

3/8/96

Comment from Wellington Region core group re consensus statement  
Gestational Diabetes:

1: Take out the word close, in par.2 ...require medical  
supervision

2: Guidelines:

would read better as ... midwives have a role to alert women  
of any factors...etc.

Looks good otherwise.

Cheers,

Sandra Sinclair.



## NZCOM CONSENSUS STATEMENT

### *Gestational Diabetes*

# DRAFT

The New Zealand College of Midwives (Inc) believes there is no data currently available which supports the practice of routine screening of all pregnant women for gestational diabetes.

Women with pre-existing diabetes mellitus require close medical supervision throughout pregnancy.

#### Guidelines:

Midwives have an essential role in alerting women to any factors in their history or health status that would suggest screening for diabetes mellitus when in a non-pregnant state.

Midwives also have a responsibility to inform women that there is considerable debate surrounding:

1. the efficiency of the screening programme for Gestational Diabetes.
2. the nature of the condition itself.
3. the benefits and risks of any treatment.
4. ethical issues relating to the diagnosis of pre-diabetic state, eg, insurance and employment opportunities.

#### References:

Title: Effective Care in Pregnancy and Childbirth, Chapter 11  
Author: Enkin, M. Keirse, Marc JNC. Chalmers, I.

Title: Gestational Diabetes  
Author: Goer, Henai.  
Source: International Journal of Childbirth Educators. Vol.15, No.1, November, 1991. Pg 20-30.

Title: Screening for Gestational Diabetes Mellitus: A Critical View  
Author: Stephenson, Michael J.  
Source: The Journal of Family Practice, Vol.37, No.3, 1993.

Title: Correspondence: 1) Parents Cenres to Health Waikato 2) Reply from Health Waikato 3) To Midwives from Health Waikato  
Author: 1) Cole, Sharron 2) Scott, David. McPherson, Ian. 3) Dunn P.

Title: Implications of gestational diabetes for the health of the mother  
Author: Anne Dornhorst  
Source: British Journal of Obstetrics and Gynaecology. Vo. 101. Pp. 286-290. April 1994.

Title: A Guide to Healthy Pregnancy & Childbirth  
Author: Auckland Home Birth Association  
Source: Auckland Home Birth Association, P O Box 7093, Wellesl y, Auckland

Purpose of New Zealand College of Midwives Consensus Statements are to provide women, midwives and the maternity services with the profession's position on any given situation. The guidelines are designed to educate and support best practice.

All position statements are regularly reviewed and updated in line with evidence-based practice.



18 JUN 1996 1906

## **Combined Meeting to discuss Screening for Gestational Diabetes**

6 June 1996

School of Medicine, Auckland University.

This meeting was convened by the New Zealand Society for the Study of Diabetes (NZSSD) and various groups were represented including NZCOMI - Emma Wolfe and myself; ParentsCentre - Sharon Cole; NZSSD - Dr Tim Cundy and Dr David Simmons and one other physician; NZCOG - two reps whose names I didn't catch; one O&G representing the Chinese community; a number of representatives of the Pacific Island community, both midwives and community workers and three Maori women representing two different groups. They had invited a representative from the NZCGP but no-one came.

The meeting was chaired by Dr Rick Cutfield, President, NZSSD.

Dr Tim Cundy presented a historical and current overview of Gestational Diabetes - paper attached. Dr David Simmons presented the South Auckland view from the diabetes pregnancy clinic at Middlemore hospital.

The purpose of the meeting was to come to some consensus about screening for gestational diabetes in pregnancy.

It was clear from the information presented to the meeting that there is a worldwide increase in diabetes across all nations and cultures and there are some racial groups which are particularly vulnerable to the onset of the condition. This includes Maori, Pacific Islanders, Indian, Chinese and other Asian races. There was obvious concern from those involved in the burgeoning 'Diabetes Industry' for some way to pick up people who had diabetes or had a high chance of developing diabetes in the future.

There was support from the doctors present and from the Maori and Pacific Island groups for universal screening of pregnant women. This apparently does not happen with some practitioners although our view was that it was highly prevalent in most places. There seemed to be a strong desire to have the midwives agree to universal screening of all pregnant women.

