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Intravenous Therapy In Surgery

An Unscientific and in the Nude Approach

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Introduction

SINCE the second year of medical school the writer has taken an extra interest in fluid and electrolyte metabolism. This resulted in a horrendous collection of notes, papers, reprints, the perusal of a few books, and the euphoric feeling of having some "know how" on the subject. Mea culpa! What a herculean blow was struck, when, once exposed to actual cases, the discovery was made of how little I knew, and of the paucity of practical values I had extracted from my chemical sojourns.

The reason (besides decerebrate thinking), probably lies in the fact that in reading material on this matter, one becomes so engrossed and tied up with the intricate (though often necessary) descriptions of physio-chemical processes, that practical conclusions are lost or difficult to form. This is what discourages the practitioner.

Before the start of the interne year I resolved to sift from the aforementioned collection a working and practical approach to therapy. Still knowing very little, I managed to draw up a fairly simple method that helped me greatly during the first two months of interning. It was from case experiences then that this article evolved into a practical plan.

The result is contained herein. What you will read is so unscientific that it would have made Pavlov's dogs stop barking. Like Gypsy Rose Lee during an encore, this paper has not only been stripped of the clothing of detailed facts but certain points are pushed to the fore, so that essentials are uncovered rather than left to the imagination. It is admitted, however, that the result is probably not as pleasing to the eye as Miss Lee.

The paper is presented in two parts:—the first contains some basic facts and it is here that the greatest stripping was done—so much so that certain statements may appear erroneous—this is one of the hazards of such a mode of presentation. In the second part treatment is discussed and you will note the effort, redundant at times, to offer a step-wise method that may be easily retained and most of all, I hope, easily applied when you are at the ward desk ready to order treatment. Again, the effort for simplicity will probably leave the contents open to criticism from the more pedantic.

For treatment of certain common surgical disorders I have outlined prescriptions. It is not intended that these should be used exactly for such cases you may handle. Rather, they are more of a guide, planned to show what solutions can be used in such situations and how you may calculate and draw them up for administration. Actually, as stated later in the paper, one can handle the majority of cases with four types of solutions.

One further word of defense—This subject of fluids and electrolytes, since its inception, has paraded around with an awesome aura of mysticism. Journals report complicated cases and yet almost every author confesses that

the case was made complicated because of the fact that basic principles were ignored at the outset of treatment.

Abbreviations are used throughout the paper. Sodium, potassium, chloride and the Carbon Dioxide Combining Power are Na, K, Cl, and CO_2 C.P. respectively. The word saline denotes isotonic or physiological, or, as some call it, normal saline. One-sixth molar sodium lactate is given as 1/6 molar lactate. The following are shortened: dextrose to "D", i.e., 10% D/W signifies 10% dextrose in water, or 5% D/S denotes 5% in saline; kilogram will appear as Kgm and liter (1000 cc) as L. The term milliequivalents per liter will be represented as mEq/L.

PART I

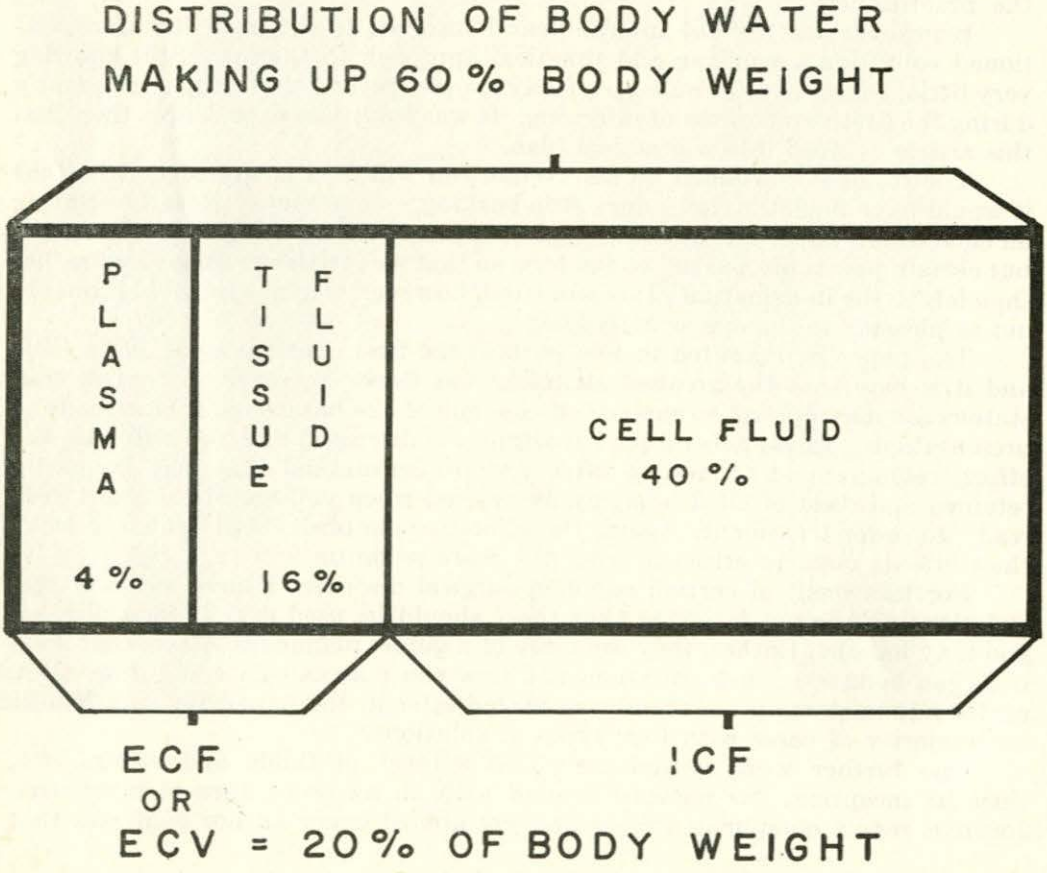
Basic Facts

These essentials are discussed under the six following headings:

1. **Body Fluids**—About 60% of the body weight is water which is distributed into various compartments of the body:

—40% within cells—the intracellular fluid (ICF)

—20% outside the cells—the extracellular fluid (ECF); the ECF is made up of fluid in two nearly chemically identical compartments—16% as the fluid in the interstitial or tissue spaces, and 4% as the plasma fluid. This 20% of the body weight as ECF is generally referred to as the extra-cellular volume (ECV).



Thus in a 70 kilogram man (knowing that 1000 cc of water weighs one kilogram), there are:

60% of 70 = 42 liters of water, of which
40% of 70 = 28 liters are in the cells
16% of 70 = 11.2 liters are in the interstitial spaces
4% of 70 = 2.8 liters is plasma fluid.

Each of these compartments contains electrolytes and each has its own particular composition of these. The important thing to remember is that the pattern of the plasma electrolytes (which is really what the laboratory reports), generally reflects the pattern of the interstitial fluid, since these two intimately interchange through the capillary membrane. The state of the plasma electrolytes THEREFORE PROVIDE SOME IDEA OF THE STATUS OF THE ENTIRE EXTRACELLULAR FLUID COMPARTMENT, which is the clinically important and guiding compartment. The importance of this point, to which there are a few exceptions, will be shown later.

2. **Daily Fluid Losses**—The following are average route losses: urine—900 to 1500 cc: skin and lungs—600 to 900 cc (insensible losses); feces—100 to 200 cc. Generally 2500 cc are regarded as the total fluid output/24 hours and thus the required daily intake.

3. **Electrolytes**—The laboratory reports values in the serum of the blood sent for determinations; as stressed above these values can be regarded as those of the entire ECF. Electrolytes are reported as milliequivalents per liter (mEq/L) of serum, or really, for practical purposes, of ECF. The average values are:

Cations		Anions	
Na	142	HCO_3	27
K	5	Cl	103
Ca	5	SO_4	1
Mg	3	HPO_4	2
	—	Organic Acids	7
	155	Protein	15
			—
			155

Note the chemical equality of the *number* of cations and anions. The value of 27 Meq/L for HCO_3 is referred to as the CO_2 combining power and reflects the amount of available base in the fluid compartment. Values for the electrolyte pattern of the intracellular fluid (ICF) are not given since we cannot measure these practically; however one should have an idea of these and look them up.

4. **A Word About Milliequivalents**—Some people ask, "Why use milliequivalents instead of mgm% and so add technical confusion?" To answer, one writer puts it, "We are not so much concerned with how much ions weigh (mgm%) but rather how many ions there are (mEq/L)". If six internes who weigh collectively 1000 pounds, call up a nurses' residence for dates, they don't ask for 1000 pounds of female (not in these parts anyway), but rather for six females. Thus there will be an equal number of cat-ions reacting with m-anions, regardless of what each individual weighs!

Applying this:

(a) the atomic weight of Na is 23
—23 grams of Na is one equivalent, or dividing by a thousand
—23 milligrams of Na is one milliequivalent.
(b) the atomic weight of Cl is 35, but nevertheless
—35 grams of chloride is one equivalent and
—35 milligrams of chloride is one milliequivalent

Thus chemically speaking, one unit of N and Cl are equivalently equal although their atomic weights differ. The important thing to note is the valence. For instance, a bivalent ion represents two equivalents. To wit: calcium has an atomic weight of 40 and a valence of 2; it therefore can combine with two ions, or rather equivalents of chloride which is monovalent. Thus 40 milligrams of calcium is 2 milliequivalents since this amount would combine with 2 milliequivalents of chloride.

The rule becomes, to change mgm% to mEq/L, use the following formula:

$$\frac{\text{mgm\%}}{\text{atomic wt.}} \times 10 \times \text{valence} = \text{mEq/L}$$

Here are a few handy values when speaking of absolute amounts rather than concentration:

1 gram of NaCl represents 17 mEq of Na and Cl
1 gram of KCl represents 13 mEq of K and Cl
If the CO_2 C.P. is reported as volumes %, divide by 2.22 to obtain mEq/L.

5. **Composition of Gastrointestinal Fluids**—In surgery the patient with a G.I.T. lesion presents the commonest situation when fluid and electrolyte balance are required. Therefore one should have a good idea of the following values since they offer a guide in the selection of electrolyte solutions for therapy—if we have an idea of what is lost in a quantity of vomitus, we can replace it by selecting a solution (or a mixture of such) with an electrolyte pattern approximating that of the fluid lost.

Study this Diagram:

From the diagram we can say that fluids of the G.I.T.:—
(a) above the pylorus contain a greater concentration of Cl than Na and a considerable amount of K; in irritative conditions of the stomach, the ratio of Cl to Na may be as high as 4:1 as compared to the resting state ratio of 3:2.
(b) below the pylrous, contain a greater concentration of Na and a large amount of K.

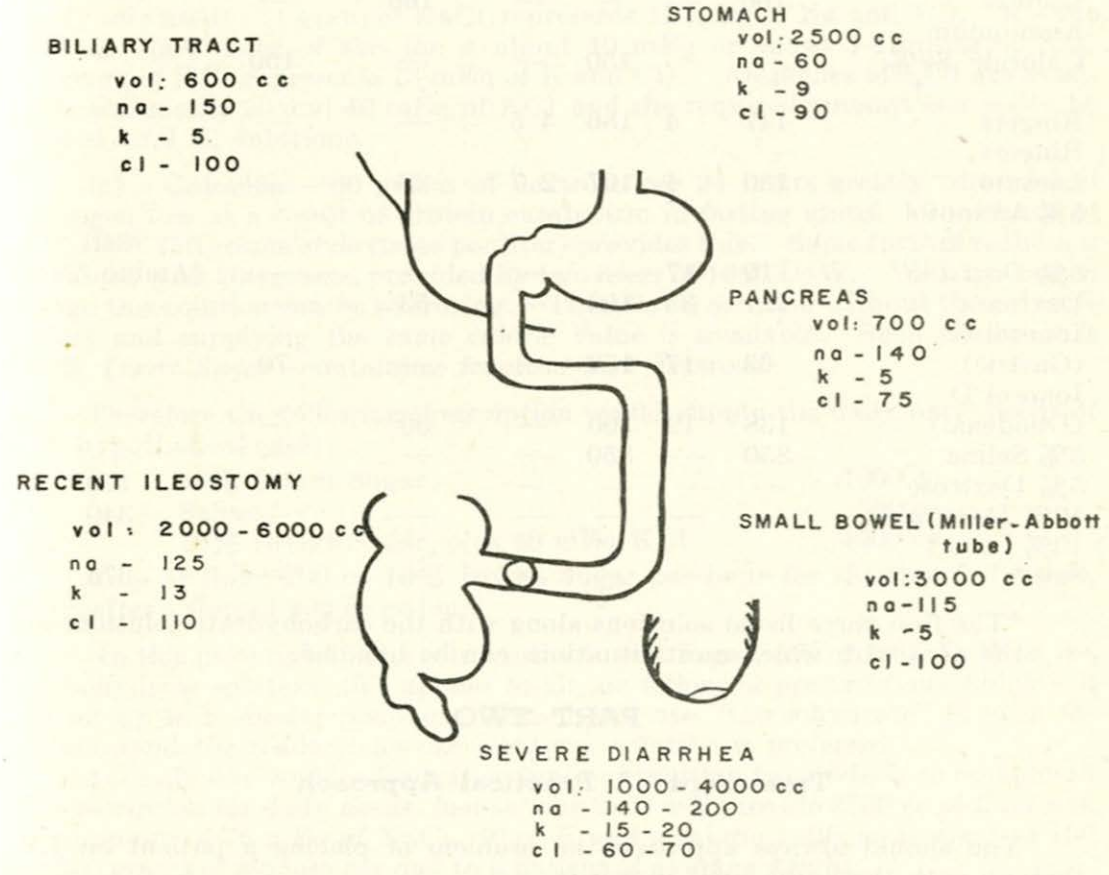
The distinction is not this clear cut. Sometimes in losses from the duodenum, i.e., suction, the losses of Na and Cl are about equal. The pylorus was taken as an arbitary point of division to drive home the point of the differences between fluids of the upper and lower G.I.T.

