

## 6. DIABETES MELLITUS

The prevalence of diabetes in the tropics is the subject of controversy based all too often on hasty generalizations. When the health services were set up in the Congo (Zaire), the analytical and biochemical detection methods reflected those in use at the time. The simple procedure of screening for protein and sugar in urine was performed in various circumstances (hospital confinement, maternity check-ups, labour recruitment). As massive glycosuria was not observed in the groups tested it would have been illogical to consider diabetes mellitus a priority when so many other endemic and epidemic diseases gave greater concern.

The traditional healers were familiar with glycosuria. If a patient urinated near an anthill (an ingenious test used among other countries in Madagascar), they could distinguish between urine that repelled or attracted the ants. They also knew of plants able to make the sugar disappear.

As time went by, diabetes mellitus received more attention. From the thirties onwards, hospitals had access to in-house and external laboratories able to measure blood glucose and urinary acetone levels. In fact each medical service did from time to time diagnose a case of diabetes mellitus, and rarely a fatal case. Knowing the propensity of specialists in tropical medicine to study and describe any undocumented disease, it seems clear that diabetes mellitus, while not unrecognized, was seen merely as a curiosity to be treated with particular solicitude. Moreover, WHO surveys of rural populations in Togo and Papua-New Guinea (1985) have confirmed that diabetes mellitus is rare in the tropics.

Since the late forties however, in various African countries including Zaire, the prevalence of diabetes mellitus has been rising steadily in the towns, especially in the part of the population with high living standards. Better detection may be one of the causes of this increase.

Diabetes mellitus is a metabolic disorder which is not only due to an insulin deficiency, as was thought when the hormone secreted by the pancreas's islets of Langerhans was discovered. It is a complex syndrome, especially noticeable in the tropics, where it exhibits unique features.

The metabolism of glucose – the cell's energy source – involves the insulin-mediated transfer of sugar into the cells. In the absence of insulin the blood plasma glucose concentration rises, leading to a series of problems. In fact, two substances are involved, insulin and glucose. Insulin production is regulated by sugar intake. Consequently, insulin is delivered in the blood by pulses, as required by the

sugar level increase, and is not present continuously. Glucose is the metabolic product of various sugars and starches in the diet. Mono- and oligosaccharides are broken down and used up quickly, polysaccharides are used more slowly and for a wider range of purposes, depending on how the food is cooked, which leads to marked differences in blood glucose levels.

Type I diabetes mellitus occurs in Central Africa, but its prevalence is low. The paediatric wards reported only a few cases per several thousand admissions, with some in diabetic coma. The mechanism or mechanisms triggering the destruction of the beta cells in the islet of Langerhans are not known. An autoimmune disorder does not seem to be involved in Central Africa, since antibody against islet cells has not been detected. The role of an hereditary predisposition is difficult to establish.

Tropical or protein-deficient diabetes – an unexpected variety apparently specific to regions with hot climates – is linked to malnutrition. Far from offering protection against diabetes, malnutrition becomes a determining cause. This form of diabetes has created a certain degree of confusion with Type I diabetes mellitus, because it is resistant to exogenous insulin and exhibits a certain opposition against keto-acidosis.

Type II – non-insulin-dependent diabetes mellitus – is the most frequent form of the disease. It is not essentially due to high lipid intake, and is sensitive to insulin. However, hypoglycaemia is a real risk and may be accompanied by oedema, which is even more serious. Vascular and cerebral complications – hypertension, cerebrovascular accidents – develop early. On the other hand, coronary ischaemia is absent. Neurological involvement is fairly frequent. Retinitis is rare, to the point that some manuals of tropical ophthalmology overlook it completely. Cataracts are seen, but may have other causes. The fasting blood glucose concentration may be remarkably high.

One hypothesis suggests that longlasting consumption of a high-starch diet (beans, peas, and lentils) may have caused the body to lose its ability to react to a diet rich in quickly metabolized sugars. This would lead to hyperglycaemia and a need for more, but less effective, insulin to carry the glucose into the cells.

Besides the classic distinction between Type I insulin-dependent diabetes mellitus of juvenile-onset and Type II non-insulin-dependent diabetes mellitus of maturity-onset, there are cases difficult to fit into either category.

In addition, fibro-calculeous diabetes occurs in a large number of tropical countries, including Zaire.