Emma and I both presented the view that screening for gestational diabetes was, in fact, screening for diabetes, either actual or probable in the future, which presented in pregnancy. This involved targeting a particular group - i.e. pregnant women. We stated the 'party' line that midwives work in partnership with women and while we were in a position to give information to women about their risk factors for developing diabetes, it was the women themselves that would make the decision as to whether they needed to be tested or not and when they would be tested - during pregnancy or after.

This had the effect of altering the idea for 'consensus' to the concept of producing guidelines for practitioners in recommending screening based on current research as well as producing an information pamphlet that could be given to women to help them decide to be screened or not and when.

There are a number of recommendations at the end of Tim Cundy's paper which the NZSSD would like us to consult our membership about with feedback to them by the end of September. They would particularly appreciate more "user-friendly" language. They will also send out a draft information pamphlet for us to comment on.

I would be happy to collate any comments from the Regions and feed back to NZSSD, perhaps having a brief moment to ratify our feedback at the National Committee meeting at Conference. Comments would need to be to me by the end of July.

Bronwen Pelvin

As far as the College's Consensus Statement goes, I'm not sure that we need to make any alterations at this point - what do you think ??

18 JUN 1996

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## Gestational Diabetes - universal screening or a "high risk" approach? A discussion paper

### Insulin dependent diabetes and pregnancy

Until recently the prospects of a woman with insulin-dependent diabetes delivering a healthy baby were bleak; the perinatal mortality rate until the early 1960s remained as high as 25-50%. In the mid 1960s the institution of regimens for strict glycaemic control (which in those days meant prolonged hospitalisation) dramatically reduced perinatal mortality rate in insulin dependent diabetes (IDDM). Such regimens are now applied universally. Technological advances, particularly self blood glucose monitoring, have meant that strict glycaemic control can be achieved as an outpatient by most women. Although not subjected to formal controlled trials, the evidence that improved glycaemic control in middle and late pregnancy can improve pregnancy outcome is so strong that it is difficult to imagine there will be any significant retreat from this position (1). However, the improved outcome may not be all attributable to improved glycaemic control. Obstetric surveillance and willingness to intervene in pregnancy has also changed over the years. Thus, induction of labour and caesarean section are both very frequent in women with IDDM, and again it is difficult to imagine that these practises are going to change substantially. There have also been significant advances in neonatal care. The most difficult challenges remaining are the high incidence of congenital malformation in infants born to women with IDDM, and the management of pregnancies in which the mother has advanced diabetic complications (1).

### Gestational diabetes : historical aspects

The clear view we have of IDDM and pregnancy contrasts markedly with the controversies surrounding gestational diabetes (GD). The concept of GD arose from observations that women with an obstetric history of high birth weight and fetal loss seemed particularly prone to develop non-insulin dependent diabetes (NIDDM) in later life (2). Studies by the Boston group in the 1960s established criteria for the 100 g glucose tolerance test in pregnancy and confirmed that women with gestational glucose intolerance are at risk of subsequently developing NIDDM. They also suggested that perinatal mortality was higher in women with glucose intolerance (3). These studies have been widely quoted as the strongest justification for screening for gestational diabetes, but the conclusions are flawed since the studies made no attempt to adjust for important variables, such as maternal age, which are significantly associated with perinatal mortality (2).

### Gestational diabetes : controversies

Sharp divisions in opinion exist about the significance of GD. It has been described as both "a major public health problem" and "a diagnosis looking for a disease". Consequently, there exist also sharp divisions of opinion about the need for detection of GD. Such a divergence of views reflects a paucity of agreed facts. Until definitive answers to the controversies surrounding GD are available, then policies for its detection and management need to be pragmatic and suited to the population being served. In this paper some of the controversial areas in GD are discussed and a New Zealand perspective presented.

### Definition

Gestational diabetes was originally defined as glucose intolerance discovered in pregnancy but reverting to normal postpartum. The definition was subsequently expanded to include glucose

intolerance arising in *or first detected in pregnancy*. It is important to understand that irrespective of the diagnostic criteria employed, the prevalence of GD in any population is directly related to the background prevalence of glucose intolerance in that population(4). For example, the Maori and Pacific Island communities, which have a high prevalence of NIDDM and impaired glucose tolerance (IGT), also have a high prevalence of gestational diabetes.

