

DISCUSSION.

DR. KARL K. KOESSLER, Chicago: Dr. Butler says that it makes no difference whether the reaction is made with syphilitic organ extract, with an alcoholic extract of a normal rabbit's heart, or with lecithin, according to Porges and Meier. I can not confirm his opinion. Although the Porges-Meier reaction is said to work with lecithin only, researches made in this line at Vienna have shown that results are obtained in only 45 per cent. of cases, while the Wassermann reaction gives about 90 per cent. The Porges-Meier method, however, involves quite a different principle from Wassermann's, being a precipitin reaction having nothing to do with the deviation of the complement. Any one who, like Dr. Butler, has worked out a great many cases, knows what a long process Wassermann's method is. Certainly it would be much simpler to use the alcoholic extract instead of the syphilitic; but comparison between positive results in each method gives preference to the original procedure of Wassermann. It is perhaps better, in accord with Levaditi's modification, to use a dried extract of the liver of a syphilitic fetus, in order that it may be preserved and a solution made when needed. On account of the enormous responsibility for the physician in making the diagnosis of syphilis he should use the method which gives the greatest number of reliable results and in uncertain cases repeat the test three times at intervals of ten days and make the positive diagnosis only within the month.

DR. WILLIAM J. BUTLER, Chicago: Time would not permit the reading of that part of the paper referring to the Porges-Meier reaction. I used the Porges reaction in a small number of cases and compared it with the Wassermann and the Klausner reactions. Recent investigations show that it is not specific, because it is found that tuberculous will give a positive reaction in about as large a number of cases as will syphilis. I did not find it positive as often as I did the Wassermann reaction in the cases of syphilis in which I used it. I notice, however, that Porges and Meier in a recent publication have compared the Wassermann and lecithin reactions and report almost as many positives with the latter as with the complement fixation method.

As to the relative value of alcoholic and salt solution extracts, Wassermann contends that the latter extracts give a more reliable reaction than the alcoholic. I can not say that in my own observation I have had an opportunity to compare the aqueous saline solution and the alcoholic extracts with the same sera; but it seems to me, comparing my previous experience with the aqueous extract, that the alcoholic works as well as the salt solution and it has the immeasurable advantage of being stable. I do not know of anything more difficult than to work with the salt solution extracts. They are most unreliable; sometimes in a few hours, or over night, a large quantity of liver extract will go wrong and be useless. Every test made is controlled by a series of normal and syphilitic sera. The use of these controls obviates any chance of error. If under these circumstances a suspicious serum is found positive, it can be said without hesitancy that the patient has syphilis.

THE NATURE AND THE CAUSE OF EDEMA.*

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INTRODUCTION.

The remarks that follow represent one of the possible applications of a principle which for some time past I have been interested in establishing experimentally, namely, that in the variable affinity of colloids for water we have the explanation of many of those physiologic

phenomena which are characterized by a storage or a migration of water.¹

If we ignore certain "vitalistic" conceptions of edema in which fantastic properties of "living" cells are supposed to account for the phenomena observed, the increased amount of water held by tissues in a state of edema is usually attributed to "changes in blood pressure," and to "alterations in the permeability of vessel walls." The alterations in the permeability of vessel walls have never been demonstrated experimentally, and all efforts to produce states of edema through simple increase in blood pressure have failed. The slight edema observed by some investigators after prolonged intravenous injections at high pressure of enormous amounts of various liquids can be explained more easily by changes brought about in the colloids than through simple pressure effects.

Here and there one meets a phrase in the discussion of edema which suggests that the author at least assumes the possibility of a cause for this condition in the tissues themselves, but I know of no experiments planned to actually prove this point, or of any attempts to define the nature of these tissue changes physico-chemically, except those of Jacques Loeb,² who, ten years ago, tried to find in an increase in the osmotic pressure of the tissues the cause of the increased absorption of water. The inadequacy of this explanation has since become apparent, but the experimental facts adduced by this author to show that the cause of edema resides essentially in the tissues are of permanent value, and should have received a recognition at the hands of pathologists which has never been accorded them.

