JUVENILE DIABETES MELLITUS AT KENYATTA NATIONAL HOSPITAL : CONTROL PARAMETERS, LEVEL OF CONTROL AND THE FACTORS WHICH INFLUENCE THIS CONTROL

by

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D E C L A R A T I O N S

CANDIDATE:

This thesis is my original work and has not been presented for the award of a degree in any other University.

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This thesis has been submitted for examination with my approval as University Supervisor.

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2. Dr. E. N. Mngola for allowing me to conduct the study among diabetes clinic patients.
3. Dr. J. A. Grayburn was the project supervisor. He has been of invaluable help in all the stages of this project. He has readily availed himself for consultation and suggestions and for this I am very grateful to him.
4. Mr. L. Nyabola of the Department of Community Medicine, University of Nairobi, helped me with the statistics and for this I am grateful to him.
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C O N T E N T S

S U M M A R Y

40 Juvenile Diabetics attending the Diabetes clinic, Kenyatta National Hospital are studied. These form 11% of the Juvenile Diabetics population and 1.7% of the total population of diabetics as registered in the Kenyatta National Hospital Diabetes Clinic Register by September, 1977.

In only ¼ of these patients is diabetes controlled (using the parameters of : - a single determination of fasting plasma glucose, a 2 hours post prandial plasma glucose and fasting urine glucosuria by both clinistix and clinitest).

Some of the causes of this poor control are investigated and an attempt is made to correlate control using the level of glucosuria to control using the fasting plasma glucose level. Literature is reviewed in connection with the merits and demerits of rigid diabetes control and criteria of diabetes control used in the past and those currently in use. Some suggestions are made as to how better control can be achieved in our population and further a few future research projects in the field of diabetes are suggested.

INTRODUCTION

It is now more than fifty years since Banting and Best first isolated insulin from a dog's pancreas. Soon after, this extract was also obtained from cows and pigs. Use of this extract on patients with diabetes gave very encouraging results and mortality from keto-acidosis and hyperglycaemia immediately dropped. Oral hypoglycaemic agents have now been in use for just over twenty years. In the control of diabetes mellitus, diet-in addition to either oral hypoglycaemic agents or insulin are used. Their use is not an overall success story and even today the World's Medical opinion is divided as regards the merits and demerits of good diabetic control.

Many people believe that good control minimizes or prevents diabetic complications (Marble,A; Hardin, R.C., et al). Others believe that control apparently makes no difference (Knowles,H.C. Jr.; Siperstein,M.D., et al). Lack of good controlled studies leaves this issue unsettled and this has led to divergent philosophies on management of diabetes. Some of the so-called complications of diabetes have been reported in patients without diabetes (Ellenberg,M.) The drugs used are not without risk and it has even been suggested that

the insulin perse might give rise to the various complications seen in diabetes (Knowles, H.C. Jr., et al).

Reported in Diabetes, March 1976, and Diabetes Outlook, vol. II No. 7, September 1976, is the American Diabetic Association policy statement advocating rigorous control of blood sugar in diabetics. The goal of therapy should be a serious effort to achieve blood glucose levels as near as possible to those of non diabetics particularly in the young and middle aged patients who are at greatest risk of developing microvascular complications. Engerman, R. et al findings that reduction of blood sugar prevents or minimizes the formation of diabetic like lesions, biochemical demonstrations of an association between hyperglycaemia and sorbital (deposited in the eyes, nerves and vascular tissue) and glycosalation of haemoglobin and basement membrane considered. The recent Prospective French study demonstrating that enthusiastically well controlled diabetics have less diabetic retinopathy than those not well controlled has also been considered.

This Policy statement has been the subject of a lot of criticisms and is said to be based on non-convincing demonstrations.

The prevalence of diabetes among the indigenous Kenya Africans has been steadily increasing over the last twenty years. In 1955, there were 14,635 Africans admitted to the King George VI Hospital (present day Kenyatta National Hospital). 17 of these (0.12%) were diabetics (Manson-Bahr). By 1959 the number of African diabetics had risen to 40 (0.36%) among total African admissions of 11,099. In 1959, Ojiambo, H.P., had 4 diabetics among 5,668 patients admitted to Kiambu District Hospital. In 1960, diabetics accounted for 0.25% of the total admissions to Central Province General Hospital, Nyeri (Hadman, D.F.). The limitations of considering hospital admissions in determining the prevalence of a disease is realised.

