matory reaction in the fibrous thickening and contraction of the mesentery.

The second condition is that of tuberculous abscess in the mesentery of the small bowel. I have had two of these in children, one of which was the size of a large orange, situated in the midline above the symphysis pubis. This was at first considered to be a distended bladder. But catheterization, however, showed a small amount of clear urine and did not in any way influence the size or position of the mass. It was found that when the patient assumed the knee-chest position and the abdominal wall became relaxed, the mass was displaced upward and could easily be moved from side to side. For this reason I thought of a mesenteric cyst.

In the second case the mass was to the left of and on a level with the umbilicus. This, however, was not freely movable. Operation revealed a large tuberculous abscess in the leaves of the mesentery in each case. The first of these was removed en masse, the second incised and drained. Each patient made an excellent recovery.

These differ from encysted tuberculous peritoneal effusions in that the latter are more fixed and even if they appear to be round in shape are usually connected to the anterior abdominal wall. These encysted effusions may occupy the midline of the abdomen.

Leo Eloesser, M. D. (Butler Building, San Francisco)— Like Doctor Cowan, it has not been my fortune to operate upon a patient for chylous cyst, but I have seen other cysts, difficult to distinguish from them.

Pancreatic cysts are usually less movable, but in 1920 I saw, with Dr. H. P. Hill, an old lady who had a movable tumor of the epigastrium, some 8 cm. in diameter, which pushed the stomach downward and to the left, and which throbbed with the aortic pulse, but was not itself expansile. We thought the cyst most likely to be of the pancreas. The next day it ruptured, so that I hurriedly opened the abdomen and found a moderately tense red cyst, covered by dilated veins, lying between the liver and stomach. The hand could be introduced between the cyst and the liver. There was apparently no connection between them. I marsupialized the cyst, and the patient made an uneventful recovery. I thought I was dealing with a pancreatic cyst, but sections of the wall contained liver cells and the pancreatic ferments were absent in the fluid. The growth was a cystadenoma of the liver.

Encapsulated intra-abdominal abscesses and large movable abdominal tumors from suppurating glands of the mesentery are not so very rare. One such tumor in a girl who was afterwards sent to the San Francisco Hospital was twice the size of the one in the above-mentioned patient. X-ray films of the abdomen taken before the administration of a barium meal, will usually reveal shadows of chalky or cheesey deposits, which permit of a diagnosis even without opening the abdomen. Large ovarian cysts may also present diagnostic difficulties. Echinococcosis wall gives a characteristic complement fixation reaction, but echinococcus infection is as rare in this country as the chylous cysts themselves.

Doctors Levison and Wolfson (closing)—We thank Doctors Cowan and Eloesser for their discussion of this paper and read with interest their cases cited. We will close the discussion with Donoghue's remarks: "In the literature on cysts of the lesser peritoneum one finds few reported, apart from those credited to the pancreas; it is often so difficult or even impossible, to recognize during operation, the precise origin of any individual cyst; there are so many possible sources from which cysts may develop that one is forced to believe that operators and writers have too often assumed their pancreatic origin without sufficient diagnostic data."

A physician should always be willing to call a consultant. This attitude is looked upon with favor by the family. In all severe cases one should have a consultant, not only to cover the patient's illness, but to guard against any legal difficulty that may follow, for example, an attempt to break a will, a claim of unsound mind. In these days of dishonesty, one must be covered at every angle.—Medical Review of Reviews.

EDEMA FOLLOWING THE USE OF INSULIN

By D. M. Ervin *

The edema of insulin is nothing more than colloids under the influence of two different types of chemicals— one the electrolyte affecting the dispersion, the other the non-electrolyte, only preventing the water from being "pulled in" to the colloid by the dispersion caused by the other.

Discussion by Paul G. Woolley, Los Angeles; T. Henshaw Kelly, San Francisco.

WITH the use of insulin edema has begun to appear in the diabetics, a fertile field for the salt retention followers who have lost no time in putting forth their favorite theory. To the colloid chemist, however, who is able to imitate in a simple manner the entire affair without the use of salts, the salt retention theory is both unnecessary and inadequate.

