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Evaluation of a prognostic index in diabetic vascular disease

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EVALUATION OF A PROGNOSTIC INDEX IN DIABETIC VASCULAR DISEASE

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An attempt is made to evaluate a prognostic index for patients suffering from diabetic vascular disease. This is an index which rehabilitation and placement service personnel can apply for assessing whether any individual presenting himself for service should be given extensive rehabilitation services. It is realized that in many instances the life expectancy of individuals who have suffered extensive vascular change through disease is not great.

A prognostic index of this type was originated by a "Sub-Committee on Special Problems of the Diabetic Blind," working under the auspices of the United Community Services Committee for the Study of the Blind in Omaha, and by extension, Nebraska. Mrs. Gus Wickstrom is chairman of this committee and David Majors, Secretary. Members of this sub-committee are as follows:

Morris Margolin, M.D., Chairman
Frank L. Eagle, M.D.
Gordon E. Gibbs, M.D.
Charles R. Hankins, M.D.
Mary Jo Henn, M.D.
Robert P. Heaney, M.D.
Robert A. Slabaugh, M.D.
Dwight Frost, M.D., Consultant on Rehabilitation
Mrs. Harry F. Schneiderwind, Executive Secretary, Nebraska Diabetes Association
Mr. Harry Hines, Director of Nebraska State Services for the Visually Impaired - Ex Officio
Mr. Robert A. Kimball, Social Worker for the Blind
Mrs. Gus Wickstrom, Chairman of the Committee of the Whole.
David Majors, Secretary for the Committee of the Whole.

This committee was charged to investigate the unique and special problems presented for the people who have diabetes mellitus and who are visually handicapped, and to investigate the service problems involving that group.

This prognostic index was originated because of testimony from the Nebraska State Services for the Visually Impaired that the diabetic blind presented an unusually large percentage of their active case load, and that rehabilitation for such persons was quite difficult.

Before a diabetic, inflicted with blindness or other complications due to his disease, can be accepted for rehabilitation, the service involved must carefully evaluate this patient as to his medical status, vocational experience, personal and social history. The assimilation and evaluation of this data may mean the difference between success and failure in his rehabilitation.

To assess the personal and social information for purposes of sound planning toward vocational rehabilitation, the answers to such questions as the following must be evaluated:

1. In the light of the patient's work history, what are his prospects for vocational rehabilitation?
2. What can the patient do in view of the resources available, either as immediate placement or training?
3. Is the patient physically and mentally ready for vocational rehabilitation? (1)

It is this third question which the rehabilitation and placement personnel find the most difficulty in evaluating. The fundamental problem is that the rehabilitation and placement service personnel have no sufficient criteria for assessing whether any individual should be given extensive rehabilitation services, realizing that in

many instances the life expectancy of a patient with extensive vascular change may not be great.

One could say that it is not morally, socially, or economically wise to subject a patient, his family, and the state's resources to extensive rehabilitation only to have the patient die within a few months after acquiring a new vocation.

It was brought out to the committee that there had probably never been conceived any real method of evaluating each individual, medically, to determine whether that person should benefit substantially from such rehabilitation. Therefore, the committee assigned itself the task of evolving such formula as could be brought into existence to begin coping with the identified lack of evaluative criteria.

Each member of the committee analyzed a different facet of the problem and through such analysis it was found that there were indeed persons who should be given rehabilitation services who could, through proper controls, extend their active years, find and continue employment.

Dr. Hankins undertook to devise a survey form that could be used in gathering the proper information needed to determine, with some degree of accuracy, the extent of vascular change of an individual, and thereby render more exact an evaluation of his life expectancy. The following survey form was thereby adopted:

"REHABILITATION SURVEY FOR THE DIABETIC PATIENT WITH VISUAL IMPAIRMENT

NAME ADDRESS

Marital status Children Date of birth

Education Occupation Employed?

Date of onset of Diabetes:

Insulin? 1) Type 2) Dosage

Oral hypoglycemic agent? 1) Type 2) Dosage

Diet: 1) Measured 2) Qualitative only 3) Free

Degree of control:

1) Excellent 2) Good 3) Fair 4) Poor
5) Very Poor

Brittle?

Frequency of hypoglycemic reactions

Severity: Mild Moderate Severe

Renal Function:

1) Albuminuria 0 1+ 2+ 3+ 4+

2) Edema? a) Yes b) No

3) Blood pressure: ____/____ on Medication? What?

4) BUN mg% or NPN mg%

5) Creatinine mg%

6) Total serum protein: gm% A/G

Cardiac / Peripheral Vascular Status:

Neuropathy:

Disabling? a) Yes b) No

Any associated disabling disease that will alter general prognosis or ability to undergo a training program:

Date_____ _____ M.D."

Dr. Heaney, using the Hankins Survey Criteria, drafted a formula to reduce the data, that could be obtained, to a simple index that could be referred to for evaluating potential for rehabilitation:

$$2(\text{serum creatinine} - 1) \quad \text{or} \quad 2\left(\frac{\text{blood urea nitrogen} - 15}{15}\right) \quad \text{or}$$

$$2\left(\frac{\text{non-protein nitrogen} - 35}{35}\right) \quad \text{plus} \quad \left(\frac{\text{diastolic blood pressure} - 10}{10}\right)$$

$$\text{plus (degree of albuminuria) plus} \quad \left(\frac{\text{duration of diabetes}}{10}\right)$$

$$\text{plus} \quad \left(\frac{\text{degree of control}}{2}\right) \quad \text{plus (degree of cardiovascular disease)}$$

plus (degree of cerebral vascular disease)

Some checking with actual case histories was done from various data existing from medical records of persons falling into the category under scrutiny. The committee felt that sufficient evidence was gained by this process to justify the conclusion that the data prescribed as "needed," and the formula for reading the data, was ready for usage on an experimental basis.

After re-evaluation and critical analysis, the committee agreed upon an index. It was found that in the initial checking of the formulated information, of case histories of persons now deceased, that the data analyzed, under the formula recommended, gave whole numbers that would indicate the following:

a. Any number up to six should be acceptable for extensive rehabilitation because these patients could be expected to be active many more years.

b. Any patient receiving a number between six and ten should be acceptable for rehabilitation with reservations, the reservations being that the likelihood of the patient's disease being in poor control, after the acceptance for rehabilitation, is not great.

c. Above the number ten, rehabilitation programs are not recommended because the indications would be, that vascular change had occurred so extensively, that ability to continue working after rehabilitation would be highly unlikely.

After this evaluation of the formula, the committee then recommended that the formula be used on a trial basis by the Nebraska State Services for the Visually Impaired. The formula was subsequently adopted by this agency for the selection of cases for rehabilitation. It was also recommended that further evaluation of the index be made, based on case studies at the University of Nebraska and Creighton Medical Schools.

If the formula is broken down into component parts, the significance of each factor can be shown:

2(serum creatinine -1): Creatinine is a metabolic product derived from muscle creatinine phosphate and is excreted primarily by the kidney, which clears the plasma of creatinine at approximately the glomerular filtration rate, which provides the basis for a good clearance test. Normal values for serum creatinine vary from .6 to 1.3 mg. per 100 ml. Elevated concentrations occur only when renal function is impaired by factors intrinsic or extrinsic to the kidney. A concentration

of greater than 1.5 mg% indicates impairment of urine formation or excretion. A rise in serum creatinine can be assumed to be due to diabetic nephropathy in a diabetic, if other causes of increased serum creatinine are eliminated.

Patients in the less than forty age group, who have had diabetes mellitus for more than twenty years, almost never escape the development of intercapillary glomerulosclerosis, and they suffer from renal arteriosclerosis associated with chronic glomerulonephritis and hypertension much more frequently.(2) The usual progress of intercapillary glomerulosclerosis appears to be slow. It is associated with arteriosclerosis, and frequently with pyelonephritis. In the early stages the condition can be suspected because of albuminuria. It causes about thirty percent of the total number of deaths of young diabetic patients who have had the disease for more than fifteen years.(3,4) The clinical manifestations of the disease include diabetes of long duration, albuminuria, leg edema, arterial hypertension, and anemia.(5)

Diseases such as glomerulonephritis, chronic pyelonephritis, amyloid disease, acute renal failure following traumatic or toxic injuries, sulfonamides, congestive heart failure, salt and water depletion, shock and obstruction of the urinary tract must be ruled out as a cause for the elevated serum creatinine.

The same type of prognostic information, derived from the serum creatinine, concerning renal function can be obtained by either the blood urea nitrogen or the non-protein nitrogen. The serum concentrations of urea nitrogen and creatinine tend to rise in parallel fashion,

in kidney disease, when the protein intake and catabolism are constant. If one uses the BUN as an index of pathological conditions, accelerated protein catabolism as a cause of its rise must be ruled out. One can therefore see that if a patient had a normal BUN or creatinine, he would receive a zero, indicating that his kidney function was adequate and probably not significantly involved with kidney vascular alterations.

$\frac{\text{(Diastolic BP-80)}}{10}$: Elevated blood pressure, when used as a part of the prognostic index in the diabetic patient, may mean peripheral vascular disease and/or diabetic nephropathy. All the blood vessels of the diabetic patient appear to be susceptible to premature aging and degeneration; this is reflected in the fact that among diabetics the death rate from cardio-vascular and renal disease is twice that of the general population.(6)

Vascular disease is by far the most common serious complication of diabetes mellitus. Atherosclerosis of medium sized arteries is the largest single cause of deaths among diabetic persons.(7) The most important clinical effects are caused by involvement of the coronary and the cerebral arteries, the arteries of the legs, the arterioles of the kidney, and the capillaries of the glomeruli and the retina.(8)

Diastolic blood pressure is used as an index of prognosis rather than systolic pressure, because the pathology involved is such that an elevation of the diastolic pressure is more significant of complications. The pathology is an atherosclerotic process involving the intima whereby subendothelial accumulations of hyaline are laid down in the arteries followed by calcium deposition, thus causing a narrowing of the lumen. The narrowed lumen will cause an elevation of the diastolic rather than

systolic pressure.

Although diastolic pressure is used as an index for complications, the systolic pressure may be elevated by arteriosclerosis. The incidence of arteriosclerosis is increased in the diabetic; thus it can be seen that both diastolic and systolic pressures may be elevated. A patient with a "normal" diastolic pressure of 80 mm Hg would get an index number of "0", while a patient with a diastolic pressure of 100 mm Hg would get a "2", indicating vascular or kidney involvement.