## DIABETES MELLITUS

<b>1. Different types</b> . . . . .	463
<b>2. Symptoms</b> . . . . .	463
<b>3. Epidemiology</b> . . . . .	464
3.1. Type I diabetes . . . . .	464
3.2. Type II diabetes . . . . .	464
3.3. Diabetes connected with malnutrition. . . . .	464
3.3.1. Protein-deficiency diabetes . . . . .	464
3.3.2. Fibrocalculous pancreas diabetes . . . . .	465
<b>4. Diagnosis</b> . . . . .	465
4.1. Glycosuria . . . . .	465
4.2. Blood sugar . . . . .	465
4.3. Oral glucose tolerance test . . . . .	465
<b>5. Treatment</b> . . . . .	466
5.1. Dietary and general health measures . . . . .	466
5.2. Insulin therapy. . . . .	466
5.3. Oral hypoglycaemic drugs . . . . .	467
<b>6. Prevention</b> . . . . .	467
<b>7. Diabetes and pregnancy.</b> . . . .	467
<b>8. Diabetes and health education</b> . . . . .	467
<b>9. Actual situation and research.</b> . . . .	468

<b>BIBLIOGRAPHY</b> . . . . .	469
-------------------------------	-----

## DIABETES MELLITUS

Diabetes mellitus is a metabolic disease characterized by an absolute or relative insulin deficiency, a protein-based hormone secreted by small units of pancreatic cells known as the islets of Langerhans. Insulin has as function to stimulate storage of nutrients by the body. Thus dietary glucose is stored as glycogen in the liver and fatty acids as lipids in adipose tissue, while amino acids are changed into proteins. Thanks to insuline glucose is providing energy for the body, especially the heart and muscles.

However insulin deficiency reverses these processes: glycogen is broken down into glucose (glycogenolysis), lipids into fatty acids (lipolysis), proteins into amino acids (proteolysis), and amino acids into glucose (gluconeogenesis). Moreover in that case, the body uses fatty acids as an energy source in preference to glucose. This leads to the production of other acids known as ketone bodies, which easily pass into the urine.

### 1. Different types

Diabetes mellitus may be essential or secondary to an organic disease of the pancreas, the adrenals, or the pituitary gland. Essential diabetes mellitus comprises not one but many diseases formerly classified, according to age at the onset, as juvenile diabetes and senile diabetes, but now reclassified on the basis of clinical presentation as type I (insulin dependent or ketosis-prone) diabetes, type II (non-insulin dependent or non-ketotic) diabetes, pregnancy diabetes, and malnutrition diabetes (WHO Expert Committee on Diabetes Mellitus, 1985).

Type I diabetes corresponds to the former juvenile diabetes, and is characterized by total insulin deficiency caused by destruction of the insulin-secreting beta cells. The body's inability to use glucose, constant production of glucose by gluconeogenesis and failure to build up glycogen reserves from dietary glucose, lead to elevated blood sugar levels (hyperglycaemia), while depletion of the stored proteins and fats is accompanied by a build-up of fatty acids, triglycerides, amino acids and ketone bodies. The blood's osmotic pressure increases, the patient urinates frequently (polyuria) and becomes dehydrated. This type of diabetes is immediately life-threatening, and the patient's survival depends on the exogenous administration of insulin.

Type II diabetes corresponds to the old classification of *senile diabetes*. Endogenous insulin secre-

tion may be either normal or slightly depressed. It is high enough to prevent the breakdown of the body's lipid and protein reserves, but often insufficient for all the body's needs since frequently the peripheral tissues are also insulin-resistant. Thus the clinical picture for this type of diabetes is quite fluctuating.

A third type of diabetes mellitus found in the tropics has recently been added to the two classic forms mentioned above. This is *tropical diabetes* or *malnutrition diabetes* (WHO Expert Committee on Diabetes Mellitus, 1985). It is distinguished from type I diabetes by its geographic distribution and aetiology.

### 2. Symptoms

Diabetes mellitus commonly starts with hyperglycaemia and leads to degenerative complications. It may be acute, subacute, or chronic. The symptoms of acute diabetes mellitus include frequent and abundant micturition (polyuria), constant severe thirst (polydipsia), excessive hunger (polyphagia), and unexplained weight loss. If insulin is totally lacking, ketoacidosis may develop with quick progress to coma. The subacute symptoms include infections of the skin and mucous membrane, vulvar pruritus, furunculosis, urinary tract infections, tuberculosis, unusual fatigue, abdominal symptoms, and disorders of vision. Prolonged hyperglycaemia eventually damages nerves and blood vessels, and reduces normal irrigation of the organs. This results in serious chronic lesions, particularly of the eyes, kidneys, heart, brain, and skin. The diabetic's defence mechanisms weaken, leaving the diabetic more vulnerable to infection.

