ir ants requires heightened awareness from all health care w rkers in the region.

This study was supported in part by the University of Natal R search Fund, the Medical Research Council of South Africa, and th > AIDS FIRCA Fogerty Foundation, USA.

We thank Kevin de Kock, CDC Atlanta, for reviewing the m nuscript; Cathy Connolly for reviewing the statistical c culations; Fikile Sibanyoni for conducting most of the HIV c unselling with the mothers; the medical and nursing staff at King E ward VIII and King George V hospitals; and the mothers for p ticipating in the study.

R erences

1. Murray C, Lopez AD. Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. *Lancet* 1997; **349:** 1436-1442.
2. Murray CJ, Lopez AD. Mortality by cause for eight regions of the world: Global Burden of Disease Study. *Lancet* 1997; **349:** 1269-1276.
3. Choolani M, Ratnam SS. Maternal mortality: a global overview. *J Indian Med Assoc* 1995; **93:** 36-40.
4. World Health Organisation. *World Health Report, May 1999.* Geneva: WHO, 1999.
5. Boerma JT, Nunn AJ, Whitworth JAG. Mortality impact of the AIDS epidemic: evidence from community studies in less developed countries. *AIDS* 1998; **12:** S3-S14.
6. Taha TE, Miotti P, Liomba G, Dallabetta G, Chiphangwi J. HIV, maternal death and child survival in Africa. *AIDS* 1996; **10:** 111-112.
7. Juneja Y, Goel U, Sood M. Changing trends in maternal mortality over a decade. *Int J Gynaecol Obstet* 1994; **46:** 265-269.
8. Ray A. Maternal mortality in a subdivisional hospital of eastern Himalayan region. *J Indian Med Assoc* 1992; **90:** 124-125.
9. Ahmed Y, Mwaba P, Chintu C, *et al.* A study of maternal mortality at the University Teaching Hospital, Lusaka, Zambia: the emergence of tuberculosis as a major non-obstetric cause of maternal death. *Int J Tuberc Lung Dis* 1999; **3:** 675-680.
10. Coovadia HM, Jeena PM, Wilkinson D. Childhood HIV-1 and TB co-infection. Reconciling conflicting data. *Int J Tuberc Lung Dis* 1998; **2:** 844-851.
11. Adhikari M, Pillay T, Pillay DG. Tuberculosis in the newborn: an emerging disease. *Pediatr Infect Dis J* 1997; **16:** 1108-1112.
12. Matambo JA, Moodley D, Moodley J. HIV seroprevalence and rapid testing in unbooked pregnant African women. *Int J Gynaecol Obstet* 1999; **66:** 289-290.
13. Khan M, Pillay T, Moodley J, Connolly C. Maternal mortality associated with tuberculosis-HIV type 1 co-infection in Durban, South Africa. *AIDS* (in press).
14. Dye C, Scheele S, Dolin P, Pathania V, Raviglione M. Global burden of tuberculosis. Estimated incidence, prevalence and mortality by country. *JAMA* 1999; **282:** 677-686.
15. Wilkinson D, Davies GR. The increasing burden of tuberculosis in rural South Africa — the impact of the HIV-1 epidemic. *S Afr Med J* 1997; **87:** 447-450.
16. Connolly C, Davies GR, Wilkinson D. Impact of the human immunodeficiency virus epidemic on mortality among adults with tuberculosis in rural South Africa. *Int J Tuberc Lung Dis* 1998; **11:** 919-925.
17. Kleinschmidt I. South African TB mortality data —showing the first signs of the HIV-1 epidemic? *S Afr Med J* 1999; **89:** 269-272.
18. Harrison A, Connolly C, Wilkinson D. Increasing prevalence of HIV infection among pregnant women attending public sector antenatal clinics in rural KwaZulu Natal. *S Afr J. Epidemiol Infect* 1999; **14:** 22-23.
19. Mofenson LM, Rodriguez EM, Hershow R, *et al. Mycobacterium tuberculosis* infection in pregnant and non pregnant women infected with HIV in the Women and Infants Transmission Study. *Arch Intern Med* 1995; **155:** 1066-1072.
20. Margono F, Mroueh J, Garely A, White D, Duerr A, Minkoff HL. Resurgence of active tuberculosis among pregnant women. *Obstet Gynecol* 1994; **83:** 911-914.
21. Gilks CF, Brindle RJ, Otieno LS, *et al.* Extrapulmonary and disseminated tuberculosis in HIV-1 seropositive patients presenting to the acute medical service in Nairobi. *AIDS* 1990; **4:** 981-985.
22. Leroy V, Msellati P, Lepage P, *et al.* Four years of natural history of HIV-1 infection in African women: a prospective cohort study in Kigali (Rwanda), 1988-1993. *J Acquir Immune Defic Syndr Hum Retrovirol* 1995; **9:** 415-421.
23. Anderson G. Tuberculosis in pregnancy. *Semin Perinatol* 1997; **21:** 328-335.
24. Jana N, Vasishta K, Jindal SK, Khurnu B, Ghosh K. Perinatal outcomes in pregnancies complicated by pulmonary tb. *Int J Gynaecol Obstet* 1994; **44:** 119-124.
25. Figueroa Damian R, Arrendondo Garcia JL. Tuberculosis in pregnant women. *Ginecol Obstet Mex* 1992; **60:** 209-216.
26. Figueroa Damian R, Arrendondo Garcia JL. Pregnancy and tuberculosis: Influence of treatment on perinatal outcome. *Am J Perinatol* 1998; **15:** 303-306.
27. Good J, Iseman M, Davidson P, *et al.* Tuberculosis in association with pregnancy. *Am J Obstet Gynecol* 1981; **140:** 492-498.
28. Hedvall E. Pregnancy and tuberculosis. *Acta Med Scand* 1953; **147:** 1-101.

