

by resorting to such a simple procedure, as might have been obtained if a decompression operation had been performed.

DR. ALBERT E. STERNE, Indianapolis: It depends entirely on what we mean by edema of the brain. We should not use that term unless we mean a cellular encephalitic process, a serous encephalitis. We should distinguish between edema and any form of hydrocephalus. Furthermore, in certain conditions, an encephalitic process frequently arises in the brain no matter what the etiology may be, whether it is traumatism, tumor, infection, exhaustion, sunstroke, or due to a drug or syphilis. Under those conditions the only thing the brain cell can do, primarily, is to swell. This is the preliminary stage of an inflammatory process. Then, if by the term edema of the brain, Dr. Jones means such a process as that, I would subscribe to such a conception. But I do not believe I should consider that edema of the brain arises in a purely local manner. Without doubt there can be intensification of the general swelling in a given territory, but I do not believe it arises in a circumscribed fashion and remains circumscribed.

DR. JOSEPH BYRNE, New York: I confess to having doubts as to the localization in the first case Dr. Jones reported. There are a few inconsistencies in the sensory manifestations. For instance, motor power and stereognosis were defective in the left hand, but the author did not specify as to these functions in the right hand. I would ask just what the condition was in that respect. If the left hand only was involved, the trouble probably would be in the commissural fibers. The second point is as to the question of vibration. There are two elements in vibration: the affective or thalamic element and the critical or cortical element. These two things should be differentiated sharply. The specific element of heat as such, as well as the element of rapidity in vibration, implies comparison and is to that extent critical or cortical, whereas the pleasant or unpleasant feeling of heat or vibration (affective element) is mediated by the thalamus. The question of edema has not been settled satisfactorily to physiologists. It is the problem of physiologists to-day just as it was many years ago, Dr. Martin Fisher's work to the contrary notwithstanding.

Another question that comes up is the mechanism of cerebral nutrition. I do not want to be overcritical, and it may be merely a play on words, but there are no lymph vessels in the brain tissue or spinal cord. There are perivascular spaces but no lymphatics. Then, another question comes up as to the rôle of a very important structure the function of which has not been defined clearly. The neuroglia is in close anatomic relation with the perivascular spaces on the one hand and with true neural tissue on the other, and hence may play an important rôle in neural nutrition. We are a long way from having a true conception of the mechanisms of cerebral nutrition.

I differ somewhat from Dr. Jones as to diagnosis and localization, although I agree with him as to his method of treatment. Our practical work in cases like this has been most successful. I believe that in all these cases a decompression on one or the other side has great influence in restoring nutrition and function.

DR. BAYARD HOLMES, Chicago: I have been interested in the possibility of reducing edema of the brain on account of its occasional appearance as a terminal factor in dementia praecox. When the two cases that were reported by Nissl came out I began to look for some method of reducing the increased intracranial pressure and edema which seemed to be present in those cases, and found that when there was a high intraspinal pressure, which presupposed, perhaps, a high intracranial pressure, it could be reduced by the intravenous injection of large quantities of a concentrated solution of glucose, the concentration being about 33⅓ per cent. or higher. And I wondered whether in such instances as those reported a concentrated solution of glucose might not be useful in reducing edema of the brain, perhaps anticipatory of any surgical interference. In the few instances wherein we used the intravenous injection of a concentrated solution of glucose the intraspinal pressure went down and remained only a short time, not to exceed twenty-four to forty-eight hours, when the intraspinal pressure rose to the high point from which it had come. In most cases we found the pressure to be from

150 to 380 mm. of water, and six hours after injection of the glucose it had fallen, in one case, to 40 and in another to 80 mm.

