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Sodium Chloride in Surgical Patients

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I. COMPARTMENTAL DISTRIBUTION OF SODIUM CHLORIDE IN SURGICAL PATIENTS PRE- AND POSTOPERATIVELY

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Normal individuals usually tolerate large amounts of intravenously administered isotonic sodium chloride. Those same persons, however, during the immediate postoperative period may develop rather serious complications from relatively slight excesses of saline solutions. The observation that surgical patients developed salt intolerance had been described at the turn of the century by Evans (1911),[1] Trout (1913)[2] and later by Matas (1924).[3] In subsequent years, however, emphasis was directed to the complications resulting from salt loss, and an era of postoperative saline administration ensued.[4,5,6,7,8,9,10] Coller et al (1938) formulated a "clinical rule" aimed at quantitatively replacing depleted saline in surgical patients.[11]

Stimulated by the studies concerning the dangers of chloride deprivation, many surgeons administered saline solution regardless of whether or not losses had occurred, and frequently excessive quantities of sodium chloride solution were administered. It was natural, accordingly, for symptoms of salt retention to become manifest and for rather dangerous clinical sequelae to develop. Since then reports have appeared cautioning against promiscuous administration of saline solutions,[12,13] focusing attention on the fact that the sick surgical patient does not tolerate excess sodium chloride[9,14] and directing efforts to determine criteria for the quantity and type of fluid to administer postoperatively.[15] Wangensteen in 1942 stressed the danger of uncontrolled water and salt administration postoperatively and described a method of determining the fluid status of patients following surgery by gravimetric means.[13]

Coller et al[16] in 1944 retracted the so-called clinical rule for chloride administration and stated that no isotonic saline solution or Ringer's solution should be given during the day of operation and during the subsequent two postoperative days.

Because controlled electrolytic and fluid administration is germaine to uncomplicated postoperative convalescence and since either hypochloremia or excessive chloride administration contribute to the development of clinical complications, it was considered pertinent to investigate the manner by which the body handles a load of sodium chloride. An equal amount of sodium chloride given to the same patients preoperatively and shortly after they were subjected to surgical intervention would permit a comparison of the metabolism of the salt pre- and postoperatively and possibly point out the mechanics of the postoperative salt intolerance.

Methods of Study

A salt tolerance test was utilized which permitted the observance of the metabolism of a load of sodium chloride over a period of twenty-four hours. All tests were performed according to the following routine. The patients were permitted no food during the test period, but salt-free oral fluid was permitted ad lib. The morning of the test (usually three to five days preceding surgery) the morning voided urine was discarded. The patient was then weighed and samples of venous blood withdrawn for the following determinations: serum protein, chloride, carbon dioxide combining power, sodium and hematocrit. Blood samples were obtained also in heparinized syringes as blanks for determining plasma volume and extracellular space. Three milliliters of 0.5 per cent solution of Evans Blue Dye and 20 milliliters of 5 per cent sodium thiocyanate were injected intravenously for plasma volume and thiocyanate space determination. Twenty minutes after the injection, blood samples were gain taken from the opposite antecubital vein for measurement of the dye dilution.

The patient remained in bed, and an

intravenous infusion of 27 grams of sodium chloride dissolved in 3 liters of 5 per cent dextrose and water was administered. An average of four hours was required for the fluid to be given.

Immediately after the completion of the infusion the patients were again weighed, blood specimens withdrawn for chemical determination, and the urine was collected during this period via an indwelling Foley catheter within the bladder and analyzed for chloride content. The chemical analyses of the blood and plasma volume and thiocyanate (available fluid) space were performed again three hours after the completion of the infusion. Urine specimens during this three hour period were measured for total volume and chloride content. Blood and urine analyses were performed again twenty-four hours after the completion of the infusion (twenty-one hours after above urine collection). It was felt that by this method a continuous record of the manner in which the organism handled the 27 grams of sodium chloride for the twenty-four hour period could be ascertained.

From the serum concentration of protein and chloride and the plasma volume and thiocyanate space one can determine the total quantity of circulating protein and the total distribution of the chloride ion within the plasma and interstitial space (the thiocyanate space theoretically measures the extracellular compartment, which represents the space in which chloride is distributed). In this presentation the plasma volume is subtract from the thicyanate volume to permit an evaluation of the interstitial volume. A comparison of the total circulating serum chloride and the total chloride content of the interstitial psaces pre- and postoperatively is thus permitted.

The salt tolerance test was repeated again on the first postoperative day. In two patients the salt tolerance test was performed on the second postoperative day, since it was felt that 27 grams of sodium chloride might be dangerous because of the precarious clinical state of the patient.

General inhalation anesthesia was given to each patient and preanesthetic opiate and barbital. Blood and fluid volume was usually maintained by administration of appropriate solutions during surgery. No saline was given postoperatively except the test dose herein described.

