Reoperative Surgery

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PREFACE

There are many excellently written and finely constructed textbooks of surgery from which the present-day student or surgeon can learn or renew acquaintance with the basic principles, therapeutic indications, and techniques of surgery. These books are directed mainly toward instructing those who encounter surgical lesions of a primary nature. Most of them deal briefly, or not at all, with methods of successfully combating impending failure, overcoming complications, or handling the problems posed by recurrence of disease.

Although techniques have been improved greatly and surgical morbidity and mortality rates have been reduced remarkably within the past few decades, nevertheless, surgeons are all too often disappointed in end results. They are faced frequently with the need to reoperate in order to bring about alleviation of symptoms or to effect a surgical cure. An apparently well-executed initial operation does not always lead to the obliteration of the offending lesion, nor does accurate technique necessarily prevent recurrence. Moreover, surgery, by altering anatomic and physiologic states, often creates new problems that must be met by still further operative intervention. And finally, procedures that seem to be adequate at the time of the initial operation later prove to be inadequate and will require additional surgical intervention.

This book has been compiled as an aid both to the established surgeon and to the surgeon in training. It is meant to be used as a supplement to the standard textbook of surgery. Here, some of the outstanding American surgeons have recorded their methods of handling the recurrences, failures and near failures, and complications of surgery. It is a glowing testimony to surgical ingenuity that so many apparent defeats can now, through reoperation, be turned into victories.

The task of editing and compiling material from many different sources is complicated by the great distances that exist between contributor and editor and by the preoccupation of the contributors with their professional duties. It becomes an even more difficult undertaking when the authors are men of such extraordinary accomplishment as those who have written this book, for their arduous surgical obligations must take precedence over their literary obligations. Despite these factors, this editor has been extremely fortunate and his task has been simplified by the splendid cooperation he has received. For this, he is deeply grateful.

An editor of a surgical text requires much assistance from his publisher. Hundreds of communications must flow back and forth from author to editor to publisher; charts, tables, and illustrations must be prepared and permission for reprinting must be obtained; innumerable changes in manuscript must be made to conform to limitations of space. In all aspects of this complex production my publisher has been most helpful and has gone far beyond a publisher's obligations to an editor.

Robert E. Rothenberg

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PREOPERATIVE PREPARATION AND POSTOPERATIVE CARE

Robert E. Rothenberg and Marvin Roberts

The preparation of a surgical patient for reoperation and his postoperative care are tasks that require a thorough knowledge of intracellular and extracellular metabolism and an understanding of pathophysiologic states. No treatise on surgery, whether it deals with primary or secondary operations, would be complete without a preliminary discussion of the various chemical actions and reactions which take place within the body in response to the trauma of a surgical procedure.

The special problems that develop in a patient who requires a second or third operative procedure demand an even greater insight into the normal and abnormal reactions attendant upon surgical trauma and the body's ability to recover from it. In this chapter, no attempt will be made to cover the entire field of preoperative preparation and postoperative care. Rather, emphasis will be placed on the special situations created by the need for reoperation, and it will be assumed that all are familiar with the standard measures employed in uncomplicated initial surgical preparation and postoperative care. It will suffice to state that every effort should be made to see that chemical balance and cardiac, hepatic, and renal function are maintained, and that measures are taken to ensure wound healing without infection. A balanced state of hydration should be carefully preserved, and an adequate intake of chemicals, calories, protein, and vitamins should be provided. The patient should be afforded respiratory support, especially if thoracic surgery has been performed, and he should receive ample supplies of oxygen and red blood cells if a deficiency exists. Gastrointestinal distention should be prevented by nasogastric tubes or a temporary gastrostomy. Catheters should be placed in the bladder in order to measure the output of urine. Early ambulation and frequent changes of position in bed should be rigorously employed.

It is widely recognized that it is essential for surgical patients to maintain adequate levels of vitamins B, C, and K. Edema of suture lines, poor wound healing, dehiscence, and failure of blood coagulation mechanisms are the penalties of deficiencies in these substances. When a patient has experienced an initial depletion because of a primary operative procedure, it is even more important to supply adequate amounts of these vitamins prior to and following a second operation. The interval between operations is often characterized by poor alimentation and vitamin absorption so that parenteral administration becomes necessary to supply sufficient amounts of these vitamins.

The use of intravenous catheters may prove to be especially helpful to the patient who has undergone previous surgical intervention and who has experienced repeated venipunctures, or in whom prolonged parenteral feedings are anticipated. Their extended use in the veins of the extremities often leads to phlebitis, and for this reason their installation into large veins such as the vena cava has been advocated. Some surgeons advise the use of small amounts of heparin or steroids to forestall the occurrence of phlebitis.

