FOUNDERS OF THE RENAL PATHOLOGY CLUB (1977)

Conrad Pirani ("instigator")
Jay Bernstein
Peter Burkholder
Ramzi Cotran
Robert Heptinstall
Michael Kashgarian
John Kissane
Kash Mostofi
Gary Stricker

Jacob Churg
Francis Cuppage
David B. Jones*
Richard Kempson
Robert McCluskey
Benjamin Spargo
(F.Silva: Secretary)
CHARTER MEMBERS (1978)

Giuseppe Andres  
Gloria Gallo  
Harrison Latta*  
Gary Hill  
Tito Cavallo  
Robert Lannagan  
C. Craig Tisher  
John Hoyer  
James McAdams  
Ralph McCoy  

Curtis Wilson  
Tatiana Antonovych  
Arthur Cohen  
Morris Karnovsky  
Alfred Michael  
Victor Pollak  
Keith Holly  
Victoriano Pardo  
Seymour Rosen
DAVID JONES  
(1921-2007)

Born: Canton, China, Dec 1, 1921  
(Father was a YMCA Missionary)
Wife: Jean and Three children
Served Active Duty: US Navy
Education: Syracuse Univ. (AB and MD: Cum Laude and Magna Cum Laude)
Internship: US Naval Hospital/Brooklyn
Residency: Syracuse/Neuropath Fellowship/Mayo
Fellow to Professor of Pathology: All at Syracuse (1948-1963)
Uremia
1 - Electrolytes
2 - Water
3 - Blood
DAVID JONES

• Medical Director, Cytotechnology, Syracuse
• Editorial Board, Clinical Nephrology
• AFIP: Consultant in Renal Pathology (1944-1991)
• USCAP:
  – First complete Renal Proffered Session (8) (1967) : First abstract by Dr. Jones (“Acid Mucoproteins of the Glomerulus: An EM Study”) (one also by S.Rosen)
  – Member, Education Co (1978-1982)
  -- Specialty Conference Moderator (1978-1981): First instituted by Kash Mostofi in 1968 (only Surgical Pathology and Pediatric Pathology had one at that time). Dr. Jones-- the second Moderator in history (How many of us got into the USCAP).
    (followed by Drs. G.Gallo, S.Rosen, A. Cohen, C.Jennette, C.Alpers, V. D’Agati, A.Fogo and B.Colvin)
Fifty-Sixth Annual Meeting
of the
INTERNATIONAL ACADEMY
OF PATHOLOGY
(Formerly International Association of Medical Museums founded 1906)

61st
March 12-15, 1967
YEAR

Course:
Pathological Physiology and Anatomy of the Central Nervous System

The Sheraton-Park Hotel
WASHINGTON, D.C.

SCIENTIFIC SESSION
Monday, March 13, 1967
8:30 A.M.

SECTION D  VIRGINIA SUITE
Chairman—DR. JOHN L. SHAPIRO
(Each presentation is limited to 10 minutes)

8:30 “Acid Mucoproteins of the Glomerulus: An Electron Microscopic Study.” DAVID B. JONES — State University of New York, Upstate Medical Center, Syracuse, New York

8:45 “Rapid Development of Chronic Glomerulonephritis in Experimental Scurvy Sickness.” MASAYUKI TAKASUGI, TOBY MORGAN, DOUGLAS WOO, and LYNN OGDEN — Medical College of Georgia, Augusta, Georgia.

9:00 “Malarial Nephropathy in the Rhesus Monkey.” SEYMOUR ROSEN, JESSIE E. HANO, and KEVIN G. BARRY — Walter Reed Army Institute of Research, Washington, D.C.


10:00 “Regeneration of the Nephron Following Hypoxic Injury.” DANIEL NEACOV and FRANCIS E. CUPPAGE — Ohio State University, Columbus, Ohio.

10:15 “Modification of Rejection of Transplanted Kidneys by Treatment of the Donor.” STEPHEN T. IMRIE and JOEL G. BRUNSON — University of Mississippi School of Medicine, Jackson, Mississippi.

