The Memphis and Atlanta Continuing Care Programs for Diabetes. II. Comparative Analyses of Demographic Characteristics, Treatment Methods, and Outcomes over a 9–10-year Follow-up Period

JOHN K. DAVIDSON, M.D., Ph.D., ROGER VANDER ZWAAG, Ph.D., CHERYL L. COX, R.N., M.S.N., HARRY K. DELCHER, M.D., ILSE MAINZER, HENRY BAGGETT, B.A., AND JOHN W. RUNYAN, M.D.

A total of 1467 black patients (911 in Atlanta, 556 in Memphis) were selected (1969–70) and followed longitudinally and prospectively until death (404 patients) or through 31 December 1979, when 676 were alive and active and 387 were lost to follow-up. The women/men ratio in each cohort was 4.7/1. Women had more excess body wt than men at maximum weight and at time of diagnosis. At selection, the Atlanta cohort was older (60.2 vs 56.8 yr), had diabetes longer (7.5 vs 5.2 yr), and had a higher initial mean random plasma glucose (MRPG) level (217 vs 195 mg/dl) than the Memphis cohort. The Atlanta cohort was on sulfonylurea/phenformin therapy, which was discontinued at entry. After 9–10 yr follow-up, the MRPG level was not significantly different from the initial level in either cohort, and the Atlanta cohort level was still significantly higher (221 vs 185 mg/dl). Mean weight loss after 9–10 yr follow-up was significantly greater in Atlanta (17.7 vs 6.8 lb). Those under good control in 1979, as indicated by random plasma glucose (RPG) of <150 mg/dl, lost more weight (means: Atlanta, 23 lb; Memphis, 8.7 lb) than those under poor control in 1979 (RPG > 300 mg/dl; means: Atlanta, 14.7 lb; Memphis, 1.3 lb). In the pooled alive and active cohorts (1979), 29.1% were under good control (RPG < 150 mg/dl); 52.9%, fair control (RPG = 150–300 mg/dl); and 18.0%, poor control (RPG > 300 mg/dl). Of the 639 alive and active patients, paired plasma glucose levels were <200 mg/dl in 207 patients in 1969–70 and <200 mg/dl in 322 in 1979. In 1979, 70 more patients on diet alone and 50 more on diet plus insulin had levels of <200 mg/dl, but five fewer on sulfonylurea/phenformin had <200 mg/dl. Observed/expected mortalities in the two cohorts were almost identical. Nine-year life table survival rates were related primarily to age at entry (73%) and secondarily to duration of diabetes (15%). The men/women death ratio for the cohorts was 1.58/1 (Atlanta, 1.54/1; Memphis, 1.66/1). Standardized mortality ratios (SMR) were highest for those on insulin therapy (Atlanta, 1.62; Memphis, 1.78), intermediate for those on sulfonylurea/phenformin therapy (Memphis, 1.52), and lowest for those on diet therapy alone (Atlanta, 1.33; Memphis, 1.06). In Memphis, SMR for sulfonylureas was 1.50, for phenformin 1.56, and for sulfonylurea plus phenformin 1.56 (for all oral agent therapy, 1.52). Differences in SMR in the three groups in Memphis were not significant (P = 0.082). Mortality outcomes revealed by SMR appeared to relate to perceived severity of diabetes in each cohort and clinical assessment of effectiveness of each mode of therapy. This appeared to reflect differences in therapeutic strategy in that 70.9% in Atlanta and 25% in Memphis were treated with diet alone, 29.1% in Atlanta and 25% in Memphis were treated with diet and insulin, and 50.4% in Memphis were treated with diet and sulfonylurea/phenformin. Since therapy was not randomly assigned at inception, and since patients were not routinely continued on one mode of therapy for the duration of the study, it was not possible to calculate prospectively and longitudinally the relative risks of mortality for each therapeutic modality. DIABETES CARE 7: 25–31, JANUARY–FEBRUARY 1984.

During the decade of the seventies, both Memphis and Atlanta conducted prospective longitudinal studies to examine the efficacy of their continuing care programs for patients with diabetes mellitus.[1,2] Both programs were developed in university-based teaching hospitals, and each provided what was perceived in that location as optimal continuing primary care for an underserved, medically indigent population of referred patients with diabetes mellitus (City of Memphis Hospital, Memphis–Shelby County Health Department, and Grady Memorial Hospital). Both used defined policies and procedures for initial evaluation, education, and treatment, and for continuing follow-up.[3-7] Both have reduced significantly the incidence and costs of hospitalization,[1,2,5,8] generally through prevention of acute complications (diabetic ketoacidosis) and of lower-extremity amputations.

