounces a-day during the last fortnight of her life. On post-mortem examination, the kidneys were found enlarged; one weighed six, the other five ounces; they were in a state of waxy degeneration. The woman had had syphilis. The capsule came off with difficulty; the texture of the organ was exceedingly firm, and of a pale colour. The separation between cortical and medullary substance was ill-defined. In the pyramids were several opaque points, which yielded a puriform fluid on pressure. When examined with the microscope, similar opaque points in the cortical substance were found to contain renal epithelium in all stages of degeneration, and numerous oleo-albuminoid granules.

Supposing this woman had chanced to become pregnant a month before she took ill, she would have been delivered just

about the time she died, and her death would in that case have been most probably attributed to puerperal convulsions from albuminuria, whereas, as we here see, the pregnancy might in reality have had nothing at all to do with it. I do not wish it for a moment to be supposed that I ignore the influence of pregnancy, and more especially of the effects of delivery, in inducing convulsions in those otherwise predisposed to them.

All I desire is, to utter a strong protest against the oft-repeated statement that puerperal convulsions are always the result of the albuminuria of pregnancy, while in reality they are much more frequently the concomitants of true kidney disease, assisted by the effects of the puerperal condition, the chief cause of the convulsions being the retention in the circulation of the excrementitious urinary products.

It is of vast importance to both mother and child to be able to diagnose these cases; for while in the albuminuria of pregnancy, unless the symptoms be severe, the case may almost be left to nature, in pregnancy, associated with true kidney disease, energetic treatment is demanded, even, as we have seen, to the induction of premature labour; for even when the pregnancy does not end in convulsions it never fails to act prejudicially on the renal affection.

I would even go further, and say that pregnancy, in many cases, is one of the exciting causes, not alone of albuminuria, but of true kidney disease; just in the same way as cardiac and hepatic affections are—by keeping up renal congestion.

Albuminuria the Result of Organic Disease of the Heart, Liver, or Pancreas.

This is an exceedingly common class, and one demanding our most careful consideration, as judicious treatment can here do a great deal for the patient. The pathology of this class of cases is very simple, mechanical obstruction inducing passive renal congestion. As the albuminuria usually first makes its appearance at a late period of the cardiac, hepatic, or pancreatic disease, it often happens that the patient succumbs to the united effects of the double complication ere any well-marked anatomical alteration in the structure of the kidneys has had any time to occur. When such is the case, on post-mortem examination the kidneys are merely found highly congested. Should death, however, not have proved immediate, there is usually some structural change met with in the renal organs, for, as we have already seen under a different heading, the continuance of renal hyperæmia gradually, but surely, leads to chronic hypertrophy, which in its turn ultimately induces a true structural change in the renal tissue. In the class of cases now under consideration, the anatomical alterations met with in the kidneys, as well as the functional derangements to which these changes give rise, must be regarded as the secondary results of the diseased liver, heart, or pancreas, as the case may be. But there is also another set of cases where, although the double complication exists, the pathology is different. I allude to those where the union of the renal and the other affection is, as it were, accidental. That is to say, each disease has originated independently of the other, certain causes having led to the renal affection on the one hand, certain other causes having induced the cardiac, hepatic, or pancreatic affection on the

other. The double complication in the latter class of cases must be looked upon as a much more serious matter than in the former.

For the sake of illustration, let us imagine the case of a patient, who, after having for years been the subject of chronic kidney disease, becomes suddenly attacked with endocarditis, ultimately leading to permanent valvular obstruction, the tendency of this being, as we know, to lead to hypertrophy of the heart. Here, then, is a case of a most grave character; for in addition to the cardiac hypertrophy we have the increased tension in the arterial system produced by the already existing kidney disease, also tending to the same result. The evil does not even stop here; for just as on the one side the kidney disease tends to increase the cardiac hypertrophy, so on the other the valvular obstruction lends its helping hand to increase and perpetuate the kidney disease.

