17 hours the damage was sometimes partially reversible. However, with 24 hours' preservation severe irreversible damage resulted. The interpretation of the microscopical changes in relation to those occurring in renal homografts is discussed.

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GLUCOSE TOLERANCE AND GLYCOSURIA IN THE GENERAL POPULATION

REPORT OF A WORKING PARTY* APPOINTED BY THE COLLEGE OF GENERAL PRACTITIONERS

In a previous report (Diabetes Survey Working Party, 1962) we described an investigation in Birmingham into the causes of glycosuria in a large and representative sample of the general population of England and Wales (19,412). The screening method was based on self-testing of the urine one hour after the largest meal of the day, using a glucose oxidase paper strip ("clinistix"). Those subjects who found glycosuria had a 50-g. oral glucose-tolerance test (G.T.T.). Using the conventionally accepted criteria in interpreting the G.T.T., we obtained results which showed that the rate for known diabetes was 0.64% and for previously undiagnosed diabetes 0.69%. These figures were in close agreement with previous surveys and indicated that the method of self-testing was as successful as any other in the discovery of undiagnosed diabetes. It might be assumed that this rate represented the total of those showing diabetic glucose tolerance, and that those who had no glycosuria on screening had normal tolerance. However, we have already reported that examination of 123 control subjects showed that abnormal tolerance was frequently met with in this supposedly "normal" group. The present paper reports on further studies of controls and seeks to establish the true prevalence of abnormality of glucose tolerance in the general population. The value of the test in the diagnosis of true diabetes is further considered.

Method

The control subjects were those who had not reported glycosuria in the original survey. They were drawn from the age–sex registers of the complete practices maintained by the family doctors (M.R.C. Committee for Research in General Practice, 1960). This was done by taking the next person of the same age and sex in alphabetical order following each person who had reported glycosuria or who had known diabetes. Members of the same family or those living at the same address were excluded. Seven out of the original ten practitioners took part in the study, and on their registers there were 461 persons who had diabetes or who had reported glycosuria out of the 584 in the original survey. An invitation to assist in this study was accepted by 247 control subjects, and on these a G.T.T. has been carried out. The additional subjects required to construct an adequate sample were drawn from the age–sex registers.

Table I compares the age and sex distribution of this constructed control group with the original survey group of patients who had either glycosuria or known diabetes. The percentage age and sex distribution of the general population is also shown for comparison. Those reporting glycosuria or with known diabetes were older than the general population, and therefore the sample of control subjects is older than the average. The percentage age distribution for the survey and control groups matches more closely for the men than for the women. There is an excess of control-group women aged 50–69 and a deficit of those aged 70 and over.

TABLE I.—*Age and Sex Distribution (With Percentages) of Survey Population, Constructed Control Group, and Population of England and Wales*, 1959

Age	Male		Female		Population	
	Survey	Control	Survey	Control	Male	Female
0–29	47 (14·4)	18 (11·1)	61 (23·8)	23 (12·6)	44·3	40·0
30–49	120 (36·8)	69 (42·6)	52 (20·3)	34 (18·6)	28·4	27·1
50–69	118 (36·2)	56 (34·6)	89 (34·0)	99 (54·0)	21·5	23·8
70+	41 (12·6)	19 (11·7)	56 (21·9)	27 (14·8)	5·8	9·1
Total	326 (100)	162 (100)	258 (100)	183 (100)	100	100

G.T.T.s were carried out for the most part 15 to 20 at a time at the practitioners' surgeries on Sunday mornings. Some were also done in hospital and in the patients' own homes. All the specimens of blood and urine were analysed in the same laboratory, and the procedure and methods were the same as those previously reported (Diabetes Survey Working Party, 1962).

*Members of the Working Party: Dr. J. M. Malins, Dr. M. G. FitzGerald, and Dr. R. Gaddie, of the General Hospital, Birmingham; Dr. K. W. Cross and Miss Mary Wall, of the Department of Social Medicine, Birmingham University; Dr. A. J. Allen, Dr. Amy M. Allen, Dr. D. L. Crombie, Dr. L. S. Gathergood, Dr. C. M. Green, Dr. R. H. Morgan, Dr. A. J. Pearce, Dr. L. A. Pike, Dr. R. J. F. H. Pinsent, and Dr. G. W. Thorpe, of the College of General Practitioners.