Long-term treatment strategies designed to control diabetes in the two programs differed. Atlanta discontinued use of sulfonylurea and/or phenformin therapy in 1970 and, in 1973, implemented a policy that limited the use of insulin[9,10] to those who were (1) in diabetic ketoacidosis or a hyperglycemic hyperosmolar state, (2) pregnant, and (3) significantly hyperglycemic (fasting plasma glucose > 130 mg/dl) and not significantly overweight (<110% of ideal body weight). The cornerstone of therapy for those in Atlanta who were not insulin dependent was aggressive low-calorie diet therapy (including 1-wk total fasts) designed to produce the loss of as much excess body weight as possible.

Memphis routinely prescribed low-calorie diet therapy for the overweight, and dietitians encouraged weight loss during follow-up, but the approach was less aggressive than the Atlanta approach, and fasting was not used. The primary therapeutic strategy in Memphis was to maintain plasma glucose levels as near normal as possible with sulfonylurea and/or phenformin (until 1978) or insulin therapy as currently recommended.[11,12]

The intent of this report is to compare the effects of alternative therapeutic methods (diet, diet plus insulin, diet plus sulfonylurea/phenformin) on outcomes (alive, dead, lost to follow-up, plasma glucose, and weight changes) for a 9–10-yr follow-up.

METHODS AND PATIENTS

Structure of the Atlanta and Memphis programs. The structures of the Atlanta and Memphis programs have been described in detail,[7,9] and the selection of the 556 black patients who made up the 1970 Memphis cohort and the 911 black patients who made up the 1971 Atlanta cohort has been reported.[13]

Patient classification. Patients were classified according to their status on 31 December 1979 as (1) alive and active: defined as those known to be alive and for whom clinical data had been collected during the year 1 January to 31 December 1979 (Table 1A); (2) lost to follow-up: defined as those for whom no clinical encounter or death was recorded for 1 yr or longer before 31 December 1979; or (3)

TABLE 1A
Demographic and plasma glucose and weight measurements by last known treatment method for patients alive and active

	No.	Entry age (yr)	Percent male	Duration of diabetes (yr)	Initial glucose (mg/dl)		Last known glucose (mg/dl)		P*	Initial weight (lb)		Last known weight (lb)		P*
Diet only														
Atlanta	315	59.0	11.4	6.7	211		213		0.806	178.4		158.1		<0.001
Memphis	66	55.8	15.2	2.8	132		138		0.427	183.9		174.5		0.012
P†		0.35	0.002	<0.001	<0.001		<0.001			0.308		0.002		
Insulin + diet														
Atlanta	122	55.9	16.4	7.8	236		242		0.586	155.6		144.3		<0.001
Memphis	73	47.9	13.7	7.7	239		223		0.339	177.6		175.2		0.563
P†		<0.001	0.801	0.975	0.858		0.181			<0.001		<0.001		
Memphis only														
Sulfonylurea(s) + diet	86	54.9	9.3	3.2	204	203	186	188	0.212	180.9	182.9	172.2	174.6	<0.001
Phenformin + diet	6	47.2	0	0	219		160		0.055	214.0		195.0		0.068
Sulfonylurea(s) + diet + phenformin	8	51.9	12.5	3.1	183		227		0.369	181.5		185.1		0.828
P†		0.243	0.697	0.236	0.194		0.226			0.142		0.217		

*P (comparison of horizontal means) = Probability of a significant difference (P = <0.01) within the Atlanta and Memphis cohorts, reference initial and last known plasma glucose and initial and last known weight.

†P (comparison of vertical means) = Probability of a significant difference (P = <0.01) between the Atlanta and Memphis cohorts, reference entry age, percent male, duration of diabetes, initial glucose, last known glucose, initial weight, and last known weight.

dead: defined as those for whom death occurred (verified by death certificate) at any point during the study, through 31 December 1979 (Table 1B).

