CURRENT THERAPY IN NEPHROLOGY AND HYPERTENSION 1984-1985

RICHARD J. GLASSOCK, M. D.

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PREFACE

Nephrology is a field of extraordinary diversity. The treatment of patients suffering from renal diseases, disorders of fluid, electrolyte, and acid-base balance or hypertension touches virtually every aspect of the broad array of internal medicine. Many advances have occurred within the last decade which afford new opportunities for management of these disorders.

In organizing this volume, I have attempted to deal with those aspects of the field of nephrology and hypertension which lend themselves to specific dissertations concerning treatment. Each chapter has been introduced by a brief analysis of appropriate diagnostic maneuvers. The contributors have been selected on the basis of their personal experience with each of the specific disorders addressed. Their recommendations stem from this personal experience balanced by a consideration of the literature and the experience of others. When specific pharmacologic agents are mentioned, generic terms have been employed. Whenever agents are discussed which have not yet been released for general marketing purposes in the United States, this fact is carefully documented.

It is readily apparent that some of the recommended therapeutic strategies mentioned in this volume are still in a process of evaluation and evolution. It is anticipated that the modes of therapy for some of the specific diseases mentioned may change significantly in the near future as new pharmacologic agents and novel strategies are developed and tested in clinical trials. Subsequent volumes will hopefully keep the reader abreast of these changes. I sincerely hope that this volume will be useful to its intended audience, that of the practitioner of medicine and specifically individuals who commonly deal with patients with renal disease and hypertension.

I am deeply appreciative of the participation of my colleagues in nephrology in the preparation of this volume. Without their dedicated efforts, the final product could never have been generated. I am also grateful for the excellent secretarial assistance of Pat Vasconcellos, the patient understanding of the publisher, Brian Decker, and the expert editorial assistance of Mary Mansor.

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February, 1984

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FLUID AND ELECTROLYTE DISORDERS

HYPERNATREMIC AND HYPEROSMOLAR SYNDROMES

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Definition of Terms

This brief dissertation on the evaluation and treatment of the hypernatremic and/or hyperosmolar states should begin with a few definitions, since the two terms are not necessarily synonymous. *Hypernatremia* is a precise term referring to an elevation in the plasma or serum concentration of sodium ions above the upper limit of normal (usually >145 mEq/L). As such, hypernatremia always delineates a state of hyperosmolality as sodium, along with its attendant anions, is the major contributor to the total osmolality of the extracellular fluid. A state of hypernatremia implies a disparity between the water and solute content of the body (namely, too little water relative to solute) and can be interpreted, therefore, as indicating a state of dehydration, even though at times the absolute total body amount of sodium and attendant anions may be above normal levels. Thus, the abnormal serum concentration of sodium ion may not reflect the true status of the volumes in the various components of the body fluids. In fact, with hypernatremia and attendant hyperosmolality of the extracellular fluid, the intravascular fluid volume may be seriously reduced.

Hyperosmolar states are usually defined as a plasma osmolality of >290 mOsm/kg water and are not neccessarily synonymous with hypernatremia unless sodium and its attendant anions are the major contributors to the hyperosmolar state per se. Other low-molecular-weight endogenous or exogenous nonionic compounds, such as glucose, urea, mannitol, sorbitol, ethanol, methanol, or glycerol, may contribute to plasma osmolality and thus influence the plasma sodium concentration in different ways. Acute increases in the extracellular fluid concentration of nonionic, osmotically active compounds which penetrate cells poorly [e.g., mannitol, sorbitol, and glucose (in the absence of insulin)] may withdraw water from within the cells and thus initially diminish plasma sodium concentration by movement of water deficient in sodium ion into the extracellular fluid compartment. The hyperosmolar states accompanying increases of extracellular fluid sodium and/or impermeant solutes are also regarded as hypertonic states. Other compounds which penetrate cells freely, such as ethanol and urea, elevate the plasma osmolality, but have little acute effect on the plasma sodium concentration in the extracellular fluid. However, as all of these compounds exert an effect as an osmotic diuretic over extended periods of time, they may induce the excretion of large volumes of an electrolyte-poor urine (osmotic diuresis) and tend to diminish total body water, thus ultimately elevating the plasma sodium concentration if inadequate amounts of solute-free water is provided orally or intravenously.

Although the plasma or serum sodium concentration can be accurately measured by flame photometry or by ion-sensitive electrodes, under certain circumstances plasma or serum electrolyte values may become difficult to measure accurately, particularly at very high or at very low concentrations. Appropriate standards must be included in these circumstances. Plasma osmolality may be estimated from the following formula:

$$(1.95) \times (P_{Na^+} + P_{K^+}) + \frac{\text{BUN}}{2.8} + \frac{\text{Blood Glucose}}{18} = \text{calculated plasma osmolality (in mOsm/kg } H_2O\text{)}.$$

For example:

$$1.95 \times (138 + 4) + \frac{14}{2.8} + \frac{90}{18} = 276 + 5 + 5 = 286 \text{ mOsm/kg } H_2O.$$