DR. WILLIAM A. JONES, Minneapolis: The discussion has brought out the points I wished to emphasize. I think there is absolute justification in the decompression of many people who have ill-defined conditions in the cortex and meninges, and I believe we will find many localized edemas, from whatever cause. One case of mine might have been a cardiovascular renal case. At all events, the patient had the condition, and the swelling was large enough so that it was circumscribed and scarified, relieving the symptoms, and the man recovered, though he had exhibited all the focal symptoms described. He may have another attack later. The question of epilepsy is very important as to the possibility of some local troubles being responsible for the epileptiform attacks, and those cases may safely be decompressed, and I think many of them would be improved if not relieved entirely. As Dr. Sterne suggests, I believe there is a swelling of the cells which surround a certain definite area in the pia-arachnoid and unquestionably in the localized territory of the cortex, but a swelling here can be relieved just as well by decompression and scarification as it can elsewhere. The astereognostic sense was limited and localized and defined very definitely. It was carefully worked out and found to have absolutely disappeared within two weeks after operation. I think that proves we struck the right point, because an exudative and perhaps inflammatory process was found, though typically edematous in appearance.

ETIOLOGY OF THROMBO-ANGIITIS OBLITERANS (BUERGER) *

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On the basis of extensive clinical observation and a variety of laboratory examinations of patients suffering from the so-called thrombo-angiitis obliterans (Buerger), some points that may prove of interest are presented herein as tentative conclusions. They may have to be revised from time to time as our knowledge of the disease deepens.

Chemical analysis of the blood of these patients has shown that:

1. There is no retention of waste nitrogenous constituents.

2. There is no marked decrease in the alkaline reserve of the blood, as demonstrated by the carbon dioxide combining power of the blood plasma (method of Van Slyke).

3. In all the cases thus far studied, the ingestion of 100 gm. of glucose, after a brief fasting period, has produced hyperglycemia.

This observation shifts the whole question of the etiology of the disease to new ground, and the old designations appear no longer appropriate.

For the present I would propose naming the disease "glycophilia," and shall so call it in the course of this paper. The similarity of the name to "hemophilia" is intended to point to sex limitation and other obscure features of the disease.

GENERAL CHARACTERISTICS OF THE DISEASE

The disease usually develops soon after puberty, rarely after the fiftieth year.

Abuse of tobacco, particularly excessive smoking of cigars, may occasionally be a contributory etiologic factor, though some patients afflicted with this disease

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have claimed they are very moderate cigaret smokers. The disease is a chronic one, usually extending over many years. Periods of activity may alternate with periods of quiescence.

TABLE 1.—CHEMICAL ANALYSIS OF THE BLOOD

Case	Age	Red Blood Cells	Platelets	Wassermann	Milligrams, 100 C.c.					Chlorides, per Cent.	CO ₂ Comb. Power, Vol. per Cent.	Sugar, per Cent.
					Nonprotein N	Urea N	Uric Acid	Creatinin	Cholesterol			
H. K.*	33	6,120,000	Neg.	25	12	1.8	2.0	188	47	0.136
M. B.*	42	5,200,000	Neg.	30	14	1.0	0.5	183	65	0.129
V. M.*	39	5,800,000	Neg.	27	12	1.5	0.4	231	0.62	70	0.136
M. S.	29	6,170,000	Neg.	22	11	1.9	0.4	231	0.57	68	0.138
A. S.	47	5,400,000	544,000	Neg.	35	18	3.1	0.8	214	0.62	57	0.102
J. C.	44	5,900,000	720,000	Neg.	25	12	3.1	0.6	185	0.56	61	0.116
W. R.	30	5,500,000	416,000	Neg.	25	12	2.0	1.0	125	0.56	48	0.119
I. K.	43	6,814,000	484,000	Neg.	23	10	1.9	1.0	150	0.62	53	0.106
C. M.	37	7,200,000	524,000	Neg.	25	12	1.3	0.8	188	0.110
M.	..	5,336,000	544,000	Neg.	25	11	2.9	1.0	214	0.70	57	0.100
H. F.	..	5,100,000	383,000	Neg.	30	14	2.9	1.0	213	0.70	78	0.120
M. G.	33	5,984,000	496,000	Neg.	27	13	1.2	0.8	200	0.60	71	0.116
L. M.	34	5,240,000	416,000	Neg.	25	11	2.5	0.8	188	0.66	67	0.103
I. B.	41	4,584,000	312,000	Neg.	25	11	2.0	0.8	188	0.66	65	0.108
L. A.	40	Neg.	25	12	1.5	0.5	200	0.56	65	0.10

* Blood cultures negative.