What holds good for cardiac affections holds equally good for obstructions to the portal circulation induced by hepatic or pancreatic disease. All these morbid conditions act and react upon each other in such a manner as to render it exceedingly difficult to define how much of any given effect is due to the one, how much to the other. Thus it often happens that a patient who has been labouring under the combined effects of cardiac and kidney disease suddenly becomes seriously worse. The swelling of the feet increases, the pulse becomes rapid and irregular, the breathing short and quick, and within twenty-four hours after being in his usual health he seems at the very gates of death. On examining such a patient carefully, we most probably find a foul tongue, a sallow complexion, an enlarged and engorged liver. Added to this a bastard condition of lung—half pneumonic, half bronchitic; râles copious; sputa sanguineolent; dulness irregular, the most distressing symptoms at the same time being the inability to take a full breath and the impossibility

to procure sleep. No sooner does the poor patient lie down and shut his eyes than he starts up in a paroxysm of suffocation. Severe as these cases may be, they are by no means always without hope. I have seen patients as bad, and worse than here described, get comparatively well—at least, so well that they have lived for several months and been able to travel many miles. Perhaps this statement will be better appreciated if I relate a case of the kind; and the one I purpose selecting is that of a well-known publisher whom I attended with two other Medical men. When first seen, in conjunction with Dr. Walshe and Mr. Courtenay, the patient's condition was briefly as follows:—Great difficulty of breathing; respirations 38; feeble and irregular heart's action; pulse 100; headache, sickness, and vomiting; sputa pneumonic, red, rusty, and tenacious; dulness over the lower pulmonary lobes on both sides of the chest; loud mitral murmur; œdema of the lower limbs; scanty and highly albuminous urine, of a specific gravity of 1010. Coupled with these, there was an engorged liver and a sallow skin. In fact, a more hopeless-looking case could scarcely be imagined; for, although the patient could not lie down to sleep, he seemed gradually drifting into a state of uræmic coma. His breath and perspiration had already a urinous odour, and that, coupled with the uræmic pneumonia, made one regard the case with anything but agreeable forebodings. Strange to say, however, under very active treatment directed to the skin, lungs, liver, and heart alternately, this patient ultimately recovered, and was able to leave his bedroom within five weeks from the time that he was in the condition above described, and within a very few weeks more he was able to take a fifty miles' railway journey with perfect impunity. During the period of active treatment the case several times assumed so severe a character that both Dr. Walshe and Dr. Williams, who also saw the patient during his

illness, agreed with Mr. Courtenay and myself in thinking that hours, not even days, would have limited his existence.

The influence of cardiac disease on renal affections is not yet all told, for it may lead to other complications than those already described—such, for example, as embolism of the kidney, of which the following case, kindly furnished to me by Dr. Bäumler, is a good illustration. It has the additional advantage, too, of being one in which the pathological condition was correctly diagnosed during life:—

E. K., aged 26, admitted into the German Hospital on October 3, 1864, with well-marked mitral disease; no dropsy; rather difficult breathing; crepitation at the base of the right lung.

October 4.—Urine scanty (about 700 c.c.), with deposit of urates; contained only a trace of albumen, a good deal of pigment, and a considerable amount of urea, so that on putting strong nitric acid to it, the urine very soon crystallised.

5th, 6 p.m.—Sudden attack of severe pain in the right side below the twelfth rib, and in the right loin. Complete collapse; extremities cold; slight cyanosis; no pulse could be felt; sickness and vomiting, diarrhœa. Rallied after the administration of stimulants.

6th.—Pain and sickness continued.

7th.—Diarrhœa stopped. A little blood in the sputa.

From the 7th to the 12th of October the prominent symptoms were in the chest, the pain in the right side having then nearly subsided.

12th.—Feels better; urine in quantity and appearance the same as on the 4th. *A little more albumen.* In the evening she was suddenly seized with vomiting, repeated purging, severe pain in the left hypochondrium, and collapse.

13th.—Pain continued very severe. (Great relief from subcutaneous injection of $\frac{1}{4}$ gr. of morphia.) The pain was greatest just below the twelfth rib, and radiated to the left loin.

15th.—No more diarrhœa. Urine 500 c.c.

16th.—Urine 1000 c.c., contained *more* albumen than on the 12th. The field of the microscope was covered with tube-casts, transparent and granular, and blood corpuscles.

22nd.—The same amount of albumen; felt better; œdema of the legs.

23rd.—Chest symptoms increased; considerable effusion into the right cavity of the pleura.

25th.—Less urine, less albumen.

27th.—Beginning jaundice.

