Some Aspects of the Pre and Post Operative Care of Surgical Patients.

by

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The first forty years of the twentieth century have seen greater advances in the science and art of surgery than the whole of preceding time. But, as the field of surgical endeavour has widened, new problems have arisen. Operations are now carried out upon patients so reduced by their disease that formerly interference would not have even have been considered. The outcome of such enterprises depends upon meticulous care in the pre and post-operative treatment, not the least important aspect of which is the maintenance of normal water and electrolyte metabolism. In this connection it is interesting to recall that as long ago as 1831 Dr. W.B.O'Shaughnessy of Newcastle upon Tyne recognised the essentials of water and electrolyte depletion and also outlined treatment. In a brief letter to the London Medical Gazette[1] he summarised the changes which occur in the blood of patients suffering from cholera. He stated that:

1. "The blood drawn in the worse cases of cholera is unchanged in its anatomical or globular structure."

2. "It has lost a large proportion of its water, 1000 parts of cholera serum having but the average of 850 parts of water."

3. "It has lost also a great proportion of its neutral saline ingredients."

4. "Of the free alkali contained in healthy serum, not a particle is present in some cholera cases, and barely a trace in others."

5. "Urea exists in the cases where suppression of urine has been a marked sympton."

6. "All the salts deficient in the blood, especially the alkali or carbonate of soda, are present in large quantities in the peculiar white dejected matters."

Dr. O'Shaughnessy then went on to state that cure was dependent upon two principles. "First, to restore the blood to its natural specific gravity (i.e., water content); second, to restore its deficient saline matters." He advocated copious enemata of "warm water holding the natural salts of the blood in solution" and also the intravenous administration of a similar fluid.

In 1850, O'Shaughnessy's brilliant work was confirmed and greatly

amplified by Karl Schmidt, but up to the end of the nineteenth century there were only occasional references to the therapeutic value of saline solutions. In 1909, however, Rogers[2] reported his experiences of the treatment of cholera with 1.35 per cent sodium chloride solution administered intravenously and intraperitoneally. He found that its use decreased the mortality of the disease from 61 to 32.5 per cent. Nichols and Andrews[3] during the Philippine epidemic of cholera in 1908 also showed the value of saline solutions by reducing the mortality during the stage of collapse by 34.6 per cent. These extremely gratifying results led to the increasing recognition of the value of the administration of water and electrolytes in the treatment of diseases characterised by the loss of abnormal amounts of body fluids.

The application of O'Shaughnessy's principles has gradually become more generally adopted as knowledge of the changes in the water and electrolyte content of the body from surgical diseases has increased. In 1912, Hartwell and Hoguet[4] showed in dogsthat, after complete obstruction of the lower duodenum, life could be greatly prolonged by the daily subcutaneous administration of physiological saline solution in amounts slightly in excess of the total volume of the urine and vomitus of the previous twentyfour hour period. They attributed its effects to the relief of dehydration which is such a marked feature of high intestinal obstruction. In 1914, Tileston and Comfort[5] described the rise in blood non-protein nitrogen which accompanies the dehydration of high intestinal obstruction, an observation which was confirmed by Cooke, Rodenbaugh and Whipple[6] in 1916. In 1918, McCann[7] described the increase in carbon di-oxide combining power of the blood which occurs from the loss of gastric secretions after obstruction of the pylorus. In 1920, MacCallum and his associates[8] reported the fall in plasma chloride concentration after complete obstruction of the pylorus and confirmed McCann's observation. They also showed that the administration of sodium chloride prevented both the fall in the chloride concentration and the rise in the carbon di-oxide combining power of the plasma, and that the administration of hydrochloric acid or sodium bi-carbonate prevented neither. Haden and Orr[9-17] in a series of brilliant investigations, made important contributions to our knowledge both of fluid therapy and of the pathology of intestinal obstruction. They confirmed the observations of previous workers that azotaemia, hypochloraemia, and alkalosis

result from high intestinal obstruction, and showed that in low intestinal obstruction these changes are not nearly so marked. They also confirmed the beneficent effects of the administration of sodium chloride solution reported by Hartwell and Hoguet[4] and by MacCallum and his associates[8], and demonstrated the ineffectiveness of many salts of sodium and chloride, other than sodium chloride. They further showed that the administration of water alone did not prevent the onset of symptoms and death from high intestinal obstruction as was inferred by Hartwell and Hoguet. They reported the increased sedimentation rate and the fall in the tissue chloride concentration after high obstruction of the gastro-intestinal tract.

The findings of these early workers have since been corroborated by a large number of clinical and experimental investigations. Valuable papers are those of Hastings, Murray and Murray[18], Gamble and Ross[19], Gatch, Trusler and Ayers[20], and many others.

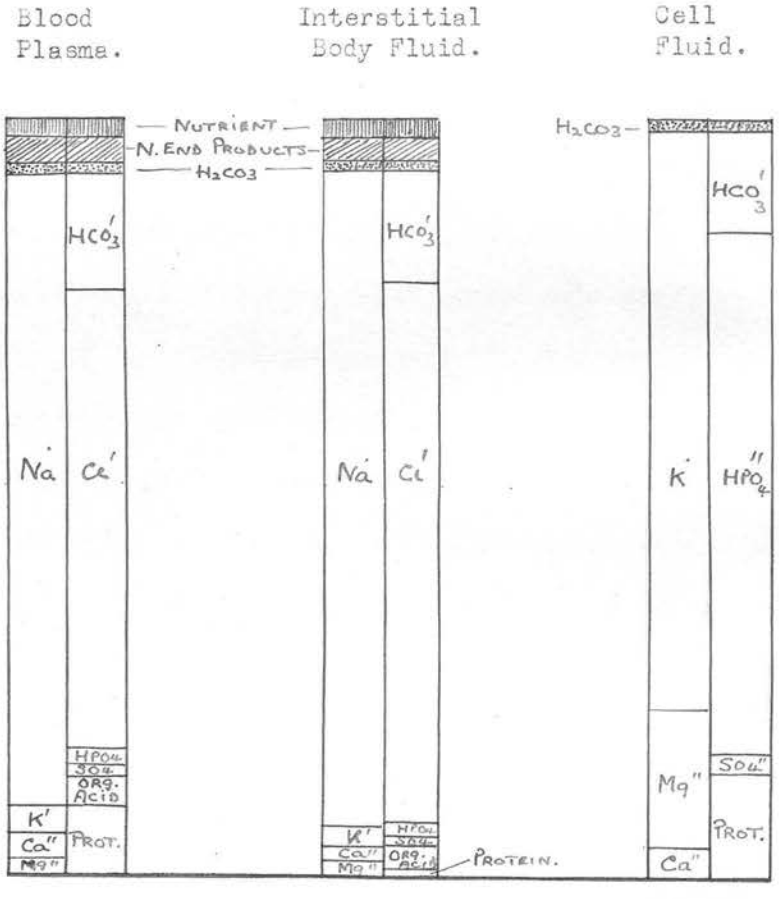
From these early papers and the very numerous investigations which have since been carried out, it has become obvious that loss of water and sodium chloride from the body so alters "the innumerable and interrelated chemical reactions that together accomplish what we call metabolism"[21] that life itself may thereby be seriously endangered. Therefore rational fluid therapy must be directed not to restoring the normal body content of any particular component of the body fluids (which indeed is separately impossible), but to the restoration and maintenance of the normal volume, composition, and distribution of the body fluids as a whole, so that metabolism may proceed under the most favourable of circumstances. It is the purpose of this paper to discuss the metabolism of the body fluids, the practical applications of fluid therapy in surgical patients, and to make some contribution to our knowledge of the therapeutics of sodium chloride.

The Body Fluids.

Water forms from 63 to 65 per cent of the total body weight[22] and exists both inside the cells and in the extracellular spaces of the body. In both fractions of the body water many substances are dissolved, the resulting complex solutions being known as the intracellular fluid and extracellular fluid.

FIGURE 1.

Chemical Composition of the Body Fluids in Terms of Acid Base Equivalence.



(Derived from J.L.Gamble, Bulletin of the Johns Hopkins Hospital, 61. 153, 193

predominant anion of extracellular fluid, the phosphate ion that of intracellular fluid. This separation of apparently similar elements chemically presents a problem of great physiological interest. At the present time all that can be said is that in the case of the cations especially the cell membrane exercises a "customs" right to reject sodium and allow the passage of potassium.

The two main components of extracellular fluid, the blood plasma and the interstitial fluid, have an almost identical chemical structure, their small differences in composition being due to the osmotic effects of the much larger quantity of protein in the plasma. The plasma proteins also form the mechanism whereby interstitial fluid may be attracted into the blood stream from the interstitial compartment.

It will thus be seen that the chemical structures of the intracellular and extracellular fluids are quite different, the cell membrane or some other unknown factor separating certain of the electrolytes of the two fluids. In spite, however, of these differences of composition, equality continually is maintained between the osmotic pressures of the two fluids and also between their hydrogen ion concentrations. The maintenance of equality between the hydrogen ion concentrations and the osmotic pressures of the two fluids is effected by the transference of water between the two fluids and by reactions which follow Donnan's law and the Henderson equation, in addition to acid phosphate, alkaline phosphate reactions etc. A discussion of these interesting reactions is outside the scope of this paper and will not be further pursued. It is necessary however to discuss more fully the functions of the body fluids.

The Functions of the Body Fluids.

Extracellular fluid obviously is the vehicle for the conveyance of materials to and from the cells. It also serves other important functions which will be examined later. Knowledge of the functions of the intracellular fluid is, at the present time, very incomplete and much of it is speculative. It is the medium in which the vital processes of the cell actually proceed, and, for the preservation of cellular life, physico-chemical conditions conditions within the cell must remain stable within narrow limits. The hydrogen ion concentration and osmotic pressure of the intracellular fluid reflect the values at which these

properties are held in extracellular fluid, the immediate environment of the cell. The organism as a whole has therefore developed special methods for the maintenance of normality in the latter in order that normality also may be maintained in the former. These may be conveniently considered under two heads. 1. Regulatory mechanisms present in extracellular fluid itself, and 2, The kidney in relation to extracellular fluid.

Regulatory Mechanisms Present in Extracellular Fluid.

a. The Control of Hydrogen Ion Concentration and Osmotic Pressure of Extracellular Fluid.