Treatment methods. Patients were classified by their last known treatment method. Both cohorts contained patients who had been treated since 1970 with diet alone, or since 1970 with diet plus insulin (sometimes transiently). Of the Memphis patients, 50.4% were treated with diet plus (1) sulfonylureas and/or (2) phenformin (until 1978) (Tables 1A and 1B).

Data collection and analysis. Data were collected during clinic visits, hospital admissions, and periodic searches of the file of death certificates in Atlanta, of the Georgia Department of Human Resources Bureau of Vital Statistics, and, in Memphis, of Shelby County, Tennessee. When data were not available from these sources, and when there was no response to three letters and three telephone calls to the patient and to a responsible friend or relative, patients were classified as lost to follow-up. Occasionally, those who were classified as lost to follow-up would reappear in the clinic or hospital and again be classified as alive and active.

Atlanta data were stored in the Emory University Univac 9080 Computer and transmitted on a 1600 BPI magnetic tape to the University of Tennessee College of Medicine for comparative statistical analysis with Memphis data on a PDP-11/70 computer.

Analyses were conducted as follows: (1) analyses on each cohort as a whole and on each cohort subdivided into alive and active, lost to follow-up, and dead, according to alternative therapeutic modalities as of 31 December 1979 were performed (Tables 1A and 1B); (2) 1979 plasma glucoses in those alive and active in each cohort were stratified at five levels and compared with 1970–71 paired plasma glucose levels (Table 2); (3) weight and plasma glucose changes from 1969–71 to 1979 in the groups under good control (1979 plasma glucose <150 mg/dl) and the groups under poor control (1979 plasma glucose >300 mg/dl) were compared; and (4) the effects of alternative therapies in lowering mean group plasma glucose levels from >200 mg/dl in 1969–71 to <200 mg/dl in the pooled alive and active groups in 1979 were compared.

Because it was not possible for many patients to attend the clinics at specified times, plasma glucose levels were measured at random, without regard to prior food intake, in venous plasma by the polarographic glucose-oxidase method. Because the clinics were frequently crowded, patients were weighed while wearing shoes and conventional indoor clothes.

Standard deviations, standard errors, and P values for differences were calculated according to standard statistical techniques for each group and each subgroup. Differences were classified as significant (S) when P was <0.01, and not significant (NS) when P was >0.01.

TABLE 1B
Demographic and plasma glucose and weight measurements by last known treatment method for patients who died

	No.	Entry age (yr)	Percent male	Duration of diabetes (yr)	Initial glucose (mg/dl)	Last known glucose (mg/dl)	P*	Initial weight (lb)	Last known weight (lb)	P*
Diet only										
Atlanta	181	64.7	24.9	8.3	188	190	0.853	171.3	153.7	<0.001
Memphis	33	68.4	30.3	6.9	161	151	0.928	165.9	158.4	0.021
P†		0.049	0.659	0.367	0.225	0.024		0.462	0.460	
Insulin + diet										
Atlanta	81	62.5	21.0	10.4	259	252	0.976	153.1	141.6	<0.001
Memphis	36	59.4	25.0	10.9	247	224	0.411	173.4	168.7	0.366
P†		0.201	0.809	0.763	0.661	0.295		0.044	0.001	
Memphis only										
Sulfonylurea(s) + diet	52	64.0	28.8	5.1	201	185	0.212	155.8	152.4	0.332
Phenformin + diet	9	67.6	11.1	4.2	128	129	0.814	168.5	166.4	0.825
Sulfonylurea(s) + diet + phenformin	12	59.4	16.7	7.5	234	259	0.628	172.4	166.8	0.425
P†		0.329	0.373	0.411	0.067	0.004		0.372	0.395	

*P (comparison of horizontal means) = Probability of a significant difference (P = <0.01) within the Atlanta and Memphis cohorts, reference initial and last known plasma glucose and initial and last known weight.

†P (comparison of vertical means) = Probability of a significant difference (P = <0.01) between the Atlanta and Memphis cohorts, reference entry age, percent male, duration of diabetes, initial glucose, last known glucose, initial weight, and last known weight.

RESULTS

Atlanta patients were significantly older (60.2 versus 56.8 yr, $P < 0.001$), had had diabetes significantly longer (7.5 versus 5.2 yr, $P < 0.001$), and had a significantly higher initial mean random plasma glucose level (217 versus 195 mg/dl, $P = 0.007$) than Memphis patients when the cohorts were selected. Initial mean weights in the two cohorts were not significantly different (Atlanta 170.6 lbs versus Memphis 174.6 lbs, $P = 0.085$). Both cohorts were predominantly women, with the women/men ratio in each cohort being 4.7/1.

