A Handbook for Pediatric Surgery

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Williams & Wilkins

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WILLIAMS & WILKINS
Baltimore/London

428 E. Preston Street
Baltimore, Md. 21202, U.S.A.

Made in the United States of America

Library of Congress Cataloging in Publication Data

Ternberg, Jessie L
A handbook for pediatric surgery.
Includes index.
1. Children—Surgery. 2. Diagnosis, Surgical. I. Bell, Martin J., joint author. II. Bower, Richard J., joint author. III. Title.
RD137.T47 617'.98 79-22941
ISBN 0-683-08150-0

Composed and printed at the
Waverly Press, Inc.
Mt. Royal and Guilford Aves.
Baltimore, Md. 21202, U.S.A.

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Preface

This manual was originally designed to help general surgical residents and pediatric residents during their monthly rotations on the pediatric surgical service at St. Louis Children's Hospital. Just as these residents need a ready source of practical points, the pediatrician in practice may have occasional need of practical thoughts regarding the management of pediatric surgical problems.

It is our aim to provide the pediatrician, general practitioner, and resident with a ready reference to one way of handling surgical problems that he may encounter only occasionally.

The manual is not meant to be a textbook and makes no effort to discuss every method of handling problems, but rather covers *our* current approaches to and various methods of giving pediatric care.

Acknowledgment

The authors would like to specifically acknowledge and thank many who contributed greatly to the completion of this Manual. Our administrative secretary, Opal J. Puttin, and our copy editor and coordinator, Linda L. Bradford, put in many extra hours of organization to reach this final copy. Vicki Moses Friedman, Supervisor of the Division of Surgical Illustration, contributed the fine drawings and figures. Finally, we are indebted to the Pediatric and Surgical House Officers who, over the years, have raised questions and advanced ideas that provided the underlying incentive for this effort.

Contents

CHAPTER 1

The Three R's of Fluid and Electrolyte Use

The use of fluids and electrolytes in the care of infants and children will be discussed under the three headings of regular, repair, and replacement, defined as follows:

Regular. These are fluids and electrolytes necessary for daily maintenance.

Repair. The fluids and electrolytes necessary to correct existing deficits are designated "repair."

Replacement. Finally these fluids and electrolytes are necessary to replace continuing losses not covered by the regular maintenance fluids and electrolytes.

REGULAR MAINTENANCE FLUIDS AND ELECTROLYTES

The quantity of regular maintenance fluid and electrolyte required in 24 hours is based upon our information about daily losses. The sources of daily water loss are:

1. Insensible--the loss of water from skin and lungs by evaporation
2. Renal--the water necessary to eliminate the osmotic load delivered to the kidneys; this loss will vary with the urine solute content and concentration
3. Sweat--the water loss varies with the environmental temperatures
4. Gastrointestinal--the water loss increases with diarrhea

The two major sources of regular, daily water loss are insensible and the renal water losses. Each is a function of the metabolic rate. The surface area and the metabolic rate of an individual can be shown to be related. It is possible therefore to base the calculations for the regular maintenance fluid requirements on an individual's surface area:

1. Insensible loss is about 500 $cc/m^2/24$ hr. This loss increases with fever, 10% to 14%/each °C of elevation.
2. Obligatory renal loss is 500 to 800 $cc/m^2/24$ hr.
3. Combined sweat and GI loss is about 200 $cc/m^2/24$ hr.
4. The regular maintenance fluid requirement on this basis is 1,500 $cc/m^2/24$ hr.

Based on weight (in pounds) the surface area can be estimated from a simplified nomogram (Fig. 1-1).