Dr. D. L. Crombie and Dr. M. G. FitzGerald were the recorders.

Definitions

The categories used to interpret the results were defined as follows:

"Diabetic" Abnormality.—All those tests in which the 1-hour capillary-blood-glucose figure exceeded 180 mg./100 ml. and the 2-hour figure 120 mg./100 ml. These have been subdivided into (*a*) florid diabetes where the fasting value exceeded 130 mg./100 ml. and (*b*) G.T.T. diabetes where the fasting value was under 130 mg./100 ml.

Lag-storage Curves.—Level over 180 mg./100 ml. at ½ or 1 hour with fasting levels under 110 mg./100 ml., 1½-hour figures under 160 mg./100 ml., and 2-hour figures under 120 mg./100 ml.

Renal Glycosuria.—Glycosuria found by "clinitest," confirmed as true glucose by clinistix, no blood glucose being above the normal values.

"Miscellaneous" Abnormality.—These tests have been subdivided into the following:

Group A, where the fasting value exceeded 110 mg./100 ml. but the 2-hour figure was below 120 mg./100 ml.

Group B, where the figure at 1½ hours exceeded 160 mg./100 ml. and the ½-hour or 1-hour figure or both these figures exceeded 180 mg./100 ml. while the fasting level was below 110 mg./100 ml. and the 2-hour figure below 120 mg./100 ml.

Group C, where the 2-hour figure exceeded 120 mg./100 ml. while the fasting and 1-hour values were normal.

Normal.—No glycosuria using clinitest, although enzyme test might have been positive. All blood values normal—that is, below 110 mg./100 ml. fasting, 180 mg./100 ml. at ½ hour, 180 mg./100 ml. at 1 hour, 160 mg./100 ml. at 1½ hours, and 120 mg./100 ml. at 2 hours.

Unclassifiable.—Results not conforming to any of the above categories.

Results

Table II shows the actual numbers in the various categories in the constructed control group.

TABLE II.—*Distribution of Categories in the Constructed Control Group*

	No.			%
	Men	Women	Total	
"Diabetic" abnormality:				
Florid	—	1	1	(0·3)
G.T.T.	14	12	26	7·5
Lag storage	27	25	52	15·1
Renal glycosuria	2	—	2	(0·6)
Miscellaneous abnormality:				
A (high fasting)	5	9	14	4·0
B (hump curves)	4	6	10	2·9
C (high 2-hr.)	11	28	39	11·3
Unclassifiable	3	7	10	2·9
Normal	96	95	191	55·4
Total	162	183	345	100

Figures in parentheses are based on small numbers.

Diabetic Abnormality.—Twenty-seven subjects were classed as showing diabetic abnormality, and Fig. 1 shows the superimposed blood-glucose values in these cases during the course of the G.T.T. Although most of the fasting values were below 110 mg./100 ml., the curves are just as abnormal as those in subjects found to have G.T.T. diabetes in the original survey. Only one subject had a fasting blood glucose in excess of 130 mg./100 ml. and must therefore be classed by us as having florid diabetes. None of the subjects had symptoms of diabetes and none is receiving insulin or oral hypoglycaemic drugs. Three have been instructed in use of a low-carbohydrate diet because of obesity, and all are being followed up.

Lag-storage Curves. — These were very common (Table II), being found in 52 cases (15%). Men and women contributed equally to the numbers, whereas in the original survey four times as many men as women had shown this phenomenon. This is explained not by any differences in the height of the blood-glucose peaks between the groups but by the fact that fewer women produced heavy glycosuria and therefore presumably did not report positive tests in the original survey (see Glycosuria).

Renal Glycosuria.—As expected this was uncommon and was recorded only twice.