The early symptoms are: pains in the calf of the leg on walking (intermittent claudication); spontaneous, uncontrollable pains in the toes, feet and legs, especially during the night, when in horizontal posture. Later on, the arteries usually become occluded and spontaneous gangrene develops. The upper extremities also may become affected.

GENERAL CHARACTERISTICS OF THE PATIENTS

All the patients we have had have been males; women seem to be immune.¹ They were all Jews of the poor classes who had emigrated from Russia, Poland, Galicia or Roumania—countries whose population is known to be especially subject to disturbances of sugar metabolism. This particular type of poor immigrant Jew has for ages been living under most insanitary conditions, closely confined and forced into intermarriage. They are hereditarily heavily encumbered; amaurotic family idiocy is specific among them; diabetes is a frequent disease; at one clinic it was associated with tuberculosis in 60 per cent. of the cases, against from 6 to 27 per cent. in other countries than those named above, according to general diabetes statistics. In one of my recent cases Irish descent was claimed. Investigation disclosed an Irish mother and a Polish Hebrew father; the son (my patient) had been left in ignorance of his father's descent.

Patients afflicted with a similar trouble are said to have been found also among the Swedes in Wisconsin and Minnesota (Ochsner), among the Japanese (Koga), in Cuba and in Persia, but the identity of

the disease found in these countries with that of these Jews has not been proved.

The majority of the patients have a high red blood count and proportionately high count of blood platelets.

In trying to find a donor for blood transfusion in these individuals, three years ago, it was seen by the serologist of our hospital, Dr. A. L. Garbat,² that they all belong to a special class.

Almost all the patients seen by us have flatfoot.

INVESTIGATION OF THE DISEASE

On previous occasions I have given expression to my conviction that the disease under discussion would turn out to be a blood disease. I determined to get more light on this question, if possible. Therefore, beginning with February of this year, during my service at the Lenox Hill Hospital (formerly the German Hospital) of New York, I arranged for the admission of a limited number of these patients and had their blood examined in every conceivable direction by Dr. Adolf Bernhard, the pathologic chemist of the hospital, to whom I am greatly indebted for his untiring cooperation (Table 1). After a series of negative results in various directions, we found a disturbance of the sugar metabolism.

The sugar tolerance in glycophilia has been studied in fifteen patients up to the present time. The results thus far obtained are embodied in Table 2.

The concentration of sugar in the blood of fasting normal individuals varies from 0.08 to 0.10 per cent. When 100 gm. of glucose are ingested, the sugar concentration of the normal individual does not rise above 0.15 per cent. at the end of one hour, and by the end of the second hour the concentration of blood sugar has again returned to the normal or to a somewhat lower level. If the concentration of blood sugar one