31st.—No albumen nor casts.

November 2.—Urine about 1000 c.c., without deposit; large amount of bile pigment; *no* albumen.

11th.—*Not a trace of albumen* in the urine. Died on the same day.

The post-mortem examination showed a small fibrinous deposit in the right kidney in a state of shrinking; the upper part of the left kidney entirely diseased; one of the branches of the renal artery plugged up; the plug was about one inch long.

Albuminuria the Result of General Affections.

This includes a large group of diseases, which with advantage may be subdivided into the two varieties of febrile and non-febrile affections. Among the diseases composing these groups may be mentioned—

1st. Small-pox, typhus, typhoid, scarlet, yellow, rheumatic, puerperal, and remittent fevers.

2nd. Pneumonia, pleurisy, bronchitis, phthisis, peritonitis, gout, scurvy, erysipelas, purpura, and cholera.

The urine in all these cases may be coagulable under three different conditions. In the first place, the kidneys may only

permit the serum of the blood to escape; in the second place, not alone may the unorganised serum escape, but even the organised blood corpuscles, thereby giving to the case somewhat the appearance of acute Bright's disease, the urine being both coagulable and smoky, or even red. Thirdly, and lastly, in the severer forms of disease, especially in those assuming a malignant type, the walls of the blood corpuscles are broken down, and their disorganised contents—hæmato-globulin—escape, giving to the urine the colour and chemical reactions, without, however, the microscopical characters, of hæmaturia. Such a state of things is occasionally seen in putrid typhus, malignant scarlet fever, and severe purpura. I shall afterwards have to relate cases, under the head of intermittent hæmaturia, which closely resemble these, in so far as the urine contains disorganised blood constituents without blood corpuscles.

As regards the pathology of the albuminuria of general affections, all that need be said is that it is essentially due to renal congestion, and that there is usually but little difficulty in diagnosing the case, from the circumstance of its coming and going with the other affection. It is true that active congestion may also supervene in the course of one or other of the forms of primary albuminuria, but even then we have it in our power to recognise its existence by a peculiar form of renal tube-cast—the granular tube-cast. Granular casts were at one time thought to be characteristic of contracted kidney, but this I have long since proved to be an error. They may occur in cases of contracted kidney, as they may occur in any form of albuminuria; but the cause of their presence is then, as it is now, active congestion.

Some idea of the appearances presented by granular tube-casts can be obtained from the accompanying illustration. The casts may be large or small, quite full, or only partially filled with granular matter, the size of the cast depends

on the part of the tube in which it was formed; the amount of granular matter it contains on the condition of the kidney.

FIG. 6.

Quantities of escaped granular matter and epithelial cells may also be irregularly scattered over the field of the microscope.

Before passing to the subject of treatment, I would again call special attention to the fact that in every form of chronic kidney disease, the organ is liable—more liable than the healthy kidney—to occasional attacks of acute congestion or of inflammation, and that under such circumstances the appearances met with in the urine become modified and complicated accordingly. Fatty casts are found mingled with blood corpuscles, waxy casts mixed up with pus cells, granular casts surrounded with oil globules, and so on, thereby throwing almost insurmountable difficulties in the path of the beginner. Care and experience will, however, soon clear the way and show that the apparent confusion is but perfect order not properly understood.

Treatment.

At no very distant date the treatment of kidney diseases was regarded as an almost hopeless task. As our knowledge of their pathology and our powers of diagnosis have, however, advanced, we have gradually gained confidence in the value of remedies in such affections, and we might almost say that it is in the power of the enlightened Practitioner not only to check the milder forms, but even to control and mitigate the symptoms arising from the severer renal affections. To be successful in treatment we must be correct in diagnosis, and taking for granted that the nature of the case has been thoroughly made out, the general principles upon which we are to found our treatment are as follows:—The first and great principle of all is to give rest to the diseased organ, for just as the function of the diseased eye is to be kept in abeyance while nature is removing the lesion, so must the diseased kidneys have rest from their function in order that their healthy condition may be restored. We cannot, unfortunately, lay up the kidneys as we can a fractured limb, nor entirely arrest their function, as we can that of a diseased eye. Nevertheless, we can reduce their labour to a minimum by making other organs for a time perform an essential part of their office.