In extracellular fluid the stability of the hydrogen ion concentration is controlled by the maintenance of the normal ratio of the concentrations of free carbonic acid and plasma bicarbonate. This normal ratio, at the pH of 7.4, is one part of carbonic acid to twenty parts of bicarbonate. The carbonic acid concentration is very accurately maintained at three volumes per cent by the respiratory mechanism, and it follows therefore that the plasma normally contains 60 volumes per cent of bicarbonate. Since the concentration of carbonic acid in the plasma is subject only to minor compensatory variations, deviations from the normal of the carbonic acid - bicarbonate ratio, known as acidosis or alkalosis, are the result of changes in the plasma bicarbonate concentration. Such alterations in the plasma bicarbonate concentration are always secondary to changes in the ratio of the concentration of fixed base to that of acids, other than bicarbonate, in the extracellular fluid. "In other words, the plasma bicarbonate value is determined simply by the extent to which fixed base stands above the sum of the concentrations of the other acid radicals."[26] Since, in extracellular fluid, the sodium and chloride ions are overwhelmingly the most important basic and acidic ions, and also the most liable to alterations both in their absolute and relative concentrations, the plasma bicarbonate concentration will practically always depend upon the ratio of the concentrations of sodium and chloride. It therefore follows that the bicarbonate concentration in extracellular fluid almost always reflects the ratio of the concentrations of sodium and chloride in that fluid.

b. The Osmotic Pressure of Extracellular Fluid.

The osmotic pressure of extracellular fluid is the sum of the osmotic

pressures of all its chemical components. In extracellular fluid chemical equivalence between the concentrations of basic and acidic ions is maintained, in the presence of fluctuations in the concentration of the chloride ion, by recippocal alterations in the concentration of bicarbonate, the total electrolyte concentration thereby remaining unchanged. It follows therefore that the osmotic pressure of extracellular fluid varies with the concentration of base, 94 per cent of which normally consists of sodium. The osmotic pressure of extracellulaf fluid therefore depends almost entirely upon the concentration of sodium in that fluid. In the presence of a fall in the concentration of chloride in extracellular fluid, it would seem that the defence of hydrogen ion concentration could be equally well effected by the excretion of an equivalent amount of sodium by the kidneys, rather than by the substitution of bicarbonate for lost chloride. That such a method is not used makes it clear that the conservation of sodium for the maintenance of extracellular fluid volume and osmotic pressure is of the greatest importance, and the significance of the hormaonal control of sodium by the supra-renal cortex becomes the more appatent.

The Kidney in Relation to Extracellular Fluid.

The composition of extracellular fluid is continually being affected by the ingestion of water and electrolytes in the food, and by the secretion of the digestive juices and the sweat. In order to maintain the necessary stability of the physicochemical properties of extracellular fluid upon which depends the successful operation of intracellular processes, some specialised organ in intimate relation to extracellular fluid is required. The kidney fulfils this requirement perfectly. Considerations of space forbid a full discussion of renal physiology in relation to extracellular fluid, and therefore only a broad general outline will be presented.

The first important function of the kidneys is the preservation of the normal electrolyte pattern of the extracellular fluids. Since basic and acidic radicals are ingested in unequal quantities it is necessary that the kidneys shall be able to excrete basic and acidic radicals in varying proportions. If, as is usually the case, acid radicals are ingested in excess of basic, the kidneys effect the excretion of the excess acidic elements by:

1. The excretion of a urine which is more acid than the blood plasma, by the formation of monobasic phosphate from dibasic phosphate, thus directly conserving fixed base.

2. By the excretion of from 10 to 20 per cent of the total organic acids excreted in the urine uncovered by base.

3. By the formation of the base ammonia from urea to complete the covering of the acid excreted, thereby further conserving the fixed base of extracellular fluid. Thus in the presence of the necessity of excreting an excess of acid radicals, an absolute saving of base is effected by excreting an acid urine and substituting ammonia for fixed base.

On the other hand it may be necessary to excrete an excess of fixed base over acid. In these circumstances it is necessary that an acid substance be present in the plasma capable of combining with the excess of fixed base to form salts which the kidneys can excrete. Carbonic acid, normally excreted by the lungs, fulfils this requirement.

The second important function of the kidneys is the preservation of the normal osmotic pressure of extracellular fluid. This is effected by the selective re-absorbtion of water and electrolytes from the glomerular filtrate. If sodium, the predominant base of extracellular fluid, is lost, as for example by the rejection of gastro-intestinal secretions, there is a corresponding loss of extracellular water. Conversely, if the intake of sodium is in excess of its excretion, a corresponding amount of water will be retained in the body. Therefore the concentration of total base in extracellular fluid and the osmotic pressure of that fluid remains practically constant, and the amount of extracellular base present in the body cannot be deduced merely from its concentration in an extracellular fluid, for example blood plasma. This accurate control of the concentration of base and the osmotic pressure of extracellular fluid, is maintained by the kidneys even in the face of considerable losses of base by the excretion of a corresponding amount of water. A point is reached however, at which the necessity for preventing further depletion of extracellular fluid volume exercises an important influence. Recently, in two normal subjects in whom extracellular electrolytes were removed by continuous gastro-duodenal drainage, it was found that, in spite of providing enough 5 per cent glucose solution in distilled water intravenously for the daily excretion of more than 1500 cc. of urine, after the plasma sodium chloride concentration of the subjects had been reduced below 450 mg.%, a rise in the plasma non-protein nitrogen concentration occurred, and that it increased with further removal of base. In each case the carbon dioxide combining power of the plasma remained within four volumes per cent of 55 throughout the experiment,

indicating that sodium and chloride were lost in equivalent amounts. The kidneys, therefore, in addition to defending the osmotic pressure of extracellular fluid, are, in some measure, also separately able to defend the extracellular fluid volume by the retention of osmotically effective substances such as urea which it normally rapidly excretes. Jeghers and Bakst[27] in a very able review of the syndrome of extrarenal azotaemia, have emphasised the relationship between the loss of body sodium and azotaemia. Basing their discussion on the researches of Kerpel-Fronius, and Gomori and Podhradszky, they point out that hypochloraemia in the absence of hyponatraemia causes neither dehydration nor azotaemia if an adequate fluid intake is provided. Hypochloraemia therefore, by itself plays no direct part in the developement of azotaemia. But, in the hypochloraemia which results, for example from vomiting, although little sodium is lost from the body, much water is lost, dehydration results, and azotaemia will follow because little water is available for the excretion of urine. Loss of sodium, on the other hand, is accompanied by a corresponding reduction in extracellular fluid volume comparable to direct water reduction and results in azotaemia even if an adequate fluid intake is provided. The authors apparently subscribe to the view expressed by Gomori and Podhradszky that azotaemia associated with hyponatraemia may be attributed to dehydration occasioned by the loss of sodium, even though the fluid intake remains adequate for the secretion of a satisfactory volume of urine.

With this conception we are not in complete agreement and will therefore state our interpretation of the relationship between loss of sodium and azotaemia. We believe that the azotaemia which accompanies the loss of sodium, and therefore of extracellular fluid, constitutes an attempt on the part of the body to prevent an excessive fall in extracellular fluid volume, since the nitrogenous waste products retained are osmotically effective and therefore add to the total volume of fluid present in the body. It must be emphasised, however, that nitrogenous waste products such as urea, uric acid etc., are only effective in increasing the total volume of fluid present in the body and do not have any selective function in confining such retained fluid in one or other compartment.

The implications of this conception of the reasons for nitrogenous waste product retention in the presence of adequate urinary excretion and hyponatraemia

are of far reaching importance. They afford an explanation of the dehydration which accompanies chronic interstitial nephritis, the azotaemia of diabetic coma, the tendency to azotaemia which exists during the severe stages of Addison's disease and many other problems. They also lead to a method of treatment based on the study of the requirements of base in these conditions. And finally it can be said that preliminary investigations have confirmed the soundness of these conceptions and hopes for the improvement in treatment so earnestly needed today for these diseases.

The third important function of the kidneys is the excretion of certain waste products such as urea. For the perfect performance of this function it is necessary that there shall be an adequate volume of water available for excretion. From data supplied by Lashmet and Newburgh[28], Coller and Maddock[29], assuming that 35 Gm. of solids are excreted daily in the urine, calcuoated the minimum volumes of urine necessary per day to effect the excretion of this weight of urinary waste products both by normal and by diseased kidneys. Their results are presented in Table 1.

TABLE 1.

Kidneys.	Maximum Concentrating Ability. Specific Gravity.	Minimum Amount of Water Required to Excrete 35 Gm. Waste Materials. cc.
Normal.	1032 - 1029	473.
Diseased.	1028 - 1025	595.
	1024 - 1020	605.
	1019 - 1015	850.
	1014 - 1010	1439.

The volumes given in Table 1 are those necessary for the excretion of 35 Gm. of waste materials by healthy and diseased kidneys under conditions forcing them to work at their maximum concentrating ability. It is probable that surgical patients during the post-operative period may excrete considerably more than 35 Gm. of waste materials in the urine.[30] It follows, therefore that the minimum volume of water necessary to excrete the urinary waste products after operation may be considerably larger than the volumes given above by Coller and Maddock. As a clinical rule, therefore, it can be stated that patients with healthy kidneys should be supplied with enough water to permit of the daily excretion of 1200 cc. of urine.

Before leaving the subject of renal physiology in relation to body fluids it is necessary to emphasise that for the performance of any of its functions, the

kidneys require an abundant supply of water. Acid base regulation, the maintenance of osmotic pressure, and the excretion of waste products can only be effected by the simultaneous excretion of an adequate volume of water.

A brief discussion of the physiology of the body fluids has been presented. It now becomes necessary to consider (1) Alterations in the normal distribution of water in the body, and (2) Dehydration.

Alterations in the Normal Distribution of Water in the Body.

It is very largely to Schechter[31], Schechter, Cary, Carpentieri, and Darrow[32], and Darrow and Yannet[33], that we owe our knowledge of the effects of changes in the distribution of water between the cells and extracellular spaces. These authors showed that if an isotonic solution of sodium chloride or glucose is injected into the peritoneal cavity of an animal, the composition of the injected fluid becomes altered to that of a fluid in ionic and osmotic equilibrium with the blood plasma. In the isotonic sodium chloride solution injections, it was found that:

1. The total base of the injected fluid remains practically unaltered, but the chemical pattern of the acidic ions altered to that of the plasma though remaining equivalent chemically to the total base.

2. That absorbtion of the injected fluid from the peritoneal cavity proceded at a constant rate.

In the isotonic glucose solution injections there was initially a marked rise in the volume of the fluid in the peritoeal cavity, electrolytes from the plasma passed into the peritoneal fluid, its glucose concentration gradually fell, and the animals appeared dehydrated and were anuric. At the end of from four to six hours sufficient absorbtion had taken place approximately to restore the original volume of fluid injected. The explanation for the initial increase in volume is supplied by Clark[34] who points out that the electrolytes which enter the peritoneal cavity with water to adjust the chemical equilibrium do so faster than the larger slowly moving glucose molecules can carry water out. This experiment is of particular importance to the present discussion because, at the end of the four to six hour period, without alteration in the total body water, the body may be considered to have lost temporarily the amount of electrolyte which has passed into the fluid in the peritoneal cavity, since that fluid was no t immediately available throughout the tissues.