By 1966, the magnitude of the problem had become sizeable necessitating the starting of a clinic at Kenyatta National Hospital wholly devoted to the problem. By 1975, December, 1666 patients had been registered (Abdullah, M.S.). Patients are recruited into the clinic as referrals from the various clinics and wards in Kenyatta National Hospital and clinics in and around the city. By September, 1977, 2,405 patients had been enrolled. On the average there are 30 patients enrolled per month. At the rate of a new diabetic per day among the population served by this hospital (mainly Africans), it can no longer be said that diabetes

is rare among Africans.

The prevalence of diabetes among the Kenya Africans has not yet been established. Basing their study on a very limited sample, Mngola, et al found a prevalence rate of the disease in the Nairobi population of about 0.43%. Vogel suggests that for Nairobi the prevalence is about 1%.

There is lack of uniformity as regards the parameter(s) of control of diabetes in the various studies or clinics that deal with this problem. Good control, bad control and even just controlled mean different things to different people. This lack of standardization of the control parameter(s) and the degrees of control or lack of control makes it difficult to compare different studies especially in trying to correlate the degree of control and the development of complications of diabetes. Root, H.F., et al (1954) categorized levels of control as excellent, good, fair or poor depending on adherence to diet, insulin administration, urine testing, blood sugar levels and the number of times that a patient had been in coma. In his "Study of the Incidence and causes of poor control in patients with Diabetes Mellitus," Stone modified Root's et al criteria such that he divided his patients into good, fair or poor levels of control. J.M.Steel, et al have in their paper on "Diabetic Retinopathy in Kenya" classified degrees of control according

to the averages of blood sugars recorded at the morning clinics. They have the degrees of good, fair or poor according to average blood sugars of 175 mg % or less, 175 mg % to 250 mg % and 250 mg % or more respectively. A patient who has had three or more hospital admissions for poor diabetic control after the initial diagnosis is considered to be poorly controlled.

Malone et al have summarized the criteria of diabetic control from the data of others:

	Excellent	Good	Fair	Poor
Fasting blood Glucose <110 mg/dl (% of blood tests)	100%	85%	70%	< 70%
2 hr. post prandial blood glucose <150 mg/dl (% of blood tests)	100%	85%	70%	< 70%
Urine glucose gm/24 hours	0	0 - 25	25 - 50	> 100
Urine specimens free of sugar (% of urines tested)	100%	75%	50%	< 50%
Urine Acetone (mg/dl)	0	0	0 - 10	> 30%

Benner et al write :- " We consider that the patient is in adequate continuous therapeutic control if when 85% of the post prandial urine specimens and, more importantly, 85% of the 24 hour urine specimens are free of sugar or when 2 hour post prandial true blood sugar values are less than 150 mg%. 85% of the times these determinations are made." Other parameters used in determining the degree of control include : - dose of insulin, tendency to keto acidosis and hospital admissions, symptoms, plasma lipids (cholesterol, β lipoproteins and high density lipids). Malone et al have pointed out the variability of the level of control in the same. patients at the same time if various parameters of control are considered. Even using one parameter the level of control is not uniform throughout the whole day and varies from day to day. 24 hour total urinary glucose excretion only indicates control in that particular day. It is increasingly being shown that the amount of glycosylated haemoglobin, especially HbA_{1C} is directly proportional to the degree of hyperglycaemia that has been prevailing for a couple of weeks or months before the determination (Koening, R.J. et al). This parameter does not fluctuate so much as the others.

In our Diabetes Clinic at Kenyatta National Hospital control and treatment are based on amount of glucosuria as determined by clinitest tablets and for those with glucosuria of 3+ or 4+ blood sugar is done using the Reflomat glucose machine. At home patients often use the various types of urinary sticks to determine their amount of glucosuria. How the amount of glucosuria by the sticks correlates to the amount of glucosuria by clinitest tabletes, or even blood sugar, has not been determined in our population.

The purpose of this study is to analyse the degree of control of the juvenile diabetics in our clinic by several different parameters and assess their relative efficacy in our population. In addition some of the factors influencing control are determined. It is assummed but not proven, that the 40 patients studied would fit into Hugh-Jones Type I group of diabetics and none would fit into the type II group of diabetics. In this clinic juvenile diabetics are those who manifest the disease at the age of 30 years and below. By September, 1977, juvenile diabetics formed 15.3% of the total diabetic population (368 out of the total of 2405). The patients studied, 40 patients, are only 11% of the total number of juvenile diabetics and 1.7% of the total diabetic population.

MATERIALS AND METHODS

In our clinic juvenile diabetics are regarded as those who got their disease at the age of thirty years and below. For the purpose of this study these were identified from the Diabetic Clinic Register. An attempt was made to study all those juvenile diabetics who attended the clinic during the period of study.

