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ORIGINAL ARTICLES.

THE DIFFERENTIATION OF THE CARDIAC INCOMPETENCY OF INTRINSIC HEART DISEASE AND OF CHRONIC NEPHRITIS.

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BY FRANK BILLINGS, M.D.
CHICAGO, ILL.

The recognition of an incompetent heart is usually not difficult. The dyspnea of exertion, the sense of post-sternal weight and constriction of the chest, the cough of exertion, and upon assuming the recumbent position, the enlarged liver with tenderness and often with pain or a feeling of weight in the epigastrium, the indigestion of portal congestion, the usually scant urine, the edema of the feet, or all of the above named conditions much aggravated until constant dyspnea with orthopnea, general anasarca, excessive physical weakness, marked palor, or palor and cyanosis give unmistakable evidence of cardiac weakness. The local signs of valvular disease may be absent or present. Signs of cardiac enlargement with dilatation of the chambers of the heart are present and may usually be recognized.

These phenomena are usually present in varying degrees of severity, in an incompetent heart. When due to intrinsic heart disease a valvular lesion or some other cause of dilatation of the heart and weakness of the heart muscle is present the chronic nephritis, cardio-vascular changes occur, and especially in the form classed as diffuse interstitial nephritis. In the latter stages especially of chronic nephritis the heart may become so dilated that it becomes incompetent, and the symptoms and signs enumerated appear. The urinary findings, the symptoms of uremia and other characteristic signs may plainly indicate the primary nephritis. In many cases, however, the differentiation can not be easily made, and in a few examples the diagnosis may remain in doubt until the side light of the dead house clears up the darkness. It is with these doubtful cases that this brief paper has to deal.

The following case will serve as a text and more clearly illustrate the subject.

Mr. W. McF.; aged 50; American; lawyer; married. Came to me on Sept. 12, 1894, and presented the following history:

Father died at 76 years; mother living, aged 72 years, and in good health. One brother died of acute pneumonitis. The father suffered from a tremulousness of the right arm, and the patient has had from birth a tremulousness of the right upper extremity, which is intensified by the presence of strangers, or by an attempt on the part of the patient to suppress it. The intentional trembling extends to the left arm also, during excitement or other emotion.

The patient has never had a venereal disease. He has never been addicted to the use of alcoholic beverages or tobacco in any form. He has always been well; has always had much to

do in his profession, and has worried and fretted much about his affairs. He has always eaten heartily of all kinds of food. He has not taken physical exercise as a recreation.

For a month he has noticed shortness of breath after exercise, and an unusual throbbing of the heart after exercise, after a hearty meal and when excited. The strength has been noticeably less, and recently the ankles have been swollen in the evening. The tremulousness of the hands has become worse. Vision is less acute. The mind becomes easily tired. He dreads the presence of strangers, and fears to go into court with his cases. He has headache, with a sense of pressure about the head and the nucha.

Examination.—Height, six feet two inches; weight 250 pounds; face pale and eyes puffy; tongue large, pale and covered with a white fur. Muscles flabby; upper extremities tremulous, and this was increased by the excitement attendant upon the examination. Abdomen large, prominent and flaccid. Liver area apparently normal; spleen not palpable. Lungs normal, with a few moist râles over posterior dependent portions. Heart area difficult to ascertain because of overlying lung. Apex beat not palpable. Heart sounds pure but weak. No endocardial murmur. Heart action irregular; intermittent pulse, 96 per minute. No accentuation of second sound over pulmonary or aortic valves. Radial pulse, soft, moderate in size, compressible and intermittent. Moderate edema of both legs. A single specimen of urine showed, sp. gr. 1025, acid reaction, brownish color, small amount of albumin, no sugar, no bile. A few hyaline and granular casts were found in the deposit of the centrifuge.

