Francis Delafield (1841-1915):
The original contributions of an American investigator to diseases of the kidney

Joslyn L. Campbell, Garabed Eknoyan
Renal Section, Department of Medicine, Baylor College of Medicine, Houston, TX - USA

ABSTRACT: The growth of our knowledge of diseases of the kidney has been the work of brilliant individuals of talent and passion, who have become some of the most respected and best remembered figures in nephrology. Unfortunately, we often forget that in many cases there were others, no less brilliant, whose vision and passion to study diseases of the kidney was no less intense but whose contributions have been forgotten. Francis Delafield (1841-1915) is one of those important but overlooked figures. An 1863 graduate of the College of Physicians and Surgeons, he continued his studies in London and Berlin, and upon his return to New York became a pioneer in histopathology in general and that of kidney disease in particular. He provided detailed and accurate microscopic descriptions of kidney pathology, made a concerted effort to correlate clinical signs and symptoms with kidney lesions and provided a nosological classification of the acute and chronic forms of what was then known merely as Bright’s disease. During his life, he was recognized as the American authority on diseases of the kidney and the City of New York acknowledged his contributions by naming a hospital after him.

Key words: Francis Delafield, Bright’s Disease, Acute tubular necrosis, Acute renal failure, Chronic renal failure, Nephrology history, Histopathology
Francis Delafield, a forgotten contributor to nephrology

Francis Delafield, a forgotten contributor to nephrology, a partial list of the contributors includes William Osler, W.H Welch, E.G. Janeway and Francis Delafield, author of the section “Diseases of the Kidney” (4).

Francis Delafield (Fig. 1) was born on August 3, 1841 in New York. He was the seventh and last child of Edward Delafield and Julia Floyd, granddaughter of General Floyd, a signer of the Declaration of Independence. From his paternal side, Francis inherited a distinguished history of contributions to medical education. His father, Edward Delafield (1794-1875), was an 1816 graduate of the College of Physicians and Surgeons. Like most American graduates of the time, Edward continued his education abroad, mainly in London, where, along with his friend and fellow classmate, John Kearney Rodgers (1793-1851), he was first exposed to ophthalmology at the London Infirmary for Curing Diseases of the Eye (The Royal London Ophthalmic Hospital). Inspired, they returned to New York in 1818, where they founded the famed New York Eye and Ear Infirmary in 1820, the oldest and continuously existing specialty hospital in U.S., still considered one of the premier specialty training centers for diseases of the eye, ear, nose and throat. Subsequently, Edward Delafield became the founder and first president of the American Ophthalmologic Society, and in 1858, president of the College of Physicians and Surgeons, in which position he was instrumental in converting it into the Medical Department of Columbia College (5).

Following the family tradition, Francis decided early on to pursue a career in medicine, graduating from the New York College of Physicians and Surgeons in 1863, during his father’s term as president. He, like most of the elite American medical school graduates, chose to further his medical education in Europe, where he studied in London and Berlin, at a time that microscopic examination of post-mortem material, or cellular pathology, was the investigative approach de rigueur under the influence of Rudolf Virchow (1821-1902), who had returned to Berlin in 1856, as chair of pathology, at the time that Delafield went there.

He returned to New York in 1866 as one of “the best equipped physicians of his day”, certainly in America. Having acquired in Berlin a conviction of the overwhelming importance of microscopic studies in pathological anatomy, he at once began to devote much of his time and attention to work in the “deadhouse” and soon came to be recognized as an authority in pathology. He became curator to Bellevue in 1866, then visiting professor there in 1875, as well as consulting physician to St Luke Hospital and pathologist at Roosevelt Hospital. Also, he became actively involved in the New York Eye and Ear Infirmary, undoubtedly because of its family ties. By 1876, he had become well recognized as an able diagnostician and that year was made adjunct Professor of Pathology and the Practice of Medicine in the College of Physicians and Surgeons. He was made full professor in 1882 and Emeritus professor in the early 1890’s. In 1886, he promoted the organization and became the first president of the Association of American Physicians and Pathologists. Early in his career, he had made the decision that it was best to

**Fig. 1 - Francis Delafield (1841-1915).**
retire from his professional and academic duties at the age of sixty and in 1901 he did just that, although he was still in perfect health. He continued to function as a consultant and in September 1901 was one of the consulting physicians for President McKinley after his assassination attempt at the Pan American Exposition in Buffalo, N.Y. He continued consulting, with marked success, until failing health compelled him to reluctantly become less active, although he never retired completely. He died on July 17, 1915, at the age of 73, in Noroton, Connecticut at the home of his elder sister (3, 6-9).