THE CAUSE OF EDEMA RESIDES IN THE TISSUES.

For a starting point in our discussion it is well to take the results of some experiments which prove conclusively that the cause of edema resides primarily in the tissues. When this is proved we can say that tissues become edematous not because water is forced into them, but because they absorb water from liquids flowing through or about them. An analysis of the changes in the tissues leading to the increased absorption of water may then be attempted.

My experiments consisted in passing a ligature about one hind leg of a frog or a toad sufficiently tightly to close both the veins and the arteries of that leg, and then placing the animal in a vessel containing enough water to cover the legs. A ligature placed just above the knee where lack of musculature makes ligation of the vessels an easy matter proved most satisfactory. In such an entire absence of the circulation the ligated leg begins to swell and in the course of a few days develops an intense edema. The edema is well marked at the end of twenty-four hours, and in two or three days the weight of the ligated leg may come to be almost twice that of the unligated leg.

The picture presented by the edematous legs is a duplicate in every way of the most intense states of edema observed clinically. The tissues are boggy, pit on pressure, and when incised allow the escape of fluid. If the ligature is left in place for two to three days small vesicles form which may enlarge into great blebs con-

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1. Fischer, M. H.: Physiology of Alimentation, New York, 1907, pp. 268, 182-187, and 267-269. Fischer, M. H., and Moore, Gertrude, *Am. Jour. Physiol.*, 1907, xx, 330.

2. Pflüger's Archiv. für die gesammte Physiologie, 1898, lxxi, 468.

taining a blood-stained serum.³ Later, these blisters rupture and allow the escape of their contents, and the weight of the ligated leg diminishes. This progressive diminution in weight continues due, in part, to loss of substance, in part to changes which no longer allow the leg to hold the large amount of water it once held,⁴ until finally the ligated leg weighs no more than the unligated. If the animal is kept sufficiently long (two to three weeks) the ligated leg is lost entirely (gangrene).

The increase in the weight of the ligated leg is entirely due to the absorption of water. This water is held in the skin, in the tissues composing the body of the leg—chiefly the muscles—and in the lymph spaces found between the skin and the underlying tissues. With this third division must be mentioned the fluid found in the blisters, which it is well to point out, in passing, are formed in the body of the skin.

As is to be expected, the development of an edema in the ligated leg of a frog or a toad kept in a little water will occur when the animal has been pithed just as well as in the normal animal, but if the frog or toad is killed, the difference in the weight of the two legs does not appear. This is not, however, because the edema does not develop in the ligated leg, but because it develops equally well in the other leg (which has through the death of the animal been deprived of its circulation). Herein is found the explanation of the fact that the tissues of dead bodies which are kept in water swell.

An objection which is likely to be lodged against my experiments is that I did not tie the ligatures sufficiently tightly to shut off both veins and arteries. That this was accomplished is proved by the fact that if the frog or toad was kept in a dry vessel the ligated leg soon dried so as to become brittle, while the unligated leg remained moist.

CHARACTER OF THE TISSUE CHANGES IN EDEMA.

It is clearly shown by these simple experiments that the severest grades of edema may exist without the presence of any "blood pressure" or a demonstrated "increased permeability of blood vessel walls." I shall try to show now that edema represents nothing but an increased affinity of the tissue colloids for water. Such an increased affinity of the colloids may be brought about in either or both of the following ways: *First*, in conditions leading to edema, various substances (particularly acids) capable of greatly increasing the affinity of colloids for water are not removed as they should be or are produced in abnormal amounts, and *second*, colloids having but little affinity for water are changed into such as have a greater affinity.

The first of these two factors plays the predominant rôle and is the only one discussed in this paper. We may regard it as settled that the amount of water held by a tissue under normal circumstances is essentially a function of its colloidal constitution for the same external circumstances which make certain so-called hydrophilic colloids either take up or give off water, make animal tissues do the same.⁵

If, now, the conception of edema as a condition in

which this affinity of the tissue colloids for water has been abnormally increased is correct, it must be possible to prove three things: (1) That protoplasm is colloidal in constitution. (2) That in the variable affinity of colloids for water we have a force of sufficient magnitude to account without strain for the maximum amount of water ever found absorbed by tissues in a state of edema. (3) That conditions leading to an increased affinity of their colloids for water exist in the tissues under circumstances associated with the development of an edema. We shall briefly consider each one of these.