He was kept quiet at his home upon a simple diet excluding meats. A more extended examination revealed diminution of the urine of twenty-four hours, with a relatively low specific gravity; lessened total solids, and one-half the normal urea. Albumin was constantly present with granular and hyaline casts. The heart presented the physical findings enumerated. The sphygmograph gave a poor and unsatisfactory tracing, because of the difficulty of applying the instrument to a wrist which was in a constant intentional tremor, and also because the heavy muscles and tendons covered the radial artery so fully that the pressure necessary to secure a result gave a tracing of no value. Strychnine sulphas, infusion digitalis and hydrogogue cathartics were used from the beginning. At first there was apparent improvement. The incompetent heart beat more regularly and forcibly, and the symptoms referable to a weak heart disappeared or became better. The urine increased in amount, but the total solids and the urea remained deficient, the specific gravity low, and the albumin and casts were always present. He resumed his work in spite of protests to the contrary during January. From the beginning of the return to work he gradually relapsed into the condition which he first presented. During February, March and April he became gradually worse, and by the beginning of May he was practically water-logged. There was general anasarca, orthopnea, cyanosis, cough, a weak wavering pulse, feeble heart action without perceptible murmurs, accentuation of the aortic second sound, diminished bulk of urine, containing much albumin and many hyaline granular and epithelial casts, with less than one-half the normal amount of urea. There was sleeplessness but no headache, no nausea

and no disturbance of vision, with normal fundus. The general appearance was that of a chronic Bright's disease, with the pallor, puffy face and heavy expression so characteristic of the disease. On April 20 he complained of pain in the left side of the neck, and within twenty-four hours the left jugular vein, extending from the angle of the jaw to the clavicle, was felt as a cord-like body, which was tender to the touch. This was diagnosed a thrombus, and the diagnosis was verified by an extension of the thrombus into the left axillary and brachial veins. This complication was attended with much edema of the left upper extremity, which continued for about two weeks. Absorption of the thrombus seemed to occur and the circulation was fully restored within a month. During the time enumerated the patient was kept quiet in bed as much of the time as he would submit to. The diet was exclusively vegetable, with some milk. The medication consisted of strychnine sulph. gr. 1/30, every four to six hours. Digitalis in infusion or tincture, off and on, Basham's mixture of iron in 3ss. every six hours, and hydrogogue cathartics, elaterin,—gr. 1/20 to gr. 1/10, every six hours, or the salines in doses sufficient to produce free, watery stools several times each day.

From the time of the absorption of the thrombus of the left external jugular, axillary and brachial veins the patient commenced to improve, and by June 15 he was entirely free from dropsy, the heart stronger, beat almost regularly, and the patient felt able to go about. The urine still contained albumin and many hyaline and granular casts, and a deficient amount of total solids and urea.

He was placed upon a diet for nephritis, excluding dark meats, and the iron and strychnia were continued. He spent the summer at the sea-shore. Upon his return home in September, 1895, his weight was 220 pounds; his color was good, and he felt perfectly well. An examination showed a steady heart action, with a good strong and soft pulse of 84 per minute. The heart presented no abnormal signs. The urine of twenty-four hours was as follows: quantity 56 ozs., sp. gr. 1022; reaction, acid; color, amber; urea 2 per cent. or 537 grains in twenty-four hours. No bile, no sugar, no albumin and no casts.

He was cautioned not to eat much meat, and to not fatigue himself by work.

He returned to his professional work, which was very exacting. He walked, for recreation and exercise, five or more miles per day. During 1896 he remained well. I saw him several times and examined the urine every month. Unusual professional care worried him much during the winter of 1896-7. In March, 1897, he again complained of the dyspnea of exertion, and the heart was not quite regular. There was a trace of albumin, but no casts in the urine at this time. Rest, with strychnia, gave him relief. He was out of Chicago for a vacation during July and August. Sept. 24, 1898, he again complained of dyspnea upon exertion and of palpitation of the heart. Weariness after mental or physical exertion and the old symptom of fear of people returned.

Examination showed some edema of feet and legs, a weak, irregular heart, and for the first time a systolic murmur at the mitral area, not transmitted, with accentuation of both the aortic and pulmonary second sounds. The urine contained a trace of albumin and a few hyaline casts, and a normal percentage of urea.

He was again placed at rest and submitted to the bed with better grace than during the preceding attack. In spite of physical and mental rest, heart tonics, free stools daily and a simple diet he became gradually worse. On December 25 he was suddenly seized with aphasia and slight loss of power in the right leg and arm. Within two hours he could talk incoherently but never regained the power of free expression by speech or writing.

The dropsy gradually increased and the heart weakness more pronounced. The heart murmur remained constant, and there was evidence of dilation of both ventricles of the heart. The urine was, as a rule, scant, from 20 to 40 ounces per twenty-four hours, with lessened solids including urea, but not much albumin and but few casts. In this attack the symptoms pointed chiefly to the heart as the chief offending organ. At no time in this attack did the urine present evidence of more than chronic venous congestion of the kidneys. He died suddenly on March 14, 1898.

