Basic concepts underlying the classification of nephritis

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SNIOR THESIS

(BASIC CONCEPTS UNDERLYING)

THE CLASSIFICATION OF NEPHRITIS

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APRIL 13, 1934
INTRODUCTION
The function of medicine is the cure of disease. All medical study therefore should be so directed as to facilitate most efficiently that end. The ideal, the most obvious cure is that brought about by removal of the cause, provided of course irreversible or non-reversible damage has not already been done.

The organs of the body are each individually susceptible to a number of pathological states which may or may not be shared by other organs and tissues of the body. The various diseases vary in those characteristics which together determine them, as etiology, course, prognosis, morbid anatomy, etc. These diseases usually can be separated into groups showing some common characteristic or characteristics which facilitate their study or handling to the end that they may be cured. So arises classification of diseases.

The ideal classification then is one based on etiology. Diagnose a disease, match it under its etiological category and the method of cure is indicated. However, there are certain obvious obstacles to this seeming state of medical Utopia. The etiology may be unknown or granting its recognition, means of removing it may be limited or altogether unavailable. Again, damage may have progressed to the point where it is irreversible and removal of the cause is in itself insufficient to restore normal conditions.

In spite of these shortcomings, be they present, classifications on the etiological basis is the classification of choice. Only insofar as the cause is apprehended...
and in the degree to which it can be combatted, so far can one go in the cure of the disease at hand—any other handling is palliative and if anything allows the morbid process, under a mask of apparent well-being, to advance farther toward the stage of irreparable damage or fatality.

What then if in the study of a disease there is no substantial etiological basis to work on? Such is the case with Bright's Disease or nephritis. Henceforth our discussion is to be limited to this specific morbid condition.

No two chemical substances react alike—for only insofar as they differ are we able to distinguish them as separate entities. In a similar manner, we may say that no two chemical compounds, toxins or organisms, react similarly. In other words, granted a delicate enough technique, we should be able, if not to determine the etiological agents directly, to distinguish them as separate entities, recognized by a specific morbid process. To lend hope and color to the above statements let me bring out the fact that Suzuki (8) has demonstrated how anatomically distinct are the renal lesions produced by inorganic salts such as those of mercury, uranium and chromium.

Let me summarize what has been said by saying that the ideal classification is one based on etiology, but failing this the most suitable mode of approach is thru the
substituted expedient of a study of the morbid process.

Volhard and Fahr in 1914 say this in regard to the place of the morbid process (taken from the monograph of Rolphe Floyd): "With full appreciation of the biological justification and importance of the principle that diseased departures from normal function are to be judged by functional standards, we still regard it as our ultimate aim to bring clinical symptoms into relation with anatomical findings; not, however, for the purpose of insisting on a controlling position of pathological anatomy, but because of the desire to better understand the abnormalities of function, that is to refer them, in the final analysis, back to definite pathological processes, to concrete pathological reactions, such as cell changes or demonstrable chemical products produced by disordered cell activity.

"Any grouping on the basis of functional distinctions must not on the pretext that functional alterations are beyond the reach of the microscope lead us to turn our backs on pathological anatomy. On the contrary, the study of function ought to tend to greater cooperation with anatomical progress. The failure of an histological method must stimulate us to elaborate a better technique; it can give no license to deny the histological basis of functional derangements.

"In other words, functional failure is not the chief,
but only a vital symptom. The same is true of the histological picture after death. The correct interpretation of functional failure and microscopic findings can only be had through their synthesis and the added appreciation of the time factor in forming an adequate conception of the disease process. Classification on any other basis is futile."

Forgetting for the moment the underlying motive in the above quotation to vindicate the pathological in respect to the clinical, it is an admission by the proponents of the most accepted pathological classification of the difficult task ahead in the complete correlation of the pathological with the clinical. It is a likely possibility that the basic etiology will be directly apprehended before or simultaneously with the complete understanding of the morbid process.

