2nd edition

surgery

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The Natio[illegible]s for Independent Study

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Preface

In keeping with the purpose of the *National Medical Series for Independent Study*, this book presents the core material of the specialty of surgery. The text is not meant to be all-inclusive and does not contain minutiae that we felt would be of little use to the reader. The authors have included not only didactic material but also facts that they find useful in clinical practice. Where controversy exists, we have attempted to present all sides fairly and to indicate factors that are essential in the decision-making process. We have also tried to stress situations in which surgeons and all others involved in patient care must work closely to make the most appropriate decisions regarding treatment.

In preparing the second edition of *Surgery*, we were able to focus on refining and updating the content and expanding the text when necessary. All of the chapters are now better organized in a more logical format. Certain chapters, such as Principles of Surgical Physiology and Liver, Portal Hypertension, and Biliary Tract, have been completely reorganized and expanded, and other chapters, such as Organ Transplantation, have been updated to reflect the rapid advances in the different fields of surgery. Two hundred study questions and explanations have also been added.

Surgery, 2nd edition, was written primarily for students and residents in general surgery, but practicing surgeons as well as physicians in other specialties will no doubt find it a useful reference. We hope that all readers will find *Surgery*, 2nd edition, represents a declaration of the state of surgical art in 1990.

Bruce E. Jarrell
R. Anthony Carabasi, III

Contents

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We would also like to express the admiration, appreciation, and affection that we feel for Francis E. Rosato, M.D., Samuel D. Gross Professor and Chairman of the Department of Surgery at Thomas Jefferson University. In our opinion, he is the ideal academic surgeon.

This work would not have been possible without the dedicated help of Mrs. Joanne Crouch. We gratefully acknowledge her assistance.

To the Reader

Since 1984, the *National Medical Series for Independent Study (NMS)* has been helping medical students meet the challenge of education and clinical training. In this climate of burgeoning knowledge and complex clinical issues, a medical career is more demanding than ever. Increasingly, medical training must prepare physicians to seek and synthesize necessary information and to apply that information successfully.

The *National Medical Series* is designed to provide a logical framework for organizing, learning, reviewing, and applying the conceptual and factual information covered in basic and clinical sciences. Each book includes a comprehensive outline of the essential content of a discipline, with up to 500 study questions. The combination of an outlined text and tools for self-evaluation allows easy retrieval of salient information.

All study questions are accompanied by the correct answer, a paragraph-length explanation, and specific reference to the text where the topic is discussed. Study questions that follow each chapter use current National Board format to reinforce the chapter content. Study questions appearing at the end of the text in the Challenge Exam vary in format depending on the book. Wherever possible, Challenge Exam questions are presented as a clinical case or scenario intended to simulate real-life application of medical knowledge. The goal of this exam is to challenge the student to draw from information presented throughout the book.

All of the books in the *National Medical Series* are constantly being updated and revised. The authors and editors devote considerable time and effort to ensure that the information required by all medical school curricula is included. Strict editorial attention is given to accuracy, organization, and consistency. Further shaping of the series occurs in response to biannual discussions held with a panel of medical student advisors drawn from schools throughout the United States. At these meetings, the editorial staff considers the needs of medical students to learn how the *National Medical Series* can better serve them. In this regard, the *National Medical Series* staff welcomes all comments and suggestions.

1
Principles of Surgical Physiology

Michael J. Moritz

I. FLUID, ELECTROLYTE, AND ACID-BASE DISTURBANCES

A. Overview

1. Fluid and electrolyte **homeostasis** is determined by the individual's intake and output and is precisely regulated by the normal, healthy body.

2. The distribution of body water is shown in Table 1-1. The electrolyte composition and volume of various body fluids is shown in Tables 1-2 and 1-3. Table 1-4 shows the daily flux—losses and requirements—of water and electrolytes.

3. In disease states, regulatory mechanisms can become impaired and imbalances occur. The surgeon frequently encounters patients with these problems, which may be worsened by the additional stress of surgery, the use of tubes that drain fluids, which are not usually excreted, and the patient's inability to tolerate oral intake of fluids and nutrients.