The clinical course, the physical signs and urinary findings in this case in the first attack pointed more to nephritis as the primary disease. The thrombosis of the left jugular and other veins, the absence of retinal changes, the primary edema of the left, the absence of the nervous phenomena of uremia, and finally the apparent recovery and status of good health for about eighteen months negated nephritis. On the other hand, the last named phenomena, the relapse of the disease, the physical findings of the heart and the condition of the urine in the last attack pointed to an intrinsic disease of the heart, as a cause of the insufficiency of that organ.

The difficulty of the recognition of the primary cause of the incompetent heart is the intimate relation which exists between the cardio-vascular system and the kidneys. In all inflammatory diseases of the kidney there are cardio-vascular changes, which are most marked in the so-called granular kidney. These fibroid changes of the arteries and hypertrophy of the heart, when most marked are easily recognized by the physical signs and the characteristic urinary findings. In these cases, too, there is not, as a rule, a tendency to that form of heart incompetency which leads to dropsy. The arteries, palpable and invisible, partake of the fibroid change, and cerebral or other hemorrhage is more likely to occur than dropsy. Headaches, nausea, vomiting, cyclical diarrhea, retinal changes, mental excitement or hebetude of mind, coma or epileptoid convulsions are the rule in the later stages of the disease. In chronic tubal nephritis, on the other hand, while cardio-vascular changes are less marked, dropsy may occur with all the phenomena of an incompetent heart. The pallor of the patient, the puffy, waxy face, the retinal changes, the headache, nausea and vomiting, the cyclical diarrhea, the epigastric pain and pressure or sense of weight, the scant urine containing much albumin, with hyaline, granular, epithelial and fatty casts, with microscopic quantities of blood at times; and frequently all the signs of a dilated heart, with endocardial murmurs and a weak, rapid and sometimes intermittent or irregular pulse are not unusual phenomena.

The incompetent heart of an arterio-sclerosis which involves the coronary arteries alone or of the arteries of the heart and kidneys or of the general arterial system, may present phenomena so nearly like the clinical findings of one or both of the inflammatory condition of the kidneys mentioned that the

differentiation becomes difficult. There may be the thickened artery, the sustained full pulse, the hypertrophied or dilated heart, the retinal changes, vomiting, diarrhea, nervous phenomena, headache, albuminous urine with deficient urea, and the presence of casts of all varieties.

An incompetent heart from valvular disease, presenting first of all the physical findings, characteristic murmurs, dyspnea of exertion, cyanosis, dropsy, beginning in the lower extremities, enlarged liver, portal congestion with consequent indigestion, tendency to thrombosis or embolism, a concentrated albuminous and often bloody urine with hyaline and granular casts, is easily recognized when classical, but may present symptoms and clinical findings which make differentiation from primary kidney disease very difficult. In other words, kidney disease with cardio-vascular changes and an intrinsic heart disease with broken compensation and resulting venous congestion of the kidney, often present common symptoms and signs, which may indicate coincident primary involvement, or a primary disease of the one with resulting disease or disturbance of function of the other. Those phenomena common to both I have already enumerated. There are special phenomena, however, sometimes present, which although sometimes common to the two are more often the result of a primary heart or kidney lesion alone, and these signs or conditions are of some aid in differential diagnosis.

If the urine, even though scant, shows a relatively low specific gravity, with diminished total solids, lessened urea, much albumin and casts of all varieties.

If the arterial fibrosis is marked and the pulse full and sustained, or without arterial thickening if the pulse is sustained.

If the dropsical effusion is poor in albumin.

If the dropsical effusion contain urea in considerable amount.

If there is a tendency to inflammation of serous surfaces, pericarditis and pleuritis especially.

If there is a tendency to persistent morning nausea and vomiting of stringy mucus.

If there is tendency to periodic diarrhea.

If there is much headache, especially severe frontal and occipital pain.

If there is a tendency at one time to unrest and mental excitement or at another to somnolence or to coma.

If cerebral hemorrhage occur.

If the dyspnea of exertion becomes of the Cheyne-Stokes type. If severe epigastric pain occur.

If the eye ground shows the changes common in albuminuria, and if the patient present a pale, waxy puffy face and heavy expression, and a general anemia, the diagnosis is in favor of nephritis.

If there is a history of antecedent disease in which endocarditis may have occurred.