There are three channels by which we can draw off from the system the excrementitious materials which normally fall to the lot of the kidneys to eliminate—namely, the bowels, skin, and lungs. By keeping up an excessive action in these, we can not only remove for a time a portion of the burden from the over-taxed kidneys, but in those cases where dropsy has already become a distressing symptom give great relief to the patient.

The value, indeed, of pressing into our service the vicarious

action of the bowels, skin, and lungs in the treatment of renal disease it is scarcely possible to overrate, for we can not only by such means give rest to the kidneys and diminish dropsy, but even mitigate the more serious symptoms which are ordinarily included under the term uræmic intoxication,(a) but which, more properly speaking, are the direct result of the combined effects of the retention in the circulation of all the excrementitious products, organic as well as inorganic, which normally fall to the lot of the kidneys to excrete. The skin and lungs are more powerful auxiliaries in the elimination of urinary products than is generally supposed, for, as shown in our first lecture, the cutaneous perspiration does not only carry off water, but many of the organic as well as inorganic urinary salts. Thus it has been found that the sweat even in health contains urea, uric acid, phosphates, and chlorides, while in disease, in addition to these, it contains many abnormal compounds, such even as the insoluble oxalate of lime. Pulmonary exhalation, too, as was then shown, may be almost of equal service, for in the expired air of even healthy men have been detected urea, uric acid, urate of soda, and urate of ammonia.

The vicarious action of the bowels is to be induced by the internal administration of mild or drastic purgatives, according to the constitution and condition of the patient. When there is much dropsy, elaterium is a favourite form of purgative, but in cases of kidney disease it is usually advisable to administer it along with hyoscyamus, as it not unfrequently brings on an exhausting diarrhœa, especially if given after the symptoms of uræmic poisoning have already set in.

The vicarious action of the skin may be induced either by the internal administration of diaphoretics or the external

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(a) The symptoms of uræmia have already been given under the head of urea.

use of the warm bath, vapour-bath, or hot air-bath. The two latter not only increase the cutaneous, but also augment the pulmonary elimination of urinary products. This is especially the case with the hot air-bath.

I may here remark that the usual practice of trying to diminish rather than to increase the urinous odour of the sweat and breath in cases of advanced kidney disease is greatly to be reprehended; for instead of trying to check, we ought, on the contrary, to assist Nature in her laudable efforts to rid the circulation of the deleterious agents that are gradually extinguishing the life of the patient.

In the next place, the employment of antiphlogistics is in many of the inflammatory forms of kidney disease of essential service. The most powerful of these is, of course, the local abstraction of blood, either by leeches or the cupping-glasses. But just as in many cases of inflamed lung the general condition of the patient prevents the employment of such active means, so also in the case of the kidney we must occasionally abandon this line of treatment, and content ourselves by merely diverting for a time the course of the circulation, either by the application of dry cupping-glasses, counter-irritation, or of hot fomentations to the loins.

I ought not to omit to mention that the unloading of the portal circulation by a smart calomel purge will often prove an important adjunct to other antiphlogistic measures.

As regards the employment of diuretics in the treatment of kidney disease, a few words are here necessary. In the first place, it ought never to be forgotten that in acute Bright's disease, as well as in the first stage of all inflammatory and congestive attacks occurring in the course of chronic kidney affections, diuretics are inadmissible. In the second place, it must be borne in mind that great care should always be observed in their selection; for a diuretic which will prove beneficial in one form and at one particular stage of renal

disease will often not only do no good, but actual harm, when administered in another form or at another stage of the same attack. Thus, whenever the albuminuria is the result of active congestion, the antiphlogistic variety of diuretic—such, for example, as a combination of bitartrate of potash and digitalis—is to be selected; whereas in the absence of active congestion, and more especially when the vital powers of the patient are low, the stimulating variety of diuretic may not only be used with impunity, but with actual advantage. The reason why the employment of diuretics often does harm in acute kidney affections, is readily understood when we recollect that they have always the tendency rather to increase than diminish the flow of blood to the already engorged organ.

