KOUSHANPOUR

# RENAL PHYSIOLOGY:

## Principles and Functions

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*An integrated analysis of renal-body fluid regulating systems*

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# PREFACE

Physiologists and clinicians are keenly aware of the truth in the century-old statement by Claude Bernard that constancy of internal milieu is prerequisite to a normal life. The mechanisms by which this constancy of internal environment or "homeostasis" is maintained involve the dynamic interplay of several organ systems, of which the kidney is the most prominent. However, few textbooks on renal function have even attempted to, much less succeeded in, explaining this dynamic interaction to medical and allied health students.

This interdependence is well demonstrated by patients with primary or secondary renal disease, who often exhibit a wide variety of clinical symptoms, seemingly unrelated to the failure of the kidney. Included in these symptoms are: hypertension, of both the arterial and the portal vein variety, fluid retention in dependent extremities, often accompanied by acute renal failure, congestive heart failure, and liver cirrhosis, as well as acid-base disturbances associated with abnormal metabolism, such as diabetes mellitus and gastrointestinal disorders. Although not readily apparent, a careful analysis of the patient's physical and laboratory findings, as well as of the physiology of renal function, would reveal that most, if not all, of these unrelated symptoms can be traced to some disturbance in normal renal regulatory functions. Therefore, to facilitate an intelligent approach to the diagnosis of the underlying cause and the management of the clinical symptoms, it is necessary to acquire a thorough understanding of the renal function in relation to its dynamic interaction with other major organ systems involved in homeostatic regulation.

This book has developed from a 20-lecture course in renal physiology given for the past 12 years by the author to medical students at Northwestern University Medical School. It is an attempt to present an integrated, quantitative analysis of renal function and its role in body fluid homeostasis. The book uses the systems analysis and synthesis approach, which represents a significant departure from the traditional and conventional presentation of the subject. The understanding that such an approach provides is not descriptive, but mechanistic; it imposes mathematical rigor on conceptual processes. It facilitates the search for key factors, alternate possibilities, and missing links that guide experimentation in fruitful directions. At each stage of progress, it summarizes in unambiguous form the current state of understanding so that deficiencies are well exposed to prod further efforts. This approach does not replace experimental ingenuity nor depth of knowledge of the subject, but facilitates and stimulates both. Therefore, application of systems analysis to the study of renal function developed in the present book represents a new and novel approach to the description of this complex physiological system.

This book consists of 13 chapters and two appendices. The first chapter presents an overview of the renal-body fluid regulating system. It not only introduces the reader to the author's approach to the subject, but also brings into focus the unique role the kidney plays in the regulation of body fluid homeostasis. Chapters 2 through 11 are devoted to a rigorous and mechanistic description of body fluids and renal function. Where appropriate, sufficient

background materials are included in each chapter so as to minimize the need for review. For example, Chapter 7 gives a detailed analysis of the biochemical and quantitative concepts necessary to understand the mechanisms of renal transport and the concentration and dilution of urine discussed in Chapters 8, 9, and 11. To better understand the role of the kidney in the regulation of acid-base balance, an extensive discussion of buffers and associated concepts as well as respiratory regulation of acid-base are included in Chapter 10. In this way, the materials in each chapter not only introduce and develop systematically some aspects of renal function, but they also provide the necessary background for the materials presented in the succeeding chapters. Furthermore, unlike other books on the subject, in which the anatomy of the kidney is treated separately, we have integrated the anatomical information with the discussion of kidney function. Also, at the end of each chapter, numerous problems are included, which are designed to further the students' understanding of the materials covered in the text.

Chapters 12 and 13 are devoted to a detailed and integrated analysis and synthesis of the renal-body fluid regulating system, in the light of what has been presented before, and from the standpoints of both normal and pathophysiological disturbances. Included are a mechanistic description of renal function in disease and the extent of its involvement in conditions such as acute glomerulonephritis, pyelonephritis, nephrotic syndrome, hypertension, liver cirrhosis and congestive heart failure. It is hoped that these clinical examples will provide a clear demonstration to the reader of the utility and relevance of the materials presented earlier and contribute to his understanding of the diverse processes which underlie a disease state.