If the heart shows physical signs of valvular disease with characteristic murmurs.

If there is an irregular heart action persistent in character.

If the pulse is weak and not sustained with or without arterial fibrosis.

If the dyspnea of exertion is attended with cyanosis.

If there is a normal amount of red cells and hemoglobin or only a moderate degree of anemia.

If the dropsical effusion begins in the lower extremities, appearing during the day.

If the dropsical effusion is relatively rich in albumin.

If the dropsical effusion contains relatively little or no urea.

If there is a much enlargement of the liver and tenderness of the left lobe.

If the urine is scant, of high specific gravity, with much lateritious deposit, little or no albumin, a high percentage of urea, although the total urea of twenty-four hours is deficient; a few casts only, chiefly hyaline or finely granular.

If there is little or no headache, no eye ground changes, and if venous thrombosis or embolism occur the diagnosis is in favor of intrinsic heart disease.

100 State Street.

DISCUSSION.

Dr. COLE of California—I am much interested in the paper read by Dr. Billings. It is very difficult to decide whether the primary condition is one pertaining to the kidneys or to the heart; this is more especially true in advanced diseases. And I am glad to know that there are others that have this difficulty in making a differential diagnosis.

I am much pleased with the finest points in the differential diagnosis which Dr. Billings has made.

Dr. WEST of Galveston, Texas—It appears to me that this is one of the most interesting and practical subjects. It is a difficult question to determine whether the primary disease is one of the kidneys or of the heart. Diseases of these organs occur so concurrently. One point in the differential diagnosis and which assists in determining which is the primary disease, is the therapeutic test. If renal symptoms due to congestion of the kidney, such as albuminuria and other evidences of disturbed renal circulation appear, the patient should be placed upon proper treatment which tend more especially to remove the results of disturbed cardiac action and disturbances of the circulation; this consists in placing the patient in a horizontal position and giving him heart tonics, such as digitalis, strychnin, etc., and stimulate the emunctories, especially the bowels and skin; under that treatment in many instances the differential diagnosis will clear up. The urine becomes more abundant, there is a lower specific gravity, the casts disappear and the differential diagnosis can be made with perfect ease. In my experience, if these cases are allowed to progress for a long time, it is then impossible to make a differential diagnosis.

Dr. BOND of Indiana—This is an interesting subject. Dr. Billings brought out clearly all the prominent points. In one case the patient had kidney disease and still further arterio-sclerosis. One common factor in both diseases is arterio-sclerosis. I doubt if it is possible to have either disease without arterio-sclerosis; this is a common observation in these cases even before albumin or casts appear in the urine or a heart murmur is observed, showing deficient waste. Some years ago, 100 cases of this kind were observed where there was a deficiency of urea found in cases not suspected of having heart or Bright's disease. I think the first evidence of this disease which is constant is a deficiency in the nitrogenous waste. Instead of twenty-five grams of urea excreted in twenty-four hours, a man was found to be passing ten or fifteen grams; he was afterward found to be passing casts and there were symptoms of arterio-sclerosis; the albumin came on especially after exercise. One year afterward the heart was found to be affected.

Dr. BROWN of Illinois—I think it is very important that we should separate the two great forms of Bright's disease, the interstitial from the chronic. There is no disease in which we so often make mistakes in the early diagnosis as in interstitial

nephritis. For five or ten years there may be no albumin, no dropsy, no casts or anything to account for the polyuria which exists, and it is very difficult to find the cause. The diagnosis is made, not upon the symptoms directed to the kidneys themselves, but upon the symptoms that are referred to other organs than the kidneys—upon the subjective symptoms.

Dr. BRIDGE of California—What Dr. Bond has said is largely true. It is a matter of no great moment in these cases to settle the question whether the lesion is one of the kidneys or of the heart, because sooner or later lesions in both will occur in the same patient. Dr. Billings has set forth, as well as could be set forth, the means of making a differentiation. An important thing brought out in the paper was that there were two kinds of lesions and the fact that they result from similar causes. There is some poison in the blood containing effete matter, which lowers the vitality of the vessels of the kidneys, whereby these various lesions occur. In his case he had afterward a thrombus in the veins due to some injury to the lining of the vein, followed by certain changes in the blood, then atheroma which caused changes in the heart, producing dilatation and finally murmurs. What is this change? What metamorphosis is there in the lining of the vessels of the heart and kidney which make it predisposed to take on these changes?