On 31 December 1979, the pooled cohorts contained 676 alive and active (Table 1A), 404 dead (Table 1B), and 387 lost to follow-up. Demographic characteristics of those lost to follow-up were not significantly different from those not lost to follow-up. Of those not lost to follow-up on 31 December 1979, 53.8% of the men and 34.0% of the women had died. The men/women death ratio for the pooled cohorts was 1.58/1 (Atlanta 1.54/1, Memphis 1.66/1).

Those who died (Table 1B) were significantly older and had had diabetes longer than those who were alive and active in 1979 (Table 1A). In the life table survival rate analysis, age accounted for 73% of the sums of squares related to death and duration of diabetes for 15% of the sums of squares related to death.[13]

Neither Atlanta nor Memphis mean initial and mean final random plasma glucose levels were significantly different (Atlanta initial 217 versus final 221 mg/dl; Memphis initial 195 versus final 185 mg/dl), but both initial and final levels were significantly higher in Atlanta (Table 1A).

Of the 676 alive and active in 1979, paired random plasma glucose levels (1969–71 and 1979) were available in 639 (94.5%), or 433 of 437 (99.1%) in Atlanta and 206 of 239 (86.2%) in Memphis. Although mean initial and final levels were not significantly different in either cohort, stratification of the random plasma glucose levels in the pooled cohorts revealed that 56.2% were lower, 9.1% were unchanged (±3 mg/dl), and 34.7% were higher in 1979.

In the pooled cohorts in 1979, 186 (29.1%) were under

TABLE 2
Stratification of 1979 plasma glucose (PG) at five levels, and comparison with paired initial PG and weight changes from 1970–71 to 1979

PG levels (mg/dl)	N	PG 1971	PG 1979	% change	Mean weight loss 1971–79 (lb)
Atlanta					
Diet only					
1979 PG <150	83	182	116	− 36.1	−24.0
1979 PG 150–199	68	224	172	− 23.0	−18.3
1979 PG 200–249	58	223	223	0	−18.6
1979 PG 250–299	42	208	275	+ 31.9	−22.9
1979 PG ≥300	60	221	356	+ 61.1	−18.6
Insulin					
1979 PG <150	26	222	123	− 44.5	−19.9
1979 PG 150–199	17	188	169	− 10.0	− 8.4
1979 PG 200–249	24	239	227	− 5.0	−12.5
1979 PG 250–299	21	215	277	+ 28.8	− 9.7
1979 PG ≥300	34	278	358	+ 28.8	− 7.3
Memphis					
Diet only					
1979 PG <150	41	115	112	− 2.5	−13.2
1979 PG 150–199	10	126	178	+ 41.1	− 4.9
1979 PG 200–249	3	226	237	+ 4.9	− 6.3
1979 PG 250–299	3	375	270	− 28.0	−14.0
1979 PG ≥300	1	100	332	+232.0	+ 3.0
Sulfonylureas/phenformin					
1979 PG <150	23	188	112	− 40.7	− 6.8
1979 PG 150–199	30	189	176	− 7.1	− 8.5
1979 PG 200–249	20	243	218	− 9.9	−16.5
1979 PG 250–299	5	151	271	+ 79.1	− 3.0
1979 PG ≥300	10	258	356	+ 37.8	+ 0.8
Insulin					
1979 PG <150	13	246	114	− 53.8	+ 2.1
1979 PG 150–199	11	237	171	− 28.0	− 2.3
1979 PG 200–249	12	213	217	+ 2.0	− 5.6
1979 PG 250–299	14	209	271	+ 29.9	+ 7.5
1979 PG ≥300	10	248	359	+ 44.9	− 3.9

good control (random plasma glucose <150 mg/dl), 338 (52.9%) were under fair control (random plasma glucose 150–299 mg/dl), and 115 (18.0%) were under poor control (random plasma glucose >300 mg/dl). Those under good control had lost more weight (Atlanta mean 23 lb, Memphis mean 8.7 lb) than those under poor control (Atlanta mean 14.7 lb, Memphis mean 1.3 lb).