1. Since the classical researches of Hofmeister, Pauli, Hardy and a few others, it is no longer questioned by any one that protoplasm represents a colloidal solution in which may be found embedded or dissolved a greater or smaller amount of extraneous material that need not be directly connected with the vital activities of the cell. Since colloids may, however, exist in several states, it is well to decide in which they are present in the cells. For our purposes it is best to distinguish between such colloids as have the power of swelling when placed in water (the so-called hydrophilic colloids) and such which do not possess this property. Many reasons can be adduced to show that protoplasm represents essentially a mixture of several hydrophilic colloids. Not only do dried albumin, fibrin, gelatin, etc., swell when placed in water, but a direct analogy can be proved to exist between the swelling of muscle, for example, and fibrin. The same conditions which make fibrin either take up or lose water, make frog muscle do the same.⁶

2. It must now be shown that in the affinity of colloids for water we have a force of sufficient magnitude to account without strain for the maximum amount of water ever found to be absorbed by tissues in a state of edema. This can be done very easily. The severest edemas found in frogs no more than double the weight of the normal leg. Some observations made on patients indicate that the severest states of human edema give values which lie far below this.

We will now, for the sake of argument, neglect the inorganic constituents of the tissues, and say that three-fourths of the body represents water, and only one-fourth various organic substances. These organic substances belong almost all of them in the group of the colloids. On the basis of the figures which I have just given, 1 gram of dry body substance would therefore equal 4 grams of moist body substance, and this in a state of edema may absorb enough water to double in weight, that is weigh 8 grams. In terms of the dry weight of the body, the colloids are therefore required to absorb in a state of edema, seven times their weight of water. It will be sufficient evidence to show how easily colloids accomplish such an absorption of water when I use as an illustration the amount of water that fibrin will absorb under appropriate circumstances. In a weak solution of acid or alkali, dry fibrin may absorb from 20 to 40 times its weight of water.

3. Lastly, it must be proved that in conditions leading to edema substances are formed which are capable of increasing what we may call the normal affinity of the tissue colloids for water. Various substances are capable of markedly influencing the amount of water held by a hydrophilic colloid.

With the exception of alkalies, we find that acids, including carbon dioxide, are the most powerful substances thus far known for increasing the affinity of colloids for water, and it is in either the retention or the abnormal

3. A circulatory system and the pressure of circulating liquids have therefore, nothing to do with the formation of blisters. The necessary elements for the formation of such pathological structures reside entirely in the tissues themselves.

4. The tissues of the leg undergo autolysis. In consequence colloids having a great affinity for water are changed into such as have a lesser affinity, or they may be reduced even to crystalloids.

5. Fischer, M. H., and Moore, Gertrude: *Am. Jour. Physiol.*, 1907, xx, 330

production of acids in the tissues as rendered possible in conditions leading to an edema, that I see the chief cause for its development. To illustrate how powerfully a little acid modifies the amount of water absorbed by a colloid, we need only recall the fact that while fibrin will scarcely swell sufficiently in distilled water to double its weight, it will hold twenty times its weight of water if a little acid is added.

The production of acids in conditions leading to edema has been shown repeatedly. In the described experiments in which one leg of a toad or a frog is ligated, the acid reaction of the tissues may be sufficiently pronounced to evidence itself with the use of any of the common indicators. In some analyses of various edema fluids made in Prof. Friedrich Müller's clinic in Munich I found that these always showed an acid reaction toward phenolphthalein, even after the carbon dioxid contained in them had very largely been allowed to escape. We know, moreover, from Hoppe-Seyler's analyses that edema fluids contain various acids—lactic, valerianic, succinic, butyric—from Strassburg and Ewald's accounts that their carbon dioxid tension is far above that of ordinary venous blood and from Araki's studies that in conditions associated with a lack of oxygen—just such a state as is produced through circulatory disturbances—organic acids are produced in excessive amounts. Ranke's observation that a muscle after exercise absorbs more water than a resting one, coupled with the fact that acids are produced during muscular activity, also finds a ready explanation through changes brought about in the state of the colloids and lends support to the ideas advanced here.