STATE OF THE ART AT THE CLOSE OF THE 19TH CENTURY

The efforts to localize diseases to anatomical seats (sedes morbi) began in earnest with Morgagni (1682-1771), with the transition of dissection from the domain of the anatomist to that of the clinician. It was this impetus to correlate ante and post-mortem findings that was to make morbid anatomy synonymous with pathology during the second half of the 18th century (10). It is in this tradition that Richard Bright (1789-1858) compiled his Report of Medical Cases (1827), containing his original descriptions of the kidney in dropsical patients with its epoch-making distinction between edema due to the diseases of the heart and the kidney. His subsequent systematic study of uremic patients on the clinical wards of Guy’s Hospital and the correlation of their clinical and biochemical findings with his observations at post-mortem constitute the very basis on which nephrology was to emerge a century later (11). Yet for all the elegance and importance of his studies, Bright failed to use the microscope in examining the kidneys whose gross appearance he described and illustrated so meticulously.

Simple lenses used as spectacles were introduced in the 14th century. Working microscopes, providing high magnification, were introduced in the 17th century, and had begun to revolutionize medicine by the first part of the 19th century. Yet their principal application was confined to the description of normal anatomy (histology), rather than that of diseased tissue (histopathology). The emergence of histopathology, whose impact continues to the present, can be credited to Johannes Muller (1801-1858) and specially to his student Rudolf Virchow, whose concept of cellular pathology was being formulated at about the same time that Richard Bright was making his observations on diseased kidneys (12). To quote Virchow from his historical essay presented on March 30, 1894 at the XI International Medical Congress in Rome: “Since (the Parisian School of Bichat, Laenec and Dupuytren) we have progressed far beyond the aims of this school (described as the School of Organism). The search of the sedes morbi has advanced from the organs to the tissues and from the tissues to the cells” (13).

Following his move from Wurzburg to Berlin in 1856, as chair of pathology, Virchow established the influential Pathological Institute, which began to attract a world-wide influx of students who were to extend the new pathological tradition of applying the microscope to the study of cellular pathology. It is amongst those students that Delafield acquired the skills he was to apply to the study of diseases of the kidney (10).

SCHOLARSHIP

Delafield was a prolific writer, with over 82 journal publications on various subjects spanning a time period of 36 years between 1869 and 1904. Some of his better known textbook publications begin in 1872 with the “Handbook of Post-mortem Examinations and of Morbid Anatomy”, which led to an expanded pathology text in 1885 entitled “Handbook of Pathological Anatomy and Histology”, written and illustrated with his colleague T. Mitchell Prudden (1849-1924). The handbook soon became the leading textbook on the subject and remained the standard text well into the twentieth century, going through sixteen editions (1936) and increasing from 575 to 1116 pages.

Throughout the writings of Delafield, one can detect the evolution and increasing clarity of his concept of the kidney and its diseases, and his laborious attempts to differentiate various forms of Bright’s disease into a systematic description of the diversity of the microscopic pathological lesions and their corresponding clinical syndromes. This lasting and unfulfilled quest for a clearer nosological classification of diseases of the kidney is best enunciated in his presentation on “Acute Bright’s Disease” to the Medical Society of New York, at Albany, on February 8, 1888: “It is now for many years that I have studied patients during life, and kidneys after death; that I have read all the available literature on the subject; that I have written and lectured concerning it; but always with the same feeling of dissatisfaction and want of success.” (14). An obsession, that was to haunt him throughout his life, and one that is essential in interpreting his evolving writings in the context of our current understanding of the functions of the kidney in health and its lesions in disease.

In this regard it is important to note that his studies constitute part of the ongoing pioneering studies of diseases of the kidney worldwide that was to lead to
the coherent scheme of F. Volhard (1872-1950) and T. Fahr (1877-1945), which has come to frame much of the current classification of diseases of the kidney (15). It is also important to remember that hypertension as a clinical entity was not yet fully established and kidney biopsies were not available (16). Moreover, pathology was still based on deductive reasoning and still awaiting the emergence of physiology and chemistry to advance it to the inductive reasoning we have come to associate with it, which was made possible only by the expansion of the basic sciences (10).

As such, much of Delafield’s pathology is descriptive and based on autopsy material, representing rather advanced disease. Nevertheless, his concerted effort to correlate signs and symptoms with kidney lesions and to determine the clinical conditions associated with different kidney lesions is clearly evident in his writings. Considered in this context, it is easier to appreciate the fame and recognition Delafield achieved in his life as the American authority on diseases of the kidney.

**DISEASES OF THE KIDNEY**

Delafield was an immensely popular and effective lecturer, prompting one of his students to compile and publish a series of his lectures in 1881, most notably that on “Diseases of the Kidney”, which gained him special notoriety. Most of the present analysis of his work is based on these lectures, which represent the culmination of the years of study that preceded his chapter on “Diseases of the Kidney” in Pepper’s textbook, his formal attempt to synthesize his observations on Bright’s disease (17, 18).