A word or two may also be added regarding the action of diaphoretics, which, as is well known, are exceedingly usefnl in chronic kidney disease. The most common form is Dover's powder, and from this containing opium, some have even ventured to administer that drug in combination with other substances than ipecacuanha. I must here, however, call attention to the fact that, although Dover's powder may be given with impunity, opium can seldom be employed in kidney affections in any other form without a certain amount of risk. More than one example of its deleterious effects in such cases has come under my notice, and not long ago(a) a fatal case actually occurred in one of our London Hospitals, where a man, aged 45, labouring under kidney disease, died after having taken only one grain of the acetate of morphia. The object of giving diaphoretics in chronic kidney disease is, of course, to keep up cutaneous perspiration. For a similar reason, patients ought always to wear warm clothing; and this rule ought never to be departed from, even in cases where

(a) *Lancet*, June 8, 1861, p. 575.

the patient is comparatively well, so well that complete recovery may be said to have taken place. In the latter class of cases, indeed, every precaution against the effects of cold ought to be had recourse to, for a patient who has once had an attack of kidney disease is ever afterwards much more liable to another attack than one who has never been so affected.

In chronic albuminuria, astringent salts, as well as acid tonics, are often of great service, especially in getting rid of the last traces of albumen after an attack of acute disease. Just as gonorrhœa often ends in a gleet, so it happens that the kidneys, after having for a long time been the seat of Bright's disease, continue to pour out small quantities of albumen long after all constitutional symptoms have disappeared. In such cases, sulphate of zinc, sesquichloride of iron, gallic acid, tannic acid, and the mineral acids have frequently a very salutary effect in checking the remnants of the albuminuria.

This action of the acids, at least, seems already to have found a physiological explanation, for Heynsius, while studying the diffusibility of albumen,(a) discovered that the exosmose of an albuminous to a saline solution is retarded by acidity, and accelerated by alkalinity; and, if such be the case out of the body, there can be little doubt, I think, that a similar, though, perhaps, a modified, effect follows the exudation of albumen from alkaline blood into acid urine.

In a recent communication, Dr. Hassall (b) has thrown some doubts upon the value of sesquichloride of iron in the treatment of renal disease, to which he had been led by finding that he obtained no ferruginous reaction from urine of patients to whom he had administered that remedy. This being entirely opposed to my own experience, I was led to inquire into the cause of the difference in our results, and I soon found, in

(a) Donders' "Archiv.," vol. iv., part 3.
(b) *Lancet*, vol. ii., p. 740, 1864.

perusing his paper, that the error had arisen from his testing the urine directly, instead of first evaporating and incinerating the residue before applying the tests for iron; iron, like most other metals, being quite undetectable in organic fluids, unless present in immense excess. Even normal urine contains iron.(f)

As diet invariably plays an important part in all treatment, I must here call attention to the influence of foods on albuminuria, as previously pointed out in the physiological part of our subject. From what was then said, it will be readily understood why patients labouring under kidney disease should receive the lightest and most digestible kinds of diet. Moreover, their meals should be frequent and small rather than seldom and abundant.

The valuable researches of Professor Parkes(g) on the influence of food on the quantity of albumen eliminated by the kidneys, form the grounds for this latter recommendation. Dr. Parkes found that more albumen is eliminated after than before a meal, and that fasting not only invariably diminishes the quantity of albumen excreted, but even in some cases may cause it entirely to disappear from the urine—a fact which should make us avoid giving the patient more food than the wants of the system actually demand. The kind of food ought also to be regulated, in some measure, according to the form of kidney affection. Thus, while in cases of fatty renal degeneration, oleaginous diets are to be avoided, in the amyloid variety of the disease these same foods may be given, if not with advantage, at least with impunity. The general constitution and condition of the patient must, however, always be taken into consideration, and the diet selected according to the special requirements of the case.

Before leaving the question of treatment, as a sequel to the general principles just enunciated, I may add a few words

(f) The Author, *Pharmaceutical Journal*, Nov., 1852.
(g) *Med. Times and Gaz.*, 1854, p. 395.

regarding the especial treatment of certain of the more common forms of albuminuria.