Chemical Methods and Calculations

Serum chloride was performed by the technique of Schales and Schales.[17] Serum protein concentrations were determined by the method of Weichselbaum;[18] serum sodium by an internally compensated Perkins-Elmer flame photometer, and the hematocrit as described by Musser and Weintrobe.[19] The carbon dioxide combining capacity was determined by the gasometric method of Van Slyke and Cullen.[20] Urine chloride concentration was ascertained by the modified Volhard-Harvey titration method.[20] Plasma volume and thiocyanate (available fluid) space was measured by the procedure developed by Gregersen and Stewart[21] adapted for the Evelyn Colorimeter. One post-injection sample was obtained, since Noble and Gergersen have demonstrated that results so obtained vary but little from the method of obtaining several blood samples and projecting the dilution values to zero time.[22] The twenty minute postinjection period for withdrawal of the blood for measurement of dye dilution was taken as the most convenient point to withdraw samples for both T1824 and thiocyanate determination. Noble and Gregersen have shown that at twenty minutes after injection the variation from the multiple sample method is only 1 to 5 per cent.[22] The thiocyanate disappearance continues quite steadily for approximately thirty minutes.[23] At twenty minutes, therefore, a compromise between the above values is achieved which permits estimation of plasma and thiocyanate volume simultaneously. Although these total values may differ slightly from those obtained by multiple sample techniques, the method was used to compare results in the same patient at different times, and therefore comparison of values in the same patient in the different states is permitted. Total circulating plasma proteins were calculated by multiplying the plasma protein concentration with the plasma volume. Total serum water chloride was calculated by determining the chloride concentration in serum water (serum minus protein) and multiplying that value by the total serum water.[24]

The interstitial chloride volume was calculated by multiplying the interstitial volume (obtained by subtracting the plasma volume from the thiocyanate volume) by the extracellular chloride concentration[24] (serum water chloride concentration corrected for Gibbs Donnan effect).

Results

Salt tolerance tests were performed on ten patients pre- and postoperatively. In seven patients plasma volume and thiocyanate space were determined permitting the calculations of total plasma and interstitial volumes and chloride content. The results can be expressed best by describing the values obtained by each component part of this study.

Serum Chloride Concentration

Figure 1 presents the percentage increase of the serum chloride concentration at various times following the intravenous injection of 27 grams of sodium chloride. During the preoperative studies it may be noted that immediately after the completion of the infusion the average increase of the serum chloride concentration is 10.3 per cent above the preinfusion value of 106 milliequivalents per liter. Following this peak during the subsequent three hours there is a gradual drop to an average 4.7 per cent increase above the preinfusion level. During the subsequent twenty-one hours the dscent of the serum chloride concentration continues and approaches the preinfusion value (averaging 107 milliequivalents per liter).

Postoperatively a somewhat different pattern of response to the 27 grams of sodium chloride is reflected by the serum chloride concentration at varying time intervals subsequent to its injection. The peak of the serum chloride level is increased to 9.5 per cent above the preinfusion level and the drop is more gradual, so that at three hours the average value is 7.8 per cent above the pre-infusion level, and it remains 4.9 per cent above the initial concentration twenty-four hours subsequent to the injection.[2]

A diminished hematocrit during the postinfusion periods reflected the hemodilution. A diminution of serum protein concentration occurred usually, but no characteristic pattern of response was observed.

The serum concentration of sodium at the different periods subsequent to the saline injection was determined in four patients, and the distribution simulates that exhibited by the chloride ion.

Alterations in Plasma Volume and Total Serum Chloride.

In order to determine whether any difference exists in the total serum chloride preoperatively from that during the postoperative period analyses of total volume in both periods were compared. Table III reveals that three hours after the infusion, before the patient had been subjected to surgery, the plasma volume was increased an average of 46.9 per cent above the preinfusion level of 2.77 liters. Associated with the increased plasma volume was an increase of the total serum water chloride of 56.1 per cent above the preinfusion total serum water chloride value of 294 milliequivalents. Subsequent to surgical intervention the plasma volume, which was approximately the same as that observed preoperatively (averaged 3.0 liters), increased but 21.3 per cent following the administration of 3 liters of fluid containing 27 grams of sodium chloride. Associated with the lesser expansion of the plasma volume was noted a lesser increase of total serum water chloride postoperatively (19 per cent).

These results imply that postoperatively less saline is retained in the circulatory compartment following the administration of 3 liters of normal saline solution and suggest that the compartmental distribution of electrolyte might be altered. This premise has been substantiated by the following data.

Alterations in Interstitial Volume and Total Interstitial Chloride

Table IV summarizes the average changes which occurred in the interstitial volume and the total interstitial chloride subsequent to the administration of 27 grams of sodium chloride as normal saline pre- and postoperatively. The average interstitial volumes were similar during the pre- and postoperative periods before the intravenous salt solution was administered. Three hours after the completion of the infusion the average interstitial volume had expanded 9 per cent during the preoperative study, while the expansion was greater postoperatively, averaging 16.1 per cent. Similarly, an increased amount of chloride was present in the interstitial space postoperatively. The average postoperative interstitial chloride volume increased 21.9 per cent, whereas the average preoperative expansion of interstitial chloride mass was 14.7 per cent above the preinfusion levels of 1,417 and 1,476 milliequivalents respectively.

Effects of Saline Administration upon Total Serum Proteins

The retention of fluid and electrolytes within the circulation is linked intimately with the concentration of proteins in the serum,[26] and Stewart and Rourke have demonstrated an increased total serum protein content subsequent to saline infusions, which they feel permits circulation of the extracellular fluid between the vascular and interstitial compartments.[27] For this reason the total serum proteins of the patients in this study were determined pre- and postoperatively preceding and three hours after the completion of the saline infusion. The data are summarized in Table V and reveal an average increase in the total serum protein preoperatively of 30.4 per cent above the preinfusion value of 194 grams. Postoperatively a slight increase (7.3 per cent) occurred subsequent to the infusion. The inability of the administered saline solution to elicit a mobilization of protein into the circulation to the same degree that occurred preoperatively is possibly related to surgical trauma[28] and protein depletion, and might be a causative factor permitting the greater increase in interstitial volume and chloride mass postoperatively.

