

## The Early Diagnosis of Disease of the Kidneys especially Bright's Disease,

Bright's disease has so many victims, and when the disease is once fastened upon the patient in the chronic form it is so generally fatal, <sup>that</sup> early diagnosis becomes a matter of the utmost importance. When asked to present this subject I first thought of limiting what I had to say to the uses of the microscope in its diagnosis. But on making some review of the more recent literature of the subject thought it better to drop this, as the main feature, and open up the whole subject of diagnosis and the early treatment. This seemed best, from the fact that within the last decade there seems to have been considerable change of opinion and thought upon the subject. I studied this subject pretty closely fifteen years ago, and thought I had some fitted notions in regard to it. For a number of years I followed it and made many examinations of urine for the Physicians of the surrounding country. When I broke down in health this was dropped and for some years I had

given the subject no special attention, now I find it a new field of investigation, presenting, not new problems, but new phases of the old problems. Some of these have placed matters in doubt that before seemed settled while some things that not thought much about have assumed grave importance.

Albuminous urine was once considered as incontestable proof of actual disease of the kidneys and with most of us it is perhaps still so looked upon. Furthermore albumen <sup>in the urine</sup> has been considered to be the first tangible evidence of serious disease of the kidneys. To day neither of these propositions will hold. It is true that when albumen is present in large amount and continuously for some weeks it furnishes evidence of kidney trouble of some kind. But it tells us very little, of itself, of the character of the disease, we may have the urine heavily loaded with albumen for several weeks continuously without very serious kidney lesions, and the patient may recover perfect health. Or the patient may die of chronic nephritis and albuminous urine not be present at any time.

With albumen this must be coincident - symptoms of grave disorder of the kidneys in order to justify a diagnosis of serious nephritis such as loss of vigor without other manifest cause. Muscular debility, headaches, swelling of the face and feet, or drowsical effusions, and a peculiar waxy palor - The nephritic cachexia.

In this case the condition may be very grave and point to early fatal results or it may be transient and quickly recovered from. The prognosis is now to be made on two points in addition. Has the disease been chronic. Has the muscular debility been of considerable duration approaching the present condition slowly or has it been rather sudden in its onset. In the first case we are dealing with a chronic nephritis that may now be in acute exacerbation. In the second we may have simply an acute affection, just here the study of the casts that may be present will often help us out. If the muscular strength has failed suddenly and we find only epithelioma casts with little or no fatty degeneration our patient may recover in a few weeks. But if the muscular debility has been of long duration and the

Casts are granular and fatty showing space epithelium in a state of fatty degeneration we must make an unfavorable prognosis. And this no matter how well the action of the heart or absence of dropsy, or fair strength of the patient. Extensive fatty degeneration of the renal apparatus is more conclusively shown by the study of the urinary sediment microscope, than in any other way short of an autopsy. ~~Blood~~, <sup>great degeneration of epithelium,</sup> hyaline casts, and many other things may be present and have better to tell of the actual state of the kidneys, ~~they speak of~~. Blood usually - if the amount is not too great, speaks of an acute inflammatory movement just then in progress, Pus, <sup>generally</sup> indicates some other disease than nephritis. Of course abscess of the kidney may occur.

When these things are found, one of two things follows. A case of acute nephritis from which the patient may recover, Or that we have a patient upon whom a chronic nephritis has become fixed long before and who is now <sup>perhaps</sup> beyond help.

Could we have discovered this latter condition in time to have rendered the patient

Different grades of this have been  
divided into a number of forms by  
different authors.

Substantial aid. This is the important question  
now for the medical profession in relation to this disease.  
It is easy, comparatively, to recognize the disease in its  
latter stages. How shall we recognize it in the very  
early stage?

Before attempting to answer this let me say that  
for the practical purposes of the physician I think  
it best to recognize three forms of the disease, which  
may, however, be blended in all kinds of ways

1st Acute Nephritis - Large soft Kidney  
2nd Waxy Nephritis Large waxy kidney ordinary  
~~and Contracted~~  
3rd Chronic Nephritis. Contracted kidney

The first - most often follows the exanthem  
or is a result of moist cold, or exposure,  
and usually yields to treatment if it is prompt,  
The second is ~~not especially~~ <sup>of necessity primarily</sup> a disease of the  
kidneys though it is in these organs that it  
does most mischief. Generally various other organs  
are similarly affected

The third is chronic nephritis from the first  
and is the disease that claims the large share of the  
victims

## Etiology

The cause of this disease is involved in the utmost obscurity. We have only surmises, we know that there is a large number of medicines that, when taken into the blood will cause inflammation of the kidneys. I may mention the mineral acids, Oxalic acid, phosphorus, arsenic, lead & chromate of potassium, also Squill Balsam copaiba, turpentine, Salicylic acid, Chlorate of potassium, Cubeb's nitrate of Potassium ~~and~~ carbolic acid ~~and~~ Iodoform and Mustard, Causticades, tar petroleum, styrol, naphthalene, pyrogallic acid are liable to produce inflammation of the kidney when applied to the skin. All of these are excreted by the kidneys after their passage produce inflammation.