In acute Bright's disease cupping, dry cupping, leeching, or fomenting the loins ought immediately to be had recourse to, according to the urgency of the symptoms and the constitution of the patient. In the beginning, diuretics must be scrupulously avoided, and even in the second stage only those of the antiphlogistic variety are admissible. The bowels are to be made to act freely; the action of the skin and lungs increased. The quickest, and in many cases the safest, way of doing this is by using the hot air bath; and it is astonishing how rapidly beneficial its employment may sometimes be. On one occasion I was very forcibly struck with this, in the case of a little child, who speedily recovered, after being almost moribund from the united effects of dropsical effusion and uræmic intoxication. The case was one that I saw along with Mr. Brown, of Finsbury-circus, and, if I remember rightly, the age of the child was not more than fifteen or eighteen months.

In fatty kidney, in addition to the means already indicated, great attention must be paid to the improvement of the general health, for in the majority of these cases the affection is more frequently due to constitutional peculiarity than to an accidental cause. Fresh air and regular exercise within the margin of fatigue, together with light, non-oleaginous foods, are to be prescribed. Change of air and hot salt-water baths ought, in the early stages of the affection, alike to be had recourse to. When tonics are given they should be of the astringent and acid kind, except when counter-indicated by any constitutional peculiarity.

In the amyloid variety of renal degeneration, which, as already shown, is frequently the result of a syphilitic taint, the judicious employment of the bichloride of mercury is often of much use; but care must be taken to stop as soon as

the mercurial fœtor of the breath becomes distinct, for such patients do not readily tolerate the reducing action of mercury. It will be often necessary to combine with this treatment the administration of port wine and light nutritious foods.

Atrophied kidney must be treated upon the general principles already enunciated, and, of course, it is one of the most suitable of all renal affections for increasing the action of the skin and lungs.

In it, as well as in all cases of confirmed albuminuria, we may occasionally be able to prolong the life of our patient from the free action of the skin being periodically induced by the Turkish bath, and when that cannot be obtained the hot vapour bath may often be substituted with advantage, care being always taken to employ the bath periodically and at short intervals; for then alone can we expect advantangeously to make the skin vicariously perform the function of the renal organs.

In the latter stages of atrophied kidney, as well as in the fatty and waxy forms of kidney disease, the symptoms of uræmic poisoning may sometimes be diminished by administering small doses of tartar emetic. Indeed, Dr. Lang, of Königsberg, recommends the use of tartarised antimony in the uræmia that follows acute exanthematous diseases.(h)

Regarding puerperal albuminuria, sufficient has already been said to serve as a guide to its treatment. All that need here be repeated is, that premature labour ought only to be had recourse to in those forms where the pregnancy is associated with true kidney disease, or where there is reason to anticipate the occurrence of convulsions either at or before the time of delivery. Premature labour may also successfully be had recourse to in the earlier months of pregnancy, when

(h) *Brit. Med. Jour.*, Jan. 21, 1859.

the health of the mother is rendered precarious by the constitutional disturbance arising from the albuminuria.

Thus, Dr. Lee related a case to the Medical-Chirurgical Society in 1863, in which he had induced the expulsion of a fourth month's fœtus in a pregnancy complicated with albuminous urine, dropsy, and amaurosis, after which the albumen gradually disappeared, and the vision of the patient improved.

When in the course of puerperal albuminuria convulsions have once manifested themselves, the readiest and safest means of subduing them appears to be by the free administration of an anæsthetic. Chloroform has been most commonly employed for this purpose, but as insensibility has usually to be kept up for a considerable time, it is much safer to employ the mixture which I proposed some years ago, and which received the approval of the Chloroform Committee of the Royal Medical-Chirurgical Society. The mixture consists of one part by measure of pure alcohol, two parts of chloroform, and three parts of sulphuric ether. The advantages of this mixture need not be here alluded to, as they were fully dwelt upon in the report of the above-mentioned committee.

As regards albuminuria associated with organic disease of the heart, liver, or pancreas, as well as in that variety the concomitant of general affections, the principles already indicated must serve as the landmarks to the treatment of the renal disturbance; whereas the other disease or diseases forming the complication are to be treated according to their special requirements. Always remembering that the removal of the cause, rather than the mitigation of its effects, ought to be the first consideration.

