From Dropsy to Bright's Disease to End-stage Renal Disease

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"BRIGHT'S DISEASE" WAS THE ONCE FAMILIAR term applied to diffuse noninfectious bilateral renal disease, usually marked by albuminuria and sometimes by the symptom complex of uremia. "ESRD" stands for "end-stage renal disease" and this acronym entered widespread use as a result of Section 2991 of Public Law 92-603 passed by Congress on 30 October 1972. This unprecedented legislation provided federal financial support to essentially all Americans with a particular chronic disease—kidney failure—so that they could receive treatments to prolong their lives. The treatments were, of course, dialysis and transplantation. Neither Bright's disease nor ESRD referred or refers to any single disease entity as defined by pathologic examination or by cause.

Knud Faber (1930, 211) in his book *Nosography*, still honored with rereadings sixty years after its publication, said about morbid categories that the clinician "cannot live, cannot speak, cannot act without them." This remains true, and in this article I will return to these names, Bright's disease, ESRD, and others, these labels placed on categories of sickness, and look at them more closely. I hope to show how the changing use of names in renal medicine reflected how physicians and others thought about kidney disease. Certainly one theme must be ways in which individual physicians initially, then the community of scientific medicine, and finally government or society, fashioned both conceptions of disease and the names which label

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the conceptions. I also will attempt to suggest how some patients have thought about their kidney disease, or at least how they experienced it. The patients' contribution to the idea of a disease remains far more elusive, less readily traced, than that of the physician. Yet, it warrants inclusion, even if fragmentary, and assumes perhaps greater importance near the end of my narrative, when renal patients become dialysis patients. My article will not provide a comprehensive account of the evolution of pathological categories, etiology, and management of renal disease, though such would be a worthy and so far undone task. I will be instead episodic and selective, as I seek to reconstruct the way in which successive generations have construed the experience of kidney disorder, and constructed its nomenclature.

Dropsy

The prehistory of Bright's disease was dropsy. From a universe of dropsical patients, Richard Bright of Guy's Hospital beginning in the 1820s would recognize a special subset, the forbears of all renal patients.

For hundreds, perhaps thousands, of years two forms of serious chronic disease dominated medical practice. They induced in their victims equally grotesque but oddly antipodal transformations. The one was consumption, which thinned and shrank the body; the other dropsy, which bloated it. Physician and nosologist William Cullen discussed the species of disease called dropsy in the 1787 edition of his popular *First Lines of the Practice of Physic*. Dropsies were "distinguished from each other according to the parts they occupy, as well as by other circumstances attending them; yet all of them seem to depend upon some general causes, very much in common to the whole." Dropsies are often associated with a scarcity of urine. "This scarcity of urine may sometimes be owing to an obstruction of the kidneys, but probably is generally occasioned by the watery parts of the blood running off into the cellular texture, and being thereby prevented from passing in the usual quantity." Cullen did not see dropsy as a "cardiac" or "renal disease." His analysis remained more general and symptomatic than local and pathological.

Let us focus on one man's dropsy. In the last year of his celebrated life, writer and lexicographer Samuel Johnson suffered with asthma

and dropsy, and told of the experience to Boswell, to his doctors, and to other friends (Johnson 1952). On 11 February 1784 he wrote to Boswell: "The asthma, however, is not the worst. A dropsy gains ground upon me; my legs and thighs are very much swollen with water, which I should be content if I could keep it there, but I am afraid it will soon be higher."

Then in late February of 1784 a remarkable event relieved Dr. Johnson's pulmonary and peripheral edemas: "Last week I emitted in about twenty hours, full twenty pints of urine, and the tumour of my body is very much lessened, but whether the water will not gather again, He only knows by whom we live and move." Boswell included this mighty postdevotional discharge of fluid in the *Life*, making it unquestionably the most famous diuresis in the history of the English-speaking people. But Johnson's suffering was not over. On July 21 he wrote to one of his physicians: "The water has in these summer months made two invasions, but has run off again with no very formidable tumefaction." Into late summer and early fall, armed with archaic diuretic remedies squill and cantharides, Johnson battled the floods, which would rise and fall. On November 4 he reported (to John Ryland) that the "water grows fast upon me." Two days later (to Dr. Brocklesby): "The water encreases almost visibly and the squills which I get here [Lichfield] are utterly inefficacious. My spirits are extremely low."