TABLE 2.—CARBOHYDRATE TOLERANCE TEST

Case	Age	Blood Sugar before Glucose, per Cent.	Blood Sugar 30 Min. to 1 Hr. after 100 Gm. Glucose, per Cent.	Blood Sugar 2 Hrs. after 100 Gm. Glucose, per Cent.	Urine before Glucose	Urine after Glucose
1. M. B. 3343	42	0.129	0.180	Neg.	Neg.
2. V. M. 3323	39	0.126	0.212	Neg.	Neg.
3. M. S. 29	40	0.138	0.246	Neg.	1st hr. trace 2d hr. trace 3d hr. trace
4. A. S. 953	47	0.102	0.198	0.198	Neg.	Neg.
5. J. C. 1272	44	0.116	0.180	Neg.	Neg.
6. W. R. 1610	30	0.110	0.189	0.120	Neg.	1st hr. trace 2d hr. trace 3d hr. trace
7. I. K. 1912	43	0.106	0.189	0.159	Neg.	Neg.
8. C. M. 2152	37	0.110	0.212	0.006	Neg.	1st hr. neg. 2d hr. trace
9. M.	..	0.100	0.174	0.100	Neg.	Neg.
10. H. F.	..	0.100	0.153	0.112	Neg.	Neg.
11. M. G. 3454	33	0.116	0.189	0.174	Neg.	Neg.
12. L. M. 2498	34	0.106	0.138	0.215	Neg.	1st hr. neg. 2d hr. trace 3d hr. trace
13. I. B. 2468	41	0.103	0.192	0.126	Neg.	1st hr. neg. 2d hr. large amt. 3d hr. trace
14. L. A. 3288	40	0.100	0.174	0.138	Neg.	1st hr. trace 2d hr. 1.5% 3d hr. trace
15. S. S. 3696	51	0.096	0.222	0.192	Neg.	1st hr. 0.6% 2d hr. 1.6%

hour after the ingestion of the glucose reaches a higher level than 0.15 per cent., hyperglycemia exists.

This hyperglycemia may be accompanied by glycosuria. In the cases studied, the fasting level of the blood sugar varied between 0.096 and 0.168 per cent.

1. In a recent case hyperglycemia was also found in a Jewess who complained of continuous cold sensations in her feet and showed absence of pulse in both peroneal arteries.

2. Meyer, Willy: Conservative Treatment of Gangrene of the Extremities, *Ann. Surg.*, 1916, **63**, 280.

In seven cases the concentration of blood sugar before the ingestion of glucose was higher than the normal figure, varying between 0.116 and 0.168 per cent. In other words, some of these patients come to us with a certain degree of hyperglycemia, without having taken the glucose.

One hour after the ingestion of 100 gm. of glucose, the blood sugar concentration in these patients varied between 0.138 and 0.248 per cent., whereas in the normal individual, as already stated, it does not rise above 0.15 per cent. Two hours after the ingestion of the glucose, the blood sugar concentration varied between 0.10 and 0.215 per cent.

One case, M., No. 2498, did not show a *marked* increase in blood sugar concentration after the ingestion of glucose, it being 0.138 per cent. one hour later. However, this case showed the highest concentration of blood sugar after two hours, at which time it was 0.215 per cent. It is the only case in our series that shows a higher concentration of blood sugar at the end of the second hour than at the end of the first hour.

In six other cases the blood sugar at the end of the two hours did not reach the original level obtained before the ingestion of glucose.

The results of these investigations indicate some disturbance in the carbohydrate metabolism of the patients. The studies are being continued in our hospital, and their progress will be reported from time to time.

The general clinical aspect of these cases in many respects shows such a close similarity to what we are accustomed to see in cases of diabetes mellitus that their classification as "near diabetics" appears justifiable, and I fully expect to find this diagnosis verified after the study of a greater number of cases.

The characteristic difference in the sugar tolerance of healthy individuals, glycophilics and diabetics is diagrammatically shown in the curves in the accompanying chart.

The probable cause of hyperglycemia in glycophilia, and also of the erythrocytosis which is frequently found combined with it, I expect to take up soon in another communication.

The chemical blood analyses of Dr. Bernhard appear to justify the above proposed change in the nomenclature of this trouble. "Thrombo-angiitis obliterans"—the name given it by Dr. Leo Buerger of New York—implies a thrombus formation, due to inflammation of the blood vessels. I do not believe, however, that the occluding masses within the small arterial blood vessels are really thrombi, nor do I believe that an inflammatory process of the blood vessel walls plays a rôle from an etiologic point of view.