All these well-known symptoms clearly apply to diabetes diagnosed in Central Africa. In a first series of clinical studies conducted in Zaire, *Haine* (1958) described 16 patients showing signs of acute diabetes. Two of these were teenage girls between 13 and 15 years of age, who had keto-acidosis when first seen. In 1962 *Bourgoignie* published a review of 68 cases of diabetes mellitus in which he noted that 70% of patients complained of polyuria, polydipsia, and weight loss, while almost half experienced intense fatigue accompanied by polyphagia. Roughly 10% were in ketotic coma when first brought for treatment. Nearly half of the patients in a more recent study by *Kandjingu* (1985), covering the period from 1960 to 1978, had the classic symptoms of acute diabetes. The other patients were seeking treatment for urinary tract infections (20%), polyneuritis (10%), or other

symptoms. Five percent of the cases were complicated by ketotic coma.

According to the same hospital statistics, the chronic complications of diabetes mellitus occurred with variable frequency and severity in Zairean diabetics. Thus, Bourgoignie asserted that slight retinopathies occurred in 40% of the diabetic population and high blood pressure in 12%, whereas in Kandjingu's series only 8% were hypertensive, 7% had retinopathies, and 4% had gangrenous lower limbs. Fatal complications such as nephropathy, strokes and myocardial infarctions were diagnosed in fewer than 2% of the cases. These percentages make no allowances for the duration of the disease. There are no mortality figures for diabetes mellitus in Central Africa.

### 3. Epidemiology

Diabetes mellitus strikes irrespective of race but with variable frequency. The number of diabetics worldwide is put at 100 million, yet precise figures on the prevalence and incidence of diabetes are hard to obtain, since approaches and diagnostic criteria vary widely and many cases are either overlooked or unreported.

Diabetes mellitus in Central Africa was long thought to be rare, and so received relatively little attention. According to the statistics of the Belgian Congo's health services, only 123 cases were diagnosed between 1922 and 1938. This apparently low figure was attributed to the relaxed lifestyle and low calorie intake. The number of cases began rising in the fifties, so fast that shortly before independence nearly 1,500 new cases were reported annually. Today diabetes is acknowledged as one of the most prevalent diseases in Zaire's departments of internal medicine. Kandjingu (1985) described 1,204 cases of diabetes in Kinshasa's University Clinics and some 5,000 cases are allegedly being treated in Kinshasa's Mama Yemo General Hospital alone. In Brazzaville, across the river, *Mbadinga* (1984) has treated more than 1,000 cases of diabetes.

The epidemiology of diabetes mellitus varies according to the type.

#### 3.1. Type I diabetes

This generally affects young subjects. It reportedly has a predilection for white people. Studies of families, especially of twins, suggest that the risk of developing type I diabetes is associated with genetically determined histocompatibility antigens and

human leucocyte antigens. HLA DR3 and DR4 are thought to help precipitate the destruction (by environmental factors such as viruses and chemicals) of the insulin-secreting beta cells, through auto-immune mechanisms. The serum of such patients contains specific anti-islet antibodies. According to Oli's observations (1981), the anti-islet antibody is almost absent from the bloodstream of diabetics in Nigeria, suggesting that African diabetes is not the typical type I diabetes.

These findings are corroborated by our observations of some 100 insulin-treated Zairean diabetics examined in 1981: none carried the anti-islet antibody, whereas it was detected in 20% of the Belgian diabetics examined simultaneously.

#### 3.2. Type II diabetes

Type II diabetes is also assumed to be hereditary. It generally strikes obese adults, but may also occur at any age, regardless of weight. Its frequency varies greatly according to country, race, and ethnic group. Thus, the prevalence of diabetes mellitus is 25% among the Pima Indians, 10% in Saudi Arabia, 20% in the US and India, and 0.5% in some African villages. In America, however, type II diabetes is more prevalent in blacks than whites, while the prevalence of diabetes in immigrated Indians ranges from 2% in Trinidad to 10% in South Africa's Cape Province. Those affected by the disease are generally (but not exclusively) obese, sedentary and big-eaters. In a sense it is the price paid for material comfort. Studies carried out in South Africa and Zimbabwe show the frequency of type II diabetes in African cities to be increasing.

#### 3.3. Diabetes connected with malnutrition

Malnutrition diabetes was recognized as a particular disease in 1985. It poses a serious health problem, occurring as it does in countries where over 70% of the population has no access to decent health care. It takes at least two forms – protein-deficiency diabetes and fibrocalculus pancreas diabetes.

Their evolution cannot be anticipated: there is a permanent risk of keto-acidosis, but improvement is also observed, with decrease of required insulin or even complete withdrawal.