10:30 RECESS AND EXHIBITS
11:00 Maude Abbott Lecture — Park Room
11:45 Business Meeting — Park Room
DAVID JONES
Over 100 Major Publications

Glomerular:
- Nomenclature, Definition, and Classification of Renal Disease
- Inflammation/Repair/Nature of scar tissue in glomeruli/mesangium
- Acid mucoproteins/EM/Sticking of leukocytes to endothelium in Acute GN
- Cell/Extracellular morphology of the glomerular stalk
- Correlations Scanning and TEM of Renal Bx and Experimental Disease
- Enzymatic Digestion of the kidney
- Formation/Healing Crescents
- Silver Stains (THE JONES Stain)
- Wegener’s
- Focal GN
- Thrombosis/Toxemia of Pregnancy/Postpartum Malignant Hypertension
- Nephrotic GN (including SEM of MPGN; MCNS; FSGS)
- MPGN: One disease or many?
- Bartter’s
- Use of the Biopsy gun
Chapter 2

The Role of Scanning Electron Microscopy in the Study of Normal and Diseased Glomeruli*

DAVID B. JONES

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FIG. 1. This is an isolated glomerulus from a patient with membranous glomerulonephritis. Note the bean-like globular configuration and the podocytes with many microvilli covering the capillaries. OTO gold-palladium; x360.
Fig. 6. Membranous glomerulonephritis. Note the flat indistinct cell junctions of pedicles on the capillaries (C) and the many spherical blebs and microvilli. These changes are uniformly present. OTO gold-palladium; \( \times 1400 \).

Fig. 7. Minimal change disease. Note the many microvilli on podocyte cell bodies (P) and the indistinct foot processes over the capillary (C). OTO gold-palladium; \( \times 4800 \).
Fig. 8. Membranoproliferative glomerulonephritis, Type I. Note the spherical blebs and the marked variation of foot processes from near normal to severe effacement (arrow). OTO gold-palladium; \( \times2600 \).
Vascular and Tubular Disease:
Nephrosclerosis and the glomeruli
Severe/malignant Hypertension (SEM; TEM; IF)
Experimental ischemic renal arterial necrosis/resolution

Injury/Repair of Proximal Tubular Microvilli/Evidence of Membrane recycling
TEM Studies of Tubules/Interstitium in Glomerular Diseases
Acute Renal Failure/EM: Basolateral surface change
Myeloma/Light Chain Diseases

Urinary Cytology: Acute allograft rejection/renal tubular epithelium
Graft and Transplantation Rejection
Clinical presentation for Renal Biopsy in transplantation
Cyclosporin toxicity

CHAPTER ON THE KIDNEY: In Anderson and Kissane
Ultrastructure of Human Acute Renal Failure

DAVID B. JONES, M.D.

Department of Pathology, State University of New York, Upstate Medical Center, Syracuse, New York

The author studied with light microscopy, scanning electron microscopy, and transmission electron microscopy 19 kidney biopsies from patients with oliguric and nonoliguric acute renal failure, two biopsies from patients with renal failure due to bilateral ureteral obstruction, and 1 biopsy with normal renal tubules. In acute renal failure, there were no intrinsic lesions of glomeruli, but lesions of varying severity were found in the proximal and distal tubules. Proximal tubule changes included diminished, bizarre or absent brush border, often with no or multiple cilia (often more severe in the straight segment of the proximal tubule); luminal surface blebs or bizarre projections; decreased, flattened, or absent basolateral interdigitations simplified cuboidal appearance; bizarre lateral interdigitations; enlarged “contracted” attachment bodies; increased cytosomes, “osmotic” or autophagic, and decreased apical vescules. Distal tubule changes included decreased basolateral interdigitations of the convoluted segment, some decrease in microvilli, increased cytosomes and luminal casts, and enlarged “contracted” attachment bodies. These changes imply severe diminution of luminal and antiluminal surface area which may decrease sodium and chloride flux and, thus, might induce renal cortical vasoconstriction by tubuloglomerular feedback mechanisms. Tubular changes resulting from partial ureteral obstruction closely resembled those of acute renal failure.