Friedrich Muller, the great German clinician, had this to say at the conclave in Meran in 1905: "Knowledge of disease does not reach its highest expression in ability to state at the bedside what pathological changes exist in the sick man; it is more profound to see the causes of illness as the starting point of our thinking. Formerly the pathological changes were regarded as the causes of symptoms, but they are better thought of as of common origin with symptoms." Here is expressed in a manner the futility with which pathological study was regarded before Virchow and
Fahr but it indicates a better basic insight as to the ultimate goal of the study of Bright's Disease, namely a clear etiological picture. Muller at this time went so far as to suggest an etiological classification but admitted its inadequacy at that time.

In summary, knowing the cause of a diseased process constitutes the ideal mode of attack toward a cure. In its absence, knowledge of the disease process itself, if it be sufficiently understood, is of great value as it in the end is specific for the corresponding cause. It may, in itself, disclose possible modes of combatting the process.

This paper shall not be concerned with analysis of modern classifications primarily, but rather will outline the development of the basic concepts which underly modern classification.
BEFORE BRIGHT
The story of nephritis before Bright is concerned with an attempt to understand the then baffling dropsies. That the kidneys were somehow involved was known for a long time. Hippocrates (460-?B. C.) mentioned a dropsy which he described as emanating from the lumbar region. (2) Aetius (500 A.D.) and Avicenna, the "Prince of Physicians"; (980-1057 A.D.), described dropsies due to renal disease.

The old physicians who began to do autopsies as a diagnostic adjunct knew as well as is known at present, several forms of nephritis and carried the hydrops back more or less clearly to nephritic diseases, as did Helmont: "Verus hydrops ascites est in renibus."

Morgagni (1682-1771) and also Bonet mention an ascites from renal alterations.

Dekker in 1694 perhaps first described the coagulation of urine by heat and acetic acid in certain cases which were obviously nephritic. (/4) Stephen Hale wrote of blood pressure in 1708 but the hemodynamometer of Poiseuille did not come out till 1828.

In 1764, Domenico Cottugno or Cotonnius (1736-1822), an Italian anatomist described a typical case of acute nephritis with edema, anuria and albuminuria. He too is given credit with first noting the coagulation of urine on heating. (2) He arrived at the conclusion that the urine of dropsical patients was albuminous while observing the
Increased urinary output with the absorption of dropsical effusions. He believed therefore that these dropsical effusions were eliminated thru the kidneys. He tested his belief as mentioned above by heat coagulation.

Wells in 1812 found the urine in postscarlatinal dropsy often bloody. In other such dropsical patients, with apparent absence of red blood cells, the urine being heat coagulable he believed blood serum to be present. He also showed that frequent pains in the lumbar region and material alterations in the parenchyma accompany coagulable urine.

Cruikshank in 1798 (Blackall in 1813 agreed with him) found that not all dropsical urines were albuminous. With these two types of dropsy, a division was made of dropsy with coagulable urine and dropsy without coagulable urine (cardiac) which proved of some importance for prognosis as well as for therapeutics. In other words, a cardiac and a non-cardiac (renal) dropsy were distinguished with as yet no proven or generally accepted relation of the latter to the kidney itself.

A. L. Leomie has this to say about this pre-Bright period: "The scattered and solitary references to morbid states of the kidney of earlier authors, although in some cases accurate descriptions of the gross pathological appearances (were made), were never as connected with
clinical histories, either in the printed record or the mind of the observer, as to suggest any relation between the two. Dropsy was regarded as the primary condition, the albuminuria dependent on hemic or other constitutional changes and the kidney affection as accidentally coincident."
RICHARD BRIGHT 1789 - 1858
one question may be asked in this place—do we always find such lesion of the kidney as to bear us out in the belief, that the peculiar condition of the urine, to which I have already referred, shows that the disease, call it what we may, is connected necessarily and essentially with the derangement of the organ? After 10 years' attentive—observation—I am ready to answer this question in the affirmative—. The above quotation is from an article by Doctor Richard Bright in 1836, Guy's Hospital Reports.

A. L. Loomis has this to say about Bright's place after discussing the inadequacy of previous authors:

"When, therefore, Bright pointed out the frequency of such renal changes, and not only their coincidence with, but pathological relations to albuminuria and dropsy, giving minute descriptions and drawings of the kidney changes, the application of the name Bright's Disease to all conditions associated with albuminuria and dropsy was an appropriate recognition of his invaluable contribution to renal pathology." Bright not only confirmed what was believed to be a possibility in many minds both before and during his time but proved it painstakingly. The microscope had again saved the day and now attention was turned from the clinical picture, with dropsy, etc., which had dominated the study up to that time to the pathological picture.

