FLUID AND ELECTROLYTE THERAPY IN SURGERY: A RATIONAL APPROACH

By Siew-Chey Ong, M.D. & Prof. L. F. Tinckler, M.D., CH.M., F.R.C.S.

(Department of Surgery, University of Singapore)

The advance in the knowledge of body fluids and electrolytes in the last two decades has served both to aid and confuse surgeons to no small extent. It is often difficult to evaluate the relevance of the multitudinous experimental and clinical facts and to apply them appropriately in practice. Methods of fluid and electrolyte management are consequently highly variable. There are those who diligently calculate every milliequivalent of an electrolyte and every cubic centimeter of water. Others follow a single rule for all cases. Still others just "give a few bottles." In most cases, our treatment owes its success to the immense capacity of the body to make adjustments. One may therefore come to regard knowledge in fluid and electrolyte therapy as convenient but not essential. Admittedly, derangement of fluid and electrolytes is seldom assumed to be the immediate cause of death, but it all too often contributes to a moribund state. The slow post-operative recovery beset with complications in some cases, and the heavy morbidity and mortality in poor surgical risks, should not always be accepted as inevitable.

THE POST-OPERATIVE PATIENT

Fluid and electrolyte therapy in the post-operative patient is a subject of much controversy. Some routinely give salt in the early post-operative period while others condemn the practice. The fallacy of setting an inflexible rule in this respect is clear. One should not regard the post-operative period as a completely new beginning and to neglect correction of antecedent deficits of fluid and electrolytes. The patient may come to operation in uncorrected fluid and electrolyte imbalance, not by choice but by necessity, and should not be managed as a routine case in the post-operative period. In the absence of antecedent deficits, it is true that it matters little whether or not salt is given in the early post-operative period. In most cases, resumption of oral intake soon corrects all deficits. The matter assumes practical importance when the patient is to rely on intravenous fluid for a long time and when there are complications.

What is the rationale of restricting salt and water in the early post-operative period? Surgeons are well aware of the effects of adrenocortical steroids (1,2,3,4,5,6) and antidiuretic hormone (7,8,9,10,11,12,13) which play an augmented role after a major operation. These hormones exert their influence on fluid and electrolyte balance mainly through their actions on the kidney. It is believed by most that as the glomerular filtrate enters the proximal tubules of the kidney, there is active reabsorption of solutes, including sodium, potassium and chloride. This results in hypotonicity of the filtrate. The relative excess of water is then reabsorbed by passive diffusion across the cellular membranes, so that the net outcome is an isosmotic reabsorption of solutes and water. Reabsorption of salt and water in the proximal tubules is believed to be independent of the influence of any hormone. As the filtrate reaches the distal tubules, sodium chloride is reabsorbed under the influence of aldosterone (14,15). For every sodium ion reabsorbed, a potassium ion or a hydrogen ion is secreted into the filtrate in exchange (16). Following reabsorption of solutes, there is again passive diffusion of water from the tubular lumen across the cellular membranes. However, for the cellular membranes here to become freely permeable to water, the action of antidiuretic hormone is required (17,18). In the absence of antidiuretic hormone, reabsorption of water in the distal tubules is impaired. When the urine reaches the collecting tubules, water is further reabsorbed by passive diffusion due to great hypertonicity of the surrounding medullary interstitial fluid. There is reason to believe that the action of antidiuretic hormone is quite important here (17, 18). The mechanism of formation of hypertonic interstitial fluid surrounding the collecting tubules is still not fully explained.