B. Common acid-base disturbances

1. **Respiratory acidosis** is caused by carbon dioxide retention due to inadequate alveolar ventilation.
   a. **Values.** Arterial blood gases show increased P_{CO_2} and decreased pH.
   b. **Compensation** by renal bicarbonate (HCO_3^-) retention takes days.
   c. **Treatment** is improved ventilation.

2. **Respiratory alkalosis** is caused by an increased loss of carbon dioxide due to hyperventilation.
   a. **Values.** Arterial blood gases show decreased P_{CO_2} and increased pH.
   b. **Treatment** is decreased ventilation (e.g., sedatives) or rebreathing the same air to decrease carbon dioxide loss.

3. **Metabolic acidosis** is due to a loss of HCO_3^- or retention of some acids.
   a. **Values.** Serum electrolyte values reveal decreased HCO_3^- levels on an SMA6. A blood gas reveals a decreased pH.
   b. **Compensation** is by increased ventilation to lower carbon dioxide levels.
   c. **A high anion gap** defines conditions resulting from the accumulation of acids. Anion gap is defined as:

$$\text{sodium } (Na^+) - [\text{chloride } (Cl^-) + HCO_3^-]$$

   with normal values equal to 15 or less.
   (1) Normal anion gap acidosis is due to HCO_3^- or Cl^- loss.
   (2) High anion gap acidosis is due to diabetic ketoacidosis, renal failure, overdose of methanol, ethanol, ethylene glycol (antifreeze), paraldehyde, or aspirin, and lactic acidosis.
   d. **Treatment** varies with the cause.
   (1) HCO_3^- loss due to diarrhea or pancreatic fistulas is treated with appropriate fluid and HCO_3^- replacement.
   (2) Acid retention requires specific therapy for each cause.
   (a) Diabetic ketoacidosis is treated with fluid replacement and insulin.
   (b) Renal acidosis is treated with HCO_3^- replacement and dialysis.
   (c) Lactic acidosis is managed with the treatment for shock (see Chapter 9 V).

Table 1-1. Distribution of Body Water

Compartment	Body Weight (%)*
Total body water	60
Extracellular fluid	20
Plasma	3–5
Interstitial fluid	15–18
Intracellular fluid	40

*Percentage decreases as body fat increases.

Table 1-2. Electrolyte Composition of Body Fluids

	Electrolyte Content (mEq/L)							
Fluid	Na^+	K^+	H^{+*}	Cl^-	HCO_3^-	Protein*	PO_4^-	SO_4^-
Plasma	142	4.5	. . .	100	25	16	2	1
Gastric juice								
High-acid	45	30	70	120	25	. . .	. . .	. . .
Low-acid	100	45	0.015	115	30	. . .	. . .	. . .
Intestinal juice	120	20	. . .	110	30	. . .	. . .	. . .
Bile	140	5	. . .	. . .	40	. . .	. . .	. . .
Pancreatic juice	130	15	. . .	. . .	80	. . .	. . .	. . .
Intracellular fluid	10	150	. . .	5	10	60	100	20

*Subject to wide variation in gastric and intestinal fluids.

Table 1-3. Volume of Gastrointestinal Tract Fluids

Fluid	Volume (ml) Produced per Day
Saliva	1500
Gastric secretions	2500
Bile	500–1500
Pancreatic juice	700
Small bowel secretions (succus entericus)	3000
Total	8200–9200

4. **Metabolic alkalosis** is due to loss of acid or loss of potassium (K^+). (Without adequate K^+, the kidney must exchange H^+ for Na^+, resulting in an acid loss in the urine.)
   a. **Values.** Serum electrolytes reveal elevated HCO_3^- and decreased K^+ levels.
   b. **Compensation** is by hypoventilation to increase carbon dioxide levels and renal HCO_3^- loss.
   c. **Treatment** varies with the cause. Loss of acid, usually from vomiting or nasogastric suctioning, requires fluid, K^+, and occasionally H^+ repletion. K^+ loss, usually due to diuretics, requires K^+ repletion.

C. **Fluid and electrolyte imbalance in surgical patients**

1. **Terms**
   a. **Volume**
      (1) Too much water is defined as overhydration or **hypervolemia.**
      (2) Too little water is defined as dehydration or **hypovolemia.**
   b. **Concentration** is described in terms of Na^+, the principal extracellular cation (i.e., **hyper-** or **hyponatremia**).

