英文影印版

Owen □ Pereira □ Sayegh

透析与移植
Dialysis and Transplantation

A COMPANION TO

Brenner & Rector's THE KIDNEY

科学出版社 ☆ Harcourt Asia ☆ W.B.Saunders

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《布伦纳-雷克托肾脏病学》配套用书

Dialysis and Transplantation

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To my wife, children, and teachers
William F. Owen, Jr.

To my wife, Sunita,
and children, Natasha and Nikhil,
for their constant support and understanding

To my mentors,
Drs. Kirpal Chugh, Nicolaos Madias,
and Andrew Levey
Brian J. G. Pereira

To my two brothers,
Dr. Sard H. Sayegh and Abdul Hafiz H. Sayegh,
for their continuous support of my career
through the years
Mohamed H. Sayegh

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PREFACE

For the past two decades, *The Kidney*, by Drs. Barry M. Brenner and Floyd Rector, has been the central resource for authoritative and current information in the field of nephrology. However, the continuing expansion in the understanding of the pathophysiology and management of renal disease, dialysis, and transplantation and rapid advances in technology led to the need for more focused accompaniments to *The Kidney*. Consequently, Dr. Brenner planned on two additional textbooks—a concise and practical text on the treatment of renal disease and hypertension and a comprehensive textbook on dialysis and renal transplantation. The first such venture was *Therapy in Nephrology and Hypertension*, edited by Drs. Hugh R. Brady and Christopher Wilcox. Our textbook, *Dialysis and Transplantation*, is the second.

The intent of our venture was to provide readers with a seamless flow of information regarding the management of the patient with end-stage renal disease (ESRD). In pursuit of this goal, we sought to provide an overview of the principles of management of the patient with ESRD as well as a more focused examination of the physiologic principles and clinical application of the different modes of renal replacement therapy. We undertook this venture with the understanding that the treatment of patients with ESRD is complex and the field is rapidly changing. The past decade has witnessed new frontiers in the understanding of the physiologic basis of dialysis and its complications and of the immunologic basis of allograft tolerance and rejection. Advances in pharmaceutical technology and biotechnology have brought new and effective hormones and immunosuppressive agents into clinical use. In addition, the evolution of dialysis technology continues at a rapid pace. Finally, the continuing efforts of professional societies to improve the quality of patient care have resulted in the development of evidence-based clinical practice guidelines and clinical performance measures. These developments have mandated a new look at the management of the patient with ESRD. Consequently, we invited distinguished scientists and educators in the fields of hemodialysis, peritoneal dialysis, critical care nephrology, and transplantation to provide an in-depth review—from the laboratory to the clinic. Each author was challenged to discuss the fundamental concepts behind the management of the patient with ESRD, to provide a comprehensive critique of clinical trials, and to present rational recommendations for clinical treatment.

Our strategy was to cover the most clinically relevant issues in dialysis and transplantation and to classify them under six broad sections—principles and practice of hemodialysis, principles and practice of peritoneal dialysis, long-term care of the dialysis patient, renal replacement therapy in the critically ill, ethical issues, and kidney transplantation. Each section was overseen by one of the editors and included a brief overview of fundamentals of each modality of renal replacement therapy, followed by topics that were critically important for the optimal management of the ESRD patient. Each chapter is self-contained and provides the reader with a thorough review of the subject along with a complete list of key references. Diagnostic and treatment algorithms have been used whenever possible. With an eye on the future, our contributors were encouraged to identify major unanswered questions, to suggest future clinical trials, and to highlight promising experimental strategies. We have applied a strong editorial policy to ensure that chapters remain balanced and that they conform to these principles.

In summary, we have used the parent textbook, Brenner and Rector's *The Kidney*, and our sister textbook, Brady and Wilcox's *Therapy in Nephrology and Hypertension*, as the springboard for a bench-to-bedside review of the management of patients with ESRD. Readers are encouraged to refer to the previous two textbooks in this family for a detailed

discussion of other issues in nephrology. We intend to maintain this textbook as a work in progress and to update it periodically as the relentless advances in the field of dialysis and transplantation mandate. During the coming years, we welcome comments, critiques, and suggestions from our readers as we strive to deliver a comprehensive and contemporary textbook on dialysis and transplantation.