For each patient a previously drawn up proforma was filled up (See appendix for proforma and reasons for the various questions and examinations). Also done for each patient were : - a fasting plasma glucose, a 2 hour post prandial plasma glucose. Fasting urine glucosuria determination (by both clinistix and clinitest) and a 24 hour total urinary glucose excretion. Glucose oxidase method (Muller, 1925) was the method used for plasma glucose determinations. (In our laboratory guiacum is used as the chromogen acceptor). Fasting urine glucosuria by both clinistix and clinitest was determined according to the manufacturer's instructions. (A handbook of chemical and biological information systems - Ames Company). For the fasting urine glucosuria, urine was collected into clean bottles without any preservative. For the 24 hour urine specimens patients were provided with two to three litre bottles into which 10 mls of strong hydrochloric acid had been added to act as preservative. Patients had been instructed to

start the urinary collection 24 hours prior to the morning of the clinic and then submit these specimens as they came to the clinic. Glucose oxidase method was used to determine the amount of glucose in the twenty four hour urine specimens. To be able to use the same spectrophotometer, and the standard solutions glucose curve that is drawn daily, it was found (by trial and error) that the urine had to be diluted twenty times more than the blood usually is. The twenty four hour urine specimens were checked as to whether they were 24 hours urine specimens or not by determining the total creatinine in them (on a 24 hour urine, 15 mg or more creatinine per kg body weight is excreted - Cook, R.E.) Only the true 24 hour urine specimens are analysed further.

All the specimens were analysed on the same day that they were submitted. Only 40 patients were fully worked up as per requirements of this study.

RESULTS

TABLE I GENERAL INFORMATION

PATIENT	CASE NO.	SEX	AGE AT ONSET (YRS)	AGE DEC. '77 (YRS)	DURATION (MONTHS)
F.M.	1	M	21	23	28
G.O.	2	M	17	17	10
M.W.	3	M	20	24	54
S.M.	4	M	18	19	16
R.M.	5	F	18	20	24
D.M.	6	M	20	27	24
J.M.	7	M	24	27	36
J.M.	8	M	24	28	58
F.L.	9	M	27	29	24
U.K.	10	F	15	18	36
E.N.	11	F	25	26	12
M.W.	12	F	28	33	60
H.T.	13	M	22	24	24
G.K.	14	M	28	24	12
B.M.	15	F	14	15	12
A.W.	16	F	13	17	48
W.W.	17	M	13	14	12
J.K.	18	M	29	30	12
P.W.	19	F	25	26	17
N.O.	20	M	24	24	8
F.N.	21	M	21	25	59
K.N.	22	M	16	19	36
K.C.	23	M	6	12	72
N.G.	24	M	18	29	132
G.K.	25	M	17	34	204
M.W.	26	M	21	21	3
R.W.	27	F	25	32	91
J.K.	28	M	29	30	16
N.J.	29	F	23	23	4

PATIENT	CASE NO.	SEX	AGE AT ONSET (YRS)	AGE DEC. 1977 (YRS)	DURATION (MONTHS)
A.G.	30	F	25	30	62
K.C.	31	M	27	33	79
P.N.	32	M	16	18	25
C.K.	33	F	NOT KNOWN	NOT KNOWN	NOT KNOWN
J.N.	34	F	22	22	6
P.M.	35	M	22	27	61
C.G.	36	M	19	22	44
E.N.	37	M	11	19	96
J.N.	38	M	25	28	38
G.G.	39	M	27	31	50
J.N.	40	M	16	21	67

In nearly all cases treatment was started soon after the start of symptoms. Total number males is 29. Total number females is 11. This gives a ratio of about 3 males for every one female in the study. Analysis of diabetic register up to September 1977, shows almost equal numbers for both male and female juvenile diabetics - 185 males and 183 females. Sexwise therefore, the study population is not representative of the juvenile population attending the Diabetes Clinic.

In this study group the disease occurred earliest at the age of 6 years and latest at 29 years (range of 23 years). The mean age of onset is 20.8 years with standard deviation of $\pm$ 5.44 years. The mean age by December, 1977, was 24.3 years (standard deviation 38.8 months) and range of 201 months (shortest is 3 months while the longest is 204 months.)