Johnson eventually succumbed on 13 December 1784, and the autopsy revealed both cardiac and renal disease (the right kidney atrophic, the left cystic). But in life Johnson, like many similar patients had *dropsy*, not Bright's disease or congestive heart failure. The experience of his disease was dreadful: a constant struggle against the drowning of his body by floodwaters from within, a struggle waged with drugs he knew to be inadequate, unpredictable, and noxious.

Bright's Disease

Widespread in private and hospital practice, hideous and lethal when advanced, dropsy captured the attention of some early nineteenth-century physicians who were remapping diseases based on morbid anatomy and physical examination. In 1827 Richard Bright of Guy's

Hospital (1789–1858) published the first volume of his magisterial *Reports of Medical Cases*. Almost one-half of this volume deals with dropsical patients, and the first 24 cases reveal Bright's discovery. Certain patients with dropsy have albumin in their urine (detected by the spoon and candle—heat coagulation), and—previously unrecognized—many of these show striking morbid changes of the kidneys at autopsy. Furthermore, many such dropsical patients experience characteristic symptoms, including vomiting, headaches, pericarditis, seizures, amounting to what later would be recognized as uremia. Bright's discovery has been hailed as one of the gleaming achievements of clinical/pathological correlation. Dropsy was ubiquitous, heat-coagulable urine little explored, the uremic symptoms variable, the patients hectically sick, the autopsies undoubtedly odorous and oozing; yet, Bright made sense out of all this, and established the basis of renal disease.

Not only was the detection of albuminuria, as Bright used it, arguably the first practical laboratory aid to diagnosis, but Bright went farther. He encouraged several physician-chemists to perform analyses of the urine and blood of some of the renal patients (Peitzman 1981). William Prout, John Bostock, and George Owen Rees showed that sometimes urea was detectable in the blood but deficient in the urine, while the albumin was decreased in the serum. In 1842 Bright received permission from the managers of Guy's Hospital to assign during the summer a number of beds to receive only renal cases, for careful prospective clinical and laboratory study. This hinted at the "metabolic ward" or clinical study unit of the next century.

Bright's way of understanding the new disease was, however, solidly of the early nineteenth century. Although he recognized that albuminuric renal dropsy could follow scarlet fever, Bright stressed in all his writings *exposure to cold* as the most important source. Cold could "suppress the insensible perspiration," leading to "sympathy" between the "checked" skin and the kidneys. This sort of language and way of thinking go back at least to Galen, and the physician of 1989 simply no longer shares them with Bright and his day. Yet, for Bright the association of cold with renal disease was not a "theory" or speculation, but indeed a repeated clinical observation: the patients he cared for and reported almost all did recount some recent exposure to cold and wet as part of their story. Cold gave way to microbial

explanations, then these to immunologic models, as accepted causes of diffuse kidney disease.

We ought to be on firmer grounds with Bright's morphological descriptions, enhanced by the magnificent hand-colored mezzotint engravings appended to his *Reports*. Dr. Bright in the initial 1827 publication, based on 24 cases, suggested three forms of that deranged kidney structure which accompanies albuminuric dropsy. The first is a kind of softening with yellow mottling. The second form is one in which "the whole cortical part is converted into a granulated texture, and where there appears to be a copious morbid interstitial deposit of an opake white substance." The third form of disease "is where the kidney is quite rough and scabrous to the touch externally, and is seen to rise in numerous projections not much exceeding a large pin's head, yellow, red, and purplish. . . . The form of the kidney is often inclined to be lobulated [and there is a] contraction of every part of the organ" (Bright 1827, 67–69). These three categories and the descriptions hold little meaning for the nephrologist of the 1980s, who sees other striking differences—mainly ignored by Bright—when viewing the colored plates. There are two bases for the difficulty. First, Bright relied on a sort of macroscopic tissue pathology of the 1820s derived from Xavier Bichat and (more directly) from Bright's colleague at Guy's Hospital, Thomas Hodgkin. Second, the renal physician today rarely sees or touches a fresh diseased kidney, and does not replicate the visual and tactile examinations at autopsy which for Bright repeatedly assured in his mind the reality and reproducibility of albuminuric renal disease.

