The Diagnosis of 'Pathological Hyperglycaemia' in Gestational Diabetes in a High Risk Obstetric Population

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In order to define a level of 'pathological hyperglycaemia', i.e. glucose intolerance that predicts perinatal morbidity among the obstetric population, 100 g glucose tolerance tests (GTTs) were performed in 660 patients attending for antenatal care at the University Hospital in Jeddah. The results were analysed in two ways: (1) patients were stratified according to the number of abnormal glucose values on the GTTs and (2) patients were placed into one of three groups according to the 100 g GTT diagnostic criteria, i.e. normal (non-GDM), abnormal with fasting blood glucose (FBG) ≥ 5.8 mmol l^{-1} (GDM), and abnormal with FBG <5.8 mmol l^{-1} (gestational induced hyperglycaemia, GIH). Although there was a stepwise association between fetal/maternal morbidity with increasing number of abnormal glucose values, no level of glucose intolerance could be defined as a threshold level for normal response. However, when stratified by FBG, GDM patients were significantly heavier (78.5 kg ± SD 14.9), had a higher incidence of both macrosomia (27.5 %) and operative delivery (25.3 %) than the other two groups (14.7 %, 14.3 %, and 15.4 %, 12.8 % in the non-GDM and GIH, respectively). It is suggested that among patients with abnormal GTT results a FBG ≥ 5.8 mmol l^{-1} identifies a threshold for true 'pathological hyperglycaemia'.

KEY WORDS **100 g Glucose tolerance test Gestational diabetes Fasting hyperglycaemia Macrosomia**

Introduction

There is no doubt that gestational diabetes mellitus (GDM) is a controversial medical disorder. While there is almost unanimous agreement that maternal hyperglycaemia in pregnancy increases the risk of fetal morbidity, there is no agreement on a threshold level for pathological hyperglycaemia, i.e. a particular degree of hyperglycaemia that is associated with a significant increase in fetal/maternal mortality. Some authors have considered that even a limited degree of maternal hyperglycemia may affect pregnancy outcome adversely.[1] Others have condemned all forms of screening for GDM, except for research purposes.[2]

Neither of these extreme views should be adopted without prior evaluation of the effect of GDM in a given population. Adopting too liberal criteria for the diagnosis of glucose intolerance will unnecessarily increase the number of women labelled as having GDM. Conversely, to abandon screening for GDM may result in increased fetal morbidity or even mortality.

The 100 g GTT, originally described in 1964 by O'Sullivan and Mahan, is still recommended as the gold standard for the diagnosis of gestational diabetes in the USA.[3,4] Recently its validity for today's American population has been questioned.[5] It could however be argued that the fetal response to hyperglycaemia is a physiological one, not influenced by racial or ethnic variables. Consideration of the power of the test to identify true negative from true positive should be related to its ability to identify cases with significantly increased risk of fetal/maternal complications. In this respect, when defining pathological hyperglycaemia in a given population, other potentially confounding variables should be considered such as maternal obesity, birth rate, fetal loss rate, the risk of certain congenital fetal malformations.[6] These are obviously changing variables that not only affect the importance of screening for GDM but also influence the diagnostic criteria that should be adopted for that population.

The maternity unit of the University Hospital in Jeddah city caters for about 2200 deliveries per annum, of mostly booked or referred cases. The population is predominantly middle social class Saudi women, with small proportions from African and other Arab countries.

Abbreviations: *AS* Apgar score, *FBG* fasting blood glucose, *GDM* gestational diabetes mellitus, *GIH* gestation-induced hyperglycaemia, *GTT* glucose tolerance test.

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We were concerned that the adoption of the O'Sullivan criteria in our unit might result in an overdiagnosis of patients labelled as having GDM, with adverse effects on maternal well-being and on the service provider.[7] Therefore the objective of the present study was to identify a level of pathological hyperglycaemia that predicts perinatal morbidity, using the O'Sullivan and Mahan test protocols.

Materials and Methods

Over a period of 14 months, all antenatal patients attending the author's (H.N.) clinic—except women with established diabetes—were requested to have a diagnostic 100 g GTT between 24 and 28 weeks of gestation. The tests were normally performed in the hospital out-patient clinic. After 12 h overnight fast, each woman received 100 g glucose in 300 ml of water. Venous blood was obtained before the solution was ingested and 60, 120, and 180 min thereafter. Plasma glucose was determined by α glucose-oxidase method[8] using a glucose analyser (Beckman, Fullerton, CA, USA).