##### 3.3.1. Protein-deficiency diabetes

This was described for the first time by *Hugh Jones* in Jamaica (Type-J diabetes or malnutrition diabetes). It is associated with past or present protein-energy malnutrition (PEM) and has, since its first observation

been found in other tropical countries of Asia (Bangladesh, India, Indonesia) and Africa (Republic of South Africa, Ghana, Kenya, Malawi, Nigeria, Uganda, Tanzania, and Zaire). Its degree of prevalence in Central Africa is unknown, due to the lack of epidemiological studies in the world. It reaches 7% in Jamaica and 33% in New Delhi. It tends to strike young, poor and usually male, subjects, between 15 and 25 years of age, and is characterized by resistance to keto-acidosis, partial resistance to exogenous insulin, and extreme weight loss. There are no signs of either calcified or chronic fibrotic pancreatitis.

### 3.3.2. *Fibrocalculous pancreas diabetes*

In fibrocalculous diabetes the pancreas becomes fibrotic and atrophic with inflammatory infiltrates and intraductal calcifications. This type has been described in tropical countries worldwide: Brazil, Jamaica, Bangladesh, India, Indonesia, Thailand, Madagascar, Nigeria, Uganda, Zaire, Zambia. In Zaire calcified pancreatitis was reported by *Bourgoignie* (1962) in 3 out of 68 diabetics, and by *Kandjingu* (1985) in 48 (about 4%) out of a series of 1,204 diabetics. *Sonnet* (1966) described the disease in detail for 20 Zairean patients. They were young (average age 12,5 years) and presented with cachexia or wasting, parotiditis, and clinical or biochemically detectable steatorrhea.

Fibrocalculous pancreatitis also exists in developed countries, where it is caused by alcohol abuse. This aetiology is unlikely in the tropics, given the patients' youth, and the disease is more probably caused by the toxicity of the cyanogenic chemicals (linamarine) contained in the leaves and roots of cassava.

Some important epidemiological clues are to be found in India and Nigeria. In India, malnutrition diabetes accounts for only 5% of young diabetics nationwide; but it is endemic in Kerala State where cassava is the staple food. In Nigeria, it is more frequent in the south, where again cassava is widely consumed, than in the north where it is not. The current consensus is that cyanide ions resulting from cassava consumption cause pancreatic lesions only if associated with PEM. Normally the toxic compound is eliminated from the body as thiocyanate, through the action of sulfur-containing amino-acids. Insufficient protein intake might slow down this elimination and increase the compound's toxicity. The thiocyanate itself hampers thyroid hormone synthesis. The association between goitre and malnutrition diabetes deserves further investigation.

## 4. Diagnosis

Diabetes is diagnosed by the clinical signs and by relatively simple urine and blood analyses. Only in doubtful cases may tolerance tests be required. The key diagnostic criterion is the blood sugar level (glycaemia). Hyperglycaemia will be indicated by sugar in the urine (glycosuria) accompanied by polyuria. In the world's leading diabetes centres, procedures to diagnose the lesions are close to those described here.

### 4.1. *Glycosuria*

Measuring urinary glucose levels is the traditional diabetes screening test. It is simple and inexpensive and will confirm a diagnosis when clinical symptoms are observed. WHO does not, however, recommend it for diagnosis, as glycosuria is not a faithful reflection of glycaemia. In fact it can lead to erroneous diagnoses, both positive and negative, for hyperglycaemia can exist without glycosuria just as glycosuria can exist without hyperglycaemia. Sugar detected in the urine, even in the absence of other signs, requires in every case to check blood sugar levels.

### 4.2. *Blood sugar*

Blood sugar can be measured in fasting or postprandial samples of plasma, venous blood, or capillary blood. However using venous blood is simplest. If accompanied by symptoms, fasting blood sugar (FBS) greater than 120 mg/dl, or postprandial blood sugar above 180 mg/dl, if accompanied by symptoms, indicate diabetes, whereas levels below 100 mg/dl are normal. Fasting blood sugar is always elevated and symptomatic in insulin-dependent diabetes. If diabetes mellitus is suspected but the fasting or postprandial blood sugar levels are not overly high, the oral glucose tolerance test should be considered.

### 4.3. *Oral glucose tolerance test (OGTT)*

The OGTT assays the body's ability to handle an overload of glucose. Measuring serum insulin during this test is seldom necessary, since the blood sugar level reflects adequately the insulin response. The test should not be used if fasting hyperglycaemia has been observed.

In its 1980 report, the WHO Expert Committee on Diabetes Mellitus recommended the following procedure:

- after a 10 to 14-hour fast the subject at rest is given a solution of 75 g of sugar in 300 ml water to drink in 5 minutes;

- blood specimens are taken before and at least 1 and 2 hours after the glucose is ingested and blood sugar levels are determined.

