

TESTING IN FAMILY PRACTICE FOR DIABETES

DONALD CROMBIE, M.D.

Birmingham

THE TESTING for diabetics in general practice can be considered under three main headings: What is diabetes? Where should we look for early potential cases? and how should we look for them?

What is diabetes?

For practical purposes 'clinical diabetes' can be arbitrarily defined as an abnormality of glucose tolerance which requires treatment for its control, whether or not it has caused the cardinal symptoms or complications.

The question "What is diabetes?" seems innocent enough but the truth is that we are not absolutely sure. We know that approximately 0.7 per cent of the general population of this country have already been diagnosed by clinicians as suffering from clinical diabetes (College of General Practitioners Working Party, 1962); we know that there are undiscovered in the general population a further 0.3 per cent with diabetic abnormality of a similar degree of seriousness. This figure is less than the generally quoted figure of 0.7 per cent (College of General Practitioners Working Party, 1963) but not all the patients who are discovered to have a diabetic type of glucose tolerance test (G.T.T.) have symptoms or need treatment. For instance, it has been established that 1 in 6 of all males over 50 and 1 in 4 of all females over 70 have 'diabetic' glucose tolerance.

The full diabetic syndrome seems to consist of the following:

1. Diminished glucose tolerance (diabetic G.T.T.).
2. Some or all of the cardinal symptoms of diabetes (thirst, polyuria, loss of weight, pruritus). The cardinal symptoms will appear only if the diet ordinarily taken by the patient overloads his carbohydrate mechanism and sugar appears in the urine in any quantity. In other words, a patient who persistently eats a diet with a very high carbohydrate content will have symptoms of overload where a patient with similar glucose tolerance but on a diet with less carbohydrate may not. This is the basis of the dietary control of mild diabetes.
3. The complications of diabetes. It may also be necessary for nephropathy, retinopathy or neuropathy to be present even in a presymptomatic phase before

the full syndrome of clinical diabetes should be diagnosed. This, however, is controversial.

There are only arbitrary definitions if biochemical criteria are used and there are no boundaries between normality and abnormality except those created artificially by definition. We can only say that individuals have varying degrees of diabeticity when this is measured by the glucose tolerance test. In sharp contrast to the infinitely graded biochemical characteristics (which in this resemble hypertension) are the specific complications, nephropathy and retinopathy and possibly others as yet undefined which are either present or absent and once established tend to progress inexorably, influenced only to a limited extent if at all by treatment. On the other hand, the biochemical abnormalities and hence also the symptoms associated directly with hyperglycaemia and glycosuria can for all practical purposes be eliminated or controlled by treatment. The complications are seldom if ever seen without evidence of present or past gross glucose intolerance.

Where should we look for diabetes?

It would be generally agreed that we must discover all patients with the clinical syndrome of diabetic intolerance and the cardinal symptoms, for these will enjoy perhaps many years of better health as a result of proper treatment.

On the other hand, we must be aware of the disadvantage of disclosing symptomless diabetic abnormality of glucose tolerance. The patient, for so he has now become, is made aware of a defect which for him may have all the unpleasant implications that diabetes can arouse in the lay mind, such as coma, blindness, and gangrene. Until further research has clearly indicated the significance of these lesser degrees of diabetic abnormality, it would seem undesirable to embark on detection drives for the purpose of uncovering these cases, whose only abnormality is that revealed by our own technology.

The kernel of the problem of diabetes detection is "How can we prevent the initiation of the complications".

Until we know firstly the mechanism by which the complications are initiated, and secondly criteria by which we can recognize the individuals among the multitude who already have or are developing diabetic intolerance in whom this may happen, we are on poor ground to argue in favour of mass detection drives based on present

biochemical tests except for the following limited purposes:

First to recognize patients with symptoms who will benefit from treatment, some 0.3 per cent only of the general population, and second to recognize patients with rapidly deteriorating tolerance. As yet the only way of detecting those with rapidly deteriorating tolerance is to do repeat glucose tolerance tests; the younger the patients the more frequent would these have to be done since the tempo of change varies inversely with age. This is why routine testing for glycosuria to detect diabetes in those under 50 is such an unrewarding procedure.

In the current follow-up studies resulting from the survey by the College of General Practitioners being carried out in Birmingham, 17 new cases of clinical diabetes have occurred in the population since the original mass testing for glycosuria two years ago. Sixteen of these new diabetics were non-glycosuric two years ago and only one comes from the group who were glycosuric but non-diabetic.