TABLE 2 PARAMETERS OF CONTROL AND RESULT

		PG (mg/dl)		FASTING URINE		CONTROL				
CASE NO.	UG GM/24 HR.	FPG	PPG	CLINISTIX	CLINITEST	UG 24 HR.	FPG	PPG	CLINISTIX	CLINITEST
1	-	220	240	DARK	4+	-	NC	NC	NC	NC
2	-	220	117	DARK	2+	-	NC	C	NC	NC
3	18	130	205	DARK	TRACE	G	NC	NC	NC	C
4	7	210	260	DARK	4+	G	NC	NC	NC	NC
5	-	345	320	DARK	3+	-	NC	NC	NC	NC
6	68	208	152	DARK	4+	P	NC	NC	NC	NC
7	-	175	135	DARK	4+	-	NC	C	NC	NC
8	62	117	110	DARK	4+	P	NC	C	NC	NC
9	30	145	220	NEGATIVE	NEGATIVE	F	NC	NC	C	C
10	8	195	210	DARK	2+	G	NC	NC	NC	NC
11	-	195	245	MEDIUM	3+	-	NC	NC	NC	NC
12	-	300	130	DARK	4+	-	NC	C	NC	NC
13	-	175	180	DARK	4+	-	NC	NC	NC	NC
14	43	50	225	NEGATIVE	NEGATIVE	F	C	NC	C	C
15	-	334	210	DARK	4+	-	NC	NC	NC	NC
16	-	165	510	DARK	4+	-	NC	NC	NC	NC
17	-	220	80	MEDIUM	2+	-	NC	C	NC	NC

Table 2 (Contd.)

18	-	80	40	LIGHT	NEGATIVE	-	C	C	C	C
19	0	85	100	NEGATIVE	NEGATIVE	G	C	C	C	C
20	1	152	85	NEGATIVE	NEGATIVE	G	NC	C	C	C
21	-	275	300	DARK	3+	-	NC	NC	NC	NC
22	189	205	255	LIGHT	2+	P	NC	NC	C	NC
23	61	370	190	LIGHT	2+	P	NC	NC	C	NC
24	-	296	310	DARK	4+	-	NC	NC	NC	NC
25	-	400	240	LIGHT	2+	-	NC	NC	C	NC
26	79	150	70	DARK	4+	P	NC	C	NC	NC
27	1	172	250	NEGATIVE	NEGATIVE	G	NC	NC	C	C
28	36	125	67	DARK	3+	F	NC	C	NC	NC
29	-	195	270	DARK	4+	-	NC	NC	NC	NC
30	-	210	330	NEGATIVE	NEGATIVE	-	NC	NC	C	C
31	44	110	260	LIGHT	2+	F	C	NC	C	NC
32	82	415	415	DARK	4+	P	NC	NC	NC	NC
33	-	405	287	DARK	4+	-	NC	NC	NC	NC
34	2	70	85	NEGATIVE	NEGATIVE	G	C	C	C	C
35	-	60	350	DARK	4+	-	C	NC	NC	NC
36	-	370	215	DARK	4+	-	NC	NC	NC	NC
37	85	25	100	NEGATIVE	NEGATIVE	P	C	C	C	C
38	150	340	255	DARK	4+	P	NC	NC	NC	NC
39	133	300	325	DARK	4+	NC	NC	NC	NC	NC
40	54	107	40	DARK	4+	P	C	C	NC	NC

PG = Plasma Glucose

UG = Urine Glucose

FPG = Fasting Plasma Glucose

PPG = Post Prandial Plasma Glucose

C = Controlled

NC = Not Controlled

Negative, Trace, 1+, 2+, 3+ and 4+ glucosuria by clinitest semiquantatively represent glucosuria of 0 mg%, ¼ mg%, ½ mg%, ¾ mg%, 1 mg% and 2 mg% respectively.

Patients with FPG of 110 mg% and below are considered as controlled. Those with FPG above 110 mg% are considered as not controlled. Patients with PPG of 150 mg% and below are considered as controlled. Those with PPG above 150 mg% are considered as not controlled. Negative, Trace and Light glucosuria by clinistix is taken as controlled while above that it is taken as not controlled. Negative, Trace and 1+ glucosuria by clinitest is taken as controlled while above that the patient is considered as not controlled.

Only about a half of the patients (21 out of 40) obeyed the instructions that they submit a 24 hour urine sample. The others submitted a specimen which was not a 24 hour specimen. In the control of diabetes a patient has to obey instructions and the degree of control very much depends on his cooperation.

By the parameter of FPG, 32 patients (80%) are not controlled and only 8 (20%) are controlled. By the parameter of PPG 27 patients (67.5%) are not

controlled and only 13(32.5%) are controlled. FPG and PPG correlate in 29 cases (5 patients controlled by both parameters while 24 patients not controlled by both parameters). In 11 cases there is no correlation. It should be noted that the FPG and PPG had been done on different clinic days.

