There have been several important and interesting advances in renal parenchymal diseases during the last decade; however, perhaps the most clinically relevant is the paradigm shift in glomerulonephritis associated with infection. The frequency of methicillin-resistant *Staphylococcus aureus* (MRSA) infections is increasing both in hospital-associated and in community settings in the United States and worldwide. Infection due to *S. aureus* imposes a high and increasing burden on healthcare resources. A growing concern is the emergence of MRSA infections in patients with no apparent risk factors. Classic postinfectious/poststreptococcal glomerulonephritis is now rarely seen in Western countries, and most cases of infection-associated glomerulonephritis are secondary to *S. aureus* infections, affecting predominantly the elderly with underlying comorbidities, primarily diabetes but, with increasing frequency, also younger people with no predisposing factors. The differential diagnosis of infection-associated glomerulonephritis and immune-mediated glomerulonephritis not related to infection can be difficult. Infection-associated glomerulonephritis may mimic IgA nephropathy, Henoch–Schönlein purpura (IgA vasculitis), C3 glomerulonephritis, proliferative immune complex glomerulonephritis of autoimmune etiology or even pauci-immune crescentic glomerulonephritis. These forms of glomerulonephritis are treated with immunosuppressive medications. Immunosuppressing patients with active infection-associated glomerulonephritis can have serious consequences. In addition to glomerulonephritis, bacterial infections can cause a wide spectrum of kidney diseases involving the tubulointerstitium and vasculature. Pyelonephritis appears to be an easy diagnosis; however, it is not always so, particularly not in immunosuppressed renal allograft recipients. Bacterial infections can also lead to vascular diseases; the most well known of these are thrombotic microangiopathies, such as the hemolytic uremic syndrome associated with Shiga toxin-producing *E. coli* infection.

This textbook is designed to present a comprehensive and the state-of-the-art but practical approach to the diagnosis and management of bacterial infection-associated renal disease. The chapters address the different types of glomerular tubulointerstitial and vascular diseases, associated with bacterial infections, describe diagnostic pitfalls, provide differential diagnosis and discuss treatment and management. Easy-to-follow diagnostic algorithms are included for practical usefulness. The chapters contain a large number of
color microphotographs, illustrations and each chapter refers to the most important up-to-date literature in the area. All chapters were written by experts in the field and include the most up-to-date clinical and scientific information at the time of the writing.

Infection-associated renal diseases are addressed in large textbooks on kidney diseases, frequently hidden in chapters discussing various forms of glomerulonephritis, interstitial nephritis or vascular disease. This book intends to be a comprehensive but user-friendly resource on renal complications of bacterial infection, which is becoming increasingly relevant now in the era of staphylococcus epidemic and emerging new resistant bacterial strains. We hope this textbook will be an important resource for nephrologists, general internists, infectious disease specialists, pathologists, and urologists. Transplant surgeons may find the chapter on transplant pyelonephritis useful.

We would like to thank our nephrologist colleagues for their input. Their dedicated interaction with us taught us more about infection-associated renal diseases than any pathology text we read. Several of them are authors of chapters in this book. Many infection-associated renal diseases, particularly interstitial diseases, such as pyelonephritis, can be diagnosed without involvement of the pathologist but the pathologist plays a crucial role in the correct diagnosis of infection-associated glomerular diseases. Still, we cannot emphasize enough that the pathologist alone is usually “lost” in the absence of close interaction with the nephrologist. Correct diagnosis of an infection-associated renal disease/glomerulonephritis (just like other forms of renal parenchymal diseases) is only possible if the pathologist and the nephrologist discuss the case in detail, considering every possible differential diagnosis, preferably above the microscope. We are particularly indebted to Drs. Lee Hebert and Brad Rovin, who for the last one and a half decades, since we have been at The Ohio State University, were our main mentors in nephrology issues. They were always available for advice in making clinicopathologic correlations in the interpretation of renal biopsies, even if they were not involved in the care of patients. Our renal biopsy reports frequently reflect their input.

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Columbus, OH, USA

Anjali A. Satoskar
Tibor Nadasdy
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