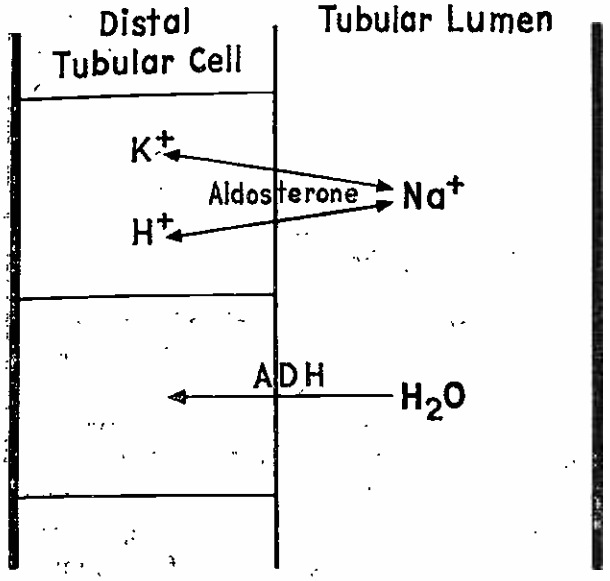

Fig. 1. Hormonal actions in distal renal tubules: Aldosterone promotes reabsorption of sodium. Antidiuretic hormone (ADH) renders cellular membranes freely permeable to water.

After a major operation, one would, therefore, expect the patient to retain salt and water. For this reason, many surgeons give no salt and a limited amount of water in the early post-operative period. Although the physiologic basis for this practice appears to be sound, there is also considerable evidence to the contrary. While the increase in the release of aldosterone after a major operation has remained beyond dispute, the importance of the role of the hormone in post-operative salt retention has been questioned in recent years. It has been shown that the characteristic post-operative decreased salt excretion is not altered in the adrenalectomized man or animal on constant doses of steroid replacement (19,20,21). It has now been suggested that post-operative salt retention and antidiuresis are phenomena secondary to deficit in extracellular fluid incident to operative trauma. Shires (22) in 1962 showed that patients who were given no salt during the operative day showed a marked positive sodium balance when presented with a salt load the first post-operative day and that patients who were given replacement of estimated deficit of salt during the operation showed little tendency to retain salt the first post-operative day when similarly presented with a salt load. The inference is that operative loss of extracellular fluid is the cause of increased post-operative aldosterone output and salt retention. The operative deficit of extracellular fluid has been demonstrated by simultaneous measurement of plasma volume, red cell mass, and extracellular fluid volume by means of I^{131} tagged serum albumin, $Chromate^{51}$ tagged red blood cells, and S^{35} sodium sulphate (23). The deficit appears to be related to the degree of operative trauma. The loss of extracellular fluid could not be located as an external loss and is presumed to be largely redistributed to the intracellular compartment. This may also explain the tendency of the chronically hypovolemic patient to become hypotensive in the post-operative period. In view of these experimental facts, it would seem reasonable not to restrict salt and water in the early post-operative period. However, it should be kept in mind that the body must be able to handle the excess of water and salt when redistribution of fluid takes place in the opposite direction several days later.

It is clear that no final word can be said about proper rational post-operative fluid and electrolyte therapy. In our present state of knowledge, it seems possible to adopt a reasonable compromise to the two divergent schools of thought. If there has been a pre-operative deficit, such as in bowel obstruction or peritonitis, it is desirable to administer a liberal amount of salt in the early post-operative period. In the absence of such an antecedent deficit, it is probably unnecessary to administer salt on the operative day. From empirical observations, most patients show no apparent ill effects from a mild hyponatremia. The urine output and gastric suction are measured. In the first post-operative day, normal saline in the amount equal to one-half of the total volume of gastric aspirate and urine output in the previous day is given. The amount of water given should be equal to total urine output and gastric loss and any other abnormal external loss, plus about 1,500 c.c. for sweating and insensible loss in the tropics. This simple method of replacement may also be used for subsequent post-operative days. The sodium concentration of the gastric suction specimen is usually not over 60 mEq./L. The concentration of urine and sweat is quite variable. It should be emphasized that every case should be individualized and that no hard and fast rule can replace good clinical

observation and judgement. Whenever indicated by the clinical picture or laboratory evidence, the amount of salt and water should be appropriately increased or decreased.

