

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

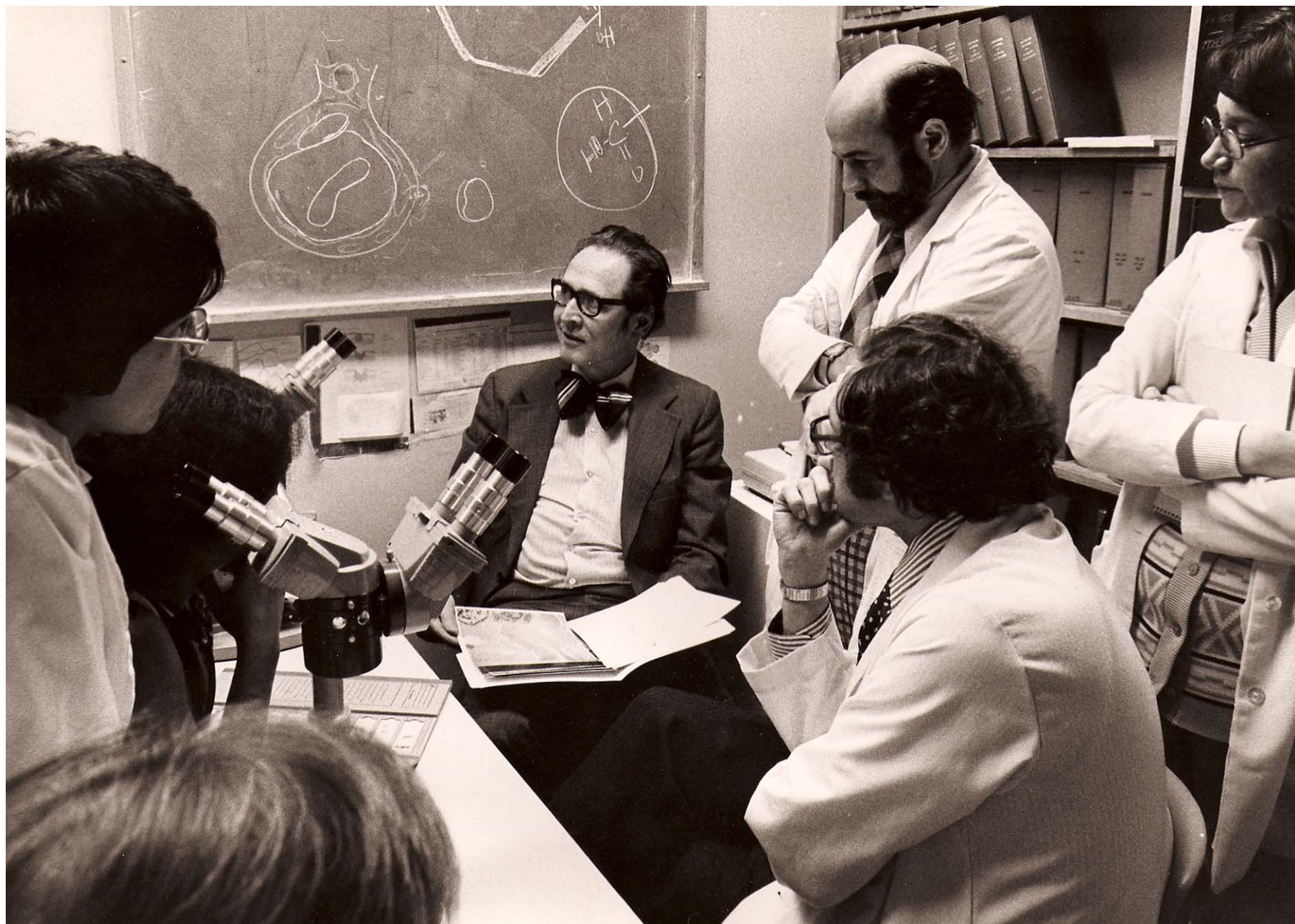
Alfred Michael

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

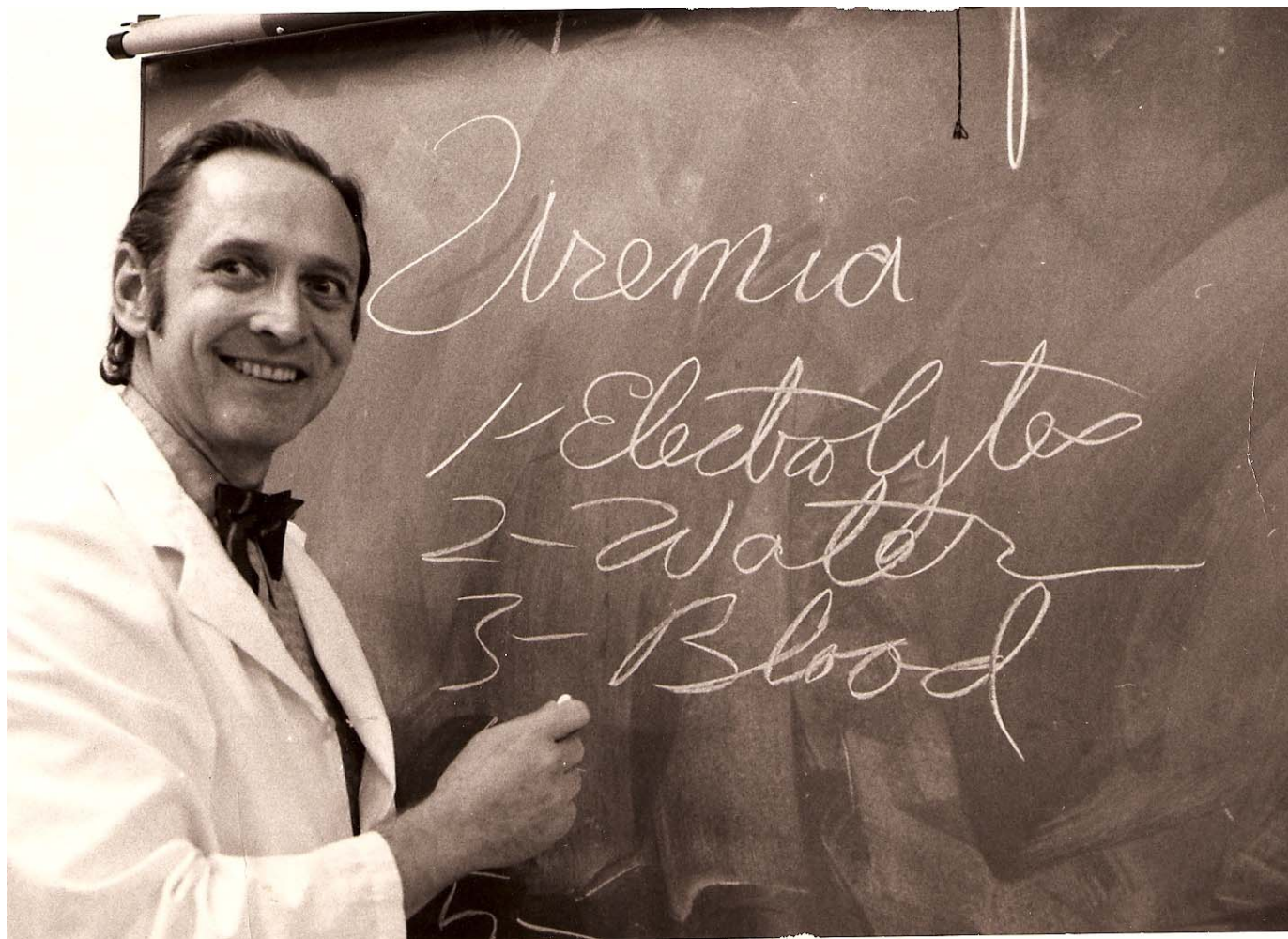
Served Active Duty: US Navy

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)



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)

Mar. 1-7, 1968, Drake, Chicago

October - ^{PROGRAM} College & ASCP meeting



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 Serum 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.
- 9:15 "Ultrastructure of Renal Proximal Tubules of the Rhesus Monkey: A Comparison with the Human." C. CRAIG TISHER and SEYMOUR ROSEN — Walter Reed Army Institute of Research, Washington, D. C.
- 9:30 "The Ultrastructural Lesions in the Kidney of a Patient who Survived 14 Days of Complete Anuria due to Acute Fatty Liver of Pregnancy." SERGIO A. BENCOSME, G. F. KIPKIE, L. S. VALBERG, S. P. HANDA, P. A. F. MORRIN, and J. C. WYLLIE — Queen's University and Kingston General Hospital, Kingston, Ontario, Canada.
- 9:45 "Sequential Histochemical Features in Experimental Osmotic Nephrosis." A. J. MONSERRAT, C. GOTELLI, and R. GARAY — Ila Catedra de Patologia, Buenos Aires, Argentina.
- 10:00 "Regeneration of the Nephron Following Hypoxic Injury." DANIEL NEAGOY 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