Accepted 27 December 2000.

MICROVASCULAR COMPLICATIONS IN SOUTH AFRICAN PATIENTS WITH LONG-DURATION DIABETES MELLITUS

Ayesha A Motala, Fraser J Pirie, Eleanor Gouws, Aslam Amod, Mahomed A K Omar

Objective. To determine the prevalence of microvascular complications in South African black and Indian patients with long-duration diabetes mellitus (DM).

Design. A retrospective analysis was undertaken of clinical records of 219 DM patients (132 black, 87 Indian) with long-duration DM (over 10 years) attending a diabetes clinic in Durban. Data recorded on each subject included demographic details (age, gender, ethnic group, type of diabetes, age of onset and duration of diabetes), presence of retinopathy, markers of nephropathy and biochemical variables. The prevalence of complications and the clinical and biochemical parameters were evaluated for type 1 and type 2 diabetes and for each ethnic group.

Results. Of the 219 patients, 47 had type 1 DM (36 blacks, 11 Indians) and 172 were classified as type 2 DM (96 blacks, 76 Indians). The mean age of onset of DM was later in blacks than Indians, both for type 1 ($P < 0.05$) and type 2 DM ($P < 0.01$). In patients with type 1 DM, the prevalence of retinopathy was 53.2% (blacks 55.6%, Indians 45.5%), persistent proteinuria was found in 23.4% (blacks 25%, Indians 18.2%) and hypertension in 34%. No ethnic difference was found except for the prevalence of hypertension which was higher in blacks than Indians (41.7% v. 9.1%, $P < 0.5$). Onset of retinopathy from time of diabetes diagnosis occurred earlier in blacks than Indians (13.0 ± 4.6 yrs v. 18.0 ± 4.6 yrs, $P < 0.05$). For the type 2 DM group, retinopathy was found in 64.5% (black v. Indian 68.8 v. 59.2%) and persistent proteinuria in 25% (black v. Indian 30.2 v. 18.4%). Hypertension was observed in 68% and was more prevalent in blacks (84.4 v. 47.4%, $P < 0.01$) There was an earlier onset of retinopathy ($P < 0.05$) and hypertension ($P < 0.01$) from time of diabetes diagnosis in blacks than Indians. In the type 1 DM group retinopathy was associated with a significantly

Diabetes Unit, Department of Medicine, University of Natal
Ayesha A Motala, MD, FRCP
Fraser J Pirie, MB ChB, FCP
Aslam Amod, MB ChB, FCP
Mahomed A K Omar, MD, FCP, FRCP

Institute of Biostatistics, Medical Research Council, Durban
Eleanor Gouws, MSc

longer duration of diabetes ($P < 0.05$) and higher glycated haemoglobin (HbA_1) ($P < 0.05$). For type 2 DM subjects there was a significant association between retinopathy and longer duration of diabetes ($P < 0.05$) and higher systolic blood pressure ($P < 0.05$).

Conclusion. This study has shown that there is a high prevalence of microvascular complications in South African patients with long-duration diabetes mellitus.

S Afr Med J 2001; 91: 987-992.