The addition of potassium to parenteral fluid therapy is often neglected. It is true that in the absence of an antecedent deficit or unusual loss, it is unnecessary to administer potassium the operative day and the first post-operative day. Destruction of cells and blood transfusions in an operation provide an amount of potassium to the body. However, on the second post-operative day, the negative balance of potassium may be considerable unless there is oliguria. Normally, most of the potassium filtered by glomeruli is reabsorbed by the proximal tubules. In the distal tubules, potassium is secreted while sodium is reabsorbed. The secretion of potassium by the distal tubules is obligatory (24,25,26). In other words, loss of potassium must occur with urine output. The concentration of potassium in gastric suction specimen varies between 10 and 15 mEq./L. The daily requirement of potassium in a patient on gastric suction and with a fair urine output is about 40 mEq. or 3 grams of KCl. Probably more often than realized, severe potassium depletion is the cause of prolonged post-operative ileus and lethargy.

GASTRIC OUTLET OBSTRUCTION

Gastric outlet obstruction is one of the commonest conditions encountered by the general surgeon. In the adult it is usually caused by a peptic ulcer or a pyloric carcinoma. The patient has vomiting of progressive severity for days or weeks and often comes in the hospital in dehydration, azotemia, and profound electrolyte derangement. The condition requires no immediate operation. The surgeon has time to prepare the patient, and there is no justification for not getting him into an acceptable state of hydration and electrolyte balance.

The patient with an obstructing pyloric carcinoma often secretes little or no free acid in the gastric juice. He loses mainly sodium, potassium and chloride by vomiting. It is quite clear that replacement should be in the form of water, sodium, potassium and chloride. The patient with duodenal or pyloric obstruction due to peptic ulcer scarring loses a considerable amount of hydrogen ions. It may not be as apparent that correction can similarly be achieved with water, sodium, potassium and chloride. Gone should be the day when ammonium chloride was advocated as part of the replacement therapy.

When the body is depleted in sodium chloride, there is a demand on the kidney to conserve this substance. The ability of the kidney to perform this function remarkably well is evidenced by the fact that a urine essentially free of sodium can be excreted. Under the influence of aldosterone, there is increased active reabsorption of sodium by the distal renal tubules. For every Na^+ reabsorbed, the tubular cells secrete a K^+ or a H^+ into the filtrate in exchange (16). K^+ and H^+ ions compete with each other for the exchange with Na^+. The competition is probably to a large extent conditioned by the availability of one or the other ion. When there is a great loss of Na^+ from the stomach, the kidney attempts to conserve Na^+ by increasing its reabsorption. Since H^+ is already depleted, the loss of K^+ is relatively great. As the condition progresses, potassium becomes severely depleted from both urinary and gastric loss, and more H^+ ions are secreted into the urine. The patient excretes an acid urine in the face of alkalosis in this paradoxical manner. Moreover, intracellular potassium ions migrate out and are replaced in part by sodium and hydrogen ions. All these factors eventually bring about a state of severe alkalosis.

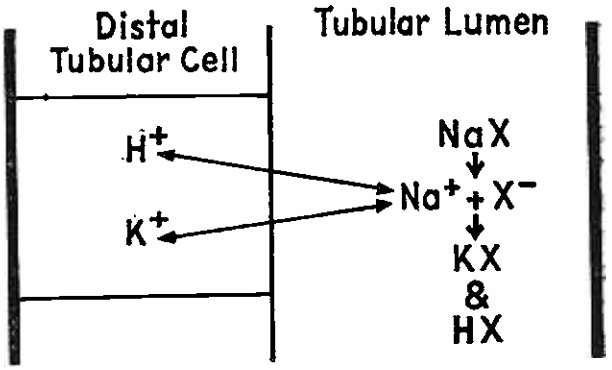

Fig. 2 a.