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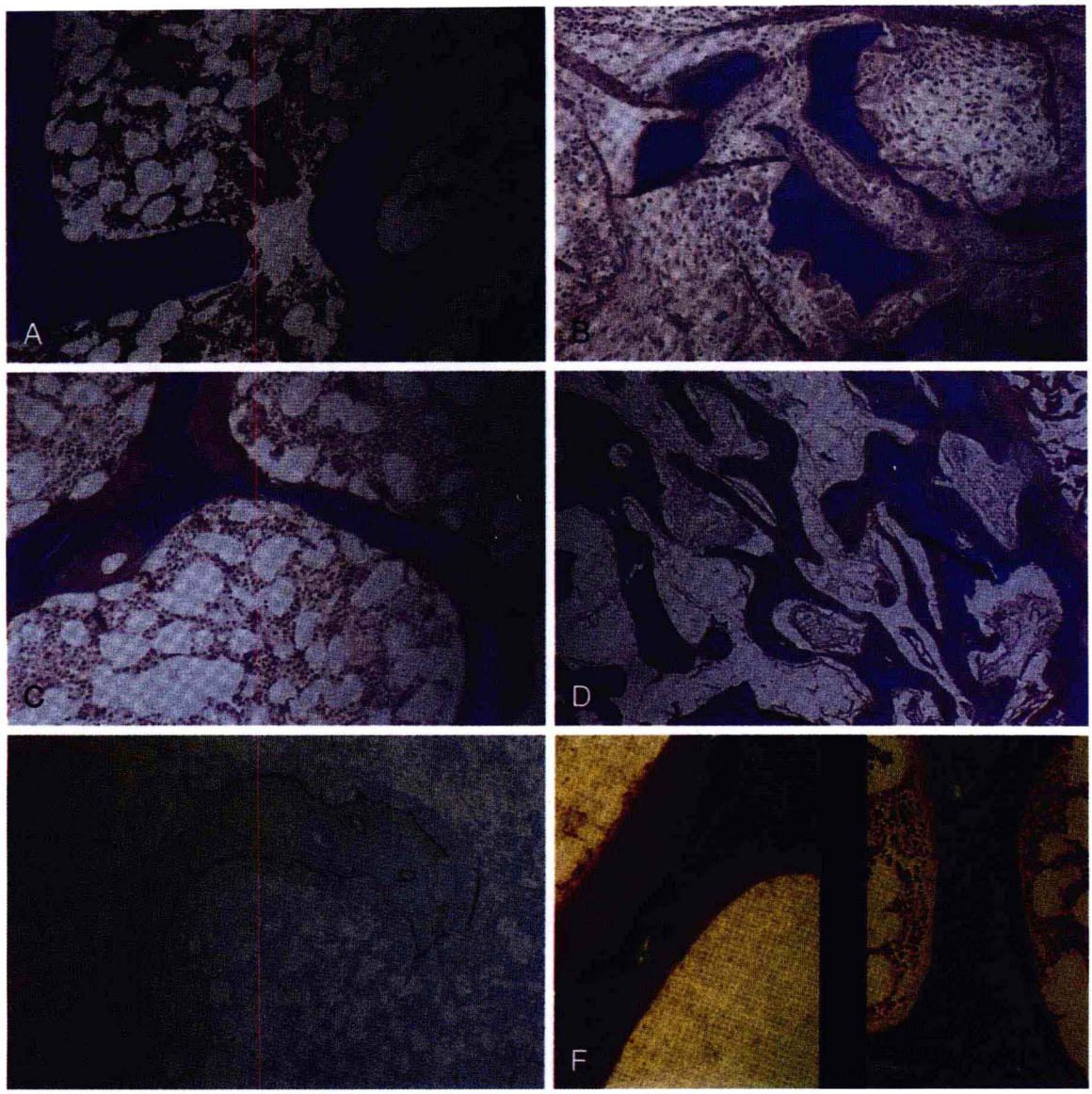

FIGURE 14–11. Color photomicrographs showing representative features of renal osteodystrophy. *A,* Normal trabecular bone. Mineralized bone matrix is stained blue; there is little unmineralized osteoid. (Undecalcified, modified Masson stain; original magnification × 100.) *B,* Osteitis fibrosa in renal failure. There is an excess of unmineralized osteoid, which is stained red and lined by osteoblasts, and numerous multinucleate osteoclasts are present. Note that marrow fibrosis is evident. (Undecalcified, modified Masson stain; original magnification × 100.) *C,* Osteomalacia with wide osteoid seams (red-staining material) in a patient with renal failure. (Undecalcified, modified Masson stain; original magnification × 100.) *D,* Low-power view of cortical and trabecular bone in a patient with long-standing renal failure and osteosclerosis. There is a loss of distinction between cortical and trabecular bone and wide osteoid seams are present *(red material).* The periosteum can be seen in the upper right corner. (Undecalcified, modified Masson stain; original magnification × 40.) *E,* Low-power view of trabecular bone stained for Al. The Al stains as a bright red band at the junction between mineralized bone (the pale blue in the center) and wide osteoid seams (paler area surrounding the bone). In this patient with Al-related osteomalacia, nearly all the forming surface is positive for Al. (Undecalcified, modified aluminum stain according to Maloney et al.) *F,* Higher-power views of two separate bone biopsy specimens from patient on dialysis who had Al-related osteomalacia before *(left)* and after *(right)* chelation therapy with weekly infusions of deferoxamine. Before therapy, there are widened osteoid seams *(red)* surrounding mineralized bone *(blue).* After therapy, there is a marked reduction in the osteoid thickness, and there is the appearance of peritrabecular fibrosis. Normal marrow elements are seen to the right of the trabeculum. (Undecalcified Goldner stain; original magnification × 160.) Except for the area shown in *B,* these figures show findings similar to those labeled in black and white in Figure 15–10. (*A–D,* and *F,* Courtesy of S. L. Teitelbaum. *E,* Courtesy of D. J. Sherrard.) (From Llach F, Bover J: Renal osteodystrophy. *In* Brenner BM [ed]: Brenner & Rector's The Kidney, 5th ed. Philadelphia, WB Saunders, 1996, pp 2187–2274.)