Frequently, the patient who has undergone a secondary surgical procedure will require the prolonged use of nasogastric intubation. These tubes may provoke esophagitis and pneumonitis, especially in elderly persons and in those with underlying chronic pulmonary disease. The use

of temporary gastrostomy drainage or feeding should be considered in all patients upon whom several operative procedures are contemplated or in whom postoperative distention can be predicted. The care of a gastrostomy tube differs from that of a nasogastric tube in that suction is not required.

It is well known that respiratory acidosis is not an uncommon finding during anesthesia and the immediate postoperative period. This response is due to inadequate ventilatory exchange. The acidosis must be overcome by increasing respiratory ventilation and establishing adequate pulmonary exchange of oxygen. This is best accomplished by tracheal suction, early ambulation, and the administration of bronchodilatory drugs. It must not be forgotten that acute respiratory acidosis may damage cellular life and myocardial function as enzymatic oxidations do not occur in an acid environment. Ventricular fibrillation may occasionally occur with severe respiratory acidosis.

Patients with chronic pulmonary disease such as emphysema, bronchiectasis, or pulmonary fibrosis may present a picture of chronic respiratory acidosis. In these patients, the respiratory response to elevated carbon dioxide levels is occasionally lost and the respiratory stimulus appears to come from hypoxia. It may be dangerous to give these patients oxygen.

Secretional ventilatory obstruction, which is one of the indications for tracheostomy, is sometimes encountered in patients unwilling to cough or in those who have lost the cough reflex.

The normal pattern of response to surgical procedures has been ably described by Moore and Ball. Essentially, the response consists of:

1. A rise in temperature and pulse rate and a drop in circulating eosinophils.
2. A period of starvation and caloric deficiency.
3. A transient physiologic decrease in urine excretion.
4. Loss of nitrogen from the body for 3 to 7 days by the urinary route (the serum protein concentration usually remains normal during these changes).
5. Loss of potassium for 2 to 5 days followed by potassium retention.
6. Decreased urinary excretion of sodium for 2 to 5 days followed by sodium diuresis.
7. Loss of weight greater than that accountable by the above changes. (The weight loss is probably due to increased fat utilization.)
8. An acute decrease in plasma calcium and magnesium, probably secondary to the associated elevation of plasma phosphate.

Operations of greater magnitude evoke responses of greater depth and duration.

It is important that these changes be recognized and that all losses and gains be estimated accurately so that one can distinguish between a normal and an abnormal response. The depletion response to surgical trauma assumes greater import when one is dealing with patients who are already depleted by illness, starvation, or previous operative procedures. Their convalescence is much more likely to be complicated if they are depleted to a dangerous level. They will have a greater incidence of phlebothrombosis and wound dehiscence and may show signs of edema, hypoproteinemia, and sodium retention. However, markedly depleted patients will respond better to good postoperative therapy and will utilize the replacement materials better than their well-nourished counterparts.

The surgeon should delay operating upon a depleted patient if proper repletion can be accomplished preoperatively. If this is not possible, every attempt should be made to attain a normal intravascular volume with a normal concentration of proteins, ions, and crystalloids. It is in this specific area that staged operations should be used, thus permitting rehabilitation before the second- or third-stage procedure is carried out. Depleted patients should not be given saline solution injudiciously as they are more prone to develop hypoproteinemia and edema.

Most patients who have been subjected to surgical procedures experience certain physiologic changes which return to normal during the course of their postoperative convalescence. However, the patient who requires reoperation soon after his first procedure will have additional metabolic changes superimposed upon the unresolved physiologic changes brought on by the initial operation. In addition, this patient may have another pathologic condition to confuse the picture. In such cases it is extremely important to interpret physiologic changes accurately so that relatively minor abnormalities or losses do not increase and cause major complications.

Major surgical trauma will produce hyponatremia, hypochloremia, and a decrease in plasma protein, osmotic pressure, and hematocrit in the first 3 to 5 postoperative days. These changes are due primarily to the shift of water and ions

between cells and the failure to excrete administered water. There is also an increase in the extracellular, sucrose, inulin, or thiosulfate space, with the greatest expansion between the third and fifth postoperative days. In a patient undergoing surgery of the gastrointestinal tract, there is a significant pooling of fluid and ions in the intestinal lumen which cannot be measured by inulin or sucrose. It has been shown that there is either no change or only a slight decrease in total body water; thus the expanded extracellular compartment occurs at the expense of the intracellular space or of intracellular dehydration. Therefore, the patient must not be deluged with water in an attempt to make up urine volume. If this is done, the clinical symptoms may mimic a complication of the initial operation, such as intestinal obstruction or peritonitis.