CONCLUDING REMARKS.

It may not be amiss, in conclusion, to point out a number of facts which in a sense are corollaries of what has been said. Attention has already been called to the fact that dead bodies kept in water become edematous. This is due to the fact that after death the tissues become acid, and absorb water from their surrounding medium. For the same reason gangrenous tissues swell if a source of water is furnished them either from without or through the blood and lymph vessels. In gangrene due to occlusion of a vein it is well-known that the tissues are ordinarily moist and swollen, while a gangrene due to occlusion of an artery is ordinarily dry.

The conception of edema offered here holds for local as well as generalized edemas. In fact, one form of local edema furnishes what amounts to experimental proof of the ideas advanced. Reference is made to the rapidly developing edema which follows the stings or bites of various animals capable of carrying formic or other acids into the wound produced by them. Formic acid is exceedingly active in increasing the affinity of various colloids for water. This explains why its introduction into the tissues—as through a flea bite—is followed by such a rapidly developing local edema. That circulatory disturbances are not primarily responsible in this case is indicated very clearly by the fact that the edematous tissues are initially white and only after resolution has commenced does an increased flow of blood occur through them. "Flea bites" can, in fact, be perfectly mimicked with a needle, a little formic acid and a hydrophilic colloid. If a gelatin plate is pricked with a needle dipped into formic acid, and a little water poured over the gelatin plate, wheals soon develop on the surface of the gelatin, which, in shape and in the rate

of development, are identical with those which follow the bite of a flea or the prick of a formic-acid-laden needle in the skin of a human being.

As another form of local edema which finds a ready explanation on the basis of the ideas advanced here, we may mention glaucoma, and the imbibition of water by the crystalline lens. Even if we do not accept the view that no circulation whatsoever occurs through the vitreous and aqueous humors, we must at least admit that a well defined circulation as maintained through blood and lymph vessels is difficult to demonstrate experimentally. Workers on the eye have therefore been unable to find the explanation of glaucoma in any disturbances in the pressure, *per se*, of circulating fluids; and no attempt has ever been made to account for the absorption of water by the crystalline lens, for here the absence of a circulation is only too apparent. The facts observed are, however, easily explained when it is remembered that the humors of the eye and the crystalline lens represent typical colloidal bodies and that not alone the production of the slightest amount of acid or an accumulation of carbon dioxid, even a mere decrease in the absolute or a slight shifting of the relative concentrations of the various salts found in these substances is followed by so great an absorption of water that the severest grades of clinical glaucoma are easily accounted for.

I wish, finally, to say a word regarding the active or passive migration (diapedesis) of the formed elements of the circulating fluids into the tissues. My reason for bringing up this point at this time is because in the later stages of edema the red blood corpuscles frequently pass over into the tissues, and I would like to protest against the present generally accepted belief that the red blood corpuscles, for instance, pass out of the blood vessels through so-called stomata. Such an idea, is, on its very surface, gross, if not absurd, for how can such holes exist in the blood vessel walls? The histologic evidence of their existence is entirely inconclusive. I would like to call attention to a property of colloids which allows a ready explanation, not only of the passive passage of such formed elements as the red blood corpuscles through the walls of the blood vessels, but also the active movements of the ameboid cells. It is perfectly possible for one colloid to pass through another without losing its identity or leaving any mark behind indicative of its passage. The passage of a liquid body through a colloid can be very nicely illustrated by the movement of a mercury drop through a solidified gelatine of the proper concentration. Under the influence of gravity a mercury drop will move anywhere through a gelatin mass, in which, of course, there are no stomata, and leave no evidence of its passage behind. Similarly red blood corpuscles can, under the influence of a pressure not exceeding that in the blood vessels, be blown into a stiffened gelatin. Here one colloidal body moves through another. Whether one such body will pass through the other is simply a matter of relative surface tensions. Under ordinary circumstances this relation is not such in the body as to allow the red or white blood corpuscles to pass into the tissues (we might say be swallowed), but in lack of oxygen, under the influence of chemical agents (chemotaxis), etc., the proper ratio may be produced.