PATHOLOGY
H&G

A. CALATAYUD
1877/66

Michael Reese Hospital

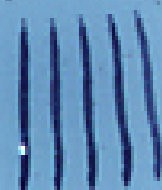


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PATHOLOGY
MASSON'S

A. CALATAYUD
1877/66

Michael Reese Hospital

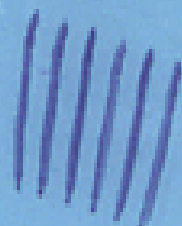


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PATHOLOGY
H&G

A. CALATAYUD
1877/66

Michael Reese Hospital



1877/66

PATHOLOGY
AB - PAS

A. CALATAYUD
1877/66

Michael Reese Hospital

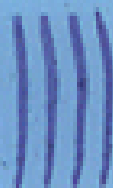


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A. CALATAYUD
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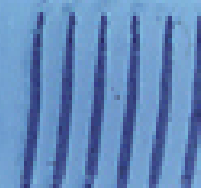


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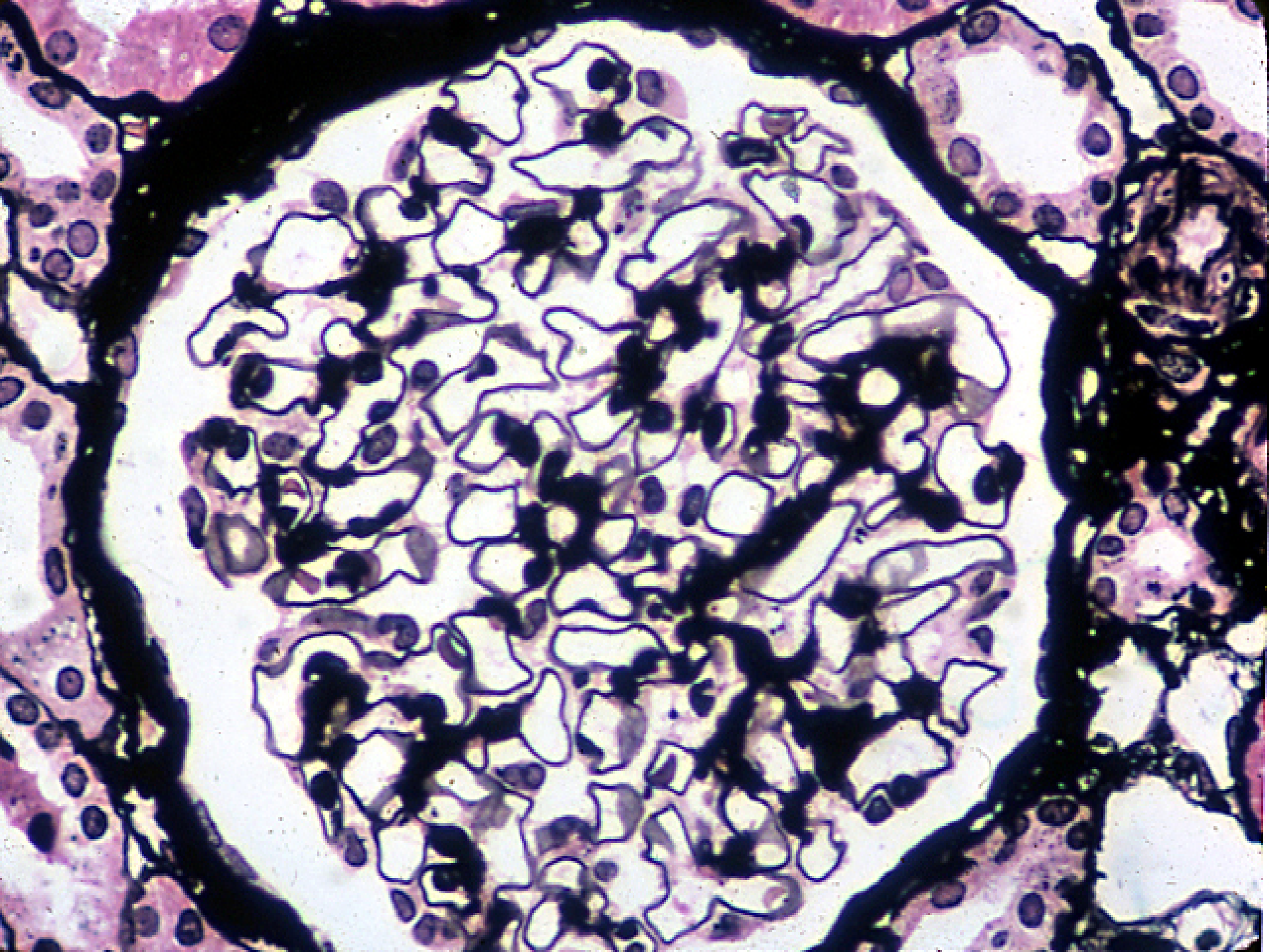
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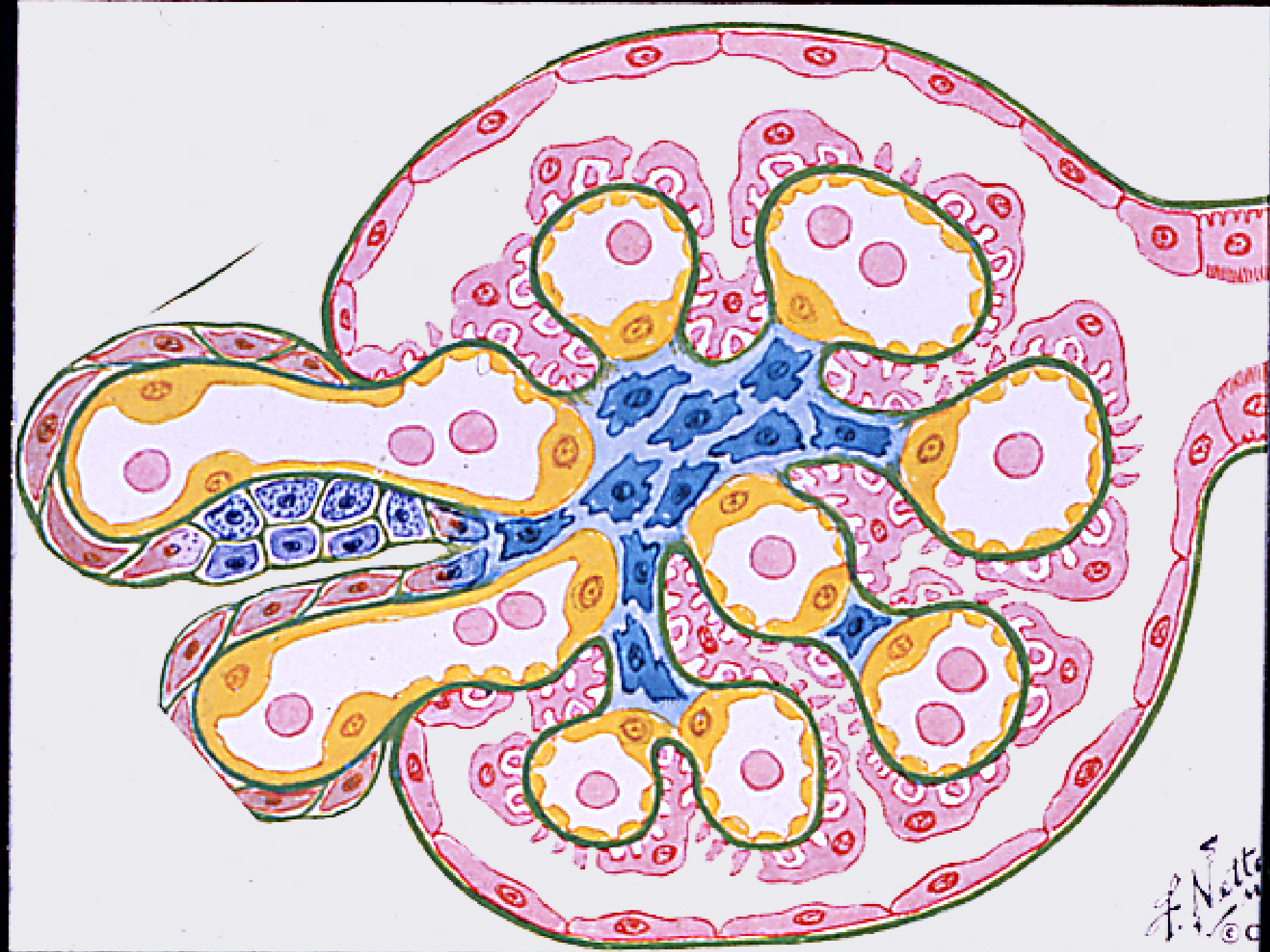
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KIDNEY DISEASE: Present Status

by 16 authors

EDITED BY JACOB CHURG, M.D.

