DR. ALFRED GORDON, Philadelphia: Syphilitic individuals, in spite of the syphilis, and not on account of it, may present symptoms resembling ordinary psychoses. They may present a picture of manic depressive psychosis, dementia praecox and others. The syphilitic patient may have all sorts of diseases, and when we do investigate for syphilis and the tests are positive we can improve the patient's condition. One often finds, on close observation, modifications of classical symptoms, and this is due to some modifying factor such as syphilis. I have no illusions as to the cure of syphilis of the central nervous system. I am not optimistic to that degree, but I am far from convinced that the last word has been spoken. I think we can often improve the condition. While my paper is largely technical, there is a practical side, namely, the findings of modifications in the typical clinical pictures and thereby a possibility for improvement by antisyphilitic remedies.

INTRAVENOUS GLUCOSE INJECTIONS IN SHOCK*

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In accordance with a prevailing opinion, the low blood pressure in "shock" is associated with a diminished effective volume of blood in the vessels, and the two factors constitute in themselves essential parts of the shock mechanism. On this assumption, benefits should result from procedures having the effect of holding water within the vessels. In normal individuals it is possible to accomplish this by effecting an entrance of glucose into the blood at appropriate rates sustained for sufficient lengths of time. Marked hydremic plethora and striking increases of the systolic blood pressure may thus be produced, and the present study has shown that the mean arterial pressure may also be elevated in this way. Glucose is, moreover, a food; it is capable of increasing the power of involuntary muscle contractions, elevating the total metabolism, combating at least one type of acidosis, and restoring depleted glycogen reserves. Has it a value in the treatment of cases of shock?

During the past two years, Woodyatt, with Sansum and Wilder, has studied the physiologic effects of sustained intravenous glucose injections at known rates, and applied the method to several clinical conditions, including two cases having features of shock. Favorable results in these cases made it appear desirable to study a longer series under conditions permitting more exact observations. This was not possible with the available clinical material, and it was decided to test the principles in cases of experimental shock in animals. Erlanger and his associates have during the summer performed some sixty experiments on shock in animals with special reference to the circulatory phenomena and the vasomotor mechanism. A large number of records for control purposes and a perfected technic were thus available. The primary purpose of the work here reported was to ascertain how the symptoms of experimental shock in dogs might be influenced by intravenous glucose therapy. Incidental observations were made on the effects of salt solutions, salt and carbonate solutions, and epinephrin. The work was done in eight days, and no attempt is made to give this report the character of a completed study. The possible differences between the conditions induced in dogs and that of shock in human cases is appreciated, as also the fact that the slow intravenous injection of glucose in postoperative conditions is not a new procedure.[1]

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PHYSIOLOGIC PRINCIPLES

When a single dose of glucose is injected into a peripheral vein, there is a temporary increase of the total quantity of sugar in circulation. The injected sugar passes rapidly out of the blood into the tissues. During its stay in the vessels, the blood volume rises and a state of hydremic plethora develops, owing doubtless to the binding of water by the extra glucose in the vessels. This is shown by a fall in the hemoglobin percentage, and a coincident fall in the blood sugar percentage following an initial rise. These facts were demonstrated by Brasol[2] in 1884, and confirmed and amplified by Biedl and Kraus,[3] Starling,[4] Kleiner and Meltzer, and others. Similarly during the period of absorption following the alimentary administration of glucose there is at first a rise but later a return to the normal of the blood sugar percentage, and simultaneously with the latter a fall in the hemoglobin percentage.[5] In shock cases it is frequently impracticable to secure the entrance of glucose into the blood through the alimentary tract, nor is the subcutaneous injection of glucose a harmless clinical procedure. If glucose is injected into the peripheral venous blood of a normal resting (unanesthetized) individual at or below the rate of 0.8 to 0.9 gm. per kilogram of body weight per hour, the injection may be sustained in most cases for hours without causing glycosuria.[6] With injections of 0.9 gm. or more of glucose per kilogram per hour, glycosuria develops, and the rates of sugar excretion then bear definite relationships to the rates of injection. The higher the absolute injection rates, the higher the absolute rates of glycosuria.