Appendix A is an attempt to introduce the student to the principles of systems analysis and synthesis and its potential application to physiological systems. Appendix B presents a mathematical background for the principle of dilution used in this book. Finally, at the end of the book we have provided answers to some of the problems given at the end of each chapter, designed to increase the understanding of the student of the principles presented in the text.

As written, this book should fulfill the needs of all types of students, including those with little or no mathematical background. At first glance, the quantitative and rigorous approach to the subject may be considered somewhat beyond the need of the medical students. Our experience at Northwestern University Medical School has proved otherwise. The materials presented and the systems analysis approach were received enthusiastically not only by the medical students, but also by the physical therapy and medical technology students. Of course, for the latter group we minimized the extent of mathematical notations, but we made no compromise in the flow and functional diagram approach.

Finally, although the author s primary goal has been to write a book which satisfies the needs of several groups of students, it could be of special interest to researchers in renal physiology as well as medical practitioners. For the latter audience, it should provide a fresh approach to a complex field hitherto not within easy grasp.

It would be impossible to acknowledge and adequately thank everyone who has helped make this book possible. The author is indebted to Professor John S. Gray, who not only introduced him to systems analysis and its application to physiological systems, but also helped with the development of some aspects of the book, especially the acid-base chapter. I wish to express my sin-

cere appreciation to many former medical students, who made invaluable contributions to the development of this book by their enthusiastic and critical feedback as well as their continuous encouragement. I can only say that they made the effort very much worthwhile. I wish to specially thank Miss Jenny L. Forman, who, as a devoted secretary, both encouraged me in the writing of the book and diligently undertook the typing of the manuscript, during all phases of its development. She also meticulously and patiently typed the final manuscript and assisted in proofreading. Special thanks are extended to Mr. Donald Z. Shutters, who skillfully rendered all the original illustrations. I also wish to express my appreciation to all the authors and publishers who kindly permitted the reproduction of the borrowed illustrations. I would like to thank the National Institutes of Health for their generous support of my research, mentioned in the book. Finally, I extend sincere thanks and appreciation to the capable staff of W. B. Saunders Company for their enthusiasm, continued assistance and cooperation during the publication of this book.

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# CONTENTS

# Chapter 1

# INTRODUCTION TO THE RENAL-BODY FLUID REGULATING SYSTEM

All cells of the body are bathed by a fluid, called the interstitial fluid, which provides the *internal environment* of the cells. Both volume and composition of the interstitial fluid must remain within narrow limits, or malfunctions result. Abnormal volumes of vascular and interstitial fluids impair cardiovascular function, and abnormal composition of interstitial fluid impairs cell function. The relevant concentrations include those of electrolytes, hydrogen ions, metabolic waste products, and even water (osmotic effects).

Numerous *disturbing factors* tend to upset both the volumes and composition of these body fluids. These include water ingestion, deprivation, or loss; electrolyte ingestion, deprivation, or loss; fortuitous fluxes of acid or alkali; and the metabolic production of waste products or the administration of toxic substances.

Clearly, there must be *active regulation* to maintain the vital constancy of the internal environment in the face of such disturbing factors. The system, in fact, has two compensating components subject to regulatory control. One is the *G.I. (gastrointestinal) system*, which can appropriately adjust intakes (thirst, appetite, etc.). The other, on which we shall focus in this book, is the *kidney*, which can appropriately adjust outputs. In the renal-body fluid regulating system, the kidney plays much the same compensating role as the bone marrow does for the hemoglobin regulator, or the lung for the blood gas regulator.