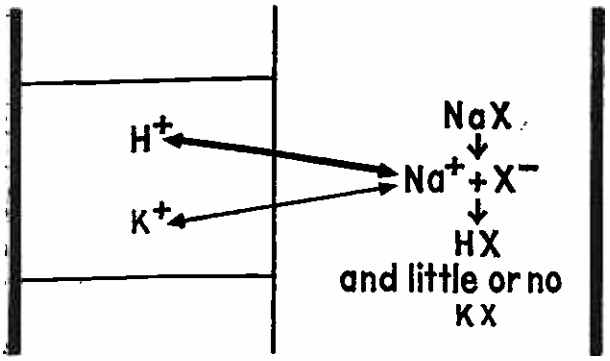

Fig. 2 b.

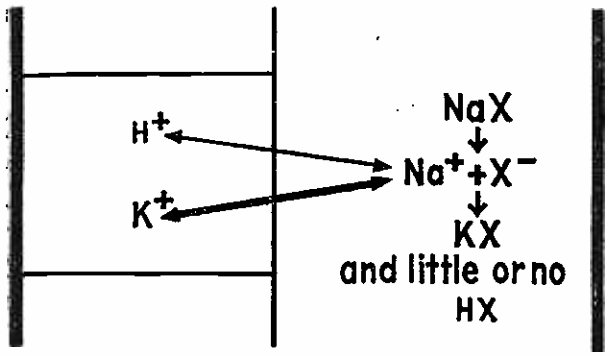

Fig. 2 c.

Figs. 2a, 2b & 2c. Competitive ion exchange in distal renal tubules.

When saline is given to the patient, the body can afford to excrete more sodium in the urine. Consequently, more potassium and hydrogen ions are conserved. There will, however, be a persistent loss of potassium in the urine since its excretion is obligatory. Furthermore, with uncorrected potassium deficit, intracellular K^+ and extracellular Na^+ and H^+ exchanges continue which potentiate extracellular alkalosis and hyponatremia. Therefore, if saline alone is given to the patient, hypokalemic alkalosis cannot be reversed completely. If, however, potassium is included in the replacement therapy, extracellular alkalosis can be effectively corrected as more hydrogen ions are conserved by the distal renal tubules and as intracellular hydrogen and sodium ions return to the extracellular fluid compartment. In practice, it is perhaps advisable to give saline first until a fair urine output is established before potassium is administered.

There is no magic formula for one to make a precise estimation of the amounts of sodium chloride and potassium needed for replacement. However, certain broad principles may guide our management. 1. The patient should not be given non-electrolyte solutions, such as 5% dextrose in water, until correction of electrolyte deficits has been largely achieved. 2. A rough estimate of an appropriate amount of salt needed for replacement may be made on the basis of serum sodium concentration and the weight of the patient. 3. Potassium must be given once a fair urine output is established even if the serum potassium concentration is normal. Potassium being largely intracellular, serum potassium is a poor index of the actual potassium balance.

A rough estimate of NaCl deficit may be made from the serum sodium concentration and the weight of the patient. Usually the total body water makes up some 60–70% of the body weight. In a 60-kg. man, the total body water is at least 60 x 60 or 36 liters. If the serum sodium is 125 mEq./L., to raise the serum sodium concentration to 140 mEq./L. will require 15 mEq. for every liter of body water or a total of 15 x 36 = 540 mEq. of sodium. This amount of sodium is contained in about 3,500 c.c. of normal saline, which has 154 mEq. of sodium per liter. The correction does not have to be accomplished in one single day. It should be recognized that replacement based on this method is only partial, since even if we succeed in bringing the serum sodium concentration up to normal, there is still an absolute deficit of salt and water that is not reflected in initial serum sodium concentration. A second estimation based on a repeat serum sodium determination is necessary if replacement is to be more approximate.

