

THE POSTOPERATIVE PATIENT AND HIS FLUID AND ELECTROLYTE REQUIREMENTS

M. T. JENKINS, A. H. GIESECKE AND E. R. JOHNSON

Proper fluid and electrolyte management during the postoperative and convalescent periods will depend to a great extent upon the patient's pre-operative status and preparation, the magnitude of surgical trauma, and the fluids administered intra-operatively. Consequently, we will first refer to some preoperative complications and then outline our intraoperative regimen, before discussing post-operative fluids.

In the preoperative period, an intensive and specific regimen may be proper for patients with some medical problems, and yet may be improper if continued up to the time of anaesthesia and operation. Examples include the hypertensive patient who is undergoing vigorous therapy with antihypertensive drugs and diuretic agents while maintained on a low-salt diet. Such a patient will probably have a severe hyponatraemia and hypokalaemia which, unless correction has been started prior to anaesthesia, will lead to resistant hypotension.

Another who is likely to develop severe hypotension under anaesthesia is the patient whose congestive heart failure has been corrected by complete digitalization and who has been rendered oedema-free by the use of diuretic preparations, causing a relative hypovolaemia.

A special problem exists in the administration of fluids to patients either in compensated or uncompensated heart failure, but still fluids cannot be withheld without endangering renal function and without affecting the microcirculation adversely.

Perhaps less spectacular, but more frequent in occurrence, are the immediate sequelae of anaesthetizing the hypoglycaemic patient, the one who has been on a "nothing-by-mouth schedule" for many hours, or who has been on gastric drainage for several days. Starvation depression of glycogen may leave the patient's liver susceptible to toxins, including anaesthetic agents, unless corrective fluid therapy is begun after surgery (Jenkins, 1970).

During the anaesthetic and operative period, undesired responses may be achieved by the infusion of too much free water, too much salt, unnecessary albumin, plasma derived from outdated blood and containing a high potassium content, osmotic diuretics, or by insufficient volume replacement.

We acknowledge the obvious fact that, throughout the world, many differing routines for administration of intraoperative fluids are followed for patients having a wide variety of diseases and surgical procedures. These routines may range from no fluids at all to blood only, and some routines include the use of albumin or mannitol or dextran on a definitely timed basis. Under any regimen a majority of patients survive, some because of fluid administration and others despite it.

Since no one regimen for fluid administration will apply to all patients with varying physical status and disease processes, we offer here our approaches to intraoperative (Jenkins and Giesecke, 1974) and postoperative therapy, applicable to adult patients having no pre-existing fluid or electrolyte imbalance. Requirements during the first 24 hr after operation will possibly vary if these guidelines for intraoperative fluids are not followed.

As a central theme of our precepts we feel that homeostatic mechanisms in the anaesthetized patient are best maintained if fluid administration helps to preserve normal renal function while replacing extracellular fluid (ECF) translocated from the dynamic pool.

BASIC PRECEPTS FOR INTRAOPERATIVE FLUID THERAPY

(A) We begin with 5% dextrose in water (D5W) up to 500 ml, and continue with 5% dextrose in balanced salt solutions (D5BS) in the following procedures:

(1) *Intra-abdominal and hip operations.* 12–15 ml/kg body weight during the first hour, and 6–10 ml/kg/hr for the next 2 hr, varied as indicated by the degree of operative manipulation (trauma),

M. T. JENKINS, M.D.; A. H. GIESECKE, JR., M.D.; E. R. JOHNSON, M.D.; The Department of Anesthesiology, The University of Texas Southwestern Medical School, Dallas, Texas, U.S.A.

arterial pressure, pulse, urine output and, in certain cases, central venous pressure or pulmonary wedge pressure. For operations beyond 3 hr, we continue with a balanced salt solution without glucose at a rate to assure urinary output of 50–100 ml/hr.

(2) *Intrathoracic (non-cardiac) operations.* 6–10 ml/kg/hr.

(3) *Extremities and major superficial operations.* 6–10 ml/kg/hr, varied after the first hour as indicated by degree of operative manipulation (trauma), arterial pressure, and pulse.

(B) We begin and continue with D5BS in these operations:

(1) *Intracranial procedures.* Balanced salt solutions are administered only in volumes sufficient to keep the venous channel (i.v.) open until the surgeon begins the closure; then begin replacement with balanced salt solutions as indicated by urine output.