Importantly, Bright allowed that the three forms he distinguished might be only stages of one process. But he seemed to favor three categories. So from the first publication on the disorder, Bright's disease was not held to be one specific entity, not even by Bright.

Although Richard Bright surely did not propose the name, the disease or diseases he described became known almost immediately as "morbus Brightii," "Bright's kidney," "Bright's disease," or "maladie de Bright." In fact, I believe that Bright's disease may be the earliest regularly used eponymous name for a disease in English. It is the only such disease name in the index to the 1844 edition of Thomas Watson's popular *Lectures on the Principles and Practice of Physic* (unless one counts "St. Vitus's Dance"). Watson did not, however,

like the term. "For this disease," he wrote, "we have no appropriate name. I wish we had. Some call it *granular degeneration* of the kidney, but the epithet granular is not always applicable. It is most familiarly known, both here and abroad, as *Bright's kidney*, or *Bright's disease*; after the eminent physician who in 1837 [sic; should be 1827] first described it, and showed its great pathological importance. These are odd-sounding and awkward terms; but in the lack of better, I must employ them."

Why did the naming of this new disease pose a problem, and lead to a new way of naming diseases? One obvious answer is the recognition, already noted, that no one morbid appearance defined Bright's disease. But I would argue there was more to it than this. Bright's disease required a new sort of naming because it represented a new, nineteenth-century, way of thinking about and defining disease. A patient had Bright's disease if he had at least some of the following: certain symptoms, such as dropsy; certain physical findings, such as a hard pulse, or a pericardial rub; certain morbid changes in the kidney, if he came to autopsy; and—perhaps most novel in the 1820s and 1830s—a primordial laboratory abnormality, albuminous urine. The last two of these elements—albuminuria, morbid changes of the kidney—could only be detected by the physician. So this sickness is no longer mainly the patient's disease: it's also the physician's disease, Dr. Bright's disease. Bright recognized that not all these elements were present in every such patient. For example, case 4 of his 1836 "Cases and observations illustrative of renal disease accompanied with the secretion of albuminous urine" provides "a strong example of the disease of the kidney passing to its most fatal period, without the slightest symptom of dropsical effusion—a state of things, which, above all, is apt to throw us off our guard" (Bright 1836). Gradually it would become clear as well that some patients might progress to renal failure without ever showing important albuminuria. Others might display profound anasarca and albuminuria although their kidneys show no defect to the naked eye or even to the light microscope. Bright's disease then may be said to have been understood as a way of getting sick through your kidneys. It was a term presumably useful, even indispensable, to most physicians. It was a term which I suppose (without strong evidence) gradually became in some way comprehensible and useful to patients.

By 1950 the term Bright's disease evidently lost much of this

usefulness; doctors found it old-fashioned. The last two monographs in English that I can verify with "Bright's disease" as part of the title both appeared in 1948, one by Henry Christian, the other by Stanley Bradley. Thomas Addis, a leading student of renal disease from 1915 to 1949, wrote two books. His first appeared in 1931 titled *The Renal Lesion in Bright's Disease*; the second in 1948 he called *Glomerular Nephritis*, though its content extended well beyond that particular form of renal disease (Addis and Oliver 1931; Addis 1948).

Not until writing this article did I perceive the vacancy left with the demise of "Bright's disease." Currently, no term mutually ratified by patients and physicians adequately expresses "getting sick through your kidneys." Without such a joint frame of reference, I often find it difficult to discuss a new diagnosis of chronic renal failure with a patient.

Before we leave Richard Bright and Guy's Hospital, let me try to evoke the *patient's* experience of Bright's disease in the nineteenth century, so that I can later draw a contrast with the present day. Here is Bright's (1827, 29–31) description of case 13 of the *Reports*, Thomas Drudget, a carman hospitalized on 7 December 1826:

> About a fortnight before his admission he was attacked with sickness at the stomach, and shortness of breath; purging then came on, and vomiting: about nine days before admission his face and legs began to swell. The urine had been deficient in quantity the whole time. He complained much of tenderness in the pit of his stomach.