Abnormalities in Serum Sodium Concentration

Hyponatremia implies a lowered serum sodium concentration. This state can occur in the presence of increased, normal, or decreased total body sodium content. Postoperative hyponatremia can be divided into two distinct categories: (1) dilutional, due to either excessive fluid intake or the internal shift of ions, (2) depletional, associated with losses in the upper gastrointestinal tract, fistulous drainage, diarrhea, or adrenal insufficiency.

There is normally a hyponatremia in the postsurgical period despite an increase in the total extracellular sodium. This has been referred to by Moore as the mild posttraumatic sodium-potassium shift. The phenomenon is explained as a dilutional hypotonicity. Sodium and free water come from the lysis of cells and fat, and the extracellular volume is increased by any sodium-free water that is given in excess of actual requirements. Again, it is important to reiterate that this response is normal and that no treatment is necessary.

Patients who have been chronically ill and have undergone surgical procedures after a prolonged period of starvation or are in an undernourished state usually display more profound hyponatremic changes. Clinically, such a person will be weak, lethargic, and hypotensive. In this situation, there is an exaggeration of the dilutional hypotonicity because of a disorder of water and salt distribution associated with chronic energy deficits. There is also a greater shift of extracellular sodium into the cells. Treatment, in which overhydration and oversalting must be avoided, may consist of the administration of small amounts of concentrated sodium, even if the total body content is normal. Starvation hyponatremia, seen most frequently in those who have lost more than 15 per cent of their body weight, should be treated by a high-caloric diet, given on a gradual basis.

Hyponatremia in the presence of circulatory insufficiency and a contracted extracellular volume must be treated with replacement of sodium in sufficient quantities. One should remember that in the presence of edema, the total body sodium must be either normal or elevated. Hyponatremia in the presence of edema is treated by restriction of water, and only rarely should small amounts of sodium be given.

Hyponatremia may also be seen in water intoxication. Coma, convulsions, disorientation, twitching of the extremities, and hemiparesis, accompanied by a rapidly falling plasma sodium concentration and a rapid weight gain, should alert the clinician to the diagnosis of this condition. Water intoxication may occur within 48 hr after surgical intervention and is encountered mainly in patients who have been given too much water (or other hypotonic solutions). It may take place during a period of posttraumatic antidiuresis, usually with a preexisting sodium chloride deficiency. The patient who exhibits mild symptoms of water overloading should have a limited fluid intake. With severe symptoms, one must judiciously administer hypertonic saline (3 to 5 per cent) or glucose solution or, occasionally, urea or mannitol.

Hypernatremia (over 145 mEq per liter) is much less commonly seen in surgical patients. When it does occur, it indicates that there is too little water for the quantity of solute present. Hypernatremia is due to either a relative water deficit despite normal renal conservation of water or failure to conserve water. A dilute urine in the presence of hypernatremia occurs in two pathologic states:

1. Neurohypophyseal insufficiency with lack of antidiuretic hormone (ADH). (A urine which is hypotonic to serum in the presence of hypernatremia, plus a positive response to exogenous antidiuretic hormone, usually indicates the presence of diabetes insipidus. Most patients with diabetes insipidus, however, will have a normal serum sodium concentration.)

2. A disorder of the renal tubules that prevents them from responding normally to antidiuretic hormone, such as (*a*) potassium depletion, (*b*) hypercalcemia, or (*c*) intrinsic renal disease.

Small volumes of maximally concentrated urine will be found in cases of relative water deficit in spite of normal renal conservation of water. Hypernatremia may be encountered in the following situations:

1. In patients with diabetes insipidus under the stress of enforced restriction of fluid intake.
2. In patients with uncontrolled diabetes mellitus in whom osmotic diuresis results from heavy glycosuria.
3. In comatose patients maintained on a high intake of protein and salt administered by gastric tube. The large load of urea and salt to be excreted may increase the obligatory renal losses of water, thus producing a negative water balance and hypernatremia.
4. In patients with nephrogenic diabetes insipidus, the so-called "water babies," with persistent dehydration and hypernatremia.
5. Mild hypernatremia has been described in association with primary excess of sodium-retaining hormones such as in patients with Cushing's syndrome or hyperaldosteronism.