Department of Pathology
Mount Sinai School of Medicine of the City University of New York
New York, New York

BENJAMIN H. SPARGO, M.D.

Professor and Associate Chairman
Department of Pathology
The University of Chicago
Chicago, Illinois

F. K. MOSTOFI, M.D.

Genitourinary Pathology Branch
Armed Forces Institute of Pathology
Washington, D.C.

AND MURRAY R. ABELL, M.D., Ph.D.

American Board of Pathology
Tampa, Florida



THE WILLIAMS & WILKINS COMPANY

Chapter 2

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

DAVID B. JONES

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Kidney Disease: Present Status

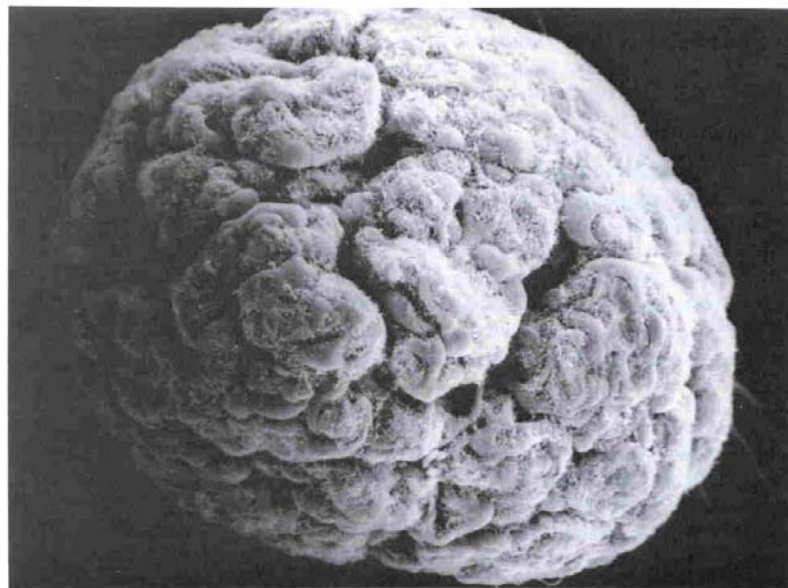


FIG. 1. This is an isolated glomerulus from a patient with membranous glomerulonephritis. Note the brain-like globular configuration and the podocytes with many microvilli covering the capillaries. OTO gold-palladium; $\times 360$.