The clinical observation that the diabetic does not have edema but does die the same cerebral and respiratory death (save convulsions) as the nephritic, is quite common but has received scant attention.

While the diabetic and nephritic comas are fundamentally identical, it is not within the scope of this paper to discuss the applicability of the Gibbs-Donnan law to the physical chemistry of coma. It is intended to discuss only the edema which appears under the use of insulin in the diabetic as a simple and natural conduct of hydrophilic colloids under the influence of two different types of chemicals, electrolytes and non-electrolytes, which both affect the water contents of the colloid.

When colloids of the hydrophilic type are placed in acids swelling takes place. This is because the colloids become under the influence of the acids more dispersed; that is, the colloid particles become smaller. The more the particles become dispersed, the more their internal force permits the water to be drawn in, and as the water is drawn in swelling takes place. It is not due to the swelling that the dispersion takes place; but it is due to the dispersion that water is permitted to be drawn in. Acids produce swelling by increasing the dispersion. Salts decrease swelling by decreasing the dispersion.

The non-electrolyte, while decreasing the swelling, does not do so by affecting the dispersion of the colloid. It is an interface or membrane equilibrium.

The power to hold water by a colloid is decreased by the presence of the sugars as by the presence of the salts, but their action is entirely different. This may be experimentally evidenced by the effect of the salts and the sugars upon the liquefaction point of gelatine under the influence of acids. The liquefaction of gelatine under the influence of acids is a dispersion of such high degree that the colloid particles lose their internal tension and become liquid. Upon this liquefaction point may be tested the effect

of salts and of glucose testing their power to change the dispersion.

Let us set up a series of tubes of gelatine of 1.5 per cent, ranging through increasing concentrations of acids as:

N	N	N	N	N	N	N	N	N	N
200	180	160	140	130	120	110	100	90	80

The tubes that are underlined are those that remain liquid; the others became solid upon standing. If salts affect the dispersion, tending to decrease it against the effects of the acid, the point of liquefaction will be moved towards the right by the addition of sodium chloride. A higher concentration of acid will be necessary to produce liquefaction in the presence of the salt than without.

M NaCl	N	N	N	N	N	N	N	N	N	N
100	200	180	160	140	130	120	110	100	90	80

M NaCl	N	N	N	N	N	N	N	N	N	N
50	200	180	160	140	130	120	110	100	90	80

Under the influence of the sodium chloride it was necessary to have $\frac{N}{80}$H Cl to bring about liquefaction. Now in a similar manner we may test the action of glucose.

Glucose

1%	N	N	N	N	N	N	N	N	N	N
	200	180	160	140	130	120	110	100	90	80

2%	N	N	N	N	N	N	N	N	N	N
	200	180	160	140	130	120	110	100	90	80

4%	N	N	N	N	N	N	N	N	N	N
	200	180	160	140	130	120	110	100	90	80

5%	N	N	N	N	N	N	N	N	N	N
	200	180	160	140	130	120	110	100	90	80

Through the entire series $\frac{N}{120}$HCl is as effective in the liquefaction of gelatine in the presence of 1, 2, 4, 5 per cent glucose as it is in the absence of glucose. Glucose does not affect the dispersion of the colloid.

On the other hand, let an interface be established by having the gelatine in the form of a cube and immersed in $\frac{N}{120}$ HCl and $\frac{N}{120}$ HCl plus 5 per cent glucose. The cube in the acid and glucose does not swell nearly so much as in the acid alone. Glucose does not affect the dispersion of the colloid, but does affect the swelling when an interface is present between the colloid and the glucose solution.

In the nephritic there is present more than the normal amount of acids; in the diabetic there is present more than the normal amount of acid and glucose.

As the acids increase in the nephritic, water is "pulled in" by the increased dispersion. If a membrane is present, as the eyeball, tenseness develops. As the acids increase in the diabetic, water is pre-vented from being "pulled in" by the increase of glucose and with the increase of dispersion with no increase of the water content, the system tends towards liquefaction or softening, as in the eyeball of the diabetic coma.