Traditionally diabetes mellitus (DM) has been regarded as a disease of urbanisation and industrialisation, and one that is still rare or unknown in rural Africa.[1,2] But, based on World Health Organisation (WHO) criteria and age-standardised estimates, King and Rewers[2] have shown that diabetes in adults is a global problem and that populations in developing countries, minority groups and disadvantaged communities in industrialised countries face the greatest risk. Data on diabetes epidemiology in Africa have shown that although the prevalence is low in some rural communities, in other countries there is a moderate prevalence comparable with that found in developed countries.[3]

It is well established that there is increased morbidity and mortality associated with diabetes complications, both microvascular and macrovascular. The natural history and clinical course of diabetes in Africa is poorly understood, in many instances because of poor follow-up.[1] Earlier reports have indicated that unlike in Western populations where the major causes of mortality were cardiovascular and renal disease, in Africa the major aetiological factors were acute metabolic and infective.[1,4-6] More recently, reports from Ethiopia[7] and South Africa[8] have indicated a changing pattern, with diabetic nephropathy playing a more important role in mortality.

In Western countries the prevalence of retinopathy ranges from 2% to 90% and that of nephropathy from 2% to 20% depending on type and duration of diabetes.[9] Reports on prevalence of macrovascular complications in Africa are limited. Previous impressions that microvascular complications are rare in Africa were probably related to shorter survival rates and inadequate screening.[1] Earlier studies (before 1990) from Africa have reported that the prevalence of retinopathy was 2.9 - 57.1% and that of nephropathy 1.0 - 30.5%;[1] however, such studies involved mixed cohorts of type 1 and type 2 diabetes patients with varying duration of disease.

Although several studies have examined subjects with diabetes duration of 10 years or more, only three reports on well-defined groups have examined diabetes outcome.[7,8,10] Such studies showed that in type 1 diabetes[7,8] retinopathy was found in 40 - 50% and nephropathy in 20 - 28%. In Ethiopian type 2 patients with DM duration over 20 years, retinopathy was found in 45.5% and nephropathy in 29.8% of cases.[10]

In a recent report[11] of an audit of primary health care services in Cape Town, a high prevalence of complications was found; however, again this report included both type 1 and type 2 diabetes patients and with varying duration of disease.

This retrospective analysis was undertaken to determine the prevalence of retinopathy and nephropathy in South African blacks and Indian patients with diabetes duration of over 10 years.

PATIENTS AND METHODS

Patients and background

The study population comprised South African black and Indian patients with diabetes duration over 10 years registered at the diabetes clinic at King Edward VIII Hospital (KEH), Durban. KEH is the major referral hospital not only for the city but also for the province of KwaZulu-Natal which represents a quarter of the total population of the country.

Patients are reviewed at the diabetes clinic at monthly, 3-monthly or 4-monthly intervals. At each visit symptomatic details are recorded. Clinical examination includes measurement of weight, blood pressure (BP) and pulse rate; dipstix urine is examined for glucose, protein and ketones; capillary blood glucose is measured with reflectance meter and venous blood samples are taken for estimation of glycated haemoglobin, plasma glucose, serum fructosamine, serum lipids, urea and electrolytes. Annually, fundal examination, visual acuity and glomerular filtration rate (GFR) are assessed. Records of such information are available from 1983.

Methods

Using November 1995 as the reference end-point of the study, information was obtained on demographic data, presence or absence of retinopathy and markers of nephropathy and biochemical variables. Demographic details included the following: age, gender, ethnic group, type of diabetes, age of onset and duration of diabetes and the date of the last clinic visit if the subject was lost to follow-up.

The information recorded regarding retinopathy included presence or absence of retinopathy; if present, whether it was background or proliferative; use of laser treatment and estimation of duration from time of diabetes diagnosis to the onset of retinopathy.

The markers of diabetic nephropathy evaluated included persistent dipstix proteinuria, hypertension, serum creatinine and GFR. The presence or absence of proteinuria over the previous six visits (18 months) was recorded. The means of three values per year for systolic and diastolic BP were calculated. A record was kept of hypertension requiring treatment from the time of diabetes diagnosis. Hypertension

w s defined using WHO criteria,[12] i.e. systolic BP ≥ 160 mmHg or diastolic BP ≥ mmHg or if the patient was receiving ar ihypertensive treatment; borderline hypertension was de ined as BP > 140/90 mmHg but < 160/95 mmHg. The most re ent serum creatinine and GFR measurements and their du ation from time of diabetes onset were noted.