During sustained intravenous injections at or below the tolerance rate, glucose passes from the blood into the tissues and there undergoes chemical change (polymerizations, oxidations, etc.) at the same rate as that of injection. There is no material accumulation of glucose itself in the tissues. Injections at subtolerant rates therefore have the effect of increasing the quantity of glucose in the blood without producing any considerable increase of the glucose in the tissues. The result is a disproportionate increase of the power of the blood to hold water, and a state of hydremic plethora develops and presumably persists as long as the injection is sustained. The water held by the blood must come from the tissues or from the injection site or both. If the water injected with the glucose is not sufficient to satisfy the glucose in the blood, some water will flow into the blood from the tissues. When the glucose injection is stopped and the last of the injected glucose passes into the tissues, the water pre-

1. Kausch: Deutsch. med. Wchnschr., 1911, **37**, 8.
2. Brasol: Arch. f. Physiol., 1884, p. 211.
3. Biedl and Kraus: Wien. klin. Wchnschr., 1896, **9**, 55.
4. Starling: Jour. Physiol., 1899, **24**, 317.
5. Compare Gilbert and Baudouin: Comp. rend. Soc. de biol., 1908, **65**, 710. Fisher and Wishart: Jour. Biol. Chem., 1912, **13**, 49.
6. Woodyatt, R. T.; Sansum, W. D., and Wilder, R. M.: Prolonged and Accurately Timed Intravenous Injections of Sugar, THE JOURNAL A. M. A., Dec. 11, 1915, p. 2967. Wilder, R. M., and Sansum, W. D.: d-Glucose Tolerance in Health and Disease, Arch. Int. Med., February, 1917, p. 311. Sansum and Woodyatt: Jour. Biol. Chem., 1917, **30**, 155.

viously held in the blood is to a large extent discharged suddenly into the urine. This whole phenomenon differs essentially from that obtainable with inorganic salt solutions, because such salts when injected also pass into the tissues, but when once there are not destroyed. There is therefore the possibility of salt accumulations in the tissues holding water there, and in part counteracting the effects of the salt in the blood stream.

With sustained intravenous glucose injections at rates higher than 0.8 to 0.9 gm. per kilogram per hour, the glucose utilization fails to keep pace with that of injection and the tendency is toward some accumulation of unchanged glucose. If the kidneys are functioning actively this glucose appears on the urinary side of the renal membrane and tends to collect water in this locality, probably for the same reason that glucose in the blood produces hydremia.[7] This tendency is expressed as polyuria. Now if the diuresis exceeds the rate of water administration, water will flow from the tissues to the blood and thence to the urine, and hydremia will be sustained, as long as the water supply is adequate. But if the kidneys do not function in this way, most of the unchanged fraction of the glucose injected remains in the body and tends to behave like a salt, causing accumulations of water in the tissues and body spaces wherever it may go. In shock cases the secretion of urine is likely to be impaired. Accordingly the most rational application of intravenous glucose injections in shock would appear *a priori* and on theoretical grounds to consist in long sustained uniform injections at subtolerant rates. Just what the tolerance limit will be in any pathologic condition cannot be stated. When the kidney function is impaired the rate of injection necessary to produce glycosuria cannot be used as a criterion. The most appropriate injection rate might be expected to lie within the normal tolerance limit at possibly 0.3 to 0.6 gm. of glucose per kilogram of body weight per hour. For a man weighing 50 kg. this would imply the injection of from 15 to 30 gm. of glucose per hour, corresponding to from 84 to 168 c.c. of 36 per cent. solution hourly, or twice these quantities of 18 per cent. solution. Although moderate accumulations of unchanged glucose in the body might do no harm or perhaps even prove desirable, it has been noted in the experiments on shock in dogs, produced by occluding the inferior cava, that a general hemorrhagic tendency supervenes when the administrations are excessive.