As to the diagnostic value of casts, I feel sure that if the Doctor should use the centrifuge carefully, he would find casts in everybody's urine. It is no question of the presence of casts, but their number.

Dr. DAVIS of Chicago—The most difficult problem presented to us is to find which is the primary disease, that of the heart or of the kidney; especially is this true when the kidney and heart trouble appear to come on simultaneously. In certain cases the phenomena present enable us to make a diagnosis as to which is the primary lesion. On the other hand, as already has been stated, we find cases in which there are evidences of cirrhosis of the kidney with cardiac lesions, due to atheromatous condition of the vessels generally. Here symptoms come on which are important in that they show us which lesion predominates, whether the renal or the cardiac. These cases require the closest scrutiny and treatment.

Dr. BRIDGE—There were two points brought out in the paper of Dr. Billings, one, heredity, or the family tendency to the disease; second, the occurrence of some persistent disease which could lead to either heart or kidney disease. Frequently Bright's disease is a family disease. Also, the persistence of any disease which could lead to endocardial or myocardial disease would lead to heart disease as against Bright's disease.

The point raised in regard to the thrombus is a good one. Whether it occurs in the blood vessel of the body or the brain it indicates a tendency to heart disease. The non-existence of hypertrophy and valvular lesions in any case would point away from the heart and toward the kidney. In Dr. Billings' case might it not have been that the earlier disease was a diffuse nephritis in a patient with a weak heart which manifested symptoms of this weak heart? Afterwards the patient regained health, and then suffered from cardiac disease which ended his life. I believe that cases of chronic diffuse nephritis recover under appropriate treatment and the evidences of kidney lesion, previously present, will entirely disappear. Perhaps it may recur again or produce changes which will produce manifestations later on in the life of the individual.

I am glad that I have heard the paper; the differentiation between lesions of the heart and the kidney is of considerable importance as bearing on both prognosis and on treatment.

Dr. WEST of Galveston, Texas—I wish to speak of another class of cases of interstitial nephritis in which there is a low specific gravity, with an entire absence of renal symptoms or disturbances of circulation. There are no symptoms at all except a slight albuminuria. By the careful use of the centri-

fuge no casts are found. I examined a man for life insurance, in whom there were no symptoms pointing to kidney disease; there was an albuminuria present. No casts were found after careful examination in several instances. One might suppose it to be a case of temporary albuminuria. I could not possibly recommend the man in spite of the fact that he gave no symptoms at all.

Dr. BABCOCK of Illinois—I should like to emphasize a single point in the differentiation between primary cardiac and primary renal cases; it relates to pulse tension. In these cases of primary renal disease there is an increase in the blood pressure which will have existed for a greater or less length of time, and if the pulse be carefully studied both by the sphygmograph and by careful auscultation of the aortic second sound much valuable information will be derived. In the case reported it was distinctly said that the primary examination of the heart showed an accentuation of the aortic and pulmonic second sounds. It seems to me that that statement was important. At the postmortem it was shown that the heart had been diseased for a long time, although the patient suffered from renal changes, as has been stated; perhaps there was a chronic diffuse nephritis which was recovered from; still the heart was the organ primarily at fault.

I have under observation a man aged 65 years who has an enlarged heart, who has renal changes, whose urine shows evidences of chronic interstitial nephritis. The arteries are also sclerosed. It has been a difficult matter for me to determine which organ was the one demanding treatment. The accentuation of the aortic second sound, the pulse tension and the fact that the heart endures exercise, also that the amount of urine responds pretty well, *i. e.*, the amount of the urine increases but the solids of the urine do not, these findings make me sure that in this man's case the heart is not the organ primarily affected. The heart is secondarily enlarged in response to the abnormal pulse tension which has existed for years. The treatment in such cases, to be successful, must be directed to the general condition largely, although if the heart is disproportionately weak, then this organ demands close attention and the greatest care.