Realising the great significance of these experiments Darrow and Yannet[33] in one series of animals (dogs, rabbits, and monkeys) repeated the intraperitoneal injection of isotonic glucose solution, and, in another series, injected 1.8 per cent sodium chloride solution intraperitoneally. They found, as in the glucose series, that approximately four hours after the intraperitoneal injection of 1.8 per cent sodium chloride solution, the fluid in the peritoneal cavity was in approximate ionic and osmotic equilibrium with the plasma and the total volume of fluid remaining was nearly the same as that injected. Thus the total amount of extracellular electrolyte had been increased without significant change in the total body water. In each experiment a weight of fluid equal to ten per cent of the body weight of the animal was injected.

As the result of these experiments, it was therefore possible to study the changes in the distribution of body water which result from the addition (1.8% sodium chloride solution injections) or subtraction (5% glucose solution injections) of extracellular electrolytes, the total water content of the body remaining practically unaltered. The results of these experiments are shown in Table 2.

TABLE 2.

5% Glucose Solution Injections. Extracellular Electrolyte Subtracted from the Body.	1.8% Sodium Chloride Solution Injections. Extracellular Electrolyte Added to the Body.
1. No local signs of pain or peritoneal irritation.	1. No local signs of pain or peritoneal irritation.
2. All the animals showed signs of mild to severe dehydration. Tongue and mucous membranes were dry, skin turgor lost, animals became languid and looked sick. They were not thirsty.	2. Animals remained well. They became thirsty but no loss of skin turgor occurred.
3. All were anuric.	3. In three out of eleven animals a definite diuresis occurred. In the other eight animals no urine was passed.
4. Changes in the Blood. The red cell count and the serum protein concentration were increased. The plasma volume was therefore decreased. (Roughly from 8 to 27 per cent).	4. Changes in the Blood. The red cell count and the serum protein concentration were decreased. The plasma volume was therefore increased. (Roughly from 4 to 52 per cent).
5. The concentration of cell protein in the red cells decreased. The intracellular water was therefore increased.	5. The concentration of cell protein in the red blood cells increased. The intracellular water was therefore decreased.
6. In all cases considerable reductions in the concentrations of chloride and sodium in the plasma occurred, obviously due to the migration of chloride and sodium into the fluid in the peritoneal cavity.	6. In all cases considerable increases in the concentrations of chloride and sodium in the plasma occurred, due to the migration of these ions from the 1.8% sodium chloride solution injected into the peritoneal cavity,

The results of these experiments have been recorded in some detail because of their importance. The following deductions emerge from them:

1. Since base is the determining factor in the production of osmotic pressure in the body fluids, and because neither the base of the intracellular fluid, potassium, nor the base of extracellular fluid, sodium, can pass freely between the cells and extracellular fluid, the maintenance of equality between the osmotic pressures of the intracellular and extracellular fluids is effected by the transference of water from one compartment to the other.

2. The clinical manifestations of dehydration are the result of reduction in the extracellular fluid volume except that the sensation of thirst is apparently the result of cellular dehydration.

3. Because of the profound alterations in the distribution of water and salt which result from the administration of 5 per cent glucose solution in distilled water intraperitoneally, the only fluid injected into the peritoneal cavity should be physiological saline solution.

4. Since the rate of absorbtion of fluids from the peritoneal cavity is relatively slow, correction of dehydration and electrolyte deficit is considerably delayed when this method of administration is used.

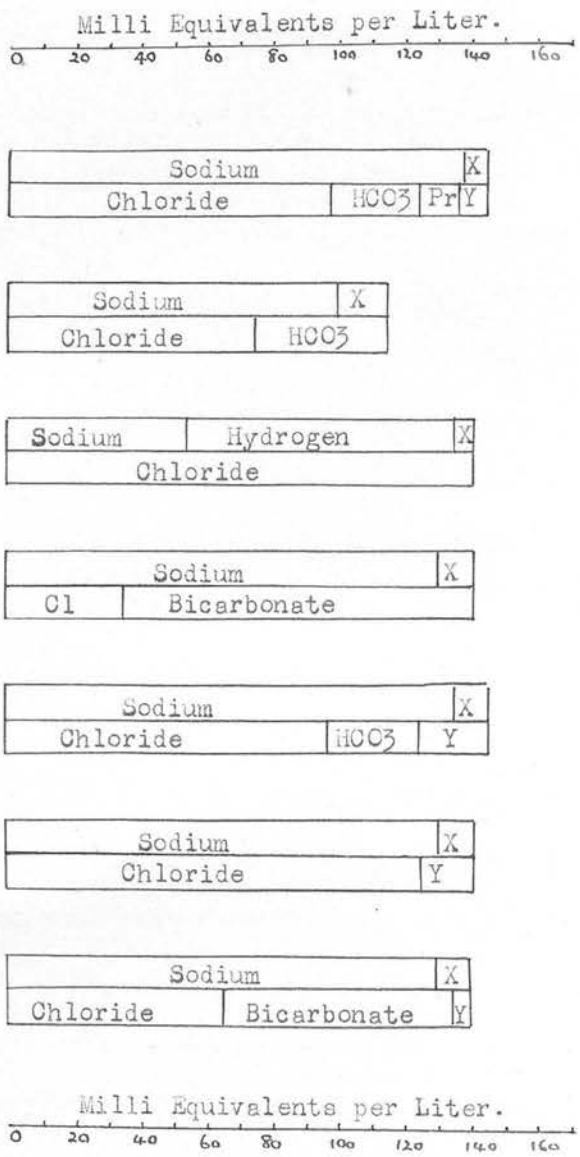
It has been shown by Gilman[35] that a decrease in blood pressure and an increase in susceptibility to the effects of haemorrhage result from the loss of extracellular electrolytes with little change in total body water. He also showed that disturbances in the distribution as well as the amount of body water probably occur as the result of disproportionate losses of water and electrolytes in diabetic coma, persistent vomiting, protracted diarrhoea, heat stroke, etc. Thus the partition of water between the extracellular and intracellular compartments of the body is of great clinical improtance and one looks forward therefore with eagerness to the day when, not only as is now possible, one can be assured of the proper total hydration of the patient, but also can be certain that the body water is ideally distributed between the cells and extracellular spaces.

Dehydration.

The term dehydration has become deeply entrenched in medical literature. But its exact meaning is not correspondingly well understood. To some it means loss of water only. To others it calls to mind a state which results from lack of water and simultaneous loss of body fluids such as vomitus. Dehydration therefore requires

FIGURE 2.

Chemical Composition of the Digestive Secretions.



Composition of the Body Fluids: X = Unnamed Basic Radicles.

Y = Unnamed Acidic Radicles.

(After R.A.McCance, Lancet, 1936, 1, 705).

definition and amplification.

Two conditions may result from fluid imbalance, (a) water deprivation, and (b) dehydration. It is of great importance clearly to distinguish between them, although clinically both frequently occur together. Water deprivation results from the inadequate administration of water and is characterised, amongst other features, by oliguria or anuria. All that is required for its correction is the administration of a sufficient quantity of water. Dehydration, on the other hand, is caused by actual loss of secretions derived from the body fluids (blood plasma, interstitial fluid, etc.,) by vomiting, diarrhoea, excessive sweating , etc., and is not accompanied by any reduction in the volume of urine secreted if water administration and assimilation are adequate. Thus patients who are dehydrated have suffered reduction in the total fluid content of their bodies, normally 65 per cent of the total body weight. Since the body fluids are solutions containing electrolytes such as sodium, potassium, chloride, etc. maintained by the kidneys in practically unvarying concentration, dehydration or reduction in body fluid volume can therefore only be prevented or corrected by the administration of solutions containing these electrolytes in proper concentrations and not by water alone.

There are many causes of dehydration such as excessive sweating, diabetes mellitus, etc. but the most important from the surgical point of view, is loss of gastro-intestinal secretions from vomiting or diarrhoea. Figure 2 depicts, in terms of acid-base equivalance, the chemical composition of the digestive secretions, and Table 3 derived from Figure 2 and information supplied by Rowntree[36] and McQuarrie[37], presents estimations of the daily volume of the various digestive secretions of an adult, the possible daily losses of water and electrolytes which may occur from obstructions at various levels, and the resultant effect of such losses on the acid-base balance of the plasma.

It will be seen that, as the result of the loss of gastro-intestinal secretions from obstructions at various levels, enormous losses of water and electrolytes may occur. Indeed, if all the eight liters of the gastro-intestinal secretions are lost, more than twice the volume of the blood plasma may be removed from the body. In spite, however, of these serious losses of secretions, all ultimately derived from the plasma, plasma volume itself is not greatly reduced

until the terminal stages of dehydration approach. This protracted defence of plasma volume is effected by the transference of interstitial fluid to the vascular compartment and persists practically to the point of exhaustion of this reserve. The extent to which intracellular water participates in the defence of plasma volume cannot as yet be definitely stated, but certain generalisations are possible.

Usually starvation coexists with dehydration and consequently some water will be liberated from the cells as the result of the necessary katabolism of the body tissues for energy requirements. Also, if during the developement of dehydration, the osmotic pressure of extracellular fluid rises water will be transferred from the cells to the extracellular fluid to equalise the osmotic pressures on both sides of the cell membrane. Such a sequence of events probably results from the loss of upper gastro-intestinal secretions because, although the volume of fluid lost is considerable, correspondingly less sodium is lost than water. For example, as the result of pyloric obstruction, 4500 cc. of fluid may be lost, all ultimately derived from the plasma. The sodium content of 4500 cc. of blood plasma is 15.3 Gm. (the normal plasma sodium concentration being 340 mg. per 100 cc.). Since the 4500 cc. of gastric juice and saliva lost only contain about 7.14 Gm. of sodium, during their secretion approximately 8.16 Gm. of sodium are left behind in the extracellular fluids, which, if not rapidly excreted by the kidneys, will raise the osmotic pressure of the extracellular fluids and cause the transference of water from the cells to the extracellular fluids. On the other hand, losses from the lower gastro-intestinal tract, such as diarrhoea or the discharges associated with carcinoma of the rectum, have practically the same sodium concentration as the plasma. Almost no alteration in the osmotic pressure of the extracellular fluids will therefore result from the loss of lower gastro-intestinal secretions, and in consequence no transference of water from or into the cells will occur. Thus it appears probable that intracellular water plays a part in the defence of plasma volume after loss of upper gastro-intestinal secretions and no part after the loss of lower gastro-intestinal secretions.

The clinical syndrome of dehydration is characteristic. Its main features are summarised in Table 4. All the clinical features will not necessarily be present in every case but in general the severity of symptoms and the number of signs present will vary with the degree of dehydration.

TABLE 4.

Symptoms and Signs of Dehydration.

Symptoms. Fatigue, thirst, weakness, anorexia. There may be muscular cramps.

Signs. The mentality is dulled. The temporal hollows are accentuated. The cheeks fall in, and the eyes are dull and sunken. The skin is dry and inelastic. There may be pyrexia.

Cardio-Vascular System. The pulse is rapid and thready, and the pulse pressure is reduced. The circulation time is increased and there may be cyanosis.

Changes in the Blood. The red blood cell count and the haemoglobin concentration are increased. Blood viscosity and specific gravity are increased.