The therapeutic conclusion is obvious. Where Na losses exceed that of Cl, we select a fluid with a relatively greater concentration of Na; the reverse holds with excessive Cl losses.

6. **Available Repair Solutions**—An impressive array of repair solutions is available on the market. Some companies have solutions "tailored" to meet the need; for instance, Abbot puts out a solution called "IONOSOL G" and this is referred to as their "gastric" solution as it has an electrolyte composition approximating that of gastric fluid. Use of these polyionic solutions is not

COMPOSITION OF GASTROINTESTINAL (G.I.T) FLUIDS

A simple diagram giving the volume of secretions per, 24 hours and the concentration of each electrolyte in meq per liter of secretion fluid.



(in obstruction and irritative condition of the stomach, the cl concentration of the gastric fluid may be as high as 150 meq.)

practical in smaller hospitals. The original manuscript of this paper included these solutions in the treatment prescriptions; however, since I have used them very little, and then only for the sake of experience and as an exercise in matching deficits, they are omitted. They are listed in the table only to familiarize the reader with them.

One can handle the majority of cases with saline, one-sixth molar lactate, 0.89% ammonium chloride, ampoules of KCl and dextrane or invert sugar.

Table listing electrolyte solutions with ion concentrations given as milliequivalents per liter

Solution	Na	K	Cl	Ca	Lactate	NH_4	Calories
Saline (Isotonic	155	—	155	—	—	—	
$\frac{m}{6}$-Sodium Lactate	166	—	—	—	166	—	
Ammonium Chloride .89% *	—	—	150	—	—	150	
Ringers	147	4	156	4.5	—	—	
Ringers Lactate	130	4	107	2.7	28	—	
5% Aminosol in 5% Dextrose	10	17	—	—	—	—	340 (Amino Acids)
Darrow's	121	35	103	—	53	—	
Ionosol G (Gastric)	63	17	150	—	—	70	
Ionosol D (Duodenal)	138	12	100	—	50	—	
5% Saline	850	—	850	—	—	—	
5% Dextrose	—	—	—	—	—	—	170
10% Dextrose	—	—	—	—	—	—	340
10% Invert Sugar	—	—	—	—	—	—	375

*The first three listed solutions along with the carbohydrate solutions are the ones with which most situations can be handled.

PART TWO

Treatment—A Practical Approach

You should always approach the problem of placing a patient on I.V. therapy with three questions in mind:

(a) **Daily Maintenance**—what does this patient require to fulfill normal daily needs?

(b) **Replacement of Previous Losses**—if this patient has lost or been denied fluids and electrolytes prior to admission, how do we go about replacing these?

(c) **Replacement of Current Losses**—how are losses sustained while in hospital replaced?

The answers to these three questions will prevent a haphazard method of prescribing.

Daily Maintenance

If a normal person were to be denied oral intake and placed on I.V. solutions, what would cover his needs for 24 hours?

(a) **Fluids**—estimate 35-45 cc/Kgm body weight as the required intake; generally this would average about 2500 cc. (In the elderly patient, it is safer to estimate 1500-2000 cc.)

(b) **Electrolytes**—NaCl—our diet usually provides 5-10 grams of salt per day; we require only about 4-5 grams under basal conditions. This requirement per day represents abut 76 mEq of Na and Cl, and since there are 155 mEq of Na and Cl in one liter of normal saline, 500 cc of this will meet this daily salt need. (1 gram of NaCl represents 17 mEq of Na and Cl). K—the average daily need of this ion is about 40 mEq or about 3-4 grams of KCl (1 gram of KCl represents 13 mEq of K and Cl). Ampoules of KCl are available containing 20 and 40 mEq of KCl and the required amount can easily be added to I.V. solutions.

(c) **Calories**—100 grams of dextrose per 24 hours greatly diminished nitrogen loss as a result of protein catabolism in fasting states. Two liters of 5% D/W (50 grams of dextrose per liter) provides this. Some further reduction is gained with 200 grams, provided by two liters of 10% D/W. With prolonged usage this solution can be sclerosing. Therefore a solution without this adverse effect and supplying the same caloric value is available. Such a solution is 10% *Invert Sugar*—containing fructose and dextrose.

Therefore the following prescription would supply the daily basic needs of our hypothetical case:

10% Invert Sugar.....1000 cc
Saline..... 500 cc
10% Invert Sugar, plus 40 mEq KCl .....1000 cc

Rate of flow: 200 cc 10% Invert Sugar per hour for the first 2-3 hours, thereafter a flow of 300 cc pr hour.

(In this prescription one can use the Invert Sugar, 5% or 10% D/W as the carbohydrate solution: this applies to all the following prescriptions which will be set up in a similar manner. Thus I will use "Carbohydrate" in such instances and the reader may use whatever solution is preferred.)

Actually one could select other types of solution from which to compound a prescription for daily needs, just so long as it will provide 2500 cc of fluid and a minimum of 76 mEq of NaCl, 40 of K and a caloric value equivalent to 100 to 200 grams of glucose per day to a patient of average weight.

Prolonged Daily Maintenance

The above prescription is satisfactory for short term therapy but if prolonged I.V. therapy is necessary, i.e., if—

(a) a patient post-operatively who cannot eat even after the second or third day

(b) a pre-operative patient markedly debilitated from malnutrition who cannot accomplish adequate oral intake

(c) severe stomatitis, oral lesions or operations making oral intake impossible—then nitrogen balance and protein requirements must be considered.

A minimum of 0.5 gram of protein/kgm. body weight/day, or in general, 50 grams of protein are required by the body daily. Protein solutions (amino acids) are available, such as 5% Aminosol in 5% Dextrose (Abbott), one liter

supplying 50 grams of protein. However, these solutions are of dubious value in correcting a negative nitrogen balance. If you wish to use Aminosol in such cases listed above, the following prescription will deliver the fluid and electrolyte requirements as outlined for daily needs as well as 50 grams of protein.

5% AMINOSOL in 5% D.....1000 cc
SALINE..... 500 cc
CARBOHYDRATE with 20 mEq KCl .....1000 cc
(N.B.: AMINOSOL contains 17 mEq/L of K.)

If an anemia is present in the patient requiring prolonged I.V. therapy, blood itself will help the protein problem as 1000 cc supply about 50 grams of protein. Be aware that in fresh blood, the serum concentration of K is about 4-6 mEq/L (as in the body); but in stored blood or plasma the K concentration may be as high as 25 mEq/L and must be allowed for in therapy.

With prolonged therapy, one should supply vitamins parenterally.

We have covered so far only *daily needs* under two situations—short and prolonged therapy, and have outlined prescriptions for both.

Replacement

What about the patient admitted in a dehydrated state from prolonged vomiting or diarrhea, or debilitated from inadequate fluid intake? How are these losses made good? The method of approach is:

(a) to decide the extent of the dehydration—how much fluid is to be replaced?

(b) to decide what proportion of the amount to be replaced is to be electrolyte containing solution, and what electrolytes?

Estimation of the Amount of Fluid to be Replaced

Two practical and roughly accurate methods can be used, depending on the situation:

(1) If the patient's usual weight is 60 Kgms. and he now weighs 57, he has probably lost 3 Kgms. of water which will require 3000 cc (1000 cc weighs 1 Kgm.) to make it good. However the patient cannot always be weighed.

(2) One can roughly estimate the losses from clinical assessment:

(a) Mild dehydration—about 2% of the body weight loss; thirst is the earliest indication of fluid loss, although not specific. This would be the type of patient who has a history of some fluid loss and complains only of some weakness and thirst and you note that his skin is dry but turgor still good; urine volume decreased and contains no acetone.

(b) Marked dehydration—about 5% body weight loss; this type of patient appears very weak and ill, with the eyes appearing sunken; the mouth and mucous membrances of the body are dry and when you "pinch" the skin you will note the poor elasticity as the pinched area remains folded. There will be a rapid heart rate, a slight temperature and a small urine volume of high concentration, positive for acetone.

(c) Severe dehydration—about 7-10%; the above picture is more pronounced along with extreme restlessness, disorientation and shock.

If our 60 Kgm. man presents with a picture of marked dehydration, there has been a fluid loss of 3000 cc (5% of body weight).

We have determined by one of two methods 3000 cc of fluid to be replaced, as well as giving the daily fluid needs, in the first 24 hours.

Method of Administration

At what rate should the 3000 cc (or whatever the calculated previous deficit is) be given? Keeping a record of the intake and an eye on the output, give the first 1000 cc in 2 hours, the second in 3 hours and the third 1000 cc in 4 hours. The first 1000 cc may be given in one hour if the patient's condition warrants it and then the daily needs are given over the next 7-9 hours.

Sometimes a very large amount must be replaced. The writer once saw a severely dehydrated patient with an estimated deficit of 8000 cc to be replaced. This meant that 8000 cc plus 2500 cc for daily needs had to be given in the first 24 hours. Since it is dangerous to give too large a replacement intravenously, the treatment was as follows:

—½ amount of the estimated replacement and the daily needs } in the first 24 hours.

—½ amount of the estimated replacement and the daily needs } in the second 24 hours.

If while following this plan during the first 24 hours, the patient vomited considerably or lost fluid through gastric suction, one should record the amount of these *current* losses and give this amount as I.V. fluid during the second 24 hours of therapy. Thus our plan for the second period of therapy should now be:

—½ amount of the estimated replacement volume of losses during the first 24 hours and the daily needs } for the second 24 hours

You will note the order of the replacement plan. Give the previous losses first, then give the current losses, if any, and lastly deliver the daily needs. Reassess the patient clinically before starting the second 24 hour therapy. If rehydration and urine volume are good, around 1500-2000 cc or more, then the original estimated amount for this period need not be given. Urinary output is a good indication of the body's fluid balance—if the body has euough fluid it will excrete the excess.

Electrolytes

We have estimated the amount of fluid to be replaced, the rate at which it should be given and in what order, but what volume of *this total* is to be electrolyte containing solutions and what electrolytes? Since not all hospitals have laboratory facilities for electrolyte determinations, and since those that do usually have to wait a matter of hours for reports, an outline for the empirical selection of electrolytes will be given. After this a method of selection will be described for those who have laboratory facilities available.

Selection of Electrolytes — Without a Laboratory

There are two guiding principles:

(a) usually anywhere from a third to two-thirds of the total amount of fluid to be replaced should be electrolyte solutions.

(b) You try to select electrolyte solutions which have a composition similar to the fluid lost—for instance, if gastric fluid is lost, use a solution with a similar composition or one with a predominance of chloride.

Refer back to Sections 5 and 6 under BASIC FACTS before reading the following illustrative cases.

(1) A 70 Kgm man with a history of marked diarrhea for 2-3 days is admitted. We estimate, by one of two methods given earlier that about a 4000 cc fluid loss must be replaced, and with oral intake denied, 2500 cc as daily needs. So we take ½ of the estimated loss of 4000 cc plus 2500 cc and decide on giving a total of 4500 cc of fluid for the first 24 hours. (You may give the entire 4000 cc replacement amount if the cardiovascular-renal status is good.)

So we decided to give 4500 cc of fluid for the first 24 hours of therapy; but what electrolytes are required?