He begins his presentations with a general description of urine and its constituents, a general discussion of dropsy and albuminuria, and a dissertation on the popular theories of uremia at the time. He asserts that the normal quantity of urine is forty to fifty fluid ounces (~1200 to 1500 mL) per day and defines oliguria (<600 mL/day) as a pathological state of varied etiology, and suppression of the urine as an output of 30-60 mL/day. The normal specific gravity of urine in a healthy person is given as ~1.020 to 1.030, while in chronic nephritis the specific gravity is said to be lower and the urine output higher (>1500 ml/day). For normal urinary solid constituents he gives 500 grams of urea and urates. He emphasizes the importance of examining the urine sediment microscopically for abnormal constituents such as blood cells, casts and crystals. Concerning dropsy, he astutely notes that “the as-

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<th>TABLE I - DELAFIELD’S CLASSIFICATION OF DISEASES OF THE KIDNEY</th>
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<td>1893</td>
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<td><strong>Acute nephritis</strong></td>
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CHF = congestive heart failure; HBP = high blood pressure; TIN = tubulointerstitial nephritis; GN = glomerulonephritis; RPGN = rapidly progressive glomerulonephritis.
sociation of dropsy with kidney disease is of such frequent occurrence that it is often difficult to convince both patients and physicians that Bright’s disease can exist when dropsy is absent”. Concerning albuminuria, he emphasizes the importance of its presence but clearly states that it “may be absent with kidney disease and present without it”, giving examples of normal physiologic states in which albuminuria may be present such as exertion, fever and paroxysmal (orthostatic). He expounds on “simple persistent albuminuria” as daily albuminuria without any other symptoms, but one that is not a “good risk” for insurance companies because “sooner or later disease of the kidneys, of the heart, or of the arteries is apt to develop”. However, it is his classification of Bright’s disease, based on cellular pathology, which is the most provocative and detailed component of his lectures and work (Tab. I). In Pepper’s textbook he prefaces it by describing Bright’s disease as: “a name given to a group of kidney diseases which are characterized by changes in the urine and by certain clinical symptoms. The clinical relationships of these diseases is so marked that it seems probable that they will always be grouped together although anatomically they are quite distinct”. In his lectures he daringly proposes that: “There would perhaps be a certain advantage if we could get rid of it (Bright’s disease) altogether, but the name is so firmly fixed that this is not likely to take place; hence the best way is to accept the term and try to use it in a rational way...not expressing any one anatomical condition of the kidneys...”

The evolution of his attempts to develop a rational classification is shown in the Table. Essentially, he separates Bright’s disease into acute and chronic nephritis. Of the acute nephritides, acute congestion is said to be “a temporary congestion of the blood vessels which may be so severe as to produce exudation of serum and escape of red blood cells”. He attributes it to heart disease but does not elaborate on it and does not mention it in his lectures. Acute degeneration is defined as an acute change in the kidneys characterized by degeneration or death of the epithelial cells of the tubules. This is said to be a later stage of acute congestion and seems to be a clear description of acute tubular necrosis. He provides a much clearer and detailed description of this entity in an article on Acute Bright’s Disease, in which he notes that this disease process can be caused by toxins such as arsenic and mercury, various infectious diseases and extensive injuries’ (14). Acute interstitial nephritis is described as “rare typically associated with cases of scarlatina, diphteria, measles” and marked by “rapid onset, high temperature, rapid loss of flesh, without changes in urine output but urine sediment positive for albumin, casts and cells”. Microscopic examination reveals that “the Malphigian bodies are unchanged, the epithelium of the cortex tubes is flattened, there are large number of leukocytes infiltrated between the tubules, the capillary veins are large and full of leukocytes”. Acute exudative nephritis, described as “an acute inflammation of the kidneys characterized by congestion, exudation of plasma, emigration of white blood cells, diapedesis of red blood cells, to which may be added changes in the renal epithelium and glomeruli”. In the microscopic description he indicates that the tubules are filled with casts and red and white blood cells, but the most significant changes are in the glomerular capillary tufts. This seems to be a reasonable description of acute glomerulonephritis. Acute productive (diffuse) nephritis is characterized as “the most serious and important form of acute nephritis” in which one sees “an acute inflammation of the kidneys characterized by exudation from the blood vessels” with two “unique histological changes different from exudative nephritis as the characteristic stamp of the lesion: a growth of connective tissue in the stroma and a growth of capsule cells of the malphigian bodies”. This seems to be a clear description of acute crescentic glomerulonephritis, whose associated symptoms he describes as “marked cerebral symptoms, high pulse tension, dyspnea, dropsy, oliguria, albuminuria and casts”, the majority of which “terminate unfavorably”. Chronic congestion of the kidneys is described as an entity that appears to be mediated secondary to circulatory or hemodynamic failure rather than a primary kidney disease. In describing this diagnosis he attributes it to “a number of morbid conditions which interfere with the circulation of the blood in the aortic system in such a way that the blood accumulates in the veins and is diminished in the arteries”, whose symptoms (congestive heart failure) predominate the disease. On microscopic examination “some of the glomeruli remain unchanged, but a considerable number are large, their capsules dilated and with thickened walls” and “the small arteries of the cortex are dilated and their walls thickened. The urine is said to be of high specific gravity with little or no albuminuria and a bland sediment”. This is quite suggestive of nephrosclerosis secondary to hypertension, with cardiac symptoms dominating the clinical presentation. Chronic degenerative nephritis described as a chronic disease of the kidney is likely an early stage of end-stage kidneys due to undetermined forms of glomerulonephritis. Chronic productive (diffuse) nephritis with or without exudation is considered as different expressions of the same disease: glomerulonephritis. The exudative form is characterized as showing “growth of capillary tufts in such numbers as they compress the interticular spaces” and “the capsule cells may be changed into connective tissues and the tufts become atrophied”,