Miscellaneous Abnormalities.—There were a large number of these (Table II), and there seems to be no justification for treating them as a homogeneous group. Of the 39

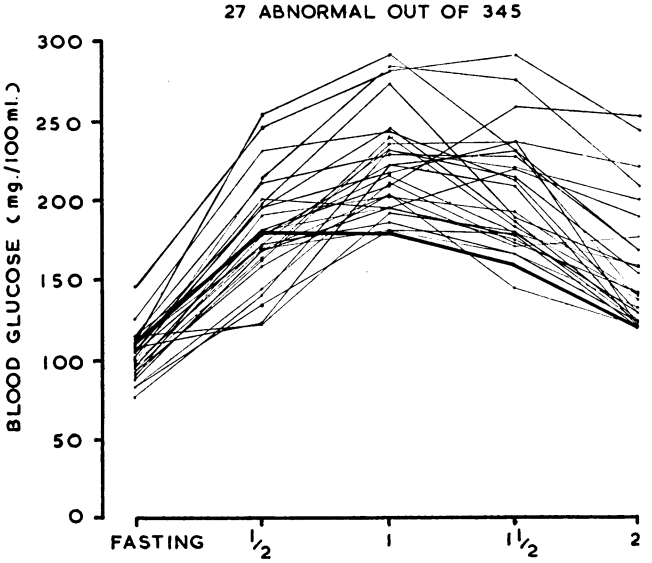

FIG. 1.—"Diabetic" abnormality of glucose tolerance in control group.

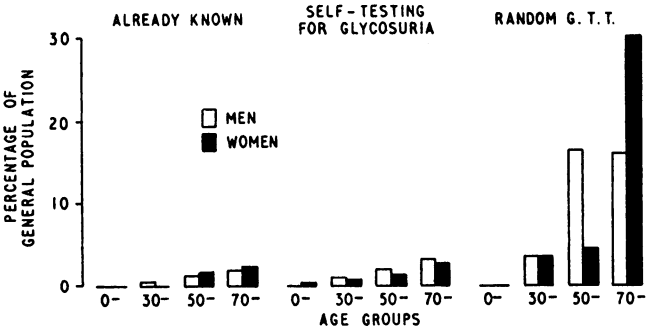

FIG. 2.—Age and sex distribution of "diabetic" abnormalities as percentage of general population. Comparison of rates in those already known to have diabetes, in those who found glycosuria on self-testing, and from the random use of the G.T.T.

cases in group C who had normal fasting and 1-hour blood sugars and abnormal 2-hour figures, 16 (41%) lay between 120 and 130 mg./100 ml. This peculiarity of the G.T.T. has already been commented on by Mosenthal and Barry (1950) and by Fajans (1960), and is regarded by them as normal.

Unclassifiable.—Ten of the curves did not conform to any of the categories, and may well have been due to technical errors.

Age and Sex Distribution of Diabetic Abnormalities.—The age and sex distribution of the subjects in the various categories from the original survey is shown in Fig. 2, the height of the histogram representing a rate of abnormality in the general population for the age-groups 0–29, 30–49, 50–69, and 70 and over. In the control group no diabetic abnormality was found under the age of 30 and 2.9%

between 30 and 50. About one in six of all men over 50 had a diabetic abnormality. The rate for women aged 50–69 was only half of this, but rose to one in four for aged 70 and over. Known diabetics and those discovered in the original glycosuria survey are also included for comparison. Since all the results are plotted on the same scale the figure illustrates how much more frequently diabetic abnormality was found by the random use of the G.T.T. than by self-testing for glycosuria.

Total Prevalence of Abnormal Tolerance.—Table III combines the results from the original survey (Diabetes Survey Working Party, 1962) with the present findings, and gives the rates of abnormality for the various categories in a large sample of the general population. The rates are shown separately for under and over the age of 50.

TABLE III.—*Estimated Percentage Prevalence of Normal and Abnormal Glucose Tolerance in the General Population*

Age:	Under 50	50 and Over	Total
Known diabetics	0·21	1·57	0·64
"Diabetic" abnormality:			
Florid	0·06	1·40	0·49
G.T.T.	1·32	13·09	5·08
Lag storage	5·18	17·94	9·26
Renal glycosuria	0·36	(1·25)	0·65
Miscellaneous abnormality:			
A (high fasting)	2·22	5·29	3·20
B (humps)	1·66	4·19	2·46
C (high 2-hr.)	11·13	10·55	10·94
Unclassifiable	2·16	3·23	2·50
Normal	75·70	41·49	64·78
Total	100	100	100

Figure in parentheses is based on small numbers.