(2) *Transurethral prostatic surgery.* 3–6 ml/kg/hr while watching closely for sudden expansion of intravascular volume by operative "washwater" absorbed through the prostatic bed.

(C) We begin and continue with D5W in volumes sufficient to keep the i.v. open (i.e. minimal fluids where operative trauma is limited) in these procedures:

(1) *Microsurgery of the ear and larynx.*

(2) *Most ophthalmic operations.*

(D) We limit the total dextrose administered to a maximum of 125g.

(E) We transfuse with whole blood or its equivalent in blood component therapy when blood loss exceeds 20% of estimated blood volume.

(F) We monitor urine output on all major trauma operations and all predicted to be lengthy procedures.

Comments on These Precepts

D5W: We begin with D5W to replace pure water loss and to reduce renal work. For example, consider the patient who has had no fluid intake for 12 hr or more before arriving in the operating theatre. He is usually thirsty whether or not he has had a belladonna premedication. His thirst results from the slight increase in serum osmolality due to water restriction, to continuing loss of water through the kidneys, and to insensible loss as sweat from the skin and moisture in exhaled air. In the hypothalamus, the osmoreceptors which trigger his thirst by signalling the change in water-solute ratio are the same osmoreceptors which send

impulses to the posterior pituitary gland, causing an increased release of antidiuretic hormone (ADH). As a result of ADH activity, the distal convoluted tubules and the collecting ducts in the kidney become permeable to water so that water moves into the hypertonic interstitium and back into the circulation. Consequently, in losing water, the urine becomes more concentrated. The administration of free water (with glucose) can obviate this ADH response.

Glucose aids in still another way by decreasing renal work requirements. The fasting patient has approximately 900 m-osmole (45g) of non-volatile solute to be excreted in the urine daily, representing breakdown of 160 g of fat and 75 g of muscle protein. Intravenous glucose, up to 125 g, will reduce the non-volatile solute to 200–400 m-osmole (10–20 g), which represents a small load even for the kidney having a limited concentrating ability (Cahill, 1970).

D5BS: We administer 5% dextrose in lactated Ringer's solution (D5LR) at the rate of 10–15 ml/kg body weight for the first hour to patients having intra-abdominal and hip operations. If the operative procedure continues into the second hour, and if there is no undue loss of blood or unusual degree of tissue manipulation and trauma, we slow the fluid administration to 6–10 ml/kg/hr. If the operation continues past 3 hr, lactated Ringer's without dextrose (LR) is substituted for D5LR. Hourly urine output should be monitored in these longer operations, and fluids should be administered at a rate to assure an output of 50–100 ml/hr, or about 1 ml/kg body weight/hr.

For intrathoracic procedures involving the lung only, we begin with D5W and continue with D5LR at the slower rate of 6–10 ml/kg/hr, realizing that higher rates of fluid administration may contribute to the inevitable localized pulmonary oedema in areas of the lung traumatized by the operative procedure. Probably smaller functional deficits of extracellular fluid (ECF) result from intrathoracic operations than from intra-abdominal or hip operations.

The routine for major operations outside body cavities or on the extremities is very similar to that for intrathoracic surgery. Rational use of fluids in any of these circumstances demands good monitoring and good interpretation of information gained from the monitors. Arterial pressure and pulse should always be monitored. Urine output is perhaps the most valuable guide to continued fluid

therapy. Central venous pressure gives us information on the heart, including the manner in which it manages the fluid load, but it is not a direct measure of the adequacy of volume replacement. The patient in congestive heart failure can have an elevated venous pressure even though his ECF volume is deficient. Conversely, in the healthy patient, serious overloads of isotonic salt solutions can be given without significant elevation of the central venous pressure. The overload is more likely to be manifested by interstitial oedema and diuresis.

For intracranial operations, we begin with D5LR administered at a rate just sufficient to keep the vein open, to assure a ready avenue for drug administration or transfusion. If the surgeon wishes to use one of the osmotic diuretic agents to control intracranial pressure, we continue to withhold fluids until the crucial part of the operation has been completed, after which fluid administration is accelerated in an effort to replace that volume which has been lost as urine.