783
a feature that is lacking in the form without exudation, in which the dominant features are “new growth of connective tissue in the stroma, permanent changes in the glomeruli, degeneration of the renal epithelium and sometimes changes in the wall of the arteries. On gross pathology, the kidneys of either form are said to be of variable size but as a rule small with thinned cortex with the microscopic anatomy revealing new connective tissues in the cortex, flattened epithelium and small tubules, cystic dilation of the tubules and glomerular atrophy”. Essentially, various stages in the progression to end-stage kidney disease, with the exudative form due to burned out crescentic glomerulonephritis and the non-exudative due to various other forms of glomerulonephritis.

CONCLUSION

The contributions of Francis Delafield, though significant and widely acknowledged in his days, have come to be overlooked and forgotten over the years. He did most of his research at the beginning the era of rapid growth in medicine, when most of the nation’s attention was focused on John Hopkins in the overwhelming shadow of the Osler Era. Actually, in his textbook, Osler makes repeated reference to Delafield, but provides a rather simplistic classification of Bright’s disease into either acute or chronic, without much attention to the various entities described by Delafield or for that matter to that of other investigators in the field. Obviously, that was all to change with the introduction of kidney biopsies in the 1950s and the emergence of nephrology as a discipline in the ensuing decade.

In reconstructing the history of nephrology it is important to recognize the contributions of this forgotten early American nephrologist for his emphasis on the study of the microscopic pathology of kidney disease, his concerted effort at providing a nosological classification of kidney diseases, and his detailed and accurate description of kidney pathology well ahead of his times. Perhaps his memoriam says it best: “nomenclature may change again and again but none of us can forget the basic facts of morbid anatomy and of the association of ante-mortem pictures with post-mortem lesions whose clear delineations we…owe to Francis Delafield” (3).

Address for correspondence:
Garabed Eknoyan, M.D.
Department of Medicine
Baylor College of Medicine
One Baylor Plaza
Houston, Texas
USA
geknoyan@bcm.tmc.edu

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Wikipedia on Francis Delafield, a physician who was the "son of the prominent John Delafield who had emigrated to America from London, England in 1783 carrying the provisional peace treaty between England and The United States." Since Delafield died in July 1915, this photo may have been supplied by Bain for obituaries. New-York tribune., July 18, 1915, Page 9 has an announcement of his death, but uses a different photo chroniclingamerica.loc.gov/lccn/sn83030214/1915-07-18/ed- swanq 7y. More on Delafield at www.ncbi.nlm.nih.gov/pubmed/14733429 Francis Delafield (1841-191... Start studying An American Tragedy. Learn vocabulary, terms and more with flashcards, games and other study tools. Part of an evangelistic family signing hymns on the street. Clyde Griffiths, the main character in this particular novel, first appears as? Fasle. She returns, pregnant, without the man with whom she ran off. Clyde's sister Esta leaves the family to elope and never returns. The Green-Davidson Hotel. What is the name of the first hotel that employs Clyde? Hegglund. Francis Delafield (August 3, 1841 â€“ July 17, 1915) was an American physician, born in New York City. His father, Dr. Edward Delafield, was the son of the prominent John Delafield who had emigrated to America from London, England in 1783 carrying the provisional peace treaty between England and The United States. While his father Edward graduated Yale in 1812, Francis graduated at Yale (1860) and at the College of Physicians and Surgeons, Columbia University (1863), and after further study abroad