The treatment of hypernatremia should be directed at the underlying pathologic state. Neurohypophyseal insufficiency must be treated with the antidiuretic hormone; other forms of hypernatremia should be treated by administering sufficient water to dilute the body solutes down to normal. A reduction of sodium chloride and solute intake may aid in decreasing the obligatory water loss.

Abnormalities in sodium concentration occurring during the postoperative period may produce a clinical picture resembling paralytic ileus or intestinal obstruction. Severe hyponatremia may lead to renal abnormalities or, in the presence of hyperkalemia, it will produce myocardial irritability.

Abnormalities in Potassium Concentration

Alteration in the extracellular concentration of potassium, the principal intracellular ion, often produces acute changes in the condition of the postoperative patient and usually requires vigorous treatment. A long-continued slow potassium loss, as in postoperative starvation, depletes the intracellular potassium gradually. This may take place in the presence of a normal plasma potassium concentration. Acute potassium losses, compounded by starvation, predispose to a low plasma concentration of potassium, especially if accompanied by alkalosis due to hypochloremia or the adrenal stimulus of trauma. In a depleted patient, the addition of potassium loss to chloride loss and stress may cause a rapid decrease in plasma potassium concentration. Such a sequence of events may lead to abdominal distention, fever, and profound weakness.

Since potassium enters and leaves the cell more rapidly than nitrogen-containing protein products, acute hypokalemia may develop in poorly regulated postoperative patients. Some of the most frequent causes of postoperative hypokalemia are:

1. Failure to administer potassium to a patient who has been on a restricted diet preoperatively and who has taken frequent cathartics in preparation for surgery of the intestinal tract.
2. Extrarenal losses from the gastrointestinal tract as the result of tube drainage, vomiting, fistulous drainage, or diarrhea. Such losses can diminish the serum potassium. It is well to know that the gastric contents may contain as little as 0.5 mEq per liter of potassium or as high as 40 mEq per liter. Because of diarrheal stools or ileostomy drainage as much as 100 to 150 mEq per day may be lost.
3. Use of drugs such as mercurials, Diuril, or carbonic anhydrase inhibitors.
4. The administration of aldosterone.

The treatment of hypokalemia is, of course, the administration of potassium. If good renal function exists, large amounts of potassium may be given parenterally without any danger. There is some danger, however, if the potassium is given too rapidly. It is advisable not to give more than 40 mEq per liter in intravenous solutions.

The clinical signs of hypokalemia are: (1) weakness and hypotonia of skeletal muscles progressing to frank paralysis; (2) dyspnea with gasping respirations in which the accessory respiratory muscles are involved; (3) cyanosis, which is usually of respiratory but may be of cardiac origin; (4) abdominal distention in the presence of hypotonia of musculature; (5) nausea and vomiting; (6) cardiac enlargement and the appearance of systolic murmurs; (7) increased pulse rate with Corrigan pulse; (8) elevated venous pressure and signs of cardiac failure;

(9) electrocardiographic changes: (*a*) depression of the ST segment, (*b*) lengthening of the Q-T interval, (*c*) depression or inversion of the T wave.

Hyperkalemia occurs only in the presence of renal failure and is accompanied by the following symptoms: (1) listlessness and mental confusion; (2) numbness and tingling of the extremities, with a sense of weakness and heaviness in the legs; (3) cold, gray pallor; (4) peripheral vascular collapse; (5) electrocardiographic changes: (*a*) elevation of the T wave, (*b*) atrial standstill, (*c*) depression of the ST segment, (*d*) increasing spread of QRST, (*e*) biphasic curve.

The successful treatment of hyperkalemia will be dependent upon resumption of renal function. However, certain measures should be taken immediately to forestall a rapid, fatal outcome: (1) administration of potassium-free solutions, (2) administration of glucose-insulin solutions to bring about glycogenesis and to force potassium intracellularly, (3) administration of exchange resins, (4) dialysis with an artificial kidney.

The relationship between potassium and digitalis must be kept in mind in preparing a patient for a second operative procedure. In a digitalized patient, a fall in the body potassium may precipitate digitalis intoxication. Toxic doses of digitalis will also interfere with the disposition of potassium within the body. Finally, the administration of potassium to a patient in congestive heart failure with digitalis toxicity will produce hyperkalemia.

Stored blood should be used cautiously as the plasma potassium level rises as this ion leaves the red blood cells. After 3 to 4 weeks of storage, the potassium concentration may reach lethal levels. This danger is heightened if stored blood is infused into the aorta near the coronary artery inflow. One of the few instances of acute hyperkalemia causing death seen by the authors occurred in a newborn infant when a well-meaning resident in pediatrics flushed an intravenous catheter with a solution containing potassium. In this instance, it is most probable that the rapid infusion of 10 ml of a solution containing potassium caused cardiac arrhythmia and instantaneous death.