## A FLOW-DIAGRAM OF THE RENAL-BODY FLUID REGULATING SYSTEM

We can acquire an initial orientation by examining a flow-diagram of this system in the steady state of normality, as shown in Figure 1-1, which identifies the major fluid compartments of the body and the principal channels of influx and efflux. Briefly, the flow-diagram specifies the pathways of material flow into and out of a subsystem, depicted by a *box*, where material transformation may take place. The material flow into and out of each subsystem is shown by an *arrow*

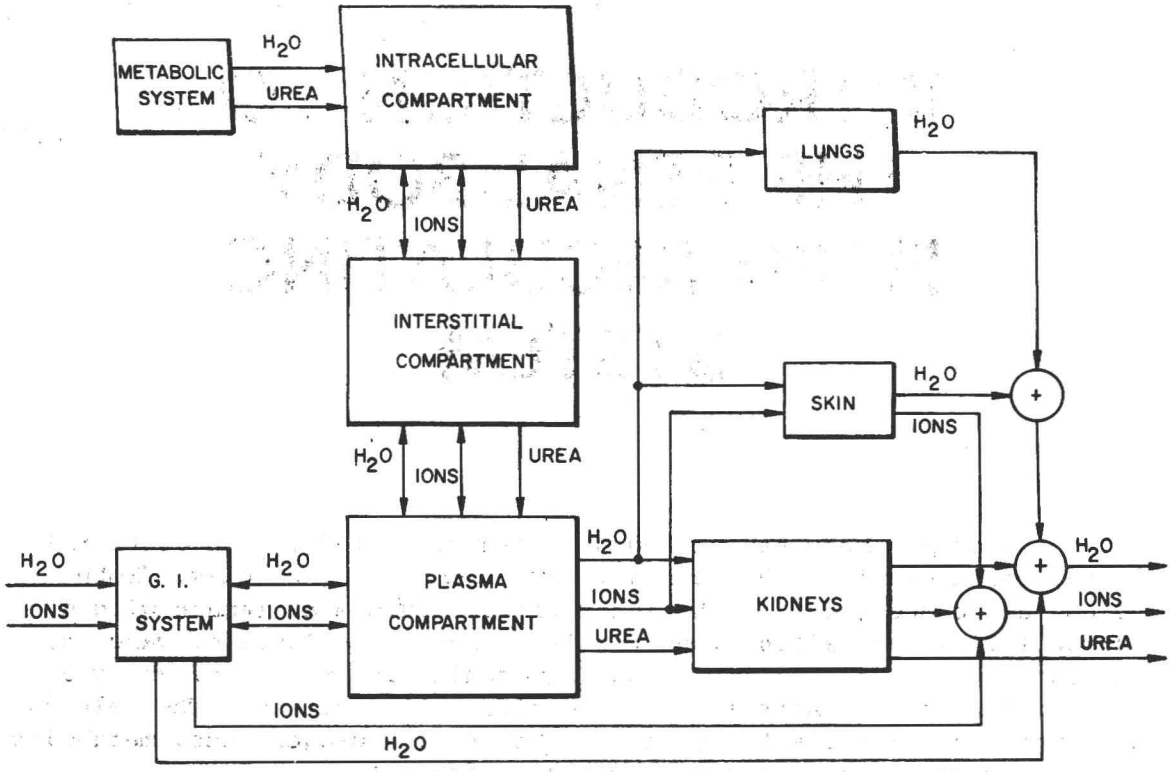


*Figure 1-1.* Flow-diagram of the normal renal-body fluid regulating system.

entering or leaving the box, on the same or opposite sides. In contrast, the *functional diagram* depicts the cause and effect, the input-output relationships for one or many subsystems and the factors influencing their functional relationships. Thus, such a diagram serves as a basis for a quantitative and eventually a mechanistic description of the system. For a detailed treatment of both the flow-diagram and functional diagram, which are liberally employed whenever appropriate in this book, the interested reader is referred to the materials in Appendix A.

You are already familiar with the *plasma* compartment, which comprises a volume of about 3.2 liters in a healthy 70 kg man. Since the plasma circulates throughout the body, it provides the medium for transporting water and solutes from influx to efflux channels, and for exchanging water and solutes with the largely uncirculated interstitial fluid compartment.

The *interstitial* compartment, of about 8.4 liters, is shown to have a two-way exchange with the plasma. This occurs in the tissue-exchanger, i.e., the systemic capillaries, where partially deproteinized plasma escapes into the interstitium from the arterial end of the capillary, and is then reabsorbed into the blood in the venous end of the capillary. The mechanism of this two-way exchange is *filtration*. The exchange fluxes amount to about 4.5 liters/min, so that 3.2 liters

of plasma are turned over every 0.7 min, and the 8.4 liters of interstitial fluid every 1.9 min. In the steady state, the escape and reabsorption rates are equal, but in a transient state they may be temporarily unequal, thus yielding a net *shift* of fluid from one compartment to the other.