EXPERIMENTAL

The solutions injected consisted of chemically pure glucose in strengths of from 18 and 36 per cent., to which on occasions epinephrin was added, of 0.9 per cent. sodium chlorid, and of an alkaline saline solution, 0.3 per cent. anhydrous sodium carbonate in 1.4 per cent. sodium chlorid solution (Fischer's solution). Whenever possible the physiologic sodium chlorid solution was injected either just before or just after the glucose as a control to the direct volume effects of the latter. The injections were made centrally into the vena saphena at a practically constant rate by means of an apparatus devised by Woodyatt.[8] Continuous records were made of the pressure in the carotid artery by means of a mercury manometer. Dogs were used exclusively. They were anesthetized with ether. In some experiments the rate of secretion of urine was followed, a catheter being passed into the bladder for this purpose.

Only one experiment has been done on a normal animal. The glucose solution (36 per cent.) was injected for a period of thirty minutes at the rate of 0.8 c.c. per minute (1.78 gm. of glucose per kilogram per hour). The injection raised the arterial pressure, but not more than from 5 to 7 mm. of mercury. The pulse amplitude, however, increased quite appreciably, indicating the development of some plethora. The maximum effect seems to have been attained during the first five to ten minutes of the injection, and was maintained for ten or more minutes after completion of the injection. The slightness of the rise of the pressure in comparison with the considerableness of the increase in blood volume, indicated by the change in pulse amplitude, probably is to be attributed in part to a diminution in the viscosity of the blood, due to hydremia, and in part to vasomotor accommodation.

The animal was then bled to the extent of 150 c.c., and after the pressure had become constant, well below the normal level, glucose was again injected at the same rate, but for a period of twenty minutes. During the injection period the pressure rose from 107 to 120 mm. of mercury, and during the succeeding ten minutes it continued to rise, finally reaching 125 mm. of mercury. It then declined to 111 mm. of mercury in the course of the next thirty minutes. The pulse amplitude was increased. Salt solution injected at the same rate raised the pressure only 5 mm. of mercury, without appreciably changing the pulse amplitude.

It is inferred from this experiment that in normal animals the compensatory mechanism tends to hold the arterial pressure close to the normal level during the plethora determined by the glucose solution; but that when, as after hemorrhage, the efforts of a normal compensatory mechanism fail to hold the pressure up, the same hydremia results in a more marked rise of pressure.

The rate of injection employed in this experiment would lead in the unanesthetized dog to a glycosuria of some 0.5 gm. per 10 kg. per hour, and this would cease within fifteen minutes after the discontinuance of the injection. In this anesthetized animal, however, the injection caused a heavy long lasting glycosuria and a coincident hyperdiuresis of such a grade that in the course of the experiment more water was excreted by way of the kidneys than was injected into the circulation.

In six experiments the effect of the injection of glucose in different doses was followed after shock had been produced by temporary partial occlusion of the inferior vena cava.[9] Our previous experience with this method of inducing shock justifies the statement that when, after deocclusion of the cava, the arterial pressure eventually begins consistently to decline, this decline proceeds, almost invariably, without break until the animal dies, though the pressure at which the decline begins may be only 20 mm. of mercury below the normal level. Despite the fact that the arterial pressure rises quite appreciably with deocclusion, we are inclined to believe that the damage that has been done to the tissues during the period of occlusion is irreversible. That is to say, despite whatever may be done, the cells of the body eventually will fail to carry on the functions necessary to the life of the organism.

7. Sansum and Woodyatt (Footnote 6).
8. Woodyatt: Jour. Biol. Chem., 1917, **29**, 355.
9. Janeway and Jackson: Proc. Soc. Exper. Biol. and Med., 1915, **12**, 193.

In such an event, the only result that could be anticipated from any form of treatment would be but a temporary improvement of certain of the functions.