I am rather inclined to explain these masses as originally being a conglomeration of red blood corpuscles due to erythrocytosis and stasis, the latter being a

sequence to the increased viscosity of the blood. They may appear to the eye of the pathologist, macroscopically and microscopically, as thrombi. Yet, I feel that microscopic lenses of higher power than we know at present will probably show them to be something different.

This is also the reason why the name "thrombophilia," which I recently proposed³ for this disease, has been discarded.

CLINICAL OBSERVATION AND TREATMENT

In my service at the Lenox Hill Hospital five beds were set aside in the early part of this year for the study and treatment of glycophilia. They have been constantly occupied by these patients, and for weeks at a time by the same men. At certain periods it was necessary to increase the number of beds to seven. I feel the need of at least fifty beds for this purpose. But to have set aside more than from five to seven would have crippled the service.

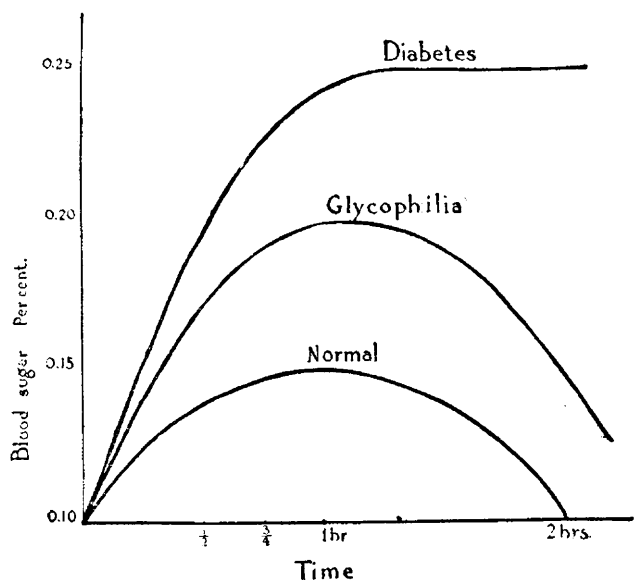
In order to handle with these limited facilities the greatest possible number of individuals, I have endeavored to shorten the time required for their treatment at the hospital. Out of the first empiric steps and the feeling of our way, a sort of routine has gradually developed, as follows:

On the patient's admission to the hospital a complete chemical analysis of his blood is made, also a complete blood count, platelet count and Wassermann test. Then the sugar tolerance test is carried out. According to our present view, a positive result places the patient in the glycophilic class, and he is retained for treatment.

In order still further to cut down the time required for preliminary examinations and to save several days of hospital attendance, the blood tests and the sugar tolerance tests have of late been made in an ambulatory fashion at the dispensary. The work there has been placed in charge of a special assistant, Dr. M. Greenberger.

A most conspicuous feature in the picture of glycophilia seems to be a considerable dehydration of the tissues. A glycophilic may be compared to a flower withering for the want of water. Our treatment, therefore, consists in supplying an abundance of water to the system.

From the start, the treatment is intensive. It aims at producing quickly the highest possible degree of hydremia. This is obtained by the simultaneous use of duodenal flushing with the help of Einhorn's duodenal tube, as suggested by McArthur⁴ of Chicago, combined with hypodermoclysis. In duodenal flushing, from 8 to 10 quarts of Ringer's solution, or a



Comparative blood sugar curves after ingestion of 100 gm. of glucose.

3. Report of Surgical Section, New York Academy of Medicine, Med. Rec., New York, 1917, 92, 262.
4. McArthur, L. L.: Surg. Clinics, Chicago, 1917, 1, 499.

solution of sodium bicarbonate (from 15 to 30 gm. of the salt per day) of body temperature, are given by the rapid drop method within twenty-four hours. Ringer's solution or a solution of sodium bicarbonate seems to have a better effect than plain water.⁵ In duodenal flushing the actual absorption of the water into the system by way of the blood has been proved by the fact that about as much urine is discharged as water is poured into the intestinal tract.