A pathological controversy dominated the scene henceforth. After Bright, workers, largely German, attempted to
work out the pathology of Bright's disease, and while consider-
erable progress was made, it still was impossible to suitably
correlate the pathological with the clinical picture. In
spite of the considerable knowledge at hand, when it came to
predicting the pathological state of the kidney from
clinical findings, autopsies revealed the unexpected often
enough so that numerous men lost faith in a solution of the
problem and reverted to purely functional and clinical class-
ification which reverting tendency persists even today. Del-
afield working in America presented a pathological classifica-
tion in 1880 which however was inadequate in the case of the
baffling chronic nephrites which were the thorns in the path
of progress. Volhard and Fahr in 1914, Germans, presented a
pathological classification which did give new strength to the
pathological movement. They analyzed chronic nephritis in
a manner which proved of definite practical value. Modern
pathological classification dates from their invaluable work.

The work leading up to their findings was long drawn out
and beset with numerous difficulties. A false division into
chronic parenchymatous and chronic interstitial nephritis was
advanced by two very eminent pathologists, Bartels and Vir-
chow which has not as yet been eliminated from the literature.
Also, their were deeply involved controversies as to the scope
of inflammation, as to whether it did or did not include de-
-11-
degeneration, and the terminology itself through persistent usage even after change of the basic ideas responsible for its introduction, added to the confusion and is still today the bugbear of the uninitiated medical student. There was also considerable doubt as to whether a single process or many were involved under the category of nephritis. Was Bright's disease a single condition or was it of plural nature?

Bright let us remember, described under "Bright's disease" only those conditions associated with urinary findings of albuminuria, etc., and with dropsy. J. B. Herrick in Osler and McCrae's Modern Medicine, 1914, has this to say in the course of discussing the terminology as it it used and its actual meaning. Nephritis, he claims, is used to mean non-suppurative inflammation of the kidney. This term is used interchangeably with Bright's Disease. Bright however, described only those cases characterized as mentioned above but did not include those cases without dropsy and also some forms, degenerative and atrophic in character rather than inflammatory.

Bright described three groups of the disease but was not altogether certain whether these were separate diseases or different stages of the same disease, though he evidently inclined to the former opinion. As to his first group, Fishberg states that from his description and figures it is clear
that at least some of the cases here described were instances of amyloid disease of the kidney. His second group, he states, includes various stages of nephritis or as Fishberg chooses to call it, glomerulonephritis, indicating a dominant glomerular role as a means of distinguishing it from other conditions usually included under the term nephritis in its broadest interpretation. His third group, Fishberg adds, are instances of chronic nephritis with secondary contraction.

Bright himself says: "Although I hazard a conjecture as to the existence of these three different forms of disease, I am by no means confident of the correctness of this view. On the contrary, it may be that the first form of degeneracy to which I refer never goes much beyond the first stage, and that all the other cases together with the second series, and the third, are to be considered only as modifications, and more or less advanced stages of one and the same disease." Amyloid disease is a separate entity, and as Fishberg states is a vascular degenerative condition simultaneously present in various organs. His second and third groups, as far as the majority of his cases run are in sequence.
BRIGHT to 1870
Bright's description of renal disease as the cause of albuminuria and edema was quickly accepted. He and his contemporaries soon developed the clinical symptomatology to a degree of completion. The question which next occupied some of the best medical minds was whether or no the variegated anatomical and clinical pictures described by Bright correspond to different disease or merely successive stages of one and the same process.

Rayer, a Frenchman, in 1840 maintained Bright's disease an inflammation of the kidney which he termed albuminous nephritis, but in his enthusiasm described six varieties of this inflammation. In 1842 ReKitansky made the first important disclosure by declaring the amyloid kidney structurally distinct and his contention was confirmed by Traube. Frerichs, however, came forward in 1851 with the statement that all Bright's disease was one process of inflammation, as did Rayer and as Bright hinted, and recognized three stages: An initial hyperëmia, a secondary period of exudation with fatty degeneration of the renal epithelium, and a third stage of connective tissue hyperplasia terminating in atrophy of the kidney. This unitaristic interpretation, because of its attempted simplification of matters no doubt, had many adherents for a long period.