A distinct depression of renal function occurs during and following major surgical procedures. Anesthesia, too, will produce a lowering of urinary flow, a decrease in glomerular filtration, a diminished renal blood flow, and decreased tubular reabsorption. These changes are directly related to the depth of anesthesia. In addition, there is a physiologic oliguria which occurs postoperatively in association with a disturbance in water metabolism. This probably involves activity of the antidiuretic hormone.

It is sometimes difficult to ascertain whether the postoperative oliguria is due to the normal obligatory retention of water, or to dehydration or acute tubular necrosis. The patient with normal water retention has a urine of high specific gravity or osmolarity, containing approximately 30 to 50 mEq sodium per liter. In these patients, the total amount of sodium excreted during 24 hr is less than the intake. In prerenal deviation or dehydration, the sodium concentration in the urine is 20 to 30 mEq per liter and there is an increased osmolarity. However, with acute tubular necrosis, the sodium concentration in the urine is 60 to 80 mEq per liter, and in addition, there is a progressively rising blood urea nitrogen. In renal shutdown, the specific gravity and osmolarity of the urine are close to that of the plasma.

Treatment is specific for each one of these conditions. In a patient with acute tubular necrosis, the fluid intake is limited to 400 to 750 ml plus extrarenal losses, whereas in a dehydrated patient, a vigorous attempt would be made to replace all existing deficits. The normal maintenance of fluid requirements, approximately 2,000 to 2,500 ml per day, is all that is necessary to "treat" the normal physiologic response to surgical trauma.

It cannot be emphasized too strongly that reoperation upon a patient with oliguria is fraught with grave danger. Every effort to restore normal kidney function should be made prior to surgical intervention.

One of the most common complications demanding reoperation during the convalescent period is intestinal obstruction. It is almost always associated with loss of fluid and ions through a nasogastric tube, into the lumen of the bowel, or by seepage into the peritoneal cavity.

The clinical picture of intestinal obstruction varies according to the level of the blockage. A high obstruction produces little or no distention but is accompanied by profuse vomiting and rapid onset of toxicity. A low intestinal obstruction produces marked distention and little or late vomiting. The biochemical picture will also vary according to the level of obstruction. Obstruction at the pylorus, because of the associated loss of chloride and potassium, will often lead to an alkalosis. The alkalosis may be pre-

dominantly hypochloremic or hypokalemic, or it may be a mixed type. Simple measurement of the pH of the urine can give vital information about the type of alkalosis present. A relatively acid urine in the face of alkalosis denotes a hypokalemic alkalosis and requires the strenuous use of potassium solutions. An alkaline urine denotes the hypochloremic or mixed alkalosis and necessitates the administration of either saline solution or ammonium chloride solution.

It is important to remember that hyperkalemia is never seen in an alkalemia and that the treatment of any alkalosis must be accompanied by the administration of potassium. Alkalosis will not be corrected without the use of potassium.

As the level of intestinal obstruction descends caudad in the gastrointestinal tract, the amount of base secreted by the intestine is increased whereas the acid ions decrease. Thus, the loss of jejunal or ileal contents either through a nasogastric tube or into the lumen of the bowel tends to produce metabolic acidosis. Such acidosis should be treated with saline solution, 5 per cent glucose in water, and sodium bicarbonate.

Obstruction of the small bowel may also give rise to an entity known as "isotonic dehydration" where the plasma sodium, chlorine, and carbonic acid concentrations may all remain normal whereas the extracellular fluid volume is reduced. Serum levels in such a patient would all be normal, but the diagnosis can be made by perusal of the intake-output balance sheets and by noting the symptoms and clinical signs. The clinical picture is dependent on the degree of extracellular volume deficit. A 2 to 4 per cent body weight loss is accompanied by weakness, apathy, stupor, and anorexia; a 6 per cent body weight loss will produce hypotension, tachycardia, and poor skin and muscle turgor; and a 8 to 12 per cent weight loss will invite shock and coma.