(1) We saw earlier that a minimum total of **76 mEq** of **NaCl** and **40 mEq** of **K** are required for **daily needs.**

(2) in this patient losses are from the lower G.I.T. and from the diagram of the G.I.T. we see that in one liter of diarrheal fluid there are 140-200 mEq of Na, 60-70 of Cl and 15-20 of K; take the lower values of these electrolytes for our calculations as follows:

We decided to replace 2000 cc or 2 liters of the **total loss** in the first 24 hours and this loss represents diarrheal fluid. Therefore in the two liters of fluid lost there were:

140 x 2 liters = 280 mEq. of Na
60 x 2 liters = 120 mEq of Cl
15 x 2 liters = 30 mEq of K,

All of which is our replacement quota. So our total of 4500 cc of fluid for the first 24 hours of herapy should provide the following amounts of electrolytes:

	Na	Cl	K
Daily needs.....	76	76	40
Replacement.....	280	120	30
TOTAL.....	356 mEq	196 mEq	70 mEq

Note that a greater amount of Na as compared with Cl must be given. Refer to the list of available repair solutions and you will note that certain solutions "fit", in that they have an electrolyte composition in which Na is present in excess of Cl. Thus the rationale and adequacy of the following prescription will be apparent in that it supplies over 300 mEq of Na and 70 of K, which are our chief concern in the electrolyte disturbances resulting from diarrhea.

The total amount of each electrolyte in this prescription does not equal exactly the amount we calculated to give. Don't be rigid in this respect; we are dealing with rough estimations—and safely on the conservative side.

PRESCRIBE:

Saline	1000 cc	
Carbohydrate with 40 mEq KC1	1000 cc	for the first
1/6 Molar Lactate	1000 cc	24 hours
Carbohydrate with 40 mEq KC1	1000 cc	
Carbohydrate	500 cc	
	4500 cc	

If shock or debilitation is associated here, blood or plasma expanders may be added to the prescription and allowed for in the quota.

Before starting the second 24 hours of therapy reassess the patient clinically; If there is still evidence of dehydration and oral intake is still being withheld, repeat the prescription (this will deliver the rest of the estimated previous fluid and electrolyte losses as well as the daily needs for this second 24 hour period.)

If, during the first 24 hours of therapy, the diarrhea persisted, try to record the approximate volume of stool lost and replace with an equal volume of saline during the second 24 hour therapy and add another 20 mEq of KC1. So, let us say that our patient passed about 1000 cc of diarrheal stool during the first 24 hours; our second 24 hour therapy, replacing the volume of stool fluid with saline, would now read:

Saline in 5% D	1000 cc	
Carbohydrate with 40 mEq KC1	1500 cc	for the second
1/6 Molar Lactate	1000 cc	24 hours
Carbohydrate with 40 mEq KC1	1000 cc	
Saline with 20 mEq KC1	1000 cc	
	5500 cc	

It is not necessary to follow strictly the order of the prescriptions as set out, so long as you correct the dehydration as you supply the electrolytes.

In correcting any state of dehydration 10 cc of 10% Calcium gluconate should be added to one of the solutions.

Potassium is usually withheld until renal output is satisfactory. A good working rule is—no K until at least 1000 cc of urine have been passed. In the above prescription you will note that we estimated 100 mEq of K to be given. As a calculation based on empiricism this is logical, but where you have such a large amount calculated to be replaced in one day, it is better to give $\frac{3}{4}$ of the amount. Another practical rule is to withhold the administration of blood to a dehydrated patient until at least 1000 cc of fluid have been given. The infusion of blood to a patient in a state of dehydration is attended with the possible danger of intravascular hemolysis.

(2) Take a case of pyloric obstruction admitted after a prolonged episode of severe vomiting and an estimated fluid loss of 4000 cc. We know that C1 loss is excessive so empirically we give part of the 4000 cc to be replaced as solutions containing C1 in excess of other ions such as in Ringer's solution. Saline is suitable because although it contains equal parts of Na and C1, its C1 concentration is greater than that of the serum. One can also use .89% ammonium chloride which has a concentration of 150 mEq of C/liter. Actually saline is the treatment of choice in the dehydrated alkalotic patient, but as

the dehydration is corrected the other solution may be incorporated in the therapy.

In this case, again, one-half the total to be replaced, 2000 cc, and the daily need, 2500 cc, giving 4500 cc will be delivered in the first 24 hours of therapy.

The amount of electrolytes to be given is estimated the same as with the first case. Since the 2000 cc of fluid calculated as replacement represents 2000 cc of gastric fluid lost, the amount of electrolytes to be included in this quantity should equal that lost in this amount of gastric fluid.

You will note that in resting states there are about 90 mEq of Cl in one liter of gastric juice, but in irritative or obstructive lesions, the concentration may be as high as 150 mEq per liter. One would be safe in assuming in our case a value of 120-130 mEq/liter. Thus our quota to be replaced would be:

130 x 2 liters =260 mEq of Cl
60 x 2 ,, =120 mEq of Na
9 x 2 ,, = 18 mEq of K

Adding this to the daily needs, our total of 4500 cc for the first 24 hours should provide:

	Na	Cl	K
daily needs	76	76	40
replacement	120	260	18
TOTAL	196 mEq	336 mEq	58 mEq

One of the following prescriptions can be used in such a case as each delivers an excess of Cl to Na and fulfill the above calculations:

Saline in 5% D ..... 1000 cc
Carbohydrate with 20 mEq KCl ..... 1000 cc for the first 24 hours
Saline in 5% D ..... 1000 cc
Carbohydrate with 40 mEq KCl ..... 1500 cc

4500 cc

OR

Saline in 5% D ..... 1000 cc
Carbohydrate with 20 mEq KCl ..... 1000 cc for the first 24 hours
Ammonium Chloride 0.89% ..... 1000 cc
Carbohydrate with 40 mEq KCl ..... 1500 cc

4500 cc

For the second 24 hours of therapy repeat one of the above if indicated after reassessment. Also, you must replace what was removed from the stomach by the gastric suction that was set up, i.e., 1500 cc were aspirated during the first 24 hours; you can replace this with 1500 cc of saline during the second 24 hours along with the daily needs for this period and the remaining one-half of the initial total replacement.

This simple approach to replacement can be applied to losses from other sites. A few of the major sites of losses is herewith listed:

(1) **duodenal losses**—through Miller Abbott suction, fistula or obstruction. Ringer's lactate or saline are the most useful. The latter should be stressed because sometimes, from upper small bowel lesions, the loss of Na and Cl are equal.

(2) **biliary losses**—through T-tube drainage or fistula. 1/6 Molar Lactate and Ringer's Lactate are recommended.

(3) **recent ileostomy**—1/6 Molar Lactate.

It must be mentioned here that (a) in replacing gastric losses in the elderly patient, one should be aware of the possibility of achlorhydria and avoid excess administration of chlorides; (b) ordinarily 600-900 cc represents the daily insensible water loss from skin and lungs. With moderate sweating or a fever over 101°F, the fluid loss will be about 1500-2000 cc and will contain from 25-50 mEq of NaCl. This must be replaced; (c) be sure that kidney function and urinary output are adequate—this is perhaps the sine qua non of therapy.

Replacement of Electrolytes—An Easy Way Out

If you are lazy or just can't be bothered with learning the composition of repair solutions and G.I.T. fluids, you will find the following Table "X" convenient as a handy and simpler guide to therapy. It eliminates the necessity of making the above calculations and enables you to handle cases with only three solutions.

In this table, there is given the percentage of each solution of the total amount to be replaced. For example, if you calculate a 3000 cc loss of gastric fluid to be replaced, then 33% of this should be replaced with 5 or 10% D/W (or 10% Invert Sugr) and 67% should be saline.

TABLE "X"

	% of D/W	% of 5% Dextrose in Saline (D/S)	% of 1/6 Molar Lactate
Gastric Losses	33	67	—
Small Bowel Losses	20	70	10
Biliary Losses	0	63	37
Recent Ileostomy or Diarrhea	15	70	15
Pancreatic Fistula	0	50	50

As for K replacement with this table, use the following rule of thumb: for each liter of gastro-intestinal fluid lost, 10-15 mEq of K should be given, along with the 40 mEq allowed for daily needs.

This replacement table and the knowledge of the daily needs will enable you to handle the greater part of cases, and eliminates all mathematical gymnastics given earlier, if you are not athletically inclined in the Betz cells.

Selection of Electrolytes—With a Laboratory

With lab facilities available, we have a better guide of what is going on, and are able to use some formulas in calculating electrolyte deficits. This, however, should not be the sole guide of therapy. The patient can be perfect chemically but moribund clinically.

The initial approach is as outlined earlier:

(a) Calculate the volume of the ECF; in other words, calculate the ECV which is obtained by taking 20% of the body weight.

(b) Estimate the amount of fluid to be replaced by one of the above two methods, and then an additional step:—

(c) Calculate the deficit of each electrolyte per liter of ECF and multiply by the calculated ECV to obtain the total deficit of each electrolyte.

This is how it works out: A patient is admitted in a state of dehydration after an episode of severe and prolonged emesis and a diagnosis of duodenal ulcer with pyloric obstruction is made. Normal weight is 62 kilograms. We take blood for electrolytes, set up 5 or 10% dextrose in saline, while awaiting the lab report, and begin gastric suction with a Levine tube.

The lab report shows—Na of 130, Cl of 84, K of 3.7 and CO_2 C.P. of 34—all values being in mEq/L. Following steps a, b, and c above, we determine the:

(a) calculated ECV—Body weight 62 kgm x 20% equals 12.4 liters.
(b) degree of dehydration—let us say that we calculated this to be 3000 cc.
(c) deficit of each electrolyte:—

—Na deficit per liter of ECF	142-130 equals	12 mEq
∴ Total Na deficit in the ECV	12 x 12.4 equals	148 mEq
—K deficit per liter of ECF	5 - 3.7 ,,	1.3 mEq
∴ Total K deficit in the ECV	1.3 x 12.4 ,,	16 mEq
—Cl deficit per liter of ECF	103 - 84 ,,	19 mEq
∴ Total Cl deficit in the ECV	19 x 12.4 ,,	235 mEq

What we have been doing is subtracting the found serum values from the normal values of these electrolytes. The value obtained represents the deficit of each electrolyte in every liter of ECF. (Recall that for practical purposes, the serum values reflect those of the ECF.) Since the volume of the ECF, (the ECV), in this patient was calculated as 12.4 liters, then multiplying the deficit per liter by 12.4 gives the total body deficit.

Thus in this case the total needs of fluids and electrolytes for the first 24 hours of therapy will be:

WATER—	daily needs	2500 cc		
	replacement	3000 cc		
		5500 cc		
ELECTROLYTES—		**Na**	**Cl**	**K**
	daily needs	76	76	40
	replacement	148	235	16
	TOTAL	224	311	56

The following prescription is compounded to total 5500 cc and deliver, nearly, the exact amount of electrolytes required:

Saline	1000 cc
Carbohydrate with 20 mEq KCl	1000 cc
.89% Ammonium Chloride	1000 cc
Carbohydrate with 40 mEq KCl	1000 cc
Carbohydrate	1500 cc
	5500 cc

Similar calculations can be made on the second day and so on.

Post-Operative Maintenance and Disturbances

This is where the bulk of the problems arise. Although it might read as complicated as Russia's Foreign Policies, the principles of therapy will be presented on basic principles as discussed earlier. This section will be outlined under four separate headings: The effects of surgery on Electrolytes; Fluid and electrolyte management for operation,; Post operative salt syndromes; Some aspects of acid-base imbalance.

The Effects of Surgery

As a result of major surgery there usually ensues:

(a) retention of salt as manifested by a decrease of the urinary output of salt for 1 to 3 days, depending on the extent and stress of the operation.

(b) loss of K; loss of this ion is great after major operations, especially within the first 24 hours. As urinary output increases after the temporary oliguria immediately after operation, the K loss is maintained and tends to deplete the patient.

(c) protein breakdown and loss as manifested by increased urinary excretion of nitrogen for 4 to 6 days.

(d) decreased urinary output; this is a transient thing in the immediate post-op period.

As a result of the above, salt infusions are avoided for 24-36 post-operative hours; some advocate a longer period, **But** where there will be loss of salt, i.e., from biliary drainage or an ileostomy, do not restrict salt but allow whatever is lost on the first post-operative day; K intake must be religiously attended to, but it is usually withheld for 24-36 hours until renal output returns to normal; adequate calories in the form of carbohydrate should be supplied to spare portein catabolism and protein may be maintained by giving blood and/or Aminsol; strict charting of intake and output is necessary.

Actual Management in the Post-operative Period

Here is a fairly standard routine based on the above principles and the harangue earlier about daily maintenance:

Day of Operation:—

1. 500 cc of 5% D/S can be started shortly before the patient is sent to the O.R.; this will facilitate the work of the anaesthetist—if you feel so inclined.
2. the anaesthetist will then substitute 5% D.W. and later, blood as required for losses during operation.
3. Upon returning the patient to his room, the infusion is allowed to continue until a total of 2000 cc of 5% D/W has been given since the start of the operation.