As it is only necessary to remove the glucose which does not affect the dispersion from the diabetic to convert the diabetic tissue into the nephritic tissue, to convert the soft flabby tissue into the tense swollen tissue, it is easily seen why edema is appearing under the use of insulin.

The eyes of the diabetic coma and the nephritic coma can readily be duplicated with gelatine inclosed in diffusion membranes and immersed in acid and acid and glucose.

With the use of insulin the dispersed unswollen colloids of the diabetic are being converted into the dispersed swollen colloids of the nephritic by the rapid lowering of the glucose content of the body. The glucose, by the use of the insulin, is being removed more rapidly than the acids, leaving behind dispersed colloid that may now swell.

It must be noted that the swelling of the colloid is a result in the conduct of colloids, the serious factor being the state of the dispersion upon which state of internal energy the colloid depends for its ability to do work and upon which the body becomes a workable machine. Whether in the nephritic or the diabetic the conception of importance is the state of dispersion. We are notified of it in the nephritic by the swelling, but not in the diabetic; yet it is just as surely and seriously there.

The edema of insulin is nothing more than colloids under the influence of two different types of chemicals—one the electrolyte affecting the dispersion, the other the non-electrolyte, only preventing the water from being "pulled in" to the colloid by the dispersion caused by the other.

DISCUSSION

Paul G. Woolley, M. D. (Pacific Mutual Building, Los Angeles)—I hesitate to go into detail in a discussion of this article for the reason that I am too poor a colloid chemist to do anything but make a mess of the thing. However that may be, I might say this much, that the fact that edema occurs after the administration of insulin has been observed of course, but so far as I know, no systematic experimental explanation of the phenomenon has been put forth until this one of Ervin's. There are many workers who will probably disagree with Ervin's interpretation and among them will be those who disagree with Martin Fischer's formulation of the cause and treatment of the so-called, and mostly miscalled, nephritides.

Ervin's experiments point to the fact that edemas of diabetes are pure edemas due to the withdrawal—to the destruction—of a substance (glucose) which protects the tissue proteins from taking up water in the presence of abnormal amounts of acids. If this interpretation is true, then it should be possible to apply it therapeutically and upon the appearance of an edema following insulin, to dissipate it by the careful use of alkalies and salt solutions.

T. Henshaw Kelly, M. D. (490 Post Street, San Francisco)—Like Doctor Woolley, I am too poorly versed in the lore of colloid chemistry to attempt a highly refined, technical discussion of Doctor Ervin's paper. However, there are one or two points that I would like to argue about.

Firstly, being a pupil of Fischer in the days of "oedema," the question of "interface" sounds to me somewhat ultra-conservative. It has been more or less the custom of those dealing in the colloidal theories of tissue structure to frown upon cell membranes, endothelia, etc.,

which might partake of the nature of interfaces, and to ascribe the changes in water content of tissue largely to internal factors or dispersion states. I am quite interested, therefore, to hear Ervin referring to interface phenomena when the glucose content of the tissue which is going to swell is already high.

Secondly, in discussing this question with Dr. Dwight Shepardson of San Francisco, he made the statement that edema had not occurred in his series of cases except when the blood sugar had been reduced to 50 or 60 mgs. or below, this marked reduction being accompanied by evidence of myocardial weakness. The myocardial weakness and the edema disappeared when the blood sugar was raised to 100 mgs.

These two points do not add any great weight to the discussion, but they interest me and may serve to draw further interesting explanations from Doctor Ervin in closing.

DOCTOR ERVIN (closing)—In answer to Dr. T. H. Kelly, I would say that I by no means limit the idea of an interface to that of a membrane. Nothing was further from my mind than to suggest that a cell should have a membrane. The membrane of the physiologists is too limited and special a case to be held seriously in biology. It is only an artificial method of effecting the coefficient of distribution of a solvent between two systems. Nor is a membrane of such a type necessary to produce Donnan-Gibbs equilibrium, a surface upon either side of which a solvent has not the same degree of solubility.

By interface I meant a surface of discontinuity across which at equilibrium the components of either system have the thermodynamic potentials equal but not necessarily the concentrations. Such a surface a gelatine cube would present when in contact with water, air, etc.