The means of three values per year were calculated for finger pr ck capillary glucose, venous plasma glucose, HbA_1 (before 19 3) and HbA_{1c} (after 1993). Retinopathy was evaluated by the re ord of findings at annual fundal examination done through di ated pupils using hand-held monocular direct op thalmoscopy. Retinopathy was classified as background, pr -proliferative or proliferative.[13]

ersistent proteinuria was defined as dipstix proteinuria on th e or more consecutive occasions over 18 months in the ab ence of infection or cardiac failure, serum creatinine was de ined as abnormal if the value was > 115 µmol/l at the last vi t, and abnormal GFR was defined if GFR < 95 ml/min in w men and GFR < 98 ml/min in men.

B chemical tests

Th methods used for the biochemical variables were as fo ows: venous plasma glucose was measured using a glucose ox lase method; cation exchange microcolumn ch matography for HbA_1 (normal range 5 - 8%); enzyme-lin ed immunosorbent assay (ELISA) technique for HbA_{1c} (n mal range 3.5 - 5.6%); serum creatinine was measured by re tion rate (normal range 53 - 115 µmol/l). GFR was assessed us g ^{99m}Tc- DTPA (normal range 98 - 150 ml/min for men, 95 - 12 ml/min for women).

Statistical analysis

Statistical analysis was performed using the SAS computer programme.[14] The prevalence of complications and the clinical and biochemical parameters were evaluated for type 1 and type 2 diabetes and for each ethnic group. Data are expressed as means ± standard deviations (SD) or prevalence (%). Ethnic differences were assessed using the unpaired Student's *t*-test for numerical data and the chi-square test for categorial data. A *P*-value < 0.05 was regarded as significant.

RESULTS

The study group comprised 219 patients (159 women, 60 men; 132 blacks, 87 Indians) identified as having had diabetes for over 10 years and who constituted 25.2% of the 870 patients registered at the diabetes clinic. Of these, 172 subjects (78.5%) had type 2 diabetes and 47 (21.5%) had type 1 diabetes.

Clinical characteristics

Table I shows the clinical and biochemical characteristics. In both the type 1 and type 2 diabetes groups the mean age of onset was later in black than Indian patients ($P = 0.03$, type 1; $P = 0.004$, type 2), and BP was higher ($P < 0.05$). While BP control appeared reasonable, overall glycaemic control, as judged by glycated haemoglobin level, was suboptimal in both ethnic groups.

Prevalence of complications (Table II)

In the type 1 diabetes group retinopathy was found in over half of the patients; the mean onset of retinopathy from time of

Table I. Clinical and biochemical characteristics of 219 subjects with long-duration diabetes mellitus (DM) (> 10 yrs)*

	Type 1 diabetes				Type 2 diabetes			
Variable	Total	Black	Indian	*P*-value	Total	Black	Indian	*P*-value
Number	47	36	11	-	172	96	76	-
Gender (F/M) (*N*)	28:19	22:14	6:5	-	131:41	72:24	59:7	-
Age (yrs)	39.5 ± 10.9	39.9 ± 11.2	38.0 ± 10.5	0.6	58.4 ± 9.4	59.1 ± 7.8	57.4 ± 11.0	0.3
Age of onset (yrs)	22.5 ± 11.0	24.3 ± 11.4	16.6 ± 7.4	0.03	39.9 ± 9.4	41.8 ± 8.0	37.5 ± 10.5	0.004
Duration (yrs)	16.10 ± 4.9	15.6 ± 3.6	21.3 ± 6.8	0.005	18.6 ± 5.7	17.4 ± 4.9	20.2 ± 6.2	0.002
Lost to follow-up (*N* (%))	16 (34)	9 (25)	7 (63.6)	0.07	57 (33.1)	32 (33.3)	25 (32.9)	0.1
Blood pressure (mmHg)								
Systolic	133 ± 13.6	135.3 ± 12.5	125.9 ± 15.4	0.04	144.7 ± 13.3	147.2 ± 12.8	141.4 ± 13.5	0.0052
Diastolic	82.1 ± 8.1	83.5 ± 7.8	77.7 ± 2.8	0.03	84.4 ± 8.4	86.7 ± 8.2	81.4 ± 7.9	0.0001
Plasma glucose (mmol/l)	10.8 ± 4.2	11.5 ± 4.1	8.2 ± 3.5	0.02	12.1 ± 3.9	12.1 ± 4.7	12.1 ± 3.4	0.0195
HbA_1 (%)‡	9.5 ± 1.5	9.9 ± 1.5	9.5 ± 1.4	0.5	9.7 ± 1.8	10.0 ± 2.1	9.3 ± 1.4	0.02
HbA_{1c} (%)§	9.8 ± 2.2	10.2 ± 2.3	8.6 ± 1.6	0.08	9.8 ± 2.1	9.8 ± 2.2	9.8 ± 2.1	0.1
Serum creatinine (µmol/l)	99.2 ± 77.1	95.7 ± 68.5	111.5 ± 105.2	0.6	132.4 ± 174.5	147.2 ± 212.7	113.6 ± 106.2	0.2
Glomerular filtration rate (ml/min)	98.6 ± 33.9	98.5 ± 31.5	99.1 ± 42.9	0.9	77.0 ± 32.9	74.6 ± 32.1	80.1 ± 34.1	0.2