Our tests of the action of glucose were begun in that stage of the experiment in which the pressure begins consistently to fall. The persistence of heavy glycosuria in the normal but anesthetized dog for hours after a twenty minute injection at the rate of 1.8 gm. of glucose per kilogram per hour indicates that such a rate of infection in shocked animals which pass no urine must result in an accumulation of free sugar in the organism. The first intravenous dose at supertolerant rates must therefore give the clearest indications of what the injection of sugar is capable of accomplishing. In the table are collected the results of the first injections with the animals in shock. In evaluating the effect of the injections on the blood pressure, account must be taken of the fact that such changes as occur are built up on a pressure curve whose general tendency is to decline with time. In the construction of this table an attempt has been made to indicate this fact. For example, it is seen in the table that in Experiment 3, in which the greatest rise of pressure was recorded (from 55 to 100 mm. of mercury), the pressure at the start of the injection was practically stationary, presumably as a result of the action of salt solution injected immediately preceding the sugar injection; while Experiment 5, in which the smallest rise was recorded (from 72 to 76), was one in which the arterial pressure was falling quite rapidly at the time the glucose injection was started. The other pressure increases ranged between these extremes. On account of the difficulties just mentioned, the determination of the duration of the sugar effect on the pressure is not a simple matter. In at least two cases the maximum pressure is attained some time after the cessation of injection; and whereas in some of the other cases the maximum pressure is attained during the period of injection, the pressure curves are of such a form as to indicate that the effect is manifesting itself not alone during the whole of the injection period, but for some time subsequently.

The table also shows the effect of the sugar injection on the pulse amplitude. This is invariably increased by the injections, and the persistence of the sugar effect is brought out more clearly by the changes in pulse amplitude than by the changes in pressure. Thus in Experiment 6, whereas the mean pressure begins to fall five minutes after the cessation of injection, and in thirty minutes has returned to its previous level, the pulse amplitude at that time has not nearly declined to its previous value. This greater persistence of the effect on the pulse amplitude than that on the arterial pressure is seen best, however, after the second and third injections.

RESULTS OF THE FIRST INJECTION WITH THE ANIMALS IN SHOCK

Experiment Number	Normal Mean Carotid Pressure	Pressure at Time of Injection*	Injection Period, Min.	Pressure Rise		Pulse Amplitude Increases		Dosage				Action Subsequent to Injection	Remarks
				To Mm. Hg	In Min.	From Mm.	To Mm.	Per Kg. per Hour, Gm.	Percentage Solution	Rate of Injection, C.c. per Min.	Injection Period, Min.		
1	159	51 and falling	73	69	45	1.5	3.0	0.57	18	1.0	73	The pressure is falling during the last 20 minutes of injection	Sugar injected immediately after salt solution, which had kept the pressure constant
2	140	64 and falling	68	96	68	3.5	4.5	3.7	36	1.85	68	Pressure immediately falls slowly, and, after 10 minutes, rapidly	Sugar injected immediately after salt solution, which had failed to maintain the pressure
3	117	55 and stationary	25	100++	45++	4.0	5.5	3.6	36	2.0	25	It would appear as though this animal was coming out of shock; the rise of pressure stopped only when the cava was again occluded	Immediately after 20 minutes salt injection which raised pressure 4 mm.
4	195	61 and falling	25	70	†	5.0	8.0+	2.2	36	1.0	25	.....	A previous salt injection had temporarily raised the arterial pressure 4 mm. Hg
5	142	72 and falling quite rapidly	23	76	14	6.0	6.5	3.0	36	2.0	23	.....	Pressure can be raised only by epinephrin despite the considerable increase in pulse amplitude by subsequent sugar injections
6	110	66 and falling	30	88	35	4.0	7.0+	4.0	36	2.0	30	Pressure falls rapidly after 5 minutes elapse	
8	122	78 and falling rapidly ‡	25	86	45	5.0	8.0+	3.7	36	2.0	25	Pressure falls rapidly	

* The pressure usually is uninfluenced by the sugar injection until from five to ten minutes have elapsed. This is the pressure at the moment the injection begins to cause the arterial pressure to rise.