For transurethral resection of the prostate, we prefer to begin with D5LR. Enough irrigating solution may be absorbed from the prostatic venous plexus to dilute the sodium concentration of the plasma to levels as low as 90–120 m-mole/litre. Depending upon the number and size of veins opened by resection and the duration of resection, administration of electrolyte-free solutions by the anaesthetist will only serve to aggravate the hyponatraemia.

Even the heartiest advocate of crystalloid therapy in the operating room will concede that no shift in functional ECF occurs during microsurgery of the ear and larynx, and in the majority of eye procedures. Therefore, minimal fluids should be administered during these procedures.

In a major operation where there is considerable surgical manipulation (trauma) or blood loss exceeding 10% of the circulating volume, changes occur in the patient's physiology leading to oliguria or anuria. These changes are due to salt losses or blood volume losses which stimulate aldosterone secretion with its subsequent effects on the kidney. Therefore, we continue with a balanced salt solution, the composition of which approximates to that of deproteinated serum. The total volume administered depends upon operative time, blood loss, and estimated translocation of ECF due to necessary trauma of tissue manipulation. Translocations represent internal distribution of ECF so

that it no longer enters into the dynamics of circulation. Such losses are essentially of isotonic fluid and are inexactly referred to as "third-space" shifts. Part of the redistribution loss may be into the tissues adjacent to the surgical wound, or it may be translocated into an area where salt tends to be sequestered, such as in the splanchnic bed during intra-abdominal operation, and/or it may represent an isotonic intracellular shift. Regardless of the areas to which redistribution occurs, there is justification for the use of a balanced salt solution during anaesthesia and operation. The first and usually the second litre of balanced salt solution will contain 5% glucose, after which lactated Ringer's solution is given without glucose.

Generally, patients so treated will continue to secrete urine throughout the operative period. This indicates that ADH is not elaborated, and also that there is no stimulus for aldosterone secretion. Maintaining the circulating blood volume with a salt-containing solution (plus blood replacement as indicated, of course) removes the principal stimulus for aldosterone secretion. Consequently, in long operative procedures (and for the trauma patient and other emergency patients), urine output should be monitored as an indication of adequate fluid replacement. Balance studies show that these patients have only a small positive sodium balance when given a salt load after operation. Patients given no salt during a major operation will retain a salt load given after surgery, since this salt is retained (aldosterone effect) to correct for the ECF deficit (translocation) acquired during operation.

For the patient who goes into a state of haemorrhagic shock during operation, we believe that volume replacement is the primary key to its successful management, employing simultaneous administration of whole blood and balanced salt solutions. Blood alone or colloid alone will not correct the deficit of functional extracellular fluid, of which part has moved from the interstitial pool into the vascular system, part has shifted into cells, and part has been sequestered and immobilized in damaged tissues (Jenkins, Giesecke and Shires, 1965).

POSTOPERATIVE FLUID MANAGEMENT

If the surgical patient's extracellular fluid volume has been maintained during operation, his renal function should remain in a normal range with a daily postoperative urinary output of 1,200–2,000 ml.

Until recent years, traditional concepts have blamed anaesthesia for the antidiuresis occurring in the early postoperative period. We feel that if urine output stopped during operation and if the patient is oliguric in the immediate postoperative period, it is because the kidney is protecting a diminished functional ECF volume (internal distributional changes), and it is not because of specific effects of the anaesthetic agent on the kidney (Jenkins, 1970).

General Comments

Orders for postoperative fluids should not be written until the patient is in the recovery room and his fluid status has been assessed. This should include his preoperative evaluation, fluid loss and gain during operation, and vital signs. Initial orders should correct any existing deficit and include maintenance fluids for the rest of the day if these can be determined reasonably. Otherwise, fluids should be ordered 1 litre at a time and a reassessment of the patient made during their administration.

Continued ECF volume depletion may occur due to further loss at the site of operative trauma, such as into intestinal wall or lumen or subjacent to the peritoneum. Such continued translocations are manifested usually as circulatory instability.

Classification of fluid disturbances.

There are four major categories of disturbance of fluid balance applicable to the postoperative patient as well as to his preoperative status: disturbances in volume, concentration, composition, and distribution. Each should be evaluated in assessing a patient's fluid status. All losses and gains of body fluid are directly from the extracellular volume phase. The intracellular fluid does not share immediately in losses involving volume alone, but does share without delay in losses involving a change in concentration or of composition (Baxter and Shires, 1966).