* Data are means ± SD, except as noted.
† P-value: Black v. Indian.
‡ *N* = 43, type 1; *N* = 149, type 2.
§ *N* = 30, type 1; *N* = 123, type 2.

Table II. Prevalence of microvascular complications in 219 patients with diabetes mellitus (DM), duration > 10 yrs (% (N))

	Type 1 DM (% (N))				Type 2 DM (% (N))			
	Total (N = 47)	Black (N = 36)	Indian (N = 11)	P-value†	Total (N = 172)	Black (N = 96)	Indian (N = 76)	P-value†
Retinopathy								
Any	53.2 (25)	55.6 (20)	45.5 (5)	0.6	64.5 (111)	68.8 (66)	59.2 (45)	0.2
Background only	38.3 (18)	38.9 (14)	36.4 (4)	0.6	44.2 (76)	47.9 (46)	39.5 (30)	0.2
Proliferative	14.9 (7)	16.7 (6)	9.1 (1)	0.5	20.4 (35)	20.8 (20)	19.7 (15)	0.9
Laser treatment	17.0 (8)	16.7 (6)	18.2 (2)	0.9	29.7 (51)	26.0 (25)	34.2 (26)	0.2
Onset from DM diagnosis (yrs)*	14 ± 4.9	13.0 ± 4.6	18.0 ± 4.6	0.039	14.4 ± 6.0	13.1 ± 4.9	16.2 ± 7.0	0.01
Proteinuria								
Persistent (p.p)	23.4 (11)	25.0 (9)	18.2 (2)	0.6	25.0 (43)	30.2 (29)	18.4 (14)	0.08
Without retinopathy	0 (0)	0 (0)	0 (0)	-	13.9 (6)	10.4 (3)	21.4 (3)	0.06
Onset from DM diagnosis (yrs)*	11.4 ± 3.2	10.3 ± 2.1	16.5 ± 2.1	0.0045	13.4 ± 4.8	13.5 ± 4.7	13.4 ± 4.9	0.9
Hypertension								
Requiring treatment	34.0 (16)	41.7 (15)	9.1 (1)†	0.046	68.0 (117)	84.4 (81)	47.4 (36)	0.001
BP ≥ 160/95 mmHg	10.6 (5)	11.1 (4)	9.1 (1)	0.9	19.1 (31)	23.9 (22)	12.7 (9)	0.07
BP ≥ 130/85 mmHg	57.5 (27)	66.7 (24)	27.3 (3)	0.021	90.2 (148)	92.5 (86)	87.3 (62)	0.001
Onset from DM diagnosis (yrs)*	9.6 ± 3.5	9.3 ± 0.3	14.0 ± 0	0.2	13.1 ± 7.1	11.7 ± 6.0	16.3 ± 8.2	0.001
Abnormal serum creatinine‡	17.8 (8)	17.1 (6)	20.0 (2)	0.8	25.2 (43)	29.2 (28)	20.0 (15)	0.2
Glomerular filtration rate (ml/min)								
< 95 (women), < 98 (men)	39.5 (17)	39.4 (13)	40.0 (4)	0.9	73.4 (124)	73.4 (69)	73.3 (55)	0.9
< 70	20.9 (9)	18.0 (6)	30.0 (3)	0.4	42.0 (71)	46.8 (44)	36.0 (27)	0.2

* Mean ± SD.
† P-value: black v. Indian.
‡ > 115 µmol/l.

diagnosis was significantly earlier in blacks than Indians (13.0 ± 4.6 yrs v. 18.0 ± 4.6 yrs, $P = 0.039$). Nephropathy on the basis of persistent proteinuria was found in approximately one-quarter of the subjects, all of whom had retinopathy. One-third of the patients had hypertension requiring treatment and the prevalence was higher in blacks than Indians (41.7% v. 9.1%, $P = 0.046$); 17.8% of patients had elevated serum creatinine and 20.9% had abnormal GFR.