The 2-hour concentration is usually the only value taken into consideration when establishing a diagnosis. If it is above 180 mg/dl, the diagnosis of diabetes is certain. If it is between 120 and 180 mg/dl, the subject is said to be glucose-intolerant and must be retested for hyperglycaemia occasionally during the next 12 months.

These diagnostic criteria have been formulated on the basis of European and North American norms. However the fasting blood sugar and the OGTT give the same results in Africans as in Europeans, provided that the subjects have been adequately nourished for three days prior to the test.

## 5. Treatment

Treating a disease means usually cure and prevention, neither of which being possible with diabetes mellitus unless the aetiology is well known. Treatment, which is palliative, aims consequently to eliminate the classic symptoms and to correct the blood sugar levels in the short term, but also to prevent long-term complications. The ultimate goal is to enable the diabetic patient to lead a normal life. Therapy will vary in response to the heterogeneity of the disease and may include diet and exercise, insulin, and oral hypoglycaemic drugs. This treatment, which is expensive and lifelong, will succeed only if the diabetic and his/her family are educated appropriately.

### 5.1. Dietary and general health measures

The therapeutic approach includes diet and exercise. It wants to maintain the individual's ideal body weight. The diet must be tailored according to the type of diabetes as well as to the subject's current weight, socio-cultural background, and likes or dislikes. An obese subject must follow a low-calorie diet because weight loss increases the efficacy of insulin's action on the target tissues. A non-obese patient should be placed on an unrestricted diet. The ban on sweetened foods and drinks has been challenged and is now considered to be pure superstition. Several, at least three, meals daily are recommended, with the same number of meals each day and same calorie content in each meal. Enriching the diet with undigestible vegetable polysaccharides (fiber) is strongly advised, because the more bulk a food contains the less it is hyperglycaemic. It is sometimes supposed that thousands of African

diabetics, totally lacking medical resources could be kept alive thanks to their generally high-fiber diets.

Physical activity is also an integral part of the African's life, so there is no need to prescribe exercise, except for obese, who constitute only a minority. The patient should be advised how to co-ordinate his or her energy expenditure and diet, and informed about the possible use of medication to avoid hypoglycaemia. Such dietary and general health-related measures are often sufficient to keep in balance non-insulin dependent diabetes. If not, an oral hypoglycaemic agent should be added. For insulin-dependent diabetes, for diabetic pregnant women, and in the event of an acute infection, insulin therapy is unavoidable when oral treatment fails.

### 5.2. Insulin therapy

Insulin is extracted from the pancreas of cattle or pigs or is prepared in laboratories as human insulin. Either rapid-acting (ordinary or regular) or intermediate-acting insulins can be used. Rapid-acting regular insulin starts working half-an-hour after the injection. Its action peaks 1 to 4 hours later and lasts a total of 5 to 8 hours.

If this insulin is used alone, 3 to 4 injections a day are required. The action of intermediate acting (*lente*) insulin starts slowly, peaks 6 to 12 hours after the injection, and lasts up to 24 hours. Two injections a day are usually enough to cover the nycthemeral cycle, but it is preferable to add an injection of ordinary insulin to cover the immediate post-injection period. The common practice in the industrialized world is to administer a mixture of ordinary and *lente* insulin in the morning before breakfast and in the evening before supper. This schedule can be implemented in Africa only if the patient has the opportunity of having several meals during the day. African diabetics, given their eating habits are generally controlled satisfactorily (in the short term) with a daily injection of *lente* insulin.

Bovine, porcine, and human insulin demonstrate similar activities; their small structural differences barely influence their action. However, these differences can be enough to induce antibody formation and the development of allergic reactions, resistance to treatment, and skin lesions (lipodystrophies) around the injection sites. These reactions are enhanced if the insulin contains impurities, hence the current tendency to use highly purified porcine or human insulin (monocompound insulins). Insulin can cause hypoglycaemic reactions (insulin shock), especially if food intake is erratic, and should be taken just before a meal.

### 5.3. Oral hypoglycaemic drugs

If diet alone fails to control blood sugar, the obese type II diabetic without a history of keto-acidosis may be given oral hypoglycaemic drugs (sulfonamides or biguanides). These substances are easily administered, do not require very regular meal times, and are active for at least 24 hours.

The hypoglycaemic sulfonamides stimulate endogenous insulin production and enhance the hormone's action on the target tissues. Unfortunately they are effective in only 20 to 30% of type II diabetics; moreover, they may give rise to allergic and idiosyncratic reactions, as well as to severe hypoglycaemic reactions. The so-called first-generation sulfonamides (tolbutamide, chlorpropamide, acetohexamide, and tolazamide), the therapeutic doses of which are high, have more side effects than the second-generation sulfonamides (glyburide, glibenclamide, glipizide, and glisoxepide) which are effective at relatively low doses.