Changes in Blood Chemistry. The plasma chloride concentration is nearly always reduced in dehydration resulting from loss of upper gastro-intestinal secretions. It may be unchanged, increased, or decreased in dehydration resulting from loss of lower gastro-intestinal secretions.

The plasma sodium concentration is usually unchanged until dehydration is well advanced. It is frequently reduced after losses of lower gastro-intestinal secretions.

The blood non-protein nitrogen concentration is increased.

The plasma protein concentration is increased if dehydration is severe.

The carbon di-oxide combining power of the plasma may be unchanged, increased, or reduced.

Renal System. There may be oliguria or anuria.
The Urine. Specific gravity increased. Albumin, red blood cells, and casts may be present. The concentration of chlorides in the urine is much reduced, often to less than 100 mg. per liter.

Fluid Therapy in Relation to Surgery.

A brief out-line of the chemistry and metabolims of the body fluids has been presented. The therapeutic implications are many and concerned mainly with the administration of water, sodium chloride, plasma proteins, and the correction of deviations from the normal of the acid-base balance of the body by the administration of either sodium lactate or ammonium chloride. Plasma protein admistration and correction of acid-base balance will not be discussed in this communication except to state that it is now possible to raise the concentration of the plasma proteins to normal by the administration of a calculated amount of plasma by transfusion; and acid-base regulation is now possible by the administration of calculated amounts of sodium lactate for acidosis and ammonium chloride for alkalosis. But, although plasma protein therapy and acid-base regulation will not here be discussed, it must be remembered that effective fluid therapy requires the simultaneous restoration to normal of the water, electrolyte and plasma protein content of the body.

For clarity of description the therapeutics of water and sodium chloride will

be discussed separately, and finally an attempt will be made to integrate the administration of both.

Normal Water Exchange.

The rationale of water administration is based upon the normal water exchange. In health the intake and output of water are so adjusted that the one balances the other, and the water content of the body is maintained at a remarkably constant level. This adjustment of intake and output is known as water balance. The components of normal water exchange are presented in Table 5.

TABLE 5.

Components of Normal Water Exchange.

Available Water.		Excreted Water.	
1. Fluids Drunk.....	800-2000 cc.	1. Water Vaporised.....	800-1500 cc.
2. Food: Diet or Body Tissue. a. Water Content. b. Water of Oxidation.	.....1000-1500 cc.	2. Water of Urine.....	1000-1500 cc.
		3. Water of Stool. about...	100 cc.

(Adapted from W.G.Maddock and F.A.Coller, Journal of the American Medical Association, 1937, 108, 1.)

Available Water.

1. Fluids drunk vary from 800 to 2000 cc. per day, more being drunk in hot than in cold weather.

2. The solid constituents of the diet contain on an average about 70 per cent of water, and, in addition, a further 20 per cent is formed when the food is oxidised for energy purposes. Each gram of solid food ingested therefore provides approximately 0.9 Gm. of water. A normal diet will thus furnish from 1000 to 1500 cc. of water per day. In starvation about 500 cc. of water become available from the tissue katabolism necessary for the energy requirements of the body.

Excreted Water.

1. Water is continually lost from the skin and in the expired air as water vapor. The process of water vaporisation is in part responsible for the regulation of body temperature. Under normal conditions of external temperature, and in states of activity which do not produce visible sweating, approximately 25 per cent of the body heat is dissipated by the vaporisation of water[38], the remaining 75 per cent being lost by radiation from the surface of the body and lungs, and conduction between the body and its surroundings. In order to dissipate one large calorie it is necessary for the body to vaporise approximately 1.7 cc. of water. Thus an adult

whose heat production is 3000 large calories per day (of which 25 per cent, or 750 large calories, need to be dissipated by the process of vaporisation) requires 1275 cc. of water for this purpose. If the external temperature is increased and therefore radiation and conduction decreased, or if the heat production of the body is enhanced by the performance of extra work, or by disease such as thyrotoxicosis, more water will be vaporised in order that the constancy of body temperature may be maintained. Indeed, it has been found that in disease as much as 2500 cc. of water may be excreted per day by the body as water vapor.[30,39,40] The process of water vaporisation is thus the flexible means of increasing or decreasing the heat loss from the body and it has priority over all other means of excreting water since large departures from the normal temperature may be extremely prejudicial or even fatal. It has also been shown that no reduction takes place in the amount of water vaporised until 6 per cent of the body water has been lost.[41] This degree of water deprivation is accompanied by oliguria or anuria, elevation of temperature, and other evidences of severe metabolic derangement. It is evident therefore that the body normally requires from 800 to 1500 cc. of water per day for the purpose of temperature regulation, and that in disease quantities considerably in excess of this may be needed.

2. The volume of water excreted in the urine is that which remains after vaporisation has withdrawn its quota from the water available for excretory purposes. If there has been severe water deprivation there may be none available for excretion in the urine. Anuria will result and urinary waste products will be retained. On the other hand, if the excretion of urine is adequate in volume (from 1000 to 1500 cc. per day), it can be inferred that water intake is adequate, since water vaporisation has already exercised its pre-emptive rights over the water available for excretion. It must be remembered however, that there may be dehydration in spite of an adequate daily excretion of urine. It is essential therefore to ensure that the volume of the body fluids is normal in addition to providing enough water for the daily excretion of from 1000 to 1500 cc. of urine.

3. The loss of water in the stool is usually less than 100 cc. but serious dehydration can rapidly occur from uncompensated diarrhoea or colostomy or enterostomy discharges.

It is evident from the preceeding discussion that the normal water requirements

in health are from 2500 to 3500 cc. per day. To give less than 2500 cc. to a seriously ill patient is to court water deprivation as the amount excreted through the skin may be much greater than normal. To give more than is necessary throws an avoidable strain on the kidneys and in addition carries with it the risk of producing oedema in dependent situations such as the lung bases. The water requirements of each patient therefore should be calculated every day and thereby frequently their recovery will be accelerated and sometimes their death will be prevented.

Practical Applications of Water Balance to Surgery.

It is only during the last seven years that the principles outlined above have been applied to the practice of surgery. Surgery owes an incalculable debt to the inspiration of Professor L. H. Newburgh and to the industry of Professor W. G. Maddock whose researches into the water requirements of surgical patients have placed fluid therapy on an accurate quantitative basis. They have repeatedly emphasised that both pre-operatively, and in the recovery period, unless an accurate record is kept, both of the intake and output of fluids, dehydration frequently occurs. For the maintenance of water balance in surgical patients it is necessary to realise the magnitude of the losses which occur before, during, and after operation.

1. Fluid Losses Before Operation.

A. Water Deprivation before Operation. Once a routine pre-operative measure, starvation and water deprivation have now been almost entirely abandoned. In some cases the very nature of the disease prevents the adequate absorption of water from the gastro-intestinal tract. Such cases demand that their water needs be fulfilled by some other method of administration. In the absence of unusual fluid losses from vomiting, high fever, etc., 2500 to 3500 cc. of water are required daily by adult patients.

B. Fluid Losses before Operation which Result from the Disease Itself. Intestinal obstruction and many other acute abdominal emergencies are accompanied by severe and protracted vomiting or diarrhoea. Osteomyelitis, acute otitis media, high fever, the uraemia of chronic prostatic obstruction, etc., may all cause severe dehydration and water deprivation. As a result many patients will present, in addition to the symptoms and signs of their particular disease, the symptoms and signs of dehydration and water deprivation. Such patients have lost at least

6 per cent of their body weight in water alone[39], and, before any surgical procedure is undertaken, this amount of fluid must be replaced. For example a 60 Kilogram (132 Lbs.) adult who presents the clinical picture of dehydration and who consequently has lost an amount of fluid equal to at least 6 per cent of his body weight, will require at least 3600 cc. (6 pints) of fluid to make good the volume lost. This volume of fluid will only replace that previously lost as the result of his condition. In addition he will therefore require water for the maintenance of normal water balance during his first 24 hours in hospital, 2500 to 3500 cc. in the absence of continued vomiting or excessive sweating. Thus during such a patients first day in hospital 6600 cc. (11 pints) will need to be administered if he is both to be rehydrated and maintained in water balance. This volume of fluid seems enormous but if fluid therapy is to be adequate and conducted on rational lines, such volumes must be given.

2. Fluid Losses During Operation.

In the absence of vomiting or the considerable losses which follow operations such as caecostomy, the fluid losses during operation are those incurred by loss of blood and by sweating.

A. Loss of Blood During Operations. The loss of blood during operations is greater than is generally realised. Maddock and Coller determined the blood losses in patients undergoing a variety of procedures. Table 6, copied from their paper[42], presents their findings.

TABLE 6.

Volume of Blood Loss.

Partial gastric resection.	274 cc.
Excission of thyroglossal cyst.	174 cc.
Repair of inguinal hernia.	147 cc.
Haemorrhoidectomy	8 cc.
Repair of inguinal hernia.	54 cc.
Appendicectomy.	14 cc.
Excission of retroperitoneal teratoma.	546 cc.
Right radical mastectomy.	1272 cc.
Subtotal thyroidectomy.	142 cc.
Subtotal thyroidectomy.	361 cc.
Repair of ventral hernia.	306 cc.

They found that "in general the blood losses were greater when large areas were exposed and there was a more or less continuous oozing of blood than with haemorrhage from spouting vessels." Such blood losses cannot but contribute considerably to shock and dehydration.

2. Loss of Water During Operations. Loss of water during operations and in the immediate post-operative period from sweating and evaporation from exposed tissues

may be very considerable. As much as 705 Gm. of water may be lost by these means during the operation alone[43], visible sweating being a marked feature when excessive losses of water occur. In such circumstances the loss of water in grams per square meter of body surface per hour is from eight to nine times that of normal subjects under basal conditions.[43,44] This remarkable increase in the rate of water loss through the skin is due to a number of causes. Operating room temperature above 80 F., excessive coverings - especially large mackintosh drapes-, straining and moving under the anaesthetic, shock, and inhalation or spinal anaesthetics operate unfavourably. Ether alone or in combination with nitrous oxide greatly increases water loss through the skin. The use of some basal anaesthetic such as avertin, in conjunction only with nitrous oxide materially reduces perspiration.[43] In the recovery period water losses from perspiration can be greatly decreased by reducing the number of coverings of the bed. The old fashioned "ether" bed should therefore be abandoned and the patient returning from the operating room should be placed in a normal bed with one or two blankets. Lighted shock cages and an excessive number of hot water bottles should be discarded as a routine measure.

In the post-operative period there may be considerable losses from vomiting, intestinal fistulas, wound drainage, etc. These naturally vary from case to case but their volume and composition must be taken into consideration when the daily volume and composition of the fluid to be administered is decided upon if water and electrolyte balance is to be maintained.

Summary of Water Balance in Relation to Surgery.

1. Pre-operatively patients must be properly hydrated.

A. In elective operations in which the patient is not dehydrated from any cause the daily intake of fluid should be from 2500 to 3500 cc.