The diagnosis of GDM was based on the O'Sullivan and Mahan criteria. Two or more of the venous plasma glucose concentrations had to be met or exceeded: fasting, 5.8 mmol l^{-1}; at 1 h, 10.5 mmol l^{-1}; at 2 h, 9.1 mmol l^{-1}; and at 3 h, 8.1 mmol l^{-1}.[4] Our protocol for the management of diabetes in pregnancy have been previously described.[9] In summary, women diagnosed as having GDM are usually admitted to the hospital for 1 or 2 days for evaluation and initiation of treatment. A standard diabetic diet is prescribed and metabolic control is evaluated by 24 h blood glucose profile (plasma glucose at fasting, and 2 h postprandial). Insulin is initiated if either fasting or 2 h postprandial glucose levels are more than 5.8 or 7.8 mmol l^{-1}, respectively, on two or more occasions. Once satisfactory metabolic control is achieved, the patient is discharged home for subsequent follow-up as an out-patient. Since home blood glucose monitoring is not usually practised in this unit, arrangements are made for patients to attend the clinic for blood glucose measurements.

In all patients, the following characteristics were noted: maternal age, parity and weight at the time of the GTT. Known risk factors for GDM were noted: first degree family history of diabetes mellitus, previous history of GDM, 'bad' obstetric history, and history of glycosuria. A 'bad' obstetric history included more than three previous spontaneous abortions, stillbirths, neonatal deaths or malformations.

After delivery the medical records were reviewed and the following outcome measures were noted: mode and week of delivery, birth weight, incidence of macrosomia, and Apgar scores (A.S.) <5 and <7 at 1 and 5 minutes, respectively. Macrosomia was defined as fetal weight over the 90th percentile of birth weight for gestational age, based on growth standards developed for the local population.[10]

The relation between the degree of abnormality in the GTTs results and the studied variables was examined in two ways. First, patients were stratified into five groups (A to E) according to the number of abnormal glucose values in the GTTs: group A had no abnormal values, group B 1, group C 2, group D 3, and group E 5 abnormal values, respectively. In the second analysis, the studied population was divided into three groups according to the GTTs results: normal patients (non-GDM), GDM patients with high fasting blood glucose (FBG $\geq$ 5.8 mmol l^{-1}), and GDM patients with low fasting blood glucose (FBG < 5.8 mmol l^{-1}). In this analysis, the latter group of patients were described as patients with gestational induced hyperglycaemia (GIH).

Statistical Analysis

Analysis of data was performed using SPSS statistical package version 0.3 for Windows (SPSS Inc., Microsoft Corp., Chicago, IL, USA) on an IBM compatible PC. Data are expressed as means ($\pm$ SD), and incidences as appropriate. Statistical analysis included descriptive procedures, linear correlation, and regression, and one-way analysis of variance (ANOVA) for ranked data with Boneveil test for in-between group analysis for statistical significance. A p value <0.05 was considered significant.

Results

During the study period a total of 702 women consented for GTT (98.8 % of the total antenatal population). Of these, 42 patients were excluded from the final analysis (29 delivered in another hospital, 5 could neither continue nor repeat the test, and in 8 patients data were incomplete). Of the remaining patients (n = 660), 130 (19.7 %) were diagnosed as having GDM diabetes. Insulin was required for metabolic control in 22 (16.9 %).

The mean maternal age of the total population was 29.0 years $\pm$ 3.1 (SD), weight 71.6 kg $\pm$ 14.7, parity 5.1 $\pm$ 3.1, and fetal birth weight 3322 g $\pm$ 582; 53 % of births were male. Risk factor(s) for GDM were present in 499 (71.1 %) patients (one patient had four risk factors while 3.0 %, 26.1 % and 70.7 % patients had three, two and one risk factors, respectively). The incidence of GDM was not significantly different among patients who had risk factor(s) for GDM compared with those who did not (20.8 % and 16.2 %, respectively).

Results of Data Analysis According to the Number of Abnormal Glucose Values on the GTTs (Groups A–E)

The mean glucose values of the GTTs results for the five groups are illustrated in Figure 1. At all sampling times the mean blood glucose values were significantly different for each group except at baseline in groups B and C.