A common error in estimating sodium deficit is to take only the extracellular water into consideration. The cellular membrane is freely permeable to water. Osmotic equilibrium must be maintained between the extracellular fluid and the intracellular fluid. As extracellular salt becomes depleted, the relative excess of water enters the cells to re-establish osmotic equilibrium. As salt is added into the extracellular compartment, intracellular water returns to the extracellular compartment to re-establish osmotic uniformity. Thus, although the administered sodium salt is mostly confined to the extracellular compartment, its osmotic effect is distributed throughout the total body water. It follows

that in estimating sodium deficit one must take the total body water into consideration.

It is less satisfactory to determine the extent of potassium deficit from serum potassium concentration. Normal serum potassium levels may coexist with significant cell depletion. However, a drop in the serum potassium level usually indicates a depletion of considerable magnitude. Schreiber and Burnell (27) claim that a depletion of 100–200 mEq. is required to lower the serum potassium level by 1 mEq./L. if the initial serum concentration is greater than 3 mEq./L., and that an additional deficit of 200–400 mEq. is required to lower the level by 1 mEq./L. below 3 mEq./L. It has also been shown by Huth and Squires (28) on the basis of balance studies that a serum potassium concentration of 3 mEq./L. means a deficit of 300–400 mEq. In practice, rapid correction should be avoided. It is rarely necessary to administer more than 120 mEq. of potassium a day. The daily maintenance requirement in gastric outlet obstruction is about 40 mEq. or 3 grams of potassium chloride.

IATROGENIC WATER INTOXICATION

Iatrogenic water intoxication occurs when a patient incapable of diuresis is acutely overloaded with a large amount of non-electrolyte solutions, such as 5% dextrose in water. It may be seen in an infant with Hirschprung's disease given repeated tap water enema. More often it occurs in a post-operative patient given a large amount of water without salt.

In water intoxication, there is profound hypotonicity of the body fluids. The blood-brain barrier is freely permeable to water but slowly so to sodium. When the extracellular fluid tonicity drops suddenly due to acute dilution, the brain cells rapidly take up water to maintain osmotic uniformity. This leads to production of cerebral edema and increased intracranial pressure. The mild symptoms are irritability and restlessness or weakness and apathy. In severe cases, stupor, coma, convulsions and death may occur. Laboratory studies will reveal low serum sodium concentration, usually below 120 mEq./L., low chloride and low or relatively normal potassium. The actual degree of hyponatremia is not as significant as the rate at which it is induced.

Mild water intoxication requires no specific treatment. It is managed by restricting intake of non-electrolyte solutions and allowing the kidney to excrete the excess of water in due course. Severe water intoxication which threatens life should be actively treated with a hypertonic solution. A suitable solution commonly used is 3% saline, which contains slightly more than three times the concentration of NaCl in normal saline. One should realize that here one is attempting to restore tonicity of body fluids in already over-expanded fluid compartments. The kidney must subsequently excrete the excess of water and salt. The danger of cardiac failure is clear. Theorectically, the amount of salt required to completely restore tonicity is rather prohibitive. For instance, a water-intoxicated 60-kg. man has a serum sodium of 120 mEq./L. Assume, conservatively, that his total body water is 70% of his weight, or 60 x 70 = 42 liters. To raise the serum sodium concentration from 120 mEq./L. to 140 mEq./L. will require 42 x 20 = 840 mEq. of sodium, or roughly 1,600 c.c. of 3% NaCl. Since we aim only at controlling symptoms and rely on the kidney to make the definitive correction, there is no indication to correct the tonicity completely. In practice, therefore, not more than a quarter of the estimated amount of salt is given. A safe rule in treating severe water intoxication in an adult is to give about 300 c.c. of 3% NaCl initially. If symptoms are controlled, further hypertonic salt administration should best be withheld. In place of hypertonic saline therapy, the use of urea has been recommended (29).

