Preface

Fluid, electrolyte, and acid–base disorders are central to the day-to-day practice of almost all areas of patient-centered medicine, both medical and surgical. Despite the steep learning curve for trainees, the underlying pathophysiology and/or management is often viewed as "settled," with the perception that there is little in this field that is new. However, there have been significant recent developments in all aspects of these important disorders. This book encompasses these new findings in comprehensive reviews of both pathophysiology and clinical management, meant for both the nephrologist and the nonspecialist physician or medical trainee.

Virtually every subject in this textbook has witnessed major developments in the last decade. New pathophysiology includes the molecular identification of "pendrin" (SLC26A4) as the apical Cl^-/HCO_3^- exchanger in β[beta]-intercalated cells [1, 2]; this transporter functions in distal chloride and bicarbonate transport, with evolving roles in the pathophysiology of hypertension and metabolic alkalosis. A host of previously uncharacterized genetic tubular disorders have recently yielded to molecular genetics, with major impact of this gene identification on the understanding of renal physiology and pathophysiology. In particular, the identification in 2001 [3] of causative mutations in the WNK1 (With No K/Lysine) and WNK4 kinases in familial hypertension with hyperkalemia (Gordon's syndrome) led to a still-evolving cascade of insight into the role of these and associated signaling proteins in the coordination of aldosterone-dependent and aldosterone-independent regulation of distal potassium, sodium, and chloride transport [4]. Characterization of multiple genes for familial hypomagnesemia led to the identification of novel magnesium transport pathways [5] and to the identification of cell-associated epidermal growth factor as a major paracrine regulator of distal tubular magnesium transport [6]. Finally, characterization of FGF23 (fibroblast growth factor-23) as the disease gene for autosomal dominant hypophosphatemic rickets [7] uncovered a major new regulatory hormone in calcium and phosphate balance [8, 9].

At the clinical level, the spectrum of the acquired causes of electrolyte disorders continues to expand. Examples include hypokalemia due to the activation of colonic potassium secretion in Ogilvie's syndrome [10], and hypomagnesemia, with or without associated hypokalemia, after treatment with the EGF antagonist cetuximab [6, 11, 12]. The management of electrolyte disorders has also evolved considerably in the last decade. Nowhere is this more

evident than in hyponatremia, with the recent availability of vasopressin antagonists [13, 14] and the increasing familiarity with relowering of serum sodium concentration in patients who have corrected too quickly [15].

The integrated analysis and management of fluid, electrolyte, and acid–base disorders can be a daunting challenge, especially for trainees. With this in mind, the last chapter includes ten real-life clinical vignettes that provide a step-by-step analysis of the pathophysiology, differential diagnosis, and management of selected clinical problems.

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