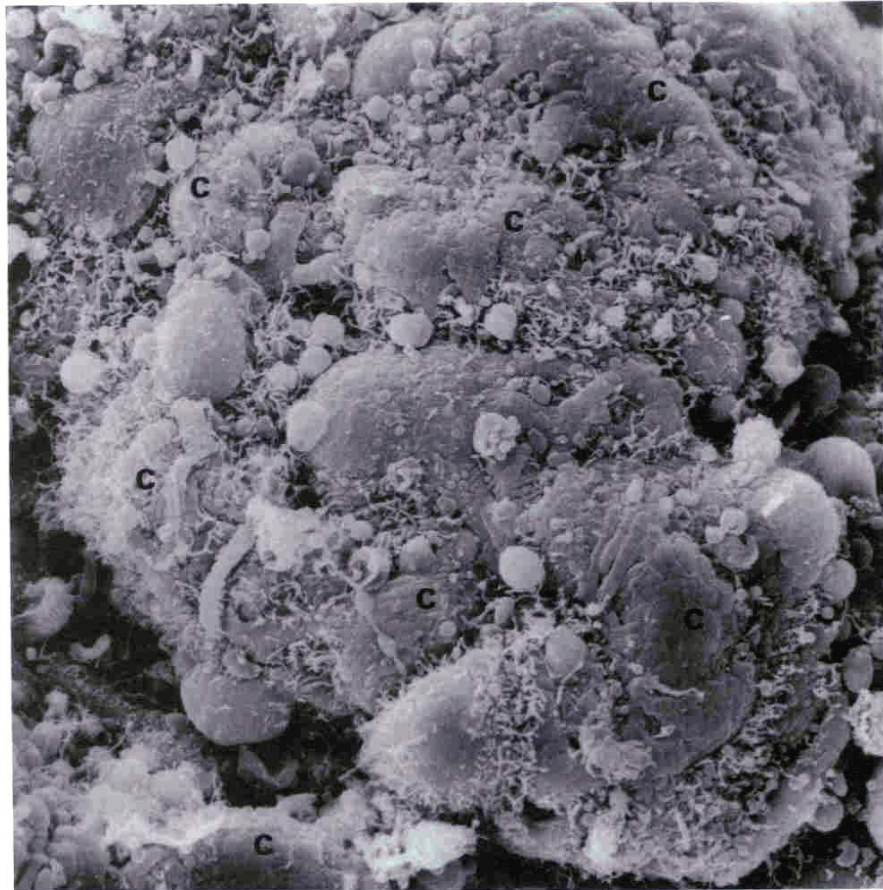


FIG. 6. Membranous glomerulonephritis. Note the flat indistinct cell junctions of pedicles on the capillaries (C) and the many spherical blebs and microvilli. These changes are uniformly present. OTO gold-palladium; $\times 1400$.

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Kidney Disease: Present Status

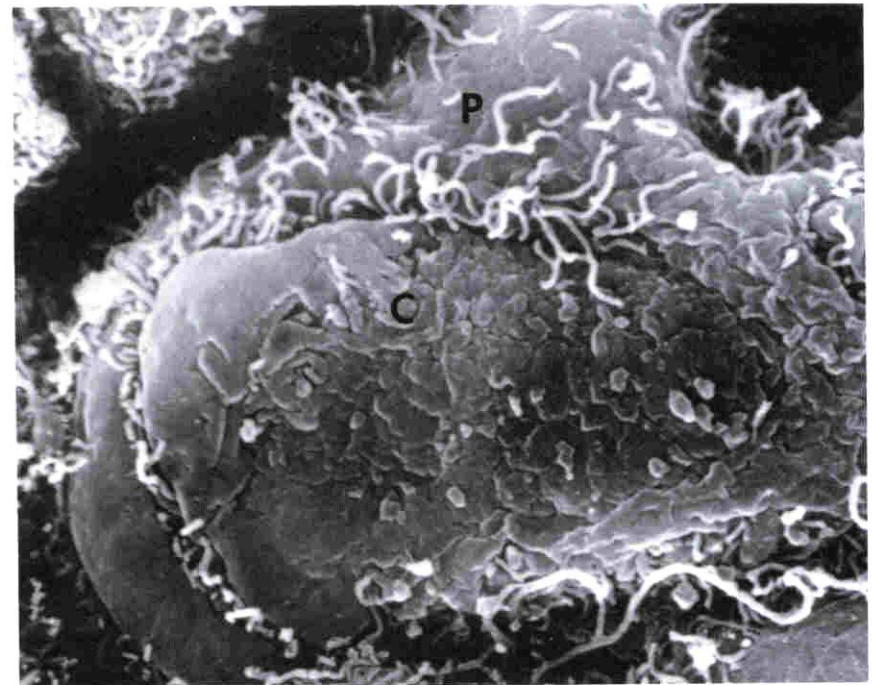


FIG. 7. Minimal change disease. Note the many microvilli on podocyte cell bodies (P) and the indistinct foot processes over the capillary (C). OTO gold-palladium; $\times 4800$.