This increased deposition of chloride in the interstitial space postoperatively could enhance the retention of that ion and prevent its deliverance to the kidneys for excretion. To investigate this hypothesis, analyses of the total urine output and its chloride content were made pre- and postoperatively.

Urinary Excretion of Chloride Following the Salt Tolerance Test

Specimens were collected during the interval of infusion, the three hour period immediately subsequent to the termination of the infusion, and the next twenty-one hour period. These specimens were analyzed separately to note whether any differences in the chloride excretion occurred during the three time intervals. Table VI summarizes the urinary volume, urinary concentration of chloride and the total amount of chloride excreted subsequent to the injection of 27 grams of sodium chloride during the preoperative and postoperative periods.

It may be noted that a somewhat greater total volume of urine was excreted postoperatively, as well as during each collecting period. This was somewhat unexpected because usually variable degrees of olugiria occur postoperatively.[29] However, since these tests were performed on the first postoperative day (the second postoperative day in two patients), it is probable that these particular patients had recovered from the immediate postoperative oliguric state. The total fluid intake varied but slightly, averaging 3.68 liters preoperatively and 3.97 liters postoperatively.

It is of particular interest that in the face of a rather parallel urine volume, the concentration of chloride preoperatively exceeded the concentration of urinary chloride postoperatively in each period studied. Preoperatively the urinary concentration of chloride immediately after the infusion avaraged 91 milliequivalents per liter (5.3 grams per cent as sodium chloride), while postoperatively the average was 61 milliequivalents per liter (3.6 grams per cent as sodium chloride). During the three hour period subsequent to the infusion, the preoperative urine chloride concentration was 162 milliequivalents per liter (9.5 grams per cent as sodium chloride) in contrast to the postoperative concentration of 75 millieuivalents per liter (4.4 grams per cent as sodium chloride). During the subsequent twenty-one hour period the preoperative concentration of chloride in the urine averaged 125 milliequivalents per liter (7.3 grams per cent as sodium chloride) in contrast to 73 milliequivalents per liter postoperatively (4.3 grams chloride as sodium chloride).

The increased urinary concentration of chloride preoperatively resulted in an increased preoperative excretion of chloride. The total amount excreted preoperatively averaged 278 milliequivalents (16.3 grams as sodium chloride) in the twenty-four hour period after the commencement of infusion, while postoperatively the total amount excreted in the twenty-four hour period averaged 153 milliequivalents (9.0 grams as sodium chloride) (Table VI). A partial explanation for the diminished chloride excretion postoperatively could be the observation that eight patients lost an average of 756 milliliters of fluid by means of abnormal drainage, as gastric aspiration, etc. Computing chloride loss on the basis that this fluid contains 5 grams of sodium chloride per liter, the postoperative patients lost an additional 65 milliequivalents of chloride. Nevertheless, during the averaged four hour necessary for the infusion to be delivered and the subsequent three hours (before a significant quantity of abnormal drainage could have exerted an influence on the chloride metabolism), the concentration of urinary chloride postoperatively was significantly lower than that observed preoperatively, indicating some derangement of urinary chloride excretion.

This series is too small to permit a significant evaluation of the influence of the extent of the surgery, age of the patient or other factors, upon the observed phenomena. The data do suggest, however, that the extent of surgery is not a significant factor, for patient 1 on whom a total colectomy had been performed, behaved similarly to patients 9 and 10, on whom cholecystectomies had been effected. These data do suggest a somewhat greater salt retention in the higher age brackets.

Discussion

The observation that during the immediate postoperative period patients do not tolerate large amounts of sodium chloride has been well documented.[9,13,14,15] The sequelae which result from excess saline administration are severe[13,30] and may contribute to the demise of the patient. Excessive salt has been administered not uncommonly because many surgeons adopted the now obsolete clinical rule for chloride replacement of Coller[11] or attempted to correct a hypochloremia which was not the result exclusively of chloride deprivation. Other metabolic derangements, such as hemodilution[31], hypoproteinemia,[32] or hypokalemia,[33] may result in a diminished serum chloride concentration.

In an effort to understand better the mechanism whereby an individual can tolerate relatively large quantities of salt solutions normally, but after receiving a general anesthetic and surgical intervention will retain administered saline in an abnormal manner, this series of experiments were undertaken. Attention was first directed to note the effects of anesthesia and surgery upon kidney function. Renal clearance studies were performed preoperatively and immediately after surgery to study the effects of surgery, including anesthesia upon renal plasma flow, glomerular filtration rate, filtration fraction and tubular excretion. Various types of alterations in the above mechanisms were observed, but in no case, except where shock had developed, could intrinsic renal dysfunction be held responsible for producing salt retention postoperatively.[34] Contrariwise, hypochloremia was observed to depress renal function.[35]

This present study was accordingly performed, utilizing a modified salt tolerance test to observe whether a prerenal chloride derangement contributed to the salt retention following surgical intervention. Soffer and his associates have utilized a three hour salt tolerance test to study salt metabolism in Cushing's disease and the response to desoxycorticosterone acetate in normal individuals and patients with Cushing's syndrome.[36] The salt tolerance test utilized in this study was devised to study saline distribution over a longer period of time (twenty-four hours) and in greater detail. Furthermore, it was desired to give as large a load of sodium chloride as could be tolerated safely to patients shortly after radical abdominal operations in an attempt to tax the mechanisms concerned so that derangements might be better observed.