The large *intracellular* fluid compartment of about 23.1 liters is also shown to have a two-way exchange with the interstitial compartment. In the steady state, these two-way fluxes for water are enormous, but they are much smaller for ions whose penetration of the cell wall is subject to severe constraints. Water is exchanged by *passive diffusion*, and the ions by *diffusion* and/or *active transport*. In transient states, the two-way fluxes of water may be temporarily unequal, thus yielding a net *shift* of water between the two compartments. The mechanism for such shifts is *osmosis*. Since the cells also constitute a *source* for metabolic water and waste products, such as urea, the metabolic system makes an extra one-way flux for water, and a one-way flux for urea, which also moves by simple diffusion.

We thus see that the three major fluid compartments, which represent body stores of water and ions, are all dynamic turnover pools. Only the circulating plasma is subject to extracorporeal influxes and effluxes, but the interstitial and intracellular compartments can respond quickly, though more or less passively, to changes in the volume and composition of the plasma.

The main channel for *influxes* of water and solutes into body fluids is ingestion via the G.I. tract. On the average, these influxes amount to about 2.5 liters of water and 7.0 g of NaCl per day. To these must be added the 0.3 liters of metabolic water (from oxidation of nutrients) and 30 g of metabolic urea (from deamination of amino acids) per day. In the steady state these influxes are matched by equal *effluxes* of 2.8 liters of water and 7.0 g of NaCl and 30 g of urea per day. Of the four channels for these effluxes, the kidneys are by far the most important, for they eliminate 1.5 liters of water, 6.2 g of NaCl, and essentially all the urea. There is also some loss of water through the respiratory tract, and small losses of water and ions via feces and skin. The water loss by these extrarenal routes amounts to about 1.3 liters in 24 hours.

Clearly, any temporary inequality between total influxes and total effluxes will alter the volume and/or composition of the critical body fluid compartments. In the present context, all influx and efflux channels, *except* the renal, constitute possible disturbance forcings, to which the kidneys respond by making compensatory adjustments of their own controlled effluxes.

## BASIC BIOPHYSICAL CHEMISTRY

Before proceeding further, we must review some basic biophysical chemistry.

The *concentrations* of substances in body fluids are expressed in several ways, each with special applications:

1. *Volume* %, e.g., milliliters of substance per 100 ml of fluid.

This is often used for blood gases, and for the water content of body fluids. For example, plasma contains 94 vol% of water, but red cells only 72 vol%.

2. *Weight %*, e.g., grams of solute per 100 ml of fluid. This mixed weight/volume unit is still widely used. Plasma proteins average 7 g/100 ml of plasma and hemoglobin 35 g/100 ml of red cells. These proteins largely account for the different water contents of cells and plasma.

3. *Molar concentration*, e.g., millimoles per liter (mM/L) of fluid. "Normal" or "physiological" saline solution contains 0.9 g% NaCl, which is (9 x 10 g/L)/(58.5 g/mole x 1000 millimoles/mole) = 154 mM/L. Note that molecular weight of NaCl is 58.5

4. *Equivalent concentration*, e.g., milliequivalents per liter (mEq/L) of fluid. The equivalent weight (or combining weight) is defined as the atomic, radical, or molecular weight divided by valence:

1 mole of urea = 1 equivalent
1 mole of NaCl = 1 equivalent
1 mole of $CaCl_2$ = 2 equivalents
1 mole of $Na_2SO_4$ = 2 equivalents

In the case of electrolytes, the sum of negative charges must equal the sum of positive charges. Since ion valence corresponds to ion charges, the sum of anion equivalents will equal the sum of cation equivalents. For this reason, the ions of body fluids are best expressed in mEq/L.