Table 1 gives the maternal characteristics for the

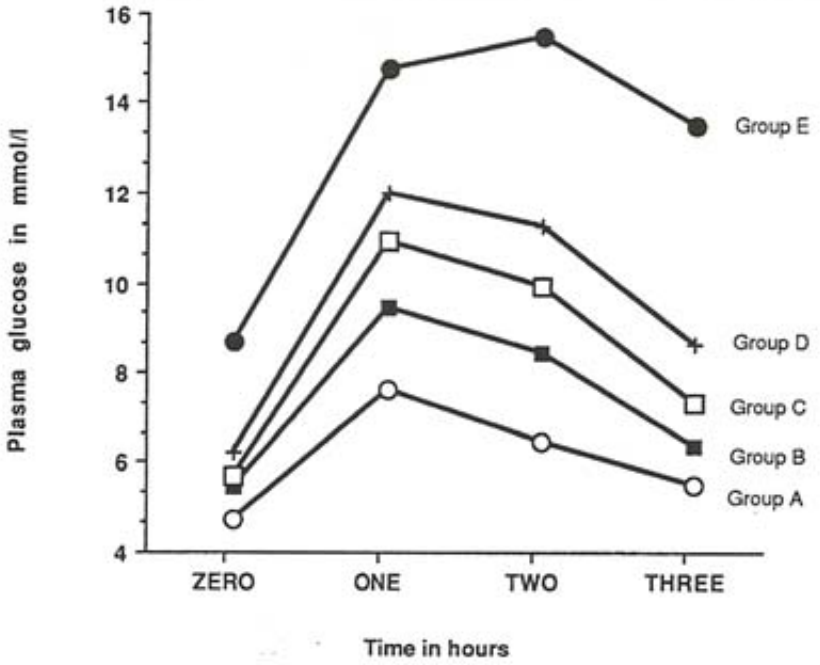

Figure 1. The mean plasma glucose level at each GTT sample time is significantly different for each group, except at zero time samples between group B-C

studied groups. ANOVA showed significant relationships between each of maternal age, parity, and weight, with increasing numbers of abnormal glucose readings in the GTTs ($p < 0.0001$, <0.0001, and <0.01, respectively). Between group analysis for statistical difference did not identify a specific category that was associated with a significant change in maternal characteristics. However patients in group 'A' were significantly younger than in the other four groups. They were also lighter than patients in group B and E and had lower parity than patients in group B and C. The distribution of risk factors between the four groups was not significantly different.

Table 2 gives the outcome measures in each group. The incidences of Caesarean section (CS) deliveries, babies weighting >4 kg, and macrosomia were significantly associated with the increase in the number of abnormal glucose readings in the GTTs. Perinatal mortality (PNM) and Apgar score at 1 and 5 minutes did not show an association. However between-group analysis showed that only patients in group A had a statistically lower incidence of CS deliveries and macrosomia than patients in each of the other four groups, in whom the incidences of those variables were not significantly different from each other.

Results of Data Analysis in the Non-GDM, GDM, and GIH Groups

The mean glucose values of the GTT results for the three groups are illustrated in Figure 2. With the exception of glucose measurements at baseline (fasting), the mean glucose values for patients in the non-GDM were significantly less than those in the other two groups. At 1, 2, and 3 h the mean blood glucose values of patients in the GIH and GDM groups were not significantly different from each other.

Table 3 gives the maternal characteristics of the groups. ANOVA showed significant positive associations between

Table 1. Maternal characteristics in the five groups of patients

	Group A ($n = 439$)	Group B ($n = 91$)	Group C ($n = 64$)	Group D ($n = 42$)	Group E ($n = 24$)
Age[a]	28.1 ± 5.7	30.6 ± 5.5[b]	30.8 ± 5.6[b]	31.3 ± 5.3[b]	32.3 ± 5.9[b]
Weight (kg)[a]	69.4 ± 14.6	75.5 ± 13.6[b]	73.9 ± 11.8	75.1 ± 15.5	82.8 ± 17.5[b]
Parity[a]	4.7 ± 2.9	5.9 ± 3.5[b]	5.9 ± 3.6[b]	5.6 ± 3.3	5.9 ± 3.2
Risk factors (%)	72.4	84.6	87.5	71.4	75.0

[a]Statistically significant association with increasing number of abnormal glucose measurements on the GTT.
[b]Statistically significant difference from group A.
Data are given as means (SD) and percentage as appropriate.