Degree of albuminuria: The prognosis of diabetic patients with proteinuria is usually held to be poor. Some years ago, the general opinion was held that persistent proteinuria in a diabetic was a benign condition and seldom lead to disturbances of renal function(Noorden and Hatlehol, 1951), but in the light of present knowledge, this can no longer be maintained. It appears that persistent proteinuria in a diabetic,which is not due to some coincidental complication, ultimately leads to renal insufficiency and uremia, provided the patient lives long enough.(9)

Albuminuria is probably due to increased permeability of the glomerular filter. The permeability of the glomerular filter seems to be increased by any factor tending to cause local or generalized asphyxia. Albuminuria is a good indicator of kidney disease, and is probably the first sign seen in diabetic nephropathy, although other causes of albuminuria must be ruled out, e.g., tuberculosis, amyloidosis, nephritis, febrile conditions, drugs, etc.

F. I. Caird recently analyzed 134 diabetics which were followed for up to ten years from the onset of proteinuria. Their survival was compared with estimates of that expected from the general population and of all diabetics. Sixty-five percent survived five years and 28% ten years from the onset of proteinuria. This represents 77% of the natural expected rate for five years, 44% for ten years, 89% and 59% respectively of the diabetic expected rate. He concluded that the prognosis for life with proteinuria is better than has been earlier believed.(10) However, the presence of proteinuria is a sign of nephropathy and leads to a decreased survival.

O'Sullivan, Fitzgerald, Blainey, and Malins reported on 100 patients with proteinuria and found that 82 had diabetic nephropathy, 73 with nephropathy alone, and nine with nephropathy and urinary tract infection. Renal biopsies done on a number of patients revealed that the histological changes of diabetic nephropathy were usually more advanced than the clinical picture suggested.(11) I feel this is good evidence that proteinuria is a reliable indication of diabetic nephropathy and its severity. Albuminuria, for index purposes, is graded one through four and the number placed directly in the formula.

Duration of Diabetes: For many years now it has been apparent that the frequency of vascular disease increases directly with the duration of diabetes.(12) Duration of diabetes is the most important single factor in the etiopathogenesis of vascular degeneration. This has been demonstrated by White and Waskaw, in their series of 200

patients in whom diabetes developed before the age of 15 years and who survived more than 20 years with the disease, nephropathy was present in 50%, calcified arteries in 75%, and retinal hemorrhages in 80%. Paul and Presley found retinopathy in 45% of patients who had juvenile diabetes of fifteen or more years duration. Vascular degeneration in long standing diabetes varies from 60% to 83% of patients. Complications due to diabetic vascular disease seldom occur before the tenth year, but are almost always present after the twentieth year. The prognosis is not altogether unfavorable. Although vascular lesions almost always are present in persons who have had diabetes for a long time, they are not always severe. (15)

This component of the index takes into account the likelihood of increasing complications as the patient increases the duration of his disease. It can be seen that after the tenth year of the disease, the patient's prognostic index will increase .1 point every year.

Degree of Control: "Most authorities feel, although there is not unanimous agreement, that the development of complications is directly related to degree of control of the diabetic patient." (16)

Much evidence has been brought forth in the past decade to support the view that the degenerative process is proportional to the degree of faulty control. A most recent article based on 391 cases comes from England (J.A.M.A. 173, 1783-1788, Aug. 20, 1960). The authors, basing their studies upon the presence of proteinuria, subdivided their cases in accordance with duration of diabetes and degree of control. With

correlation of these two factors, they found that those under good control to excellent control had either none or minimal proteinuria even after twenty years duration. A number of those poorly controlled developed proteinuria within five years and nephropathy was present in 100%. by the end of twenty years. Although time does have a bearing, control is the most important factor.(17)

Kinsell has suggested that a major factor in the vascular degeneration of diabetics may be in the continuous disturbance of intracellular and extracellular osmotic relationships resulting from extreme fluctuations in blood sugar over months and years. The continuous chemical and physical alterations may be related to the proliferative and degenerative changes in the eyes, kidney, heart, central nervous system, or in the peripheral blood vessels. Relapses and spontaneous remissions in the progression of the vascular disease at irregular intervals could then be explained according to fluctuating blood sugar concentrations.(18)

Severity of diabetes and degree of control are hard to define accurately. For insurance purposes, the diabetic patient is considered under good control if he is on a well balanced diet adequate to support normal weight and actively voids specimens which are sugar free most of the time, and has shown periodic blood sugar levels not above 160 mg. fasting or 250 mg. past prandial by the Folin Wu method.(19)

For index purposes, degree of control takes into account brittleness, frequency of hypoglycemic reactions, adherance to diet, and blood sugars (measured directly or by urine sugar values). Degree

of control is graded excellent - 0, good - 1, fair - 2, poor - 3, very poor - 4.

Degree of Cardio - Vascular Disease: Among diabetics, death from cardiovascular disease is out of all proportion to that of the general population. Coronary thrombosis as a cause of death in diabetics less than forty years of age, is ten times as frequent as in non-diabetics of the same age.(20)

Rogers and Holcomb have recently evaluated the complications of diabetes mellitus. In their study of 114 patients, the principal cardiovascular disorder was arterioscleratic heart disease (ASHD). They found ASHD to be twice as prevalent in the diabetic group as the general population. ASHD was detected clinically in 39% and by autopsy in 5 patients. "The ASHD was manifest by angina pectoris in 11 patients, by myocardial infarction with or without angina pectoris in 12, and by otherwise inexplicable cardiomegaly or congestive heart failure in sixteen patients." Ninety-one percent were older than 50 years when the ASHD became evident; only three patients were younger than 40. The same distribution of ASHD is found among non-diabetics in whom, however, the incidence is less than half as great. The average survival time after clinical onset of ASHD was 5.4 years in the deceased group, and 5.0 years in the 15 living patients whose status is known. Other types of clinical heart disease encountered were hypertensive in four patients, aortic stenosis in two. Mitral valve or luetic lesions were not found. Sustained systolic hypertension (>160) was found in 30%

of the 114 patients and in 35% of those over age 50. There were significant correlations with cardiomegaly (present by X-ray in 58% of the hypertensives, but in only 18% of the whole study group) and with proteinuria (in 64% and 35% respectively). Therefore, systolic hypertension was an unfavorable sign, although there was no correlation with ASHD, diabetic retinopathy, or with leg ischemia.(22)

Bradley and Bryfogle have recently analyzed the survival of diabetic patients after suffering myocardial infarction. They found that the presence of diabetes mellitus has been associated with high mortality from acute myocardial infarction: 60.8% following all attacks, and 57.8% after the first attack. The diabetic with acute myocardial infarction has a prognosis similar to that of "poor risk" cases from the general population. It was found that late survival of diabetic patients after the initial attack of myocardial infarction was also decreased, since fewer than 20% lived five years and only 3.6% for ten years.(23)

Table from Joslin Clinic 1950 - 1956:

Causes of death among diabetics:

ASHD	33%
Other heart disease	15%
Nephropathy	10%
CVA	13%
Gangrene	10%
Diabetic coma	1%
Infection	5%
Other	11%

This part of the index is measured by anginal pain, heart murmur, X-ray, EKG, hypertension, etc., and graded on a one through four basis.

Cerebral Vascular Disease: The term cerebral vascular disease denotes any disease in which one or more of the blood vessels of the brain is primarily implicated in a pathological process. Cerebral vascular disease causes pathological effects of two types: 1) ischemia with or without infarction; and 2) hemorrhage. The incidence of cerebral vascular disease is not increased in diabetes, but it can be seen that any evidence of cerebral vascular disease or disabling neuropathy would have a decided effect on the life expectancy and rehabilitation potential of the diabetic patient. Cerebral vascular disease is graded one through four for index purposes, e.g., a patient with a history of CVA would receive a four and patients with minor ischemis syndromes a lesser number.

In order to demonstrate how this prognostic index can be applied, several case histories of patients who have died of the complications of diabetes mellitus will be presented. In each case the prognostic index will be calculated at various times during the patient's life. By evaluating patients who have died of their disease and by giving them index numbers prior to their death, we can correlate the index number with the remaining years of life.

The following case histories are based on a study of case records from Dr. Morris Margolin and from the University of Nebraska Hospital and Dispensary.

Case 1 - Miss G. P.: This young girl was first seen in 1949 when the patient was 15 years of age.

History: She was a known severe diabetic for 12 years. At this time she was not following her diabetic diet and her disease was under only fair control. She had been taking 65 units of NPH insulin daily. Her chief complaint during this office visit was a "boil" on her right side.

Physical Exam: BP 118/80. U.A. revealed 2+ proteinuria. There was a reddened, tender, inflammed area approximately 3 inches in diameter on her right side. In the center of this area was a large vesicle filled with pus. The remainder of the physical exam was within normal limits.

Therapy: Incision and drainage of carbuncle, antibiotics, insulin and diet adjustment.

The index number at this time is calculated in the following manner:

BUN (not reported) +0 +2 +1.2 +0 +0 +0 = 3.2
(The patient received a 2 for the 2+ proteinuria and a 1.2 for the duration of the disease.)

The patient was next seen in 1953 for evaluation of her diabetes. At this time she was still under only fair control of her disease as judged by urine sugar determinations and frequency of insulin reactions. Physical exam revealed the blood pressure to be 134/88. The U.A. revealed 2+ proteinuria.

Index: BUN (not reported) +.8 +2 +1.6 +0 +0 +0 = 4.4
(She now receives .8 for her diastolic pressure of 88, a 2 for her 2+ proteinuria and 1.6 for the duration of her disease.)

The patient was next seen in June of 1957 with the following additional history: The patient had lived out of the state for the intervening four years. A letter from her physician to Dr. Margolin at this time revealed that the patient had been hospitalized 3 months prior for insulin shock and chronic pyelonephritis. Physical exam was reported to reveal a blood pressure of 180/105, hypertensive and diabetic retinopathy, 4+ proteinuria, and many pus cells in the urine.

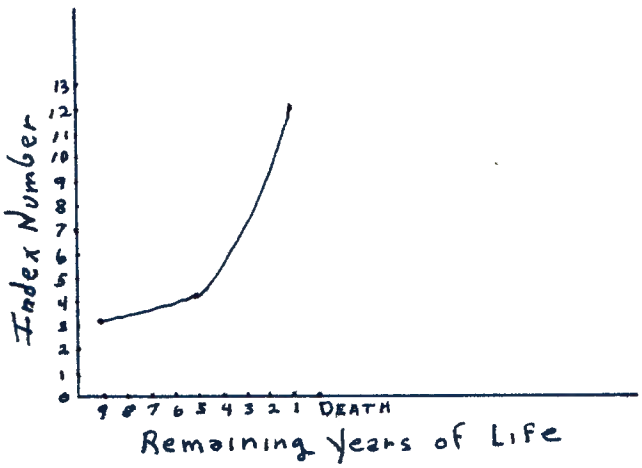
Therapy: Gantrisin®, salt limitation, insulin and diet adjustment.

Index: 1.6 /2.5 /4 /2 /2 /0 /0 = 12.1
(She receives a 1.6 for the BUN elevation, 2.5 for the abnormal diastolic pressure, 4 for her 4/ proteinuria, 2 for the duration of her disease, and 2 for poor control.)