After discontinuing sulfonylurea and/or phenformin therapy in 1970, 70.9% of those alive and active in 1979 in Atlanta had been treated with diet therapy alone. Although both weight and plasma glucose levels decreased in the majority, plasma glucose levels increased and weight levels decreased in many, presumably as a result of persistent hyperglycemia and glucosuria.

A comparison of the relative effectiveness of the three therapeutic modalities used to lower the plasma glucose level revealed that, of the 639 alive and active in 1979, 207 patients had paired plasma glucose levels of <200 mg/dl in 1969–70 and 322 patients (115 more) in 1979. In 1979, 70 more on diet and 50 more on insulin had levels of <200, but 5 fewer on sulfonylurea/phenformin had <200 mg/dl.

Standardized mortality ratios in the different demographic and treatment groups have been reported.[13]

DISCUSSION

The magnitude and diversity of the clinical, statistical, financial, and ethical problems inherent in the design, conduct, and analysis of investigations that propose to study the natural history of diabetes prospectively, longitudinally, and randomly in large populations from the time of diagnosis (inception) to the time of development of complications (morbidity) and to the time of death (mortality), and that propose to measure the safety and effectiveness of available therapeutic modalities (diet, insulin, sulfonylurea) became well-known during the seventies. The recent recognition that diabetes is heterogeneous in origin[9,10,14] suggests that the strategy of therapy to optimally control the plasma glucose level differs in the two types of diabetes [non-insulin-dependent (NIDDM) and insulin-dependent (IDDM)], and this has made the problems to be resolved even more complex.

Four prospective longitudinal studies that lasted more than 10 yr have been reported. One study dealt with the natural history of IDDM in 108 patients,[15–17] and one study dealt with the natural history of NIDDM in 1017 patients.[18–20] Two studies contained both insulin-dependent and non-insulin-dependent individuals: 4398 patients in one study[21,22] and 292 patients in another.[23,24] Reports from the Joslin Clinic[25,26] have dealt with morbidity and mortality in those with IDDM and NIDDM, with as much as 50 yr of follow-up in some patients.

The dearth of available data on the natural history of diabetes and the influence of various therapeutic modalities on outcomes prompted Knowles et al.[27] to suggest that additional prospective longitudinal data should be gleaned from clinics with good initial and follow-up records spanning periods of up to 25 yr after diagnosis. This and a companion article[13] are responses to that suggestion.

The Atlanta and Memphis cohorts reported here contained a total of 1467 patients. Although not inception cohorts (entry at time of diagnosis), both were prospective and longitudinal from time of entry. Thus, the groups are suitable prospectively and longitudinally for determination of target events cited in the numerators (dead, lost to follow-up, alive and active 1979, and plasma glucose and weight changes) but are not suitable for the determination of the statistical risks of target events (numerators) when related to time of diagnosis of diabetes, i.e., inception status (denominators).[28] In those alive and active on 31 December 1979, the mean duration of diagnosed diabetes was 15.4 yr (Atlanta mean 16.0 yr, Memphis mean 14.4 yr), and the mean age was 65.6 yr (Atlanta mean 67.2 yr, Memphis mean 62.7 yr).

Demographically, both Atlanta and Memphis cohorts were black and predominantly women (women/men ratio in each cohort was =4.7/1). Previous reports from Atlanta[9] and Memphis[5] have shown an increased prevalence of diabetes in women and have shown that women with diabetes have more excess weight than men. In Atlanta, mean maximum body weight as a percent of ideal body weight[9] was, for black women, 168% and, for black men, 135%. Mean percent of ideal body weight at time of diagnosis of diabetes was, for black women, 154% and, for black men, 116%.[29] Ninety-seven percent of those in the Atlanta cohort were above ideal body weight at the time of diagnosis, and only 3.3% of those in the Atlanta cohort were diagnosed before age 20 yr.[9] These observations, taken together, confirm and extend the observations of West[30–33] that the vast majority of those in the United States who have diabetes are, at the time of diagnosis, overweight and non-insulin-dependent.