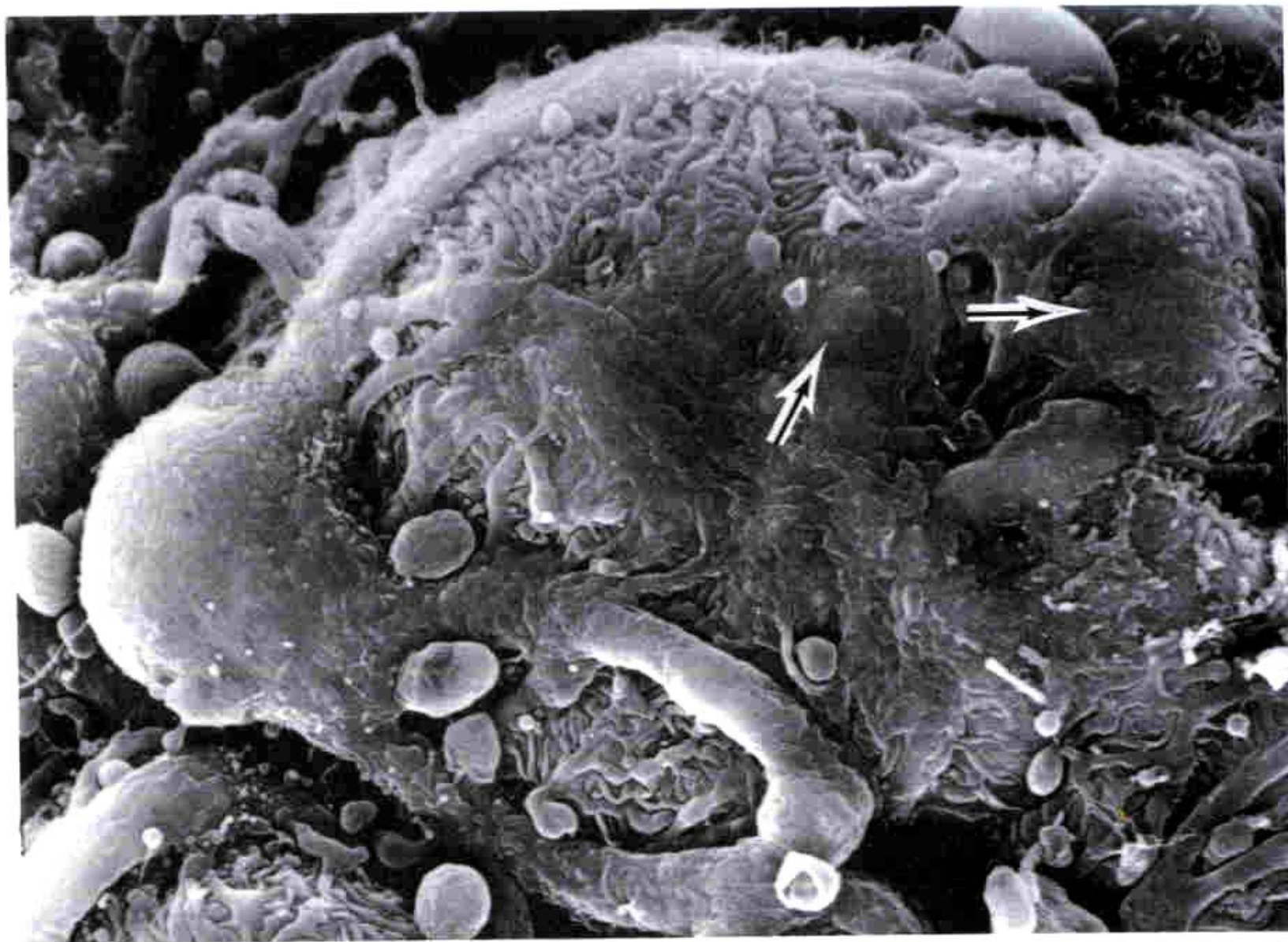


FIG. 8. Membranoproliferative glomerulonephritis, Type I. Note the spherical blebs and the marked variation of foot processes from near normal to severe effacement (*arrow*). OTO gold-palladium; $\times 2600$.