Glycosuria.—In the control group 15% of all the subjects passed glucose detectable by the glucose oxidase paper test (clinistix) at 1 hour, and in 6% this was also detectable by the tablet form of Benedict's test (clinitest). With either method a higher proportion of the men had glycosuria (Table IV). This difference is presumably due to renal causes, since there was no difference between the sexes in the height of the blood-glucose readings. More men than women reported glycosuria in the original survey, and hence there was an apparent excess of men in many of the categories.

TABLE IV.—*Percentage with Glycosuria at 1 Hour Using Clinistix and Clinitest. Men and Women Compared in Various Categories*

	Clinistix		Clinitest		No. in Category	
	M	F	M	F	M	F
Normal	8·1	3·2	—	—	98	95
G.T.T. diabetes	42·9	41·7	35·7	25·0	14	12
Lag storage	48·1	12·0	22·2	4·0	27	25
Other abnormality	34·3	10·0	17·4	4·0	23	51
Total	21·0	9·3	9·3	3·8	162	183

The sex difference in the total is significant. $P<0.05$ for clinistix and $P<0.01$ for clinitest.

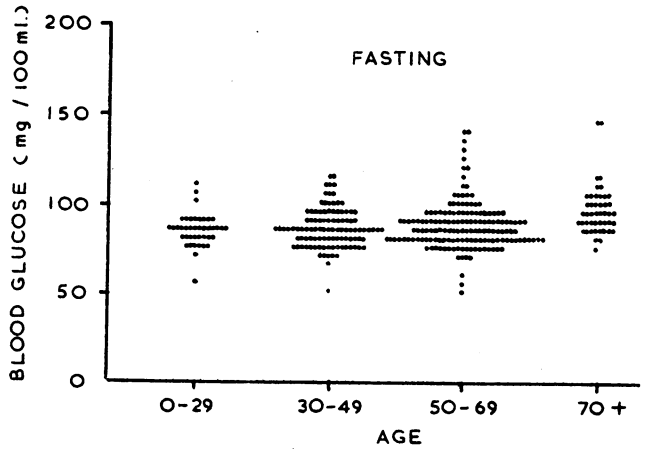

FIG. 3.—Blood-glucose values fasting in different age-groups.

Blood Sugar and Age

Figs. 3, 4, and 5 plot the distribution of blood glucose in the various age-groups fasting, at 1 hour, and at 2 hours after 50 g. of glucose, and the mean and standard deviations are given in Table V. The mean fasting value was surprisingly constant (about 87 mg./100 ml.), until 70 and over, and then the increase (99.1 mg./100 ml), though

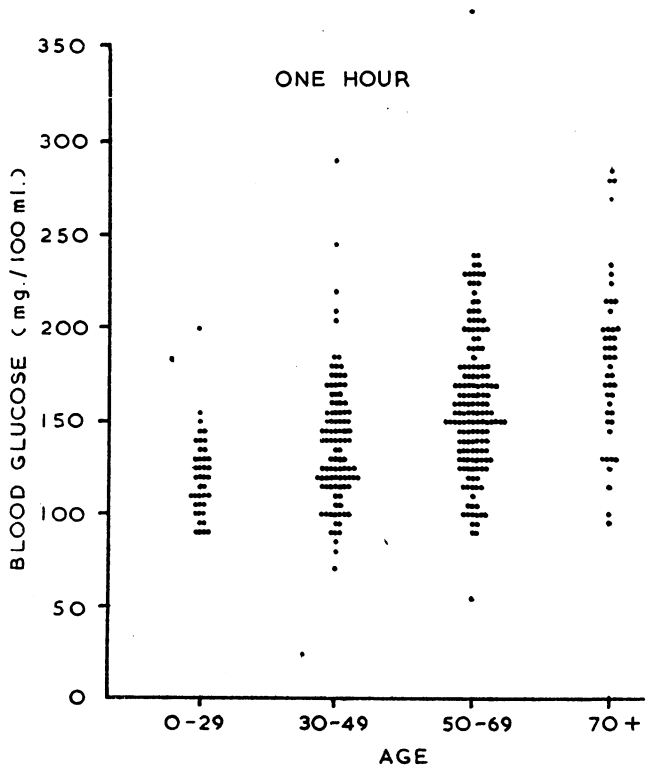

FIG. 4.—Blood-glucose values one hour after 50 g. of glucose in different age-groups.