In November of 1957 her records reveal that her control was far from adequate with frequent insulin reactions, glycosuria and failure to take her prescribed medicine. Her condition was essentially the same as in June and her index number remained the same.

In 1958 the patient applied to the State Rehabilitation Service because of retinopathy; however, later in the year she died of uremia.

If we now plot her index numbers against her remaining years of life, the following graph can be demonstrated:



It can be seen that if it had been necessary for her to apply for rehabilitation in 1949, she would have received an index of 3.2 and be deemed a good candidate for rehabilitation. From the graph it can be shown that she had nine remaining years of life. In 1953 her index of 4.4 would have been acceptable and she then had five years of remaining life. If in 1957 the patient had applied for rehabilitation she would have been turned down, and rightly so, for she died within one year.

Case 2 - Mr. V. C.: This patient was first seen in 1943 when the patient was 42 years of age.

History: A known diabetic for five months. His chief complaint on this first visit was drowsiness, weakness, polydipsia and polyuria.

Physical exam: Blood pressure 106/78. The remainder of the physical exam was within normal limits.

Laboratory: U.A. reveals 1/ proteinuria, 3/ glycosuria, blood sugar 250 mg%.

Therapy: Diabetic diet adjustment.

Index: BUN(not reported) /0 /1 /0 /0 /0 /0 = 1

He was next seen in 1944 when it was necessary to start insulin to control his blood sugar. After insulin and diet adjustment, he was asymptomatic with no insulin reactions or excessive glycosuria. His control was felt to be good. Physical exam was unchanged. The U.A. revealed no proteinuria at this time so the patient now receives an index of zero.

1946: Patient evaluated in office and history reveals that he has not been following his diet or taking insulin. Physical exam essentially normal except for blood pressure of 138/90.

Therapy: Insulin and diet adjustment.

Index: BUN(not reported) /1 /0 /0 /2 /0 /0 = 3
(For his diastolic blood pressure rise he receives a 1 and for his poor control a 2.)

1951: On this visit the history revealed very poor control with glycosuria and failure to follow diet. The physical exam revealed a blood pressure of 132/94. The U.A. revealed 3/ proteinuria.

Therapy: Stricter control with insulin and diet alterations.

Index: BUN(not reported) /1.4 /3 /.8 /2 /0 /0 = 7.2
(A diastolic pressure of 94 gives the patient a 1.4, the 3/ proteinuria a 3, the 8 year duration a .8, and the poor control a 2.)

1952: Patient comes to office with history of ankle edema, dyspnea, orthopnea, glycosuria, and poor control as far as diet and insulin are concerned. Physical exam reveals a blood pressure of 150/110, rales in the lung bases bilaterally, a heart enlarged to percussion and lung dullness in the bases. X-ray reveals an enlarged heart and evidence of pulmonary congestion.

Therapy: Digitalis, low sodium diet, rest, insulin and diet adjustment.

Index: BUN(not reported) #3 #3 #1 #2 #2 #0 = 11
(Diastolic pressure of 110 gives the patient a 3, 3# proteinuria reported earlier is 3, the duration of the disease a 1, poor control a 2, and an enlarged heart and congestive failure is given a 2.)

1952: After therapy and better control, the patient was relieved of obvious congestive failure. He did, however, have occasional episodes of ankle edema and was treated with digitalis and diuretics as needed. Blood pressure now 176/108, proteinuria 3#.

Index: BUN(not reported) #2 #3 #1 #1 #2 #0 = 9
(2 for diastolic pressure, 3 for albuminuria, 1 for duration of disease, 1 for fair control, and 2 for an enlarged heart and history of congestive failure.)

This patient was then lost for follow-up, but is reported to have died in 1957.

We can now construct a graph similar to Case 1:

This patient could have been accepted for rehabilitation without reservation prior to 1951. In 1951, the patient received an index of 7.4, placing him in the "acceptable with reservation category." He could have been accepted for rehabilitation at this point if the future prospects for adequate control were good. In this case, the control was poor to fair and it can be seen how the patient developed complications with a resultant rapid increase in index valuation. I think we can look back on this case and say that he would have been a poor candidate for rehabilitation due to his history of poor control. If he had been accepted, his ability to work must be considered in view of his 3/ proteinuria and hypertension, for they probably indicate fairly severe diabetic nephropathy in this instance.

Case 3 - Mr. W. B.: Initially seen in 1952 when the patient was 29 years of age.

History: A known diabetic for 19 years. When the diabetes mellitus was first discovered, the patient had been placed on a diabetic diet and insulin; however, during the past several years he has had no dietary control. At this time, he was taking 10 units of regular insulin twice a day, but was testing his urine only occasionally. For six months prior he had been developing dimness of vision on the right. This difficulty had been diagnosed as cataracts and retinal exudates. There was a history of dependent edema in the evenings.

Physical exam: Blood pressure 190/100. Diffuse enlargement of the thyroid, questionable heart enlargement, and pitting edema of the lower extremities was noted.

Laboratory: NPN 45 mg%. U.A. revealed 3/ proteinuria.

Therapy: Immediate hospitalization to place the patient under control.

Index: $\frac{(45-35)^2}{(35)}$ + $\frac{100-80}{10}$ +3 +1.9 +2 +0 +0 = 9.5

Hospitalization: The patient was brought under control after nine days of hospital care. His diet and insulin were regulated, the patient being dismissed on 40 units of NPH and 10 units of regular insulin in the A.M. While in the hospital, his diastolic pressure ranged from 72 to 112 mm Hg. No antihypertensive drugs were given. NPN 45 mg%. A BMR was taken and reported to be within normal range.

Index after hospitalization: .6 +2 +3 +1.9 +0 +0 +0 = 7.5

1952: Office visit - patient gives a history of frequent insulin reactions when working and much difficulty with control of urine sugar. Blood pressure 188/120. U.A. 3+ proteinuria. Patient advised to reduce insulin while working.

Index: .6 +3 +4 +1.9 +1 +0 +0 = 10.5

1953: Office visit - History reveals that the patient has been having excellent control since the last visit with no insulin reactions and urines, which are clear most of the time. There is, however, a complaint of diminishing vision. Physical exam reveals a blood pressure of 210/128 and pitting edema of the lower extremities.

Index: .6 +4.8 +4 +2 +0 +0 +0 = 11.4

This patient died in 1955.

This is a case of a young man with a long history of poorly controlled diabetes who is beginning to have visual impairment due to vascular degeneration. At the time the patient was first seen his index number of 9.5 was in the range of "acceptable for rehabilitation with reservation." With hospitalization and adequate control, his index number dropped two points, but was still not in the range of being a good candidate for rehabilitation. The two following visits reveal erratic control and, as can be seen, a marked increase in his index number.

If he had presented himself for rehabilitation due to his blindness, he probably should have been turned down due to his long history of poor control. As the graph reveals, he had a limited number of useful years remaining.

Case 4 - Mr. L. M.: Since this patient had a long, progressive course of vascular degeneration, he will be evaluated at frequent times over an 18-year span.

1936: Age 61. History reveals a known diabetic for 17 years. At this time he was asymptomatic and under excellent control. Physical exam revealed a blood pressure of 140/90, but was otherwise within normal limits.

Index: BUN (not reported) ∕1 ∕0 ∕0 ∕1.7 ∕0 ∕0 = 2.7

1943: Essentially no change from last evaluation. Patient under good control and asymptomatic. Urine analysis reveals mostly negative sugars. Blood pressure 130/60.

Index: BUN (not reported) ∕0 ∕0 ∕2.4 ∕0 ∕0 ∕0 = 2.4

1945: A routine office visit for evaluation reveals many pus cells in the urine. Patient asymptomatic. Blood pressure 164/80. U.A. 1∕ proteinuria. Therapy consisted of prostatic massage and Sulfathiazole.

Index: BUN (not reported) ∕1 ∕0 ∕2.6 ∕0 ∕0 = 3.6

1946: History reveals consistent good control. Physical exam shows a blood pressure of 190/92 with negative heart and lung findings. An enlarged prostate is noted. U.A. 1∕ proteinuria.

Index: No BUN reported ∕1 ∕1 ∕2.7 ∕0 ∕0 ∕0 = 4.7

1950: Age 74 years. Patient under good control. Blood pressure 152/68; the remainder of the physical exam is within normal limits. U.A. 1∕ proteinuria.

Index: BUN (not reported) ∕0 ∕1 ∕3.1 ∕0 ∕0 ∕0 = 4.1

1954: Admitted to hospital with persistent heartburn and "shocklike" condition. Patient had been undergood control prior to hospitalization. Physical exam: blood pressure 142/70, U.A. 3∕ proteinuria, NPN 51 mg%, EKG indicates recent posterior myocardial infarction. Therapy: oxygen, anti-coagulants, analgesics, and sedatives. The patient was dismissed after a 19-day hospitalization with the report that he tolerated the infarction well.

Index: $\frac{(51.0 - 35)}{35} \times 2$ ∕0 ∕3 ∕3.5 ∕0 ∕4 ∕0 = 13.5

(The patient is given a 4 for his myocardial infarction.)

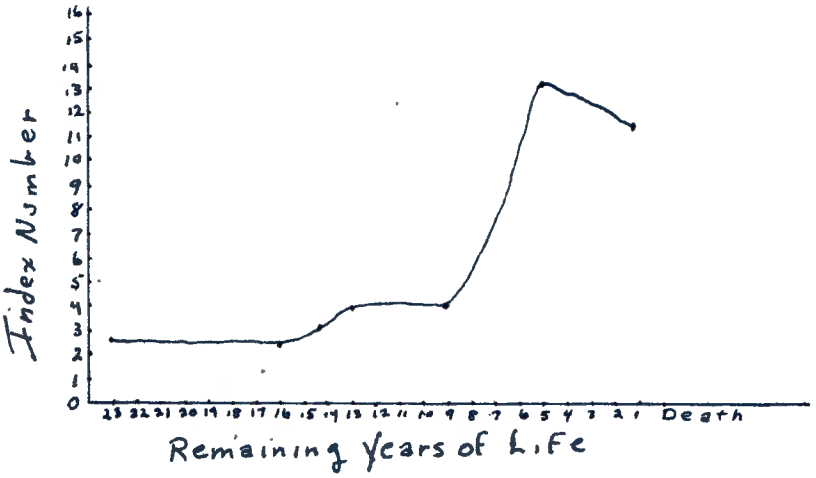
1958: Since the myocardial infarction four years prior, the patient was under good control with insulin and diet management. At this time he did have a complaint of chest pain on exertion. Physical exam within normal limits. Blood pressure 138/60. U.A. 2∕ proteinuria.