B. If the patient has been dehydrated, as for instance from vomiting, the fluid lost must be made good by the administration of a volume of saline solution at least equal to 6 per cent of the patients body weight.

C. The temperature of the operating room should be about 75 F.

2. Strict haemostasis should always be observed. Large oozing surfaces should be covered with towels soaked in saline and the bleeding controlled by pressure.

3. After the operation the patient should be placed in a normal bed with one or at most two hot water bottles at his feet.

4. The output of all fluids must be accurately measured and charted.

In addition to a moist tongue, the most valuable clinical index of adequate water administration is the volume of urine secreted by the patient. If it is less than 500 to 600 cc. and is highly concentrated, then the patient is suffering from water deprivation. If the patient is anuric, water balance must be definitely established before any other measures are taken to promote the secretion of urine. By the application of these rules, surgical patients can be maintained in water balance, a state which will lower both the morbidity and the mortality of surgical procedures.

Normal Sodium Chloride Exchange.

The importance of sodium chloride in maintaining the normal volume, distribution and structural integrity of the body fluids has been emphasised. It forms an essential component of the diet, normally from 8 to 12 grams being ingested daily. Variations in the intake of salt are very largely a matter of individual taste. In the absence of sweating 0.24 to 0.41 Gm. per day of the salt ingested are lost through the skin[45], and the daily loss in the faeces is approximately 0.2 Gm.[46] The excess of the salt ingested over that lost in the faeces and through the skin, after being used for acid-base adjustments and for the maintenance of body fluid volume, is excreted in the urine which may contain a maximum concentration of about 2 per cent of sodium chloride.[47] Thus salt balance is normally maintained by the body in the face of considerable variations in its intake.

Excessive Ingestion of Sodium Cjloride.

If a small excess of salt over the normal daily intake, such as 10 Gm., is given to an adult in ordinary good health, accelerated excretion by way of the urine will remove it all in 24 hours. If a large excess is given, such as 50 Gm, even normal subjects may find dificulty in excreting it, and water and salt retention may occur. This however only will occur when an inadequate volume of water is available for its excretion. In fact, contrary to the general belief, I am prepared to state definitely that if sufficient water is provided for the formation of an adequate volume of urine, enormous excesses of sodium chloride may be given to normal subjects without retention of salt resulting. In order to prove this contention the following experiment was carried out in four normal adult males.

For 24 hours after admission they were allowed to be out of bed but were

TABLE 7.

Guy Wicktermann, healthy male aged 27.

Date. May. 1938.		3	4	5	6	7	8	9	10
Weight. Kg.		64.12	64.91	66.05	67.58	68.40	67.54	65.83	64.89
Diet.	H_2O content & H_2O oxid.^{N}	cc.	1147	1147	1147	1147	1147	1147	1147
	H_2O drunk.	cc.	400	400	400	3000	3000	3000	3000
	Calories.		2750	2750	2750	2750	2750	2750	2750
	Salt content	Gm.	10.2	10.2	10.2	10.2	10.2	10.2	10.2
I.V.	H_2O content	cc.	2000	2000	2000	2000	2000	2000	-
	Salt content	Gm.	54	54	54	54	54	54	-
Urine	Volume.	cc.	1360	890	570	3890	5792	6464	3625
	Sp. Gr.		1022	1028	1028	1020	1016	1014	1016
	Salt content	Gm.	24.2	16.8	10.6	68.8	107.4	81.2	39.7
Skin.	H_2O vapour.	cc.	1200	1200	1200	1200	1200	1200	1200
	Salt loss.	Gm.	0.3	0.3	0.3	0.3	0.3	0.3	0.3
Faeces.	Weight.	Gm.	340	374	243	-	86	452	533
	Salt loss.	Gm.	0.2	0.2	0.2	-	0.2	0.2	0.2
Total Fluid Intake		cc.	3547	3547	3547	6147	6147	6147	4147
Total Fluid Lost.		cc.	2730	2277	1891	5090	7035	7890	5092
Total Salt Intake.		Gm.	64.2	64.2	64.2	64.2	64.2	64.2	10.2
Total Salt Lost.		Gm.	24.7	17.3	11.1	69.1	107.9	81.7	40.2
Gain/Loss Weight.		Kg.	0.79	1.14	1.53	0.82 *	0.86	1.71 **	0.94

* Definite clinical oedema.

** Oedema no longer present.

given a definite diet of known calorie value and known salt content. The volume of water which would be formed from its oxidation was estimated and a definite volume of water was given to drink. The urine was collected, its specific gravity determined and also its salt content. The faeces were inspected daily and weighed but their salt content was not determined. It was decided to estimate the daily loss of salt in the faeces to be 0.2 Gm. and that through the skin to be about 0.3 Gm. giving a total estimated loss of salt through other channels than the urine of 0.5 Gm. per day. The patient was accurately weighed every morning at 9 a.m.

On the second day the subject was put to bed and in addition to the measures outlined above 2000 cc. of 2.7 per cent sodium chloride solution was administered intravenously daily. Table 7 presents the results of one of these experiments. Careful examination of the results of this experiment reveals the following;

1. An arbitrary amount of 1200 cc. of water was considered to have been the daily insensible perspiration of the subject. Of course this is only an approximation but was probably reasonably close.

2. The water content of the stool was considered to be fifty per cent of its weight.

3. The volume of urine secreted during the period of small fluid administration fell rapidly. The kidneys were called upon to perform work against a steep osmotic gradient. It is an interesting speculation whether it is possible in the healthy human being to produce anuria by the administration of hypertonic solution of salt.

4. For the first three days the weight rose rapidly. This period was that in which little water was administered by mouth and the kidney ~~only~~ theoretically had only 347 cc. of water available for excretion in the urine.

5. During the last three days of the experiment the weight of the patient fell in spite of the fact that the same amount of sodium chloride was being administered to the subject on the first two of these days. But, adequate water was being given for the excretion of the large amount of salt administered. The kidneys had therefore no very severe osmotic gradient to surmount and the excess sodium chloride was therefore excreted.

6. Clinical oedema was present on the fourth day of the experiment and disappeared on the sixth day.

7. The daily plasma sodium chloride concentration did not vary beyond normal limits.

The practical applications of this experiment are important and obvious.

1. Salt and water retention will occur during the administration of saline solutions if an adequate volume of water is not provided for the excretion of the excess salt. The numerous reports in the literature of clinical oedema resulting from the administration of saline solutions to patients usually are the sequel to inadequate coincident water administration.[48,49,50].

2. It is particularly to be noted that excessive salt administration and retention do not result in an abnormally high concentration of sodium chloride in the plasma,[51] and the plasma sodium chloride concentration cannot therefore be used as an index of excessive salt administration.

Inadequate Ingestion of Sodium Chloride.

If the intake of salt is suddenly stopped in normal subjects significant amounts will continue to be excreted in the urine for several days[48,52]. The daily amount excreted however, rapidly decreases and usually by the fourth day is less than 1 Gm. At the same time the plasma sodium chloride concentration remains within normal limits (560 to 630 mg. per 100 cc.). If, in addition to salt deprivation, sodium chloride is lost as for instance by vomiting, the plasma sodium chloride concentration falls below normal and the daily loss of salt in the urine becomes always less than 0.5 Gm. The kidney is thus able directly to conserve the remaining salt in the body.

Ideal Sodium Chloride Intake.

There is really no ideal sodium chloride intake applicable to all sections of the population and to all climates. Certain generalisations are however possible. In temperate climates at normal temperatures a daily intake of from 6 to 10 Gm. is perfectly adequate. It will allow for acid-base adjustments and for a reasonable excess. If however the occupation of the individual entails working strenuously in hot temperatures such as in mines the loss of salt through the skin should be counterbalanced by the ingestion of extra salt. In the tropics the same factors apply. Make good the salt lost through the skin from sweating by taking an extra ration in the diet.

Abnormal Loss of Sodium Chloride.

The body may loose large amounts of salt (1) From the gastro-intestinal tract, and (2) From the skin and in the drainage from wounds. In addition the formation

of ascitic or pleural fluid removes salt from the normal extracellular fluids since such abnormal collections contain sodium chloride in approximately the same concentration as in the plasma. Table 8 presents the concentrations of salt in the gastro-intestinal secretions, the sweat, and the ascitic fluid of heart disease. It will be noted that in general the concentration of salt in these various secretions is about the same as that of the plasma, or from 5 to 6 Grams per liter. Although no accurate information is available concerning the salt content of exudations from wounds it is probable that it also approximates closely to that of the plasma.

TABLE8.

Concentration of Sodium Chloride in Various Body Secretions.

Secretion.	Range of NaCl Concentration Gm./ Liter.	Average NaCl Concentration Gm./ Liter.	Source of Information.
Vomitus.	1.2 - 6.2	3.3	Dick, Coller, Maddock.[52]
Gastro-duodenal drainage.	1.7 - 7.9	5.7	Analysis of 100 twenty four hour undiluted specimens.
Hepatic Bile.	3.5 - 6.4	5.1	Analysis of 30 twenty four hour specimens.
Intestinal fistulous drainage.	4.7 - 4.9 3.1 - 6.6 3.0 - 8.8	5.2	Welch, Masson, Wakefield.[46] Analysis of 2 specimens. Karr and Abbott.[54]
Diarrhoeal Stools.	3.7 - 5.2	4.3	Analysis of 3 specimens.
Sweat.	3.4 - 7.8 1.2 - 4.6 4.3 - 8.3 1.2 - 3.5	4.7	Camerer.[55] Dill, Jones, Edwards, Oberg.[56] Talbert and Haugen.[58] Moss.59.
Ascitic Fluid.	5.0	5.0	Analysis of 1 specimen.
Blood Plasma.	5.6 - 6.3		Atchley, Loeb, Benedict, and Palmer.57.

Clinical Aspects of Salt Depletion.

Any disease associated with the loss of salt containing secretions will lead to depletion of the sodium chloride content of the body and will therefore result in dehydration. Among such conditions are pyloric and intestinal obstruction, acute poisoning, eclampsia and hyperemesis gravidarum, continuous gastro-duodenal drainage for the treatment of peritonitis etc., cholodochostomy, enterostomy, enteritis, ulcerative colitis, infantile diarrhoea. neoplasma of the large bowel and rectum and other diseases in which diarrhoea is a prominent feature, high fever and other conditions in which sweating is profuse. However caused, salt depletion presents

two main problems for solution.

1. The restoration to normal of the salt content of patients depleted as the result of disease before coming under medical care.

2. The prevention of salt depletion in patients while under treatment.

Restoration to Normal of the Salt Content of Patients Suffering from Sodium Chloride Depletion.