(1) Disturbances in volume are not diagnosed definitively by chemical analyses, but may be revealed by physical signs. Generally, we feel that ECF volume is acceptable if the patient's urinary excretion is 50–100 ml/hr or roughly 1 ml/kg body weight/hr. A moderate ECF deficit causes sleepiness, anorexia, slow responses to questioning, depression of sense of thirst, disinterest in surroundings, orthostatic hypotension, "sticky" skin (diminished elasticity of subcutaneous tissue), weakness, apathy, tachycardia, collapsed superficial

veins, and small pointed tongue with longitudinal furrows. These signs are more pronounced with a severe ECF deficit which results also in stupor, sunken eyeballs, cold extremities, atonic muscles, recumbent hypotension, hypostatic cutaneous lividity, and fall in body temperature.

Laboratory results are not diagnostic of volume changes, although in volume losses they may show an elevation of haemoglobin concentration and also of haematocrit and red blood cell count. Urea may be elevated because of decreased renal blood flow. Urinary sodium and chloride tend to be low or absent unless there is renal disease or other conditions interfering with tubular reabsorption of sodium chloride (Moyer, 1957).

(2) Changes in concentration are measured in the extracellular fluid, using sodium as the principal osmometer. The sodium ions principally determine the tonicity of body fluid compartments, and they account for 90% of the osmotically active particles in the ECF.

Hyponatraemia and hypernatraemia may be diagnosed by clinical examination and also by laboratory tests. Acute hyponatraemia (sodium less than 130 m-mole/litre) is characterized by c.n.s. signs of water intoxication, and by increased intracranial pressure. If not corrected, oliguric renal failure will develop relatively soon.

To treat a moderate hyponatraemia with an associated volume deficit, isotonic sodium chloride may be used initially if there is also a metabolic alkalosis. However, if there is an associated acidosis, correction should be with one-sixth molar sodium lactate. Hyponatraemia with a volume excess may be treated by restriction of water.

The kidney has little ability to compensate for water shortage, and hypernatraemia can occur even when renal function is normal. Central nervous system signs are present in hypernatraemia. Body temperature is elevated and may reach a lethal level. C.n.s. haemorrhages may occur when the ECF hyperosmolarity causes a shift of intracellular water to the ECF. A high serum sodium (above 150 m-mole/litre) indicates a significant deficit of total body water.

Iatrogenic hypernatraemia results from several circumstances: infusion of salt-containing solutions to replace water losses; excessive glucose administration which requires a large volume of water for its excretion; high protein feedings which produce an osmotic load of urea; and indiscriminate infusions of osmotic diuretic agents and their

obligatory excretion with a large volume of water accompanying the increased sodium output.

Hypernatraemia may follow excessive water losses due to fever and sweating, losses through tracheostomies in a dry environment, and from evaporation through a large burned area. Renal water losses may be significant after hypoxic damage to the distal tubules and collecting ducts, resulting in large volumes of solute-poor urine.

For the treatment of severe symptomatic hypernatraemia with an associated volume deficit, infuse 5% D/W slowly to avoid reducing ECF osmolality too rapidly, causing convulsions and coma. Correction may also be accomplished with less danger of convulsions by infusing half-strength sodium chloride or half-strength lactated Ringer's solution.

(3) Alteration of the concentration of other ions in the ECF will produce compositional change without significant change in osmolality. The parameter of fluid balance which particularly requires laboratory data is that for compositional changes. Examples include changes in pH, glucose, and lactic acid. Laboratory findings are diagnostic before the appearance of physical signs such as Kussmaul's respiration, carpopedal spasm, or hypoglycaemic coma (Jenkins and Giesecke, 1968). In many patients compositional changes can be anticipated, such as the hypochloraemic, hypokalaemic alkalosis which may occur in the patient allowed water by mouth when he has a gastric tube on suction.

(4) Distributional changes are internal losses of ECF into non-functional spaces representing, therefore, a type of volume change. There are several examples: the sequestration of isotonic fluid subjacent to the peritoneum in peritonitis, varying up to several litres; the swelling of burned tissue; oedema following muscle trauma; and ascites. These represent fluid in extracellular non-functional spaces. Another internal site for functional loss of isotonic fluid is into cells, occurring in haemorrhagic shock. All distributional shifts cause a contraction of the functional ECF volume.