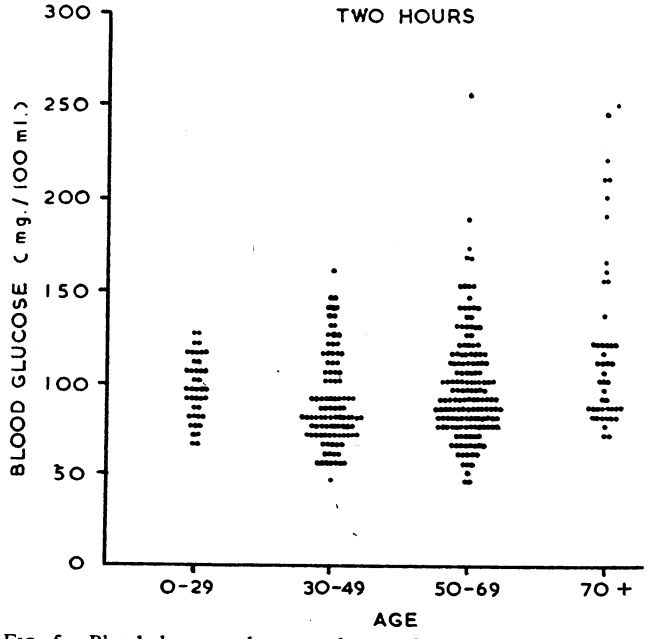

FIG. 5.—Blood-glucose values two hours after 50 g. of glucose in different age-groups.

significant, was comparatively slight. There was no difference between the sexes. There were five subjects under the age of 70 whose values exceeded 120 mg./100 ml. These were from the miscellaneous group A and may not have fasted properly—that is, fasting values over 110 mg., other values normal.

TABLE V.—*Mean Capillary Blood Glucose (mg./100 ml.) for Various Age-groups, Fasting, 1 Hour, and 2 Hours After 50 g. of Glucose*

Age-group	No. of Cases	Fasting		1 Hour		2 Hours	
		Mean	S.D.	Mean	S.D.	Mean	S.D.
0–29	41	85·8	8·8	122·1	22·0	97·7	16·7
30–49	103	87·8	11·4	141·2	34·7	90·2	24·7
50–69	155	89·7	13·7	162·0	40·6	98·5	31·0
70+	46	99·1	16·3	186·0	43·3	118·8	44·3

One hour after the glucose (Fig. 4) a very wide range of readings was found and the mean blood-glucose values increased steadily with advancing age. At 2 hours, however, the pattern approximates to that observed in the fasting state, with the mean increased only at age 70 and over. The mean values at 2 hours did not fall as low as the mean fasting values, but there was a substantial minority in whom the 2-hour value did fall below the fasting value.

These readings are only from those who did not show glycosuria in the original detection drive. If those with glycosuria and with known diabetes are added (3.4% of the general population) then a further 11 points (3.4% of 345) would be added, and this makes no significant difference to the mean values.

Finding Abnormal Glucose Tolerance

Table VI shows the estimated percentage of the general population who would be found to have abnormal glucose tolerance or glycosuria, using various testing techniques. The results are based on the findings from the survey and control groups and have been corrected for age and sex to provide an estimate for an average National Health Service practitioner's list of 2,500 patients.

TABLE VI.—*Estimated Percentage of Population with Glycosuria or Abnormal Glucose Tolerance in the Various Categories, Using Different Methods of Discovery*

Method of Discovery	"Florid" Diabetes	"G.T.T." Diabetes	Non-diabetic Glycosuria
Already known	0·64 (16)	—	—
Glycosuria on enzyme test 1–2 hours after main meal, excluding those already known	0·30 (8)	0·39 (10)	1·82 (45)
Glycosuria on enzyme test 1–2 hours after 50 g. glucose, excluding those already known and those with glycosuria after a meal	* [1]	2·31 (58)	10·99 (275)
Discovered after routine G.T.T. on patients aglycosuric after 50 g. glucose	0	2·38 (60)	—
Total	0·94 (24)	5·08 (128)	12·81 (320)

Figures in parentheses are the estimated numbers in a practice of 2,500 patients.
* No rate can be calculated, as only one case was discovered.