More recently we have made the patient buy the tube before he enters the hospital. Here he is taught its use, and as soon as he has learned it, he is sent home to continue the treatment there for from three to five weeks, provided it is a mild case; if he represents a more serious case, he remains at the hospital for additional hypodermoclyses. We are now about to transfer to the dispensary the giving of hypodermoclyses, and we are arranging for three treatments per week for each patient, under special supervision. Of course, only patients who are able to walk can be subjected to such ambulatory treatment.

Hypodermoclyses are given with the maximal quantity of Ringer's solution the patient can endure, and repeated daily, if possible. Intravenous infusion of Ringer's solution is not resorted to, because the aim is the attainment of the greatest possible penetration of fluid into the body tissues. Extensive literature shows the latter to remain quite dry when the liquid is introduced intravenously.

The injection of such quantities of fluid is made principally for the purpose of reducing the viscosity of the blood and thus enable it to pass again through the narrowed arterial channels.

During this treatment the condition of the patient occasionally runs the following course: The incessant, most excruciating pains, not controllable by any other means, decrease in intensity. At first only a few hours of sleep can be enjoyed by the patient while sitting upright, for the night, in a chair. Soon he goes to bed and keeps the affected leg hanging out; then he risks lifting it to a horizontal posture; eventually he rests comfortably in bed, free from pain.

The foregoing refers to patients without local gangrene, either of the superficial or deeper tissues. If gangrenous lesions of the skin and subcutaneous tissues are present, it has been found best, as regards saving time in the treatment, and particularly in alleviating the pain, to touch the ulcerative spot *lightly* with the actual cautery. The sealing of the hypersensitive sore does away with a good deal of pain. Under the scab, often healthy granulations form which tend to more rapid healing. Sometimes the eschar stays on until cicatrization of the formerly obstinate wound is completed. The light, dry gauze dressing is changed as little as possible during the time that the artificial hydremia is being maintained.

In the majority of cases the deeper tissues, particularly periosteum and bone, are affected. Often it will be seen that what at first glance appeared to be a paronychia is a necrotic osteoperiostitis of, say, the third phalanx of the toes or fingers. This may have the tendency to invade the central cavity of the bone, and later involve the interphalangeal joint; or it may be that a seemingly superficial gangrene, for instance,

over the os calcis laterally or at the calx itself, runs straight down to the bone. If conservative treatment is at all contemplated in a case like this, it is unwise to treat these wounds with all kinds of dressing, until the demarcation line has formed. Circumcision of the necrotic tissue with a knife, and the use of the actual cautery on the surface of the fresh wound, are better. Although with the latter procedure it will take a long time before the wound heals, the patient's pains are often much lessened.

When it comes to operating—enucleation or amputation—I prefer to undertake the operation after the patient's system has been thoroughly permeated with fluid. It may then be sufficient to enucleate or amputate a toe or a finger, where formerly larger parts of the extremity had to be sacrificed. In other cases, amputation below the knee will give a good result, while without the preceding hydration of the system, amputation above this joint would probably have been required. It almost seems as if this temporary saturation of the system with fluid would enable us to operate more peripherally, with greater and more frequent success, than could be obtained formerly. Further experience is needed definitely to determine this point. Probably the degree of improvement in the circulation and the condition of the blood will differ in different individuals.

Without doubt there are abortive cases of the disease.

A most remarkable consequence of the treatment in some cases has been the restoration of pulsation in arteries over which, before the treatment, no pulse could be felt. Koga observed the return of almost normal pulse in ten out of thirteen Japanese cases, after a course of hypodermoclysis. I have seen it in only a few instances. Still, I have seen it, and the mere fact that pulsation does return in previously pulseless major arteries—or, expressed in other words, that occluded arteries become again patent, and within a comparatively short time—makes me look with doubt on the theory that ascribes the occlusion to thrombosis. It rather inclines me to attribute the occlusion to stasis, as far as the arteries are concerned. In the veins the occlusion is probably a true, though secondary, thrombosis.