POST-TRAUMATIC ACUTE RENAL FAILURE

Acute renal failure may occur as a sequela of multiple traumatic injuries and hypotension of considerable duration. Acute renal tubular necrosis is the usual lesion found. The clinical diagnosis is made when there is severe oliguria or anuria after blood volume and hydration have been restored. One must also exclude direct injuries and obstruction of the urinary tract. The subsequent cause of death is usually congestive heart failure or hyperkalemia. If the patient can be kept alive long enough, there is hope of eventual recovery. Renal tubules regenerate remarkably

well. The turning point toward recovery is indicated by onset of diuresis. The treatment in the critical oliguric phase is centered around preventing overloading of the body with fluid, minimizing the rise of serum potassium, and supplying the most possible calories.

The amount of fluid given a day should be roughly equal to the insensible loss and sweating, plus whatever urine output there is, minus the endogenous water supply. Sweating and insensible loss are difficult to estimate and data for tropical conditions are meager. One may conservatively assume 1,000 c.c. a day as a start. In the course of 24 hours, metabolic oxidative processes in the body yield about 300 c.c. of endogenous water. Therefore, the amount of fluid required a day is around 1,000–300 = 700 c.c. plus urine output. One should be prepared to vary from this rough estimate whenever it is indicated by clinical signs and laboratory studies. Since the loss of salt is minimal, the fluid given should be in glucose water unless otherwise indicated. The operation of Kreb's cycle requires at least 100 grams of carbohydrate a day. It is important to meet this minimum requirement so that gluconeogenesis from fat, which enhances acidosis, can be minimized. To supply 100 grams of glucose in a limited volume of fluid, one has to resort to the use of 10% or 15% solution. If excessive tachypnea results from metabolic acidosis, sodium lactate or bicarbonate may be given for symptomatic relief. No potassium should be given. If the patient can tolerate oral intake, it is desirable to give about 20 grams of protein a day. Protein will undoubtedly give rise to nitrogenous waste products and potassium, but the body will break down at least that amount of its own protein if it is not available from exogenous source. Fat in the diet has both palatable effect and caloric value.

Even with the most careful quantitative estimation of fluid, one cannot be certain of the exactitude of replacement. The patient should be weighed daily. A weight loss of about 0.5 kg. a day is expected. Gain in weight or failure to lose weight means overhydration. Excessive loss in weight indicates insufficient fluid intake. Many adjuvants are now available for treatment of hyperkalemia, such as ion resin exchange, peritoneal lavage, and artificial kidney. There is still no substitute for careful fluid therapy.

In the diuretic phase, it is important not to lag too far in the supply of fluid, sodium, potassium and chloride.

SEVERE DIARRHEA IN ULCERATIVE COLITIS AND PSEUDOMEMBRANOUS ENTEROCOLITIS

The two conditions, ulcerative colitis and pseudomembranous enterocolitis, though rare in this part of the world, do confront the surgeon on occasions. In acute exacerbation of ulcerative colitis, the need for parenteral fluids, in addition to blood, is urgent. The loss of large amounts of water and electrolytes through unremitting diarrhea in a critically ill patient must be aggressively replaced.

Pseudomembranous enterocolitis is a relatively new entity which appeared with the advent of antibiotics. In the full-blown case, a pure culture of staphylococcus aureus (micrococcus pyogenes var. aureus) is obtained from the stool. Experimentally, it has been established by Tan et al (30) that the disease is caused by an overgrowth of staphylococcus aureus in the intestinal tract in the absence of normal intestinal flora. Prolonged administration of wide-spectrum antibiotics, such as tetracycline, to which the staphylococcus is resistant but to which normal intestinal bacteria are sensitive, in a patient who has undergone sulfonamide bowel preparation, may induce the disease. Diarrhea is characteristically severe. It is possible to lose as much as 14 liters of fluid a day. Shock from dehydration and hypovolemia is the usual mechanism of death.