For the same reason that red and white corpuscles are capable of passing into and through the tissues, bacteria may be taken up by either fixed or motile cells.

Pathologists have often wondered how a bacterium may enter the tissues at one point, and leave behind no evidence of entrance. This is because one colloid body may pass through another: from the alimentary tract, for instance, into the walls of the intestine and through this into one of the circulatory systems, and again out of these through the substance of the kidney into the urine.

In this way an infectious agent may wander through any of the tissues of the body, provided the proper surface tension relationships are present, without leaving behind any mark indicative of its passage. Through discussion of the opsonins special interest has recently been attached to the methods by which the surface tension relationships of bacteria to fixed or motile cells may be altered under physiologic and pathologic conditions. An opsonin is nothing but a substance to which has been given an evanescent name, which is capable of so altering the relationship of surface tension of bacterium to cell as to allow the former to be taken up. Many conditions are capable of markedly changing surface tension relationships, and there can be no doubt that we shall on some day not far distant become acquainted with innumerable simple chemical bodies which will, when added to mixtures of leucocytes and bacteria act as our now mysterious "opsonins." We must, however, carefully distinguish between the mere inclusion of an infectious agent and its destruction. Many workers on the opsonins take the swallowing of a bacterium by a cell and its destruction to be synonymous. This is a grave mistake and can not be too vigorously guarded against. Inclusion and destruction are two separate processes, and their mechanism must be separately considered.

DISCUSSION.

DR. A. J. CARLSON, Chicago: I am not familiar with the pathologic side of edema as such. Of course, when one ligates a frog's leg, as Dr. Fischer has done, one produces very abnormal conditions. As far as I know, in the case of local edema at least, there is no evidence of the formation of this specific substance. I think, however, that there is something in this suggestion, but it is only one of the factors in the tissue cell. I do not know that we can throw away the conception of such terms as secretion, inasmuch as terms like that stand for a mass of material the nature of which we do not understand. Nobody, of course, pretends to say that such terms are positive. I think that perhaps two more factors must be considered in edema, namely, the lymphatics and also the blood vessels.

DR. A. D. HIRSCHFELDER, Baltimore: The conditions of edema in which the limb is ligated and both circulation and lymph flow are cut off are to be differentiated sharply from those in which there is some residual circulation. In the former case, as shown by Loeb ten years ago, differences of osmotic pressure are possible, and probably actually occur. I am under the impression that Loeb did demonstrate some difference between the osmotic pressure of the tissues of the frog's legs above the ligatures as compared with the parts below them. In the latter case the circulation is always rapid enough to allow osmotic interchanges to go on, and marked differences in osmotic pressure do not take place. I therefore think that this is the difference between the two forms of experimental dropsy. The injection of formic acid and other substances certainly brings about injury of the endothelial walls of the capillaries quite as much as it does the very interesting phenomenon of absorption by colloids.

DR. W. H. WELCH, Baltimore: Are the phenomena Dr. Fischer finds applicable to all forms of edema? It can hardly be that a single explanation can apply to all varieties of edema, the mechanical as well as the cachectic. We shall

probably not have a clear understanding of edema, of the events, for example, which intervene between the occurrence of passive hyperemia and the accumulation of fluid in the tissues, before the physiologists have settled many disputed matters connected with the production and absorption of lymph. There are certain striking peculiarities of pulmonary edema which must be taken into account on any hypothesis framed to explain this variety. As is well known, general pulmonary edema can appear very suddenly and can disappear with equal rapidity. Can the factors which Dr. Fischer believes to be operative explain these features of pulmonary edema?