Electrolyte requirements for maintenance can also be expressed as follows: Na^+, 50 $mEq/m^2/24$ hr; and K^+, 40 $mEq/m^2/24$ hr. This is about 3 mEq $Na^+/kg/24$ hr in the infant, decreasing to about 1.5 mEq $Na^+/kg/24$ hr in the child. The Na^+ and the K^+ requirements will be slightly less in the neonate than for the infant. A maintenance fluid can be prepared by adding KCl (20 to 30 mEq $K^+/100$ cc) to 0.25 N saline which will provide the appropriate amounts of sodium and potassium when regular maintenance fluid is provided. Commercial solutions are also available with electrolyte concentrations that will provide appropriate kinds and amounts of electrolytes when maintenance fluid is administered (see Table 1-1). Glucose should be added to maintenance solutions to correct osmolarity (see Table 1-1). In addition, the administration of 5 gm glucose/100 ml maintenance fluid will usually suffice to prevent ketosis. A higher concentration of glucose may be necessary to prevent hypoglycemia in neonates.

pounds M^2	3 0.1	6 0.2						
pounds M^2	12 0.3	18 0.4	24 0.5	30 0.6	36 0.7			
pounds M^2	40 0.8	50 0.9	60 1.0	70 1.1	80 1.2	90 1.3	100 1.4	110 1.5

Fig. 1-1. Nomogram for estimation of surface area.

Table 1-1. Composition of IV fluids

SOLUTION	DEXTROSE (gm/L)	mEq								mOsm/L
		Na^+	K^+	Ca^{++}	Mg^{++}	Cl^-	ACETATE	HPO_4	LACTATE	
5% Dextrose in water	50									278
10% Dextrose in water	100									556
5% Dextrose in saline	50	145				145				568
N saline (0.9%)		154				154				308
0.5 N saline		77				77				154
5% Dextrose in 0.5 N saline	50	77				77				432
Ringer's lactate		130	4	4		111			27	276
5% Dextrose in Ringer's lactate	50	130	4	4		111			27	554
0.2 N saline		31				31				62
5% Dextrose in 0.2 N saline	50	31				31				340
0.67 N saline		103				103				206
5% Dextrose:										
Isolyte P (McGraw)	50	25	20	3		22	23	3		345
Ionosol MB (Abbott)										
Electrolyte solution #48 (Baxter-Travinol, Cutter)										
5% Dextrose:										
Isolyte M (McGraw)	50	40	35			40	20	15		400
Ionosol T (Abbott)										
Electrolyte solution #75 (Baxter-Travinol, Cutter)										

Table 1-2

	MILD DEHYDRATION	MODERATE DEHYDRATION	SEVERE DEHYDRATION
% WEIGHT LOSS	5%	10%	15%
BEHAVIOR	Irritable	Extremely irritable	Lethargic
SKIN TURGOR	↓	↓↓	↓↓↓
SKIN COLOR	Pale	Pale to grey	Grey to mottled
PULSE	Normal to ↑	↑	↑↑
BP	Normal	Normal	↓
URINE OUTPUT	↓	↓↓	Oliguric
URINE SPECIFIC GRAVITY	Greater than 1.010	Greater than or equal to 1.020	
BUN	↑	↑	↑↑
HCT	No change	↑	↑↑
TSS	No change	↑	↑↑

REPAIR FLUIDS AND ELECTROLYTES

The problems are: (1) degree of dehydration, (2) type of dehydration, and (3) disturbances of acidosis/alkalosis.

Degree Of Dehydration

The degree of dehydration is an estimated quantity based on weight loss, clinical evidence, and laboratory evidence.

Weight loss is a reliable measurement of the deficit if the loss is acute. In any situation where fluid is lost because it is sequestered, i.e., it is not available as functional extracellular fluid (ECF) (third-space phenomenon), weight loss has no relevance.

Clinical evidence is based on alterations in appearance, behavior, pulse, blood pressure (BP), and urine output.

Laboratory evidence is based on changes in the hematocrit (HCT), total serum solids (TSS), blood urea nitrogen (BUN), and urine specific gravity. Dehydration can be described as mild, to moderate, to severe. For purposes of treatment this is related to weight loss as follows:

1. Mild dehydration, 5% weight loss
2. Moderate dehydration, 10% weight loss
3. Severe dehydration, 15% weight loss

When the weight loss is unknown, clinical and laboratory evidence can be used to estimate the degree of dehydration (Table 1-2).