Commercial drug firms have made various polyionic solutions available, labeling them for specific situations, such as "upper GI tract" "lower GI tract." Our experience has been that it is better to use basic solutions, knowing exactly what each liter of solution contains. The solutions are utilized according to the needs of the patient. Keeping in mind the fact that a liter of gastric juice contains approximately 80 to 120 mEq potassium, we would replace such a loss with 5 per cent glucose in water and 5 per cent glucose in saline solution in a ratio of 1:2 or 1:1. This would constitute adequate replacement as 1 liter of saline contains 154 mEq sodium and 154 mEq chloride. It is well to note that 0.9 per cent saline is isotonic but not physiologic for it is hyperchloremic in relation to plasma. Similarly, knowledge of the chemical composition of small bowel, biliary tract, and pancreatic secretions will aid in the proper replacement of chemical loss with solutions such as normal saline, ammonium chloride, sodium bicarbonate, or hypertonic saline.

In estimating fluid losses or degrees of dehydration, it should not be forgotten that obese patients are less able to afford fluid losses than thin patients. The amount of total body water varies inversely with the amount of fat present, thus the old adage "he's fat enough to lose that weight without any effect" is erroneous. The loss of 10 to 12 per cent of total body water will produce grave disturbances of normal physiologic processes while 20 per cent loss may cause death.

Venous bleeding into the gastrointestinal tract, either during surgical procedures or in the postoperative period, will lead to isotonic volume reduction. This reduction in blood volume will bring about a compensatory hemodilution, with replenishment of the plasma volume by intracellular and extracellular water. If the hemorrhage is large, there may be a drop in plasma sodium concentration. The ideal treatment in this situation is transfusion before transcapillary filling has taken place. In emergencies, the use of colloids, such as plasma, dextran, or polyvinyl pyrrolidine, may suffice.

Abnormalities in Calcium Concentration

Although only 1 per cent calcium is found in the extraskeletal tissues, changes in the concentration of this ion may affect the actions of sodium and potassium. There is usually a hypercalcinuria in the postoperative period, with a slight tendency toward a negative balance. However, in the presence of metastatic malignant tumors or hyperparathyroidism, 500 to 1,000 mg calcium may be excreted in the urine, with a marked negative balance. Calcium deficits may also be seen in acute pancreatitis, generalized peritonitis, massive infections of subcutaneous tissues, and as a complication of duodenal, pancreatic, and small intestinal fistulas.

The signs of calcium deficit may be present with a normal serum calcium concentration. This may be seen in patients with pyloric obstruction who develop alkalosis. Alkalosis will decrease the physiologically active fraction of the total serum calcium. The symptoms of calcium deficit will range from a numbness and tingling of the nose, ears, circumoral region, and tips of the fingers and toes, to severe painful muscle spasms. Signs of calcium deficiency are noted within neuromuscular and gastrointestinal systems. There will be exaggerated tendon reflexes, muscle cramps, respiratory stridor, carpopedal spasm, and convulsions. There may also be severe abdominal cramps and rigidity of the abdominal wall.

The hypocalcemia seen in acute pancreatitis is probably due to formation of insoluble calcium compounds along with the onset of alkalosis, because of repeated vomiting or gastric drainage. Treatment should consist of the administration of calcium salts, potassium chloride, and ammonium chloride to correct the metabolic alkalosis.

Hypocalcemia may be a cause of lack of response to volume repletion in shock. The administration of 1.0 gm calcium gluconate, repeated once, will usually suffice to indicate whether or not calcium deficiency is an important component of the shock picture.

Hypercalcemia is most frequently seen in patients with neoplastic disease, with or without metastatic osseous involvement. The clinical manifestations of this abnormality are: (1) drowsiness, lethargy, mental confusion, muscular weakness, and coma; (2) anorexia, nausea and vomiting leading to water and electrolyte losses, and constipation; (3) polydipsia, polyuria with further loss of water and electrolytes, albuminuria, and renal failure; (4) tachycardia and cardiac rhythm disturbances; (5) potassium depletion with alkalosis.

The treatment of hypercalcemia is to reduce the daily intake of calcium to 200 mg and to increase the urinary output to 1.5 to 2 liters per day. It may also be necessary to discontinue medications such as steroids, nitrogen mustard, estrogens, or androgens. Castration, intensive radiation, or surgical removal of the tumor may also be required to reduce the hypercalcemic state.

A rare cause of acute hypercalcemia, carrying with it a high mortality because it so often is undiagnosed, is hyperparathyroid crisis. The treatment of this condition is immediate surgical intervention to remove the cause of the hyperparathyroidism.

Abnormalities in Magnesium Concentration

It has only been within recent years that upsets in magnesium metabolism have been related to clinical situations. It is now known that magnesium deficiency may lead to tetany and convulsions.