Although initial mean random plasma glucose levels did not change significantly in either cohort during follow-up, stratification of 1979 plasma glucose levels and comparison with paired 1969–71 plasma glucose levels revealed that the 1979 levels in Atlanta were lower in 67% on diet and in 55% on insulin, and higher in 33% on diet and 45% on insulin. The 1979 levels in Memphis were lower in 40% on insulin and 83% on sulfonylurea/phenformin, and higher in 60% on insulin and 17% on sulfonylurea/phenformin. Of the 66 on diet therapy alone in 1979 in Memphis, 44 had mild diabetes (initial mean plasma glucose 115 mg/dl), which had not worsened (1979 mean plasma glucose 112 mg/dl). Mean weight loss during follow-up in this group was 13.2 lb. Of the other 22 in Memphis on diet therapy alone, 1979 plasma glucose levels were lower in 18% and higher in 82%.

By 1979, 86% of the alive and active in Atlanta had lost weight (90% of those on diet therapy alone, 72% of those on diet plus insulin therapy), and 61% of the alive and active in Memphis had lost weight (58% of those on diet therapy, 54% of those on diet plus insulin therapy, and 68% of those on diet therapy plus sulfonylurea/phenformin).

Stratification of 1979 mean plasma glucose levels for the combined cohorts and comparison with paired 1969–71 levels revealed that those with 1969–71 levels <200 mg/dl

increased from 32.4% to 50.4% for those on diet therapy alone and from 9.3% to 36.8% for those on diet-plus-insulin therapy, but decreased from 65.9% to 60.2% for those on diet-plus-sulfonylurea/phenformin therapy.

Those under the best metabolic control (random plasma glucose <150 mg/dl) in both cohorts lost significantly more weight (Atlanta 23 lb, Memphis 8.7 lb) than those under the worst metabolic control (random plasma glucose >300 mg/dl; Atlanta 14.5 lb, Memphis 1.3 lb).

Of the 36% in Atlanta and the 39% in Memphis who had higher plasma glucose levels in 1979 than in 1969–71, two-fifths had gained a significant amount of weight (mean 19.7 lb). Of the remaining three-fifths with higher 1979 plasma glucose levels, weight loss probably occurred because of poor metabolic control and increased glucosuria secondary to non-adherence to prescribed diet therapy, or diminished insulin secretion as NIDDM progressed to IDDM, or both.

Taken together, these observations suggest that weight loss in those with NIDDM as a result of restricted caloric intake is more effective in lowering the plasma glucose level than either insulin or sulfonylurea therapy.[9,10,33–35] Since aggressive diet therapy with weight reduction in those with NIDDM is probably safer and less expensive,[34] it appears to be the preferred treatment method.

Since patients were not randomized to the different treatment groups at inception, and since therapeutic strategies in the two cohorts differed, it was not possible to determine whether or not mortality outcomes were related to different treatment methods.

Standardized mortality ratios (SMR) were highest for those on insulin therapy (Atlanta 1.62, Memphis 1.78), intermediate for those on sulfonylurea therapy (Memphis 1.52), and lowest for those on diet therapy alone (Atlanta 1.33, Memphis 1.06). In the Memphis cohort, the ratio of observed/expected deaths on insulin therapy was 36/20.3 (SMR = 1.78), on sulfonylurea/phenformin therapy was 73/48.1 (SMR = 1.52 for all oral agents; 1.50 for sulfonylurea, 1.56 for phenformin, 1.56 for sulfonylurea plus phenformin), and on diet therapy alone was 33/31.2 (SMR = 1.06). These differences were not statistically significant (P = 0.082).[13] The choice of therapy in the two cohorts appeared to be based on the different strategies of therapy as related to the severity and duration of disease. This in turn was reflected in the SMR differences.

These observations, taken together with the observations of others that macrovascular lesions appear to be late manifestations of long-standing diabetes rather than being simply a product of the magnitude of hyperglycemia,[24] suggest that it may be imprudent to place sole reliance on correction of hyperglycemia in attempts to diminish mortality rates from cardiovascular disease in those with NIDDM. Although control of hyperglycemia, especially if symptomatic, is an important therapeutic objective, it seems likely that risks of mortality from cardiovascular disease(s) also may be reduced by correction of hypertension,[36,37] overweight,[38] and lipid abnormalities, and by avoiding tobacco smoking. Additional studies designed to examine the influence of these factors in NIDDM from time of diagnosis to death need to be initiated.

ACKNOWLEDGMENTS: The authors thank James Grimsley and Shirley Langella for secretarial assistance, James Johnson for administrative assistance, and Rosa Lewis for assistance in the medical records search.