In the preoperative studies the manner in which the salt load is distributed can be summarized as follows. The administered salt remains in the plasma for a relatively short period of time. The peak of the serum concentration of chloride, noted immediately after the completion of the infusion, decreased rapidly in the subsequent hours, and almost reached the preinfusion level twehty-four hours later. The test dose of water and salt three hours after its administration increased the plasma vöIume 46.9 per cent and the interstitial volume 9 per cent, with approximately 35.6 per cent of the administered chloride present in the serum and 46.8 per cent noted in the interstitial space.

Postoperatively the serum chloride concentration does not quite reach the same high peak as observed preoperatively, and the drop is more gradual. At twenty-four hours after the infusion it remained 4.9 per cent above the preinfusion level. In contrast to the preoperative changes a much smaller increment in volume and salt content was observed in the plasma, and a far greater retention of chloride occurred in the interstitial space, which retained 67.1 per cent of the administered chloride and expanded 2.1 liters. Expanded available fluid volumes have been reported following surgery.[37]

The somewhat increased salt mass in the interstitial space might induce hypertonicity in this compartment. If this be so, an influx of water must occur into that compartment in answer to osmotic demands, the fluid coming from the intracellular compartment.[38] To investigate the possible translocation of fluids between the different compartments, in this experiment, studies of total body water were performed utilizing the dilution technique with deuterium oxide as the tracer isotope.

The technique used was that of injecting 25 grams of deuterium oxide and thirty minutes later withdrawing a blood sample. (The values may be somewhat high because it has been demonstrated that equilibrium does not occur until one hour after injection.) The serum sample was permitted to come to equilibrium with hydrogen, and the supernatent hydrogen gas was analyzed for deuterium content with the mass spectrometer. The authors are indebted to Mr. Jack Johnson of the Department of Physiology and Dr. Alfred Nier of the Department of Physics for these determinations. Analyses were performed upon two patients four hours after their infusion during the preoperative state. Table VIII presents the distribution of the body water. Total body water equals 50 per cent and 57 per cent body weight respectively. This fits in well with the findings of Dr. Francis Moore, who observed that water constituted 50.3 per cent of the body weight of women (presented before Halsted Club, Minneapolis, Minnesota, December 3, 1949). It may be noted, however, (Table VIII) that the water under these circumstances was more or less evenly distributed between the intra- and extracellular compartment - an abnormal situation revealing an intracellular dehydration and an interstitial edema. Since it has been demonstrated that under certain conditions the thiocyanate space is larger than the extracellular space as measured by the inulin dilution,[39] the possibility exists that a certain quantity of chloride considered interstitial may be within the intracellular compartment.[38]

Urinary studies of chloride excretion revealed an increased concentration of chloride in all periods during the preoperative period with a resultant total excretion of 278 milliequivalents (60.3 per cent of the administered dose) twenty-four hours after the completion of the saline injection (including the salt excreted during the infusion period). Postoperatively 153 milliequivalents of chloride were excreted (35.5 per cent of the administered chloride). Since man, unlike the dog, normally lags in the excretion of administered sodium chloride,[25,29] this data denotes that the lag is prolonged subsequent to surgical intervention.

It is interesting in this respect that Coller and his associates observed a 46 per cent retention postoperatively following the administration of a salt load similar to the one of this report given at five intervals during the thirty hour postoperative period.[16] Elman et al have noted that when postoperative patients are given 9 grams of sodium chloride per day an average retention of 40 per cent of that administered occurred.[29]

The possibility of losses occurring during surgery without replacement on the surgical day could be a factor for the diminished chloride excretion; but no great disproportion of measured body chloride (plasma and interstitial chloride) between the pre- and postoperative periods was noted.

The diminution of chloride excretion subsequent to operative intervention, accordingly, might be the retention of the anion within the interstitial compartment. Normally saline given intravenously will be distributed between the vascular and interstitial compartment in answer to osmotic demands and the Gibbs Donnan relationship.[38] The unequal distribution observed postoperatively suggests some derangement in the normal distribution ratio. Dissociation between changes in volume of the extracellular fluid and changes in plasma volume occur following dehydration with and without salt loss,[40] Freis and Kenny have demonstrated recently that in an edematous patient with a normal pregnancy, the relationship of plasma to "available fluid" volume remains the same as normal individuals, in contrast to the eclamptic pregnant woman where a deviation from the normal relationship between the compartments occurs. There is a resultant marked disproportion between the "available fluid" which increased without a corresponding increase and not infrequently a decrease in the plasma volume[41].