In the type 2 diabetes group the prevalence of retinopathy was 64.5%; the mean onset of retinopathy from time of diagnosis was significantly earlier in blacks than Indians (13.1 ± 4.9 yrs v. 16.2 ± 7.0 yrs, $P = 0.01$. Nephropathy based on persistent proteinuria was found in 25% of these patients; 6 (3.5%) had no retinopathy. Hypertension was observed in 68%, with a higher prevalence in blacks than Indians (84.4% v. 47.4%, $P = 0.001$); the mean onset from time of diagnosis was earlier in blacks than Indians (11.7 ± 6.0 yrs v. 16.3 ± 8.2 yrs, $P = 0.001$) An elevated serum creatinine was found in 25.2% and abnormal GFR in 42% of type 2 diabetes patients.

Risk factors and complications (Table III)

Analysis of known risk factors for microvascular complications showed that in type 1 DM subjects retinopathy was associated with significantly longer duration of diabetes ($P = 0.046$) and higher HbA_1 ($P = 0.028$). For the type 2 DM group, when compared with subjects without retinopathy, the duration of diabetes was longer ($P = 0.04$) and systolic BP higher ($P = 0.0125$) in subjects with retinopathy.

Discussion

This study of South African black and Indian patients with long-duration (> 10 years) type 1 and type 2 DM shows an increased prevalence of retinopathy and nephropathy. Ethnic comparisons demonstrate that the prevalence of hypertension was higher and the onset of retinopathy earlier in blacks than Indians.

It is difficult to compare the results of this study with other studies from Africa since most studies have examined mixed cohorts of type 1 and type 2 diabetes patients, with varying duration of disease.[1]

For type 1 diabetes comparison is only possible with two outcome studies from Africa.[7,8] When compared with the recent South African report of 36 patients with mean diabetes duration of 13 years, the prevalence of retinopathy is similar (53.2% v. 52%), persistent proteinuria lower (23.4% v. 28%) and hypertension higher (34% v. 22%).[8] In Ethiopian subjects, Lester[7] reported prevalence rates of 40.7% for retinopathy and 20% for persistent proteinuria and hypertension respectively in a cohort of patients with diabetes duration of over 10 years. The apparent low prevalence rates in other African studies is probably accounted for by the varying diabetes durations of

Table III. Relationship between microvascular complications and known risk factors in 219 patients with long-duration diabetes mellitus (DM)

	Type 1 DM (N = 47)				Type 2 DM (N = 172)			
	Retinopathy		Persistent proteinuria		Retinopathy		Persistent proteinuria	
Risk factor	Yes	No	Yes	No	Yes	No	Yes	No
Blood pressure (mmHg)								
Systolic	133.6 ± 13.9	132.5 ± 13.7	140.8 ± 17.1	130.1 ± 0.9	146.6 ± 12.9	141.3 ± 13.6†	146.9 ± 11.8	143.3 ± 14.0
Diastolic	80.7 ± 7.5	83.7 ± 8.7	85.6 ± 8.2	80.8 ± 7.8	84.8 ± 7.8	83.7 ± 9.4	85.6 ± 7.8	83.7 ± 8.8
Capillary glucose (mM)	10.8 ± 2.4	10.2 ± 3.2	10.5 ± 2.3	10.5 ± 2.9	11.7 ± 2.6	11.7 ± 2.5	11.7 ± 2.9	11.7 ± 2.4
Plasma glucose (mM)	11.7 ± 4.3	9.8 ±3.9	10.7 ± 3.3	10.8 ± 4.5	12.4 ± 3.5	11.5 ± 4.5	11.9 ± 4.0	12.2 ± 3.8
HbA_1 (%)	10.2 ± 1.7	9.2 ± 0.8†	10.3 ± 2.3	9.6 ± 0.9	9.9 ± 2.0	9.4 ± 1.5‡	9.6 ± 1.7	9.8 ± 1.9
HbA_{1c} (%)	10.1 ± 2.3	9.5 ± 2.2	10.2 ± 2.8	9.7 ± 2.0	9.9 ± 1.9	9.6 ± 2.4	10.2 ± 2.3	9.6 ± 1.9
Age of onset (yrs)	23.6 ± 10.8	21.2 ± 11.5	23.2 ± 10.4	22.2 ± 11.4	40.2 ± 9.8	39.5 ± 8.8	39.2 ± 9.0	40.3 ± 9.7
Diabetes duration (yrs)	18.3 ± 5.2	15.4 ± 4.3†	17.4 ± 6.3	16.8 ± 4.4	19.2 ± 6.2	17.6 ± 4.3†	19.2 ± 5.1	18.3 ± 5.9