This work was supported in part by the Georgia Department of Human Resources Diabetes Control Program, Contract No. 4270300673 (Dr. Davidson) and the Howell Foundation Diabetes Research Fund (Dr. Davidson), and in part by grants from the Robert Wood Johnson Foundation, Princeton, New Jersey (Dr. Runyan). The opinions, conclusions, and proposals in the text are those of the authors and do not necessarily represent the views of the Georgia Department of Human Resources or the Howell Foundation, or the Robert Wood Johnson Foundation.

From the Department of Medicine, Emory University School of Medicine, Atlanta, Georgia, and the Department of Community Medicine, University of Tennessee College of Medicine, Memphis, Tennessee.

Address reprint requests to John K. Davidson, M.D., Ph.D., Diabetes Unit, Department of Medicine, Emory University School of Medicine, 69 Butler Street, Southeast, Atlanta, Georgia 30303.

REFERENCES

[1] Runyan, J. W., Vander Zwaag, R., Joyner, M. B., et al.: The Memphis diabetes continuing care program. Diabetes Care 1980; 3:382–86.

[2] Davidson, J. K.: United States of America: The Grady Memorial Hospital experience. *In* Diabetes in Epidemiological Perspective. Mann, J. I., Pyorola, K., and Teuscher, A., Eds. London, Churchill Livingstone Press, 1983:243–49.

[3] Davidson, J. K.: Diabetes in socioeconomically deprived neighborhoods. *In* Diabetes Mellitus: Diagnosis and Treatment. Vol. 3. Fajans, S. S., and Sussman, K. E., Eds. New York, American Diabetes Association, 1971:207–10.

[4] Davidson, J. K., Alogna, M. T., Goldsmith, M. P., and Borden, J.: Assessment of program effectiveness at Grady Memorial Hospital. *In* Educating the Diabetic Patient. Lawrence, P., and Steiner, G., Eds. New York, Springer Publishing, 1981:329–48.

[5] Runyan, J. W., Phillips, W. E., Herring, O., et al.: A program for the care of patients with chronic diseases. JAMA 1970; 211:476–79.

[6] Runyan, J. W.: Decentralized medical care of chronic disease. Trans. Assoc. Am. Phys. 1973; 83:237–44.

[7] Runyan, J. W.: The Memphis chronic disease program: comparisons in outcome and the nurse's extended role. JAMA 1975; 231:264–67.

[8] Vander Zwaag, R., Mason, W. B., Joyner, M. B., and Runyan, J. W.: Cost of chronic disease care. J. Chronic Dis. 1980; 33:713–20.

[9] Davidson, J. K.: Educating diabetic patients about diet therapy. Int. Diabetes Fed. Bull. 1975; 20(2):1–5.

[10] Davidson, J. K.: Plasma glucose lowering effect of caloric restriction in obesity-induced insulin treated diabetes mellitus. Abstract. Diabetes 1977; 26 (Suppl. 1):355.

[11] American Diabetes Association: Policy Statement on the University Group Diabetes Program controversy. Diabetes Care 1979; 2:1–3.

[12] Council on Scientific Affairs: Guidelines for the non-insulin-dependent diabetic: hypoglycemic treatment. JAMA 1980; 243:2078–79.

[13] Vander Zwaag, R., Runyan, J. W., Davidson, J. K., Delcher, H. K., Mainzer, I., and Baggett, H. W.: A cohort study of mortality in two clinic populations of patients with diabetes mellitus. Diabetes Care 1983; 6:341–46.

[14] National Diabetes Data Group: Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. Diabetes 1979; 28:1039–57.

[15] Knowles, H. C.: The problem of the relation of control of diabetes to the development of vascular disease. Trans. Am. Clin. Climatol. Assoc. 1964; 76:42–47.

[16] Knowles, H. C., Guest, G. M., Lampe, J., Kessler, M., and Skillman, T. G.: The course of juvenile diabetes treated with unmeasured diet. Diabetes 1965; 14:239.

[17] Knowles, H. C.: Control of diabetes and the progression of vascular disease. *In* Diabetes Mellitus, Theory and Practice. Ellenberg, M., and Rifkin, H., Eds. New York, McGraw-Hill, 1970:666–73.