Additional key words: cytoskeleton microfilaments, toxic and ischemic nephropathy.

The light microscopic lesions of acute renal failure (ARF) in the human kidney have been well documented (3, 5, 21, 24, 27). Solez, Morn-Maroger, and Smir (27) have particularly well described not only the changes recognized by other authors, but also emphasized both the loss of periodic acid–Schiff (PAS)-positive stained brush border and the difficulty in distinguishing between proximal and distal convoluted tubules.

There have been relatively few studies of the transmission electron microscopic (TEM) lesions of human ARF. Delacourt and Pederson (6, 7) described normal cells alongside of necrotic cells and shedding of brush border in some affected tubule cells. Olsen (25, 26) described a well preserved brush border of proximal tubules but had the impression that infoldings were reduced. He was not sure about the latter finding as he did not know from which location in the nephron the cells came. Dunnill and Jeremie (11) described tubular cells as being simple epithelial cells with few intracytoplasmic organelles and showing degenerative changes or necrosis.

The pathogenesis of ARF has been attributed to several factors including glomerular changes, tubular obstruction, back leakage through necrotic tubules, and renal cortical vasoconstriction (29). There has been some animal experimental data to support each of these mechanisms (6, 9, 10, 13, 18, 28, 30, 34). Of particular interest is the hypothesis of “tubuloglomerular feedback” in which proximal convoluted tubular injury results in decreased sodium, chloride, and water resorption (18, 34).

When the resulting excessive sodium and chloride load reaches the macula densa–juxtamedullary extraglomerular periarteriolar vasoconstriction results and glomerular filtration falls (18, 34). Welling and Welling (35–37) have shown that normal rabbit proximal tubular cells have an highly adapted to the tremendous sodium, chloride, water flux of normal proximal tubular function, showed that the brush border of proximal convoluted tubules increase the apical surface area of the proximal convoluted tubule three times (35). This would provide a large area for passive absorption of sodium. Since the found the basal-lateral cell surfaces were 20 times larger than the surface resting on the basement membrane (39). The large basal-lateral surface is the site of active sodium transport and passive chloride flux (8). Welling et al. (36) used computer-assisted analysis of serial sections of proximal tubules of the rabbit to postulate a complex interdigitating microvilli branching from the basement membrane processes of the proximal tubular cells. Using Dull (12), using collagenase digestion, examined the basolateral surface of rabbit proximal tubules with scanning electron microscope. They confirmed the presence of complex interdigitating microvilli on much of the basal surface of the proximal tubule membrane. Structural changes closely correlate with functional changes; significant brush border and basal infoldings were lost. Experimental toxic and ischemic ARF in is due to loss of proximal tubule brush border microvilli viewed by TEM and scanning electron microscopy (SEM) (8, 10, 39). A biopsy from a child with ARF demonstrated extensive tubular injury with marked brush border loss and moderate tubulus dilation.
Fig. 2. SEM view of a near normal proximal convoluted tubule exhibiting the common apical bleb artifact (arrow) but no rounding brush border microvilli. Specimen was prepared from Figure 1. x6,800.
DAVID JONES: Collaborations

Venkatachalam (Comments from Venk)**
H. Rennke
N.G. Levinsky
USCAP
“And gladly wolde he lerne, and gladly teche”

Geoffrey Chaucer
The Canterbury Tales (1387)
(As told to Dr. Silva by Dr. M. Schwartz)
What we have loved, Others will love, and we will teach them how.

The Prelude Wordsworth
As they say....

“Happy is the man (and woman) that findeth wisdom” (Proverbs)

“There were giants in the earth in those days” (Genesis).
A MOMENT OF SILENCE IN MEMORY OF DR. DAVID JONES