The question of cardiac inefficiency when the blood sugar is below 80 mg. does not in itself affect the nature of the physico-chemical system. I have a patient who will develop edema when the urinary sugar is below 2 per cent. This is clearly a cardiac condition and yet not an underfed heart. The dispersion of the colloid is there when the urinary glucose is 5 per cent, but not the swelling usually associated with dispersion. With proper care of the heart the edema disappeared with the urine negative for glucose.

Another instance of this point I had occasion to observe in a renal diabetic. This patient had been placed upon 75 units of insulin per day in an effort to free the urine of glucose. The blood sugar during the time I had occasion to observe her ranged between 75 and 85 mg. At no time was there present any edema. It is one of the physiological facts of note that the heart muscle is capable of continuing its work upon small quantities of glucose and oxygen.

Again the softening of the eyeballs cannot be explained upon cardiac failure. It does not occur in such. More and more diabetes takes a place as a disease of structure rather than of oxidation.

In answer to Doctor Woolley I might say that the reversal of a colloid in hydration capacity and dispersion is not so simple as it first appears. The linkage of the amino acids may be indicated by a complicated chemical formula which we will omit.

When an acid stronger than the acid group of the amino acid is added, there takes place a distribution of the amino group between the two acids.

After this, though we may add an alkali, we are only adding one more component to enter into the distribution and we only in part reverse the first change.

Not only do we fail to reverse the dispersion to its identical former self, but we fail likewise to restore the emulsion broken by the dispersion.

About the logomachy of the pathologists, I have no concern. It would seem, however, that the introduction of physico-chemists' ideas, that are concise and well defined, into pathology would go a long way toward concise and well defined ideas of pathology and more frequent use of the expression, "we do not know."

If you look at a bachelor, you become a little more reconciled to your husband.—Lady Astor.

LABORATORY AIDS IN THE DIAGNOSIS AND CONTROL OF ECLAMPSIA

By HENRY A. STEPHENSON *

The Editorial Councilor—himself an eminent obstetrician—who evaluated Stephenson's contribution for the editor, says: "I have read this paper very carefully and find it most excellent, a paper which any obstetrical journal would be glad to get. It will make a very creditable article for CALIFORNIA AND WESTERN MEDICINE."— Editor.

The more elaborate tests used for the determination of kidney and liver function have no great value in eclampsia.

Blood pressure readings and routine urinalysis, including a study of the urinary sediment, give us the most valuable information in the diagnosis of this group of toxemias.

DISCUSSION *by M. H. Ross, Los Angeles; T. Addis, San Francisco; J. M. Slemons, Los Angeles; Frank W. Lynch, San Francisco.*

DURING or immediately following the termination of pregnancy, there occurs in a small percentage of patients a chain of symptoms consisting of headache, edema of the extremities, visual disturbance and epigastric pain. Albumin and casts are seen in the urine. The blood pressure is usually elevated. This condition has been called pre-eclamptic toxemia. The addition of convulsions followed by coma is termed eclampsia.

There is some question as to whether the pre-eclamptic toxemia is the forerunner of eclampsia or whether it is a distinct entity. According to Harris, the after effects of the pre-eclamptic condition are more pronounced and last for a longer period than do those of the eclamptic condition. Harris questions whether eclampsia would always follow, were the patients with pre-eclamptic toxemia allowed to proceed with the pregnancy. Few of us have the courage to allow pre-eclampsia to continue.

Other observers go further and attempt to classify toxemia with the above symptoms as "hepatic" or "nephritic," according as the symptoms and findings point to primary liver or kidney disturbance. This differentiation seems difficult in the light of our present knowledge as we shall try to show later, and, from the standpoint of immediate treatment, is unnecessary.

The incidence of the disease among obstetrical patients is about 2 per cent. Some investigators have reported an incidence as high as 3 per cent, but certainly in this country this figure seems too high. However, Stroganoff states that 24,000 mothers and children die each year in Europe and the United States as a result of the condition. This is indeed a very large number and justifies renewed efforts to prevent or control the disease. It is with this thought in mind that I have reviewed the recent literature on strictly laboratory methods in the diagnosis and control of eclampsia.

The details of these laboratory procedures are

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