In 1852 Johnson in England differentiated a number of
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diseases dependent on the state of the renal epithelium. In 1853 Wilke also upheld the plurality view. In 1856, Traube in Germany produced adequate clinical criteria to gain admission that the amyloid kidney (confirming Rokitansky) and chronic passive congestion of the kidney from heart failure were both independent conditions. It had now become definitely established that Bright's Disease was of a plural nature.

The most devastating outgrowth of this period was the concept of chronic parenchymatous nephritis and chronic interstitial nephritis, as was mentioned before. It was the result of an attempted pathological approach with a dysappreciation of the significance of the pathological changes—it was a purely quantitative appraisal of changes in an organ of such complex nature that a qualitative appreciation should be imperative before any conclusions could be drawn.

The concepts were introduced and fostered by two eminent pathologists, Virchow and Bartels. Virchow in 1858 suggested classification depending on three changes—alteration of the tubular epithelium and waxy changes in the glomeruli, and a newgrowth of interstitial connective tissue. Especially as the result of a systemic treatise by Bartels in 1877 it became customary to consider chronic Bright's disease as consisting of two varieties, chronic parenchymatous and chronic interstitial
nephritis.

Chronic parenchymatous nephritis was described as consisting of an inflammation of the epithelial cells of the kidney which, in accord with Virchow's conception of parenchymatous inflammation, went through the stages of cloudy swelling, fatty degeneration, and finally disintegration. The clinical manifestations of parenchymatous nephritis were notably renal edema, oliguria and marked albuminuria. Chronic parenchymatous nephritis included here those cases of chronic glomerulonephritis (Fishberg) in which edema was outstanding and besides chronic or true nephrosis.

Chronic interstitial nephritis, on the other hand, was thought to consist in a primary proliferation of the interstitial connective tissue of the kidney, which by pressure caused secondary atrophy of the parenchymatous tissue. The clinical picture of chronic interstitial nephritis was dominated by cardiovascular phenomena, notably increased arterial tension, arteriosclerosis and cardiac hypertrophy, with marked tendency to cerebral hemorrhage. Chronic interstitial nephritis was simultaneously applied to primary sclerosis and to those cases of chronic glomerulonephritis dominated clinically by cardiovascular phenomena.

The untenability of such a division of chronic nephritis is already apparent in that it splits up the modernly accepted
divisions. Weigert in 1879 demonstrated its untenability in a primarily anatomical study. He pointed out that interstitial changes were present in any instance and that this connective tissue proliferation is undoubtedly secondary to parenchymatous changes. Weigert considered then that in all cases the changes are primarily in the renal parenchyma. His objections were rendered valid by most of the leading students of the time. (Fishberg)

Rolfe Floyd has this to say in regard to this division. He states that in many instances symptoms and lesions follow the division as given but that in many, they do not. Albuminuria and edema occur with small kidneys when the main changes are not epithelial and also cardiac hypertrophy and uremia occur with large white kidneys. Practically all chronic nephritis is interstitial in the sense that there is definite stromal increase. The very important effect of glomerular changes is not adequately recognized, which effect will be taken up later, nor does the relative amount of epithelial and stromal change correspond to a relative development of symptoms supposed to be characteristic of each.
1870 to 1914
This period is marked largely by the acceptance of a primarily vascular sclerosis of the kidney and the idea is advanced that the glomerular lesion is the lesion indicating chronic inflammatory changes. Mention has been made of the plural or heterogeneous nature of Bright's Disease. Grainger Stewart in 1871, in fact, entitled his monograph "Bright's Diseases." Another entity was now separated. The development of the significance of the vascular lesions was initiated by the work of Gull and Sutton, 1872, who studied arterial diseases. They showed that primary change in the smaller renal arteries caused atrophy and could exist with or without renal symptoms. They regarded "arterio-capillary fibrosis" a primary general disease and Bright's Disease associated with it as secondary. As a matter of fact, the conception of renal atrophy secondary to sclerosis (vascular) is now regarded as established. Ziegler in 1880 also maintained a primary atrophy due to sclerosis as existing.