DR. H. G. WELLS, Chicago: One thing which Professor Welch just said has always troubled me in applying most of the questions in relation to edema forms; that is the formation of pulmonary edema, particularly the alveolar form in which edema occurs where there are practically no cells and where there is nothing to cause fluid to run. Why should there be an accumulation of fluid in the alveolar space where there are no cells to attract the fluids? To provoke the alveolar fluid? But there is too little of it to account for the rapid production of edema.

DR. W. B. CANNON, Boston: Dr. Fischer stated that gelatin was capable of taking up fifteen to twenty times its weight of water. As I understand it, that is the dry weight. I should like to know whether the initial amount of water normally associated with the colloids would still allow enough more to be taken to account for the results we see.

DR. MARTIN H. FISCHER, Livermore, Cal.: It seems to me that when marked edema can be produced, as it is certainly produced in these frogs, it is going out of the way to hunt trouble to insist that the lymphatic circulation must play a rôle. The burden of proof rests on this side of the argument. I am much more inclined to believe that the lymphatic circulation is the consequence of tissue activity than that it causes tissue activity. There is no circulation in these ligated frog's legs and the edema has to be explained without a circulation.

So far as the physiologic functions of the endothelium are concerned we may, of course, if we wish, use the word "secretion," but the word is not an explanation. I believe that we are going to find the explanation of the secretion of fluid into the alveoli of the lungs and into tissue spaces generally in changes in the colloids of the surrounding tissues. These after having become edematous seem to suffer a secondary change, an autolysis perhaps, through which their affinity for water is decreased, and they drop a part of their water, as it were "secrete" it into the alveoli or tissue spaces. I do not care to discuss this point further because I am experimenting on it.

To illustrate my remarks to-day, I spoke only of such colloids as can absorb water and maintain their shape. This was done to prove that colloids have an intense affinity for water independently of any osmotic conceptions. As a matter of fact the lymph and different body fluids often have enormous amounts of colloids suspended in them, and these particles in such pseudosolution also have an immense affinity for water, which I have not at all considered in my paper.

I consider it quite immaterial whether an edema is produced slowly or rapidly—whether the circulation is shut off in a moment by tying a string around the leg, or the leg is deprived of oxygen more slowly, the condition affecting only the rate of development of the edema and having nothing to do with its essential cause.

The question whether the ideas advanced are applicable to all forms of edema is one that must be tested and answered by the future. My answer now is "yes." There are a large number of edemas. The simplest forms develop in consequence of heart lesions, thrombosis, and things of that sort. When edemas similar in nature to these are produced in frogs the acid formed is so high that it shows itself readily when litmus paper is touched to the muscles. The edemas of nephritis, for instance, I explain by saying that poisonous substances are produced or retained in this condition which act on the tissues of the body and so alter their metabolism that substances capable of increasing the affinity of the colloids for water, such as acids, are produced. These acids would

then influence the colloids of the tissues. Various irritant oils when painted on the skin will produce an inflammatory edema, but these oils when applied directly to such colloids as gelatin or egg albumin, do not increase the affinity of these substances for water. We know, however, that there are a number of poisons which produce changes in the tissue that are entirely analogous to those produced through direct lack of oxygen. Potassium cyanid when present in a tissue produces all the changes characteristic of lack of oxygen—the production of acids and the swelling and bursting of the cells affected. But the details of how edema is produced by such indirect means has to be worked out.

All my remarks had to be more or less dogmatic. I do not believe that my ideas must necessarily account for everything in the pathology of edema. Just now it looks to me as though changes in the colloids would explain 97 per cent. of it. Osmotic ideas seem to account for less than 3 per cent. Some day probably with new conceptions in physical chemistry, my own 97 per cent. will be reduced to 40, 30 or 20 per cent.