NOTE: To be quantitatively useful measurements, weight loss, HCT, and TSS require knowledge of the normal values immediately before the onset of the dehydration problem.

Type Of Dehydration

The type of dehydration will be described as isotonic, hypertonic, or hypotonic based on the tonicity of the body fluid in the dehydrated patient relative to the normal osmolarity of ECF.

Isotonic dehydration (Fig. 1-2). The *net* electrolyte and water *loss* is *isotonic*, therefore the osmolarity of the remaining ECF is unchanged. Since no correction of ECF osmolarity is required, no redistribution of water between ECF and intracellular fluid (ICF) is necessary. The result is decreased ECF volume. The serum sodium concentration will be within the normal range.

Causes. Causes include any situation in which the composition of the net fluid and electrolyte loss or sequestration is equivalent to ECF.

"Third-space" problems predominate:

1. Crush injuries
2. Burns
3. Peritonitis
4. Volvulus

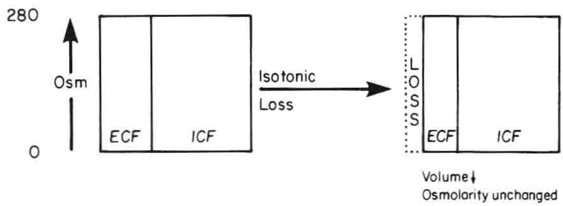

Fig. 1-2. Isotonic dehydration.

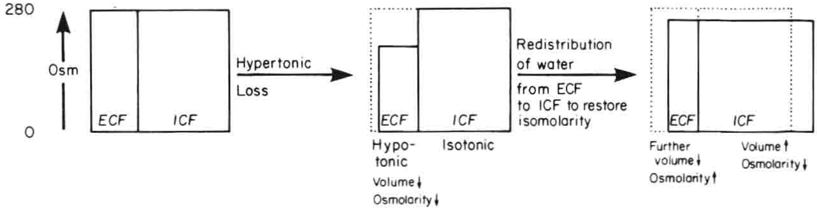

Fig. 1-3. Hypotonic dehydration.

5. Intussusception
6. Low intestinal obstruction

Treatment. The principal aim of treatment is restoration of the ECF volume. Use a balanced salt solution, e.g., lactated Ringer's solution (see Table 1-1).

Hypotonic dehydration (Fig. 1-3). The net electrolyte and water *loss* is *hypertonic*. The osmolarity of the remaining ECF is therefore decreased. Water will then be redistributed between ECF and ICF:

ECF - Water → ↑ Osmolarity of ECF
ICF + Water → ↓ Osmolarity of ECF

1. The decrease in ECF volume is greater than the volume of the water lost because of the redistribution of ECF water to ICF space.
2. The additional decrease in ECF volume is proportionate to the increase in ICF volume.
3. The serum sodium concentration will be 130 mEq/L or less.

Causes. Hypotonic dehydration results when the net fluid and electrolyte loss by the patient is hypertonic to the fluids received by the patient. The fluid loss itself can be hypotonic as in diarrhea or isotonic as in the burned patient.

Treatment. The ECF deficit is managed first by administration of a balanced salt solution such as Ringer's lactate (see Table 1-1). Subsequent repair treatment is aimed at correction of the sodium deficit. The total deficit is calculated on the basis of total body water. For example: A serum sodium of 125 mEq/L represents a deficiency of 10 mEq sodium/L body water. If it were possible to correct this deficit without providing any additional water, we would convert the patient from