First Day Post-operative:—

An attempt is made to keep the urine volume between 1000-1500 cc daily with a specific gravity of 1.015 or less. However a urine volume as low as 600 cc for the first or second days need not be alarming. As a general rule calculate the amount of fluid required as: the urine output plus 1000-1500 cc for insensible

losses. Let's say there was an urinary output of 800 cc and we allow 1500 cc for a total of 2300 cc. This could be given as carbohydrate alone or a small amount of salt can be included as follows:

10% G/W	1000 cc
Saline	300 cc
10% G/W	1000 cc
	2300 cc

Second Day:—

If oral intake is impossible or inadequate, deliver the daily requirements of electrolytes; the required 2500 cc daily intake of fluid can probably be allowed as urine volume should be around or near 1000 cc daily. Give:

Carbohydrate	1000 cc
Saline in 5% D	500 cc
Carbohydrate plus 20 mEq KCl	1000 cc
	2500 cc

This provides about the daily requirement of fluid and electrolytes.

The above postoperative prescriptions suffice for cases where there are no abnormal losses in contrast to biliary or bowel surgery where continuous drainage of G.I.T. secretions exist. These losses must be charted and replaced each day, being worked into the above prescriptions.

For example, 400 cc of bile is passed via a T-tube in the common biliary duct during the first 24 hours; this volume is replaced during the second 24 hours and Table "X" shows that 63% of this volume should be saline and 37% one-sixth molar lactate (or deliver the equivalent of salt contained therein). K is added in amounts according to volume of secretions lost, as given with Table "X".

Needless to say that as soon as possible oral feedings should be started. When doing so be sure that they contain sufficient salt and K to cover the daily need and drainage losses of these electrolytes. If their intake cannot be entirely accomplished by mouth, then the difference must be made up by parenteral fluids.

Post-operative Salt Syndromes

The awareness, recognition and management of various sodium upsets seems to be the most difficult encountered by attending staff. They are the most frequent of all electrolyte disturbances in the surgical patient. It should be emphasized here that derangements of normal sodium metabolism can produce severe shock. The attending circumstances and clinical pattern of these disturbances may be divided into three syndromes, the first two of which being the most important because of their greater frequency of occurrence.

(1) Sodium-Water Deficiency—this syndrome is called "Isotonic Dehydration" in that it is characterized by the loss from the body of a fluid which is identical, or nearly identical, in chemical composition to ordinary ECF, the extracullular fluid. It is as if you took a glass of scotch and water and poured half of it down the sink! The drink although less in quantity still contains the same alcoholic percentage. What is the practical implication of this analogy? It is obvious that if a fluid of identical nature is removed from the ECF com-

partment, the remaining ECF and therefore plasma will not be altered in chemical composition. In other words, when reports come back from the lab, the serum **concentration** of Na, Cl and bicarbonate will be normal.

This is the sort of picture you can get from small bowel losses because its composition does, somewhat, resemble that of the ECF, whether these losses be from Miller-Abbott suction or because of fluid trapped in obstructed loops of small bowel. The loss of a mixture of gastric juice, bile and pancreatic secretions may have a net effect of the same type. If there has been losses from such situations and you get normal chemistries back from the laboratory instead of an expected decreased serum Na and a lowered CO_2 C.P., think of this syndrome of "Isotonic Dehydration."

Even if you don't think of it, the history and physical signs of dehydration should obviously make you say that all is not normal despite what the report says. For treatment of such cases, we simply replace what we estimated as lost with fluids of a similar composition, using Table "X" for this purpose.

(2) Sodium Deficit with relative Water Excess—this is the grandaddy of them all. The majority of salt disturbances fall into this syndrome, and are usually caused by the loss of a considerable amount of G.I.T. fluids replaced only by water again; it is like somebody half emptying a glass of Scotch and then filling up the glass again by adding water and repeating the procedure a few times—the drink become distinctly flat and so does the patient when this syndrome occurs clinically. It is seen in the vomiting patient who has been allowed to drink water ad lib; or in the patient with a Levine tube down receiving glasses of chipped ice from a sympathetic nurse, or else receiving infusions of dextrose in water (D/W) instead of Na containing solutions.

What happens? The net effect is a deficit of NaCl and bicarbonate in the ECF compartment with a relative water excess. Because the ECF is now hypotonic, fluid from this compartment migrates into the cells, by osmosis. Thus the ECF becomes contracted and the patient can go into shock because the circulating blood volume, as a **part** of the ECF, is also reduced in volume—thus peripheral vascular collapse and shock can ensue.

Where else does this happen? In common duct surgery, especially in the elderly; or the patient undergoing surgery with a questionable cardiac status, where the attending staff usually make a fetish of withholding Na infusions or restricting the patient to a salt-free diet despite continuing salt losses post-operatively.

What is the clinical picture? Here you have an obese 50 year old female with some hypertension who underwent a cholecystectomy and exploration of the common duct for jaundice due to gallstones.She took the operation well and for the first 3 to 4 days post-op. she received 2500 cc of fluid daily, either parenterally or by mouth, containing practically no Na because of the fear of aggravating the hypertension by overloading the circulation. Meanwhile the T-tube in the common duct drains merrily on to the extent of 400-600 cc of bile per day. About this time, the patient is not doing so well despite the success of the operative procedure. And then, about the fifth or sixth day, she goes into peripheral vascular collapse—unobtainable B.P., barely palpable pulse, cold moist skin, etc. Why? You rule out possible causes of shock in this post-operative period. A review of the intake-output record will provide a clue— the collapse was due to a severe sodium deficiency. If you had been closely watching the urinary output you would have noted a steady decrease in volume preceding the collapse.

The treatment for this emergency is the infusion of 400-500 cc of 5% NaCl solution as well as other anti-shock measures. Results are often dramatic. This hypertonic solution is necessary because too great a quantity of isotonic saline, or 1/6 Molar Lactate would be required to restore the serum Na to the normal level, in an already hydrated patient. Most writers state that when the serum Na is down to 120 mEq/liter or the chlorides as low as 80 mEq/liter, hypotension and shock can be anticipated and the use of hypertonic salt solutions is indicated. Of course, if there is dehydration associated with the severe deficiency, D/S should also be given along with the hypertonic NaCl.

(3) Salt-water Surplus—this should rarely happen; it results from excessive administration of saline post-operatively because of a disregard for the intake-output record. Marked thirst and pulmonary edema are the main hallmarks of this state. Electrolyte-free solutions, mercurials as diuretics and a restricted intake to about 1500 cc of salt-free fluids per day is the treatment.

The Above in Brief

1. Where you have a large loss of salt post-operatively, don't absolutely restrict NaCl—replace the loss on a volume for volume basis.
2. Whenever oliguria develops in the 4 to 6 day post-operative period, i.e., 500-600 cc of urine/day, in the face of previous good renal function, think of a low salt level and do laboratory determinations.
3. If the oliguria is associated with a lowered serum Na and Cl allow 2500-3000 cc as the daily intake with at least 20-25 grams (or 300-400 mEq) of NaCl, plus whatever is lost from abnormal routes.
4. If oliguria is not associated with low serum salt levels, restrict the daily intake to 1500 cc without salt, but if salt losses are occurring, be sure to replace them on a volume for volume basis.
5. Think of salt deficiency whenever shock occurs in the 4-6 day post-operative period in the absence of other causes. If it occurs, don't wait for laboratory determinations—give 300-500 cc of 5% NaCl.
6. If from routine determinations, the serum Na is reported below 130 mEq/L or the Cl around 80 mEq/L, but clinical hydration is within normal limits, give 150-200 cc of 5% NaCl to avert disaster; if hydration is poor, give D/S as well.
7. Patients with Levine or Miller-Abbott tubes invariably complain of a dry mouth and throat, despite adequate replacement therapy. If you must give cracked ice to wet their mouths, do so with frozen saline or Ringer's.

It should be mentioned that hyponatremia (low serum sodium) is not always an indication of sodium deficit. This may be present in patients with cardiac and liver disease. That is why one should pay as much attention to the history and the clinical state as to the lab reports.

Acid-Base Imbalance

The two commonest exhibitions of this in surgery are metabolic alkalosis and acidosis.

Metabolic Alkalosis

The commonest cause of this is the loss of chloride ion through loss of gastric contents either by vomiting or by gastric suction. There results an increase in the blood pH, an elevated CO_2, C.P., a decreased serum Cl and an alkaline urine, the latter being a compensatory mechanism by the kidneys so as to rid the body of excess available base. This alkalosis, associated with a low serum Cl concentration is referred to as hypochloremic alkalosis.

This picture can be complicated by inadequate treatment. For example, a patient is admitted with pyloric obstruction and has been vomiting for some days; he is dehydrated, excreting an alkaline urine and has an elevated CO_2 C.P., a decreased serum Cl and some small decrease of Na and K. He has a gastric tube with suction and is getting I/V saline and/or ammonium chloride in water to restore serum Cl level. But in 2-4 days we find the alkalosis persisting or even increasing despite a normal serum Cl. What is even more paradoxical is that the pH of the urine is now on the acid side. The cause of this paradox lies with the K concentration which will be found to be low. K **must** be included in the treatment because it has an influence on the renal tubular absorption of bicarbonate; a low serum K results in an increased reabsorption of bicarbonate, thereby accounting for the persistent and refractory alkalosis and acid urine. This alkalosis associated with a low K is often referred to as hypokalemic alkalosis.

Metabolic Acidosis

This occurs whenever available body base is decreased and one usually finds a decreased CO_2 C.P., a decreased serum Na and K. Essentially it appears in conditions where Na loss is greater than Cl, such as:

(a) severe diarrhea
(b) long tube intestinal suction, i.e., Miller-Abbott tube
(c) small bowel fistula or drainage
(d) biliary drainage or fistula; pancreatic fistula
(e) post-op. enterocolitis from too vigorous antibiotic therapy
(f) recent ileostomy
(g) and sometimes with too vigorous preoperative preparation of the bowel by purging.

As we have been trying to stress, the **type** of plasma electrolyte abnormality which will **result** from losses from the G.I.T. will **depend** on the **character** and **composition** of the fluid lost.

Sometimes losses from the small bowel, or a mixture of losses of, say gastric, biliary and pancreatic secretions can lead to an "isotonic" loss and no resulting acid-base imbalance ensues. This was mentioned already under the post-op. syndrome of "Isotonic Dehydration."

Treatment of Acid-Base Imbalances

Never be in too great a rush to correct acid-base upsets per se; if you go about correcting the calculated electrolyte deficits by the methods shown earlier you won't have to bother with the following added frills, unless the imbalance is extreme, and active correction is indicated; then the following procedure can be carried out:

1. For decreased CO_2 C.P. in metabolic acidosis—to raise the CO_2 C.P. one mEq, then 4.2 cc of one-sixth Molar Lactate for each liter of the ECF are required. (The ECF or ECV, you'll recall, is obtained by taking 20% of the body weight.)

   Example: A 70 Kgm man with a CO_2 C.P. of 15 mEq/L, the normal CO_2 C.P. being 27 mEq/L.

   70 x 20% equals 14 liters of ECF

   27 x 15 equals 12, the deficit of base per liter of ECF

   Therefore 12 x 4.2 x 14 equals 705 cc of one-sixth Molar Lactate required to restore the CO_2 C.P. to normal.

2. For increased CO_2 C.P. in metabolic alkalosis—to lower the CO_2 C.P. one mEq, then 4.2 cc of 2% Ammonium Chloride for each liter of the ECV are required.

   Example: A 70 Kgm. man with a CO_2 C.P. of 35 mEq/L, the normal CO_2 C.P. being 27 mEl/L.

   70 x 20 equals 14 liters of ECF

   35 x 27 equals 8, the excess of base per liter of ECF

   Therefore 8 x 4.2 x 14 equals 470 cc of 2% Ammonium Chloride required to reduce the CO_2 C.P. to normal.