Enzyme testing after the main meal of the day uncovers all the unknown "florid" diabetics with symptoms and 55 additional patients who will need watching; while enzyme testing 1–2 hours after 50 g. of glucose following a 12-hour fast will probably uncover no further "florid" diabetics but adds another 333 patients to the group who will need watching, or a total of 388. A routine G.T.T. adds 60 cases with "diabetic" tolerance but no glycosuria without uncovering further "florid" cases.

Redhead (1960) has shown that testing with clinitest instead of enzyme test 1–2 hours after the main meal of the day will uncover nearly all the "florid" diabetics with symptoms but will disclose only half as many patients with non-diabetic glycosuria who would need watching.

Discussion

It has already been shown in Birmingham (Diabetes Survey Working Party, 1962) that the incidence of newly discovered diabetic abnormality of the G.T.T. (0.69%) is much the same as that of known diabetes (0.64%), this being the result normally found in detection surveys whatever technique is used. In addition there was a substantial group (0.83%) with glycosuria as a result of the lag-storage effect or of renal glycosuria, both of which conditions are regarded by us as innocent and unrelated to diabetes. It did not seem likely that the G.T.T.s of those who had failed to find glycosuria in the survey would add greatly to the sum of abnormalities discovered, and the result was a surprise for which no explanation has been found. In the event the estimated incidence of abnormal tolerance was raised to 6.2%, excluding miscellaneous abnormalities of the G.T.T., some of which may be diabetic in character. This high figure is far above the percentages recorded in surveys, including our own. The most obvious explanation of this discrepancy is that subjects with G.T.T. diabetes may have no glycosuria after a meal and so escape the initial survey detection.

In the survey of Wilkerson and Krall (1947) and of Kenny, Chute, and Best (1951) postprandial blood sugars were taken as well as urine specimens, but the incidence of discovered abnormality was 0.6% and 0.5% respectively in the tested populations. It appears that a meal is considerably less provocative of hyperglycaemia than a 50-g. dose of glucose taken fasting. Before suggesting that 6.2% of the population have diabetes the accepted interpretation of the G.T.T. may be questioned, for the bulk of the diabetic abnormalities (5.08%) are due to G.T.T. diabetes, which is seldom associated with clinical manifestations. As yet there is no clear evidence to show how often G.T.T. diabetes leads to the clinical disease.

Wilkerson and Krall (1953) and Wilkerson, Krall, and Butler (1959), using one- to two-hour-postprandial venous blood-sugar levels, have followed a "blood-sugar suspect" group in their Oxford Survey from 1946 to 1953 and consider that the rate of development of diabetes in this group is eight times that of a normal control group. The majority of those who had become "diabetic" still had fasting blood-sugar levels below 130 mg./100 ml. when they were reclassified, and would by our standards remain in the suspect group of G.T.T. diabetes. At present it seems probable that the rate of conversion to clinical diabetes from G.T.T. diabetes will prove to be high, but it is interesting to find that all the new cases of clinical diabetes that have so far arisen in our survey population have come from the group who were normal on testing.

The high incidence of G.T.T. diabetes in a normal population (13% over the age of 50) has to be remembered when judging whether diabetes is significantly associated with other diseases, particularly those which occur mainly in later life and confine the patient to bed. Immobilization is usually regarded as the cause of an increased incidence of abnormal glucose tolerance as age advances. Blotner (1945) found that the G.T.T. was commonly abnormal in both adults and children in hospital, and no doubt the percentage of abnormal tests in an elderly hospital population would be much greater than our already high figure. Mosenthal and Barry (1950) found no difference between the tests of a group aged 61 to 76 and those of younger subjects. While it is true that there is no steady increase of mean blood-glucose levels with increasing age, the pre-

valence of abnormal tolerance tests clearly rises after the age of 50.

Table VI gives some indication of the problem which a practitioner could create for himself by testing the urine of every patient in his practice. Assuming that a finding of glycosuria can be followed by a glucose-tolerance test, the largest group, that of non-diabetic glycosuria, could be diagnosed and discarded.