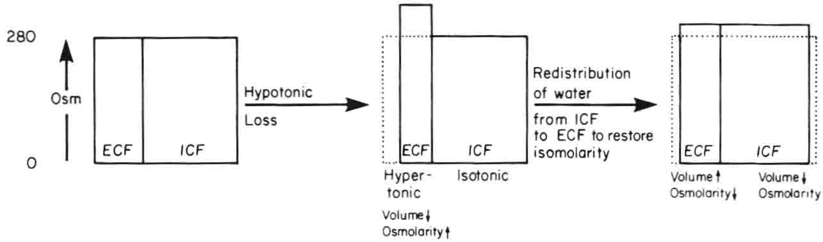

Fig. 1-4. Hypertonic dehydration.

hypotonic to isotonic dehydration. Continued administration of a balanced salt solution will expand the ECF and correct the sodium deficit. Normal sodium chloride can also be used. It will expand the ECF and correct the deficit more quickly.

Hypertonic dehydration (Fig. 1-4). The *net* electrolyte and water *loss* is *hypotonic*. Osmolarity of the remaining ECF is increased:

Water shifts from ICF to ECF → ↓Osmolarity of ECF
↑Osmolarity of ICF

The ECF volume is increased in proportion to the decrease in ICF volume. The net effect is that both ECF and ICF volumes are decreased, but the ECF decrease is moderated by the water shift from the ICF.

The serum sodium concentration is 150 mEq/L or greater.

Causes. Although any situation involving hypotonic fluid loss, e.g., diarrhea, can result in hypertonic dehydration, the severe problem is usually caused by the administration of excess electrolytes to the patient with isotonic or hypotonic loss.

Treatment. The ECF volume is not decreased as much in these patients when compared to the ECF volume in patients with isotonic or hypotonic dehydration. Resuscitation by ECF volume repletion therefore is seldom required. Hypotonic electrolyte solutions are used for correction because the electrolyte requirement is low. These fluids must be administered slowly and evenly to avoid acute swelling of the brain. Follow the BP closely. If BP increases, further slowing of the fluid delivery is indicated.

The terms "isonatremic," "hyponatremic," or "hypernatremic" could be substituted for isotonic, hypotonic, or hypertonic dehydration, especially as the important criterion for the various types is the serum sodium concentration, and basically sodium is the ion responsible for the osmolarity of the ECF. History is extremely important in the evaluation of the dehydrated patient. Reconstruction of the probable losses and type of intake can help in the assessment of the type and degree of dehydration.

Basic in all three types of dehydration is ECF volume depletion. Note that for the degree of dehydration based on weight, the ECF volume is significantly less for the patient with hypotonic (hyponatremic) dehydration and greater for the patient with hypertonic (hypernatremic) dehydration. Dehydration in the surgical patient is usually isotonic or hypotonic and ECF deficit is the first concern. If the state of dehydration is assessed as severe, initial repair is *resuscitation:*

1. Use balanced salt solution or normal sodium chloride solution.
2. Start with ¼ of the calculated maintenance volume (375 cc/m^2) and administer it over a 30 to 45 minute time span.
3. Repeat if there is no, or poor, response by the patient.
4. Follow with 0.5 N sodium chloride solution at 3,000 cc/m^2/24 hr or Ringer's lactate solution at 3,000 cc/m^2/24 hr.

The repair fluid will also cover the regular maintenance requirements during the first 24 hours. This should be an adequate period of time for correction in most cases of isotonic or hypotonic dehydration. Continued observation of the patient's response is necessary and the rate of delivery and the solution used are changed if indicated.

When a large amount of fluid is administered rapidly, do not use fluids containing glucose. However, remember to include glucose-containing fluids as a part of the calculated fluids. Potassium can be added to fluids when urine output is established (20 to 30 mEq/1,000 cc).

Acidosis/Alkalosis

Repair fluids may also be needed to correct disturbances of acidosis/alkalosis. The hydrogen ion activity of body fluids is measured as pH. The pH of body fluid is a function of the ratio of a metabolic component (HCO_3^-) to a respiratory component ($PaCO_2$).