A question raised was that of the rate of development of edema. I consider it one of the best arguments in favor of the views advanced that colloids are capable of changing their affinity for water in the short periods of time sometimes allowed tissues to develop an edema. In five minutes fibrin will readily take up or give off an immense amount of water. Were I to add a little calcium chlorid to a tube filled with swollen fibrin, it would make the fibrin shrink to the bottom in a few minutes, the change is so rapid. The reverse experiment can also be made. This very rapidity I consider one of the best arguments against the osmotic conception of edema. As a matter of fact, the most rapidly developing edemas observed clinically take more than minutes. One of the substances which is most markedly capable of influencing the affinity of fibrin for water is calcium. Calcium is about the only drug that ever does any good in many of the so-called urticarial diseases. Since urticarial eruptions are essentially local edemas, is it not probable that through administering calcium salts by mouth, we increase the calcium content of the tissues and thereby lessen the tendency of the tissues toward a development of these local edemas?

THE CARE OF PATIENTS AFTER ABDOMINAL SECTION;

WITH ESPECIAL REFERENCE TO THE PERIOD OF TIME THEY SHOULD BE KEPT RECUMBENT.*

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BUFFALO.

Having heard so much in discussion and having read so much in medical journals about allowing patients to get out of bed after abdominal operations in from five to ten days, it has occurred to me to ask why it is needful to do these things; what are the results from doing these things; and, wherein is the new idea an improvement on the old.

I am just old-fashioned enough to believe that it is harmful, in a vast majority of the cases generally subjected to severe abdominal operations, to get patients up in the short periods of time which are being advocated by so many at present; and I still adhere to my plan of keeping most patients recumbent for two or three weeks. With the exception of appendectomies, not acute, I would say that no patient having an abdominal section should sit up before sixteen or eighteen days, and for the following several reasons:

1. From two to three weeks are required to secure more perfect and more solid union of the incision, thus

preventing postoperative hernia. Early in my operative experience I used only the through and through silk-worm gut suture, then in vogue, in closing abdominal wounds. These sutures were removed in from seven to ten days, and the patients generally sat up in two weeks. Either I or someone else had the felicity of closing postoperative ventral hernias for about one in every three of the patients who were thus treated, and I assure you I had no small proportion of them on my hands, with all their discontentments, complaints and scoldings. No doubt all of you, as I have myself, have been obliged at various times to re-open an abdomen in a week or ten days after the first operation, and often the catgut or other suture material is cut, the incision may be opened down to the peritoneum by the fingers or handle of a scalpel with the greatest ease; although the parts are adherent, the line of union is so tender that separation by pressure is easily done. In another week or ten days you will find the effort to separate the line a very different proposition; the line of union is strong and resists separation.

During the past twelve to fourteen years I have used the tier suture and kept my patients recumbent for eighteen days, and I have not seen a postoperative hernia in 1 per cent. of my cases. I admit that buried animal sutures play a part in the success, but I believe that the same care in suturing and allowing patients to be about in a week will be followed by frequent hernias.

2. The majority of subjects of abdominal section are reduced physically and need rest, in order to build up bodily vigor. Especially if nervous exhaustion is present the principles of the rest treatment should be carried out and the patient placed in a better condition to proceed to normal recovery.

We are liable, in surgical work, to lose sight of the patient and his general bodily condition and center our attention on the surgical lesion for which we operate. Does the removal of the surgical lesion which has made a patient ill, or which has invalidated him, restore his health? No; no more than putting out a fire restores the building which it has demolished. It is simply the removal of a cause of invalidism, but it does not at once tend to set in motion the normal performance of all the functions of assimilation and upbuilding of blood, muscle and nerve which the patient needs to be well.

A sick person undergoes operation for the purpose of getting well, not to be sent home in a week or ten days and remain sick. I know personally of many such who are still sick and ailing, simply because their body was never given the chance to build up. Patients expect to be well as soon as the operation is finished. They always expect that. The surgeon allows them to return to their homes and their usual round of duties—the result being that they use their strength as fast or faster than they can make it, and remain semi-invalids. I am speaking now of patients who are really sick and debilitated when operated on. A person in full vigor, with an operation for strangulated hernia, or some similar acute condition is a different proposition. Such patients will, if they avoid the danger of hernia, recuperate in spite of getting about so soon.

The shock to the digestive apparatus, in the debilitated is often severe and requires from a week to ten days before the normal functions of these organs are re-established. In the meantime decrease in quantity of ingested food and consequent lack of nourishment has

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