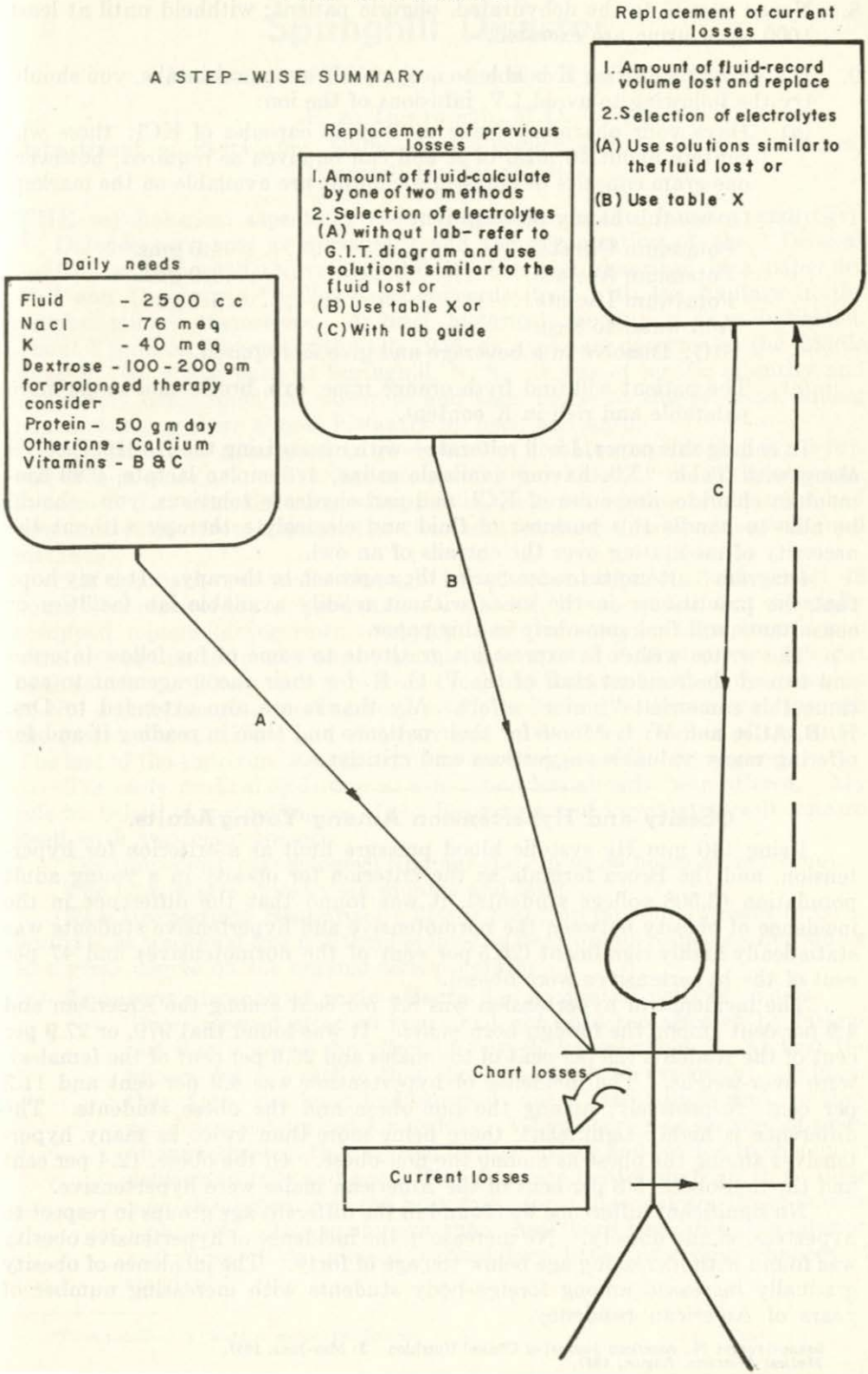
Potassium

Before ending, a few lines on K metabolism should be inserted. What is to be said will be brief and should prompt some extra reading.

1. About 90% of the K in the body is found in the fluid of the cells—in the intracellular fluid (ICF).
2. The kidney is unable to conserve K as it can Na.
3. It is now appreciated that in starvation, dehydration, conditions causing loss of fluid and salt from the body, major operations, infusions of large amounts of saline without K—all lead to a transfer of K from the cells to the ECF and subsequent elimination by the kidney, which does not conserve K.
4. If serum determinations are done at the time when this transfer is occurring, the lab report will show a "normal" serum concentration of K, even though there is an absolute loss from the body cells, kidney excretion continuing to add to the depletion.
5. Therefore in such conditions as cited in point 3, above, you should be aware that a K deficiency may exist despite what the lab report shows.
6. There are many symptoms and signs given which are manifestations of low serum potassium, but I doubt if they can be delineated clinically, at least with ease, since hypopotassemia is usually associated with other derangements. The development of an unexplainable paralytic ileus or marked muscle flaccidity may lead you to suspect the condition. There are some E.C.G. changes which are more specific, and are useful for a rapid diagnosis.
7. As for administration, you can add from 40 to 80 mEq of KCl to a 1000 cc of 5% or 10% D/W; it should not be given faster than 25 mEq per hour.

THE APPROACH

A STEP-WISE SUMMARY



8. **Never** give K to the dehydrated, oliguric patient; withhold until at least 1000 cc of urine are excreted.

9. If a patient requiring K is able to accomplish some oral intake, you should try the following to avoid I.V. infusions of the ion:

   (a) Have your pharmacy make up 3 gram capsules of KCl; these will contain about 40 mEq of K and can be given as required; however, one gram capsules of potassium chloride are available on the market.

   (b) Or use this handy prescription:

   Potassium Citrate.....1.0 gms.
   Potassium Acetate.....1.0 gms.
   Potassium Lactate.....1.0 gms.
   Add water to 8 cc.
   SIG: Dissolve in a beverage and give as required.

   (c) The patient will find fresh orange juice, oxo, broths and soups more palatable and rich in K content.

In ending this paper, I will reiterate—with memorizing the BASIC FACTS along with Table "X", having available saline, 1/6 molar lactate, 0.89 ammonium chloride, ampoules of KCl and carbohydrate solutions, you should be able to handle this business of fluid and electrolyte therapy without the necessity of meditating over the entrails of an owl.

Diagram 3 attempts to summarize the approach to therapy. It is my hope that the practitioner in the areas without readily available lab facilities or consultants will find some help in this paper.

The writer wishes to express his gratitude to some of his fellow internes and two of the resident staff of the V. G. H. for their encouragement to continue this somewhat "junior" effort. My thanks are also extended to Drs. H. B. Atlee and W. I. Morse for their patience and time in reading it and for offering many valuable suggestions and criticisms.

Obesity and Hypertension Among Young Adults.

Using 140 mm Hg systolic blood pressure limit as a criterion for hypertension, and the Broca formula as the criterion for obesity in a young adult population (3,508 college students), it was found that the difference in the incidence of obesity between the normotensive and hypertensive students was statistically highly significant (26.5 per cent of the normotensives and 47 per cent of the hypertensives were obese).

The incidence of hypertension was 8.1 per cent among the American and 4.9 per cent among the foreign-born *males*. It was found that 979, or 27.9 per cent of the students (29 per cent of the males and 23.9 per cent of the females), were over-weight. The incidence of hypertension was 4.9 per cent and 11.3 per cent, respectively, among the non obese and the obese students. The difference is highly significant, there being more than twice as many hypertensives among the obese as among the non-obese. Of the obese, 12.4 per cent and the non-obese, 6.3 per cent of the American males were hypertensive.

No significant difference was found in the different age groups in respect to hypertension and obesity. No increase in the incidence of hypertensive obesity was found with increasing age below the age of forty. The incidence of obesity gradually increased among foreign-body students with increasing number of years of American residency.

Szent-Gyorgyi, N., American Journal of Clinical Nutrition. 5: May-June, 1957.
Medical Abstracts, August, 1957.

Springhill Disaster

(Psychological Findings in the Surviving Miners).

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THE psychological aspects of the civil population (i.e. families and Civil Defence personnel at Springhill) and the organization of the "Distress Centre" at Springhill (November 2-6/56) has been described in a paper by Weil and Dunsworth.* This paper concerns itself with the findings in the rescued miners themselves. A brief historical account is here indicated. About 5 p.m. on November the 1st, 1956, an explosion occurred in the middle levels of No. 4 coal mine at Springhill, N. S. It was of terrific intensity and completely demolished the concrete and steel house at the pit-head, killing miners working there almost instantly by blast and burn.

Rescuers who attempted to go down No. 4 shaft were blocked by debris and cave-in quite near the surface and several were killed by gas in the attempt. It seemed that over 120 miners trapped in the lower reaches of the mine had been lost, either from explosion or the resultant gases (carbon monoxide and methane).

An approach was made through No. 2 mine adjoining, and almost 48 hours later some of the trapped miners worked their way up and met especially equipped miners (draegermen, carrying their own oxygen equipment) who were attempting to reach them. At this time a total of 35 men were brought to the surface. These men were from the group working on the upper reaches of No. 4 and though some had been fatally overcome in their ascent through the gasses, the survivors told of others still in the lower reaches of the mine. The last of the survivors were lifted 36 hours later, a total of 88 rescued.

The early medical and surgical evaluation has already been offered. My role on behalf of my colleagues of the Department of Psychiatry, will concern itself with two main themes:

1. The immediate psychological findings (i.e. up to 36 hours after rescue).
2. The later stages (mostly six months and later).

The Immediate Response:—This is based on far too few cases to be as accurate as we would like but there seemed to be two main responses, depending to a great degree on the organic cerebral state:

(a) **Apparent absence of toxic effects** (i.e. no clinical signs of anoxia)

(i) Those rescued after 40 hours had not been too affected by fatigue but they showed the effect of "psychological shock." At first the miners did not realize that there had been an explosion. As far as they knew the hoist had stopped (not a rare occurrence), so they sat down and waited for awhile. When there was no sign of operations resuming, they started for the surface and started to encounter debris and falls and gas. The gases are lighter than air so as these men clambered over the rock falls, some died *quickly* (from carbon monoxide). It was obvious then that there had been an explosion and most of the miners realized how little were their chances of survival.

*To be published in Canadian Psychiatric Journal.

On rescue their reaction was one of relief. They were not yet overly fatigued and the characteristic reaction was *anxiety with a marked pressure to talk.* Though their families told them to rest and take sedation, our psychological teams found it best to let them talk it out as a way to dissipate anxiety and grief (not only in disasters either!)

On later questioning we have found that most of these miners were restless and anxious for a week or more after their rescue.

(ii) The findings on those rescued after 84 hours were coloured by fatigue, and the much more disturbing waiting for such a long period one mile underground. Briefly, these men, mostly under a very clear-thinking "overman," clustered around an air source and awaited rescue. From time to time some members growing more anxious would attempt to ascend the mine, almost invariably a few would die quickly from the gasses, others would return to the original level and yet others would find a niche higher up the mine.

Throughout this time the mine was filthy with smoke and gas and dust, there was no food and no water fit to drink, and much of the time was spent in darkness. There was little or no panic. Some of the miners despaired but most of them, especially the older men, kept their heads. There was an added factor of marked fatigue, not only from lack of sleep but from over-exertion, since many of these men had come up from the lower levels. Since the hoist was not working and the main slope was full of deadly gas, they had crawled up slopes, at times nearly vertical, to be rescued.

Eight months later we find these miners' memories are coloured, e.g. three men of the same group do not give the same story. Talking to G, he states how he kept his head and was not afraid; while C, the next miner to be interviewed, spontaneously offered "G was scared, he made his will and lost all hope of rescue."

In our interviewing we have not probed too deeply. We did not want to open old wounds, since even after eight months many of the events have been forgotten or coloured in retrospect, and perhaps it's better that way.

Most of the above concerns itself with late findings, but of this group of miners we saw three or four very shortly after the rescue and it was very difficult to establish whether their symptoms were completely psychological or associated with cerebral anoxia.

Firstly, they showed marked fatigue. They looked awful; dehydrated, hungry but unable to eat, restless and aimless. They wanted rest but it wouldn't come.

They were extremely irritable. They had lost most of their usual control. One man we were called to see was threatening to kill his wife and child. He was extremely grateful and cooperative to our I.V. injection of promazine HCL ("Sparine" — Wyeth).

Clinically, in retrospect, these men suffered from a moderate degree of this syndrome for weeks or months. They had all lost weight, and some have not even now regained it. "I was too tense and jittery to eat," was the frequent remark. Their sleep was broken with dreams of happenings in the mine. They were easily aroused to anger, even men who always before were "mild and even-tempered."

Some developed fatigue and numerous aches and pains and these proved the greatest problem. It must be noted that almost all miners had enough exposure to cause temporary "blackouts," the men themselves did not remember but their buddies recalled "bringing them around."

(b) **Toxic effects**: — I, personally, saw two of these. One was very delirious, and had been underground for three days; he required special psychiatric handling for 12 hours at the "Distress Centre" and then spent two months at the Victoria General Hospital Psychiatric Pavilion. He still shows the effects. The other, an older man, keeps saying he's "very well" but his memory is badly disturbed. He came to medical attention in a peculiar way. He actually had been rescued after 40 hours underground (i.e. mid-afternoon November 3/56), was examined through the hospital and after a while sent home. After a few hours sleep he awoke, found he had his miner's lamp still with him and went down to the mine lamp house, was there found to be confused and was sent back to the hospital. He was confused in time sense but next day when I saw him he seemed improved. In retrospect, especially since eight months later he shows definite organic "loss" on psychological testing, he apparently fooled us by saying how well he felt and we did not check closely enough to evaluate his memory. This brings up a most interesting possibility, especially for compensation purposes. How many miners, especially the older group (the patient referred to is in his sixties) suffered undetected sub-clinical cerebral damage and will show symptoms later?