Later workers on the small renal arteries contributed the following. Friedman demonstrated elastic hyperplasia in the intimas of atrophic kidneys and noted its coincidence with hypertension. Jores confirmed Friedman and regarded the elastic hyperplasia the first stage of sclerosis. He showed sclerosis of the smallest renal arteries as characteristic of primary renal atrophy in contrast to its absence or meagre
development in secondary contraction. Fahr confirmed these results and regarded this articular lesion as the cause of hypertension. The lesion is really an elastic-hyperplastic thickening of the intima, rather than a purely elastic affair.

As a lesson in interpretation of clinical and pathological correlation Van Leyden in 1881 showed that with symptoms of a contracted kidney the lesion might be a large white kidney, an atrophy secondary to a primary enlargement, a waxy kidney, or a primarily atrophic kidney. Arterial changes causing such atrophy, he goes on, might be part of a chronic nephritis or they might be primary. In other words, a qualitative appreciation is vital to proper interpretation. He explains that vascular lesions when they occur early may give cardiac symptoms predominating over the renal symptoms before gross atrophy is apparent.

Klebs, Cohnheim and Nauwerk emphasized the importance of the glomerular lesion. Nauwerk described and considered constant the glomerular changes in acute cases. He believed tubular atrophy then dependent on damage to the corresponding glomeruli. Von Kahlden, on the other hand, believed the tubular changes essential and primary in acute cases. In 1907 Lohlein showed glomerular changes the basis of Bright's Disease. He believed both tubular changes and the chronicity of renal disease dependent on preceding glomerular changes. In a
large number of cases he failed to find chronic parenchymatous nephritis without tubal glomerular lesions. He distinguished focal inflammation from diffuse nephritis. "The investigation of Lohlein, confirmed in this country by Bell and Hartzell, have demonstrated clearly that the highly variegated clinical and anatomical pictures of chronic glomerulonephritis are later stages of acute glomerulonephritis, the tubular, interstitial and vascular changes all ensuing subsequent to the stage of an acute glomerulitis." Fishberg, Senator, 1880-1906 recognized that there were acute cases with lesions of the tubules and glomeruli alone and others with stromal changes as well. In the later stages he recognized all cases showed stromal changes.

Friedrich Muller in 1905 first suggested the use of the term nephrosis for the degenerative varieties of renal disease, as separate from the primarily inflammatory varieties. A definite classification at that time, however, was impractical as the nature of degenerative changes was still in question, and in the minds of many still is. A classification was first shown feasible by Volhard and Fahr in 1914. They adapted the term nephrosis, as suggested by Muller, for the primarily degenerative diseases.

Christian, in a recent article (10), says this as to the place of nephrosis: "The present concept of a duality of
lesion (initial glomerular and primary vascular) places very little importance on changes in the tubules except for those who regard so-called nephrosis primarily as a form of tubular degeneration, a view not held by a large number of investigators. Yet Volhard and Fahr, Fishberg, Elwyn, Bennett, Van Slyke and O'Hare, for instance, include nephrosis in their classifications. Let us mention here the squabble over what constitutes inflammation and what degeneration, where does one begin and where does the other end, what is nephritis and what is nephrosis? Aschoff and Oertel, according to Oliver, go off on the tangent of trying to make inflammation an all-embracing term. In other words, according to their view, nephritis should include also what are considered by many primarily degenerative states. Ribbert, on the other hand, admitting the presence of inflammatory processes, believes dysfunction can be the result only of degeneration (regressive changes) and so insists that all types of nephritis be called nephrosis. "Nephritis and nephrosis are all things to all men."- Oliver. There is however a middle ground to fall back on, and most of the leading writers either describe in detail or at least mention a true degenerative lesion or nephrosis, primary in the tubules.