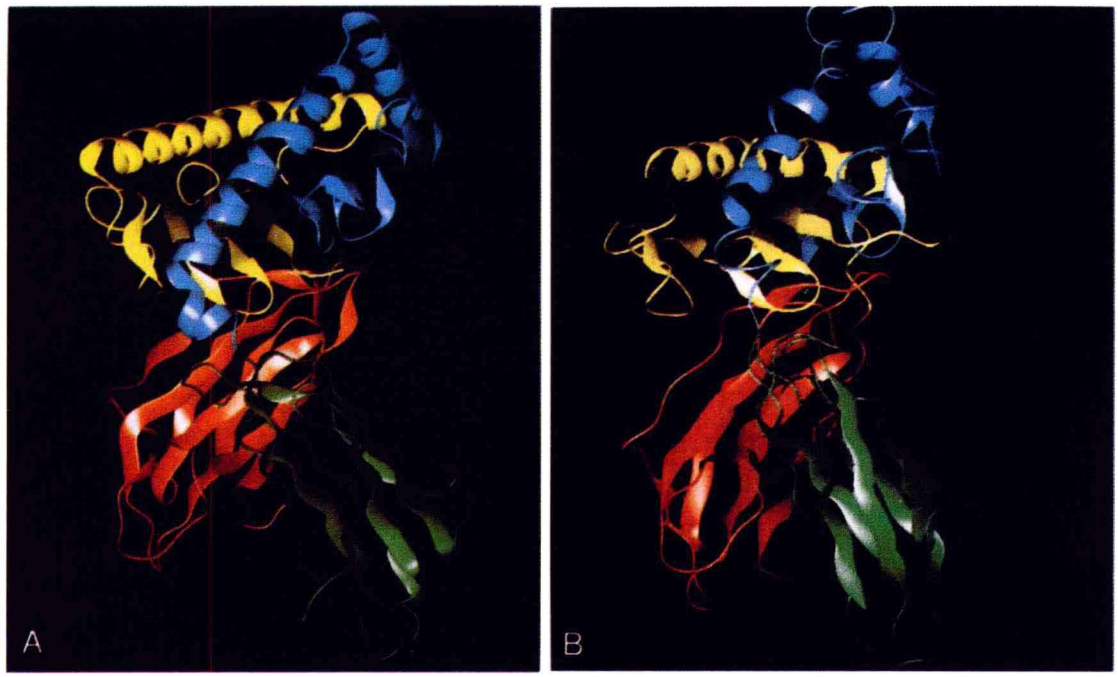

FIGURE 25–2. Computer model of HLA *(A)* class I (HLA-B27) and *(B)* class II (HLA-DR1) structures. The peptide-binding region, made up of two α helices supported by a floor of β strands, is at the top of both views. For HLA-B27, α_1 domain is yellow, α_2 domain is blue, α_3 domain is green, and β_2 microglobulin is red. For HLA-DR1, the α_1 domain is yellow, α_2 domain is red, β_1 domain is blue, and β_2 domain is green. The colors are the same for homologous domains in the two proteins. (From Germain R: MHC-dependent antigen processing and peptide presentation: Providing ligands for T lymphocyte activation. Cell 1994; 76:288.)