At this point we should point out the problems in evaluation. We have found that assessment of cerebral anoxia and its effect on the personality is very difficult. This not only goes for adults but even in children. I am of the firm opinion that sub-clinical states exist and help produce problems in behavior, especially in those whose previous personalities were not too well integrated.

We feel the clinical psychiatric evaluation plus psychological projective testing and ward behavior, with adequate reports from family and friends is a more accurate assessment than our existing neurological findings.

Actually the human personality must have great strengths. There were 88 miners rescued and we have examined 16 to date, a small number, considering the psychological trauma received. We were struck by the same findings in the civil population (Weil & Dunsworth).

The Later Findings

Over the past four months we have examined 14 survivors specifically referred for psychiatric assessment and also interviewed four miners without psychiatric symptoms. Many interviews have been held, and in most cases psychological testing done (by Victoria General Hospital Staff Psychologists) and the ward behavior reviewed. In some cases we have interviewed friends and families, and in almost all cases taken cross references (i.e. from one miner concerning other members of his group). By far our greatest problems were those miners who developed fatigue, eye symptoms, and numerous muscular aches and pains. Many of these had adequate cause for their original symptoms, but these symptoms have continued in an appreciable number of miners. In fact in a few cases they have become worse. We feel there are many reasons for this but we will list only a few:

Most of the miners seen have *denied fear and anxiety* while trapped. It could be that experienced miners would not feel these emotions but we doubt it. We will hazard a hypothesis that these men, after a few hours realized their predicament, and not wanting to show feelings in front of their buddies repressed these feelings and these later came out in physical symptoms.

Basic anxiety thus produces somatic symptoms; it is interesting to note the absence of pure anxiety reactions.

The economic trauma. Unconsciously these men, especially the younger ones with growing families, feeling poorly, were unable to admit improvement. In two cases despite two long periods of hospitalization, almost daily psychiatric interviews, psychotherapy and special rehabilitation measures, their symptoms continued. Doctor A. H. Shears of the Rehabilitation Centre agreed with our comment, "it's as if they have to be 200% well." Economically, there is the problem of unemployment. The only trade they know and want is mining, and compensation offers at least a partial measure of economic security.

We wish to emphasize that this reaction is *not* conscious. We did not find *one* malingerer, in fact the miners we met have been a fine group of men with high principals and good social and work records.

DISCUSSION:

In the relief of rescue there was a

(1) RELATIVE LACK OF EARLY PSYCHIATRIC SYMPTOMS; therefore early psychiatric disorders were *missed* and *prevention of psychiatric disorders missed.*

(2) The rescued miners used *repression* of their feelings and therefore diagnosis was harder. With repression slipping, anxiety became *converted into physical symptoms.*

(3) Marked difficulty was encountered in assessing the organic cerebral element.

(4) The pre-accident personality colours the final picture; e.g. wartime experience indicates that many personalities are more susceptible to psychic injury and thus, even with slight trauma, a major, prolonged disability in the psychiatric sphere appears if the personality is predisposed. If to this is added an economic factor the chances of a psychological disability are greatly increased.

IN SUMMARY:

In any disaster, major or minor, the doctor can fulfill a tremendous need to a survivor or his family. In either personal or national tragedy the physician by using fairly simple techniques can do a great deal to help with the immediate anguish, anxiety, guilt and fatigue, and prevent very severe consequences. He can follow these simple steps:

(1) In anxiety and guilt, permit the patient to talk it out — LISTEN.

(2) When it has subsided, reassure the patient and his family and THEN offer sedation or tranquilizers orally, or in more severe cases, parenterally.

(3) We would emphasize the early investment of time and the patience of listening. Reassurance and interviewing should be done with the patient privately, then the doctor can explain things and reassure the family.

ACKNOWLEDGEMENTS

I wish to acknowledge the contributions and technical assistance of my colleagues from the Department of Psychiatry, the Department of Psychology, especially Dr. Mary Laurence and Doctor C. A. Gordon, Department of Medicine at the Victoria General Hospital.

The Tonsils and Adenoids: Their Function and Disposition

A. G. Shane, M.D.

THIS topic has been discussed many times at meetings and in the journals and yet these easily accessible, much removed organs still have an air of mystery about them. At the outset let me state we do not know a great deal about the function of the Tonsils and Adenoids, yet a great deal of time has been spent in their study and removal.

The Tonsils and Adenoids are an integral part of the protective Waldeyer's ring at the ostium of the foregut and also of the sub-epithelial lymphoidal system of the respiratory and alimentary tracts, which also include the peyers patches of the intestines and our old and sometimes much maligned friend, the appendix. Rather than call these vestigial organs of no known function and serving no useful purpose, except to fall under the surgeon's knife, they have a function to perform similar to the other lymphoid tissue aggregations and like them undergo atrophy after 40 years of age; since these are specialized parts of the lymph system it is necessary to understand in a general way the nature and purpose of the lymphatic system as a whole.

The lymphatic system consists of a series of fine permeable capillary tubes which are situated beneath the skin, mucous membranes and gastro-intestinal tract, which are interrupted at intervals by the lymph nodes. The capillaries contain a fluid similar to tissue fluid and being permeable absorb end products of cell metabolism from the surrounding tissue which is not absorbed by the circulating capillaries of the blood stream. These smaller lymphatic capillaries unite to form larger efferent vessels which contain valves and thus limit fluid flow in one direction only. These larger vessels are interrupted at intervals by lymph nodes and from these the lymph is returned by the efferent vessels to the blood stream.

The lymph nodes consist of a supporting frame work enclosing spaces lined by reticulo endothelial cells and containing a tightly packed mass of free cells, the lymphocytes.

From this very meagre description, one can see that the lymphatic system is an open secondary circulatory system and not a closed one like the vascular system, because the tissue fluid so collected is passed into the blood stream by the endolymphatic duct. The circulation of this tissue fluid is slow and depends chiefly on muscular contraction or activity. The tissue fluid which passes through the lymph nodes and from here to the blood stream is altered, in that some of the fluid is removed by filtration, and extraneous materials such as inert foreign matter and micro-organisms are removed by the phagocytic action of the reticulo endothelial cells, and at the same time new lymphocytes are added to the efferent fluid for the blood stream.

Summing up one can say that the lymphatic system returns those proteins from the tissues to the blood stream which cannot be absorbed by the capillaries; it removes some of the waste products of cell metabolism and removes foreign materials and micro-organisms by a process of phagocytosis and filtration. We all know about the red lines that appear on the arm when a finger is acutely infected, along with tender glands in the axilla and that in chronic infections

the lymph glands are enlarged and the surrounding tissue infiltrated with lymphocytes at the focal point, thus minimizing or preventing extension of the infection to the blood stream.

From what has been described, and other evidence, we may say that the lymph nodes help, to, produce antibodies that will control bacterial and viral infections and their toxins, and manufacture new cells of the phagocytic, reticulo-en-dothelial and polymorphonuclear varieties. This results in the body acquiring a degree of immunity, with each succeeding attack. With increasing age the resultant immunity of the patient to bacterial infections increases and therefore there is less need for the immunizing function of the lymphatic system so that the adenoids, tonsils, and appendix atrophy and almost disappear in a large proportion of the population.

The tonsils and the adenoids are a specialized part of the sub-epithelial lymphatic system and these lymphoidal collections or aggregates have no efferent lymphatics like other lymph nodes. They are placed in intimate contact with inspired air or ingested food and are directly exposed to infection and bear the first impact. An important feature of these sub-epithelial concentrations is that they are found where bacterial activity is greatest. The tonsils and adenoids are admirably situated in this respect, the adenoid with 5 or 6 prominent parallel leaves, exposes a maximal surface to the inspired current of air with its content of micro-organisms. The tonsils situated on either side of the oropharynx consist of masses of lymphoid tissue with deep crypts and are directly exposed to the food we eat and in children to the various combinations of contaminated articles entering the mouth. The crypts contain particles of food, digested or in the process of digestion, desquamated cells, bacteria, and at times hard calcareous masses of these materials.

We all know that tonsillitis is a localized infection of the tonsils, and the degree to which these tissues are damaged is shown by the fact that approximately 40% of children between the ages of 2-5 years suffer from recurrent attacks of tonsillitis and adenoiditis. These tissues bearing the first impact of infection, show their greatest activity during this early period of life when immunities are being developed. In older age groups when the defensive mechanisms have been developed these tissues tend to atrophy and almost disappear, leading sometimes to disastrous results from an overwhelming infection.

A very great amount of time and effort has been spent in removing tonsils and adenoids as well as appendices. Probably these operations are the most commonly practiced, but we have not learned a great deal from their removal, except to say that if the appendix is removed that person will no longer suffer from attacks of appendicitis either of the acute or chronic variety. If the tonsils and adenoids are removed, we can say that this person will no longer have attacks of acute or chronic tonsillitis, or of Quinsy. The cervical glands will not become grossly enlarged and he will be less susceptible to diphtheria, Scarlet Fever or Rheumatic Fever. He will, however, still be liable to attacks of pharyngitis and upper respiratory infections. We know that when the adenoids are removed in children under 2 years of age, the tonsils will enlarge and eventually will have to be removed together with a regrowth of adenoidal tissue.

A. J. Wright concludes that the sub-epithelial and lymphoid tissues are primarily concerned with the production of natural immunity by virtue of a mechanism for the slow introduction of samples of infecting organisms into the lymph stream. These samples transverse the lymph gland at the angle

of the jaw and here come in contact with the reticulo endothelial cells and lymphocytes. They undergo changes and are passed into the blood stream with the production of subsequent immunity.

In our everyday life these special organs may be overcome by repeated or massive infections and instead of acting as a filter, and passing on this infected material in altered form, they become overwhelmed and themselves become a centre of infection. Having failed in their natural function, and become centres of infection rather than barriers and producers of properly digested end products giving rise to immunity, they have to be removed.

In the removal of tonsils and adenoids, we try to obtain as complete a removal of tonsil and adenoid tissue as possible with a minimal degree of local trauma. We achieve this by the dissection method of tonsillectomy and by curettage of the adenoids. For many children, the complete removal of the adenoids is more important than the tonsillectomy. The removal of adenoids by the caged adenotome alone would leave many operators astounded if they were to elevate the palate and view directly the remains of the adenoids, or use their fingers afterwards to feel redundant tissue left behind. I have heard the remark passed that some residual adenoidal tissue should be left behind to keep the nose and throat moist; this I fear is wishful thinking.

We also try to avoid psychic trauma to the child, with its sequelae of fear and night terrors. These post-operative symptoms are allayed somewhat by the use of pre-operative sedation and full anaesthesia. A frank discussion and explanation of the operation with the child and parents beforehand is also helpful. We all err in not taking enough time to talk to the child and to make him familiar with the hospital ward and operating room. In those cases which I have taken the time to discuss the operation with the child and parents not forgetting to mention that the little patient will be sick the first day, and that the throat will be sore for a few days, I have had a smoother post-operative period than cases in which this has been neglected.

We do not remove tonsils on account of size alone; size itself is of no major importance unless it is creating dysphagia, respiratory or phonatory disturbances. You all know the child who presents with tonsils, large as walnuts, meeting each other in the mid line or actually pushing each other out of the way, sitting with his mouth open, snorting and talking as though his mouth were filled with marbles. This child cannot eat solid food without difficulty because he is not able to get larger pieces between these projecting masses of lymphoid tissue. In a child under 2 years of age, the main offending organ is the adenoids. X-ray will show in many cases a complete block of the post nasal space. All children wish to breathe through their nose and many will make desperate attempts to do so. Such youngsters cannot feed well, and have a mucoid or purulent nasal discharge. They may develop bronchitis or bronchopneumonia, and some of these children have attacks of nasopharyngitis with temperatures of 103-105°:, giving rise to many unexplained febrile illnesses. In the 2-4 age group, both tonsils and adenoids may be enlarged giving the combined symptoms of respiratory, phonatory and swallowing difficulties and it is in this age group, we achieve, if I may be allowed the words — "brilliant and dramatic results" from surgery.

In recurrent attacks of tonsillitis, with temperature elevation requiring antibiotic therapy, the removal of the tonsils and adenoids reduces the incidence or severity of sore throat to a marked degree.

One attack of Quinsy is sufficient reason to remove both tonsils and adenoids.

Cases of chronic upper respiratory infection, colds that persist or recur with great frequency, and lower respiratory infections associated with congestion and hypertrophy of the tonsils and adenoids, in which one presupposes that the infection is initiated in these structures and also in cases of chronic recurrent bronchitis and bronchectasis, the tonsillo-adenoidectomy operation is fully justifiable in order to improve the child's general health.