Table 2. The outcome measures in the five groups

	Group A ($n = 439$)	Group B ($n = 91$)	Group C ($n = 64$)	Group D ($n = 42$)	Group E ($n = 24$)
Birth wt (g)[a]	3277 ± 590	3342 ± 515	3425 ± 583	3430 ± 551	3616 ± 626
Delivery wk	39.1 ± 2.8	39.1 ± 2.4	39.0 ± 2.1	39.1 ± 1.4	38.7 ± 3.1
Babies > 4 kg (%)[a]	9.1	6.6	10.9	14.3[b]	20.8[b]
Macrosomia (%)[a]	13.7	19.8[b]	17.2	28.6[b]	33.3[b]
CS (%)	14.4	26.4[b]	20.3[b]	19.0[b]	29.2[b]
Elective	5.8	8.3	5.7	11.4	20.0
Emergency	8.1	18.1	14.6	7.6	9.2
AS < 5 at 1 min	4.3	4.3	3.1	4.7	0
AS < 7 at 5 min	1.4	1.1	1.6	0	0
PNM (%)	3.2	2.2	3.1	0	4.2

[a]Statistically significant association with increasing number of abnormal glucose values on the GTT.
[b]Statistically significant difference from group A.
Data are given as mean (SD) and percentage as appropriate.
PNM, perinatal mortality; AS, Apgar score.

each of maternal age, parity and weight, with increasing level of glucose intolerance. Between-group analysis showed that patients in the GDM group were significantly heavier than those in the other two groups, and significantly older than patients in the normal, but not than those in the GIH, group. However there was no significant difference in parity between the three groups.

Table 4 gives the studied outcome measures. There was no difference in PNM rate, or the incidence of babies born with A.S. < 5 and 7 at 1 and 5 min, respectively. In the non-GDM group there were 16 perinatal deaths; 11 of them were associated with complications of extreme prematurity (gestational age range 22–26 weeks), 2 developed respiratory distress syndrome, 1 neonatal death was due to bilateral fetal polycystic kidneys, and the remaining 4 were attributed to perinatal asphyxia, but no postmortem examination was performed. In the GDM group, 2 of the 3 perinatal mortalities were due to complications of prematurity (26 and 28 weeks), and the third was unexplained intrauterine fetal death at term of a 4040 g baby boy. ANOVA showed a positive association between each of: absolute

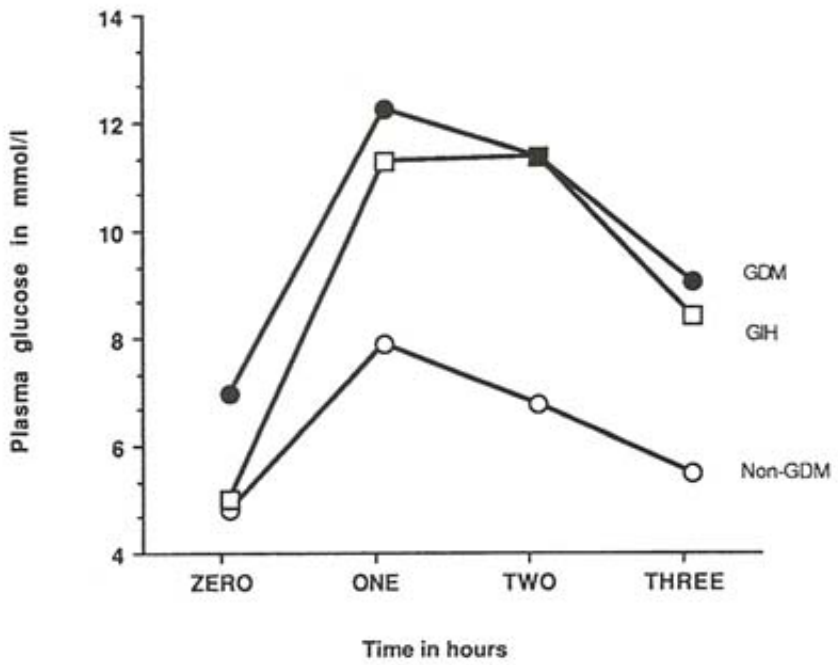

Figure 2. The mean plasma glucose levels of the non-GDM group—except at zero time—are significantly less than in the GDM and GDM groups

Table 3. Maternal characteristics

	Non-GDM ($n = 530$)	GIH ($n = 39$)	GDM ($n = 91$)
Age[a]	28.5 ± 5.7	30.3 ± 5.1[b]	31.6 ± 5.7[b]
Weight (kg)[a]	70.5 ± 14.6	70.1 ± 11.7	78.5 ± 14.9[b]
Parity[a]	4.9 ± 3.0	6.0 ± 3.7	5.7 ± 3.3
Risk factors (%)	74.5	84.5	78

[a]Statistically significant association with GTT results.
[b]Statistically significant difference from non-GDM.
Data are given as means (SD) and percentage as appropriate.

birth weight, the incidences of CS deliveries, babies weighting >4 kg, and macrosomia with increasing degree of glucose intolerance. However between group analysis showed that patients in the GDM group had a significantly higher incidence of CS deliveries (whether elective, often due to previous CS, or emergency), babies >4 kg, and macrosomia compared to patients in each of the GIH and normal group, in whom the incidences of such variables were not significantly different from each other.