Table 1-4. Normal Daily Fluid and Electrolyte Losses and Requirements

Substance	Losses/24 hr					Requirements/24 hr/kg of body weight
		Insensible				
	Urine	Skin	Lungs	Feces	Total	
Water	1200–1500 ml†	200–400 ml†	500–700 ml‡	100–200 ml	2300–2600 ml	35 ml
Sodium	100 mEq§	40 mEq/L of sweat	. . .	. . .	80–100 mEq	1 mEq
Potassium	100 mEq‖	. . .	. . .	. . .	80–100 mEq	1 mEq
Chloride	150 mEq§	40 mEq/L of sweat	. . .	. . .	100–150 mEq	1.5 mEq
Bicarbonate	. . .	. . .	. . .	. . .	40 mEq*	0.5 mEq

*Lost in urine and lungs (as CO_2).
†25 ml/kg of body weight.
‡10 ml/kg of body weight.
§Varies with intake and with volume of urine and sweat. Autoregulated via renin–angiotensin–aldosterone system.
‖Aldosterone increases excretion.

(1) Note that there are three factitious causes of hyponatremia:
(a) Dilutional hyponatremia (see I C 2 e)
(b) Hyperlipidemia
(c) Severe hyperglycemia

(2) With severe hyperglycemia, the serum Na^+ should be adjusted upward 3 mEq/L for every 100 mg/dl of glucose above normal [e.g., a serum glucose of 800 mg/dl (700 mg/dl above normal) will factitiously decrease serum Na^+ by about 21 mEq/L].

2. **Types of imbalance.** Any imbalance can be iatrogenic due to error or inattention.
   a. **Isotonic dehydration** is a proportionate loss of water and salt. This is the commonest imbalance in surgical patients. Common causes are:
      (1) Loss of blood (externally or internally)
      (2) Loss of gastrointestinal fluids from vomiting, nasogastric suction, fistula drainage, or diarrhea
      (3) **Third-space losses.** The third space, after intracellular and extracellular spaces, represents any soft tissue space. Injury or inflammation that leads to tissue swelling and fluid sequestration represents a third-space loss. Examples include burns, pancreatitis, peritonitis, bowel obstruction, retroperitoneal surgery, and cellulitis.
   b. **Hypernatremic dehydration** is the loss of water in excess of salt. It is caused by excessive loss of hypotonic fluid, for example, by perspiration, diabetes insipidus, osmotic diuresis (e.g., mannitol or hyperglycemia), and diarrhea.
   c. **Hyponatremic dehydration** is the loss of salt in excess of water. The commonest causes are renal salt wasting (e.g., diuretics or Addison's disease) and gastrointestinal losses.
   d. **Hypernatremic overhydration** is rare and due to the overzealous administration of hypernatremic fluids, such as fresh frozen plasma.
   e. **Hyponatremic overhydration** is common and generally is due to water overload with normal total body Na^+ (**dilutional hyponatremia**). It is usually **iatrogenic**, caused by the replacement of losses with inappropriately hypotonic fluids. It can also be due to the **syndrome of inappropriate antidiuretic hormone (SIADH) secretion**, which causes overconservation of free water. The treatment is **water restriction**.

3. **Physiologic responses**
   a. **Dehydration** causes activation of several neuroendocrine systems.
      (1) **Renin** is released by the juxtaglomerular apparatus, which senses the decreased circulatory volume. Renin, in turn, activates **angiotensin I**, which is then converted to angiotensin II.
      (2) **Angiotensin II**, a potent vasoconstrictor, also stimulates aldosterone and antidiuretic hormone (ADH) secretion.
      (3) **Aldosterone** acts on the renal tubules to conserve Na^+.
      (4) **ADH**, which is also secreted in response to an increase in plasma osmolarity, causes renal water conservation.
   b. **ADH secretion** is also increased by any significant stress, including trauma, burns, hemorrhage, sepsis, or major surgery. For example, increased ADH secretion persists for up to 5 days after surgery, accounting for the fluid retention that occurs postoperatively.
   c. **Hypervolemia** suppresses aldosterone and ADH secretion, allowing renal diuresis of salt and water. It also causes release of atrial natriuretic factor, which magnifies the diuresis.