A great deal more might be written regarding albuminuria, but I think enough has been said for my present purpose, which has been merely to point out the general principles which ought to guide our diagnosis and treatment. Every

one must feel convinced that the employment of the microscope is as indispensable to treatment as to diagnosis. Thus Aitken says :—

"Without the microscopic examination of the urine from day to day it is impossible to distinguish between a case likely to improve under treatment, and one which may be viewed as hopeless, and without the daily use of the microscope the treatment becomes at the best but merely guess work." (i)

It must be borne in mind, too, that as one swallow does not make a summer, neither does one tube-cast in kidney disease at any time suffice to establish an exact diagnosis. This can be easily understood when it is recollected that the deposits in the urine are but the representatives and results of the morbid change occurring in the renal tissue at the moment of their detachment; and as disease is never stationary, it is only by comparing the character of the renal *débris* of one period with that of another that we are enabled to establish the exact character of the affection, and discover whether it is marching on to a fatal termination or gradually retrograding to a point within the boundaries of healthy action.

(i) Aitken's "Science and Practice of Medicine." 2nd ed., vol. ii., p. 324.

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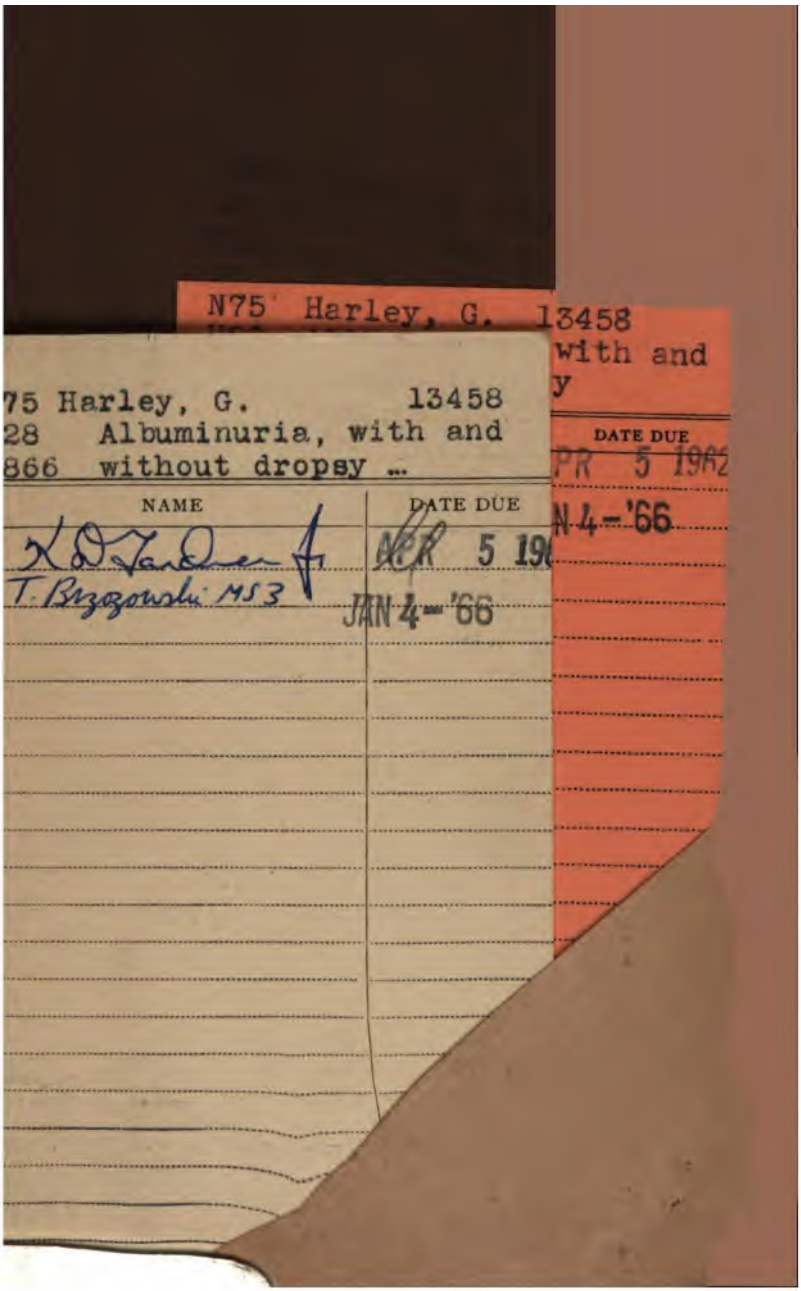
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