Comparing the FPG and Fasting urine glucosuria by clinistix shows that the two parameters agree in 31 cases (6 patients controlled by both parameters while 25 patients not controlled by both parameters). In 9 cases they disagree. It should be noted that the FPG and the Fasting urine specimens were obtained on the same morning but a few hours apart.
Fasting urinary glucosuria by clinistix gives 13 controlled patients (32.5%) and 27 not controlled (67.5%)

Comparing the FPG and Fasting urine glucosuria by clinitest shows that the two parameters agree in 32 cases (5 patients controlled by both parameters while 27 patients not controlled by both parameters). In 8 cases they disagree.
Fasting urinary glucosuria by clinitest gives 10 controlled patients (25%) and 30 not controlled (75%).

TABLE 3 AN ATTEMPT TO CORRELATE CLINITEST AND CLINISTIX FINDINGS ON THE SAME SAMPLE OF FASTING URINE

CLINITEST	CLINISTIX: NEGATIVE	LIGHT	MEDIUM	DARK
NEGATIVE	8	1		
TRACE				1
1+				
2+		4	1	2
3+			1	3
4+				19

Dark glucosuria by clinistix corresponds to 4+ glucosuria by clinitest tablets in most cases and in a few cases either 3+ or 2+. Negative glucosuria by clinistix corresponds to negative glucosuria by clinitest tablets. The erroneous impression is given that trace glucosuria by clinitest is equivalent to dark glucosuria by clinistix. This is most likely due to the small numbers involved in this study. Clinistix and clinitest glucosuria determinations are semiquantative methods and although glucosuria fails to relate to the plasma glucose (Service, F.J., et al),

clinitest tablets are widely used in indicating the level of control in diabetics. The lack of predictable correlation between plasma glucose and glucosuria is due to the fact that glucose renal threshold in patients is not a constant fixed value in each patient or between the various patients. It takes time for plasma glucose to be expressed as glucosuria and even the double void method of urine collection does not solve this problem. A third void urine specimen has been suggested (Service, F. J. et al).

Table 3 brings out the correlation between Clinistix and clinitest tablets. Table 2 shows that the clinistix and clinitest tablets correlate in 35 cases and only disagree in 5 cases. Most of our Out-patient diabetics use the sticks for urine sugar determinations. It is suggested that calibrating the sticks to replace the clinitest tablets as a rough parameter of control for the Out-patient diabetics will give an easier more handy method which is just as effective as the clinitest tablets. The calibration could be done by using total 24 hour urinary glucose excretions in diabetics.

The general impression is that a ¼ of the patients in this study group are controlled while in the remaining ¾ diabetes is not controlled. Although the study group is not a representative sample of the total diabetic population in our clinic, probably the same conclusion applies for the entire population. This clinic population is therefore an ideal set up for studying the effects

on the body tissues of controlled hyperglycaemia versus those of uncontrolled hyperglycaemia.

Table 4 CONTROL IN RELATION TO NUMBER OF ADMISSIONS AFTER THE INITIAL ADMISSION

CASE	CONTROL STATUS BY PPG	NUMBER OF ADMISSIONS	DURATION AFTER DIAGNOSIS	COMMENTS
1	NC	0	-	-
2	C	0	-	-
3	NC	2	2 years	Hypoglycaemic coma - Unexplained
4	NC	1	1 year	Hypoglycaemic coma - Unexplained
5	NC	2	1 year, 2 years	Hyperglycaemia due to poor control
6	NC	>10	All along	Hyper and Hypo states. No fixed abode
7	C	0	-	-
8	C	0	-	-
9	NC	0	-	-
10	NC	1	Same year	Failing vision
11	NC	0	-	-
12	C	2	4 years, 5 years	Pneumonia and Pharyngitis respectively
13	NC	3	-	All in hypoglycaemica coma
14	NC	0	-	-
15	NC	0	-	-
16	NC	3	-	Injection abscesses x 2. Failing vision
17	C	0	-	-
18	C	0	-	-
19	C	1	1 year	Hyperglycaemia
20	C	1	6 months	Hypoglycaemic coma

CONTROL IN RELATION TO NUMBER OF ADMISSIONS AFTER THE INITIAL ADMISSION (Contd)