The practical implications of all this seems to be as follows:

Routine detection by urine testing should be confined to those over 50 in whom diabetes is eight times commoner than in those below this age, and in the high risk groups; that is individuals who are or have been obese, women who have borne more than six children or a baby weighing 10 lb. or more at birth, those who have close relatives who are known diabetics.

These groups can be established in two main ways. Firstly by the general practitioner keeping an age/sex register and a disease index in which he can also keep lists of his 'at risk' groups. Secondly by the general practitioner with help from the local authority establishing the 'at-risk' individuals by circulating a suitable questionnaire by post.

However, far more important to my mind than all this is the necessity for the general practitioner to be aware of the possibility of diabetes during all his routine contacts with his patients. Any vague ill health in any 'at-risk' patient surely warrants at the least a routine urine test for sugar and albumin and a look at the retinae. To this group can be added patients with peripheral vascular disease or neuropathy particularly associated with ulceration or sepsis of the skin of the lower limbs, patients with visual defects, coronary disease or cerebrovascular disease, women with vulvitis and men with balanitis.

Finally and probably most important of all is for the general prac-

itioner to exploit the unique privilege which he has of influencing the way of life of his patients. For instance, his 'at-risk' patients can be helped to avoid obesity by proper dieting.

It may be that prophylactic treatment with the oral hypoglycaemic agents may prevent or delay the onset of complications in those who might otherwise develop diabetes by preventing further deterioration of tolerance, but it is a frightening thought that up to one in six of all males over 50 and one in four of all women over 70 may need to take treatment if diabetes is to be prevented in this way.

How should we look for diabetics?

The particular groups at risk defined above can be screened in various ways. Their urine can be tested for sugar or the blood glucose level estimated. It should also be possible to screen a population by questioning for the cardinal symptoms.

In the College of General Practitioners Survey (1962) each patient was given a glucose-oxidase paper strip contained in a glass vial with silica-gel in the base and sealed by a plastic push-on cap. Attached to each vial was a questionnaire on which the patients were asked to record their names, ages and the results of the tests. The vials were issued with a strong stamped addressed envelope in which they were to be returned to the practitioner after use. The instructions on the use of the enzyme tests were as follows:

Pass urine about one hour after meal. Dip the test stick in fresh urine for five seconds. Read the colour after holding in the air for one minute, and put ring round the answer.

NO CHANGE

BLUE

OTHER COLOUR

Then place the stick in the bottle attached to this card and return it to your doctor in the envelope provided.

During the first three months of the survey vials were given for each member of the family of every patient attending the general practitioners at routine surgery consultations or home visits. In this way a personal contact was made through a member of the family with about 75 per cent of the practice population. The remainder were contacted by post. Reminder letters were sent out during the subsequent three-month period to all those who had not returned their urine tests, and the residual patients still not co-operating were visited personally by the general practitioner, students, or health visitors. All patients reporting a positive test were asked to attend hospital for interview, examination, and a G.T.T. Those unable to travel were tested at home. Not surprisingly, a small proportion of the tests were carried out in unorthodox ways.

Glycosuria

It is known that testing one to two hours after the main meal of the day with Clinistix, which reliably detects down to 0.1 per cent of glucose, will disclose 4 per cent of the population with glycosuria; of this 4 per cent one in ten will have previously undiscovered 'clinical diabetes'.

If a less sensitive test than Clinistix is used, such as the copper-reduction test Clinitest, which reliably detects down to 0.25 per cent of glucose, then only 2 per cent or less of the population will be positive. At the same time for all practical purposes all the previously unknown 'clinical diabetics' will be included in this reduced total. These rates will all rise if the testing is restricted to the at-risk groups, but there will obviously have to be some system of assessment to distinguish the minority of those with 'clinical diabetes' among the total discovered to have glycosuria. This assessment is usually dependent on questioning for the cardinal symptoms of diabetes and estimating the blood glucose level.