Metabolic water exchange.

Normal water exchange for the patient is between 2 and 3 litres each day. Water by mouth is in the range of 1,000–1,500 ml daily. Ingestion of solid foods, such as meat, fruit, and vegetables which are 60–97% water, provides another 600–700 ml. Oxidation of foodstuffs contributes about 14 ml water for each 100 calories of fat, carbohydrate, or protein metabolized.

Obligatory losses of water amount to 1,700 ml, including 900 ml of water from normal urinary function in excreting non-volatile solutes such as urea, mineral salts, and other metabolic products. For removal of body heat another quantity of water, amounting to 800 ml, is lost through the lungs and skin. Greater losses may occur in a hot environment or due to fever.

The patient will gain from 350 to 800 ml water from excessive catabolism. This will vary according to the degree of surgical trauma and complications which occur during convalescence, and it will require a greater volume of urine to excrete these products of catabolism.

Postoperative metabolism.

Unless he has an infection and/or elevated temperature, the bedfast postoperative patient is usually assumed to have a level of metabolism about halfway between the basal state and that of normal activity, significantly higher than resting metabolism because of tissue injury (Holliday and Segar, 1957). It must be recognized that injuries of great magnitude cause an increased calorie expenditure, perhaps as high as 6,000 calories per day for the patient with an extensive burn. The postoperative patient usually has an accelerated nitrogen loss not entirely explained by protein metabolism (Kinney, Long and Duke, 1970). Even large infusions of carbohydrate will not entirely inhibit the massive catabolism of body nitrogen in the traumatized patient, and there is some question whether an attempt to match the protein loss by infusion of amino acids would be of benefit to him (Cahill, Felig and Marliss, 1970).

Caloric needs.

The "average" adult weighing 70 kg and having a body surface area of 1.7 m² has daily caloric needs of approximately 2,500 calories for non-strenuous physical activity (1,500 cal/m² body surface). The same afebrile adult patient at bed rest can be sustained on 2,000 calories, but it is neither feasible nor possible to meet this caloric requirement with intravenous glucose solution alone. To do so would require 553 g of glucose, with the emphasis that parenteral glucose (USP) is in a monohydrated form and has only 91% caloric effect, and that the caloric equivalent of monosaccharides is 3.75 cal/g and not the 4 cal/g usually ascribed to carbohydrates (Weisberg, 1962). Reportedly, the maximal speed of administration of glucose to normal adults without producing glycosuria is approxi-

mately 0.5 g/kg body weight/hr (3 hr for the injection of 1 litre of 10% glucose—100 g and 375 cal). If the patient's liver is glycogen-deficient, glycosuria will result when glucose is administered at even slower rates (Gardner et al., 1950).

During the immediate postoperative period, a time of relative starvation, 400–600 calories as glucose each day spares the major proportion of muscle catabolism and produces the proper substrate for those tissues which are obligatory utilizers of glucose. The brain requires 100–150 g glucose daily. Other glycolytic tissues (erythrocytes, leucocytes, bone marrow, renal medulla and peripheral nerve) metabolize 30–40 g glucose to lactate and pyruvate, which are remade into glucose by the liver and kidney. Reparative tissues, particularly fibroblasts and phagocytes, are also mainly glucose utilizers (Cahill, Felig and Marliss, 1970).

Urine output.

During long operations and for all major trauma patients, we rely on hourly urine output to reflect an adequate functional ECF. In the postoperative phase, urine output is usually a reliable index of volume replacement, but we realize that it may be misleading. If an excess of glucose has been infused it will cause an osmotic diuresis. Patients who have sustained great injury and shock may have incipient acute renal damage indicated by high urinary volumes. The same may apply to many patients with chronic renal disease.

In balancing intake and output, urine balance for the convalescent patient should not be replaced on a ml-for-ml basis. Balance studies have shown that ECF sequestered from the dynamic areas of the circulation (non-functional) during operation and/or trauma will be slowly mobilized and excreted by the kidney during the first few postoperative days (Shires and Jackson, 1962). An output of 3,000 ml on one postoperative day may simply represent diuresis of fluids given during the operation, or may represent excessive postoperative infusion of fluids. Urine output will progressively increase if these large volumes are completely replaced, until a status resembling diabetes insipidus results.