We should mention in this paper the status of functional classifications. In France the tendency developed to
disregard pathology as a basis of classification and there was emphasized instead the importance of functional changes. Brault and Dieulafoy regarded the intensity of irritation and the duration of the process as the principal factors determining postmortem findings. Chaufford and Laederich, 1909, regarded postmortem findings incidental to the intensity and duration of the inflammation. As Fishberg points out, there is a present day tendency to "functional thinking." This tendency, as was mentioned, has been especially marked in France. Due largely to influence of Widal and his followers, nitrogen retaining (azotemic) and chloride retaining (chloremic) types of renal disease are recognized. Fishberg discusses this division. The so-called functional classification he says, is not based on a unitary principle and is moreover irrational in practice. He states that only one type of renal function is known, that in which all the excretory functions are damaged. This classification, he states further, is actually a distinction between those renal diseases in which the function of the kidney is impaired and those in which it is intact, but there exists an extra-renal cause for edema formation which is associated with chloride retention. Thus, he goes on, it is not uncommon in acute glomerulo-nephritis, to find in the same patient both nitrogen retention in consequence of renal insufficiency and chloride
retention as a result of an extrarenal factor which leads to edema formation. In other cases, there is at first edema formation with chloride retention despite good renal function, but as the disease progresses renal insufficiency with nitrogen retention develops coincident with the clearing up of the edema. Such functional classifications as have been advanced he concludes are not satisfactory therefore.
PRESENT DAY CLASSIFICATIONS-CONCLUSION
Here is a quotation from Clifford Allbutt's preface to his "Diseases of the Arteries." There is a temptation "to put a resemblance of order and completion upon data and inference which in the nature of things must as yet be fragmentary and scattered; or to work up into artful categories and systems motions which as yet should be left as guesses or suggestions. By leaving some data in the background and bringing others into high relief, by painting a little color here and throwing a little shadow there, it is not difficult to construct an argument far more taking, far more readable than pages of outlines still vague, of meanings still tentative of facts still insecure and unbalanced." Such a quotation applies most completely to the attempted classifications of nephritis as presented today. As evidence to its inadequacy, one has only to consider the modes of treatment, the rational presented therefore and the results obtained therefrom. Treatment is instituted on a basis which lies, in the course of the morbid process, far removed from the original or inciting cause or causes.

The classifications of Bright's Disease presented in recent years, notably those of Christian and Addis have, according to Fishberg, not been shown to possess any special advantage and have not been widely used.

Christian (10) has attempted to show the similarity of
all classifications, and in this he includes his own, Volhard and Fahr's (1914), Elwyn (1926), Bennett (1929), Widal, Lemierre and Vallery-Radot (1929), Van Slyke (1930), Addis (1931), Fishberg (1931), Mosenthal (1931), and O'Hare (1931).

The apparent differences in the various classifications, he states, are more a matter of words than of concept. All classifications take into account a concept of time and so are acute or chronic. There is, to be sure, a less definite, so-called subacute group of various authors. Pathologically there is accepted a group of diseases based on a primary glomerular lesion—glomerulonephritis (Fishberg). There is also accepted a vascular group. As Christian states, there is no acute form recognized in the essential vascular condition. He therefore states as the basis of all classifications a division into acute and chronic nephritis with an initial glomerulitis and chronic nephritis condition with an essential vascular lesion. Any further subdivision depends on the fancy of the author. He fails to mention nephrosis in spite of the fact that the overwhelming majority of the classifications he considers take it into account. The classification of Addis and Oliver attempts to evade the issue over nephritis and nephrosis by disregarding these inveigling terms and suggest a classification with the same clinical differentiation.
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of its groups (nephritis, nephrosis, vascular) but base it on a rather constant urinary finding, hemorrhage, its presence or absence, as a substitute for a pathological admission. They regard hemorrhage as the "in vivo" indicator of the type of activity of the renal lesion. Their classification is thus into the similar grouping, hemorrhagic, non-hemorrhagic, and arteriosclerotic Bright's Disease.

I have attempted in this paper, without bringing in too much confusing detail or a too analytic interpretation of specific classifications to state as simply as possible the basic concepts back of these classifications as a group. I have expressed the general opinion that an etiological understanding is the end goal of the study of nephritis and should not be lost sight of in side-tracked controversies of minor significance. In its broadest sense perhaps the meaning of the "morbid process" includes within its bounds the etiological agents themselves.
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