Patients without gangrene are discharged as soon as their pains have subsided. When this goal is not reached, ligation of the deep femoral vein below Poupart's ligament, or amputation, is performed. In spite of one very good result of arteriovenous anastomosis between the femoral vessels in Scarpa's triangle, in one of my patients, aged 31, I could not make up my mind thus far to have this operation take the place of a regular intermediate step, before amputation is resorted to.

However, be it specially emphasized, none of these patients are cured; they are merely improved, whether or not operation is done. In either event, their trouble returns sooner or later. This, at least, has been the rule so far. Of course, there may be fortunate exceptions. The reason for this fact is that the underlying cause of the disease is as yet unknown, and until it is known, we cannot hope to bring more than temporary relief to these patients. Our treatment, conservative as well as radical, thus far has been only palliative. True, after amputation of the leg, the old tormenting pains subside as by magic; but the disease goes on nevertheless. Frequently the other extremity after a while shares the fate of the one amputated, and still

5. In making up Ringer's solution we have employed the tablets furnished by the firm of Burroughs Wellcome & Co., dissolving two tablets in 1 pint of water. Each tablet contains: calcium chlorid, 0.05 gm.; potassium chlorid, 0.05 gm.; sodium chlorid, 2.25 gm.; sodium bicarbonate, 0.025 gm., and glucose, 0.25 gm. One tablet dissolved in 250 c.c. of water forms Ringer's solution.

later on, the upper extremities may be attacked. Proof of the continuance of the disease after the discharge of the patients, with their symptoms improved and their wounds healed, is the fact that they are usually unable to do much walking without getting pains and becoming excessively tired.

We put these discharged patients on a mild anti-diabetic diet and advise them to take up work that will not unduly tax the muscles of the respective extremity, although the latter advice is usually difficult to follow for this class of mostly poor patients. They are further instructed to report back for blood examination at varying intervals, according to conditions present.

Much work remains to be done to clear up this fascinating affliction. I am convinced that it is primarily a disease of the blood of endocrine origin—due principally, perhaps, to a disturbance in the normal function of the suprarenals—and not a disease of the blood vessels. Should we succeed in tracing it back to its original cause and then find a way of correcting the disturbance, not only untold suffering would be spared these pitiful patients and their families, but vast sums of money saved to the country at large.

CONCLUSION

Although the number of cases studied is small, the results thus far obtained definitely show that there is something defective in the carbohydrate metabolism of these patients.

Regarding the theory I have advanced above, that one or more glands of internal secretion are pathologically affected and cause the disturbance, I fully realize that it rests as yet on slender support. But I take kindly to this theory, because it tends to remove the disease from the realm of the mysteriously obscure, and brings it into the purview of an already thoroughly explored field of medicine, thus making available for its elucidation a vast treasure of experimentation already laid down in literature. However, even with this help, the task of finding a path through the many contradictory observations and apparent inconsistencies is altogether too big for individual effort, and recognizing this to the full, it seemed to me best to make known at this time what we have found thus far and to urge the cooperation of all those who are called on to treat this class of patients.

The best way to make real progress in this matter would be to get some philanthropist interested in the subject and induce him to equip liberally and endow a commission, composed of specialists from the various fields of medicine involved and having for its sole object the study of glycophilia.