DAVID JONES

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

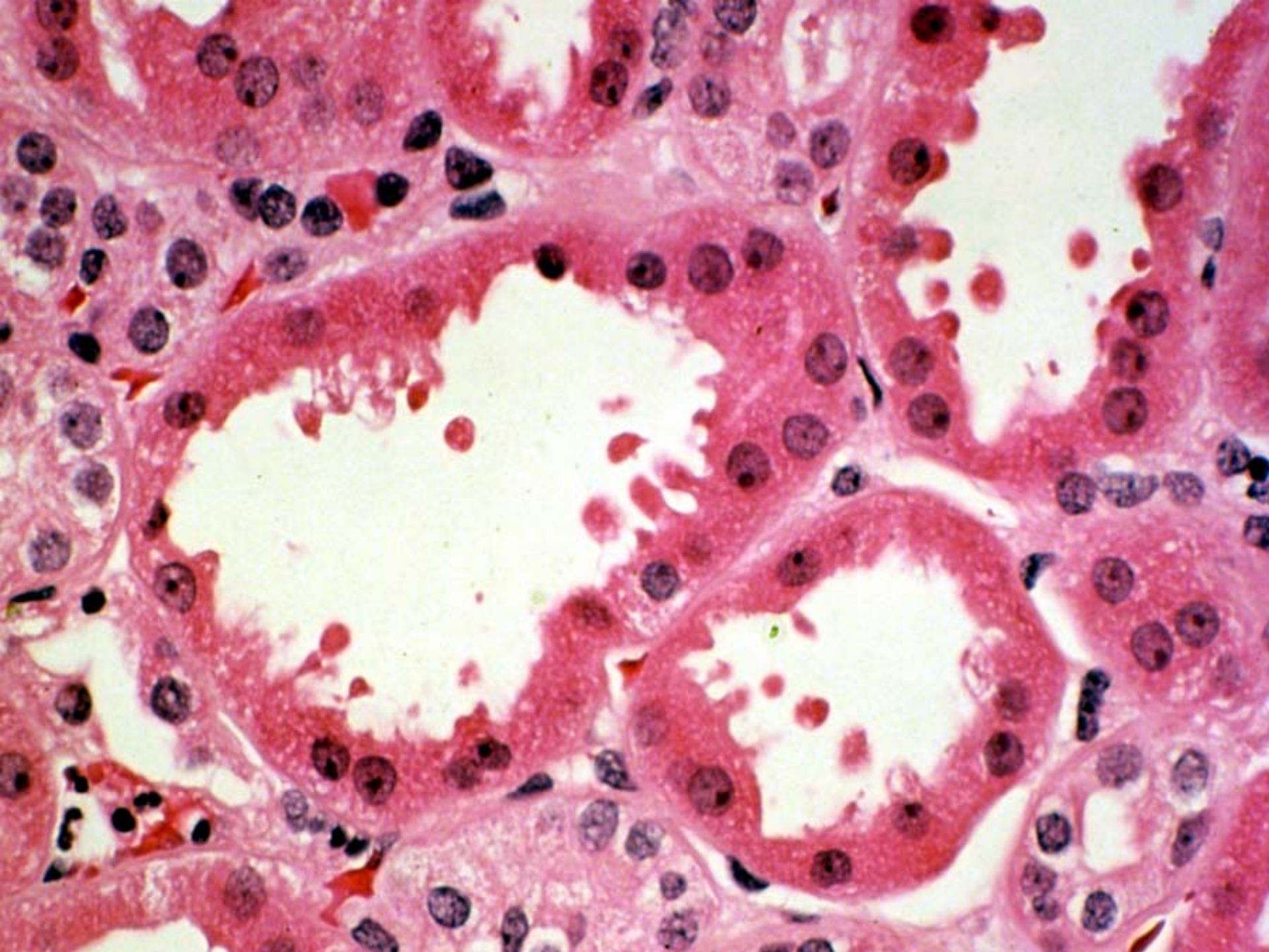
Urinary Cytology: Acute allograft rejection/renal tubular epithelium

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 14 biopsies with near normal 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 tubular 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 basal-lateral interdigitations simplified cuboidal appearance; bizarre lateral interdigitations; enlarged "contracted" attachment bodies; increased cytosomes, "osmotic" or autophagic; and decreased apical vacuoles. Distal tubule changes included decreased basal-lateral 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, Morel-Maroger, and Sraer (27) have particularly well described not only the changes recognized by other authors but emphasized both the loss of periodic acid-Schiff (PAS)-positively 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. Dalgaard 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 basal 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 Jerrome (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 (2, 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-juxtaglomerular complex, arteriolar vasoconstriction results and glomerular filtration falls (18, 34). Welling and Welling (35-37) have shown that normal rabbit proximal tubular cells have a structure highly adapted to the tremendous sodium, chloride, and water flux of normal proximal tubular function. They showed that the brush border of proximal convoluted tubules increase the apical surface area of the proximal convoluted tubule 36 times (35). This would provide a large area for passive absorption of sodium. Also, they found the basal-lateral cell surfaces were 20 times the surface resting on the basement membrane (35). The large basal-lateral surface is the site of active sodium transport and passive chloride flux (8). Welling and Welling (36) used computer-assisted analysis of surface micrographs of proximal tubules of the rabbit to postulate a complex interdigitating microvilli branching from the apical processes of the proximal tubular cells. Evan, Hay, and Dail (12), using collagenase digestion, examined the basal-lateral surface of rabbit proximal tubules with scanning electron microscope. They confirmed the presence of complex interdigitating microvilli comprising much of the basal surface of the proximal tubular cells. Structural changes closely correlate with functional changes, significant brush border and basal interdigitating defects might be expected in ARF.

Experimental toxic and ischemic ARF in the rat results in loss of proximal tubule brush border microvilli as viewed by TEM and scanning electron microscope (SEM) (9, 10, 39). A biopsy from a child with de-

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FIG. 2. SEM view of a near normal proximal convoluted tubule exhibiting the common apical bleb artifact (arrow) but retaining surrounding brush border microvilli. Specimen was prepared as in Figure 1. $\times 6,800$.

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