The Spearman correlation test was used to study the relationship between each of the maternal variables (age, parity, and weight), fasting, and 2 h GTT blood glucose values on one hand, with fetal weight at birth and birth weight percentile on the other. In the three groups there was a positive correlation between maternal weight and each of fetal weight at birth and the birth weight percentile ($p < 0.04 - r = 0.22$ (for non-GDM), $p < 0.02 - r = 0.36$ (for GIH), $p < 0.0001 - r = 0.37$ (for GDM) groups, respectively). No such correlation was found in case of either maternal age or parity. Similarly, there was no correlation between fetal birth weight and either fasting or 2 h blood glucose values except among patients in the GDM group ($p < 0.027 - r = 0.096$, and p 0.034, $r = 0.092$, respectively).

Discussion

The prevalence of GDM is variably reported ranging from <1 % to as high as 18 %. In the present study the incidence of GDM was nearly 20 %.[6] However, it must be emphasized that this figure is drawn from a hospital rather than a general population, hence it is not the true incidence of the diseases among our local population. Nevertheless, it reflects the importance of GDM as a clinical disorder and is compatible with local epidemiological data which have shown an average prevalence rate of glucose intolerance among Saudi non-pregnant females of 5.9 %.[11] It is posible that many of those women diagnosed as having GDM are patients with pre-existing but asymptomatic non-insulin-dependent diabetes who were diagnosed for the first time during pregnancy. However the results of the present study highlight several important facts.

Firstly, the incidence of risk factors for GDM are very widespread among our local population, a finding that would probably be encountered in similar developing societies that are exposed to rapid modernization. In this respect maternal risk factors for glucose intolerance which are also risk factors for fetal morbidity, such as advanced age, obesity, and high parity are also common findings among our local obstetric population. Furthermore, a history of obstetric complications such as neonatal death, stillbirths, abortions, and congenital malformations are not uncommon findings, and are more

Table 4. The outcome measures in the three groups

	Non-GDM ($n = 530$)	GIH ($n = 39$)	GDM ($n = 91$)
Birth wt (g)[a]	3288 ± 578	3308 ± 467	3527 ± 614
Delivery wk	39.1 ± 2.8	39.0 ± 1.6	38.9 ± 2.3
Babies > 4 kg (%)[a]	9.1	7.7	17.6[b]
Macrosomia (%)[a]	14.7	15.4	27.5[b]
CS (%)[a]	14.3	12.8	25.3[b]
Elective	6.2	5.0	10.0
Emergency	8.1	7.8	15.3
AS < 5 at 1 min	2.4	0	4.4
AS < 7 at 5 min	1.4	0	1.1
PNM (%)	16 (3)	0	3 (3.3)

[a]Statistically significant association with GTT results.
[b]Statistically significant difference from Non-GDM and GIH groups.Data are given as means (SD) and percentage as appropriate.
PNM, perinatal mortality; AS, Apgar score. See text for details of in-between groups analysis.

usually due to previous lack of antenatal care rather than glucose intolerance. Therefore it is important that all the pregnant population are tested for pathological hyperglycaemia, since the prevalence of the so-called clinical or historical risk factors for development of GDM are so high that they cannot be relied upon as screening markers to identify patients at particular risk of developing GDM.