This shows very plainly that these organs are exposed to injury whenever they excrete from the organism substances which are abnormal and of an irritative character. The supposition becomes tenable, and plausible, that chronic inflammation of the kidney results from some change in the functions of the tissues, or glandular service of the body, which delivers abnormal products to the kidneys for excretion. This supposition places the causes of the kidney disease, in a disease which precedes it - of which little or nothing is known. For a long time it has been known that persons are in a state of health failure long before any disease of the kidney can be determined from physical signs. Delafield says

that such persons do not

excrete the normal amount of urea, and also says further that the normal amount of urea is not formed, and is not present in the blood to be excreted. Flint, Stumpf, and almost all who have written within a decade state that the normal amount of urea is not excreted by these persons. They may show neither albumen nor casts. Health is failing. Headaches are common. Muscular debility is gradually approaching, the appetite becomes capricious, Vascular Tension is continuously high. Occasional nose bleed. Mental work has become particularly tiresome &c yet - the patient is not sick. He looks a little anemic and doesn't feel very strong.

Now is the time to make diagnosis and begin treatment. With this set of symptoms we must exclude Anemia (by the continuous high arterial tension), Neurasthenia by the general condition of the nervous system and In this the arterial tension is high in many cases, <sup>large tumors</sup> vascular system, various other affections by the failure of their prominent symptoms.

When we have done this, we must before making our diagnosis determine by <sup>quantitative</sup> examinations for urea determine that it is continuously, or at least generally, below the normal amount.

The urine urea is usually less than the normal amount of urea but low arterial tension

taking into consideration the amount  
and kind of food ingested

during the twenty four hours. With this point settled  
and the other symptoms present we have chronic Bright's  
disease of the most dangerous type, though there may  
be neither albumen, casts <sup>worm-like</sup> or dropsy. There will,  
may be, come later, perhaps all of them, perhaps only  
part of them.

I will quote some passages from the "Annual  
of the universal Medical Sciences" written by  
Francis Selsfield of New York. He quotes this  
from Dr Thomas, <sup>Rev 47th</sup> I believe that the contracted  
Kidney is always caused by the presence in the blood  
of some material which has to be daily excreted  
in large quantities, and which, in its passage  
through the kidneys, gives rise to an increased  
formation of the fibrous stroma and other  
subsequent changes."

Continuing Dr Selsfield says "We find that  
alcohol tends to produce it, (This is denied by  
Dr Flint, unless the amount is very excessive and  
then changes the injury to the kidneys to exposure)  
and we know that in such patients, from chem-  
ical examinations of the urine, the kidneys  
have to excrete a goodly portion of what has  
been taken in."

"Again Dr. Leafield says, "we often find that patients who have to do a large amount of mental work, or who have a good deal of mental worry, are apt, in time, to suffer from contracted kidney. The writer has noticed such patients for years, and invariably found that they have passed, large quantities of phosphate daily. These patients do the bulk of their mental work in the morning, and the effete material the kidneys have to excrete is passed somewhat later. He has further observed that later on, after excessive work, or very great worry, the phosphate are accompanied by albumen."

"In gout there is a tendency to the formation of urates and uric acid in the blood. The kidneys have for years excessive work to do performing in excreting these substances, and the writer believes that in consequence of this, a granular state of the kidneys is induced."

"In Plumbism we find that the patients pass urine containing lead in solution daily, and after this has continued for a time albumen begins to appear."

"Many patients, especially dyspeptics,

pass large quantities of oxalates daily, such patients ~~pass albumen~~ later on, pass albumen also, and ultimately have Bright's disease".

"The conclusion the writer has come to with regard to the contracted kidney is this: that it is invariably preceded by the presence of large quantities of some material in excess, such as phosphate urates, oxalates, lead, alcohol in the urine; that after a time a small quantity of albumen will be found accompanying it; that the presence of these constituents constantly in the blood, present therefore excretion gives rise to the hyperplasia of fibrous tissue and subsequent changes".

I will also quote the following page 480 of the same work, quoted from Semmola.

"True Bright's disease consists of a chronic morbid state distinguished by: (1) Its etiology, the slow action of damp cold on the skin, (2) By impairment leading to entire abolition of the functions of the skin, depending on progressive ischaemia with atrophy of the sudoriparous glands, progressive atrophy of the layer of Malpighi, and proliferation of the connective tissue of the derma. (3) By a chronic

Molecular alteration of the albuminoïds of the food; distinct alteration of their diffusibility, and hence in assimilability and necessary elimination through all of the excretaries, and chiefly through the kidneys. (4) By progressive diminution of the combustion of the albuminoïd which shows itself by progressive diminution of the formation of urea, so that a progressive diminution results in the quantity of urea eliminated by the urine in the 24 hours, without any accumulation of this principle in the blood or elsewhere. The blood in Bright's disease (in patients not in the uremic condition) contains less urea than that normally found. (5) By a subcutaneous serous infiltration, surely and slowly progressive and having no relation to the hydrocephalus. (6) by a special cachexia not in relation to the losses of albumen but to general impairment to assimilation. (7) By a secondary development of an inflammatory process in both kidneys."