CASE	CONTROL STATUS BY PPG	NUMBER OF ADMISSIONS	DURATION AFTER DIAGNOSIS	COMMENTS
19	C	1	1 year	Hyperglycaemia
20	C	1	6 months	Hypoglycaemic coma
21	NC	1	2 years	No comment
22	NC	Many times	All along	Injection abscesses x 3. Financial Problems
23	NC	4	All along	Hyperglycaemia
24	NC	>5	All along	Hyperglycaemia - sometimes in coma
25	NC	4	All along	Hyperglycaemia
26	NC	0	-	-
27	NC	1	Two years	Poor control following delivery
28	C	1	8 months	Hypoglycaemic coma
29	NC	0	-	-
30	NC	1	2 years	Hyperglycaemia
31	NC	1	1 month	Hypoglycaemia
32	NC	2	1 and 2 years	Hypo and Hyper states
33	NC	4	1,2,2,5, years	Poor control following delivery
34	C	0	-	-
35	NC	2	2, 4 years	Hyperglycaemia
36	NC	1	2 years	Hyperglycaemia
37	C	0	-	-
38	NC	1	1 year	Hyperglycaemia
39	NC	1	2 years	Poor control due to TB
40	C	1	5 years	Hypoglycaemia with transient hemiparesis

Table 4 brings out the point that in cases like ours where diabetics are poorly controlled in most cases, the number of admissions after the initial admission cannot be used as a parameter of control. Hypoglycaemic reactions and coma are common in this study group (similar finding reported in "Diabetes Mellitus in the Tropics.") A study to determine the aetiology of hypoglycaemic reactions in our diabetics is suggested.

Taking PPG as the control parameter, factors affecting control will be reviewed :-

SEX :

3 out of the 11 females (27%) are controlled while 10 out of the 29 males (34%) are controlled. These numbers are too small for the application of statistical analysis. With pregnancy, females will be expected to be poorly controlled (Table 4).

DURATION OF THE DISEASE:

Range in the studied population : - 3 months to 17 years. The patient who has had diabetes for three months is controlled while the one who has had the disease longest is not controlled.

Duration of the disease does not seem to influence the control status.

AGE AT ONSET OF THE DISEASE:

Range : 6 years to 29 years. The patient who has had the disease since the age of 6 years is now 12 years and is not controlled. The two patients who have had the disease since the age of 29 years

are controlled. On the whole age at onset does not appear to influence control or the lack of it. The role of growth hormone in diabetes does not appear to have been resolved. The current view seems to be that the high levels of this hormone in diabetics is as a consequence of the metabolic disturbance (Vigneri, R. et al). The hormone is diabetogenic (Harrison's Principles of Internal Medicine) but the higher levels in juvenile diabetics does not seem to be a cause of poor control of diabetes.

LEVEL OF EDUCATION AT ONSET OF THE DISEASE AND LEVEL AT PRESENT OR LEVEL EVENTUALLY ACHIEVED:

These seem to have no effect on the control or lack of it. A University graduate and a forth year University student are not controlled while a first year University student is controlled. The patient with the least schooling is one who went up to Class 4 and apparently stopped there when the disease manifested itself.

24 patients stopped schooling at the level at which their disease developed. Most of these developed their disease and stopped schooling at class 7 and a few at form IV but there are also some who developed the disease at class 6, form I or II. This seems to have made them stop their schooling. At the end of Class 7 and Form IV there are public examinations and stress might be implicated as having precipitated the disorder.

Students need not stop education just because they have developed diabetes. A lot of public health education is required to prevent this catastrophe.

FAMILY HISTORY OF DIABETES MELLITUS:

Table 5

CASE	RELATIVE(S) WITH DIABETES	OUTCOME OF MANAGEMENT
2	Grandfather	Controlled
9	Father	Not controlled
22	Mother	Not controlled
23	Mother	Not controlled
24	Sister	Not controlled
37	Three brothers	Controlled

6 patients (15% of the study group) have a positive family history of diabetes mellitus. The others probably form the propands of the disorder in their respective families. A positive family history or the lack of it does not seem to influence whether a patient is controlled or not. Basically diabetes is a genetic disorder and the recent demonstration of common HLA antigens in acute onset juvenile diabetics (Cudworth, A.C. et al) renders further support to this statement. Possession of these antigens seems to predispose the individual to infections with diabetogenic viruses (Cudworth, A.C., et al; Steinke, J., et al; Gamble, D.R.). This is especially so for Coxsackie B 4 viruses.

INSULIN AND ITS ADMINISTRATION :

Case 23 had a diabetic mother who measured insulin for him and injected him. All the other patients measured their own doses and injected themselves except case 26 who initially had hyperglycaemic osmotic vision disturbance and had to rely on staff in his local health centre to administer insulin to him. He later improved and is one of the controlled 13. Most of the patients accurately demonstrated the dose of insulin that they were on but when given a different strength of insulin most could not accurately measure the dose.

Insulin storage facilities are non ideal. Only 4 patients kept their insulin in refrigerators. 27 kept theirs either in a cupboard or box. 10 of these were controlled. 6 kept theirs in an open space. None of these was controlled. 3 kept theirs in a tin with cold water. One of these was controlled. Storage period ranged from two weeks to many months.