Parenteral fluid replacement in severe diarrhea is quite simple. The fluid lost, for practical purposes, can be assumed to have the same concentrations of electrolytes as serum. The loss can be adequately replaced volume per volume with normal saline containing 5 mEq./L. of potassium. However, the difficulty is with determination of the magnitude of loss. A careful record must be kept of the measured or estimated loss through diarrhea. Frequent weighing of the patient is of help. The importance of careful clinical

evaluation of the state of hydration of the patient cannot be over-emphasized.

INTESTINAL FISTULAE

Fluid and electrolyte management in intestinal fistulae is not complicated. The fluid lost should be collected and measured. The most precise method of replacement is to actually determine the concentrations of electrolytes in the fluid and replace it accordingly. It is seldom necessary to resort to such a method since the usual electrolyte concentrations of gastro-intestinal secretions are known:—

TABLE I
Gastrointestinal Secretions (31)

	Na (mEq./L.)	K (mEq./L.)	Cl (mEq./L.)
Small bowel	111.3	4.6	104.2
Bile	148.9	4.9	100.6
Pancreas	141.1	4.6	76.6
Ileostomy (recent)	129.5	11.2	116.2
Ileostomy (adapted)	46.0	3.0	21.4

The fluid lost may be replaced with normal saline with 5 mEq./L. of potassium. In the case of pure pancreatic fistula, one half of the sodium lost may be replaced with normal saline and one half with sodium lactate or bicarbonate. Any way of re-introducing the fluid lost into the intestinal tract is preferable to parenteral fluid therapy.

A recent ileostomy acts more like an intestinal fistula than an acceptable substitute for terminal outlet of the intestinal tract. The concentrations of electrolytes as well as volume of fluid lost from a recent ileostomy may be considerable, especially in ileostomy dysfunction (Table I). The severe potassium depletion and dehydration that may result will further impede proper function of the ileostomy and recovery of the patient. When oral replacement is not practical, parenteral therapy should be carried out promptly.

INTESTINAL OBSTRUCTION AND PERITONITIS

Fluid and electrolyte derangement in high intestinal obstruction is more severe than in low bowel obstruction. In general, the fluid lost by vomiting and trapped in the bowel closely resembles serum in electrolyte concentrations. Fluid replacement is essentially similar to that for severe diarrhea. The difficulty lies in estimation of quantitative deficit. It seems reasonable to assume that when clinical signs of dehydration appear, the body has lost at least 6% of the body weight in fluid. The fluid in the intestinal tract should be considered as external loss. Experimentally, when intraluminal pressure of intestine in dog is increased beyond 70–80 cm. of water, absorption becomes decreased (32).

In severe generalized peritonitis and in strangulated bowel obstruction, the problem is usually not one of simple deficits of electrolytes and water. There is loss of plasma and blood cells from the circulating blood. The surgeon is familiar with the appearance of the gasping moribund patient mottled with bluish discolorations in severe peritonitis. At this stage, no measure is likely to reverse the hypovolemic and septicemic shock. Correction of severe hypovolemia must be carried out when there is still time.

In restoring the circulating blood volume in severe peritonitis, dextrose water is ineffective. The dextrose administered is soon either utilized or excreted. Water freely penetrates cellular membranes and is in no time distributed to all fluid compartments, including the peritoneal interstitial spaces and peritoneal cavity, and little remains intravascularly. Normal saline is slightly more effective and probably suffices for mild cases. However, the fluid is also lost rapidly to the inflamed peritoneum due to increased capillary permeability. To decisively and effectively restore the circulating blood volume, one should administer blood or plasma in addition to saline.

THE PROBLEM OF NUTRITION

It is perhaps true that starvation of the post-operative patient for one or two days does not affect recovery. However, surgical complications are often unpredictable, and one is always faced with the possibility of having to maintain the patient indefinitely on parenteral fluids. It is important to utilize intravenous fluids also as a means of meeting nutri-

tional requirements other than water and electrolytes. There is evidence that if the post-operative patient is provided with adequate amounts of calories, protein and electrolytes, the characteristic post-operative phase of negative nitrogen balance can be minimized, if not completely abolished (33).