His urine coagulated to heat. Drudget was treated with cupping over the chest, mercurials, magnesium sulphate, tincture of camphor, potassium supertartrate, jalap, and capsicum, some of which brought temporary improvement. But on the 17th he mentioned to some of his ward mates of a headache. "About eight o'clock it was observed that he lay in bed making a very singular noise, and on going to him he was in a state of profound apoplectic stertor. Mr. Stocker was immediately called; took away twenty ounces of blood from the temporal artery, gave him ten grains of calomel, and a colocynth injection. He had one or two fresh attacks, accompanied with so much convulsion that he could scarcely be held in bed." Bright orders more bloodletting, an enema, and a cantharidis plaster to the neck, but to no avail: another case enters the *Reports*. The *sectio cadaveris* shows an

intracranial bleed as the terminal cause of death; the kidneys are pale and soft with a "motley granulation."

Edematous and convulsive, poor Drudget may stand in for all the victims of advanced Bright's disease before the invention of dialysis. I might have chosen a patient that Robert Tyson, an American authority on Bright's disease, presented to students on 20 October 1892 at University of Pennsylvania Hospital. She was a 55-year-old woman whose massive "collection of fluid ruptured the skin." She had albuminuria and granular casts in the urine, and was treated with milk and caffeine (Swan 1890–1893). Tyson sometimes used pilocarpine, often his beloved "hot-air bath," and even venesection in refractory cases of Bright's disease (Tyson 1881, 120–23, 137–47). Or I might have described Thomas Addis's (1948) patient from the 1930s, the young physicist whose case (possibly a composite) is poignantly and instructively narrated in *Glomerular Nephritis*. Over many years Addis helped prolong this patient's useful life with diet, simple medications, small Southey's tubes to drain tissues, and encouragement. Eventually, edema and nausea announce that the disease has entered a conclusive stage, confirmed by changes in urinary sediment, albuminuria, and measures of azotemia. Finally, Addis offers paraldehyde to ease the death of the young man, edematous, nauseated, and exhausted with terminal uremia. Any of these cases and countless others recorded reveal the patient's experience of renal disease in earlier times, and the physician's struggle to cure or palliate it.

Nephritis in Threes

The names that came to compete with Bright's disease, or designate its subcategories, indicate the way that nineteenth-century authorities wished to think about the disease. That way remained increasingly anatomic. The term "nephritis" existed well before the writings of Bright and his contemporaries, and appears for example in Cullen's (1800) *Nosology*. The suffix "itis" even by then conveyed the idea of inflammation—that is heat, redness, pain, swelling, loss of function. Bright strongly suggested that his disease might be a state of congestion or inflammation seated in the kidney, and elsewhere (e.g., pericardium). Though not all subsequent physicians and pathologists would agree on the essentially inflammatory nature of Bright's disease,

the term "nephritis" entered use by 1840 and the task of classifiers reduced to adding the appropriate modifiers.

The story of the superceding and competing classifications is far too tedious to explore except in the broadest of strokes. Oddly, nephrologists and pathologists looking at altered kidneys have always favored as much as possible a tripartite organization, either seeking simplicity or emulating Dr. Bright. Virchow in 1858 suggested "parenchymatous nephritis," "interstitial nephritis," and "amyloid degeneration" (Bartels 1877; Tyson 1881, 79–84). George Johnson in 1873 proposed the separation of an acute form (acute nephritis); and three chronic varieties: "red granular kidney," "large white kidney" and "lardaceous kidney" (which is the same as amyloid kidney). Osler in his influential text favored "acute Bright's disease," "chronic parenchymatous nephritis," "chronic interstitial nephritis" with amyloid dispatched to its own pathological category (Osler 1909, 686–703). In the twentieth century, the extremely influential monograph by Volhard and Fahr in 1914 provided a fresh—but still trinitarian—organization: degenerative diseases, "the nephroses"; inflammatory diseases, "the nephritides"; and arteriosclerotic diseases, "the nephrosclerosis." Thomas Addis (1928) of Stanford University offered a modification of this last framework which gained some popularity: "hemorrhagic Bright's disease," "degenerative Bright's disease," and "arteriosclerotic Bright's disease." (Even today nephrologists seek first to place a new case of renal parencyhmal disease into one of three broad categories: glomerular disease, tubulo-interstitial disease, or vascular disease.)