Index: 2 ∕0 ∕2 ∕3.9 ∕0 ∕4 ∕0 = 11.9

(No BUN was reported at this time, but the index number of 2 is given here because of the prior recording of 51 mg%.)

1959: Patient died of uremia.

The usual graph plotting remaining years of life against index number is now demonstrated.



This man's case history and resultant graphical picture point out several pertinent points. 1) At the time he was first seen, he had an index number of only 2.7 and, as is shown, he lived 23 years. 2) The patient had a 14-year span after his first visit in which his index rose only slightly, leading one to believe that he had only minimal vascular degeneration. 3) Nine years prior to his death, his index number rose rapidly as a result of his infarction. 4) It is likely that had this patient been a younger man, he would have been acceptable for rehabilitation purposes ten years prior to his death and the service personnel would have had no indication of severe cardiovascular degeneration which resulted in a myocardial infarction during the next year. It is almost impossible to predict myocardial infarctions before they occur and this may be a pitfall of this prognostic index.

Case 5 - Mr. W. P.: This patient was first seen in 1956 at the age of 64 years.

History: A known diabetic for 35 years. Patient had been on 25 units of NPH insulin every A.M., but had been irregular as to diet. His chief complaints at this time were diplopia, nausea, vomiting and glycosuria.

Physical exam: Height 5'6", weight 172 pounds, blood pressure 120/80. Remainder of physical exam was essentially within normal limits.

Therapy: Admitted to hospital. While in the hospital, diabetic retinopathy was discovered with many hemorrhages and exudates. An electrocardiogram revealed left ventricular strain. Due to a complaint of burning in the tips of his toes for the last few years, X-rays of his lower extremities were taken which revealed extensive vascular calcification.

Index: (after hospital workup)

BUN (not reported) ∕0 ∕0 ∕3.5 ∕2 ∕3 ∕0 = 8.5
(2 for poor control and 3 for left ventricular strain, plus peripheral vascular calcification.)

After a ten-day hospitalization, the patient came under good control with insulin and diet adjustment.

Index: (upon dismissal)

BUN (not reported) ∕0 ∕0 ∕3.5 ∕0 ∕3 ∕0 = 6.5

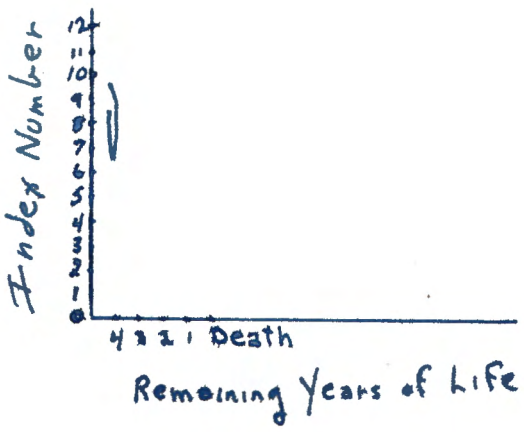
1956: Readmitted to hospital several days following dismissal. Chief complaint was episodes of insulin shock, hyperglycemia with glycosuria, nausea and vomiting. The patient was very capricious about his diet, insulin and activity. He remained in the hospital for seventeen days until his diabetes was again controlled. While in the hospital his diastolic pressure ranged from 80 - 100 mm Hg. A NPN was reported at 40 mg%.

For index purposes, we must give this patient a 2 for poor control because of his past history, even though he left the hospital in a regulated condition.

Index: $\left(\frac{40 - 35}{35}\right)2$ ∕1 ∕0 ∕3.5 ∕2 ∕3 ∕0 = 9.8

This patient died in 1960.

This case report reveals how a patient, by not maintaining good control, passed from the category of "acceptable for rehabilitation" to "acceptable for rehabilitation with reservation." Had this man applied for rehabilitation (this is unlikely at his age) when his index was 5 (or even 3 after hospitalization) he would have been accepted for rehabilitation. Only a few days later he passed into a new category with an index number of 6.3, which would make him acceptable for future rehabilitation only with reservation. With his clinical history of poor control and evidence of left ventricular strain, plus no indication of future prospects for good control, he probably should have been turned down for rehabilitation, and rightly so, for he lived only four years longer. He subsequently died after undergoing two amputations for diabetic gangrene. The standard graph is shown below:



Case 6 - Mrs. R. E.: Initially seen in 1952 at the age of 50 years. This is a case of an eight-year duration of progressive vascular disease so index numbers will be evaluated annually or bi-annually.

1952: History - A known diabetic of 17 years duration. At this time she was out of control with 4+ glycosuria on all tested urine specimens.

Physical exam: Blood pressure 164/92, weight 234 pounds. Remainder of physical exam was within normal limits.

Index: (prior to hospitalization)
0 +1 +0 +1.7 +2 +0 +0 = 4.7

Index: (after hospital control)
0 +1 +0 +1.7 +0 +0 +0 = 2.7

1954: History reveals poor control over preceding two years. Complains of glycosuria on most urine specimens. Physical exam essentially negative. Blood pressure 170/90. U.A. 1+ proteinuria. Therapy: Diet and insulin adjustment with sedatives.

Index: BUN (not reported) +1 +1 +1.9 +2 +0 +0 = 5.9

1956: Patient gives a history of poor control since last index evaluation. Admitted to hospital due to dizziness, sore throat, coughing, ear aches, and mastoid tenderness. Blood pressure 168/110, 2+ proteinuria noted. An EKG revealed no abnormality. Therapy consisted of insulin and diet adjustment, antibiotics and sedation.

Index: BUN (not reported) +3 +2 +2.1 +2 +0 +0 = 9.1

1958: Patient continues to give a history of poor control with many insulin reactions, glycosuria, pneumonia, kidney infection, etc. On an office visit in January of this year the patient complained of dyspnea, orthopnea, weakness cough, polyuria and polydipsia. Physical examination revealed a blood pressure of 206/92 and slight evidence of lung congestion. Urine sugar 4+, 2+ proteinuria. Therapy: Insulin and diet adjustment, antihypertensives, and digitalis.

Index: (No BUN reported) +1 +2 +2.3 +2 +2 +0 = 9.3
(The patient receives a 2 for evidence of congestive heart failure.)

1958: Several months after the last evaluation, the patient was again admitted to the hospital for severe left epigastric pain. Physical exam revealed a left inguinal hernia. Barium enema and electrocardiogram were non-contributory. BUN 25 mg%. Therapy consisted of herniorrhaphy under local anesthesia, insulin and diet management, digitalis, and antihypertensive drugs.

Index: $\frac{(25 - 15)}{15}2$ #1 #2 #2.3 #2 #2 #0 = 10.3

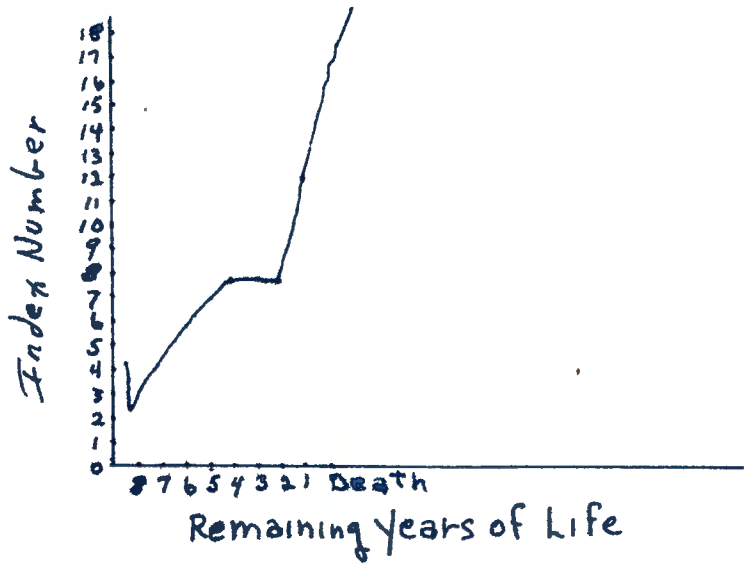
1959: Patient was admitted to hospital twice during 1959 for numerous complaints, e.g., abdominal pain, extremity pain, diarrhea, kidney infection, etc. Blood pressures during the year ranged from 200/88 to 200/110. The pressure could be controlled with antihypertensive drugs, but the patient was not faithful in taking the drugs. Blood urea nitrogen 25 mg%, U.A. 4+ proteinuria.

Index: 1.6 #1.5 #4 #2.4 #2 #2 #0 = 13.5

1960: Patient was discovered unconscious with resultant left arm and leg numbness and distorted speech. The left arm and left leg subsequently became paralyzed and a diagnosis of cerebral thrombosis was made. Blood pressure 118/112, BUN 41 mg%.

Index: $\frac{41 - 15}{15}$ x 2 #3 #4 #2.5 #2 #2 #4 = 20.5

This patient died in August 1961.



This case history exemplifies a steady progression of vascular degeneration cummulating in a cerebral vascular accident. Prior to 1954, (or only six years prior to her death) she would have been acceptable for rehabilitation purposes. She had a steady rise in her index number until 1956, at which time she reached a valuation which was near "unacceptable for rehabilitation." In 1958, the index rose to 10.3 and, as can be seen from the graph, she lived only three more years, dying in 1961 after a CVA. Had this patient been given rehabilitation in1952, when her index was at the lowest point, she would have lived only nine more years and, as pictured by her case history, a rather stormy period indeed. In 1954, with an index of 5.9, she should probably be viewed as being in the "6 - 10" category due to her history of poor control. I suspect the wisest course would have been to refuse this patient rehabilitation services from 1954 on, due to her history of poor control, disabling neuropathy, and rapid rise in index number from 1952 to 1954.

Case 7 - Mr. H. D.: Initially seen in 1959 at the age of 59 years.

History: A known diabetic of 32 years duration with a history of difficult control. Presently on PZI insulin, 65-70 units per day. Frequent insulin reactions occur between 3:00 and 6:00 A.M. His chief complaint on this visit is loss of vision beginning five years prior with almost total loss of vision at this time.

Physical exam: Blood pressure 142/80. The left eye reveals a scar in the macula with a few aneurysms. A vitreous hemorrhage was present in the right eye and the fundus was not visualized. The dorsalis pedis pulsation was not present in either foot and the legs sufussed a dependant position.

Laboratory: U.A. 1/ proteinuria, 3/ sugar, blood cholesterol 360 mg%, NPN 35 mg%, EKG read as coronary insufficiency.

Index: $\frac{35 - 35}{35} \times 2$ /0 /1 /3.2 /2 /3 /0 = 9.2

(Coronary insufficiency plus absent pedal pulses accounts for the three in cardiovascular status.)

Therapy: Admitted to hospital for control of diabetes. After a seventeen-day course of hospitalization, the patient came under good control and was dismissed on 45 units Lente in A.M. and 20 units Semi Lente insulin in the P.M.