The body requires about 100 grams of carbohydrate a day for the operation of Kreb's cycle. If that minimum requirement is not met, excessive breakdown of fat will occur for conversion to glucose with the production of ketone bodies. The basal caloric requirement of an adult is about 20–25 Cal. per kg. One liter of 5% dextrose in water contains 50 gm. of glucose or 200 Cal. When it is necessary to administer a considerable amount of normal saline, it becomes a problem to meet the basal carbohydrate requirement, not to mention the basal caloric requirement. The problem can be partially solved by the use of 5% dextrose in normal saline instead of pure normal saline. Similarly, 10% dextrose in water can be substituted for 5% dextrose in water if necessary.

When a patient is maintained on intravenous fluids containing only electrolytes and glucose for a long period of time, hypoproteinemia and hypovolemia will result. The severity of hypovolemia is often out of proportion of the extent of weight loss. Even though the hemoglobin level may be normal, an absolute anemia is invariably present. The patient withstands infection and additional surgical trauma poorly. Plasma or protein hydrolysate may be used to counteract the severe negative nitrogen balance, but repeated frequent administration is necessary to bring about an apparent effect. For rapid improvement in the patient's general condition, there is no substitute for blood transfusions.

PARENTERAL SOLUTIONS

Surgical intravenous solutions should be kept as simple as possible. The use of special solutions like Darrow's and "special 3" has its disadvantages. First of all, many who use these solutions do not know or remember their compositions. Secondly, the amounts of electrolytes given are often dictated by the specific compositions of the solutions. In general, the following solutions suffice for practically all surgical conditions:—

TABLE II
Parenteral Solutions

Solution	Na (mEq./L.)	Cl (mEq./L.)	K (mEq./L.)	Cal./L.
Normal Saline	154	154	—	—
3% NaCl	517	517	—	—
5% Dextrose in normal saline	154	154	—	200
5% Dextrose in 0.2% saline	34	34	—	200
5% Dextrose in water	—	—	—	200
10% Dextrose in water	—	—	—	400
1/6 M sodium lactate	167	—	—	—
10% KCl	—	13 mEq./10 c.c.	13 mEq./10 c.c.	

When one is concerned primarily with hydration, normal saline may be used. Otherwise, 5% dextrose in normal saline is preferred for its caloric value. In cirrhosis or hepatitis, 10% dextrose in water may be used instead of 5% dextrose in water for additional calories. The use of 1/6 M sodium lactate is rarely necessary in surgery except for symptomatic relief in metabolic acidosis. 3% saline supplies salt in excess of water from a physiologic point of view. Where it is not advisable to administer a large volume of water in order to give the salt, such as in water intoxication or severe hyponatremia in pyelonephritis, 3% NaCl may be used. 5% dextrose in 0.2% saline is quite suitable for maintenance therapy in pediatric patients since it contains about 3 mEq. of sodium per 100 c.c. of water. In general, for every 100 c.c. of water required by an infant for maintenance, about 3

mEq. of sodium is needed. The corresponding potassium requirement is about 2 mEq. Concentrated potassium solution should be available in the surgical ward. 10% KCl solution, which contains 1 gram of KCl in 10 c.c. or 13 mEq. of potassium in 10 c.c. is suitable for general use. To avoid cardiac arrest, potassium must be given in diluted form. Preferably, not more than 20 mEq. should be given in a pint of solution. A more concentrated solution is likely to cause a burning sensation in the vein.

SUMMARY

A rational approach to the management of fluid and electrolyte problems in surgery is emphasized. Based on known physiological, biochemical, and clinical facts, practical methods of management in various conditions — the post-operative state, gastric outlet obstruction, iatrogenic water intoxication, post-traumatic acute renal failure, severe diarrhea, intestinal fistulæ, intestinal obstruction and peritonitis — are discussed.

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