The life saving properties of physiological sodium chloride solution are widely recognised, but there can be no doubt that in spite of a quarter of a century of clinical and experimental investigation, the actual administration of sodium chloride remains very largely empirical. Yet the dangers of excessive[60,61,62,63] and also of inadequate salt therapy are well known.[64,65,66] To my knowledge only two attempts have been made to administer sodium chloride quantitatively instead of empirically and by trial and error. The first was by Haden and Orr,[67] who "estimated that in the presence of the toxaemia of intestinal obstruction that sodium chloride as an initial dose of 1 Gm. per kilogram of body weight should be administered." Their suggestion does not seem to have been at all generally adopted as there are apparently no data in the literature relating to its use. The second attempt to administer sodium chloride quantitatively was a definite advance. Falconer and Lyall[68] gave known amounts of sodium chloride to patients suffering from hypochloraemia resulting from vomiting and followed the consequent changes in the plasma sodium chloride concentration. They concluded that " in hypochloraemia about 20 grammes (from 15 to 30 grammes) of salt are required on the average to raise the plasma chlorides by 100 mg. per 100 c.cm." Neither estimate is entirely satisfactory although each constituted a definite advance. How then shall the ideal amount of sodium chloride and water be administered to patients whose body fluids have been depleted by disease? Before attempting this important question it is essential to clarify the aims of fluid therapy.

When sodium chloride solution is administered to a dehydrated patient an attempt is made to restore to normal the water and electrolyte content of his body in order that all those vital processes which are collectively called metabolism may proceed unhindered by alterations in acid-base balance, accumulations of waste products, reduction of circulating blood volume etc. The methods of achieving this

ideal have been to a large extent obscured by the excellence of the defence of blood volume and by the astonishingly accurate control which the body exercises over the pH, osmotic pressure etc. of the body fluids. The defence of blood volume prevents significant alterations in the plasma protein concentration, haemoglobin concentration and red blood cell count until dehydration is well advanced. The preservation of the normal osmotic pressure likewise renders the plasma sodium concentration valueless as an index of the degree of dehydration. Because if the concentration of sodium in extracellular fluid is temporarily increased by loss of water in excess of sodium, the body attempts to restore the normal osmotic pressure of its fluids by the excretion of the excess sodium remaining and by the transference of water from the cells to the extracellular fluids. The concentration of sodium in extracellular fluid therefore remains practically stable and is by itself no clue either to the amount of sodium or water which the body has lost. Similarly although extracellular fluid volume may be increased, sometimes even to the point of causing clinical oedema, the concentration of sodium in the extracellular fluid remains practically unchanged. It therefore allows of no deductions concerning either the amount of sodium or of water which the body has retained. Thus the red blood cell count, the haemoglobin concentration, the plasma protein concentration, and the plasma sodium concentration are all unreliable indices of dehydration.

The concentration of chloride in extracellular fluid is however reduced in dehydration from loss of upper gastro-intestinal secretions. If chloride could be lost without sodium or water, the percentage reduction in the chloride concentration of extracellular fluid would be equal to the percentage decrease in the chloride content of that fluid. This concept expressed in the form of an equation is as follows:

$$\%\ \text{Reduction in chloride concentration of extracellular fluid.} = \frac{\text{Amount of chloride lost} \times 100}{\text{Original total extracellular chlorides}}.$$

In practice, however, when the body looses chloride it also looses sodium and water and in consequence it appears that there should be no direct relationship between the amount of chloride lost and the resulting reduction in the concentration of chloride in extracellular fluid. White and Bridge[69] however, in dogs depleted of chlorides by experimental intestinal obstruction showed "that chloride was lost from the tissues in amounts directly proportional to the decrease in the blood" and "that the total dechlorination of the blood and tissues corresponds closely to the amount of chloride actually recovered in the vomitus and urine." It has also been shown by

Merritt and Fremont-Smith[70] that variations in the chloride concentration of the plasma are accompanied by like variations in the chloride concentration of the cerebrospinal fluid. It is therefore probable that the plasma chloride concentration is a satisfactory index of the chloride concentration in the other extracellular fluids of the body.

From the above it seems reasonable to postulate that if the plasma chloride concentration is 20 per cent below the normal about 20 per cent of the body chlorides have been lost. And , if the normal salt content of the body were known, the administration of an amount of salt equal to the percentage reduction in the normal salt content of the body, as indicated by the percentage reduction in the plasma chloride concentration, should restore the chloride content of the body to normal and therefore also to normal the chloride concentration of the body fluids.

The normal salt content of the human body has been investigated by various workers. Their estimates are presented in Table 9.

TABLE 9.

Total Salt Content of the Human Body.

Authority.	Actual Statement of Authority.	Calculated NaCl content of a 60 Kg. adult. Gm.
Magnus-Levy.[71]	Chlorine = 0.1227 per cent of the body weight.	121.7
Lotka.[72]	Chlorine = 0.16 per cent of the body weight.	158.4
Falconer and Lyall.[68]	The total chloride content of the body is about 90 Grams.	90.0
Sherman.[73]	Chlorine = 0.15 per cent of the body weight.	148.8

The figure given by Sherman is the most recent and authoritative and has been apparently confirmed by our experimental work, and it was therefore adopted as a basis for calculation. Sherman states that the chlorine content of the body is 0.15 per cent of the body weight. Expressed in terms of sodium chloride the salt content of the body equals 0.248 per cent of the body weight, or 148.8 Gm. of sodium chloride in the body of a 60 Kg. adult.

We are now in a position to express in the form of simple equations the number of grams of salt which must be administered to a patient whose body chlorides have been depleted to restore the chloride content of his body to normal and also to normal the chloride concentration of his body fluids.

TABLE 10.

Administration of Sdoium Chloride to 4 Experimental Subjects and 3 Patients with Hypochloraemia.

Subject.	Weight.	Initial Plasma NaCl con.n	Initial Plasma CO2 comb. power.	Formula Calculation Optimal Retention to restore plasma NaCl to 560mg.%	NaCl Given.	NaCl lost during period of adminis.n			NaCl Actually Retained.	Final Plasma NaCl con.n	Final CO2 comb. power of plasma.	Clinical Rule Optimal Reten.n to restore NaCl of plasma to 560 mg. %.
						Urine.	Stool.	Total.				
	Kg.	mg.%.	Vol.%.	Gm.	Gm.	Gm.	Gm.	Gm.	Gm.	mg.%.	Vol.%.	Gm.
R.W.'	56.7	384	75.1	44.2	46.7	1.4	-	1.4	45.3	516	63.6	43.9
S.T.	73.2	436	67.3	40.1	42.8	0.5	-	0.5	42.3	554	56.3	39.9
G.W.'	75.4	308	53.9	84.1	80.1	14.7	?	?	?	531	53.5	83.6
H.A.	76.9	454	49.5	36.1	38.2	0.7	-	0.7	37.5	545	60.7	35.8
J.W.	59.8	447	61.4	29.9	26.2	1.1	-	1.1	25.1	546	58.9	29.7
T.J.	72.7	427	59.9	42.7	40.6	-	-	-	40.6	564	48.0	42.5
W.P.	67.7	436	57.3	37.1	33.7	1.5	-	1.5	32.2	543	58.3	36.9

' R.W. and G.W. are the exceptions mentioned in the text.

1. % of body salt lost $= \frac{(\text{Normal plasma chloride con}^{n} - \text{Actual plasma chloride Con}^{n})100}{\text{Normal plasma chloride con}^{n}}$

2. The total salt content of the body = 0.248 per cent of the body weight.

From 1 and 2,

3. The number of Gms. of salt needed to restore the body chlorides to normal = per cent of the body salt lost X total salt content of the body.

$$= \frac{\text{Normal plasma Cl} - \text{Actual plasma Cl.}}{\text{Normal plasma Cl.}} \times 100 \times 0.248 \text{ \% of body weight (Gm.)}$$

$$= \frac{560^{*} - \text{Actual plasma chlorides}}{560} \times 0.00248 \times \text{body weight (Gm.)}$$

* The normal plasma chloride concentration expressed in terms of sodium chloride varies from 560 to 630 mg. per 100 cc. The lower limit of normal was selected for experimental purposes because (1) It constitutes an absolute criterion of restoration to normal and (2) Failure to reach this standard denotes incomplete restoration, either because of failure on the part of the formula, or, as is pointed out in the text, because some patients are at least temporarily unable to do so.

This rather cumbersome formula is, in fact, much simpler than it appears to be when applied to an actual case. For example, if a patient weighing 60 Kg. has a plasma chloride concentration of 410 mg. per cent, from the formula:

$$\text{The number of Gms. of salt needed to restore his body chlorides to normal} = \frac{560 - 410}{560} \times 0.00248 \times 60{,}000.$$

$$= 39.86 \text{ Gms.}$$

Which if given in the form of physiological saline (B.P. 0.9 Gm. NaCl per 100 cc.) would amount to 4421 cc.

In order to test the accuracy of this formula four normal subjects (R.W., S.T., G.W., and H.A.) and three patients (J.W., T.J., and W.P.) were intentionally depleted of chlorides by continuous gastro-duodenal drainage. When a satisfactorily low plasma sodium chloride concentration had thus been attained, salt was given intravenously in the form of physiological saline in amounts approximately equal to that calculated by the formula to be necessary to restore the plasma sodium chloride concentration to 560 mg. %. The results of these experiments are shown in Table 10* and they demonstrate, with the exceptions of R.W and G.W., the close correlation between the amount of salt actually retained and the calculated optimal retention or formula calculation. Taking into account the differences between the optimal and

* More complete data concerning these experiments are published elsewhere.[48]

TABLE 11.

Administration of Sodium Chloride to 11 Patients with Hypochloraemia.