Magnesium deficiency is usually due to malnutrition, or to some condition which prevents the absorption of magnesium, or to an increase in magnesium excretion because of the persistence of an acute infection. Intestinal malabsorption secondary to radiation therapy, intestinal obstruction with drainage of intestinal contents, or severe malnutrition caused by alcoholism can bring on a magnesium deficit.

Elevated magnesium levels are occasionally seen in severely wounded patients, in shock states, and in renal failure. The signs of a hypermagnesium state are respiratory failure and lethargy which progresses into coma.

Abnormalities in Nitrogen Balance

An important consideration in preparing a patient for reoperation is the rebuilding of protein. It is well known that surgical trauma produces a negative nitrogen balance proportional to the severity of the stress. (As much as 15 gm nitrogen may be lost in the urine in the early postoperative period.) Thus, it is essential when contemplating a second-stage procedure of major proportions not to prolong the postoperative starvation period beyond 3 to 4 days. Following this period, every effort at caloric restoration should be made. An untoward response to the initial surgical procedure is characterized by a profound and prolonged post-stress phase of negative nitrogen balance. Failure to regain a positive nitrogen balance will lead to hypoproteinism or generalized protein deficiency.

Patients with postoperative hypoproteinism may develop serious complications. They have a decreased plasma volume with an increased interstitial fluid compartment tending to generalized tissue edema. This may directly affect wound healing or the healing of an anastomosis. Furthermore, the hypoproteinemic patient is more susceptible to shock because of a dimin-

ished tolerance to blood loss. Some other consequences of the hypoprotein state are: (1) lowered antibody production with decreased resistance to infection, (2) liver dysfunction and fatty infiltration, (3) delayed healing of fractures.

Acute changes in the plasma protein concentration are most commonly due to changes in the hydration and salt content of the body. Excessive amounts of water and sodium, particularly during the postsurgical "sodium-saving" period, will lead to a decreased plasma protein concentration. This is particularly true in a previously depleted patient. *Chronic* lowering of the plasma protein concentration is most often caused by liver disease.

In evaluating a patient nutritionally, it must be remembered that a total protein concentration of 6.0 gm per 100 ml of blood does not necessarily signify the absence of a protein deficiency. The absence of certain essential amino acids and a contracted blood or plasma volume can lead to the false conclusion that the patient has adequate protein.

The daily requirements for the normal patient who is in positive protein balance and is at bed rest are 1.0 gm protein per kg and 30 cal per kg body weight. However, after a major operative procedure with its accompanying trauma, 2 to 3 gm protein per kg and 60 to 90 cal per kg are needed.

The nutritional requirements of the patient who must undergo a second or third operation must be evaluated with special care inasmuch as the primary pathologic changes or the preceding surgical procedure may have resulted in exceptional nitrogen losses. For example, nitrogen loss through ileostomy drainage ranges from 0.5 to 2.0 gm per liter; diarrheal stools may cause the loss of 2.0 to 6.0 gm nitrogen per day; purulent discharges from a previous surgical wound may carry away 0.7 to 1.4 gm nitrogen per day; and exudates from extensive burns may cause the loss of anywhere from 3.5 to 5.2 gm nitrogen daily.

It is a most difficult task to replace these nitrogen losses and replenish the deficits in the nutritional store of protein. By far the best method is through oral feedings. Unfortunately, many patients who require reoperation are unable to take oral feedings, and parenteral routes must be employed. Various solutions and substances are available which will supply protein intravenously. Some of these are protein hydrolysates (Amigen), albumin, plasma, and blood.

The human body will utilize most of the infused protein for energy requirements rather than for replenishing plasma proteins and rebuilding tissues. The main value of intravenous supplementation through administration of protein hydrolysates lies in preparing the depleted preoperative patient. (For the best utilization, 200 cal per gm nitrogen should be given.)

It should be emphasized that intravenous protein hydrolysates and oral protein are rather poorly utilized and retained during the immediate postoperative period. Even huge quantities of protein are unable to reverse a negative nitrogen balance during this period even though the degree of negativity can be lessened.

Patients who have recently undergone major operative procedures, who have sustained multiple injuries, who have severe postoperative complications, or who enter the hospital in a state of protein depletion will require inordinate amounts of protein. In addition, they may have sodium and potassium deficits and vitamin B and C deficiencies which need to be rectified. As Cannon has pointed out, deficiencies in sodium, potassium, and vitamins B and C may lead to significant alterations in protein and nitrogen metabolism.