Index: 0 /0 /1 /3.2 /0 /3 /0 = 7.2

Later in the year, the patient was again admitted to the hospital with the chief complaint of constipation. At this time, the physical exam revealed cardiac enlargement, venous congestion, and evidence of fluid in the lung bases bilaterally. Blood pressure 124/64. The twenty-day hospital course was characterized by very poor control with episodes of hyperglycemia, insulin shock, signs of congestive failure, and kidney infection. The patient was treated with antibiotics, digitalis, and diuretics.

Index: BUN(not reported) /0 /1 /3.2 /2 /4 /0 = 10.2

(2 for poor control and 4 for coronary insufficiency, absent pedal pulses, congestive failure and an enlarged heart.)

1960: Re-entered hospital because of new hemorrhage in left eye. History since last evaluation reveals excellent control. Blood pressure 168/88. An electrocardiogram reveals digitalis effect, but no coronary insufficiency at this time. The patient was hospitalized for seven days, during which the control was excellent.

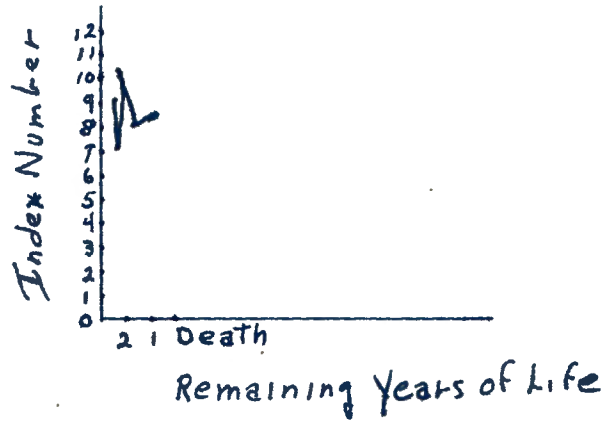
Index: BUN (not reported) /0 /1 /3.2 /0 /4 /0 = 8.2

(History of coronary insufficiency by EKG, but none at this time, gives patient a 4.)

1961: Hospitalized again with chief complaint of a painful right foot. A history of pleural effusion and thoracocentesis was elicited. Physical exam revealed an edematous, inflammed and tender foot. Laboratory: Serum creatinine normal. EKG was compatable with a diffuse non-specific type of myocardial infarction or ischemia. A chest X-ray revealed an enlarged heart, with fluid accumulations in the chest. Therapy: diuretics, demerol, Buergers exercises, and transfer to University Hospital.

The patient subsequently died in University Hospital with the diagnosis: 1) Diabetes mellitus. 2) Myocardial infarction. 3) ASHD. 4) Arteriosclerosis obliterans.

Index: 0 /0 /1 /3.3 /0 /4 /0 = 8.3



This patient exemplifies how careful control can lower one's prognostic index. However, at no time did the patient's index number fall below seven. With this man's clinical history, he would have been a poor candidate for rehabilitation on the basis of the index, and rightly so, for he lived only two years.

Case 8 - Mrs. B. S.: Initially seen in 1947 at the age of 59 years.

History: A known diabetic for 10 years, currently on a diabetic diet and PZI insulin. Complaints on this visit were anorexia, weight loss, polydipsia, polyuria, and painful, numb legs and feet. Systemic review revealed blurring vision, frequent colds, back pain, palpitation, and history of a cholecystectomy in 1940.

Physical exam: Blood pressure 148/80, weight 200 pounds, height 5'4". Bilateral flank tenderness and somewhat diminished deep tendon reflexes were noted. The fundi were not remarkable.

Laboratory: Sugar 2/, proteinuria trace, U.A. loaded with pus cells.

Diagnosis: 1) Diabetes mellitus 2) Pyelonephritis

Index: BUN (not reported) ∕0 ∕0 ∕1 ∕2 ∕0 ∕0 = 3

1948: Control since last index far from adequate. The patient frequently stops insulin, does not follow diet and apparently disregards physician's instructions. The pyelonephritis is apparently chronic and requires antimicrobral therapy frequently. Complains of dimness of vision, leg and foot pain. Physical exam reveals a blood pressure of 138/62, slight edema of feet, and injected conjunctiva. All peripheral pulses are palpable.

Index: BUN (not reported) ∕0 ∕0 ∕1.1 ∕2 ∕0 ∕0 = 3.1

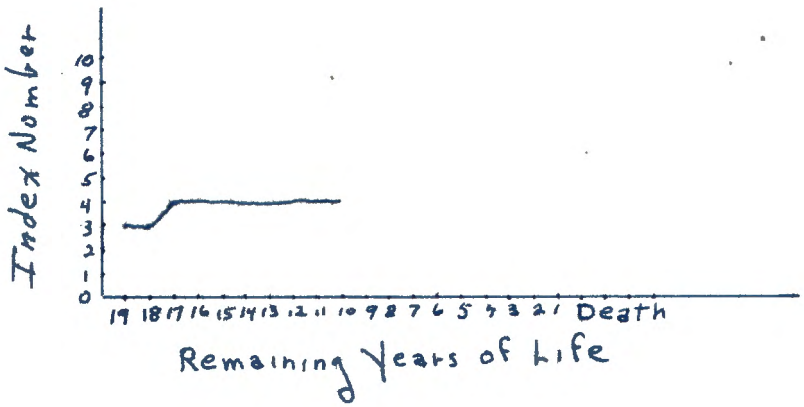
1949: Admitted to hospital because of back ache, labored respirations, nausea and vomiting. Physical exam: blood pressure 110/70. An increase in respiratory rate, increase in breath sounds and crepitant rales were noted. Laboratory results reveal a 1∕ proteinuria leukocytosis and left shift. Diagnosis: 1) Pneumonitis and pyelonephritis. Therapy: antibiotics with insulin and diet adjustment.

Index: BUN (not reported) ∕0 ∕1 ∕1.2 ∕2 ∕0 ∕0 = 4.2

1950: Readmitted to hospital. History reveals poor cooperation in relation to diet and erratic insulin therapy. She complains of epigastric pain, backache and dysuria. Physical exam reveals tenderness over the gall bladder. X-rays reveal cholelithiasis. Urine sugar 4∕, proteinuria 1∕. Therapy: antibiotics, analgesics. Surgery refused.

Index: No BUN reported ∕0 ∕1 ∕1.2 ∕2 ∕0 ∕0 = 4.2

Patient died in 1959 of a myocardial infarction post-operatively.



This patient is somewhat of a paradox for she exhibited consistent poor control and numerous complaints over the years, but as can be seen from her graph, she held a steady index number for nine years. She was lost to follow-up in 1950, but is known to have died in 1959. Possibly, if we had the date, we could apply the prognostic index after 1950, and demonstrate a marked increase in vascular degeneration. I think it is significant that the patient lived 19 years following the initial index number of 3.

Case 9 - Mr. A. F.: Patient was diagnosed as having diabetes mellitus in 1942, when he was 57 years of age.

History: Patient entered University Clinic with the history that he fell on his right hip. One week following, he began to have pains in the low back and abdomen. Systemic review and past history were non-contributory.

Physical examination: Blood pressure 160/90. Abdominal exam reveals hepatomegally and questionable tenderness in the right lower quadrant. No back or flank abnormalities were noted.

Laboratory: U.A. 3/ proteinuria.

Diagnosis: Diabetes mellitus.

Therapy: Diabetic diet.

Index: No BUN /1 /0 /0 /0 /0 /0 = 1

1943: Patient had numerous visits to University Clinic during year for apparently minor aches and pains, but diabetic control was described as good throughout. The patient was following his diet and was now on 5 - 10 units of P.Z.I. daily.

1954: In the interim of 1943 - 1954, the patient was seen privately by Dr. Margolin.

History: A known diabetic for 12 years, taking 20 units of Lente insulin daily and following diet. Patient denied any

insulin coma or diabetic acidosis. The chief complaint on this visit was pain in the anterior portion of the right chest and lumbar back pain for seven months. Systemic review reveals difficulty in lifting legs when walking, weakness, and difficulty in maintaining his balance in a darkened room.

Physical examination: Blood pressure 150/80. The right eye was aphakic, the left eye normal. The fundus of the left eye revealed A-V nicking, but no exudates or hemorrhages. The heart demonstrated infrequent premature contractions. Back and extremity exam normal. The neurological exam revealed slight weakness of grip left hand, abnormal finger-to-nose test with the eyes closed, a positive Romberg, and negative ankle clonus on the left.

Laboratory: Negative for proteinuria. X-ray revealed degenerative arthritic change involving the cervical and dorsallumbosacral spine. EKG - possible left heart strain.

Diagnosis: 1) Diabetes mellitus with neuropathy. 2) Possible ASHD. 3) Osteoarthritis.

Therapy: 20 units Lente insulin daily.

Index: BUN (not reported) ∠0 ∠0 ∠1.2 ∠0 ∠2 ∠0 = 3.2
(2 for possible left heart strain.)

1955: Patient was seen numerous times in 1955 and was undergood control, taking 65 - 70 units Lente insulin daily. He had some difficulty in regard to using U-40 insulin in a U-80 syringe and vice versa, and in sticking to his diet during religious holidays, but control for the most part was excellent. Blood pressure 150/80. Urine negative for protein.

Index: (No NPN) ∠0 ∠0 ∠1.3 ∠0 ∠2 ∠0 = 3.3

1956: Patient has now had diabetes for 14 years, taking Lente insulin 55 - 60 units daily. Physical exam reveals a cataract in the left eye, but no retinopathy noted. Heart is now slightly irregular with a grade I systolic murmur at the apex. Blood pressure 150/80. Neurological exam reported to be normal at this time. No proteinuria. NPN 41 mg%.

Index: $\frac{41 - 35}{35} \times 2$ ∠0 ∠0 ∠1.4 ∠0 ∠3 ∠0 = 4.7
(Patient receives a 3 for prior EKG findings plus development of heart murmur.)

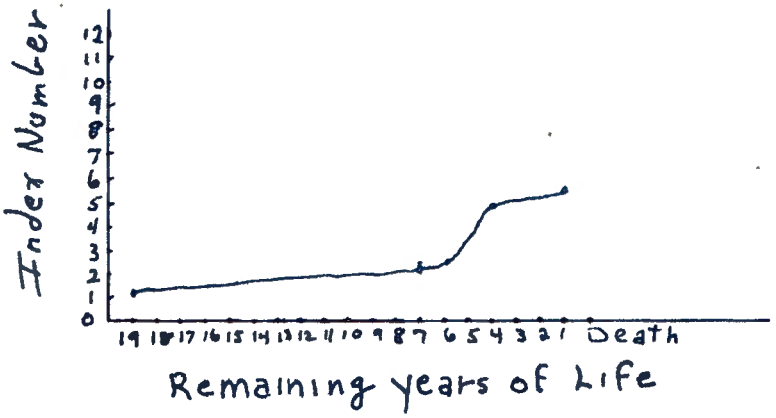
1957: Patient was given a trial of carbutamide during 1956 and responded well, going from 70 - 80 units of Lente insulin to 30 - 40 units Lente insulin daily. Control throughout 1956 was excellent, the patient being very faithful in his clinic visits. There was no history of insulin reactions and the urine sugar usually ran 0 - 1/.