Subject.	Weight.	Initial Plasma NaCl Conⁿ.	Initial Plasma CO2 comb. power.	Formula Calculation Optimal Gms. Retention to restore plasma NaCl to 560mg.%	NaCl Given.	NaCl Lost during period of administration. Upper G.I. Tract.	Urine.	Stool.	Total.	NaCl Actually Retained.	Final Plasma NaCl conⁿ	Final CO2 Comb. power plasma.	Clinical Rule. Optimal Retention to restore plasma NaCl concentratⁿ.
	Kg.	mg.%	Vol.%		Gm.	Gm.	Gm.	Gm.	Gm.	Gm.	mg.%	Vol.%	Gm.
O.M.	63.3	404	57.6	43.7	55.8	7.3	7.7	-	15.0	40.8	559	52.0	43.4
B.S.	39.0	449	63.6	19.2	28.4	1.2	9.2	-	10.4	18.0	493	67.1	19.0
J.C.	65.5	479	45.7	23.4	27.5	1.4	2.6	-	4.0	23.5	586	44.7	23.3
D.C.	60.4	513	59.8	12.6	15.8	-	1.3	-	1.3	14.5	564	53.9	12.5
D.E.	58.1	345	100.0	55.3	100.6	38.1	6.2	-	44.3	56.3	606	73.0	55.0
L.A.	62.0	372	75.0	51.6	70.1	5.5	14.9	-	20.4	49.7	566	60.0	51.3
C.K.	48.9	356	48.0	44.1	56.8	1.1	0.7	17.9	19.7	37.1	528	49.8	43.9
S.L.	21.8	479	-	7.8	8.7	-	0.9	0.8	1.7	7.0	536	-	7.8
L.B.	54.0	464	-	22.9	25.7	0,3	5.4	-	5.7	20.0	554	-	22.8
B.M.	34.0	437	-	18.5	21.3	-	0.2	-	0.2	21.1	513	-	18.4
G.B.	45.3	352	-	41.7	61.5	4.8	10.9	-	15.7	45.8	510	-	41.6

actual salt retentions, accurate restoration to normal of the plasma sodium chloride concentration was achieved. R.W., our first case, apparently failed to reach a plasma sodium chloride concentration of approximately 560 mg. per cent in spite of an actual salt retention of 45.3 Gm., whereas the optimal salt retention was 44.2 Gm. This we believe was because his plasma sodium chloride concentration was determined only 12 hours after the completion of the salt administration, and in the subsequent cases we found that the plasma sodium chloride concentration frequently continued to rise for a period of 24 to 36 hours after the termination of the salt administration, and did not reach a maximum until after that period. Falconer and Lyall[68] reported the same observation in their series of cases. The case of G.W. was especially instructive because in him water intoxication was produced intentionally and he he is almost certainly the first case of deliberately induced water intoxication in medical literature. After a period of study of the condition of water intoxication 34 Gm. of salt in 1.7 per cent solution in distilled water was administered by Levine tube into his stomach. This cured his water intoxication with dramatic rapidity, but had the unfortunate result that it provoked diarrhoea in which approximately 2000 cc. of salt laden fluid were lost. Thus from the point of view of accurate salt administration for the restoration of depleted plasma chloride concentration, he was not a satisfactory and truly illustrative case.

After the experimental investigation outlined above it was decided to apply the formula to the treatment of diseased patients with hypochloraemia. Eleven such patients were treated and the results of the study are shown in Table 11. They included instances of pyloric obstruction (1), intestinal obstruction (3), rectal polypus with profuse discharge (1), paralytic ileus (2), and patients who had been on gastro-duodenal suctionwithout accurate replacement of the drainage losses with physiological saline (4). Their optimal salt retention was calculated from the formula and approximately that amount of salt was administered intravenously. If the patient was loosing chlorides abnormally, for example by vomiting, during the period of correction of the hypochloraemia, these abnormal losses were replaced with equal volumes of physiological saline solution by the "volume for volume rule" discussed later in this paper. If the patient was dehydrated and the amount of saline given was not sufficient to correct this condition the extravolume required was administered after the termination of the experiment. With the exceptions of the

patients B.S., B.M., and G.B., the results again demonstrate that, taking into account the difference between the optimal and actual salt retnetions, there was a very close restoration to normal of the plasma sodium chloride concentrations. Patients B.S. and G.B. failed to attain a normal plasma sodium chloride concentration, although in each case approximately the optimal salt retention had been achieved. Further salt was administered to both but in neither did the plasma sodium chloride concentration increase appreciably, and in both the excess administered was promptly excreted in the urine. These two cases were apparently unable to attain a higher plasma chloride concentration. The explanation of this occurence is not at the moment clear. Each had been suffering from prolonged hypochloraemia and it is assumed that their bodies and kidneys had become accustomed to a lower electrolyte concentration in their body fluids. After their recovery they were reexamined in six weeks and it was found that their plasma sodium chloride concentration had risen to normal levels, their bodies having apparently become gradually reaccustomed to normal electrolyte concentrations in their body fluids. Patient B.M suffering from intussusception also failed to reach a normal plasma sodium chloride concentration after treatment. In this case at the moment we have no adequate explanation.

From these results in experimental subjects and in patients we believe that the principles outlined provide a reasonable and satisfactory basis for the administration of salt to patients whose body chlorides have been depleted by disease. For reasons as yet undiscovered certain cases appear to be unable to attain a normal plasma sodium chloride concentration. However the majority of patients suffering from hypochloraemia will be restored to normal by the administration of salt on the basis of the formula.

For ease of calculation and because the plasma sodium chloride concentration of 600 mg. per 100 cc. constitutes a good average normal, we advocate that salt administration to patients suffering from hypochloraemia should be based on the following formula:

$$\text{Number of Gms. of NaCl needed to restore body Chlorides to normal.} = \frac{600 - \text{Actual plasma chloride con}^{n}}{600} \times 0.00248 \times \text{Wt.Gm.}$$

From the clinical point of view such a formula is cumbersome and not easily remembered. We have therefore evolved several working clinical rules which make the calculation of salt needs a simple matter.

TABLE 12.

Sodium Chloride Replacement in Patients with Hypochloraemia.

Wt.Lbs.	Plasma Sodium Chloride Concentration in mg.%.							
	320	360	400	440	480	520	560	600
5.	2.6	2.3	1.9	1.5	1.1	0.8	0.4	-
10.	5.3	4.5	3.8	3.0	2.3	1.5	0.8	-
15.	7.9	6.8	5.6	4.5	3.4	2.3	1.1	-
20.	10.5	9.0	7.5	6.0	4.5	3.0	1.5	-
25.	13.2	11.3	9.4	7.5	5.6	3.8	1.9	-
30.	15.8	13.5	11.3	9.0	6.8	4.5	2.3	-
40.	21.0	18.0	15.0	12.0	9.0	6.0	3.0	-
50.	26.3	22.5	18.8	15.0	11.3	7.5	3.8	-
60.	31.5	27.0	22.5	18.0	13.5	9.0	4.5	-
70.	36.8	31.5	26.3	21.0	15.8	10.5	5.3	-
80.	42.0	36.9	30.0	24.0	18.0	12.0	6.0	-
90.	47.3	40.5	33.8	27.0	20.3	13.5	6.8	-
100.	52.5	45.0	37.5	30.0	22.5	15.0	7.5	-
120.	63.0	54.0	45.0	36.0	27.0	18.0	9.0	-
140.	73.5	63.0	52.5	42.0	31.5	21.0	10.5	-
160.	84.0	72.0	60.0	48.0	36.0	24.0	12.0	-
180.	94.5	81.0	67.5	54.0	40.5	27.0	13.5	-
200.	105.0	90.0	75.0	60.0	45.0	30.0	15.0	-
220.	115.5	99.0	82.5	66.0	49.5	33.0	16.5	-

Gms. of Sodium Chloride to the nearest 0.1 Gm.

1. For those accustomed to record the weight of their patients in pounds:

Administer 0.2 Gm. of salt per pound of body weight for every 100 mg. per cent that the plasma chlorides need to be raised to reach the normal.

The last column in Tables 10 and 11 gives the calculated salt needs of the respective subjects based upon this clinical rule. (The normal in calculating salt needs from the clinical rule was for purposes of comparison taken to be 560 mg. per cent.) The close approximation between the calculated optimal salt retentions derived from the formula and the clinical rule is apparent and we therefore have confidence in recommending the use of the clinical rule for practical purposes.

2. For those accustomed to recored the weight of their patients in Kilograms.

Administer 0.5 Gm. of salt per Kilogram of body weight for every 100 mg. per cent that the plasma chlorides need to be raised to reach the normal.

This rule is not quite as close an approximation to the formula as the first clinical rule. It results in the administration of a small excess of salt over the calculated ideal amount. For example in patient O.M.. Table 11, a salt retention of 49.4 Gm. instead of 43.7 Gm. would be calculated necessary to raise the plasma sodium chloride concentration to normal. It is probable, however, that the use of the Kilogram rule will not result in excessive salt administration as the excess would be eliminated by the kidneys.

In order further to simplify salt administration to hypochloraemic patients Table 12 has been constructed, based upon the formula:

$$\text{Number of Gms. of Salt needed to restore body chlorides to normal.} = \frac{600 - \text{Actual plasma Cl con}^{n}}{600} \times 0.00248 \times \text{Body Wt. (Gms.)}$$

By its use the ideal salt retnetion in a patient with hypochloraemia may be readily ascertained if his weight in pounds and his plasma sodium chloride concentration in mgs. per cent is known.

The Administration of Sodium Chloride to Patients who have Lost Lower Gastro-Intestinal Secretions.

In spite of the valuable researches of Hartmann[74], Hoag and Maples[75,76]. and Hamilton, Kajdi and Meeker,[77] and many others the administration of sodium chloride to patients who have lost lower gastro-intestinal secretions cannot as

yet be placed on a completely satisfactory basis. Very frequently, as in infantile diarrhoea, the serum electrolyte picture consists of reduction of water and total base with bicarbonate deficit, while the chloride concentration is normal or even increased. In the light of our present knowledge we believe that a patient suffering from dehydration resulting from the loss of lower gastro-intestinal secretions should receivean amount of physiological salt solution equal to 6 per cent of his body weight, with in addition an adequate amount of 5 per cent glucose solution in distilled water to correct and prevent water deprivation. The problems of water and salt administration to such patients require reinvestigation and researches are planned for this purpose.

The Prevention of Salt Depletion in Patients while Under Treatment.

After operations, especially those on the gastro-intestinal tract, significant amounts of sodium chloride may frequently be lost by vomitingor in the discharges from fistulae, drainage from the common bile duct etc. Also the modern pre and post operative treatment of ileus often requires for a period continuous gastro-duodenal aspiration. Not uncommonly therefore chloride insufficiency develops is such patients while under treatment.

It will be remembered that in Table 8 the sodium chloride concentration in various secretions was presented, and it was noted that on an average it was about the same as that of the plasma or, 5 to 6 Gms. of salt per liter. Such a concentration is less than that of physiological saline and consequently Dick, Maddock, and Coller[53] suggested that sodium chloride deficiency should not develop in patients losing gastro-intestinal secretions if such losses were replaced by equal volumes of physiological saline or Ringer's solution. The investigation discussed below was carried out for the purpose of substantiating or disproving this contention.

Procedure.

Three groups of four patients each were studied. The daily loss of gastro-intestinal secretions and of urine were carefully measured and their salt content determined. The plasma sodium chloride concentration was also determined after the operation and daily thereafter. The patients were also weighed daily. All gastro-intestinal secretion losses for each 24 hours period were replaced in the succeeding day by equal volumes of physiological saline or Ringer's solution. The data obtained in the first two groups are presented in Table 13 (replacement

TABLE 13.

Replacement of Upper Gastrointestinal Secretion Losses with Equal Volumes of Ringer Solution
1 Liter = 7.5 Gm. NaCl.