D. Diagnosis of fluid–electrolyte disturbances

1. **A history of fluid losses** makes dehydration likely.
   a. **External losses** (e.g., bleeding, melena, vomiting, or diarrhea) are usually obvious.
   b. **Internal losses** (e.g., fluid lost into obstructed bowel, pancreatitis, or internal bleeding) may be subtle.

2. **Physical examination** must be attentive to:
   a. Vital signs. Orthostatic hypotension (pulse and blood pressure) must be checked.
   b. Changes in the patient's weight
   c. Skin turgor
   d. Moistness of mucous membranes
   e. Venous filling

3. **Laboratory tests** should always include:
   a. Hematocrit
   b. Serum Na^+, K^+, HCO_3^-, Cl^-, and glucose.

c. Blood urea nitrogen (BUN) and creatinine
d. Chest x-ray
e. **Other tests** if needed
(1) Serum osmolarity (normal: 285–295 mOsm/L). Serum osmolarity can be approximated by the formula:

$$\text{serum (mOsm/L)} = 2 \times Na^+ \text{ (mEq/L)} + \frac{\text{glucose (mg/dl)}}{18} + \frac{\text{BUN (mg/dl)}}{2.8}$$

(2) Urine Na^+ and osmolarity

4. **Indirect measurements of circulating volume** (i.e., central venous pressure and pulmonary arterial and wedge pressures) may be useful for diagnosis or for titrating treatment (rehydration).

E. Calculating the amount of the deficit

1. **A volume (water) deficit** can be estimated clinically from the patient's body weight and appearance or can be calculated from the serum Na^+ level.
a. **Clinical estimates**
(1) In **mild dehydration**, the patient has lost 3% of total body water and complains of thirst.
(2) In **moderate dehydration**, 6% of total body water is lost, and clinical signs of dehydration are evident:
(a) Marked thirst and dry mucous membranes
(b) Absent groin or axillary sweat
(c) Loss of skin turgor
(3) In **severe dehydration**, 10% of total body water is lost, clinical signs of dehydration are marked, orthostatic changes or hypotension may be present, and the patient may be confused or delirious.
b. **In hypernatremia**, the water deficit can be calculated as follows: Given that normal serum Na^+ = 140 mEq/L and total body water = 0.6 × body weight (i.e., 60% of body weight), then

$$\text{water deficit} = \frac{\text{observed } Na^+ - 140}{140} \times 0.6 \times \text{body weight (kg)}$$

2. **Electrolyte deficits** are calculated from laboratory tests.
a. **Na^+, Cl^-, and HCO_3^- deficits** are calculated using the following equation:

$$\text{Deficit} = \begin{matrix}\text{[normal value} - \\ \text{observed value (mEq/L)]}\end{matrix} \times \begin{matrix}\text{electrolyte distribution in} \\ \text{body compartment (\%)}\end{matrix} \times \text{body weight (kg)}$$

where the **Na^+ distribution = 60%**, the **Cl^- distribution = 20%**, and the **HCO_3^- distribution = 50%**. (Despite the fact that Na^+ is primarily an extracellular ion, the Na^+ space is considered to equal total body water because Na^+ controls total body osmolality. Therefore, 60% is used as the Na^+ distribution.)
b. **K^+ deficits** are incalculable. **With a normal blood pH,** the estimate is:
(1) For every 1.0 mEq/L decrease in the K^+ concentration between normal and 3.0 mEq/L, consider the total body deficit as 100–200 mEq.
(2) For every 1.0 mEq/L decrease in the K^+ concentration below 3.0 mEq/L, consider the total body deficit as **another** 300–400 mEq/L.

F. Management of fluid, electrolyte, and acid–base imbalance

1. **Priorities**
a. **Correct shock and restore blood volume to normal** (see Chapter 9 V).
b. **Correct deficits.**
(1) Acid–base imbalance
(2) Serum osmolarity
(3) Electrolytes
c. **Define replacement therapy.**
d. **Define maintenance requirements** for fluids and electrolytes.
2. **Deficit correction** is defined as the fluid and electrolyte therapy necessary to correct existing deficits. Deficit correction has **top priority** in fluid and electrolyte therapy.