The biguanides increase peripheral glucose uptake, reduce nutrient absorption in the intestine, and enhance the peripheral action of insulin. They may cause gastrointestinal disorders and lactic acidosis, and are used only while waiting to switch to insulin if sulfonamides fail.

## 6. Prevention

In most cases of diabetes, the exact causes remain a mystery. This makes prevention difficult.

Preventing type I diabetes in man is not yet possible, although successful trials have been performed on laboratory animals (BB rat).

It would theoretically be easier to prevent a large number of cases of type II diabetes, for example, by avoiding a sedentary lifestyle and obesity. This may explain the differing prevalence in town and country. An epidemiological study carried out recently in some Tanzanian villages showed that the prevalence of diabetes was 0.5% among the industrious Sukuma villagers, but rose to 2.5% among the less active Hayas (Ahren, 1984).

Because of its aetiology, malnutrition diabetes, of all the currently known forms of essential diabetes, is probably the easiest to prevent. A protein-rich, well-balanced diet combined with improved methods for removing dietary toxins is fundamental for such prevention. The retting of cassava – a fairly simple procedure to eliminate cyanogens – should be improved, standardized, and practised wherever cassava is eaten.

Our understanding of diabetes mellitus has progressed considerably in the past ten years. While there still is no cure, the possibilities for prevention look much brighter. Today's therapeutic treatments allow the diabetic to lead a normal life. In the Third World, although still heavily handicapped by poverty, diabetics could nonetheless benefit from basic treatment if given some organisational help.

## 7. Diabetes and pregnancy

The association between diabetes and pregnancy deserves a separate study in view of its possible effect on fertility.

A pregnant and hitherto non-diabetic woman may suffer from the so-called diabetes of pregnancy, which generally occurs during the final months in the form of simple glucose intolerance without other symptoms. This condition is temporary and corrects itself after delivery, but it is a risk factor for the foetus.

A current diabetic will also have a high-risk pregnancy. Before insulin could be used, few young diabetic women became pregnant. Diabetes could also cause complications for both mother (exacerbation of vascular complications, pyelonephritis) and child (prematurity, perinatal mortality, congenital malformations). *White's* studies in the USA and *Pedersen's* in Denmark showed that strict surveillance of the metabolism of a diabetic pregnant woman offered the best prospects of bringing the pregnancy to term. The prognosis has improved considerably with the advent of insulin therapy, but it still remains poor for the foetus, with perinatal mortality ranging from 6 to 20 per cent. In some very specialized centres, however, this rate was further lowered thanks to intensive insulin therapy.

Studies of diabetic pregnancies in Africa are extremely rare, few doctors are familiar with their treatment, and rigorous monitoring is often hard to perform. Consequently, both diabetes in women of childbearing age and diabetes of pregnancy are real problems, particularly since female fertility has great social importance in this part of the world.

## 8. Diabetes and health education

Diabetes mellitus is a chronic disease which, if poorly treated, can often result in very disabling complications. In the past it was equated with suffering, deterioration and death within a fairly short space of time; but today diabetics can lead normal healthy lives as

long as some basic health-care practices are observed and the disease is managed correctly.

It is an established fact that careful diabetic control, by keeping blood sugar levels as close to normal as possible throughout the patient's life, can prevent the development of the major complications of diabetes. To inform patients fully and correctly of the risks incurred through ignorance or negligence is thus of paramount importance.

A newly identified diabetic, with no experience of the disease, is suddenly on unknown and thus distressing territory. To dispel this distress the patient needs simple and straightforward education not only about the disease itself but also about the basic dietary rules to follow. He or she must also become familiar with testing his or her urine and blood for sugar and with the administration of insulin or oral hypoglycaemics.

This calls for close collaboration between medical personnel and the patient, who is asked to record in a suitable notebook the various results and events related to the disease, hereby gradually learning to manage part of his treatment independently.

This education must be tailored to each patient's ability to understand the development of symptoms, as well as to his psychological make-up, age, family, professional and cultural background, his social class and language. Education must particularly be adapted to the type of diabetes of the patient.

The patient's family must be taught the fundamentals of the disease and its implications, with emphasis on the importance of diet, close surveillance in case of intercurrent disease, and the correct response to emergencies such as hypoglycaemia (hypoglycaemic or insulin shock) or diabetic coma.

Health workers and health centre or dispensary staff obviously need quite advanced training to enable them to educate the patients correctly. They need to understand fully the fundamentals of diabetes and the responses appropriate to the various situations involving diabetics. Some patients cannot easily accept their disease. Nursing personnel must encourage them, explain-

ing that, constant monitoring and correct care can now allow diabetics to lead normal lives, as long as they participate actively and intelligently in their own treatment.

