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Preface

Robert G. Dluhy

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Renal Sodium Excretion and Edematous Disorders

Pierre-Yves Martin and Robert W. Schrier

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Body sodium and water homeostasis is regulated by multifactorial renal and extrarenal systems. Following a description of these systems, four common edema states are reviewed, with special emphasis on the pathophysiologic mechanisms leading to sodium and water retention.

Natriuretic Hormones

Eric A. Espiner, A. Mark Richards, Timothy G. Yandle, and
M. Gary Nicholls

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Natriuretic peptides act as endocrine and paracrine hormones to regulate extracellular fluid volume and blood pressure at all levels of the circulation. Atrial and brain natriuretic peptides, circulating hormones secreted in response to increased stretch within the cardiac atrium and ventricle, respectively, induce comparable natriuresis, vasodepression, and inhibition of aldosterone via the guanylate-cyclase receptor, NPR-A. C-type natriuretic peptide acts via a different guanylate-cyclase receptor, NPR-B, to affect vascular cell growth and remodeling. Possible complex interactions among all three natriuretic peptides are reviewed. Although the importance of natriuretic peptides is still being assessed, data from animal studies strongly support an important role for these hormones in cardiovascular homeostasis.

Secondary Aldosteronism

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Dalila B. Corry and Michael L. Tuck

Secondary aldosteronism occurs in states of low effective arterial blood volume, which activates the renin-angiotensin concentration and stimulates the distal reabsorption of sodium by the kidney to restore blood volume. This article discusses the effects of secondary aldosteronism on sodium and potassium in the body. Normal aldosterone function is reviewed, followed by a focus on ischemic hypertensive conditions, including renovascular hypertension, malignant hypertension, renin-producing tumors, pregnancy, chronic renal failure, and iatrogenic secondary hyperaldosteronism.

Idiopathic Edema: Pathogenesis, Clinical Features, and Treatment

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David H. P. Streeten

Idiopathic edema is usually orthostatic. It is most evident in the feet or abdomen after prolonged standing or sitting and in the fingers and eyelids after recumbency overnight. It occurs almost exclusively in post-pubertal women and is associated with discomfort in the areas of fluid accumulation (including symptoms of the carpal tunnel syndrome, nonarticular rheumatism, and headaches, sometimes with pseudotumor cerebri), and weight gain with excessive increments from morning to evening. The pathogenesis, diagnosis, and treatment of idiopathic edema are discussed.

Diabetes Insipidus

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Gary L. Robertson

Diabetes insipidus, characterized by the excretion of copious volumes of unconcentrated urine, results from a deficiency in the action of the antidiuretic hormone arginine vasopressin and can be caused by any of four fundamentally different defects, including impaired secretion (neurohypophyseal diabetes insipidus), impaired renal response (nephrogenic diabetes insipidus), excessive fluid intake (primary polydipsia), or increased metabolism of the hormone (gestational diabetes insipidus). Differentiation between their causes, pathophysiology, and treatment methods is essential for effective management and is best achieved by a combination of hormonal, clinical, and neuroradiologic observations. Understanding of the genetic forms has advanced greatly and may soon lead to improved methods of prevention, diagnosis, and treatment.

Potassium Homeostasis and Hyperkalemic Syndromes

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Barbara A. Clark and Robert S. Brown

Acute hyperkalemia can be a life-threatening consequence of a variety of pathologic, pharmacologic, and iatrogenic disorders.

Recognition and prompt therapy often reverse the electrophysiologic complications within minutes. Therefore, physicians must have a thorough understanding of potassium homeostasis. The causes of hyperkalemia, intracellular-to-extracellular shifts of potassium, excess exogenous potassium loads, and pseudohyperkalemia are discussed.

Primary Aldosteronism

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W. Reid Litchfield and Robert G. Dluhy

The mineralocorticoid-excess state caused by primary aldosteronism usually causes hypokalemia and moderate to severe hypertension. A directed approach to the patient with suspected primary aldosteronism is essential. Appropriate use of biochemical and diagnostic imaging studies can identify the etiology of the primary aldosteronism in an efficient and noninvasive way in most cases. Precision in defining the etiology of the mineralocorticoid-excess state logically leads to therapeutic strategies that usually cure or improve the hypertensive state.

Apparent Mineralocorticoid Excess

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John W. Funder

Apparent mineralocorticoid excess is a congenital syndrome of sodium retention and hypertension with suppressed renin and aldosterone and normal cortisol levels. Patients with the syndrome have, however, highly abnormal levels of urinary cortisol to cortisone metabolites, indicating a reduced or absent activity of 11 β -hydroxysteroid dehydrogenase 2, the enzyme responsible for conversion of cortisol to receptor-inactive cortisone. Very recently, the gene for 11 β -hydroxysteroid dehydrogenase 2 was cloned, and mutations leading to absent or markedly reduced enzyme activity were found in 10 of 11 patients to date.

Disorders of Magnesium Metabolism

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Jerry L. Nadler and Robert K. Rude

Magnesium depletion is more common than previously thought. It seems to be especially prevalent in patients with diabetes mellitus. It is usually caused by losses from the kidney or gastrointestinal tract. A patient with magnesium depletion may present with neuromuscular symptoms, hypokalemia, hypocalcemia, or cardiovascular complication. Physicians should maintain a high index of suspicion for magnesium depletion in patients at high risk and should implement therapy early.

Dietary Factors and Blood Pressure Regulation

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Thomas J. Moore and John A. McKnight

This article reviews the potential effects of dietary constituents and manipulations on blood pressure. Dietary approaches with

documented efficacy are discussed, as are the effects of other nutritional factors, which, after study, demonstrate no proven change on blood pressure. The effects of dietary ions (micronutrients) and of individual dietary macronutrients also are reviewed. The article concludes by examining the evidence for effects of weight loss, alcohol restriction, and vegetarian diets and offers ideas for future research.

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