Data are means ± SD.
† $P < 0.05$: yes v. no.
‡ $P = 0.052$: yes v. no.

the subjects studied and possibly shortened survival rates.[1] The observed prevalence of retinopathy and persistent proteinuria in this study is compatible with reports from the Western literature.[9,13,15]

It is interesting that although no ethnic difference was observed for the prevalence of retinopathy and persistent proteinuria, hypertension was more prevalent in blacks than Indians. This finding is in contrast with earlier reports on patients with shorter duration type 1 diabetes which showed no ethnic difference,[16] but it is compatible with other reports which highlight the high prevalence of hypertension in black Africans[1] and with epidemiological evidence indicating that in South Africa hypertension is more prevalent in blacks (25%) than Indians (14%).[17]

The finding of later onset of diabetes in blacks than Indians is compatible with previous South African studies in which onset was found to be later in blacks than Indians[16,18] and Europids.[19] However, studies from North America found similar age of onset in black and white children.[20]

Despite the later onset of diabetes, the onset of retinopathy from time of diagnosis was earlier in blacks than Indians. The reasons for this are unclear since there was no difference in glycaemic control between the two groups. It is possible, however, that the higher prevalence of hypertension in blacks is responsible for the acceleration of retinopathy.

In type 2 diabetic subjects the prevalence of retinopathy and persistent proteinuria is similar to rates reported in the Western literature.[9,13,15] As for type 1 diabetes, comparison with other African studies is difficult because of mixed cohorts of type 1 and type 2 diabetes and varying diabetes duration in such studies.[1] Notwithstanding, in the Ethiopian report[10] on 121 predominantly type 2 patients with diabetes of over 20 years' duration the prevalence of retinopathy (45.5%) was lower than in this study, while that of persistent proteinuria (29.8%) was similar. The lower prevalence of retinopathy (55.4%) and persistent proteinuria (5.3%) found in a recent audit of South African blacks can be explained by the varying duration of diabetes in the patients studied (mean duration 8 years, range 0 - 28 years); moreover, that report included type 1 subjects.[11] Ethnic difference was only observed for prevalence of hypertension, which was higher in blacks than in Indians and might reflect the high background prevalence in blacks.[17]

As for type 1 diabetes, the onset of retinopathy from time of diagnosis was earlier in blacks than Indians; the difference may be accounted for by the higher BP levels in black patients. What is also apparent from this analysis is that in the group as a whole glycaemic control is poor; this clearly highlights the need for more aggressive intensive management to optimise control.

In conclusion, this study has shown that in South African black and Indian patients with long-duration diabetes there is a high prevalence of microvascular complications. Previous impressions that microvascular complications are rare in Africa were probably related to shorter survival rates and inadequate screening; as survival rates improve and there are greater numbers of African patients with long-duration diabetes, the pattern will probably be similar to that observed in the Western world.

References

1. McLarty DG, Pollitt C, Swai ABM. Diabetes in Africa. *Diabet Med* 1990; 7: 670-684.
2. King M, Rewers M. Global estimates for prevalence of diabetes mellitus and impaired glucose tolerance in adults. *Diabetes Care* 1993; 16: 157-177.
3. Motala AA, Omar MAK. NIDDM in Africans: it is increasing? *IDF Bulletin* 1995; 40: 23-26.
4. Castle WM, Wicks ACB. A follow-up of 93 newly diagnosed African diabetics for 6 years. *Diabetologia* 1980; 18: 121-123.
5. Lester FT. Diabetes mellitus in Ethiopians: mortality. *Ethiop Med J* 1984; 22: 61-66.
6. McLarty DG, Kinabo L, Swai ABM. Diabetes in tropical Africa: a prospective study, 1981-7. II Course and prognosis. *BMJ* 1990; 300: 1107-1110.
7. Lester FT. Clinical features, complications and mortality in type 1 (insulin-dependent)