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ABSTRACT OF DISCUSSION

DR. V. L. SHRAGER, Chicago: It is very sad that this condition is not better understood, particularly clinically. It does not matter so much that we do not know what the cause is at this time. At Mount Clemens, where I have been a patient myself, many patients were treated for rheumatism when they had only the early stages of thrombo-angiitis obliterans. Flatfoot is frequently associated with angiitis obliterans, which suggests an anatomic theory. Oppenheim and a few others have found that these patients have a congenital smallness of the blood vessels. Many of them, whether treated or not, develop epigastric distress and excruciating pain. I have seen cases of spasm of the epigastric region entirely ignored. The angiospasm is very much the same as a cramp in the calf of the leg. I must say in defense of the Ringer solution that while it does not modify the disease, it is a blessing to

the patient. I have never known any of them that did not get absolute relief. I have extended the use of Ringer's solution to trophic diseases of the foot and other diseases. I have treated several cases of diabetic arterial changes, and I have found that Ringer's solution gives them a great deal of relief.

DR. WILLY MEYER, New York: Dr. Buerger has made careful pathologic studies of the type of the blood vessel occlusion. I so far would disagree with him with reference to the interpretation of his findings. Our laboratory examination has shown the hyperglycemia often before the ingestion of glucose, surely soon after. I believe that these patients are "near-diabetics." Such assumption will explain easier also the cases with open blood vessels and gangrene, as in diabetes. From the standpoint of symptomatic treatment we can best improve the condition of these patients by pouring as much fluid into their system as possible, particularly by using the duodenal tube. Nobody can drink 8 to 10 quarts of fluid a day for a number of weeks. That the fluid is actually absorbed and not to greatest extent discharged by the rectum is shown by the large amount of urine voided by these patients. As a matter of course, some fluid is retained in the system.

Regarding surgical treatment, it seems unwise in patients with superficial gangrene to wait for further developments. We should act immediately and touch the gangrenous spot lightly with the cautery; under the eschar the wounds often heal. If we remove the nail in a patient with what appears to be a paronychia, we will often find under the same an osteoperiostitis, not infrequently combined with osteomyelitis, of the third phalanx, which seems to be a reactive inflammation to localized bone gangrene, and if treated properly will greatly relieve the pain. A most interesting observation, which will be valuable if corroborated by further clinical data, is, that if we pour a large amount of liquid into the system of these patients, a required amputation can likely be done more peripherally; in other words, we may with it, for instance, more often be enabled to amputate below the knee rather than above the knee without recurrence of gangrene. If we should be able to find the cause of this disease and correct it, certainly great expense would be saved to the community at large, not to mention the unspeakable suffering of these patients and their families. For the trouble is comparatively frequent, especially in the large centers of our country, and those stricken by the disease always spend months and years in hospitals.

Mortality Among Women from Causes Incidental to Child-Bearing.—Between the ages of 15 and 44 the diseases and conditions incidental to child-bearing account for more deaths of women than does any other disease or class of diseases except tuberculosis. This statement is made by Louis I. Dublin, of the Metropolitan Life Insurance Company, in an article on a study of mortality among women from causes incidental to child-bearing, and it is based on the records of death of a large number of insured women in the industrial department of the Metropolitan company. During the six year period, 1911 to 1916, covered by the study, it is estimated that 14,694,260 insured women between the ages named were under observation. From this number 10,056 deaths occurred from diseases and conditions incident to childbirth, in the ages between 15 and 44. Most important of these diseases was septicemia, which was responsible for 4,321, or 43 per cent., of the total. Albuminuria and convulsions caused 2,654, or 26.4 per cent., of the total. It will be seen that these two conditions accounted for 69.4 per cent. of the total deaths from causes incident to childbirth. The figures relating to mortality on account of childbirth in the Metropolitan company are steadily improving, due, as is said, to the extension and improvement of the prenatal and post-natal nursing service inaugurated in 1909. In 1916, of a total of 160,843 women policy holders visited during illness, 41,572, or 25.8 per cent., were cases resulting from diseases or conditions incidental to the puerperal state. In the period between 1911 and 1916 the death rate among white policy-holders from these causes fell 10.7 per cent., the decline in mortality from septicemia being 17.3 per cent.