Preface

The kidney is one of the most complex organs. Once it fails, it can no longer carry out the complicated tasks performed by the normal kidney. The ensuing disorders of calcium (Ca) and phosphate (P) metabolism are common and difficult problems in chronic kidney disease (CKD). This book is intended for medical students, house officers, and general nephrologists. As such it tries to address issues involved in Ca and P metabolism in a concise and yet comprehensive way.

Normal Ca and P metabolism as well as problems frequently encountered in patients with CKD are discussed in the first two chapters. Unfortunately, information on normal Ca metabolism in persons older than 35 years of age is not available; therefore, many problems of management of Ca in older age groups of CKD patients could not be addressed in this chapter. The third chapter discusses secondary hyperparathyroidism. In 1966 Briker suggested that calcium is the main factor responsible for secondary hyperparathyroidism. Thereafter, calcium was used as a main drug to suppress parathyroid hormone (PTH). Recent studies have indicated that phosphate is the main factor in regulating PTH in chronic renal diseases. Chapter 3 addresses treatment of secondary hyperparathyroidism with calcitriol and its derivatives. Although use of these products is controversial, one should realize that they are effective in suppressing PTH. However, these products will increase Ca and P absorption in end-stage renal disease patients who do not have a route for excreting Ca and P. Further, plasma Ca and P do not reflect total Ca and P in the body. Uremic toxins are discussed in Chapter 4. A large number of toxins can affect Ca and P as well as calcitriol (vitamin D end-product) metabolism. Only a few toxins have been determined to affect Ca, P, and calcitriol metabolism. Many other toxins need to be studied for their effect on Ca, P, and calcitriol metabolism. Abnormal function of calcitriol in patients with renal failure has been discussed to some extent in the past. The toxins may exert effects on other hormones, for example, thyroid, estrogen, and other related hormones. Current knowledge of the metabolism of calcitriol and its action in CKD is discussed in Chapter 5. Renal osteodystrophy is discussed in Chapter 6. Bone diseases are a common incidence in patients with renal failure, and the chapter

discusses normal bone physiology and abnormal bone disorder in renal failure patients. Nephrolithiasis is discussed in Chapter 7. The human kidney often forms kidney stones, although this issue is not entirely caused by abnormal kidney function. Abnormal Ca and P metabolism could be one of the main factors.

Express deep appreciation to all of the authors, who contributed to this book in a short time and who are experts in their own fields. My colleague and friend Eric Young especially helped me enormously in editing this book, which hope I will help readers to manage disorders of Ca and P metabolism. I dedicate this book to my deceased parents: Yen and Jane Hsu, who supported me through medical school and sent me to the United States to pursue a career in medicine. What I have and have accomplished today, I owe in large measure to them.

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