There is another group of children in which the direct symptoms are vague, which may require removal of tonsils and adenoids. In this group the tonsils and adenoids are regarded as foci of infection for systemic disease, e.g. nephritis, rheumatic fever, osteomyelitis, chorea, and dermatitis. In these cases one depends on the physician concerned and acquiesces to his wishes, One may also class with this group, children who do not seem to be in robust health, are prone to feeding problems, and fail to gain weight; if no medical cause can be found to account for their poor health they may have their tonsils and adenoids removed. A good number of these children will do well afterwards and I personally feel that one should not hesitate to remove them.

I mention diphtheria carriers only to state that if after Penicillin therapy, K.L.B. are still grown, the tonsils should be removed.

In cases of tuberculosis of the glands of the neck, the tonsils are the portal of entry and the surgical opinion at present is that the glands should be dealt with first, followed by tonsillectomy later.

In those cases of non-specific grossly enlarged cervical glands in which tonsils and adenoids have apparently lost their power to assist in their protective function and have themselves become the main offenders, I believe the operation should be performed.

There is a large group of children who suffer from a nasopharyngeal syndrome; this consists of conduction deafness and recurrent attacks of otitis media, either of an acute or chronic nature. This state of affairs may be caused by the hypertrophy of the adenoids themselves or by a direct spread of infection to the Eustachian tube from infected adenoids, tonsils or sinuses. One attack of acute otitis media is not in itself a criterion for removal of tonsils and adenoids; with the judicious use of the antibiotics at our disposal in adequate dosage, the ear will heal, but if there are repeated exacerbations of acute otitis media, we are justified in doing the tonsillo-adenoidectomy operation There are many children with a chronic discharging ear, presenting an anterior inferior perforation or central perforation and a mucoidal discharge, who will usually do well with their tonsils and adenoids removed. Such children should be put on protective antibiotics, before and after operation, because the affected ear will flare up and become worse during this time. The patient will also be liable to acute exacerbations from time to time especially during attacks of acute rhinitis.

Some cases of sinusitis in children will be helped by removal of their tonsils and adenoids, rather than putting them through a rigorous routine of antral washout and radical antrum procedures with doubtful results; one should try a tonsil-adenoidectomy first. Many cases of deafness in children, not associated with a previous attack of otitis media, in which the tympanic membrane is markedly indrawn and presenting conduction deafness will have their hearing ability improved by this operation.

I have left to the last the most important section of this topic, which is the history and advice of the referring physician. There are a few cases in which I feel I can safely say by examining the child that he needs the tonsils and adenoids removed, but in most of them I cannot say this on direct exam-

ination alone. A large number of tonsils do not appear to be infected to me. I know that we are given certain signs, such as large tonsils, tonsillar pits filled with a whitish creamy, pussy, fluid, and injection along the anterior pillars, but to me these are signs of small consequence. Many large tonsils cause no difficulty to the patient, and you can squeeze whitish debris out of almost any tonsil. People who smoke or live in a large city, have not only injection along the anterior pillars, but also of most of their upper respiratory tract. If the tonsils are causing dysphagia, respiratory or phonotary troubles, then I am able to say by looking at such tonsils that they ought to be removed, but of the others, and they are in the majority, I depend on the history. This information is readily obtained from the parents as is the case history and advice of the child's paediatrician or family physician. He knows how many times he has had to see this child for sore throats, persistent head colds and the other symptoms I have outlined above and if he feels that they require removal, I am pleased to oblige him. I feel that there is no age barrier, but under 3 years of age I usually remove the adenoids alone, while over 3 years I feel that both the tonsils and adenoids should be removed. The Radiologist can give you much help in the child under 4 or 5 years of age by an X-ray of the nasopharynx which may show anything from a partial to a complete block by adenoidal hypertrophy.

There are a few definite contra-indications to tonsillo-adenoidectomy.

I do not remove tonsils or adenoids during an attack of acute tonsillitis or during an acute upper respiratory infection. We feel it is best to wait for about 6 weeks before operation.

In a proven case of Haemophilia or other blood dyscrasia, the operation is contra-indicated.

Age is only a relative contraindication, in that the operation is usually not done under 3 years of age or over 60 years of age.

During an acute epidemic of poliomyelitis, one certainly must not remove these organs. If there is no polio epidemic then I feel the operation can be done quite safely, but due to press reports and public opinion, I myself do not operate during the summer months, unless absolutely necessary. This last contraindication may have to be revised in future with the general use of Salk Vaccine.

If one regards some or any debris in the tonsillar crypts as evidence of tonsillitis, the disease would be universal. The diagnosis of chronic tonsillitis cannot be made on a single morphological change, but only by the sum of all the changes including the history and examination. Enlarged tonsils in children are physiological and not necessarily a result of chronic inflammation. This hypertrophy may be hereditary and often occurs at the age of 4 or 5 years with subsequent regression in some cases at puberty. If there is any doubt in your mind as to the necessity of removing these organs, a wait of 2 to 3 months will do no harm. The removal of tonsils and adenoids is an elective procedure and not an emergency. Most children will do well if properly selected for operation both to your own and the parents satisfaction.

BIBLIOGRAPHY

Wright, A. T., 1950, J. Laryng. and Otol., Vol. LXIV.
Scott-Brown, W. T., 1952, Diseases of the E.N.T., Butterworth.
Wilson, T. G., 1955, Diseases of the E.N.T. in Children, W. M. Heinemann.
Ellis, Maxwell, 1954, Modern Trends in Diseases of the Ear, Nose and Throat. Butterworth.

The Chronic Neurological Disease Survey Halifax County 1957

PRELIMINARY REPORT

JOHN O. GODDEN

IN the summer of 1957 a survey was made of many sources of medical information to bring to light instances of diagnosed cases of selected neurological and ophthalmological disease in Halifax County. For the previous ten years pertinent records of selected diagnoses were abstracted from the record systems of all local hospitals, clinics, nursing services and departments of public health. The results of this survey produced file cards on 8,000 patients. In order to find these, our student surveyors reviewed approximately 200,000 diagnostic cards and scanned 20,000 charts at 21 sources. The cases found in the record systems at these sources were augmented by material derived from death certificates and still-birth forms in the files in the Department of Public Health for the same ten year period.

An example of the distribution of neurological diagnoses is given below for multiple sclerosis, conditions allied to multiple sclerosis and other well defined neurological diseases. This arrangement of diagnostic material is the one chosen for the modified questionnaire being prepared for circulation to the physicians of this country.

Multiple Sclerosis		124
	Suspected Multiple Sclerosis	56
Conditions Allied to Multiple Sclerosis		
1.	Transverse Myelitis	3
	Myelopathy	2
	Encephalomyelitis	0
2.	Cerebellar Ataxia Degenerative	1
	Hereditary Ataxia	0
3.	Acute Retro Bulbar Neuritis } Optic Neuritis }	18
8.	Primary Lateral Sclerosis	6
5.	Paraplegia	17
Other Neurological Diseases		
1.	Guillain-Barre Syndrome (Infectious Polyneuritis)	3
2.	Myasthenia Gravis	4
3.	Tic Doloreaux	80
4.	Syringomyelia	11
5.	Mongolism	71

The companion survey in Charleston, South Carolina has proceeded far enough to give report of the results of clinical screening of multiple sclerosis and allied entities produced by their combined hospital-physician survey. Correspondence with the practitioners in Charleston County was carried on with 152, or 80%, of the 191 doctors living in the community. Letters were not sent to retired practitioners, radiologists, laboratory and other non-clinical workers. This correspondence yielded 130 replies, approximately 85% of those circularized having responded and 35 of the 152 or approximately 23%

giving information relating to cases of multiple sclerosis or retro bulbar neuritis. When the cases identified in the hospital survey and those contributed by the practising physicians were carefully scrutinized the following numbers in the various diagnostic categories remained. The same figures produced by the hospital survey alone for Halifax County are shown for comparison.

	Charleston	Halifax*
Possible M. S.	86	180
"Primary Lateral Sclerosis"	12	6
Paraplegia	10	17
Cerebellar Ataxia	23	6
Myelitis, Myelopathy, Encephalomyelitis.	29	9
Optic and Retrobulbar Neuritis	27	18

In Charleston, all sources produced 187 cases in which the symptoms might conceiveably be due to multiple sclerosis. Thirty-six of these were discarded at once after reading the case notes. Eighty-five had died at some time during the ten year period under survey and 38 were discarded because they were non-residents. Of the remaining 79 cases 63 have been seen by the neurologist and 15 still remain to be seen at the time of writing. Out of this material 38 cases of multiple sclerosis were living on the prevalence day and 7 had died. From these 38 cases living on "prevalence day" the prevalence rate for Charleston County on December 31st, 1955 is 38 per 188,722 or 20 per 100,000.

The first phase of the neurological survey in Halifax County is completed. The student resea chers did an excellent job and much of the ultimate success of this study will be due to the thorough and persevering efforts of these four medical students during the summer of 1957. However, a second phase of the survey depends for its success on the active co-operation of the practising physicians of Halifax County. In Charleston County 26% of all cases discovered were contributed by the practising physicians. Eight-five per cent of the physicians approached answered the questionnaire and 23% of those were able to give valuable information. If the results in Charleston County and Halifax County are to be comparable the medical profession in Halifax County should take an active interest in providing this valuable information about the incidence of these diseases in our area. The process of statistical and neurological scrutiny sharply reduces the number of suspected cases. The initial case-finding efforts must be as thorough as is humanly possible in order to allow the final result to approximate the true incidence of the disease. During the months of March and April the practising physicians of Halifax County will be approached again with a final definitive reporting form. Doctor R. S. Allison of Belfast, Northern Ireland, who visited Halifax in November, will return in June prepared to examine all suspected cases of multiple sclerosis and certain other neurological diseases to assure comparability with the Charleston County material. This survey, if it is completed successfully, will place Halifax and Halifax County with other medical centres that are currently co-operating in a global effort to define the specific features of the epidemiology of these mysterious neurological diseases.

*Data not yet available for practising physicians.

Letters to the Editor

To the Editor
Nova Scotia Medical Bulletin

Dear Sir: Re Chiropractors

A letter was written for the January issue of the Bulletin to inform the members of the medical profession of what had taken place in making effective the directive of the Annual Meeting 1957, viz. to oppose proposed legislation by the Chiropractors.

Since that time, as was anticipated, yet another private bill has been introduced to the 1958 session of the Legislative Assembly. This is Bill No. 33 which was given first reading on February 20 and second reading on February 25. Second reading, which included the recommendation to refer it to the Committee on Law Amendments, resulted in a two hour debate and a standing vote which resulted in 22 for further reference and 14 against. The debate and the vote both crossed party lines. An announcement in the press states that the public hearing before the Law Amendments Committee will be held Wednesday, April 9th, at 8.00 p.m.

Following meetings of our Nucleus Committee on Legislation, a meeting of the Full Committee was called on February 13th, during which a discussion of the matter was again undertaken. Doctor D. I. Rice, Chairman of the Committee on Public Relations, is working as an ex-officio member of the Committee on Legislation. As a result, the reasons for the stand of the Medical Profession have been placed squarely before the public. This has been further enhanced by two editorials in the Halifax Chronicle-Herald, both of which were completely independent opinions on the matter.

The Nucleus Committee of the Committee on Legislation has met on several occasions and the physician members of the Legislative Assembly have been well briefed as to the problem and developments. The President of each Branch Society has been telephoned and brought up to date and has agreed that the Branch will approach their local representatives in the Legislature to inform each officially and through individual physicians the dangers inherent to the public in the proposed legislation. Five Branch Societies have reported on what has been done. It would appear that legislators have more appreciation of the facts involved than formerly.

The Nucleus Committee on Legislation has met with the Legislative Committee of the Provincial Medical Board. During the discussion plans were formulated for presentations to the Law Amendments Committee on April 9th.

The Premier and the Minister of Public Health have received reprints of correspondence and the contributed article "Of Cabbages and Kings." An important contributed Editorial—"Chiropractic and Medicine" appeared in the February issue of the Bulletin.

The Legislature recessed on March 7, 1958, to reassemble on April 8th. This is an interval of approximately four weeks. During that time more can be done to further inform the public about the truths of this matter. Physicians are urged to avail themselves of every opportunity to make the public and the legislators aware of the real dangers which are inherent to the proposed legislation.

J. McD. CORSTON, M.B.,
Chairman, Committee on Legislation.

Secretary's Page

C. M. A. MEETING — JUNE 16-20, 1958

Plans and the programme for this meeting will be presented in the C.M.A.J. in the near future.

Housing Accommodation for C.M.A. Meeting

Applications for housing accommodation are now being received by the Committee on Housing and it would appear that a good attendance can be expected.