5. *Osmolar concentration*, e.g., milliosmoles per liter (mOsm/L) of fluid. Osmolality is defined as the number of moles multiplied by the number of dissociating ions:

1 mole urea = 1 osmole
1 mole $Na^+Cl^-$ = 2 osmoles
1 mole $Ca^{++}Cl^-Cl^-$ = 3 osmoles
1 mole $Na^+Na^+SO_4^=$ = 3 osmoles

The *colligative* properties of a solution (freezing point depression, boiling point elevation, potential osmotic pressure, etc.) are functions of osmolar concentrations. The normal osmolar concentration of plasma is about 290 $\pm$ 10 mOsm/L. Normal saline (0.9 g%, 154 mM/L, 154 mEq/L) has an osmolality of 308 mOsm/L, and therefore is not *iso-osmolar* with plasma. The above were all expressed as "bulk" concentrations, i.e., quantities per unit volume of fluid bulk. Sometimes it is more meaningful to use "water" concentrations, i.e., quantities per unit volume of only the water portion of the fluid. The conversion is simply:

$$\text{"Water" concentration} = \frac{\text{"Bulk" concentration}}{\text{Volumetric fraction of } H_2O \text{ in the fluid}} \qquad (1\text{-}1)$$

Thus, the lower the water content of a fluid the more the "water" concentration exceeds the "bulk" concentration. We shall make these distinctions in the following way:

| *"Bulk" Concentration* | *"Water" Concentration* |
|---|---|
| g% | g% in $H_2O$ |
| mM/L (Molar) | mM/L $H_2O$ (Molal) |
| mEq/L | mEq/L $H_2O$ |
| mOsm/L (Osmolar) | mOsm/L $H_2O$ (Osmolal) |

Table 1-1 lists the major plasma electrolytes and their concentrations expressed in different units.

*Osmosis* is the flow of water across a membrane from a solution on one side to a solution on the other side, the latter containing a higher osmolality of solutes to which the membrane is impermeable. The water moves from the higher to the lower concentration of *water* just like a diffusion process. But since the water is the *solvent*, and not just a dissolved solute, the water flows as a convection process, analogous to filtration.

The dependency of osmosis on solute concentration is related to the change in the *chemical potential* of water caused by addition of solute. The chemical potential or molal free energy for pure water ($\mu$) is defined as the ratio of a change in total free energy of water ($\Delta F_{H_2O}$) to a change in the number of moles of water ($\Delta n_{H_2O}$), at constant ambient temperature and pressure. Expressed mathematically,

$$\mu = \frac{\Delta F_{H_2O}}{\Delta n_{H_2O}} \qquad (1\text{-}2)$$

It so happens that the chemical potential of water in a solution is lower than that of pure water. Therefore, when a solution is separated from pure water by a membrane (permeable only to water), a chemical potential difference between the two sides develops. This difference in chemical potential can be abolished by at least three processes. (1) The free distribution of solutes on both sides of the membrane. This has the effect of equalizing the chemical potential of water on both sides. However, this is not possible if the membrane is permeable only to water. (2) The diffusion of water through the membrane, thereby equalizing the chemical potentials on both sides. (3) The application of mechanical pressure to the solution in order to increase its chemical potential to the level equal to that for pure water. The mechanical pressure thus applied is called the *osmotic pressure* and is a measure of the difference between the chemical potential of the solution and that of pure water.

The osmotic pressure of a solution as defined above is a measure of the lowering of the chemical potential of pure water by the addition of solute. Since the lowering of the chemical potential depends only on the number of solute particles added, the osmotic pressure depends on the number of particles in that solution and not on their size or weight.