The observation that a significant quantity of protein is mobilized into the plasma (30.4 per cent increment) at a three hour period subsequent to the infusion of 3 liters of isotonic saline solution preoperatively while subsequent to surgery a much less pronounced response (7.3 per cent increment) occurred, could be an accountable feature producing the observed alteration of fluid distribution. Stewart and Rourke have observed that an influx of serum protein following large saline infusions occurs which maintains a serum protein concentration of nearly 6 grams per 100 milliliters. They interpret this mobilization of plasma protein as a means to increase the oncotic pressure of the circulation in response to the added load of fluid and electrolyte, and to thereby maintain a physiological relationship between the vascular and interstitial compartment.[27]

Abnormalities of fluid and electrolyte distribution are known to occur when plasma proteins are subnormal,[26,30] and certain abnormal states, occurring postoperatively, frequently cannot be corrected until the serum protein concentration approaches normal.[32] Freis and Kenny believe the serious translocations of fluid in eclamptics to be a failure of increased plasma protein to cope with the added fluid load.[41]

The dissimilarity of protein response observed in this study to the same stimulus pre- and postoperatively might be due to a body protein loss, which is a result of surgery. It has been observed that from 10 to 20 grams of nitrogen are excreted per day following surgery,[28,42,43] which apparently represent catabolyzed tissue protein.[43,44,45] The possibility exists also that as a result of the anesthesia and surgery, the body had been metabolically traumatized and the mechanism which elicits the mobilization of plasma protein had been blocked.[46]

The adrenal cortex elaborates hormones capable of decreasing renal electrolyte excretion and enhancing the catabolism of protein. Salt withdrawal normally has been shown to stimulate the adrenal gland with a resultant salt retention and protein catabolism.[47] Salt administration counteracts these mechanisms mediated via the adrenal gland[2] with a resultant salt excretion and protein anabolism.[47] It is possible that the adrenal stimulation as a result of surgical trauma[46] vitiates the action normally induced by a salt load, with a resultant salt retention and blocking the body's ability to mobilize plasma protein.

In summary, this data demonstrates a different pattern of response in the distribution of chloride administered to the same individual pre- and postoperatively. In the postoperative state a larger portion is delivered to the interstitial space, and a disproportion between plasma and interstitial volume develops. Therefore, the interstitial electrolyte is apparently not as readily delivered to the kidney for excretion. Failure of plasma protein mobilization and probably the action of the adrenal' gland may contribute to this mechanism.

Conclusions

1. A salt tolerance test is described which was utilized in ten patients to study the metabolism of 27 grams of sodium chloride administered before and after a major surgical procedure as a means of identifying the nature of postoperative salt intolerance.

2. Preoperatively the high serum chloride noted immediately after the chloride infusion decreased to the preinfusion level in twenty-four hours. Three hours after the infusion the plasma retained 35.6 per cent of the administered chloride, and the interstitial space retained a moderate quantity of the electrolyte (46.8 per cent). Postoperatively an increased serum chloride concentration persisted for twenty-four hours, a smaller amount of chloride was identified in the plasma (11.9 per cent), but a much greater quantity shifted into the interstitial space (67.1 percent) where it was apparently retained, not being delivered readily to the kidneys for excretion.

3. The mobilization of proteins into the serum in response to the salt load preoperatively may have been a factor in retaining the material in the serum. Failure of such a response postoperatively may be an accountable feature, permitting the diffusion of chloride into the interstitial space.

4. A diminished concentration and quantity of urinary chloride was observed postoperatively compared to that noted preoperatively in each period studied.

5. Some of the mechanisms for the different distribution of chloride pre-

and postoperatively and their clinical implications are discussed.

Table III

ALTERATION OF PLASMA VOLUME AND TOTAL SERUM CHLORIDE IN 7 PATIENTS SUBSEQUENT TO THE ADMINISTRATION OF 27 GRAMS OF SODIUM CHLORIDE (463 MILLIEQUIVALENTS OF CHLORIDE) DURING THE PRE- AND POSTOPERATIVE PERIOD

	Preoperative	Postoperative
Average Plasma Volume (Liters) Preinfusion	2.77	3.0
Average Plasma Volume (Liters) 3 Hours Postinfusion	4.07	3.64
% Increase of Plasma Volume	46.9	21.3
Average Serum Chloride (Milliequivalents) Preinfusion	294	289
Average Serum Chloride (Milliequivalents) 3 Hours Postinfusion	459	344
% Increase of Serum Chloride	56.1	19

Table IV

ALTERATION OF INTERSTITIAL VOLUME AND TOTAL INTERSTITIAL CHLORIDE IN 7 PATIENTS SUBSEQUENT TO THE ADMINISTRATION OF 27 GRAMS OF SODIUM CHLORIDE (463 MILLIEQUIVALENTS OF CHLORIDE) DURING THE PRE- AND POSTOPERATIVE PERIOD

	Preoperative	Postoperative
Average Interstitial Volume (Liters) Preinfusion	12.2	12.4
Average Interstitial Volume (Liters) 3 Hours Postinfusion	13.3	14.5
% Increase of Interstitial Volume	9	16.1
Average Interstitial Chloride (Milliequivalents) Preinfusion	1,476	1,417
Average Interstitial Chloride (Milliequivalents) 3 Hours Postinfusion	1,694	1,728
% Increase of Interstitial Chloride	14.7	21.9

Table V

ALTERATION OF TOTAL CIRCULATING SERUM PROTEIN IN 7 PATIENTS SUBSEQUENT TO THE ADMINISTRATION OF 27 GRAMS OF SODIUM CHLORIDE IN 3 LITERS OF FLUID DURING THE PRE- AND POSTOPERATIVE PERIOD

	Preoperative	Postoperative
Average Total Serum Protein (Grams) Preinfusion	194.0	206.0
Average Total Serum Protein (Grams) Postinfusion	253.0	221.0
Average % Increase	30.4	7.3

Table VI

THE AVERAGE URINARY EXCRETION OF CHLORIDE IN 10 PATIENTS SUBSEQUENT TO THE INTRAVENOUS ADMINISTRATION OF 27 GRAMS SODIUM CHLORIDE (463 MILLIEQUIVALENTS OF CHLORIDE) AS AN ISOTONIC SOLUTION PRE- AND POSTOPERATIVELY