† This was immediately followed with a stronger solution of sugar for seventeen minutes by which the pressure was raised only to 62 mm. Hg. Then epinephrin was added to the sugar. This raised the arterial pressure to 97 mm. Hg; but when the epinephrin was withdrawn, while the sugar injection was continued, the pressure fell in the course of twenty-nine minutes from 97 to 49 mm. Hg.

‡ Salt solution for twenty-five minutes did not appreciably modify the pressure curve. Ten minutes later, while the pressure was rapidly falling, sugar injection was begun. The pressure continued to fall but at a decreasing rate, and had just begun to rise at the end of the twenty-five minute period of sugar injection. As indicated, it continued to rise for twenty minutes.

The dosage of glucose has ranged between the extremes of 0.57 gm. per kilogram per hour and 4 gm. per kilogram per hour, that is to say, between a so-called subtolerant dose and one five times exceeding the tolerant dose. All injections resulted in a rise of pressure, which invariably was much greater than that caused by the control injection of salt solution, which often, indeed, gave an inappreciable response. Owing, however, to the difficulties mentioned, it would take a much larger series of experiments than we have performed to determine the optimum dose of glucose.

After a beginning has been made in the induction of shock by occlusion of the cava, urine secretion ceases despite the injection of sugar in doses which, in the normal animal, cause a marked diuresis. Urine was not secreted even when the alkaline hypertonic salt solution was injected.

A few tests have been made, but usually quite late in the course of the experiments, of the effect on the arterial pressure of injecting an alkaline saline solution. These tests were made only casually, and no hard and fast conclusions can be drawn from them. So far as we have gone, however, at the rate of injection employed, no obvious effects could be discerned.

We also have occasionally combined epinephrin, in concentrations ranging from 1 : 100,000 up to 1 : 25,000, with the glucose solutions. These injections also have been made late in shock. While they raised the pressure further, and possibly increased the pulse amplitude more, than the injection of simple glucose solutions, their effect was very transient and they seemed to leave the pressure at a lower level than it would have reached in the same time if nothing had been injected.

In only one case have we succeeded by the injection of sugar in turning a falling pressure into a rising pressure tending to attain the normal level. The animal in which this occurred was particularly resistant to the effects of occlusion of the cava. Whereas a two-hour partial occlusion of the cava in our experience ordinarily suffices on deocclusion to leave the arterial pressure well below the normal level, and to start the pressure on a downward course within forty or fifty minutes, in this case the arterial pressure returned to the normal level after deocclusion within twenty-five minutes. After a second period of occlusion lasting forty-five minutes, the arterial pressure was still rising fifty-five minutes after deocclusion. The same was true after a third period of occlusion. It was only after the fourth period of occlusion, which lasted one and three-quarter hours, that the arterial pressure after deocclusion started on a downward course. It was then, after a preliminary test injection of saline which held the pressure practically level, that the sugar was injected. The pressure rose from 55 to 90 mm. of mercury during the injection period of twenty-five minutes, and continued to rise in the after-period, finally reaching 100 mm. of mercury, and it was still rising when, as it was believed that the animal had recovered from shock, the cava was again clamped in order again to reduce the animal to shock.

Previous experience in the production of shock indicates that when it is induced by temporary partial occlusion of the inferior vena cava, vasomotor tone, in the stage in which we have made the first sugar injections, is either below normal or at the lower limit of normal. In order to determine whether or not the difficulty in obtaining lasting improvement as a result of the injection of sugar is attributable to the damaged state of the vasomotor mechanism, in one experiment the effect of sugar injection has been tested in shock produced by temporary partial occlusion of the descending aorta. In shock thus produced, we judge from experience that the vasomotor tone, at the stage with which we are now concerned, is either normal or above normal. The experiment ran quite smoothly; but here again the action of the sugar, while quite distinct, was as ephemeral as in the majority of our experiments.

In this experiment, urine secretion stopped shortly after the aorta had been clamped, and none was formed until about two hours had elapsed after deocclusion, when, apparently under the stimulus of the sugar injection, 1 c.c. was passed. This is the only instance in which urine was formed after the production of shock in this series of experiments. Macroscopically the urine did not contain blood.