Though not brought out in this study, non ideal insulin storage facilities especially if prolonged has been shown to decrease insulin potency (Stephenson et al; Krogh et al). Often the Pharmacy can only provide one type of insulin and not the others and one strength and not the other. This has been Shown to confuse patients and could be one cause of poor control.

URINE TESTING AT HOME:

7 patients do not examine their urine at home. 2 of these 7 are controlled. 16 patients use one or other of the various types of sticks (mostly clinistix or uristix but sometimes bililabstix as well). In the presence of consistently heavy glucosuria 9 of these 16 patients either adjust their dose of insulin or take less carbohydrates. 7 took no action on their urinary findings. 5 of these 16 patients are controlled. The remaining 17 patients used clinitest tablets but when these were not available they used the urinary sticks. 6 of these 17 patients take no action as regards their urinary findings. Thus in total 16 patients in the study population test their urine and take no action on their findings. The 13 controlled patients comprise of 5 patients from the group that test their urine by one method or the other and take action on their urinary findings while the other 8 are among those who either do not test their urine or test their urine and do nothing about their findings.

It is noted in this study that testing urine for glucosuria does not appear to affect control of diabetes one way or the other. The wide usage of the sticks is noted.

DIET:

Most patients tried to conform with the general instruction that they eat more proteins and vegetables and less of carbohydrates. Financial problems (25%

of the patients in this study group) often prevent patients from adhering to the recommended diet. Carbohydrates are relatively cheap whereas proteins and vegetables are at times costly. Strict adherence to a specific number of calories per day is not often emphasized for our diabetics and hence no patients ever weighed their food. They eat just enough unless they are warned at the clinic that they are putting on weight.

Faulty diet might be the cause of general poor control in our diabetics and this trend can be seen to continue until per-capita income improves to enable people to eat what they want or what is requested of them. Some degree of lack of control might therefore of necessity have to be accepted.

INFECTIONS:

These are not expected to be the cause of chronic poor control but case 39 does claim that his lack of control started in 1975 when he developed pulmonary tuberculosis after he had been a diabetic for only two years. Acute infections in a diabetic often lead to acute metabolic decompensation with the development of hyperglycaemia and ketoacidosis sometimes. This was observed with infections of common cold, infection abscesses and phanyngitis. The role that glucagon plays in the hyperglycaemia of acute infections has been pointed out (Thorner, M.O. et al). No reference has been found for the possible effect of chronic tropical parasitic infections

in relation to poor control of diabetes in the tropics.

MISCELLANEOUS FACTORS:

Obesity is not a problem among the patients in this group (most of them had ideal body weights by European/American standards). There were no patients on drugs that worsen hyperglycaemia. Endocrine disorders in which hormones antagonistic (directly or indirectly) to the action of insulin occur were not detected. Occupation, marital status, and home distance from clinic were not seen to influence control or the lack of it.

D I S C U S S I O N

In Paediatric practice in Kenya diabetes is uncommon. Both in our population and in other parts of the world the prevalence of diabetes has been shown to increase with age. Most diabetics, especially the young and middle aged ones eventually develop complications after sometime. African diabetics in Kenya are no exception (Mngola - Health and Disease in Kenya). In the indigenous Kenyans (diabetics and non diabetics alike) coronary arteries disease is rare unlike among the Americans and Europeans (Chukwuemeka,A.C. et al; Bradley,R.F.,et al; Robertson,W.B.). What the high fibre diet has to do with atherosclerosis is now being studied.

In this study it has been shown that the majority of our diabetics are not controlled. A diabetic is either controlled or not controlled. Table 2 does show that the various parameters of control in use in our clinic do not always agree in placing a patient as either controlled or not controlled. This variability has been pointed out in other studies. The day to day variation in the degree of control has also been pointed out. The need for a less variable parameter is greatly felt. Until such a parameter is eventually found (HbA_{1C} might be the answer), in the control of diabetes one has to choose one parameter and apply it uniformly to all patients. Fasting plasma glucose is a less variable parameter but its incovenience to the

patient excludes it from routine use. The 2 hour post prandial plasma glucose is even less reliable and it can be seen to vary with many factors. In our clinic and in most of other clinics world all over control is based on the amount of glucosuria using the clinitest tablets. Service,et al have demonstrated the inadequacy of glucosuria as a reflection of the blood glucose but until "a non invasive procedure " for determining plasma glucose is invented one day, control based on glucosuria has to continue. Methods for determining glucosuria especially by the urinary sticks (commonly used by our patients) using the glucose oxidase should be perfected and their interpretation made more meaningful both to the patient and the doctor.