The point is that nineteenth-century physicians interested in Bright's disease relied on gross and microscopic pathology to organize their thinking and teaching about the disorders. Increasingly, examination of the urinary sediment aided clinicians, for the sick kidney accommodatingly sheds bits of itself into the urine. Thus, all later nineteenth-century texts on Bright's disease contain extensive discussion of *casts*, often carefully illustrated. Sediment examination extended histologic diagnosis to the living patient, as would renal biopsy beginning in the late 1940s. Chemical examination, such as blood urea measurement, though precociously commenced by Bright's collaborators, lay nearly dormant until the invention of simpler assays in the early twentieth century.

Functional Diagnosis and Bright's Disease

Although pathology remained the underpinning of organized knowledge and diagnosis in Bright's disease, clinicians of course continued to struggle at the bedside with its obvious functional derangements—dropsy and uremia. In the last decade of the nineteenth century and the early part of the twentieth, some investigators subjected Bright's disease to a more subtle and formal sort of "functional diagnosis," using the laboratory. Methods and thinking analogous to those applied to the stomach and heart were applied to the kidney. In the late 1890s Alexander von Koranyi used cryoscopy to measure the "power" of renal concentrating ability in health and in renal disease (Faber 1930). As other workers added tests of dye excretion, test meals, and urea loads, the term "renal insufficiency" entered the language of Bright's disease (Faber 1930, 112–171; Fishberg 1930, 39–54). The "insufficient" kidney lacked normal power and reserve; further weakening later became known as "renal failure," which meant frank retention of urea and other substances usually discharged by the renal filters.

Thomas Addis, mentioned earlier, represents one of several figures transitional between the dominantly anatomic and dominantly functional ways of envisioning renal disease. An appealing and enigmatic figure, Addis (1881–1949) was a Scots physician with sound chemical training hired as a young man by the new Stanford University medical school in 1911. There he took up in the clinic and in the laboratory a lifelong study of Bright's disease. He titled both his Harvey Lecture of 1928 and his monumental book of 1931 (with pathologist Jean Oliver) *The Renal Lesion in Bright's Disease* (Addis and Oliver 1931). By "renal lesion" he meant both the type of disordered structure *and* the amount of functional loss. His scheme for pathologic classification gained validity from his method of standardizing and quantifying the urine sediment examination; that is, the classification and the "Addis count" were inseparable in his mind, and the clinical usefulness of the one depended upon the other. "Renal lesion" as loss of working renal mass Addis estimated by a timed measurement of urea content of blood and urine expressed as the "urea ratio" (urine urea per unit time/plasma urea concentration, or UV/P). Now this ratio can claim physiologic meaning as the urea clearance. But Addis thought of this number as the amount of "still functioning renal tissue." In a letter

to Alfred Cohn of 15 May 1934 about a patient, Addis reports "her old 'ratio' figures which show that she then had 69% of the amount of renal tissue proper for her size" (Cohn papers 1934). He did *not* state that her urea clearance was 69% of normal. Indeed, Addis occasionally decried the introduction of too much physiology into medicine, and in 1931 urged a return to the "straight and narrow road of morphology" (Addis and Oliver 1931, 4–5).

But physiologists such as Homer W Smith and A. Newton Richards in the 1930s and 1940s would build a language of clinical renal physiology based largely on the mathematical idea of clearance and the measurement with it of glomerular filtration rate. Physiological analysis increasingly displaced structural thinking as a medical subspecialty called "nephrology" matured (Peitzman 1986). Eventually, measurement of creatinine succeeded urea in importance, and the nephrologist of the 1980s uses mainly the serum creatinine as the cardinal indicator of renal health. An elevated creatinine value in a patient *is* "renal failure," and translates—if thought about more—into a decreased glomerular filtration rate. It does *not* call to mind an anatomic image of shrunken renal tissue, as did the urea ratio for Addis, who frequently *saw* such kidneys at autopsies. For the nephrologist of the late twentieth century, the serum creatinine does, however, have associations, is more than an abstract number. To the renal fellow called to see a new consult in the emergency room, a creatinine of "ten" (milligrams per deciliter) conveys far more than twice the urgency of a creatinine of "five." It means "decide about dialysis now," instead of "call me back when the work-up is underway." As with the tone of a clarinet, qualitative changes occur as one goes up the scale of serum creatinines.