Hours after infusion	Preoperative	Postoperative
Average Urine Volume (Milliliters)		
Immediately	607	950
3 hours	248	559
24 hours	1,470	1,490
Total	2,325	2,999
Concentration of Chloride in Urine (Milliequivalents per liter)		
Immediately	91	61
3 hours	162	75
24 hours	125	73
Total Urinary Chloride (Milliequivalents)		
Immediately	68	49
3 Hours	36	31
24 Hours	174	73
Total	278	153

Table VII

DISTRIBUTION OF CHLORIDE 3 HOURS AFTER THE INTRAVENOUS ADMINISTRATION OF 27 GRAMS OF SODIUM CHLORIDE (463 MILLIEQUIVALENTS OF CHLORIDE) PRE- AND POSTOPERATIVELY

	Preoperative	Postoperative
Total Increment of Serum Chloride (Milliequivalents)	165	55
% of Administered Dose	35.6	11.9
Total Increment of Interstitial Chloride (Milliequivalents)	217	311
% of Administered Dose	46.8	67.1
Excreted Chloride in Urine* (Milliequivalents)	97	53
% of Administered Dose	20.9	11.4

* Calculated for patients on whom volume studies were performed.

Table VIII

DISTRIBUTION OF BODY WATER 4 HOURS AFTER ADMINISTRATION OF 3 LITERS 0.9 PER CENT SODIUM CHLORIDE

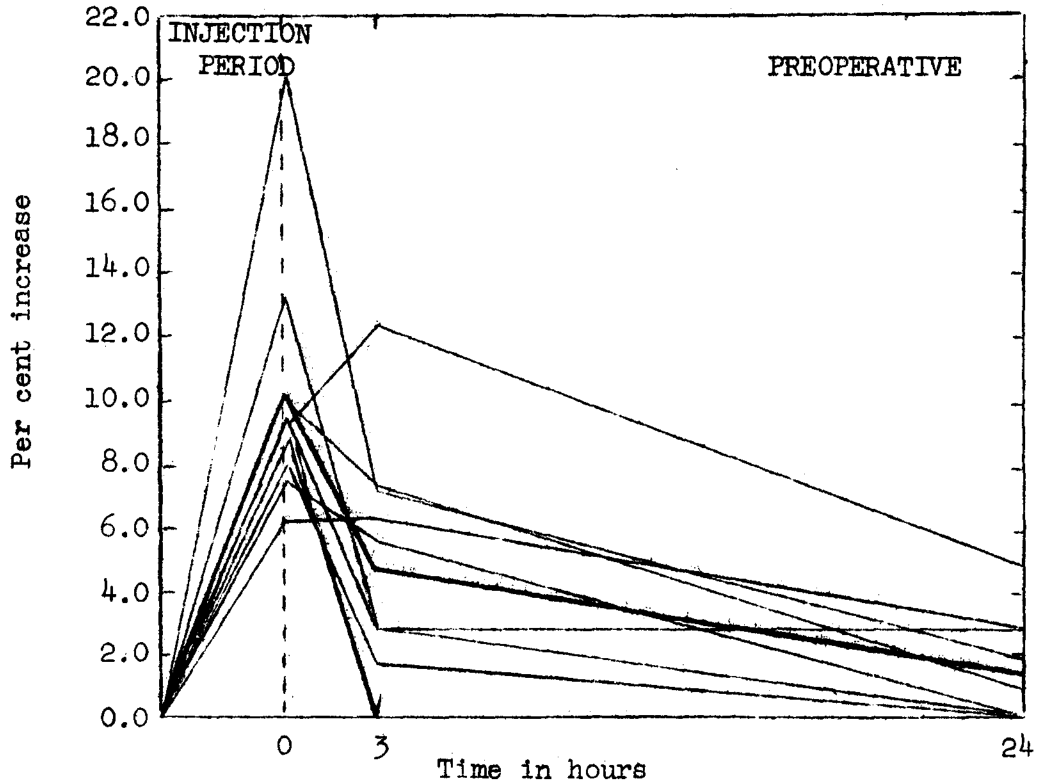
Case No. Table I	Total Body Water (Liters)	% of Body Wt.	Extra-Cellular Water* (Liters)	% of Body Wt.	Intra-Cellular Water** (Liters)	% of Body Wt.
9	32.1	50	16.7	26.0	15.4	24.0
10	33.3	57	16.7	28.3	16.6	28.1