Any screening procedure which reduces the proportion of people who will need further reassessment (for instance Clinitest) has an advantage over others, but this has to be balanced against the relative ease with which various tests may be used. The best of both worlds can be obtained by initial screening with Clinistix and retesting the positive urines (4 per cent) with Clinitest. Self-testing by the patients with Clinistix is possible and this obviously has economic and other advantages. This method was used by the College of General Practitioners Working Party in their survey of 19,412 of their patients in Birmingham and proved satisfactory. In this survey advantage was taken of the National Health Service lists of the practitioners who took part, which defined the population to be studied with accuracy and precision. The request to patients to carry out a urine test came directly from the family doctor and the patients' response was excellent, 75 per cent returning their tests without any prompting. Eventually 95.5 per cent of the total population had their urines tested.

The mechanics of detection drives need not involve the practitioner in more than the designing of suitably worded letters of explanation to his patient, with instructions in some detail, if self-testing by the patient is used. Most of the routine of such drives can be carried out by the doctor's secretary. The apparently tenuous link between the patient and his National Health Service doctor is shown by the success of these methods to be far stronger than is usually believed.

This aspect of the doctor-patient relationship can be put to good use in the planning of surveys of all kinds.

There are many advantages to the patient if detection drives are brought to the public via their general practitioners. Some of these reasons have been given; for instance, the likelihood that there will be better co-operation by the patients, and at less cost in materials and manpower than by other methods. Also, immediately after glycosuria is discovered, the patient can consult his own doctor who will have all the information, is in the best position to arrange any further tests, and can allay immediately any undue anxiety. Such testing can also strengthen the relationship between the doctor and his patient, on which all good general practice depends. The practitioner is in the best position to relate any glycosuria to other factors which might influence the assessment of the situation. These include whether or not there is any family history of diabetes, past or present obesity, the previous obstetric history, and a knowledge of the patient's past medical history. He can naturally progress from assessment to therapy if this becomes necessary.

Blood glucose

In America, random blood glucose estimations have sometimes been used instead of urine testing as a screening procedure. This introduces problems in large detection drives, since the patient has to attend a centre in person for the test, and because the results cannot be related to eating habits. The interval between the test and the preceding meal may vary; results are harder to interpret.

Questioning for symptoms

In theory this should be a highly selective method of screening a population. All with symptoms would be generally accepted as being in need of treatment. So far as is known there has been no carefully controlled drive based on questioning as the primary screening method. There are reasons why it may be unsatisfactory and possibly expensive. Most market research, based on questioning, demands that the questions and answers be interpreted by a skilled worker and the same might be true in this field, but it would surely be worth finding out.

Cost of detection

In the College of General Practitioners Survey, based on self-testing by the patient, the cost of detecting a patient with clinical diabetes in the 75 per cent of the population who returned their tests without

any reminder was £6, and for those who required reminder letters or follow-up visits this rose to £17 10s. 0d. The cost of eight glucose tolerance tests must be added to each of these figures, since on average only one glycosuric in eight was a clinical diabetic. (No costing of the time of the doctor and planning staff was made.)

REFERENCES

- College of General Practitioners Diabetes Survey Working Party (1962). *Brit. med. J.*, **1**, 1497.
College of General Practitioners Diabetes Survey Working Party (1963). *Brit. med. J.*, **2**, 655.
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Congenital Abnormalities

Members and associates who took part in the collection of material for the survey of congenital abnormalities will be interested to learn that a preliminary report under the title "Seasonal Variation in Congenital Abnormalities" has been published in *The British Journal of Preventive and Social Medicine*.

A seasonal variation in congenital abnormalities had been reported in the past by workers in several parts of the country. For instance McKeown and Record showed that the incidence of anencephaly in Birmingham and in Scotland was higher during the winter than in the summer and Guthkelch reported a similar seasonal trend for spina bifida in the Manchester Children's Hospital.

The College's survey embracing 12,602 defects is far more comprehensive than any previously reported series and shows winter excesses for cataract, anencephaly, spina bifida, oesophageal atresia and congenital dislocation of the hip, and a summer excess in aortic or pulmonary stenosis or abnormality of the aortic and pulmonary arches, abnormality of the lower end of the gut, and partial absence or defect of limbs. These variations in seasonal incidence are statistically significant but there is so far no obvious reason why they should occur; particularly, why should more babies be born with oesophageal atresia in the winter and more with defects of the lower gut in the summer?

Much more information must be collected and further analyses made of these findings which at present appear to be inexplicable.

*Slater, B. C. S., Watson, G. I., and McDonald, J. C. (1964). *Brit. J. prev. soc. Med.*, **18**, No. 1.