From this quotation from Semmola we see that he regards the nephritis as <sup>the</sup> local

In chronic

Coats 638 "Albuminuria is the most important clinical sign of Bright's disease"

639 "In chronic Bright's disease the blood is at an excessive pressure"

"XXXX The occurrence of albumen seems definitely related to the structural changes in the kidney"

465 "We may suppose that the altered blood has greater difficulty in passing through the capillaries generally than normal, and this would induce increased tension in the arteries"

Soumis

Page 527 "Urea is abnormally increased in amount in all febrile and nervous affections Pyrexia and Diabetes. It is abnormally decreased diminished in Nephritis anaemia cholera and Starvation and may be entirely absent in acute yellow atrophy of the liver?"

expression of a constitutional disease which is certainly very obscure in its character, ~~so~~ His expression of this is in wide disagreement with a number of other writers, but on one point, available in early diagnosis he is in agreement with most others who have written lately, and that is, the constancy of the diminution of urea for sometime ~~of~~ before the appearance of the evidences of kidney lesions. He claims that the normal amount of urea is not found.

Thomas claims the same, also that certain salts are present in abnormally large quantities

Flint - claims that the amount of urea excreted is abnormally small

Strumple claims the same

This is also claimed by Geiger, by Combe and Remond.

On this point Loomis has the following passage page 529 Urea is abnormally increased in amount in all febrile and nervous affections, Pyrexia and diabetes. It is abnormally diminished in nephritis, anæmia, cholera, and starvation, and may be entirely absent in acute yellow atrophy of the liver

Dr C S Bond of Richmond Ind. in an article in  
the American Journal for January 1890 presents  
a carefully written article bearing upon this point  
and detailing in tabular form his observations on  
fifty cases of Bright's disease during a period of  
five years. In all of these cases he made frequent examinations  
for urea which he gives, with other points of the symptom-  
atology, course of the disease and treatment. I will  
make some quotations from his paper

Read from Page 29

Years ago I found, during my examination of urine brought me from various quarters, with the clinical histories of patients, that albuminous urine, even when the albumen was in considerable amount, was not a sufficient proof of disease of the kidneys. This was then contrary to the books, as we say, and to a large extent is so to day. Albumen even when accompanied with a sprinkling of hyaline casts is not yet sufficient for the diagnosis of grave disease of the kidneys. Indeed this character of casts show only that albumen is passing. The casts must bring away something that shows disease. Renal Epithelium in large amount shows a desquamation process, with the nuclei enlarged and the cells crowded they are a token of inflammation. More crowded and granular - a severe type. If they are also filled with fat granules we have a process of destruction of the renal structures. Blood in connection with blood casts tells us of an active inflammatory process. This generally indicates other lesions somewhere between the kidneys and the urethra - occasionally from abscess of the kidney &c.

When possible this evidence should be had

for its value in determining the character or quality of the renal affection and for prognosis as well as during treatment, as well as for diagnostic purposes in the first instance.

The great difficulty with this examination lies in the fact that so few give the subject the time and study to become expert in the nice distinctions required in the ~~study~~ determination of the condition of the epithelium. The determination of blood and pus are easy even to a novice and there are often of great value.

Now I want to make some quotations on the subject of Albuminoid urine - There will be both negative and affirmative.

Cornil & Rauvier Page 641 state that "General or partial interstitial nephritis is frequently seen without albuminuria, which is evidence that an inflammatory lesion of the connective tissue of the kidney has no direct connection with the presence of albumen in the urine."

They describe some forms of nephritis in which there is generally no albumen present, or only toward a fatal termination when the blood

pressure has become impeded

Sa Costa ~~page~~ "Medical Diagnosis" page 706 describes what he calls "Simple Abunuenia" which he says is "unconnected with any ~~serious kidney~~ marked structural lesions, unless it be congestion." He describes it as occurring in many pathological conditions. The amount, he says, is comparatively small, and the constitutional conditions are such as to connect so similar to those in Bright's disease that the serve to correct errors of diagnosis. He follows this description with this significant sentence "Yet the most valuable aid in forming a judgement is derived from the Microscopic investigation of the urinary sediment" &c

Biererott - Diagnostic - describes a physiolog-  
ical abunuenia. This he says is not of very  
frequent occurrence, but may occur when there  
is low arterial tension or any interference with  
the circulation in the kidneys and in simple passive  
congestion.