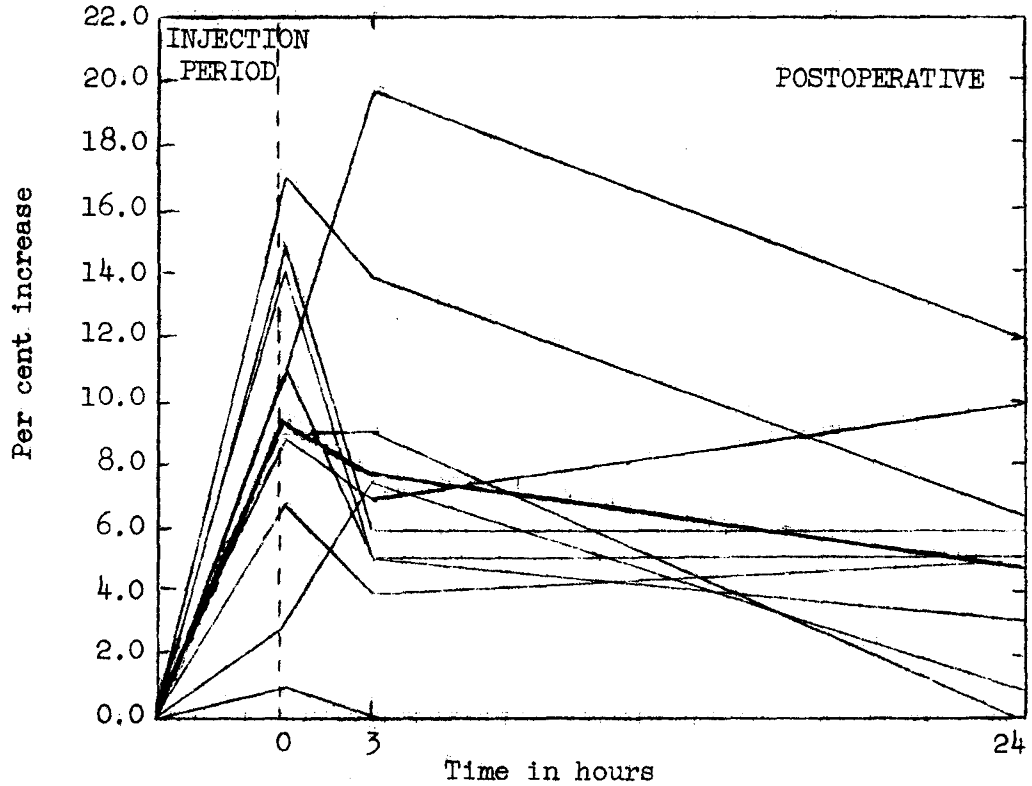
* Thicyanate space

** Extracellular water subtracted from total body water

Figure 1

THE PERCENTAGE INCREASE OF THE SERUM CHLORIDE AT VARYING TIME INTERVALS FOLLOWING THE INTRAVENOUS ADMINISTRATION OF 27 GRAMS OF SODIUM CHLORIDE IN 3 LITERS OF 5 PER CENT DEXTROSE IN DISTILLED WATER





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II. MEDICAL SCHOOL NEWS

Coming Events

January 6 - Clarence M. Jackson Lecture, Tinsley R. Harrison, Southwestern Medical College, Dallas, Texas - "The Evaluation of Cardiac Murmurs" - Medical Science Amphitheater, 8:15 p.m.

January 26-28 - Continuation course in Pediatrics for General Physicians.

January 30-February 11 - Continuation course in Neurology for Internists, Psychiatrists, and Pediatricians.

February 16-18 - Continuation course in Cancer for General Physicians.

February 16 - E. Starr Judd Lecture - "Growth in the Field of Anesthesia" - Dr. Henry K. Beecher, Harvard University Medical School; Museum of Natural Science Auditorium, 8:15 p.m.

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University Receives McClure Bequest

Almost one-half million dollars was presented to the University of Minnesota on December 23, 1949 from the estate of the late Silas McClure. The gift, which was accepted for the University by President James L. Morrill, is to be used for medical research.

Mr. McClure, a Minneapolis businessman who died in February, 1949, had previously given the University funds for medical research in the memory of his late wife, Katherine McClure.

Mr. McClure had been active in business in Beaver Dam, Wisconsin, before coming to Minneapolis in 1921. He was President of the Electric Machinery Manufacturing Company in this city until his retirement in 1944.

III.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

January 8 - January 14, 1950

No. 272

Sunday, January 8

9:00 - 10:00 Surgery Grand Rounds; Station 22, U. H.

10:30 - 11:00 Surgical Conference; Re-exploration of Colonic Cancers; F. J. Lewis; Rm. M-109, U. H.

Monday, January 9

8:00 - Fracture Rounds; A. A. Zierold and Staff; Ward A, Minneapolis General Hospital.

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; M-109, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:00 - 11:50 Physical Medicine Seminar; E-101, U. H.

11:00 - 11:50 Roentgenology-Medicine Conference; Veterans Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Eustis Amphitheater, U. H.

12:00 - 1:00 Physiology Seminar; Use of Ultrasonic Echoes for Detecting Tissue Density Changes and Measuring Tissue Thickness; J. J. Wild; 214 M. H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - 1:20 Pathology Seminar; 104 I. A.

12:30 - 1:30 Surgery Problem Case Conference; A. A. Zierold, C. Dennis and Staff; Small Classroom, Minneapolis General Hospital.

1:30 - 2:30 Surgery Grand Rounds; A. A. Zierold, C. Dennis and Staff; Minneapolis General Hospital.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Public Health Seminar; Subject to be announced; 113 Medical Sciences.

4:00 - Medical-Surgical Conference; Bldg. 1, Main Conference Room, Vets.Hosp.

5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; D. Creevy, O. J. Baggenstoss and Staffs; M-109, U. H.

8:00 - Clinical Research Club Meeting; In Vitro Studies of Some of the Newer Antibiotics, Dr. Burton Waisbren (Dept. of Medicine); Treatment of Cerebrovascular Accidents by Cervical Sympathetic Block, Fredrick Van Bergen (Dept. of Anesthesia); Construction and Use of an Intra-Arterial Transfusion Apparatus, Claude Hitchcock (Dept. of Surgery); Business Meeting - election of officers; Eustis Amphitheater, U. H.