One of the most interesting phenomena brought to light by this series of experiments, and seen best after several injections have been made, is a decline of the pressure, subsequent to the injection, to a level well below that obtained previous to the injection, despite a pulse amplitude which is higher under the low pressure after the injection than it was under the higher pressure preceding the injection. The increased pulse amplitude undoubtedly is a measure of the increase in blood volume. Presumably, therefore, the pressure is lower after than before the injection, despite an increase in blood volume. This condition probably is made possible through a diminution in the vasomotor tone and a decrease in the viscosity of the blood, which together defeat the tendency of the plethora to increase the arterial pressure. It suggests that a relatively inactive vasomotor mechanism responds in the same way to its adequate stimuli as does the normal mechanism. The normal center is, so to speak, keyed to hold the arterial pressure at the normal level. The damaged center is keyed to hold the pressure at a level below the normal. And if the activity of the damaged center is constantly diminishing, the level at which it tends to hold the pressure will also constantly diminish. The reactions of the center, therefore, to increased blood volume, no matter what the new pressure normal may be, will always be such as to return the disturbed pressure to that normal. If this is the case, the combination in the late stages of shock of an increased blood volume with a lower pressure would be accounted for.

Attention should be called to the tendency exhibited by overdoses of glucose solutions to diminish the coagulability of the blood. As a result of this action, wounds which have ceased bleeding may become covered with an ooze of blood, sometimes quite considerable in amount, which remains fluid for some time. In none of our experiments was this secondary hemorrhage serious, but it is a danger which should be kept in mind when sugar injections are employed in the treatment of shock in the wounded.

SUMMARY

1. Glucose injected intravenously at rates varying between 0.57 and 4 gm. per kilogram per hour for from twenty to sixty minutes into anesthetized dogs reduced to a state of "shock" (by partial temporary occlusions of the inferior cava or aorta) has been observed uniformly to increase the mean arterial pressure.

2. The injections have uniformly produced a marked increase in the pulse amplitude, indicating a condition of plethora.

3. The increase in pulse amplitude has usually been more striking than the increase in arterial pressure.

4. In one case the increase in pressure determined by the injection of glucose continued after the cessation of the injection until the pressure was approximating the normal.

5. A subtolerant dose has raised the arterial pressure and increased the pulse amplitude as effectively as many of the injections made at more rapid rates.

6. With the more rapid injections, a marked hemorrhagic tendency may develop in animals in this condition.

7. No other palpable deleterious effects were observed.

CONCLUSION

On theoretical and experimental grounds supported by some clinical evidence, it would appear that intravenous injections of glucose at appropriate rates are of distinct benefit in certain phases of shock.

SYMPTOMATOLOGY OF THE NERVOUS SYSTEM

IN CHRONIC INTESTINAL TOXEMIA *

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Present medical progress tends toward diminishing the number of disease entities rather than toward their multiplication. In no field is this more strikingly apparent than in the newly found and rapidly developing relationship between mental and nervous conditions and disturbances of the gastro-intestinal tract.

The symptomatology referring directly to the intestinal tract is not within the scope of this paper. However, it is difficult to find any case in which, on close investigation, symptoms referring directly to the intestinal tract are absent. The error in diagnosis as to the fundamental condition can frequently be explained by the fact that the gastro-intestinal disturbances are so often considered secondary to a disturbed nervous system, when in reality the opposite is true.

As an example of one of the commonest misapplications of diagnosis we mention neurasthenia. It is perfectly true that we may have a neurasthenia brought on by causes not connected directly with the intestinal tract, and in these a functional disturbance of the stomach and intestine may follow. Church and Peterson mention that neurasthenia may be caused by organic conditions such as phthisis, Bright's disease, diabetes, gout, rheumatism, and uremic and toxic states generally. The fact that chronic gastro-intestinal conditions are almost invariably followed by neurasthenia of differing intensities is not mentioned.