Dr. ANDERS of Philadelphia—The subject of Dr. Billings' interesting paper is a large one and I simply would like to refer to one or two aspects of the same. The differential diagnosis, as drawn by the author of the paper, readily permits a differentiation of chronic parenchymatous nephritis and organic disease of the heart. Closely with the relationship between the heart and kidney disease stands another condition, namely, arterio-sclerosis. I should like to emphasize the fact that in cases of so-called interstitial nephritis in which lesions of the heart are associated they are from one common cause. One primary fundamental cause is general arterio-sclerosis. Now, the troubles with the kidneys are secondary to the arterio-sclerosis; the same is true of the cardiac condition. In either case compensation ruptures and the left ventricle begins to weaken. In the case reported by Dr. Billings this occurred; the left ventricle was already weakened and so the phenomena reported appeared secondarily. This exists in cases in which primary condition of arterio-sclerosis is followed by rupture of compensation. Few are seen for the first time until after compensation has ruptured; here the presence of organic murmurs can be differentiated from murmurs due to primary valvular disease.

Dr. BILLINGS—I chose this subject because it was very interesting to me, partly because of the difficulty in the diagnosis of these cases, as already has been stated in the paper. It is quite important that a differentiation should be made. A man with an incompetent heart due to intrinsic heart disease, in some cases with a water-logged condition, may be given a hope of prolongation of life. In kidney disease do not forget that when the disease of the kidney pro-

gresses to such a stage that the heart becomes incompetent and the cardio-vascular changes are sufficient to produce this water-logged condition, the patient can not long exist. It is true that therapeutic tests may be of value, but the therapeutic test is like the dead-house test. A man with kidney disease with changes in the heart and blood vessels, who is in a water-logged condition, does not have perfectly normal urine, as can be proven. If a nephritis occurs and continues long enough to have cardiac changes, incompetent heart, bronchitis, general anasarca, etc., he may have hemorrhages in the brain and elsewhere. There has been a condition mentioned in which arterio sclerosis, hypertrophy of the heart and sclerosis of the blood vessels, and other evidences of Bright's disease occurred and yet no Bright's disease. I have tried to write my findings of the case, and also stated in my conclusions that in kidney disease the arterial sclerosis was marked and the pulse full and sustained. I am gratified that the paper has received the amount of discussion it has.

DIABETIC GANGRENE.

Presented to the Section on the Practice of Medicine at the Forty-ninth Annual Meeting of the American Medical Association, held at Denver, Colo., June 7-10, 1898.

BY N. S. DAVIS, JR., A.M., M.D.

PROFESSOR OF PRINCIPLES AND PRACTICE OF MEDICINE AND OF CLINICAL MEDICINE, NORTHWESTERN UNIVERSITY MEDICAL SCHOOL, ETC.

The following cases of gangrene associated with diabetes which have been observed by me during the last few years, inspired me to look through the rather meager literature of the subject with care.

J. G., 62 years old, was a ruddy and hearty Englishman, who was habitually careless and negligent of himself and his health. In July, 1892, I found that he was a diabetic. As he promptly improved, gaining in strength and flesh, he did not persevere with the prescribed treatment. He was not seen a second time until September. The last joint of the middle finger of his left hand was then purplish red, swollen and painful. The skin on the ball of the finger was hard, slightly wrinkled and almost black. Pain was constant but not intense. The rest of the finger looked normal except at the first joint a red line of demarcation was discernable. This finger had not been scratched or injured. The gangrene was evidently spontaneous. His urine contained 1.3 per cent of sugar and had a sp. gr. of 1032. A week later he voided 2 quarts of urine, a quart less than before. It contained only a trace of sugar. Sensibility was entirely wanting in the last phalanx of the affected finger except at the sharply defined line of demarcation. The last phalanx was blue-black and perfectly mummified. Pain gradually disappeared. By the 24th of September the quantity of urine voided was normal, having a specific gravity of 1028 and containing no sugar. Sugar did not reappear until the end of October, when it occurred in 1.3 per cent. The urine had a specific gravity of 1031. For several days prior to this examination he used farinaceous food freely. When he again adhered to a moderately rigid diabetic diet, sugar disappeared from his urine. The last phalanx gradually underwent spontaneous amputation and separated completely from the healthy tissue the first of November. The stump healed nicely. I saw him occasionally during the next three months. He gained in flesh, strength and vigor. There was no return of glycosuria, although in January he suffered from a mild eczema of prepuce and scrotum. This man had tortuous temporal arteries and nodulation of the left radial. A sphygmographic tracing showed the low round-topped waves produced by atheromatous arteries. When his finger was first found to be gangrenous amputation was advised, but he refused to submit to it, and when he learned later that spontaneous amputation might take place, was still more unwilling to have the knife used.