ESRD—Disease of Entitlement?

The introduction of dialysis and renal fellows into this discussion leads to the last term to be investigated: "ESRD"—end-stage renal disease. It is, perhaps, the strangest of all labels applied to those with diffuse renal disease.

In 1948 Henry Christian published a small book on renal disease which he could still title *Bright's Disease*. In the same year appeared Thomas Addis's *Glomerular Nephritis*, which movingly recounts one

patient's decline and death from terminal uremia. One wonders if either author had noted the appearance in 1947 of a slim and somewhat obscure paperbound monograph called *New Ways of Treating Uremia* by Willem Kolff. Kolff in the 1940s was the most successful of several workers who devised practicable artificial kidneys (hemodialysis). In its first decade or so of regular use, hemodialysis mainly aided patients with acute and reversible renal failure. Then, in the early 1960s engineer Wayne Quinton and physician Belding Scribner perfected the arterial-venous shunt, which allowed repeated dialysis treatments and the indefinite life support of persons with advanced irreversible renal failure and uremia (McBride 1984). (Scribner first became fascinated with renal disease during an elective course for senior medical students with Thomas Addis at Stanford.)

In 1972 Congress included within Public Law 92-603 a provision by which "chronic renal disease [is] considered to constitute disability" (Sec. 2991). This law provided federal financial support for almost all Americans requiring chronic dialytic treatment. Within a few years the "end-stage renal disease program" under Social Security was formed to implement and regulate this support. "End-stage renal disease" was defined in 1974 as "that stage of renal impairment that cannot be favorably influenced by conservative management alone, and requires dialysis and/or kidney tranplantation to maintain life or health" (*Federal Register* 1974). Although clinicians may have used the phrase "end-stage renal failure" or the like before the passage of P.L. 92-603, with the initiation of the federal program ESRD became a disease *defined by eligibility to receive funding for a particular treatment,* a disease of entitlement.

The typical patient with ESRD today is a 55-year-old person with diabetes complicated by retinopathy and nephropathy. When the patient's creatinine reaches the range of 5 to 7 (milligrams per deciliter), surgical preparations are made for chronic hemodialysis or peritoneal dialysis. The patient then has ESRD. Dialytic treatments may begin when the person reports morning nausea or occasional vomiting, perhaps skin itching or some other early uremic symptom. There are exceptions, of course, and some patients do present with advanced uremia and edema. But ESRD is not the same as "uremia," since most patients will usually begin the dialytic treatments soon enough to avoid all but the early and subtle symptoms. Nor is ESRD equal

to "chronic renal failure" (CRF), since many patients with creatinines of 4 or 5 are said to have "CRF" but still not ESRD.

ESRD, a legislative phrase, *can* lay claim to being a disease. It has become something patients can *have,* although relatively few use the term. One who did, a dialysis patient in Georgia, wrote: "Possibly the name is most frightening, end-stage renal disease. It sounds like the end of the world. That's the way I felt [upon first learning of it]. I cried and cried" (Jones 1984). ESRD is a disease nephrologists can write books about, such as *End-stage Renal Disease, An Integrated Approach* (Stone and Rabin 1983).