If the solution on one side is pure water and that on the other side is one *osmolal* strength of a completely impermeable solute, the osmotic pressure that develops is 22.4 atmospheres, or 17,024 mm Hg. Normal saline of 308 mOsm/L $H_2O$ thus has a *potential* osmotic pressure

TABLE 1-1

Conversion of Plasma Electrolyte Concentrations to mEq/L, or mg%

| Electrolytes | Calculated as | Atomic Weight | Valence | Equivalent Weight | Conversion Factors (mEq/L from mg%: DIVIDE; mg% from mEq/L: MULTIPLY) | Plasma Concentration (normal ranges) mg/100 ml | mEq/L |
|---|---|---|---|---|---|---|---|
| CATIONS | | | | | | | |
| Sodium | Sodium | 23 | 1 | 23 | 2.3 | 310-335 | 136-145 |
| Potassium | Potassium | 39 | 1 | 39 | 3.9 | 14-21.5 | 3.5-5.5 |
| Calcium | Total Calcium | 40 | 2 | 20 | 2.0 | 9-11 | 4.5-5.5 |
| Magnesium | Magnesium | 24 | 2 | 12 | 1.2 | 1.8-3.6 | 1.5-3.0 |
| ANIONS | | | | | | | |
| Bicarbonate | $CO_2$ Content | | | 22.26 | 2.2 | 53-75(av. 62) vol% | 24-33(av. 28) |
| Bicarbonate | $CO_2$ Combining Power | | | 22.26 | 2.2 | 53-78(av. 65) vol% | 24-35(av. 30) |
| Chloride | Chloride | 35.5 | 1 | 35.5 | 3.5 | 350-375 | 98-106 |
| Chloride | Sodium Chloride | 58.5 | 1 | 58.5 | 5.8 | 570-620 | 98-106 |
| Phosphate | Phosphorus | 31.0 | 1.8 | 17.2 | 1.7 | 2-4.5 | 1.2-3.0 |
| Sulfate | Sulfur | 32.0 | 2 | 16.0 | 1.6 | 0.5-2.5 | 0.3-1.5 |
| Protein | Protein | | | | 0.41 | 6-8 grams | 14.6-19.4 |

The phosphate is calculated as phosphorus, with a valence of 1.8. The reason for this is that at normal pH of the extracellular water, 20 per cent of the phosphate ions are in a form with one sodium equivalent ($NaH_2PO_4$), and 80 per cent are in a form with two sodium equivalents ($Na_2HPO_4$). The total valence is therefore (0.2 x 1) + (0.8 x 2) = 1.8.

[From Goldberger, E. (1975).]

of 5244 mm Hg. We say potential, for osmotic pressure that can actually develop depends not only on osmolality, but also on the permeability characteristics of the membrane system used.

The membranes of body cells have permeability constraints, such that NaCl cannot pass through the membrane, although $H_2O$ can easily pass. Hence, cells with an intracellular concentration of impermeable solutes of 154 mOsm/L $H_2O$ and a concentration of impermeable solutes of 154 mOsm/L $H_2O$ in the interstitial fluid are in *osmotic equilibrium*, so that there is no osmotic flow, or shift of water, between them. A solution of impermeable solute having an osmolal concentration of 154 mOsm/L $H_2O$ is said to be an isotonic solution. A solution of 154 mOsm/L $H_2O$ of a *permeable* solute, such as urea, is *iso-osmolar*, but *not isotonic*, for it will not develop an osmotic pressure across the cell membrane.

Whenever the intracellular fluid is exposed to hypertonic (or hypotonic) interstitial fluid, water will flow out of (or into) the cells until the osmolality becomes equal again on both sides. In short, any inequality of impermeable osmolal concentrations can be rectified only by the osmotic shift of water into or out of the cells. Or, to say the same thing another way: *All changes in intracellular fluid volume* (except growth, of course) *are the result of changes in the osmolality of the interstitial fluid.*

The normal efflux channels already identified vary in the osmolality of their fluids. For example, the pulmonary efflux consists of water *vapor*, with zero osmolality. Skin efflux, even in heavy sweating, is hypotonic. Effluxes from the G.I. tract (vomiting and diarrhea, for example) are generally isotonic. Since the kidneys must be able to compensate for both hypo- and hypertonic fluxes, it is not surprising to find that urine osmolality may be adjusted, as needed, from 1/6 isotonicity to 4 times isotonicity.