Serum electrolyte studies.

Although usually it is thought unnecessary to determine serum electrolytes during the days immediately after an operation, we feel that they should be followed daily if the patient is maintained on intravenous fluids. There are good

physical signs of changes in sodium concentration, but changes in potassium may become suddenly disastrous without prior warning. The total extracellular potassium is small ($4.5 \text{ m-mole/litre} \times \text{ECF} = \text{about } 63 \text{ m-mole}$ in the 70 kg adult male), but is critical to cardiac and neuromuscular function. Potassium losses in the urine following trauma may reach 90–100 m-mole/litre.

For the non-surgery patient the normal daily dietary intake of potassium varies from 50 to 150 m-mole, and most of this is excreted in the urine. Usually the kidney continues to excrete potassium even if hypokalaemia exists, but it is unwise to administer potassium during the first 24 hr after surgery unless there is a measured deficit. A small quantity of potassium may prove fatal if administered during oliguria, or to the patient having the more insidious high-output renal failure (HORF). HORF is prone to occur in patients who have had one or more episodes of hypotension during a long operation, and in trauma patients who have suffered haemorrhagic hypotension. The chief danger to HORF is not recognizing renal failure with uraemia and hyperkalaemia, because urinary volume is normal (Baxter, Zedlitz and Shires, 1964).

Hyperkalaemia is such a serious complication and so difficult to treat that an approach to its treatment will be reviewed here. Temporary suppression of the myocardial effects of a sudden rapid increase in potassium can be accomplished by the intravenous administration of a solution containing 80 m-mole of sodium lactate, 100 ml of calcium gluconate, and 100 ml of 50% D/W. This dextrose stimulates the synthesis of glycogen, which enhances the cellular uptake of potassium. Insulin may also be given, limited to one unit per 5 g or more glucose; rebound hypoglycaemia should be anticipated. The sodium lactate will elevate the pH and shift potassium intracellularly, and the calcium gluconate tends to counteract the myocardial effects of hyperkalaemia.

This regimen may not be sufficient to treat severe hyperkalaemia except to allow time while preparing for definitive removal of excess potassium by dialysis. A cation-exchange resin in 10% D/W administered by rectum may control a slow rise of potassium, but is not sufficient for the acute treatment of life-threatening hyperkalaemia (Shires and Canizaro, 1974).

Hypokalaemia may also be a problem for the patient maintained on intravenous fluids, and it

may result from excessive renal excretion, movement of potassium into cells, administration of potassium-free fluids, and loss in gastrointestinal secretions. If the patient is not oliguric, there is reason to give slowly 40 m-mole potassium daily in intravenous fluids while the patient is on a "nothing-by-mouth" regimen after the first 24 hr following operation and/or surgical stress.

Disturbances in calcium metabolism generally do not occur in the uncomplicated postoperative patient except during long periods of skeletal immobilization. Hypocalcaemia may occur in pancreatitis, with necrotizing infections, in renal failure, and as a result of hypoparathyroidism. Whether calcium should be administered to the patient receiving multiple blood transfusions is still questioned. If the transfusions are infused slowly, the binding of calcium by the citrate in ACD solution usually is compensated for by the mobilization of calcium from the bone.

Hypercalcaemia may occur as a result of hyperparathyroidism and from malignant bony metastases. If the serum calcium load is elevated in the preoperative patient, he should be placed on a low-calcium diet. In the postoperative period his ECF volume should be well expanded by adequate hydration, otherwise the early symptoms of anorexia, nausea, and vomiting will progress to stupor and coma followed by death.

For those patients who exhibit disturbed (hyperactive) neuromuscular or cerebral activity in the postoperative period, magnesium deficiency should be considered. Magnesium replacement may be important for the patient who requires protracted parenteral fluid therapy because of gastrointestinal dysfunction. As much as 2 m-mole magnesium/kg body weight/day may be infused with other fluids in the patient having a deficiency.