The pH of body fluid is altered as a result of metabolic and/or respiratory changes as follows:

	pH	HCO_3^-	$PaCO_2$
Metabolic acidosis	↓	↓	↓
Respiratory acidosis	↓	↑	↑
Metabolic alkalosis	↑	↑	↑
Respiratory alkalosis	↑	↓	↓

NOTE: HCO_3^- and $PaCO_2$ change in same direction. (1) For metabolic acidosis or alkalosis, pH, HCO_3^-, and $PaCO_2$ change in same direction. (2) For respiratory acidosis or alkalosis, HCO_3^- and $PaCO_2$ change opposite to pH. (3) For metabolic acidosis and metabolic alkalosis the primary change is in the metabolic component or HCO_3^-. (4) For respiratory acidosis and respiratory alkalosis the primary change is the respiratory component or $PaCO_2$.

Metabolic acidosis results from a decrease in HCO_3^- and can be caused by:

1. A *loss of HCO_3^-*, e.g., GI loss in short-gut syndrome
2. The *addition of strong acid*, with utilization of HCO_3^- as buffer, e.g., lactic acid accumulation secondary to ischemic hypoxia in volvulus

The mechanisms of defense are:

1. *Buffers* to remove H^+, e.g., hemoglobin, phosphate
2. *Respiratory*--by hyperventilation to decrease $PaCO_2$, thereby decreasing change in ratio of HCO_3^- to $PaCO_2$
3. *Renal*--by excretion of acid (primarily as NH_4^+) and generation of HCO_3^- to replace that lost

Respiratory acidosis results from an increase in $PaCO_2$ as a result of alveolar hypoventilation. Defense mechanisms are aimed at increasing HCO_3^- by buffers and renal generation, and by the renal excretion of acid.

The problems caused by acidosis are primarily hypotension and hypoxia as a result of depressed ventricular function of the heart and decreased peripheral vascular resistance.

Metabolic alkalosis results from an increase in HCO_3^- and can be caused by:

1. The administration of HCO_3^-

2. The loss of H^+ from ECF
3. A loss of ECF and Cl^- in excess of HCO_3^-

The mechanisms of defense are:

1. *Buffers* to supply H^+
2. *Respiratory*--by alveolar hypoventilation, resulting in an increase in $PaCO_2$, thereby decreasing the change in the ratio of HCO_3^- to $PaCO_2$
3. *Renal* excretion of HCO_3^-; note that renal excretion of HCO_3^- is modified in the following situations:
   a. *When there is ECF deficit*, ECF volume is protected by Na reabsorption (1) in proximal tubule with HCO_3^- as the accompanying anion and (2) in the distal tubule in exchange for H^+, generating HCO_3^-
   b. *When there is K^+ depletion*, renal HCO_3^- threshold is increased, with shift of H^+ into renal tubule cell, followed by its secretion; this accounts for paradox of acid urine associated with alkaline blood
   c. *When there is hypercapnea*, increase in $PaCO_2$ causes an increase in renal HCO_3^- threshold

Respiratory alkalosis results from a decrease in $PaCO_2$ and is caused by hyperventilation.

The mechanisms of defense are:

1. *Buffers* which supply H^+ and thereby decrease HCO_3^-
2. *Renal*--decrease in generation of HCO_3^- and decrease in net acid excretion and retention of H^+

The problems are:

1. Neuromuscular irritability, e.g., tetany and hyperactive reflexes
2. Decrease in ionized calcium

In acid-base disorders, the treatment of the underlying disease is of primary importance. The type and quantity of repair solution are based primarily upon the degree and type of dehydration present. The preservation of volume and osmolarity of body fluids predominates over the preservation of neutrality. Correction of volume and osmolarity problems facilitates the patient's own regulatory mechanisms to correct the acid-base disorder. Appropriate treatment of underlying problems such as ischemia, hypoxia, or pyloric obstruction must also be accomplished or the regulatory mechanisms will eventually be ineffective. However, when function is severely impaired by pH, the acid-base disorder should be treated directly. In general, acidosis is the only problem requiring direct treatment. $NaHCO_3$ solution is