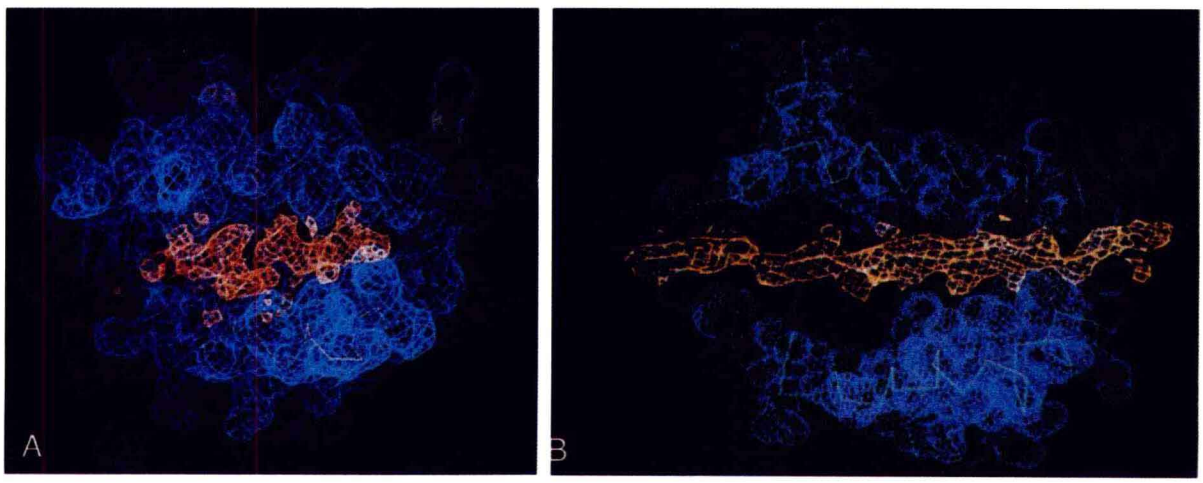

FIGURE 25–3. Peptide binding to HLA class I (HLA-A2) *(A)* and class II (HLA-DR1) *(B)*. The view is looking down on the molecule as a T lymphocyte might "see" it. The two α helices forming the rim of the peptide binding site are *blue*, and electron densities corresponding to bound peptides are shown in *red*. (*A* from Bjorkman PJ, Saper MA, Samraoui B, et al: Structure of the human class I histocompatibility antigen, HLA-A2. Nature 1987; 329:506; *B* from Brown JH, Jardetzky TS, Gorga JC, et al: Three-dimensional structure of the human class II histocompatibility antigen HLA-DR1. Nature 1994; 364:35.)

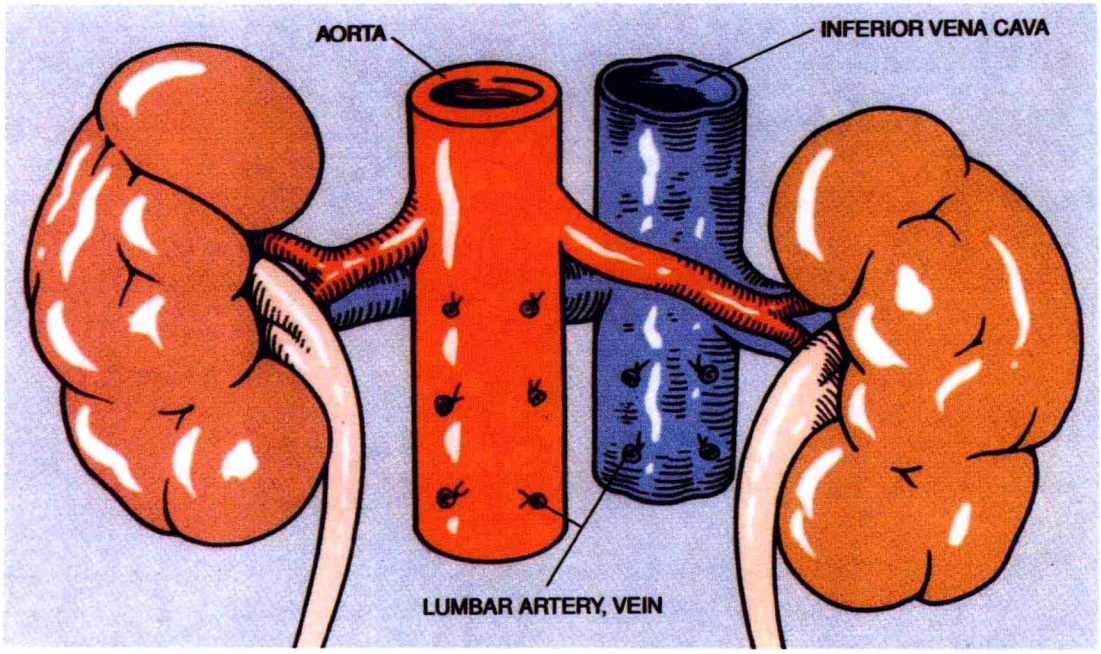

FIGURE 28–8. Pediatric en bloc kidneys. (From Shapiro R: Renal transplantation. *In* Starzl TE [ed]: Atlas of Organ Transplantation. Philadelphia, JB Lippincott, 1992, p 4.11.)