[18] The University Group Diabetes Program: A study of the effects of hypoglycemic agents on vascular complications in patients with adult onset diabetes. Part I: Design, methods and baseline characteristics. Part II: Mortality results. Diabetes 1970; 19 (Suppl. 2):747–830.

[19] Knatterud, G. L., Klimt, C. R., Levin, M. E., Jacobson, M. E., and Goldner, M. G.: Effects of hypoglycemic agents on the vascular complications in patients with adult-onset diabetes. 7. Mortality and selected non-fatal events with insulin treatment. JAMA 1978; 240:37.

[20] Knatterud, G. L., Klimt, C. R., Goldner, M. G., Hawkins, B. S., Weisenfeld, F., Kreines, K., and Haddock, C.: Effect of hypoglycemic agents on vascular complications in patients with adult-onset diabetes. 8. Evaluation of insulin therapy: final report. Diabetes 1982; 31 (Suppl. 5):1–81.

[21] Pirart, J.: Diabetes mellitus and its degenerative complications: a prospective study of 4,400 patients observed between 1947 and 1973. Part 1. Diabetes Care 1978; 1:168–88.

[22] Pirart, J.: Diabetes mellitus and its degenerative complications: a prospective study of 4,400 patients observed between 1947 and 1973. Part 2. Diabetes Care 1978; 1:252–63.

[23] Kannel, W. B., Hjortland, M., and Castelli, W. P.: Role of diabetes in congestive heart failure: the Framingham study. Am. J. Cardiol. 1974; 34:29–34.

[24] Kannel, W. B., and McGee, D. L.: Diabetes and glucose tolerance as risk factors for cardiovascular disease: the Framingham Study. Diabetes Care 1979; 2:120–26.

[25] Marks, H. H., and Krall, L. P.: Onset, course, prognosis, and mortality in diabetes mellitus. *In* Joslin's Diabetes Mellitus. 11th edit. Marble, A., White, P., Bradley, R. F., and Krall, L. P., Eds. Philadelphia, Lea & Febiger, 1971:209–54.

[26] Cochran, H. A., Marble, A., and Galloway, J. A.: Factors in the survival of patients with insulin-requiring diabetes for 50 years. Diabetes Care 1979; 2:363–68.

[27] Knowles, H. C., Meinert, C. L., and Prout, T. E.: Diabetes mellitus: the overall problem and its impact on the public. *In* Diabetes Mellitus. Fajans, S. S., Ed. (DHEW Publication no. NIH 76-854.) Washington, D.C., U.S. Government Printing Office, 1976:11–32.

[28] Kaplan, M. H., and Feinstein, A. R.: A critique of methods in reported studies of long-term vascular complications in patients with diabetes mellitus. Diabetes 1973; 22:160–74.

[29] Hamwi, G. J.: Therapy: changing dietary concepts. *In* Diabetes Mellitus: Diagnosis and Treatment, Vol. 1. Danowski, T. S., Ed. New York, American Diabetes Association, 1964:73–78.

[30] West, K. M., and Kalbfleisch, J. M.: Influence of nutritional factors on prevalence of diabetes. Diabetes 1971; 20:99–108.

[31] West, K. M.: Diet therapy of diabetes: an analysis of failure. Ann. Intern. Med. 1973; 79:425.

[32] West, K. M.: Diet and diabetes. Postgrad. Med. 1976; 60:209.

[33] West, K. M.: Epidemiology of Diabetes and its Vascular Lesions. New York, Elsevier, 1978.

[34] Davidson, J. K., Delcher, H. K., and Englund, A.: Spin-off cost/benefits of expanded nutritional care. J. Am. Dietetic Assoc. 1979; 75:250–57.

[35] Davidson, J. K., and Goldsmith, M. P.: Diabetes Guidebook: Diet Section, 3rd edit. Atlanta, Davicone, Inc., 1982.

[36] Hypertension Detection and Followup Program Cooperative Group: Five-year findings of the Hypertension Detection and Followup Program. I. Reduction in mortality in persons with high blood pressure, including mild hypertension. JAMA 1979; 242:2562–71.

[37] Christlieb, A. R.: The hypertensions of diabetes. Diabetes Care 1982; 5:50–58.

[38] Van Itallie, T. B.: Obesity: adverse effects on health and longevity. Am. J. Clin. Nutr. 1979; 32:2723–33.