Finally, both patients and health personnel should be given precise and practical instructions about the diabetics's behaviour, hygiene and diet, the tests to be carried out and the treatment to be followed.

Some excellent brochures have been published for diabetics and health professionals in Zaire. These include the *Guide pour diabétique africain* (Guide for African Diabetics), by J.L. De Plaen, and the more recent *Livre du diabétique* (The Diabetic's Handbook) by Dr. M. De Clerck, both published by Imprimerie St Paul, at Kinshasa.

## 9. Actual situation and research

The first reports mentioning low glucose tolerance in children with protein-energy malnutrition (PEM) were published more than twenty years ago. This type of intolerance was long thought to be a minor complication arising from a much more serious alteration in health status of these patient.

In recent years, however, the problem has been approached from another angle. Studies of the incidence and characteristics of diabetes in developing countries have raised the hypothesis that infantile malnutrition may actually damage the endocrine pancreas, thereby leading to the later development of diabetes. Malnutrition diabetes is estimated to account for 10 to 30 per cent of diabetes cases in these countries.

It is thus important to identify the morphological changes in the pancreas and the irregularities in glucose homeostasis caused by PEM as to determine whether the lesions are reversible. Epidemiological, clinical, and anatomopathological studies are still needed, first to improve understanding of this type of diabetes and then to prevent and/or treat it.

B. Nyomba, R. Bouillon and W. Okitolonda



## BIBLIOGRAPHY

- AHREN B. & CORRIGAN C. (1984), Prevalence of diabetes mellitus in north-western Tanzania, - *Diabetologia*, 26, pp. 333-336.
- AUBRY P. & NYIONGABO TH. (1991), *Diabète sucré tropical*, Journées médicales de Bujumbura d'actualisation en médecine tropicale.
- AUBRY P., ATTIA Y., BARABE P. & al. (1988), Distribution géographique et pathogénie des pancréatites chroniques calcifiantes en zone tropicale, - *Gastroentérol. Clin. Biol.*, 12, pp. 420-424.
- BAJAJ J.S., *Diabetes mellitus in developing countries*, - Interprint, New Delhi, p. 444.
- BARBEZAT G.O. & HANSEM J.D.L. (1988), The exocrine pancreas and protein-calorie malnutrition, - *Pediatrics*, 42, p. 77.
- BEHEYT P. & JORIS H. (1963), Le diabète au Congo, - *Rev. Méd. Liège*, 18, pp. 108-115.
- BOURGOIGNIE J., SONNET J. & DECHEFF G. (1962), Etude clinique du diabète sucré du Bantou de la région de Léopoldville, - *Ann. Soc. Belg. Méd. Trop.*, 42, pp. 261-294.
- CUISINIER-RAYNAL J.C. (1989), Le diabète tropical, - *Presse Méd.*, 18, pp. 703-705.
- CUISINIER-RAYNAL J.C., ETTE N., DUCROS M., GRAND-PIERRE G. & DARRACQ R. (1989), Le diabète sucré de l'adulte en zone tropicale. Facteurs alimentaires et insulino-sécrétion, - *Méd. et Nut.*, 25, pp. 31-36.
- DANO P., TINE M., KLOTZ F., RENAMBOT J. & AUBRY P. (1982), A propos du diabète intermédiaire de l'adulte au Sénégal, - *Dakar Médical*, 27, pp. 247-252.
- DE CLERCK M. (1986), *Livre du diabétique*. Imprimerie St Paul, Kinshasa.
- DODU S.R.A. (1958), The incidence of diabetes mellitus in Ghana, - *W. Afr. Med. J.*, 7, pp. 129-134.
- DODU S.A.R. & HATHORN M. (1966), Diabetes in Accra, - *Ghana Med. J.*, 5, pp. 2-7.
- HAINÉ J. (1958), Considérations à propos du diabète sucré du Congolais en milieu rural, - *Ann. Soc. Belg. Méd. Trop.*, 38, pp. 33-44.
- KANDJINGU K., BIELELI E., BIDIINGIJA M. & TSIANI K. (1985), Etude clinique du diabète sucré à Kinshasa, - *Méd. Afr. Noire*, 32, pp. 53-61.
- KEELEY K.J. (1957), Incidence of diabetes mellitus in the Bantu, - *East Afr. Med. J.*, 31, p. 1284.
- MAC MILLAN D.E. & GEEVARGHESE P.J. (1980), Dietary cyanide and tropical malnutrition diabetes, in: PODELSKY S. & VISWANATHAN M., eds, *Secondary Diabetes, the spectrum of the diabetic Syndrome*, Raven Press, New York, pp. 239-247.
- MBADINGA-MUPANGU (1984), Quelques aspects épidémiologiques du diabète sucré en milieu congolais (à propos de 1108 cas suivis de janvier 1973 à décembre 1980), - *Afr. Méd.*, 23, pp. 13-15.
- MEDECINE D'AFRIQUE NOIRE (1979), Le diabète sucré en Afrique noire (IXème Journées Médicales de Dakar), *Méd. Afr. Noire*, 26, pp. 717-789.
- MOHAN V. & ALBERTI K.G.M.M. (1992), Diabetes in the tropics, in: ALBERTI K.G.M.M., DEFRONZO R.A., KEEN H. & ZIMMET P., *International Textbook of Diabetes Mellitus*, John Wiley, Chichester, New York, pp. 177-196.
- MONTENY V.A.R. (1964), Considérations sur la rareté du diabète sucré dans le Nord-Est du Congo, - *Ann. Soc. Belg. Méd. Trop.*, 44, pp. 969-982.
- OLI J., BOTAZZO G. & DONIACH D. (1981), Islet cell antibodies and diabetes in Nigerians, - *Trop. Geogr. Med.*, 33, pp. 161-164.
- OMAR M.A.K. & ASMAL A.C. (1982), Peptide response to glucagon in Black and Indian insulin-dependent diabetic, - *South Afr. Med. J.*, 61, p. 395.
- PAYET M., SANKALE M., PENE P., BAO O. & TRELLE H. (1960), Les principaux aspects du diabète sucré en milieu africain à Dakar, - *Bull. Soc. Pathol. Exot.*, 53, pp. 903-910.
- PITCHUMONI C.S. & THOMAS E. (1973), Chronic cassava toxicity; possible relationship to chronic pancreatic disease in malnourished populations, - *Lancet*, ii, p. 1397.
- SONNET J., BRISBOIS P. & BASTIN J.P. (1966), Chronic pancreatitis with calcifications in Congolese Bantu, - *Trop. Geogr. Med.*, 18, pp. 97-113.
- TULLOCH J.A. (1963), *Diabetes mellitus in the tropics*, - E. & J. Livingstone, Edinburgh, p. 294.
- WHO (1980) - WHO Expert Committee on diabetes mellitus, second report, - *WHO Techn. Rep. Ser.*, n° 646, 79 p.
- WHO (1985) - Diabetes mellitus, report of a WHO Study Group, - *WHO Techn. Rep. Ser.*, n° 727, 113 p.
- ZUIDEMA P.G. (1955), Calcifications and cirrhosis of the pancreas in patients with deficient nutrition, - *Doc. Med. Geogr. Trop.*, 7, pp. 229-251.