Magnesium excess may occur in the presence of acidosis in patients with poor renal function, if they have ingested large quantities of magnesium-containing antacids and laxatives. Treatment is directed first toward the acidosis and then to repletion of ECF volume deficit if such exists. Acute findings (lethargy, weakness, widened QRS complex) may be controlled temporarily by infusion of 5–10 m-mole of calcium chloride or calcium gluconate.

Parenteral hyperalimentation.

It is beyond the scope of this paper to discuss the concept and methods of complete nutritional

support for the starving patient, using elemental diets or parenteral hyperalimentation to counteract nitrogen losses in the catabolic state (Dudrick et al., 1972). However, it should be recognized that this represents an impressive contribution to nutritional support for many postoperative patients, such as those having small bowel fistulae, continuing pyloric obstruction, pancreatitis and/or pancreatic fistulae, or inanition for any reason.

The patient maintained on parenteral hyperalimentation, who is scheduled for another operation may present several problems. For example, the serum potassium level may be within normal range although the intracellular potassium may be low. Unless the patient's formula includes more than 100 m-mole of potassium per day, he will probably have a low intracellular level and will suffer a serious cardiac depression following induction of anaesthesia. In contrast, another patient may have a low serum potassium (and alkalosis) because of its shift to the intracellular space resulting from the large glucose infusion. The accompanying glycosuria probably will not respond to insulin but will require additional potassium, frequently necessitating more than 200 m-mole per day.

Relative glucose intolerance may occur with hyperalimentation, so it becomes important to monitor both serum and urine glucose levels intraoperatively to anticipate postoperative hyperglycaemic coma. Non-ketotic, hyperosmolar hyperglycaemia may develop in a patient even at normal rates of infusion following additional severe surgical stress or trauma.

The possibility of magnesium deficiency exists for the patient with protracted dysfunction of the gastrointestinal tract, even though magnesium is provided in the hyperalimentation formula. In the postoperative period a deficiency of magnesium will be manifest by neuromuscular and central nervous system hyperactivity.

In the United States the use of fat emulsions has met with little success, and because of the accompanying side effects they are no longer approved by the Federal Drug Administration. By contrast, reports from Europe indicate successful and wide use of fat emulsions. Blood coagulation problems may present the greatest hazard caused by their use.

SUMMARY

Consideration must be given to the patient's preoperative fluid and electrolyte status and his intra-

operative maintenance in determining proper fluid and electrolyte management during the immediate postoperative and convalescent periods. Because of this, we have referred to some preoperative factors which may cause problems after induction of anaesthesia. Also, we have outlined our basic precepts for intraoperative fluid therapy and have commented on the rationale for these precepts, defining the use of 5% glucose in distilled water as an aid in depressing antidiuretic hormone, and the use of balanced salt solutions to bolster intravascular volume and to obviate the secretion of aldosterone.

In commenting on postoperative fluid management, we note that a patient should not experience oliguria or anuria as a result of anaesthesia, but should maintain a normal urine output unless complications have occurred.

In classifying fluid imbalance, we have categorized disturbances in volume, concentration, composition, and distribution. Volume changes are revealed primarily by physical signs, and changes in concentration occur with changes in sodium ions which account for 90% of the osmotically active particles in extracellular fluid. Alteration of concentration of other ions in the ECF produces compositional changes without significantly affecting osmolality. Distributional disturbances represent internal losses of ECF into non-functional spaces, from where eventually it is slowly mobilized and excreted by the kidney.

The bedfast postoperative patient usually has a level of metabolism significantly higher than the resting state, because of tissue injury. His accelerated nitrogen loss is not explained entirely by protein metabolism, and it cannot be corrected entirely by parenteral alimentation. However, infusion of glucose spares a portion of muscle metabolism and provides the proper substrate for glycolytic tissues.

In the postoperative phase urinary output is usually a reliable index of volume replacement, but there are still important reasons for following serum, and in some cases, urine electrolytes. There are good physical signs for changes in sodium concentration, but changes in potassium may become disastrous with little preliminary warning.

Although the subject of parenteral hyperalimentation is beyond the scope of this paper, it is noted that the patient maintained on this regimen will present some special problems if scheduled for

anaesthesia and operation. These problems relate to serum and intracellular potassium levels, relative glucose intolerance leading to hyperglycaemic coma, and magnesium deficiencies resulting in convulsions and coma.

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