B. J. B., 62 years old, a lawyer of prominence in one of the smaller cities of Michigan, consulted me in November, 1894. He first learned that he had glycosuria in 1892, when he was examined for life insurance. He was a large eater, moderate drinker, and eminently sedentary in habits. Ten years before he weighed 200 pounds; when he was first seen by me he weighed 177. For two years he had not varied more than two or three pounds from this weight. He felt in good health, except that for some months there had been an ulcer on a callous

spot on the ball of his right great toe. An offensive slough of small dimensions had separated from it. When I saw it healing had begun. There was a shallow ulcer in the center of a yellowish callous. Almost no pus came from it. It caused no pain and was only tender when the raw flesh was touched. The toe appeared healthy. A sample of his urine had a specific gravity of 1040 and contained 2.5 per cent. of sugar. His arteries were hard but not nodulated. His heart was not hypertrophic.

I did not see this patient again until September, 1895. The ulcer under his toe never healed entirely. Several times it seemed almost well but became worse again. Alongside of the callus on which the ulcer was located was a superficial blister filled with bloody serum. He had recently had a similar blister on the sole of the foot. Both had come without recognizable cause. His urine contained a trifle less sugar. His general health was good; he weighed 180 pounds.

The next visit I received from him was in June, 1896. The ulcer remained unhealed. It was sometimes scabbed over and at others was covered by a small amount of pus. Its diameter varied from one-third to one-half of an inch. A sample of his urine contained 3 per cent. of sugar. In October of the same year, this patient was seen again. He thought his toe was well until about the first of September, when he was much worried by business cares and lost flesh, the ulcer opened again, and in a symmetrical place upon the left big toe a large blister formed. When the latter was opened and the dead overlying skin removed, a considerable excavation with raised edges and suppurating surface was disclosed. I did not see him again for a year, until last October. Both ulcers were about the same. The whole of the left big toe was purplish and slightly swollen. It was moderately tender and painful. In other ways he felt well. A sample of his urine had a specific gravity of 1033 and contained 2 per cent. of sugar.

This patient was seen by me at such long intervals that he can not be said to have had systematic treatment. Moreover, he was not abstemious as regards food, drink or tobacco. He kept the affected toes as clean as possible, and applied successively a great variety of antiseptic salves and powders. He had persistent glycosuria, but of very moderate grade of severity. He rarely made more urine than was normal, and the diabetic state caused no appreciable loss of flesh or ability for mental work. This case is interesting because of the character and chronicity of the ulcers, and finally, because of the symmetrical distribution of the ulcers upon both feet. When last seen by me, I feared the whole of the left great toe was about to become gangrenous.

Katherine E., 58 years old, born in Ireland, was first seen by me in July, 1895. For five months her right foot pained her. I found the toes and, to a less extent, the distal third of her foot reddened and painful. The pain ceased almost completely whenever she remained on the bed or lounge. There had been slight suppuration about one toe nail, and a superficial ulcer on one toe joint. Both these lesions had disappeared before I examined her foot. She had lost flesh during several months. She had never been stout, but was decidedly thin when she came to me. Her appetite was poor. She experienced no thirst. Her bowels were constipated. As far as she had observed her urine was normal. A week later the foot was less swollen and painful, but the toe next to the little one was numb, purplish red and looked as if about to become gangrenous. A sample of her urine had a specific gravity of 1028, and contained 1.5 per cent. of sugar. Before the end of August the last two phalanges of this toe were mummified. A line of demarcation developed at the second joint. On the toe next to the big one a moist slough formed over a space a quarter of an inch in diameter. She was making from three to four pints of urine daily. The glycosuria rapidly diminished and by the first of September ceased. Her foot remained about the same, causing varying degrees of pain. Slowly the mummified toe separated from the healthy tissue. The middle of December it was attached only by a few shreds of tissue, which separated with a little traction. The ulcer on the other toe was slowly improving. The stump of the amputated toe soon healed perfectly. A trace of sugar was discoverable in her urine at this time. In January the ulcer on the second toe grew worse, although there was no glycosuria. By the first of February it had opened into the joint. Toward the end of the month I picked from the opening a small piece of dead bone. After this the wound slowly healed. In March, the following month, a gangrenous slough formed on the ball of the great toe of the same foot. The whole foot from the instep to the toes was a dull purplish red. The toes were slightly swollen. The capillary circulation was extremely slow in the skin of the foot. Pain was not constant or considerable. Her urine contained a trace of sugar. During the summer she was comfortable, able to work and rarely troubled with pain in her