It has been the custom, over the years to house members of general council, executive members, speakers and a percentage of the exhibitors at the "headquarter" hotel or hotels, and this procedure will be followed this year.

As the number of these "VIP's" will approach three hundred, it is plainly seen that the two main hotels in Halifax will be filled with guests.

Maritime members of the C.M.A. particularly those in Nova Scotia are asked to consider securing their own accommodation.

This is not meant to scare anybody away from the meeting, as plenty of accommodation is available for *any number* of people who intend to attend the meeting. The thought is that some members would rather stay with friends than be billeted in tourist homes or a university residence. *However, do not hesitate to apply for accommodation, as the Housing Committee will be only too pleased to secure it for you.*

M. R. Macdonald, M.D.,
Chairman, Housing Committee,
Canadian Medical Association.

Reunion of Graduates Dalhousie Medical School

Plans are progressing to have such a reunion during the C.M.A. meeting. It will take place on Thursday evening, June 19, at 6.30 p.m. Detailed information will be available for the April issue of the Bulletin.

Medical Society of Nova Scotia — Annual Meeting, 1958

The Executive Committee at its meeting in August, 1957, decided to approach the Refresher Course Committee with a request that Friday, October 24, be made available for the Annual Meeting of The Medical Society of Nova Scotia. The reason for this was to avoid any conflict with the C.M.A. meeting in June. This request has been granted. The decision is much appreciated. The Refresher Course will be October 20-23 inclusive, followed by the Annual Meeting of The Society on October 24, and Saturday, October 25th.

Income Tax Returns

The attention of members is drawn to the February 15th issue of the C.M.A.J. where on P. 289 is published the text of a memorandum approved by the Department of National Revenue for the guidance of doctors relative to the year 1957.

REPORTED INFECTIOUS DISEASES — NOVA SCOTIA

Summary for month of January, 1958

	NOVA SCOTIA				CANADA	
	1957		1958		1957	1958
	Cases	Deaths	Cases	Deaths	Cases	Cases
Brucellosis	0	0	0	0	9	8
Diarrhoea of Newborn	0	0	0	0	0	0
Diphtheria	0	0	0	0	17	7
Encephalomyelitis Infectious	0	0	0	1	1	0
Food Poisoning	0	0	0	0	0	0
Gastroenteritis (1) Infectious	0	6	0	1	50	55
Hepatitis—Infectious Including Serum Hepatitis	0	0	0	0	0	0
Impetigo of Newborn	0	1	0	0	0	0
Influenza (if unusual number of cases)	94	3	382	5	307	856
Meningococcal Meningitis and Meningococcimia	0	2	0	1	18	43
Pertussis	0	0	103	2	608	815
Poliomyelitis (paralytic)	0	1	0	0	6	3
(non-paralytic)	0		0	0	2	2
Scarlet Fever and Streptococcal Sore Throat	290	0	819	0	910	1508
Tuberculosis (pulmonary	6		5		420	847
(non-pulmonary)	0	4	0		44	56
Typhoid and Paratyphoid Fever	0	0	0	0	23	24
Venereal Disease (syphillis	2		5	2	143	223
(gonorrhoea)	40	1	38		1117	1506
Anthrax	0	0	0	0	0	0
Cholera	0	0	0	0	0	0
Psittacosis	0	0	0	0	0	0

(Continued on next page)

REPORTED INFECTIOUS DISEASES (Concluded)

	NOVA SCOTIA				CANADA	
	1957		1958		1957	1958
	Cases	Deaths	Cases	Deaths	Cases	Cases
Rabies	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0
Tetanus	0	0	0	0	0	0
Trichinosis	0	0	0	0	0	0
Tularemia	0	0	0	0	0	0
Other rare diseases	0	0	0	0	0	0
Other (if unusual number of cases)	0	0	0	0	0	0

(1) amoebic and bacillary dysentery and salmonellosis.

Remarks: Over the past year the Department of Public Health has been working on a revision of Regulations in Respect of the Communicable Diseases for the Province of Nova Scotia. The new regulations have now been completed and have been put into the hands of all practising physicians in the Province and others. Among the changes in the revised regulations is the change in the list of notifiable diseases. Some of the less important diseases such as chickenpox have been dropped from the list and certain diseases which were not previously on the list, and which are considered to be quite important such as impetigo of the newborn, have been added. It is hoped that this revised list, which, for the most part, contains the preventable communicable diseases, will be an improvement over the previous list. It will eliminate some unnecessary work, as well, for the practising physician, upon whose shoulders rests the onus of the reporting.

The list of diseases in the above table is the new list of notifiable diseases. The table gives the number of cases of each diseases officially reported to the Department of Public Health for the month of January 1958 and for January 1957. The number of cases for Canada are given for comparison. The table also gives the deaths from these communicable diseases which were filed by the Vital Statistics Department during the same months. It is planned to give this information to the Nova Scotia Medical Bulletin each month along with any other notes of interest regarding communicable diseases.

It must be noted that the above figures are only tentative and therefore subject to some change. Also the above table is incomplete due to the fact that the report forms containing the new list of notifiable diseases are not yet being used. It is hoped that the table will improve as the months go by.

It is in the interest of the medical practitioner to report his cases faithfully so that the information in the above table will be accurate.

In addition to the above information there were two deaths from pneumococcal menigitis reported in January, 1957. There was one death from meningitis (type not known) in Nova Scotia during the same month.

Tuberculosis deaths for the month of January 1958 are not available at this time.

DALHOUSIE UNIVERSITY — MEDICAL RESEARCH COMMITTEE

Abstract of Clinical Research Meeting, October 30, 1957

The Genesis of Abortion

W. R. C. TUPPER, M.D.

A multi-disciplinary study of the causation of spontaneous abortion was begun a few years ago. Most of the findings of this study were of a negative nature but one positive finding has opened up an intriguing field of speculation. Certain histological changes in the placentas of aborting women not previously reported were found. By using differential stains, the Pathologist was able to find in the placentas of approximately 70% of spontaneous abortions the presence of lesions of a collagen nature. Since it is well known that the blood serum in collagen diseases contains a substance which will agglutinate the red blood cells of sheep, we felt that if our assumption of the above was correct, then the serum of aborting women should also contain this agglutinating substance. We set about to prove this. Using a modified form of the Rose-Waaler test we were able to show that the serum of a large percentage of aborting women did contain a substance which would agglutinate the red blood cells of sheep and which was aborted when placental extract was added to the serum. Controls for the above investigation were made up of normal unmarried girls, pregnant women at term and normal pregnant women of three months gestation.

It is a bizarre suggestion that two conditions with such divergent clinical pictures as spontaneous abortion and collagen disease would appear to unite as a pathological entity. However, the histological lesions are similar, the serological response to the disease process appears to be the same and the antibody absorption phenomenon almost identical.

Further investigation is being done on this problem.

DALHOUSIE UNIVERSITY MEDICAL RESEARCH COMMITTEE

Abstract of Clinical Research Meeting, January 22, 1958

Diagnostic and Therapeutic Experiences with Radioactive Iodine

B. STEWART, J. STAPLETON and W. MORSE

Observations on the diagnostic use of radioactive iodine in thyroid disease obtained at the Victoria General Hospital from August 1956 to June 1957 were reported (see July 1957 issue, N. S. Medical Bulletin).

Results of radioactive iodine therapy in thirteen cases of hyperthyroidism treated at the Victoria General Hospital between March and July 1957 were also reported. Seven of these patients were rendered enthyroid and continued to have normal function at their most recent evaluation (maximum observation period nine months). Five patients showed continuing hyperthyroidism and a second therapeutic dose of radio iodine has been administered to these cases. One patient has developed hypothyroidism. It is not known at present whether or not this will be permanent.

Indications for radio iodine therapy and the value of diagnostic studies with radioactive iodine in predicting the effects of therapy were discussed.

Dalhousie University Medical Research Committee

Abstract of Clinical Research Meeting, November 13, 1957

Serum Cholesterol Research to Marine Oil in Cases of Atherosclerosis

C. M. HARLOW, LEA STEEVES and A. MYRDEN

A marked decrease in the amount of ordinary fats in the usual American or Western European diet, without any change in the amount of calories or vitamins, lowers the serum cholesterol level. The fall is rapid in the first few days, but after a few weeks there is an approach to a new plateau (Keys). Such low fat diets usually contain less cholesterol and animal proteins, but the change in the serum cholesterol level in man does not depend upon this fact (Keys and Anderson). The responsible agent is clearly either in the quality of the fats or in the ratio of fat to carbohydrate calories.

It is tempting, then, to ascribe the effect of low fat diets to the decrease in animal fat, especially since some vegetable and marine oils given in large amounts may depress the serum-cholesterol level (Kinsell) (Bronte-Stewart). It appears that the effect of dietary fats on the serum cholesterol should be related to chemical composition rather than to origin.

For the past year we have conducted at Camp Hill Hospital a small Research project in which we investigated the effect of North Atlantic seal oil on a group of patients with atherosclerosis and hypercholesterolemia.

The group included cases of ischemic heart disease and peripheral atherosclerosis with claudication. All patients were placed on a low fat diet (5%) for two weeks to one month. During that time there was a definite lowering of the cholesterol level (340 mg% to 280 mg%). The patients were then given 26 cc. of seal oil twice a day. After a further two months, the cholesterol level decreased to around 200 mg%.

It is interesting that we were able to lower the cholesterol level of 10 patients with atherosclerosis and hypercholesterolemia on a low fat diet and that a further lowering of the cholesterol could be obtained by adding 50 grams of seal oil (450 calories) to the low fat diet.

ABSTRACT

It has been the author's practice for a good many years to advise patients with severe eczema-dermatitis that it is wise to use old well washed soft cotton, such as pieces of pillow case or sheeting, rather than new gauze. The former are likely to be much less irritating in a mechanical sense than the new gauze, and it is not too important that the dressing should be absolutely sterilized. In Archives of Dermatology and Syphilology 61: 118 (Jan.) 1950, Lester Hollander reports on a case where the areas of dermatitis were noticeably irritated within ten minutes of the application of any one of three makes of sterile gauze. However gauze patch tests applied to the normal skin of the same patient produced no reaction. Attention to this little detail may prove very useful to the general practitioner in giving advice to the patient with dermatitis. I have not seen the matter discussed elsewhere.

Received from

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DALHOUSIE MEDICAL RESEARCH COMMITTEE TOPICS FOR CLINICAL RESEARCH MEETINGS*

March to May, 1958

March	12	Observations on Post-maturity in the Halifax Area by I. A. Perlin.
March	26	Observations on Carcinoma of the Uterus and Vulva by M. G. Tompkins, R. B. Thompson and S. C. MacLeod.
April	9	Palliative Therapy in Breast Cancer by G. W. Bethune.
April	23	Incidence of Psychiatric and Organic Symptoms in a Small Town by J. O. Godden and C. B. Stewart.
April	30	The Pathological Changes in Sudden Unexpected Death in Infancy by C. P. Handforth and Ruth Faulkner.
May	14	Blood Vessel Surgery: (1) Experimental Cranial Artery Surgery in Dogs by W. D. Stevenson and H. H. Tucker. (2) Freeze Drying of Blood Vessels by J. J. McKiggan, F. G. Dolan and C. E. van Rooyen.

*Held in Victoria General Hospital Auditorium at 5 p. m. All those interested are welcome.

Obituary

The death of Dr. Bernard Woodworth Skinner, age 68, of Mahone Bay, N. S., occurred on February 2 in the Dawson Memorial Hospital. Death was due to a heart attack.

Dr. Skinner was born at Weston, Kings County, and was the son of John W. and Eunie Robblee Skinner. He was a graduate of Acadia University and served in World War I with the First Canadian Expeditionary Force, with the rank of Captain. He returned to Canada and finished his medical course at McGill University and then went to Mesopotamia with the British Armed Forces and remained there for several years. Returning to Canada in 1921, he opened a practice in Hubbards, N. S., but in 1931 he moved to Mahone Bay. Dr. Skinner was a charter member of the Kiwanis Club. He was also interested in the March of Dimes, St. John's Ambulance, Red Cross, Anti-tuberculosis League, Boy Scouts, Little League and the Board of Trade, of which he was a charter member.

He is survived by a daughter, Betty (Mrs. William Porter), and a brother, Jean R. Skinner of Aylesford, and a sister Evelyn who is Mrs. Kenneth Illsley.

The Bulletin extends sincere sympathy to Doctor E. I. Glenister of Halifax on the death of his father, Ernest S. Glenister, retired Halifax Customs official and World War One veteran, on January 24th; to Doctor and Mrs. D. Bruce Keddy of Lunenburg on the death of their infant son, Donald Norman, on February 6th; and to Doctor G. B. Wiswell of Halifax on the death of his brother, A. C. Wiswell on March 6th.