Tuesday, January 10

8:00 - 9:00 Fracture Conference; Auditorium, Ancker Hospital.

8:15 - 9:00 Roentgenology-Surgical-Pathological Conference; Craig Freeman and L. G. Rigler; M-109, U. H.

8:30 - 10:20 Surgery Seminar; Small Conference Room, Bldg. I, Veterans Hospital.

9:00 - 9:50 Roentgenology Pediatric Conference; L. G. Rigler, I. McQuarrie and Staffs; Todd Amphitheater, U. H.

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and E. T. Bell; Veterans Hospital.

12:30 - Pediatric-Surgery Rounds; Drs. Stoesser, Wyatt, Chisholm, McNelson and Dennis; Sta. I, Minneapolis General Hospital.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

1:00 - 2:30 X-ray Surgery Conference; Auditorium, Ancker Hospital.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III, Veterans Hospital.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.

3:30 - 4:20 Clinical Pathological Conference; Staff; Veterans Hospital.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

5:00 - 6:00 X-ray Conference; Presentation of Cases by Veterans Hospital Staff, Doctors Fink, O'Loughlin, et al; Todd Amphitheater, U. H.

5:00 - 6:00 Porphyrin Seminar; C. J. Watson, Samuel Schwartz, et al; Powell Hall Amphitheater.

Wednesday, January 11

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

8:30 - 9:30 Clinico-Pathological Conference; Auditorium, Ancker Hospital.

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans; Room 1AW, Veterans Hospital.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker; Veterans Hospital.

Wednesday, January 11 (Cont.)

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Medicine Case; O. H. Wangensteen, C. J. Watson, and Staffs; Todd Amphitheater, U. H.

12:00 - 1:00 Radio-Isotope Seminar; Halvor Vermund; 113 Medical Sciences.

3:30 - 4:30 Journal Club; Surgery Office, Ancker Hospital.

4:00 - 5:00 Infectious Disease Rounds; Veterans Hospital, Main Conference Room, Bldg. 1.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; E-101, U. H.

Thursday, January 12

8:30 - 10:20 Surgery Grand Rounds; Lyle Hay and Staff; Veterans Hospital.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-109, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:50 Surgery-Radiology Conference; Daniel Fink and Lyle Hay; Veterans Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

11:30 - 12:30 Clinical Pathology Conference; Steven Barron, C. Dennis, George Fahr, A. V. Stoesser and Staffs; Large Classroom, Minneapolis General Hospital.

12:00 - 1:00 Physiological Chemistry Seminar; To be announced; 214 M. H.

1:00 - 1:50 Fracture Conference; A. A. Zierold and Staff; Minneapolis General Hospital.

2:00 - 3:00 Errors Conference; A. A. Zierold, C. Dennis and Staff; Large Classroom, Minneapolis General Hospital.

4:15 - 5:00 Bacteriology and Immunology Seminar; Immunologic Studies on Mouse Mammary Cancer; David Imagawa; 214 M. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

5:00 - 6:00 X-ray Seminar; Review of Meeting of Radiological Society of North America; Todd Amphitheater, U. H.

7:30 - 9:30 Pediatrics Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Tempory West Hospital.

Friday, January 13

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

Friday, January 13 (Cont.)

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:20 Medicine Grand Rounds; Veterans Hospital.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:00 - 12:00 Surgery-Pediatric Conference; C. Dennis, O. S. Wyatt, A. V. Stoesser, and Staffs; Minneapolis General Hospital.

11:45 - 12:50 University of Minnesota Hospitals General Staff Meeting; Experimental Studies on Cardiovascular Disease--Rheumatic Type; T. R. Hamilton and J. T. Syverton; Powell Hall Amphitheater.

12:00 - 1:00 Surgery Clinical Pathological Conference; Clarence Dennis and Staff; Large Classroom, Minneapolis General Hospital.

1:00 - 1:50 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

1:00 - 3:00 Pathology-Surgery Conference; Auditorium, Ancker Hospital.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

3:00 - 4:00 Neuropathology Conference; F. Tichy; Todd Amphitheater, U. H.

3:00 - 6:00 Demonstrations in Cargiovascular Physiology; M. B. Visscher, et al; 301 M. H.

4:00 - 5:00 Clinical Pathological Conference; A. B. Baker; Todd Amphitheater, U.H.

4:15 - 5:15 Electrocardiographic Conference; G. N. Aagaard, Reuben Berman, and Ernst Simonson; 106 Temp. Bldg., Hospital Court, U. H.

5:00 - 6:00 Otolaryngology Seminar; Todd Memorial Room, U. H.

Saturday, January 14

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; M-109, U. H.

8:00 - 9:00 Pediatric Psychiatric Rounds; Reynold Jensen; 6th Floor, West Wing, U. H.

8:00 - 9:00 Surgery Literature Conference; Clarence Dennis and Staff; Small Classroom, Minneapolis General Hospital.

8:30 - 9:30 Surgery Conference; Auditorium, Ancker Hospital.

9:00 - 11:30 Neurology Conference; Hypothalamic Syndromes; Veterans Hospital Annex.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-221, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, U. H.

Saturday, January 14 (Cont.)

9:00 - 11:30 Surgery-Roentgenology Conference; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:00 - 12:00 Anatomy Seminar; Species Distribution of the Satellite Body; Harold Brody; Cytological Studies of Developing Muscle; Richard H. Swigart; 226 I. A.