For purposes of simplification we have separated the symptomatology of the nervous system in connection with chronic intestinal toxemia into four classes, namely, cases involving (1) the mental system, (2) the sensory system, (3) the motor system, and (4) the sympathetic system. These classes of symptoms may occur separately or in combination, usually the latter, so that we can designate them only according to the predominant symptoms. It would be impossible to say why the toxin resulting from these intestinal conditions should have a selective affinity for any one part of the nervous system, just as it is impossible to ascribe reasons for the localization in other toxic states resulting from tuberculosis, diabetes, gout and syphilis.

It is a significant fact that in practically all of the cases considered in this article the nervous manifestations have either cleared up or have been markedly improved by treatment directed toward the intestinal toxemia. A great many of these patients have been treated by the usual therapy for disorders of the nervous system, without result. This is not in the nature of positive proof, but is very strong evidence as to the etiology of the disturbances of the nervous system in these cases.

In considering the cases exhibiting mental symptoms, we have divided them into two groups, intellectual and psychic. Under the intellectual group we find sluggishness of mentality, dulness and stupidity, loss of concentration, loss of memory and mental incoordination. In the psychic group are included irritability, lack of confidence, excessive and useless worry, exaggerated introspection, hypochondriasis, phobias, depression, melancholic states, obsessions, delusions, hallucinations, suicidal tendencies, delirium and stupor. Of 518 cases studied for this paper, 201 exhibited irritability, 317 depression, 4 obsessions, 1 dual personality, 5 hallucinations, 4 delusions, 12 hysterical coma, 74 deep melancholia, etc.

There is no group of mental symptoms that can be said to be diagnostic of intestinal toxemia. The mental symptomatology is rather complex and may assume any one or more of the symptoms of a primary mental condition, or of syphilis. These must necessarily be excluded. As an example of mental disturbance illustrating both intellectual and psychic symptoms, the following case history is of interest:

REPORT OF CASES

CASE 1.—*History.*—Miss L. E., aged 46, a schoolteacher, had been constipated sixteen years following an attack of nervous prostration. The symptoms, which began at this time and had become progressively worse up to the time when she was first seen, were intense headaches, hemicranial in type; she felt as if she had two brains, one side normal and the other side abnormal. Severe melancholia, with violent outbreaks, which was progressive, kept the patient from teaching. Twelve years ago curettage was done. Eight years ago, after a diagnosis of uterine fibroids, a hysterectomy and appendectomy were performed with no improvement. For five years she took morphin, in increasing dosage, for relief from pain in the head. She always felt uncomfortable in the abdomen, especially when standing. Ability of mental concentration was partially lost. She was much depressed.

Examination.—The roentgen ray revealed a water-trap stomach with one third retention at seven hours. The small intestine was intensely rapid. Meal was in the colon in one and three-fourths hours. Both flexures and transverse colon were very low. The urine was constantly normal except for a large excess of indican. Blood pressure for many years was around 240. The Wassermann reaction was negative. Blood analysis, chemically, showed low urea nitrogen, uric acid and blood sugar. Slightly high freezing point of serum. Subsequent chemical blood analysis showed a similar picture with the exception of a high uric acid retention. Examination of the optic fundus revealed a normal picture. This patient was in a deplorable condition, mentally and physically, and life had become a burden to her. She could work only with frequent long periods of rest.

Treatment.—She was operated on by Drs. Draper and Lynch in August, 1914. The pylorus and duodenum were normal except for some slight thickening. The gallbladder was thickened and contained one stone, the size of a marble. The stone was removed. The transverse colon showed many diverticula, one of which was solidified. This was removed. The sigmoid found bound down in a scar from a previous operation was freed. A partial colectomy (developmental reconstruction) was done. End-lateral anastomosis was effected. A partial Coffey operation was limited to three suspensory sutures to the left. There was no postoperative shock.

After an initial improvement in all symptoms the patient relapsed and came to see Dr. Satterlee four months later. She had returned to work at school against all advice. She was incapacitated again. She was sent to a sanatorium for

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