diabetic patients in Addis Ababa, Ethiopia, 1976 - 1990. *QJM* 1992; **301:** 389-399.
8. Gill GV, Huddle KR, Rolfe M. Mortality and outcome of insulin-dependent diabetes in Soweto, South Africa. *Diabet Med* 1995; **12:** 546-550.
9. Hanssen KF. Determinants of microvascular complications in diabetes: an overview. In: Pickup J, Williams G, eds. *Textbook of Diabetes*. Oxford: Blackwell Scientific Publications, 1991: 519-525.
10. Lester FT. Clinical status of Ethiopian diabetic patients after 20 years of diabetes. *Diabet Med* 1991; **8:** 272-276.
11. Levitt NS, Bradshaw, D Zwarenstein MF, Bawa AA, Maphumolo S. Audit of public sector primary diabetes care in Cape Town, South Africa: high prevalence of complications, uncontrolled hyperglycaemia, and hypertension. *Diabet Med* 1997; **14:** 1073-1077.
12. World Health Organisation Expert Committee. Arterial Hypertension. Report of a WHO Expert Committee. *World Health Organ Tech Rep Ser* 1978; No. 628.
13. Kohner EM. The lesions and natural history of diabetic retinopathy. In: Pickup J, Williams G, eds. *Textbook of Diabetes*. Oxford: Blackwell Scientific Publications, 1991: 575-588.
14. SAS Institute. *SAS/STAT User's Guide*. Release 6.03 ed. Cary, NC: SAS Institute, 1988.
15. Viberti GC, Marshall S, Beech R, *et al*. Report on renal disease in diabetes. *Diabet Med* 1996; **13:** suppl 4, 6-12.
16. Omar MAK, Asmal AC. Complications of early-onset insulin-dependent diabetes mellitus in blacks and Indians. *S Afr Med J* 1984; **65:** 75-78.
17. Seedat YK. Race, environment and blood pressure: the South African experience. *J Hypertens* 1983; **1:** 7-12.
18. Omar MAK, Asmal AC. Patterns of diabetes mellitus in young Africans and Indians in Natal. *Trop Geogr Med* 1984; **36:** 113-138.
19. Kalk WJ, Huddle KRL, Raal FJ. The age of onset and sex distribution of insulin-dependent diabetes mellitus in Africans in South Africa. *Postgrad Med J* 1993; **69:** 552-556.
20. LaPorte RE, Tajina N, Dorman JS, *et al*. Differences between Blacks and Whites in the epidemiology of insulin dependent diabetes mellitus in Allegheny county, Pennsylvania. *Am J Epidemiol* 1986; **123:** 592-603.

Accepted 9 September 2001.

SPECIAL ARTICLE

AN ASSESSMENT OF GROWTH IN HIGH AND LOW SOCIO-ECONOMIC STATUS SCHOOLCHILDREN IN SOUTH AFRICA

G J Louw, S M Naidoo

'We are guilty of many errors and many faults, but our worst crime is abandoning the children, neglecting the foundation of life. Many of the things we need can wait. The child cannot. Right now is the time his bones are being formed, his blood is being made and his senses are being developed. To him we cannot answer "Tomorrow". His name is "Today".'

(Gabriela Mistral, 1948, World Health Organisation, 1997

The patterns of physical growth (height, weight, length of trunk and limbs, circumference of trunk and limbs, and limb breadths) and function (grip strength of both hands and neuromuscular reaction time) of Cape Coloured (specifically mixed origin) schoolchildren from urban and rural areas and contrasting socio-economic status (SES) levels, were measured. The mixed longitudinal study accumulated data over more than a decade, and included 929 male and 1 160 female pupils of high SES (HSES) in the Cape Town urban area, aged 5 - 20 years, and 954 male and 1 030 female pupils of low SES (LSES) in rural areas of the Little Karoo, aged 5 - 19 years. Means for every anthropometric character were calculated and matched against age for each of the four groups, for comparative purposes. Standard deviations were recorded for each character. Figs 1 - 16 show the means for each of the anthropometric characters, matched with age groups, for HSES and LSES boys and girls.

The results reflect the importance of positive intervention in the growth and development of the LSES children at different periods for the girls and boys, namely pre-pubertally in girls, particularly during the period of possible pre-pubertal growth spurt (8 - 10 years of age), and continuously and consistently for the boys, from 5 to 19 years and onwards. Intervention should take the form of improved diet, increased exercise, and plenty of time spent outdoors in the sun.

Department of Human Biology, University of Cape Town
G J Louw, DVSc
S M Naidoo, BSc