Index: .3 /0 /0 /1.5 /0 /3 /0 = 4.8

1960: The patient's condition remained stable for all practical purposes during the preceding two years. Excellent control was achieved by faithful clinic visits and diet and insulin adjustment. Blood pressure 150/70.

Index: .3 /0 /0 /1.8 /0 /3 /0 = 5.1

Patient died in 1961 of a heart attack.



This man's graph would indicate that he was made of good vascular stock, for he lived 19 years after the diagnosis of diabetes mellitus was made. Even though he was too old for rehabilitation, at no time did this patient present enough evidence of vascular degeneration to raise his prognostic index above 5.1

Case 10 - Mr. E. M.: Initially seen in 1959 at the age of 36 years.

September 8, 1959: Chief complaint - loss of vision and evaluation of diabetes (referred by Services for Blind).

History: Diabetes mellitus discovered thirteen years ago in naval service; undiagnosed until patient went into coma. Presently on 50 - 55 units NPH insulin. Reactions usually noon or afternoon. Spills much sugar. Complains of weakness in legs when walking and on arising in morning.

Physical exam: 2 hr Post cibum 180/, 24 hr U.A. sugar 4/, albuminuria 3/, Hb 11 gms., blood pressure 168/84, weight 130 pounds, height 5'9", negative serology. Eyes - advanced diabetic retinopathy with retinal detachment on left. Vision (right) 20 - 340. PMI 5th interspace outside MCL. Extremities - pedal vessels palpable 3/, feet warm. EKG normal.

Diagnosis: 1) Diabetes mellitus, uncontrolled; 2) retinopathy advanced; 3) intracapillary glomerulosclerosis; 4) atherosclerosis with beginning calcification in femoral region with neuropathy involving upper thigh.

September 9, 1959: CBC normal, chest X-ray normal, AP views of lower extremity reveals early calcific plaques in the lower femoral arteries. Some pain in peroneal region on over extension of thigh and legs.

Therapy: 1) Adjustment of diet and insulin; 2) Vitamin B_{12}.

Index: (BUN not reported) /0 /3 /1.3 /2 /2 (femoral / enlarged) (calc. heart)

/0 = 8.3

September 30, 1959: Return visit. Chief complaint - edema of lower legs for three days, which is worse towards evening. Blood pressure 164/80, 1/ pitting edema from mid tibia down. Hb 8 gms.

Therapy: Bevatine 100 micrograms; Esidrex 50 mg. b.i.d.; feosol 2 tabs. t.i.d. pc.

October 23, 1959: History of three reactions occurring at night in last three days. U.A. - sugar negative, protein 100 mg., 2 hr PC 180/.

Therapy: Insulin changed to 40 units Lente in A.M. and 12 units in P.M.

Still complains of numbness in legs.

December 23, 1959: Decreased pain in feet. Chief complaint - chills and sweating since lunch today. Protein in urine 3+. Therapy: bed rest.

January 9, 1960: Protein 1+. Complains of recent U.R.I. Therapy: Tussaminic® for cough. No pain in legs at present.

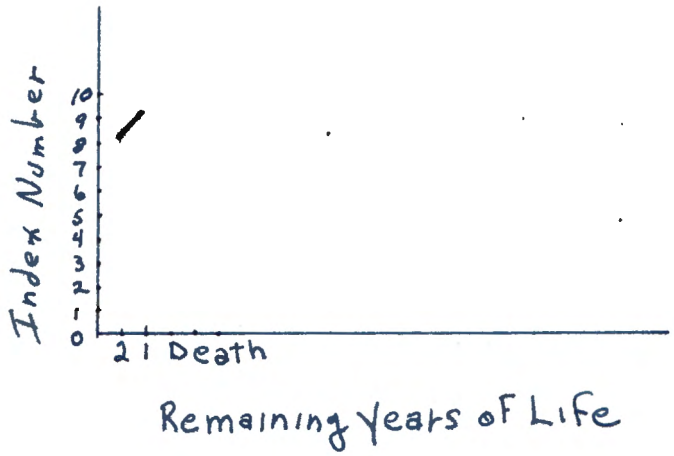
February 23, 1960: Chief complaint - pain in feet and subjective sense of cold feet, sexual impotence, heart negative, pedal pulsations good, protein 3+, sugar 4+, blood pressure 150/98.

Therapy: Increase insulin to 45 units in A.M. and 15 units in P.M.

Index: BUN not reported ✓1.8 ✓3 ✓1.3 ✓2 ✓2 ✓0 = 9.1

This patient died in 1961.

Here is another example of a patient who was initially evaluated and found to be in the "acceptable with reservations" category. His index rose from 8.3 to 9.1 in the period of months which would indicate him to be a poor candidate for rehabilitation. The fact that he was deemed a poor candidate is proven by his death, which occurred in approximately one year.



Case 11 - Mr. J. H.: Initially seen in 1958 at the age of 25 years.

June 11, 1958: Age 25. Diabetic for 16 years. Taking 40 units regular insulin in A.M. Takes regular insulin in P.M. as needed. Usually eats in restaurants. Physical exam reveals diabetic retinitis with hemorrhage. U.A. reveals 2+ albuminuria.

Therapy: Diet and insulin adjustment. Has had prior poor control.

Index: (No BUN) +0 +2 +1.6 +1 +0 +0 = 4.6

July and August 1958: Blood pressure 136/100, albuminuria 2+, heart examination negative, fair control, blood pressure 182/118 to 204/120.

Therapy: Serpasil .25 mg. t.i.d.

Index: (BUN not reported) +4 +2 +1.6 +0 +0 +0 = 7.6

September 1958: Has been in Ottumwa Hospital for twelve days. Placed on Diural 500 mg. b.i.d. Fair control at present, although blood pressure 162/120.

November 1958: Hospitalized at University Hospital. Chief complaints - hypertension and poorly controlled diabetes.

History: Duration of diabetes sixteen years. Frequently spills 3+ and 4+ sugar. Has had hypertension for three to four months. Presently on chlorthiazide.

Physical exam: Blood pressure 170/120. Eye examination reveals hemorrhages, waxy exudates, aneurysms, narrowing of the arterioles, and A-V nicking. Diagnosis - retinitis proliferans and an early lens opacity on the left.

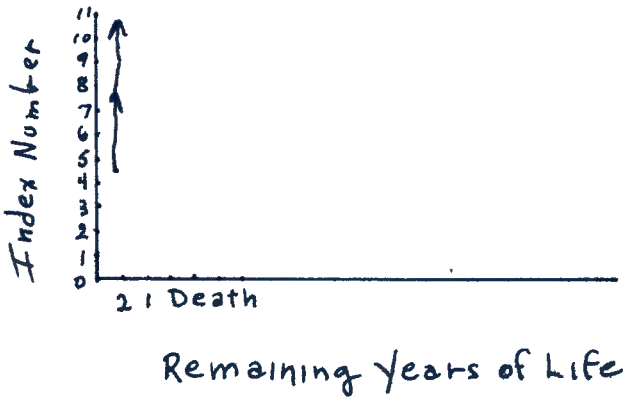
Laboratory: NPN 60 mg%, albuminuria 2+, creatinine 1.6, EKG - possible LVH.

Index: 1.2 +2 +4 +1.6 +2 +0 +0 = 10.8

This patient died in 1960.

This is a rather tragic story of a young man with marked visual impairment, who had a rapid increase in vascular degeneration in the

course of a few months. He would have been acceptable for rehabilitation early in 1958, but most surely would have had to be turned down later in the year 1958.



Let us now apply the formula to four living patients, currently being followed by the University Clinic, in order to speculate on their life expectancy and prospects of rehabilitation, if needed.

Case 12 - Mr. J. H.: Initially seen in 1960 at the age of 31 years.

History: A known diabetic for 16 years. Currently on 50 units of PZI and 20 units of regular insulin daily. He denies any history of insulin reactions or diabetic coma. The patient was asymptomatic on the first visit, but had been told by his private physician that his blood sugar was 450 mg%. The systemic review reveals a "hemorrhage in his left eye with decreased vision."

Physical exam: Blood pressure 110/78. Right eye: increased tortuosity of the vessels in the eye with an AV ratio of 1:2, a black area was noted on the retina in the temporal area. Left eye: a small wooly exudate is noted in the left nasal retina. Heart exam reveals a grade II systolic murmur at the apex. The liver is noted four centimeters below the right costal margin.

Laboratory: 2+ proteinuria, normal BUN, chest X-ray and EKG normal.

Index: 0 +0 +2 +1.6 +2 +0 +0 = 5.6

January 1961: The patient was placed on a 2000 calorie diabetic diet and his insulin dosage adjusted regularly in the clinic. He clinitests more regularly now and states that he runs "greens" most of the time. The patient is apparently under better control now then when seen initially. Proteinuria 2+, BUN normal.

Index: 0 +0 +2 +1.7 +1 +0 +0 = 4.7

August 1961: Patient now under good control, running negative sugars most of the time and following diet.

Index: 0 +0 +2 +1.7 +0 +0 +0 = 3.7

January 1962: On this clinic visit, the patient had a blood sugar of 290 mg%, but this was believed to be on the basis of a lingering cold and productive cough. Otherwise, the patient was under good control with PZI 46 units and 10 units of regular insulin daily. Proteinuria 2+.

Index: 0 +0 +2 +1.8 +0 +0 +0 = 3.8

This man's case history to date indicates how good control can lower one's index number. After being brought under control, his index dropped from 5.6 to 3.8 over a two-year period. Since this man has evidence of diabetic retinopathy, he may be a candidate for rehabilitation. With his index of only 3.8, and prospects for good control, he probably should be accepted. However, the patient also evidences signs of intracapillary glomerulosclerosis (2+ proteinuria). Therefore, before the patient is accepted for rehabilitation, he should probably have an extensive workup, looking for evidence of vascular degeneration elsewhere, such as peripherally and the heart.

Case 13 - Mr. C. S.: Initially seen in University Hospital Clinic in 1956 at the age of 29 years.

History: A known diabetic of two years duration. The patient has had erratic control for he could not afford insulin and took the drug only intermittantly. He was first seen with polyuria and polydipsia.

Physical exam: Blood pressure 130/80. The remainder of the exam was within normal limits.

Laboratory: F.B.S. 240 mg%. N.P.N. 33.5 mg%.