Moreover, ESRD like other diseases encompasses a set of characteristic signs and symptoms. These are a mixture of late consequences of renal failure rarely seen until dialysis lengthened the lives of renal patients and complications of the treatment itself. Examples include chronic anemia, renal bone disease, aluminum toxicity, dialysis-induced hypotension, and especially clotting and infections of catheters and shunts for dialysis. These last complications harry the days of dialysis patients. Today, since ESRD treatment is started before most patients suffer from engulfing edema or florid uremia *the experience of ESRD is mainly the illness of dialysis. The treatment becomes the sickness.* When renal patients write about their experiences—and a fair number do—they write mostly about *being a dialysis patient,* about the hours on the machine, about their remarkable ability to cope and prevail. Here are some examples:

> Next I spent almost five years on home hemodialysis with my mother as "nurse," two years on CAPD, then back to the machine, in-center. I had a parathyroidectomy in 1976 and had my "closest call" that year with a case of pulmonary edema (Jones 1984).

> I personally am convinced that the physical aspects are very uncomfortable, and I could gladly do without nausea, cramps, and hypotension so bad I have to crawl into bed after a sudden drop in pressure—because if I stood up I would pass out (Sand 1986).

> Home dialysis, like many things in life, had its high and low moments. There were times of sharing friendship and thankfulness for extended life and its meaning and pleasures. There were also times of despair, crisis, pain and weariness (Campbell and Campbell 1978).

> I suppose the longer an individual is on dialysis, the more he

> incorporates the process into his life. When I am at dialysis I am more willing to take an active role, and when I am away from the unit, the thought of dialysis has an easier time escaping my thoughts (Levi 1984).
>
> The first time I saw the kidney machine I thought it looked like a big washing machine. I said "I hope you all aren't going to put me in that machine!" (Rosenberg 1980)

The physician's experience of renal disease has changed as well. Relying on sequential laboratory measurements, early dialysis, and potent diuretics, the nephrologist today only rarely encounters fulminant uremia, or dropsy bursting through the skin. Indeed, the renal fellow only a few times in a year enjoys the palpable fulfillment of seeing his dialysis machine staunch seizures, penetrate the coma, defeat severe uremia. The chronic dialysis patient has been called a "marginal man," suspended between the worlds of the sick and the well, beneficiary and victim of a half-perfected technology. The renal physician, oddly, shares with his ESRD patient an incomplete, unresolving medical encounter, as he struggles more with the woes of chronic dialysis than with the woes of uremia. The nephrologist has even lost touch, literally, with the diseased kidney itself, the old granular kidney of Dr. Bright. Autopsies of ESRD patients seldom occur, and the renal doctor now sees kidneys only microscopically, a biopsied bit at a time, or views shadowy reniform images on an ultrasound screen.

Be assured that I am *not* calling for more uremic seizures and deaths, or a return to Southey tubes. Clearly, the dialysis machine *is* lifesaving. It is the routinization and scale of that lifesaving, with its daily imperceptibility, that are both extraordinary and numbing.

This article has dealt mainly with names. The naming of diseases, I argue with Knud Faber and many others, remains essential to the modern physician who must select a diagnosis and recommend treatment. To most patients, the name of the illness matters less than does getting well, but sometimes contains meaning. Samuel Johnson in 1784 wrote: "My diseases are an Asthma and a Dropsy, and, what is less curable, seventy five." "End-stage renal disease," wrote the dialysis patient in Georgia two hundred years later, "it sounds like the end of the world." "Dropsy," "Bright's disease," "nephritis," "ESRD": each of these names is to some extent elusive, ambiguous.

None refers to a well-defined etiologic or pathologic entity. These terms suggest to me an increasing complexity in the encounter of renal patient and of physician. Evident and clear to both, *"dropsy"* was disease as symptom. *"Bright's disease"* was more the physician's abstraction, with two of its key elements—albuminuria and renal structural change—invisible to the patient. Yet, I believe "Bright's disease" could nonetheless make sense to patient and doctor talking together. The still evolving histologic nomenclature of *"nephritis"* and *"glomerulonephritis"* represents a language of the renal specialist, too esoteric for patient or general doctor. Finally, *"ESRD"* is a diagnosis often uncovered by autoanalyzer, defined by need for dialysis, and formally bestowed by government.

Each term has had its use, its particular reality, and its message. Each reflects in some way the experience of the sickness felt and questioned by the patient. Each reflects as well an experience of the disease observed, contemplated, classified, and treated by the physician—treated with good intent, and with the imperfect medicines and machines of the day.

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