## DISSERTATION AT THE UNIVERSITY OF KINSHASA

- NYOMBA BULANGU LUKUKI (1986), *Interactions between insulin and the vitamin D endocrine system*, Louvain.
- NKANGA NGILA (1984), *Etude clinique de diabétiques*, Université Nationale du Zaïre.

## ANNOTATED BIBLIOGRAPHY

- BOURGOIGNIE J., SONNET J. & DECHEFF G. (1962), Etude clinique du diabète sucré du Bantou de la région de Léopoldville, - *Ann. Soc. Belg. Méd. Trop.*, 42, pp. 261-294.

Clinical and biological features are analysed for 68 cases of diabetes mellitus observed over 30 months between 1959 and 1961 at the University hospital of Leopoldville (Kinshasa). Although the frequency is lower for Africans than for Europeans, the number of cases is not unimportant and is increasing progressively. It is a non-obese insuline dependent diabetes with little acidosis although the glucose level regulation is highly disturbed. There is a slight tendency to high levels of cholesterol in blood but rarely retinopathy and seldom circulatory impairment by endarteritis. Diabetes of Africans is generally very sensitive to insuline and is associated to a high frequency of pancreatitis with calcifications.

As the socio-economic situation makes it chimerical that a diet should be followed, the practical treatment will be to

give intermediate insulin preparations with a slower action than ordinary insulin.

An important bibliography is complementing this contribution.

KANDJINGU K., BIELELI E., BIDIINGIJA M. & TSHIANI K. (1985), Etude clinique du diabète sucré à Kinshasa, - *Méd. Afr. Noire.*, 32, pp. 53-61.

On 1,204 records of diabetes patients selected between 1960 and 1978 at the University clinics of Kinshasa, the authors have analyzed various epidemiological features of the disease in Zaire.

Their conclusion is that diabetes mellitus is quite frequent among low-income populations, in the form of insulin-dependant diabetes. The bibliography covers 25 contributions.