Secondly, with such background of a high-risk population it is not unexpected that a large number of patients will have positive GTT results. However in order to optimize medical and clinical care the criteria used for the diagnosis of GDM should be able to identify true cases of pathological hyperglycaemia, i.e. hyperglycaemia that is associated with significant increase in fetal and maternal morbidity. This cannot be done through changing the test cut-off points, which will only be a trade-off between its sensitivity and specificity without improving its resolution, i.e. ability to identify true negative from true positive. A better approach would be to correlate the test results with the outcome measure of interest.[12]

Our data showed a positive association between increasing degree of glucose intolerance with not only fetal/maternal morbidity but also with some other independent maternal confounding risk variables, namely, maternal age, weight, and parity. However as expected it was difficult to identify a threshold level of glucose intolerance that could be considered as a marker for pathological hyperglycaemia. Patients with even one abnormal glucose value (group B)—who by the O'Sullivan criteria are considered normal—could not statistically be identified from those in other groups with more than one abnormal values (groups C–E). Failure to define such a critical threshold of glucose intolerance has previously been observed in similar studies.[13] This however has led to GDM being considered as an ill-defined entity since true cases of pathological hyperglycaemia are being diluted with mild and probably insignificant cases of glucose intolerance that have very little, if any, relation to perinatal morbidity.[14]

Several reports have previously shown that GDM patients with normal fasting blood glucose have very low and non-specific increase in fetal morbidity.[15,16] The significance of FBG has also been previously emphasized in the recommendation of the first international workshop on GDM.[17] The results of the present study provide further evidence to the significance of FBG. Fetal/maternal morbidity did not show significant increase until FBG $\geq$ 5.8 mmol l^{-1}, despite the fact that the post glucose load plasma glucose levels in the GDM and GIH groups were not significantly different.

Taken together, the present data emphasize the importance of fasting blood glucose measurements in the interpretation of GTTs results. This could partly be explained by the fact that on undertaking oral GTTs, the fasting glucose values may exhibit more consistency than measurements performed following the intake of a glucose load. In this respect it has been suggested that the nature, concentration, and rate of consumption of the glucose drink are important variables that affect the reproducibility of GTT.[18–20] Furthermore, a recent study on glucose intolerance in pregnancy has shown a significant racial and ethnic variation in plasma glucose values following the administration of glucose load that was not present in the fasting state.[21] A further significant finding in this study that highlights the confounding role of obesity in relation to fetal macrosomia as an outcome measure is the relation of maternal weight to fetal birth weight and the incidence of macrosomia. It is possible that in severe forms of glucose intolerance, as in the GDM group, maternal obesity may have an additive effect to that of hyperglycemia. While in the non-diabetic and mild cases of glucose intolerance, as in our GIH group, maternal obesity may have a primary influence on fetal birth weight. Among the local population the role of obesity should not be ignored, since it is a widespread disorder that affects more than 60 % of the female population.[22]

The results of the present study suggest that while a full GTT—at least for the time being—should continue to be performed as the diagnostic tool for glucose intolerance in pregnancy, fetal morbidity does not seem to be significantly increased unless the FBG $\geq$ 5.8 mmol l^{-1}. A similar finding has been observed in a previous study from Kuwait, in which the 75 g glucose load was used for the diagnosis of glucose intolerance.[23] In this study the perinatal mortality, but not fetal macrosomia, among women with glucose intolerance in pregnancy showed significant increase only if FBG was $>$ 5.8 mmol l^{-1}. This suggests that, regardless of the nature of the test being used, a fasting hyperglycaemia ($\geq$5.8 mmol l^{-1}) is an important marker for increased fetal/maternal morbidity.

In the present study more than 19 % of patients were considered as having GDM when the O'Sullivan criteria were applied. If the FBG value were taken in consideration, 13.8 % would be considered GDM and 5.9 % as GIH. The latter subgroup of population (GIH patients) had very little if any increase in perinatal morbidity over non-diabetic women. Although a beneficial effect of therapy on the GIH group cannot be ruled out, all patients who were enrolled in this study were treated with the same protocol during pregnancy. It is thus unlikely that medical decisions alone could have resulted in the significant difference in perinatal outcome that was observed between the GDM and the GIH groups. We acknowledge that in an ideal situation, the results of the GTTs tests should have been concealed from the physician. However for obvious ethical reasons this would be difficult to do. However further studies are needed especially in two main areas: firstly, to identify who, and how many, patients with an initial diagnosis of GIH (FBG $<$ 5.8 mmol l^{-1}) would develop fasting hyperglycemia and, secondly, to screen for possible subtle effect(s) of GIH on the fetus.

In conclusion it is suggested that, in a population with

a high prevalence of obstetric risk factors, the ability of the 100 g GTT diagnostic criteria to define GDM needs to be refined. Patients who have a FBG < 5.8 mmol l^{-1} (GIH), may not be considered as cases of true 'pathological hyperglycaemia'. This approach will allow optimum utilization of clinical resources, and results in a better definition of GDM as a disease entity.

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