Patient.	24 Hrs. Ending. 1938	Body Weight. Kg.	G.I. Tract Losses. cc.	Ringer Solution I.V. cc.	Sodium Chloride Losses: Urine. Gm.	G.I. Tract Gm.	Total Gm.	Total to be repl.d Gm.	NaCl Given. Gm.	Plasma Chloride Concentration mg.NaCl/100
W.W.	4-1	81.77	-	-	-	-	-	-	-	538
	5-1	80.00	1960	0	0.73	9.97	10.70	9.97	0	548
	6-1	78.82	1100	2035	0.54	6.88	7.42	6.88	15.36	549
	7-1	77.81	315	1133	2.10	2.03	4.13	2.03	8.55	573
	8-1	76.96	0	318	0.35	0	0.35	0	2.30	561
			3375	3486	3.72	18.88	22.60	18.88	26.21	
J.B.	10-1	73.00	-	-	-	-	-	-	-	596
	11-1	74.50	480	0	3.86	2.65	6.51	2.65	0	545
	12-1	73.23	505	485	5.19	3.30	8.49	3.30	3.66	533
	13-1	72.33	280	522	0.48	1.57	2.05	1.57	3.81	548
	14-1	72.07	0	318	0.32	0	0.32	0	2.40	546
			1265	1325	9.85	7.52	17.37	7.52	9.87	
A.G.	10-1	51.64	-	-	-	-	-	-	-	586
	11-1	-	510	800*	1.61	3.51	5.12	3.51	4.80	520
	12-1	49.65	955	517	1.02	7.18	8.20	7.18	3.90	520
	13-1	48.78	985	945	0.49	7.49	7.98	7.49	7.13	526
	14-1	48.25	930	963	0.34	5.95	6.29	5.95	7.27	502
	15-1	49.38	150	956	0.27	0.88	1.15	0.88	7.22	538
			3530	4181	3.73	25.01	28.74	25.01	30.32	
M.L.	24-1	52.91	-	-	-	-	-	-	-	571
	25-1	52.15	860	0	4.82	5.60	10.42	5.60	0	505
	26-1	49.85	880	835	0.51	4.62	5.13	4.62	6.30	530
	27-1	49.03	770	900	0.17	4.41	4.58	4.41	6.79	535
	28-1	49.76	300	787	0.06	1.26	1.32	1.26	5.93	521
	29-1	49.06	260	317	1.13	1.57	1.70	1.57	2.39	531
			3070	2839	5.69	17.46	23.15	17.46	21.41	

* 300 cc. of Physiological saline and 500 cc. of blood.

TABLE 14.

Replacement of Upper Gastro-Intestinal Secretion Losses with Equal Volumes of Physiological Saline. L Liter = 8.5 Gm. NaCl.

Patient.	24 Hrs Ending. 1938	Body Weight. Kg.	G.I. Tract Losses. cc.	Phys.[1] Saline I.V. cc.	Sodium Chloride Losses: Urine. Gm.	G.I. Tract. Gm.	Total. Gm.	Total to be repl.[d] Gm.	NaCl Given. Gm.	Plasma Chloride Concentration Mg.NaCl per cent
E.K.	18-2	-	-	-	-	-	-	-	-	556
	19-2	49.36	720	0	2.45	2.94	5.39	2.94	0	528
	20-2	47.86	1140	708	1.10	7.50	8.60	7.50	6.03.	528
	21-2	48.26	290	1167	1.06	1.75	2.81	1.75	9.90	540
	22-2	47.67	250	287	1.11	1.49	2.60	1.49	2.44	540
	23-2	47.44	250	478	1.26	1.01	2.27	1.01	4.06	564
			2650	2638	6.98	14.69	21.67	14.69	22.43	
E.M.	10-3	59.06	900	0	1.21	6.46	7.67	6.46	0	490
	11-3	57.25	840	865	1.65	4.94	6.59	4.94	7.35	492
	12-3	56.85	920	835	1.51	5.69	7.20	5.69	7.01	505
	13-3	56.30	1020	922	0.70	5.30	6.00	5.30	7.84	490
	14-3	56.50	0	1030	0.80	0	0.80	0	8.76	513
			3680	3652	5.87	22.39	28.26	22.39	30.96	
M.M.	21-2	42.01	-	-	-	-	-	-	-	571
	22-2	42.62	220	890*	6.85	0.56	7.41	0.56	5.86	533
	23-2	41.32	350	243	1.42	2.23	3.65	2.23	2.07	540
	24-2	41.65	390	354	1.11	2.45	3.56	2.45	3.10	541
	25-2	40.26	0	385	1.53	0	1.53	0	3.27	545
			960	1872	10.91	5.24	16.15	5.24	14.30	
M.A.	5-4	54.46	-	-	-	-	-	-	-	568
	6-4	-	650	0	2.17	4.12	6.29	4.12	0	490
	7-4	53.26	1020	669	3.12	6.40	9.52	6.40	5.96	500
	8-4	51.29	1140	1010	1.59	7.63	9.22	7.63	8.59	512
	9-4	50.87	215	1150	0.98	1.08	2.06	1.08	9.79	535
	10-4	50.92	220	393	0.76	1.14	1.90	1.14	3.34	545
			3245	3222	8.62	20.37	28.99	20.37	27.40	

* 465 cc. of Physiological saline and 425 cc. of Blood.

TABLE 15.

Replacement of Upper Gastro-Intestinal Secretion Losses with Equal Volumes of Physiological Saline Solution Plus 1000 cc. of Physiological Saline During the First 24 Hours.
1 Liter Physiological Saline = 8.5 Gm. NaCl.

Patient.	24 Hrs. Ending. 1938.	Body Weight. Kg.	G.I. Tract Losses. cc.	Phys. Saline I.V. cc.	Sodium Chloride Losses. Urine. Gm.	G.I. Tract. Gm.	Total. Gm.	Total to be repl'd Gm.	Total NaCl Given. Gm.	Plasma Chloride Concentration. mg. NaCl per cc.
T.D.	17-3	66.20	120	1050	3.77	0.80	4.57	0.80	8.91	561
	18-3	65.15	110	120	1.01	0.76	1.77	0.76	1.02	568
	19-3	63.62	335	110	1.33	2.27	3.60	2.27	0.93	578
	20-3	64.03	0	342	0.50	0	0.50	0	2.91	584
			565	1622	6.61	3.83	10.44	3.83	13.77	
E.R.	20-3	47.29	630	1010	0.36	4.50	4.86	4.50	8.60	563
	21-3	45.30	1020	630	0.46	7.53	7.99	7.53	8.36	563
	22-3	44.11	1890	1020	0.13	12.36	12.49	12.36	8.65	553
	23-3	43.21	2500	1920	0.09	19.28	19.37	19.28	16.32	530
	24-3	43.17	3000	2510	1.14	20.76	20.90	20.76	21.33	528
	25-3	42.54	2330	3080	0.41	15.38	15.79	15.38	26.20	611
			11370	10170	1.59	79.81	81.40	79.81	86.46	
L.W.	22-2	58.09	-	-	-	-	-	-	-	579
	23-2	59.22	320	1060	3.08	1.96	5.04	1.96	9.01	543
	24-2	57.09	310	325	9.02	1.82	10.84	1.82	2.76	546
	25-2	54.45	600	314	1.97	4.01	5.98	4.01	2.67	594
	26-2	55.46	0	582	0.64	0	0.64	0	4.91	592
			1230	2281	14.71	7.79	22.50	7.79	19.35	
M.L.	12-4	40.08	-	-	-	-	-	-	-	602
	13-4	40.94	480	1600*	1.33	2.87	4.20	2.87	11.41	578
	14-4	39.08	640	486	4.81	3.69	8.50	3.69	4.13	568
	15-4	38.87	370	632	1.59	2.07	3.66	2.07	5.37	569
	16-4	37.64	390	722	2.12	2.17	4.29	2.17	6.14	558
			1880	3440	9.85	10.80	20.65	10.80	17.05	

* 1050 cc. Physiological Saline Solution and 550 cc. Blood.

with Ringer's solution, 1 liter ≡ 7.5 Gm. Na Cl) and Table 14 (replacement with physiological saline solution U.S.P. 1 liter ≡ 8.5 Gm. NaCl). Careful examination of Tables 13 and 14 will show that a fall in plasma sodium chloride concentration occurred during the post-operative period in spite of the volume for volume replacement of gastro-intestinal secretions by Ringer's or physiological saline solutions. However there was usually an increase in plasma sodium chloride concentration on the last day or two of the period of study, or on the third or fourth post-operative days. In no case was there any evidence of any excessive salt administration such as marked increase in weight, oedema, etc.

In an attempt to prevent the fall in plasma sodium chloride concentration during the first day or two after operation which we had observed in the first two groups of patients, 1000 cc. of physiological saline was administered intravenously immediately before operation to a third group of patients. The data obtained in this study are presented in Table 15. It will be noted that the patients in this group maintained a much more nearly normal plasma sodium chloride concentration during the post-operative period and that the initial drop was not so marked as in the first two groups of patients.

We therefore have confidence in advocating "that patients undergoing operations should receive on the day of operation 1000 cc. of physiological saline intravenously, and that thereafter during their convalescence all abnormal losses of gastro-intestinal secretions should be carefully measured and replaced by equal volumes of physiological salt solution." If, in certain cases, such as patients undergoing caecostomy, enterostomy, etc., it is probable that the losses will exceed 1000 cc. more than 1 liter of physiological saline should be administered.

SUMMARY AND CONCLUSIONS.

Water and sodium chloride administration have been separately discussed because, although their functions depend the one upon the other, their conjoint discussion would be a matter of considerable difficulty. There remains therefore only the necessity of integrating their administration.

1. To Patients Dehydrated by the Loss of Upper Gastro-Intestinal Secretions.

A. Administer intravenously a volume of physiological salt solution containing the calculated amount by formula or clinical rule of salt necessary to restore the plasma sodium chloride concentration to normal.

B. For the daily water needs of the patient give 2500 to 3500 cc. of water if necessary intravenously in the form of 5 per cent glucose solution in distilled water.

C. If the patient loses salt containing secretions either during the period of salt replacement or subsequently, replace all such abnormal losses by equal volumes of physiological saline solution intravenously.

2. To Patients in Normal Water and Electrolyte Balance before Operation.

A. Maintain normal water balance by the daily administration of 2500 to 3500 cc. of water, if necessary intravenously in the form of 5 per cent glucose solution in distilled water.

B. On the day of operation administer in addition 1000 cc. of physiological saline solution intravenously, and thereafter replace all abnormal losses by equal volumes of physiological saline solution intravenously.

3. To Patients Dehydrated by the Loss of Lower Gastro-Intestinal Secretions.

A. Administer intravenously an amount of physiological salt solution equal to 6 per cent of the patients body weight. Thereafter replace all abnormal secretion losses by equal volumes of physiological salt solution intravenously.

B. Maintain normal water balance by the daily administration of 2500 to 3500 cc. of water, if necessary intravenously in the form of 5 per cent glucose solution in distilled water.

* * * * * * * * * * * * * * * * * * *

It gives me great pleasure to acknowledge my debt to and the inspiration of others.To Professor Sir John Fraser in whose wards my early work on water balance was carried out. To Professor D.M.Dunlop who made that work possible. To Professor W.G.Maddock of the University of Michigan for much encouragement and inspiration. And last but by no means least to Robert M. Bartlett with whom I worked a full year in community of purpose.

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