Index: 0 +0 +0 +0 +2(poor control) +0 +0 = 2

1958: Patient asymptomatic. He has been followed closely in diabetic clinic and has been sticking to his diet. He takes NPH insulin 14 - 20 units daily. Urine sugars are mostly 0 to 1+.

Index: 0 +0 +0 +0 +0 +0 +0 = 0

1960: Patient remains under good control. Essentially no change from last evaluation. Index = 0

1962: Patient being followed regularly with good control. Index = 0.

This is a case of a young diabetic who has had the disease only six years. He will soon be reaching the tenth year of his disease when the signs of vascular degeneration frequently present themselves. It is very important for this man to continue getting a "0" for degree of control, for this is our only way of preventing a rise in the index number due to vascular degeneration.

Case 14 - Mr. A. S.: Initially seen in 1960 at the age of 33 years.

History: A known diabetic for 22 years. He had been on no special diet. He had been under fair control prior to 1959, but during this year, he developed ulcers and his control deteriorated. He gives a history of diminishing vision for one year.

Physical exam: Blood pressure 180/100. On fundoscopic exam, mild diabetic retinopathy is noted.

Laboratory: 3+ proteinuria, BUN 25 mg%, IVP and EKG normal.

Impression: Uncontrolled diabetes and hypertension.

Index: $\frac{25 - 15}{15} \times 2 + \frac{100 - 80}{10} + 3 + 2.2 + 2 + 0 + 0 = 8.7$

July 1961: Patient hospitalized in University Hospital for fifteen days due to poor diabetic control, renal hypertension, and diabetic neuropathy involving the legs. Physical examination revealed numbness and paresthesia in the lower legs. The peripheral pulses were palpable. Blood pressure 160/90, BUN 30 mg%, proteinuria 3+, serum creatinine 3.3. Discharge diagnoses were diabetes mellitus juvenile type, diabetic neuropathy, renal hypertension, and Kimmelsteil - Wilson disease.

Index: $\frac{30 - 15}{15} \times 2 + 1 + 3 + 2.3 + 2 + 0 + 0 = 12.3$

1962: Condition is essentially the same as in prior evaluations; however, the index will rise .1 point due to another year of diabetes. Index 12.4.

This man is a poor candidate for rehabilitation for he obviously has severe diabetic nephropathy and a history of poor control. At this point, he needs an EKG, chest film, etc., to evaluate his cardiovascular status, for if this is significant, it could kill the patient before his renal pathology does.

Case 15 - Mr. V. H.: Initially seen in 1960 at the age of 29 years.

History: A known diabetic of 23 years duration. He gave a history of frequent insulin reactions (one per week). There was also a complaint of mild shortness of breath on exertion. Systemic review revealed failing eyesight for approximately one year. He was told that he had hypertension in 1959 and was placed on Serpasil.

Physical examination: Blood pressure 204/110. Examination of the eyes revealed only light perception on the left. Fundoscopic examination revealed hemorrhages, exudates and microaneurysms. The optic disk was not identified on the left. Grade one pretibial edema was noted. The peripheral pulses were equal throughout.

Laboratory: 3/ proteinuria.

Impression: Juvenile diabetes with poor control, retinopathy, and nephropathy.

Index: BUN (not reported) /3 /3 /2.3 /2 /0 /0 = 10.3

1960: Hospitalized at University Hospital from August 26, 1960, to September 4, 1960, due to poor control, retinopathy, neuropathy, nephropathy, and hypertension. He was treated in the hospital with insulin and diet adjustment, diuretics and hypertensive drugs. The blood pressure ranged, however, from 200/110 to 100/100. Serum creatinine 2.5 mg%, 3/ proteinuria. Discharge diagnoses: 1) Diabetes mellitus, poor control; 2) grade three diabetic retinopathy; 3) diabetic nephropathy; 4) diabetic neuropathy; 5) hypertensive heart disease (mild).

Index: (2.5 - 1)2 /3 /3 /2.3 /2 /2(hypertensive heart disease) /0 - 15.3

1961: Again hospitalized with pyelonephritis. Blood pressure ranged from 110 - 70 diastolic. BUN now 80 mg%. Discharge diagnosis: 1) Intercapillary glomerulosclerosis with hypertension secondary to azotemia. Since the BUN is probably distorted by pyelonephritis, the last serum creatinine will be used.

Index: (2.5 -1)2 /3 /3 /2.4 /2 /2 /0 = 15.4

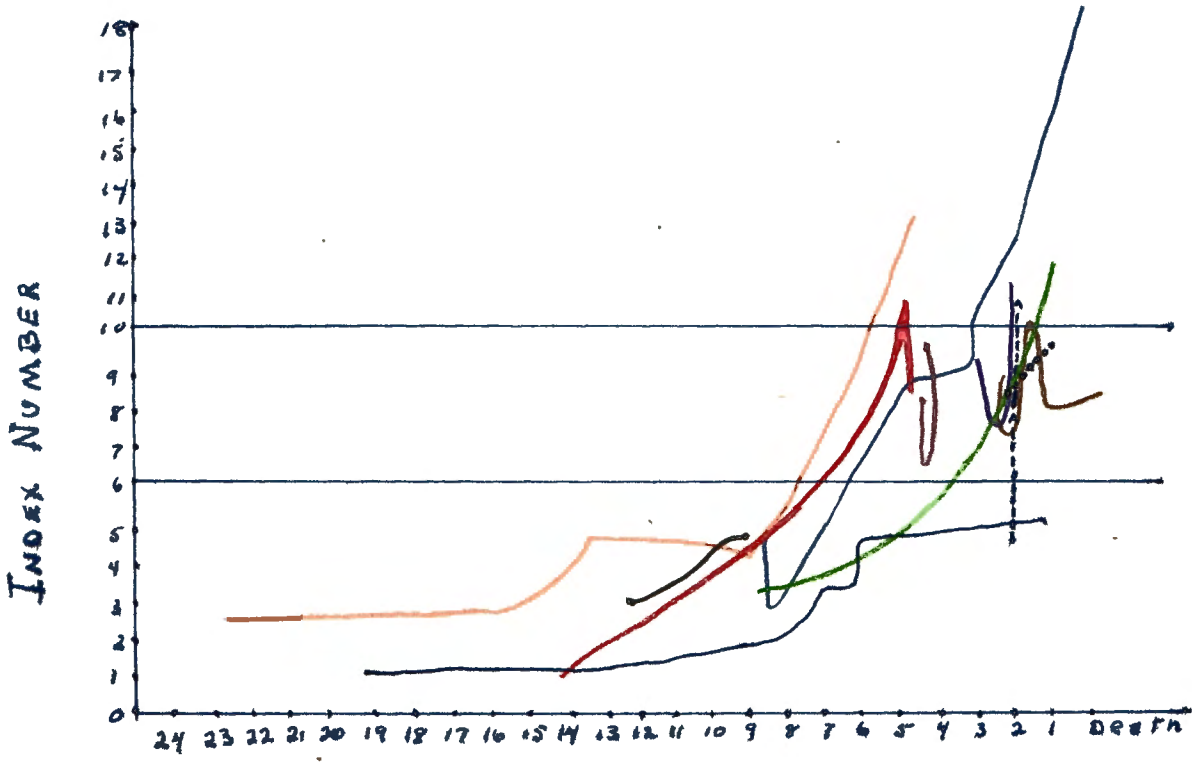
1962: The patient continues to be followed in the diabetic clinic with little change in condition. Proteinuria is now 4/, the BUN 35 mg%. Quality of control remains the same.

Index: 3 /3 /4 /2.5 /2 /2 /0 = 16.5

This is a case of a young man who has had diabetes for a long time and has developed severe vascular degeneration. There is a very good possibility that it will be necessary for him to ask for rehabilitation due to his diabetic retinopathy. This patient will have to be turned down for rehabilitation for his outlook for remaining years of life is poor. He now has a prognostic index of 16.5, which, as we shall see later, indicates a very short duration of life.

In evaluating the prognostic index, several case histories of diabetic patients have been presented. Eleven of these diabetic patients have died due to complications of their disease; the remaining four are being followed at present in the University Clinics. The information on the deceased diabetics was obtained from the office records of Dr. Morris Margolin or University Hospital. The patients ranged in age from 15 to 64 years at the time of initial evaluation. Twelve of the patients were males and three females. In each case, the prognostic index was applied to the patients in various stages of their life. Inasmuch as possible, all pertinent facts relating to their disease were presented in order to apply the prognostic index. In each case, when the individual died of his disease, a graph correlating index numbers with remaining years of life was presented.

The following graph is a composite of the individual graphs from the dead diabetics. The graph correlates index numbers with remaining years of life. Parallel lines are drawn on the graph to separate the index numbers into three categories: zero to six, six to ten, ten and above. From this graph, some general observations can be made.



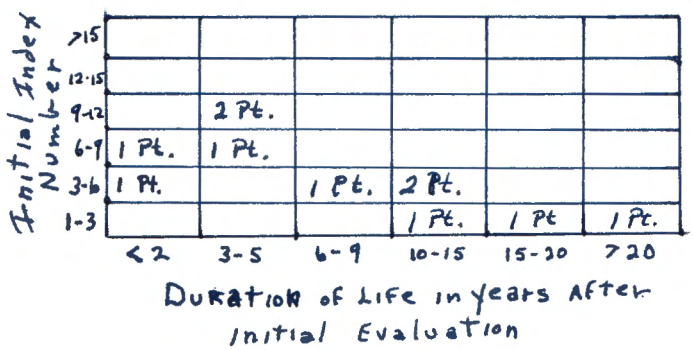
REMAINING YEARS OF LIFE

Case No. 1
Case No. 2
Case No. 3
Case No. 4
Case No. 5
Case No. 6
Case No. 7
Case No. 8
Case No. 9
Case No. 10
Case No. 11

In the eleven dead diabetics studied, the duration of life, from the initial evaluation, ranged from two to twenty-four years. From the graph it is seen that the average duration of life of those patients evaluated when their index number was below six was 12.5 years. The average length of life after receiving an index number of six was less than five years. None of the patients, after receiving an index number of six, lived over eight years. The average length of life of those patients after receiving a ten or above during the course of their life lived on the average only 3 years.

Vascular degeneration in diabetes mellitus appears to take two courses, as evidenced by prognostic index numbers. The patient either has a long slow rise in index or he has a rapid rise (note the steep rise in index number in the above-six group). There seems to be no direct linear relationship between index number and remaining years of life, e. g., apparently death inducing vascular degeneration develops rapidly after a smoldering course.

The following graph correlates length of life remaining after the initial index with the index number received at the time of initial evaluation. This graph reveals the spread of the cases studied.