Lack of control of diabetes is often multifactorial. This is especially so when one considers a group other than individual patients. This study has not identified "the cause of poor control" among our juvenile diabetics. It is suspected but not proven that faulty diet plays a very big role in this. Cruickshank refers to the diet for a diabetic in the tropical countries as "of necessity high in carbohydrates and low in proteins and fat, frequently, varies in quantity with the changing fortunes of the patient who tends to live a day to day existence." 25% of the patients in this study population had socio-economic problems and often could not conform to a

diabetic diet. Diet problems also prevail in the advanced countries (Stone; Turnbridge; Wharton, et al.). The value of repeated education sessions among the diabetics has been emphasized by Graber, A.L., et al., Stone and Williams. Patients' morale and motivation should be boosted often. Work-load and the often changing doctors as regards the diabetes clinic staff behoves the nurses, social workers, nutritionists and the organ of Diabetes Association to carry on most of the patients' education.

Difficulties in measuring the dose of insulin especially when a patient is changed from one strength to another, deteriorating in insulin patency (Pringle, M., et al.; Stephenson, et al.; Krogh, et al.), and actual omission of the dose for one reason or the other can all be seen to affect the degree of control. Insulin antibodies are not determined in the study but the observation that some of the patients require very high doses of insulin may indicate that this problem exists. Monocomponent insulin is not readily available in our country but even in countries where it is available it is said not to have eradicated the problem of insulin antibodies. The problem of hypoglycaemia, dose of insulin and meals (quantity and regularity or more often irregularity) is a real one as seen

in Table 4 and requires further study. Introduction of a single type of insulin (which should be always available) preferably of 100 units per ml. for the outpatients, and insulin syringes calibrated in tens up to 100 mark can help greatly in alleviating some of the problems connected with insulin administration.

SUGGESTIONS FOR RESEARCH PROJECTS IN THE FIELD OF CONTROL OF DIABETES

1. Determination of Normal levels of the HbA_{1C} in Kenyan Africans - People who live mainly on high carbohydrates diets. Are the levels higher than in the western world ?
2. Levels of HBA_{1C} in the diabetics and the relationship to diabetes control.
3. In the absence of refrigeration, alternative methods for storage of insulin for the out-patients and the peripheral hospitals.
4. Meal patterns (especially the single heavy evening meal for the workers) and insulin administration.
5. Diets studies in the various communities in the country to establish the daily intake of carbohydrates, fats and proteins so that if dietary modifications are requested of a diabetic, there is already an established known framework on which to base the desired changes.

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APPENDIX

Proforma and reasons for the various considerations:-

1. Name of the patient.

2. Sex of the patient.

Hormonal differences and other physiological situations like pregnancy and parity might affect the degree of control on sex basis.

3. Age at onset of disease and age at time of study :-

With maturity goes responsibility. This may mean better chances of control. With the passage of time the patient is expected to become familiar with the disease, accept the situation and appreciate the big role that he has to play in his own management. This should lead to better control.

4. Level of education at onset and furthest attained :-

Find out whether level of education correlates with degree of control. Ideally education should lead to better control.

5. Family History of diabetes :-

If present this might serve to supply company and consolation. Hopefully this will lead to better control. In a wider population it might identify families with particularly difficult diabetes to control.

6. Measurements of Heights and weights to rule out obesity and overweight. Physical examina-

tions to rule out thyrotoxicosis, acromegaly, Cushing's syndrome, Phaechromocytoma, infectious and enquiring as regards history of certain drugs like steroids and thiazide diuretics.

In all these conditions hyperglycaemia has been demonstrated to be worsened in those who have diabetes predisposition.

7. Marital Status.

Marriage involves commitment and determination. Is this spirit reflected in the control of diabetes ?

8. Who measures the dose of Insulin, who injects, testing whether the patient can measure a requested dose of insulin given different strengths and insulin storage facilities -

- Insulin potency deteriorates especially if the hormone is kept under adverse conditions.

9. Home distance from Nairobi and Socio-economic status.

These give rough indications of whether a patient can afford to come to clinics as often as necessary and whether he can afford the extra expenses that being a diabetic compels him to.

10. Diabetic diet and whether foods ever weighed.

11. Whether urine ever tested at home, with what it is tested and what action is taken as regards the findings.

- Somebody has said that if patients are going

to test urine and do nothing about it when something needs to be done, then they might just as well not examine the urine.

12. Number of admissions and in what state - General information and see whether this correlates with the degree of control.

13. Determinations of :-

a) Fasting Plasma glucose.

b) A 2 hour post prandial plasma glucose

c) Fasting urine glucosuria - by clinitest and clinistix

d) Total 24 hour urinary glucose excretion.