## TYPICAL FORCINGS AND RESPONSES OF THE RENAL-BODY FLUID REGULATING SYSTEM

The disturbance forcings which produce water and electrolyte imbalances include fortuitous *gain* of fluids via the influx channels, fortuitous *loss* of fluids through the efflux channels, and combinations of these. In practice, most of these forcings occur intermittently and are usually short-lived. Hence, they are properly called *pulse* forcings,* rather than step forcings, and are followed by *recovery*. The compensatory response of the kidneys, therefore, is to *accelerate* the recovery process and thereby speed up the restoration of volumes and compositions of body fluids toward normal.

Since fortuitous fluid gained or lost may contain different proportions of water and electrolytes, the above forcings are subclassified as *isotonic*, *hypotonic*, or *hypertonic* forcings. Thus, on the influx side, we may have fortuitous gain of isotonic,

*See Appendix A for classification of forcings and their characteristics.

hypotonic, or hypertonic fluids, and on the efflux side, we may have fortuitous loss of isotonic or hypotonic fluids. The fortuitous loss of hypertonic fluid occurs only in patients with the syndrome of inappropriate secretion of antidiuretic hormone (SIADH), which will be discussed in Chapter 3.

Table 1-2 summarizes the responses of the renal-body fluid regulating system to the forcings just described. For each forcing, the deviation from normal is indicated by (+) for increase, (-) for decrease, and (0) for no change. The direction of water shift between the extracellular (interstitial plus plasma compartments) and intracellular compartments is indicated by an horizontal arrow (→ or ←).

### *INFLUXES*

A. ISOTONIC. Oral intake or parenteral infusion of a large volume of isotonic saline increases the plasma volume, causing secondary transfer of fluid into the interstitium. The net result is a uniform expansion of the extracellular fluid volume. Since the ingested fluid is isotonic, there is no change in osmolality of the interstitial fluid and hence no net osmotic shift of water into or out of the cells. These characteristic changes in the body fluid compartments produced by fortuitous isotonic fluid influx are termed *isotonic hydration*. Unless otherwise specified, both "tonicity" and "hydration" refer to the extracellular fluid compartment.

The kidneys respond to this extracellular volume expansion by increasing their excretion of both salt and water, producing an increase in urine volume (diuresis). This controlled diuresis rapidly returns the extracellular volume back to normal.

B. HYPOTONIC. Ingestion of a large volume of plain water increases the plasma volume and dilutes plasma osmolality. Fluid then shifts from the plasma into the interstitium. This increases the extracellular volume and decreases its osmolality *(hypotonic hydration)*. The reduced interstitial osmolality causes osmotic shift of water into the cells, causing them to swell and diluting their osmolality. This is called *water intoxication* of cells. The kidneys respond by increasing the excretion of a dilute urine (reduced osmolality), thereby returning the intracellular and extracellular volumes and osmolalities back to normal.

C. HYPERTONIC. Oral or parenteral intake of large amounts of hypertonic fluid increases plasma volume and osmolality. This causes osmotic shift of water into the plasma from the interstitium and diffusion of salt in the opposite direction. The net result is an increase in the volume and osmolality of the extracellular fluid *(hypertonic hydration)*. This induces osmotic shift of water out of the cells, thus reducing their volumes but increasing their osmolality. The kidneys respond by excreting a concentrated urine, thereby restoring the normal state.

TABLE 1-2

Changes from Normal in Fluid Compartments and Controlled Renal Effluxes in Response to Typical Forcings

| Forcings | Effects on Fluid Compartments | | | | | Terminology | Renal Effluxes | |
|---|---|---|---|---|---|---|---|---|
| | Extracellular | | Water Shift | Intracellular | | | | |
| | Vol. | Osmolal. | | Vol. | Osmolal. | | Vol. | Osmolal. |
| 1. Influxes | | | | | | | | |
| a. Isotonic | + | 0 | 0 | 0 | 0 | Isotonic Hydration | + | 0 |
| b. Hypotonic | + | - | → | + | - | Hypotonic Hydration | + | - |
| c. Hypertonic | + | + | ← | - | + | Hypertonic Hydration | + | + |
| 2. Effluxes | | | | | | | | |
| a. Isotonic | - | 0 | 0 | 0 | 0 | Isotonic Dehydration | - | + |
| b. Hypotonic | - | + | ← | - | + | Hypertonic Dehydration | - | + |