With enzyme testing after a meal as the screening method the "follow-up" group would consist of seven cases of florid diabetes, which certainly needed discovery, and 10 cases of G.T.T. diabetes, which would entail a positive effort to recall them for an annual review. If clinitest rather than the enzyme test were used the number of positive results could be reduced by about half (Redhead, 1960) but would include nearly all the florid cases. The testing of *fasting* specimens by clinitest would, for practical purposes, reveal only florid diabetes, but more than half of these cases awaiting diagnosis will not be found by this method.

Summary

A total of 345 oral 50-g. G.T.T.s were carried out on a sample of the general population. The subjects were chosen from those of the survey population (19,412) who had not found glycosuria using clinistix after the largest meal of the day. They were approximately matched for age and sex with those who had found glycosuria.

Twenty-seven had a diabetic abnormality, 52 had lag-storage curves, and 63 had other miscellaneous abnormalities. Only 191 had completely normal glucose tolerance, though a further 10 were unclassifiable and two had renal glycosuria.

Under conditions of the G.T.T. one hour after glucose 21% of the men and 9.3% of the women now had glycosuria detectable by clinistix; 9.3% of the men and 3.8% of the women had glycosuria detectable by clinitest.

No diabetic abnormality was found under the age of 30 and only 2.9% at age 30–49. At 50 and over, however, about one in six of all men and at 70 and over about one in four of all women had a diabetic abnormality.

The mean fasting blood-glucose value was constant (87 mg.) until 70. At 70 and over the increase, although significant, was comparatively slight (99.1 mg.). One hour after the glucose the mean blood-glucose values increased steadily with age and showed a wide range of readings. At two hours the mean values still exceeded the fasting values and showed a greater range.

If the results from the investigation of those with diabetes or glycosuria (584) in the 19,412 who constituted the survey population are combined with that of the control group subjects (345) we estimate that the prevalence of diabetic abnormality in the general population is 6.2%, or 14.5% of those over 50.

We gratefully acknowledge grants from the Nuffield Provincial Hospitals Trust, the British Diabetic Association, the Lawson Tait Memorial Fund, and the Research Foundation of the College of General Practitioners. We also acknowledge technical assistance given by the staff of the Biochemistry Department, General Hospital, Birmingham, and also by Mrs. M. Jones and Sister E. S. Streather.

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"IDIOPATHIC" PES CAVUS

AN INVESTIGATION INTO ITS AETIOLOGY

BY

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Pes cavus is common and often one of the most severe of foot deformities; yet our knowledge of its causes is scanty and we can only guess at the mechanisms by which it is produced. Prompted by this lack of knowledge, it was decided five years ago that a "pes cavus clinic" should be set up so that a systematic prospective investigation into the causes of "idiopathic" pes cavus could be undertaken. This clinic started at the Royal National Orthopaedic Hospital in 1958 with the intention that all new patients should be thoroughly examined and investigated and that the clinic should be kept going indefinitely so that we could learn more of the development of the deformity and its prognosis.

This interim report follows a review of the first 77 new patients coming to the clinic.

A great deal has been writen about pes cavus, but most authors refer to the mechanism of production of the deformity or its treatment and very few to the possible cause of the deforming mechanism. Turner (1952) was probably the first to emphasize that there was an underlying abnormality of the central nervous system in so-called idiopathic cases. This association had been noted before by Plowright (1928) in one large family, some of whom had peroneal muscular atrophy while others had pes cavus without detectable neurological disorder. Spillane (1940) studied another family, some of whom suffered from peroneal muscular atrophy and Friedreich's ataxia, and he showed that patients with these disorders may have children who have pes cavus but are otherwise clinically normal. O'Connor (1959) expressed the opinion that the underlying cause was to be found in some abnormality of the spinal cord.

The many theories propounded for the local mechanism producing the deformity are listed in Table I. By far the most popular and generally accepted opinion is that of Duchenne (1867). He believed that the cavus foot and claw toes are produced simply by weakness of the interossei. This is an attractively simple theory and one which is generally assumed to be correct. Duchenne based this belief upon the study of 12 patients in whom he showed that electrical stimulation of the interossei gave a reduced or absent response. However, these investigations were