From the data presented, it appears that this prognostic index, if utilized properly, could be a useful adjunct to the rehabilitation personnel when they are confronted with a diabetic patient. I think we can predict fairly accurately how long a diabetic has to live with this prognostic index. I do not believe that this formula should be the only criteria for there are certainly exceptions to the case and many pitfalls. This formula must be used in conjunction with the referring physician's opinion as to the life expectancy of the patient.

The formula itself is only grossly accurate and needs to be applied to many patients over several years duration. One of the drawbacks to this procedure is that diabetics are difficult to follow closely for many years. Another drawback is the paucity of data on which to evaluate a patient regularly. As it frequently happens, specific lab tests, X-rays, EKG, etc., are only ordered when abnormality is suspected. Due to this practice, gradual rises in index number are not seen because the patients pass through mild degenerative disease to obvious degenerative disease without benefit of laboratory procedures which might have shown abnormality before clinical signs appear.

In order to supply the best medical care possible and to honestly evaluate diabetic patients for rehabilitation purposes, possibly we should evaluate each diabetic annually after the tenth year of his disease. If every year we obtained a careful history in regard to his cardiovascular and renal status, and performed a BUN, chest X-ray, EKG, etc., we could actively treat the complications before they become obvious.

I realize full well that our therapy for these complications is largely supportive and somewhat limited, but the performance of these routine tests regularly would drive home the point of good control in our patients.

One of the pitfalls in this formula is the patient who has an index number of less than six for many years, and then suddenly dies of a myocardial infarction. Maybe this type of patient could be detected with a careful history, physical, chest X-ray, and EKG.

As was pointed out earlier, one cannot apply the prognostic index to a diabetic patient at any one time and draw an opinion on one evaluation. The total picture is necessary and I believe that the rehabilitation personnel should obtain as much of the patient's medical history as possible, and plot prognostic indices as done in this paper. By this method, the rehabilitation personnel cannot only evaluate the patient at one time, but can get a picture of the patient's progression. This is quite important in regard to the likelihood of future good control.

I am sure that readers of this paper will say that the prognostic indices applied in some parts of the prognostic index are somewhat arbitrary. Possibly in the future use of this index, some definite standards should be applied, e.g., should left ventricular strain by EKG receive a 2 or a 3 for index purposes; is one insulin reaction per week good control or fair control?

I believe that this index, interpreted wisely and in conjunction with sound medical advice, could be a valuable asset to our state rehabilitation services.

Special acknowledgment goes to Dr. Morris Margolin, my advisor, who has been extremely helpful in the production of this paper, and to Mrs. Carl Reerink, the typist.

SUMMARY:

A prognostic index for patients suffering from diabetic vascular disease has been presented, an index which rehabilitation and placement service personnel can apply for assessing whether any individual presenting himself for service should be given extensive rehabilitation services or not, realizing that in many instances the life expectancy of individuals who have suffered extensive vascular change through disease is not great. The prognostic index $2(\text{serum creatinine} - 1)$ or $2\left(\frac{\text{blood urea nitrogen} - 15}{15}\right)$ or $2\left(\frac{\text{non-protein nitrogen} - 35}{35}\right)$ plus $\left(\frac{\text{diastolic blood pressure} - 80}{10}\right)$ plus (degree of proteinuria) plus $\left(\frac{\text{duration of diabetes}}{10}\right)$ plus $\left(\frac{\text{degree of control}}{2}\right)$ plus (degree of cardiovascular disease) plus (degree of cerebral vascular disease) was broken down into component parts and the significance of each factor examined. The prognostic index was then applied to 14 diabetic patients, eleven of which have died due to the complications of their disease and four which are currently being followed. In the eleven diabetic patients studied who died of their disease, graphs correlating index number with remaining years of life were presented. The duration of life of these patients from the time of initial evaluation ranged from

two to twenty-four years. The average duration of life of those patients evaluated when their index number was below six was 13.5 years. The average length of life after receiving an index number of six was less than five years. The average length of life of those patients after receiving a ten or above during the course of their life lived on the average only three years.

From this data, it can be concluded that a prognostic index number up to six should be acceptable for extensive rehabilitation because they could be expected to be active many more years. Any number from six to ten should be acceptable for rehabilitation with reservations and viewed in light of control from the point of rehabilitation on. Above the number ten, rehabilitation problems are not recommended for the duration of life appears to be limited.

It is believed that this prognostic index could be a valuable asset to the state rehabilitation services if used wisely and with sound medical advice.

BIBLIOGRAPHY

(Sequentially Arranged)

1. Clay, Frances, Towards Competence in Serving the Blind, Journal of Rehab. 26: 14-6, March - April 1960.

2. Wilson, J.L., Root, H.F., and Marble, A., Diabetic Nephropathy, Clinical Syndrome. New England J. Med. 245: 513-517, 1951.

3. Root, H.F., Diabetes and Vascular Disease in Youth, Am. J. Med. Sc. 217: 545-553, 1949.

4. Brun, C., and others, Diabetic Nephropathy, Kidney Biopsy and Renal Function Tests. Am. J. Med. 15: 187-197. 1953.

5. McCullogh, E.P., Vascular Complications of Diabetes Mellitus. Cleveland Clin. Quart. 26: 97-105. July 1959.

6. Winter, F.C., Diabetic Retinopathy; Degenerative Vascular Complications of Diabetes and Discussion of Clinical Aspects of the Disease. J.A.M.A. 174: 143-6, 10 Sept. 60.

7. Warren, S., and LeCompte, P.M., The Pathology of Diabetes Mellitus, 3d.ed. Philadelphia Lea and Fibiger, 1952, 336 pp.

8. McCullogh, E.P., Vascular Complications of Diabetes Mellitus, Cleveland Clin. Quart. 26: 97-105, July 1959.

9. E.L. Mahallawy MN, Sabour MS, Osman LM, Clinical and Diagnostic Aspects of Diabetic Nephropathy, Scot. Med. J. 5: 335-41 Aug.60.

10. Caird, F.I., Survival of Diabetics with Proteinuria Diabetes 10: 178-81, May - June 61.

11. O'Sullivan, P.J., and others, Proteinuria in Diabetes, Quart. J. Med. N.S., 29: 63-0 Oct. 1960, abstract.

12. Winter, F.C., Diabetic Retinopathy, Degenerative Vascular Complications of Diabetes and Discussion of Clinical Aspects of the Disease, J.A.M.A. 174: 143-6 10 Sept. 60.

13. White, P., and Waskow, E., Clinical Pathology of Diabetes in Young Patients. South, M.J. 41: 561-567, 1948.

14. Paul, J.T., and Presley, S.J., Complications of Long Term Diabetes Mellitus. Ann. Int. Med. 49: 142-150, 1958.

15. McCullogh, E.P., Vascular Complications of Diabetes Mellitus, Cleveland Clin. Quart. 26: 97-105 July 1959.

16. Winter, F.C., Diabetic Retinopathy, Degenerative Vascular Complications of Diabetes and Discussion of Clinical Aspects of the Disease, J.A.M.A. 174: 143-6 10 Sept. 60.

17. Kramer, D.W., Atheromatosis - Its Relationship to Diabetes Mellitus, Nebr. Med. J. 45: 249-56 May 60.

18. Ranke, E.J., Diabetic Retinopathy and the Pituitary, AMA Arch. Ophth. 6: 859-63 Nov. 59.

19. Sheridan, J.T., The Insurability of Persons with Diabetes Mellitus, Diabetes 9: 494-9, Nov. - Dec. 60.

20. Root, and others, Coronary Atherosclerosis in Diabetes Mellitus; Post Mortem Study J.A.M.A. 113: 27-30 1939.

21. Rogers, W.R., Halromb, Lengthy Diabetes: Causes and Effects AMA Arch. Int. Med. 105: 746 - 51 May 60.

22. McCullogh, E.P., Vascular Complications of Diabetes Mellitus. Cleveland Clin. Quart. 26: 97-105 July 1959.

23. Bradley, R.F., and Bryfogle, J.W., Survival of Diabetic Patients after Myocardial Infarction, Am. J. Med. 20: 207-216 1959.

BIBLIOGRAPHY

(Alphabetically Arranged)

1. Bradley, R. F., and Bryfogle, J.W., Survival of Diabetic Patients after Myocardial Infarction, Am. J. Med. 20: 207-216 1959

2. Brun, C., and others, Diabetic Nephropathy; Kidney Biopsy and Renal Function Tests. Am. J. Med. 15: 187-197. 1953.

3. Caird, F.I., Survival of Diabetics with Proteinuria Diabetes 10: 178-81, May - June 61.

4. Clay, Frances, Towards Competence in Serving the Blind, Journal of Rehab. 26: 14-6, March - April 1960.

5. E. L. Mahallawy MN, Sabour MS, Osman LM, Clinical and Diagnostic Aspects of Diabetic Nephropathy, Scot. Med. J. 5: 335-41 Aug.60.

6. Kramer, D.W., Atheromatosis - Its Relationship to Diabetes Mellitus, Nebr. Med. J. 45: 249-56 May 60.

7. McCullogh, E.P., Vascular Complications of Diabetes Mellitus. Cleveland Clin. Quart. 26: 97-105. July 1959.

8. O'Sullivan, P.J., and others, Proteinuria in Diabetes, Quart. J. Med. N.S., 29: 63-0 Oct. 1960, abstract.

9. Paul, J.T., and Presley, S.J., Complications of Long Term Diabetes Mellitus. Ann. Int. Med. 49: 142-150, 1958.

10. Ranke, E.J., Diabetic Retinopathy and the Pituitary, AMA Arch. Ophth. 6: 859-63 Nov. 59.

11. Rogers, W. R., Halromb, Lengthy Diabetes: Causes and Effects AMA Arch. Int. Med. 105: 746-51 May 60.

12. Root, and others, Coronary Atherosclerosis in Diabetes Mellitus; Post Mortem Study J.A.M.A. 113: 27-30 1939.

13. Root, H.F., Diabetes and Vascular Disease in Youth, Am. J. Med. Sc. 217: 545-553, 1949.

14. Sheridan, J.T., The Insurability of Persons with Diabetes Mellitus, Diabetes 9: 494-9, Nov. - Dec. 60.

15. Warren, S., and LeCompte, P.M., The Pathology of Diabetes Mellitus, 3d.ed. Philadelphia Lea and Fibiger, 1952, 336 pp.

16. White, P., and Waskow, E., Clinical Pathology of Diabetes in Young Patients. South, M.J. 41: 561-567, 1948.

17. Wilson, J.L., Root, H.F., and Marble, A., Diabetic Nephropathy, Clinical Syndrome. New England J. Med. 245: 513-517, 1951.

18. Winter, F.C., Diabetic Retinopathy, Degenerative Vascular Complications of Diabetes and Discussion of Clinical Aspects of the Disease, J.A.M.A. 174: 143-6 10 Sept. 60.