Protein hydrolysates are the easiest, most economic, and possibly the best means of administering protein intravenously. For example, 1.0 gm of protein hydrolysate is equivalent to 0.75 gm of protein, or 1,000 ml of 5 per cent protein hydrolysate is equivalent to 6.0 gm of nitrogen. Reactions to their use are uncommon and are usually related to the rate of administration. The amino acids in the hydrolysate appear to be metabolized in the liver in a manner similar to that of amino acids which have reached the liver by way of the portal vein following digestion. Approximately 11 per cent of the infused amino acids and peptides are excreted unchanged in the urine within 48 hr, but most of this loss is the peptide fraction which is poorly utilized. It is important to realize that parenterally administered proteins will not be used with maximal efficiency unless calories are also supplied. For this reason, calories should be provided by giving solutions containing 5 per cent glucose, fat emulsions, or alcohol.

Whole blood is a poor source of utilizable protein although it contains plasma proteins and hemoglobin; 500 ml of whole blood contains 3.1 gm nitrogen in the plasma protein fraction and 10.0 gm nitrogen in the cells. The life of red blood cells is approximately 100 to 120 days, and the nitrogen from the hemoglobin is not free for

use until the red blood cells are broken down. In patients with anemia, the administration of whole blood will increase the total red blood cell mass and will spare protein from the body store by providing preformed hemoglobin.

Whole blood is also a poor source of calories (87 cal per 500 ml) and is extremely expensive if used for that purpose.

Before using whole blood as a means of treating a patient who is in a depleted state, one should give careful consideration to the hazards of its employment. Serum hepatitis will ensue in 3 per cent of patients who have been given transfusions, and the mortality from hepatitis is approximately 1 per cent. The chances of this complication are directly proportional to the number of units given. Post-transfusion febrile reactions, probably due to leukocyte agglutinins in the recipient, are not uncommon and may undo the benefits that had been expected from giving the blood. It must be kept in mind, too, that allergic reactions occur in 1 to 2 per cent of all patients who receive blood. A rare but catastrophic complication is blood group incompatibility. Finally, citrate toxicity may ensue when large quantities of blood are given. This condition should be suspected in the presence of liver disease, chronic renal insufficiency, or hypothermia. An increased serum citrate may result in cardiac and circulatory depression with hypotension, narrowed pulse pressure, and electrocardiographic changes of hypocalcemia.

Human serum albumin is available as a 25 per cent solution and is a valuable tool in the specific treatment of hypoalbuminemia. This solution contains one-seventh as much sodium as the osmotically equivalent volume of citrated plasma. It can be given with sodium chloride to correct this deficit and thus becomes a useful adjuvant in the treatment of patients with hypoproteinemia and edema. The chief therapeutic use of albumin is the emergency treatment of shock due to surgical procedures, hemorrhage, or infection. It is an excellent method of treating shock associated with hemoconcentration, such as occurs in burns.

Serum albumin is always given intravenously and may be diluted with saline, dextrose, or sodium lactate. It can be mixed with plasma or blood. Reactions from serum albumin are rare, but overloading should be avoided.

Human serum albumin is not a good source of protein for routine nutritional use as 30 per cent is retained in the plasma whereas most of the remainder passes into the intracellular fluid. It therefore does not enter directly into cellular metabolism. It is also a poor source of calories (50 cal per 250 ml) and is expensive. The albumin remains in the vascular system for a long period of time, but its availability for use in metabolism is slow.

Plasma has been widely used in the emergency treatment of surgical shock and in the treatment of extensive burns. Although it has proved effective in these situations, there are certain disadvantages to its use in patients who have undergone reoperation: (1) It is expensive. (2) It is a poor source of calories (60 cal per 250 ml). (3) Its protein is not immediately available for nutritional purposes.

The danger of serum hepatitis following the use of plasma has disappeared since the use of pooled stored plasma.

Fibrinogen is supplied as dried powder. It should be used specifically in cases of hypofibrinogenemia or afibrinogenemia following obstetric complications or pulmonary surgery where manipulation of the lung may release thromboplastic substances into the blood stream. The addition of 200 ml sterile water to a vial of fibrinogen gives a solution isotonic with plasma. There are no serious immediate or delayed reactions to its use, although there is a slight possibility that serum hepatitis may ensue.

The solutions that are used most efficaciously to provide parenteral calories are: (1) Dextrose and fructose, either in 5 or 10 per cent solution. Each gram of carbohydrate provides 4 cal. (2) Fat emulsions provide 9 cal per gm. Their prolonged use for more than 14 days may produce disturbed liver function and gastrointestinal bleeding. (3) Alcohol.

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