Since IGT and NIDDM are disorders of insidious onset, there exists always, in any community, a number of people with undiagnosed IGT and NIDDM. Under the revised definition of gestational diabetes, such women identified in pregnancy, are diagnosed as having "gestational diabetes". Thus one difficulty in assessing the significance and risks of gestational diabetes has been that it can encompass a very broad range of abnormalities; from undiagnosed but overt NIDDM through to trivial elevations in blood glucose, present only in late pregnancy. It would seem unlikely that the risk to the pregnancy would be equal across this whole range of glucose intolerance.

Because "gestational diabetes" covers this wide spectrum, retesting and reclassification postpartum is necessary, in order to clarify the true glucose tolerance status. Reclassification, based on the results of a standard 75g glucose tolerance test is made into the categories of diabetes (usually NIDDM), IGT or normal. As indicated previously, the proportion of women with abnormal glucose tolerance antedating their pregnancy depends very much on the population being studied. In some communities the proportions are very high. For example, at National Women's Hospital in Auckland, 31% of women with GD when retested 6 to 8 weeks postpartum had either IGT or NIDDM in 1994-5. In the majority of cases these abnormalities must have antedated the pregnancy. Furthermore, almost as many women with NIDDM had it first identified in pregnancy as "gestational diabetes" as were known to have NIDDM before becoming pregnant. This high rate of newly diagnosed diabetes at National Women's Hospital is because the hospital serves large Maori, Pacific Island, Indian and Chinese communities. These are all groups in which NIDDM is very prevalent at younger ages.

#### Diagnosis of Gestational Diabetes

The diagnosis of gestational diabetes is based on the result of a glucose tolerance test. In many countries, including Australia, New Zealand and the USA, a screening test is employed at 26-28 weeks gestation, at which a blood sugar is measured one hour after the ingestion of 50 g glucose. This test is done non-fasting, and if the blood glucose is above a particular threshold (currently in New Zealand this is  $\geq 7.8$  mmol/L, although other values have been advocated) then the woman proceeds to a formal fasting 75 g glucose tolerance test. There have, however, been suggestions that the non-fasting screening test be abandoned and the fasting glucose tolerance test be the only test performed (5).

Glucose tolerance differs in pregnancy from the non-pregnant state and a variety of forms of the pregnancy glucose tolerance test have been used. The Boston group devised a 3-hour 100 g test and a modification of this was used widely in New Zealand until 1992. In that year the NZSSD followed Australian and European suggestions in recommending a 2 hour 75 g test. A variety of diagnostic criteria for GD have been proposed (Table) . Langer has argued that disputes over the different criteria are a "tempest in a teapot", since they are all very similar (6). However, the more stringent the criteria, the greater are the numbers of women diagnosed as having GD. For



example, if New Zealand was to change its diagnostic threshold for the 2 hour blood glucose value from 9.0 mmol/L, as currently recommended by the NZSSD, to 8.0 as recommended by the Australian Diabetes in Pregnancy Society, then the proportion of women diagnosed as having GD could increase by a third (7). The women included in the diagnosis when stringent criteria are used, but excluded by more liberal criteria, are likely to be those with lesser degrees of hyperglycaemia. Since there is considerable controversy about the benefit of diagnosing such women, information on the level of glycaemia that constitutes a significant fetal risk is very much needed.

International Diagnostic Criteria for  
Gestational Diabetes

	O'Sullivan USA 1964	NDDG USA 1979	Carpenter USA 1982	Sacks USA 1989	EASD Europe 1989	Melbourne Aus 1986	ADIPS Aus 1991	NZSSD NZ 1992
<u>Glucose Load</u>	100	100	100	100	75	50	75	75
<u>Fasting</u>	5.0	5.9	5.3	5.3	5.4	-	5.5	5.5
<u>1 hour</u>	9.2	10.6	10.0	9.6	10.5	9.0	-	-
<u>2 hour</u>	8.1	9.2	8.7	8.4	9.1	7.0	8.0	9.0
<u>3 hour</u>	7.0	8.1	7.8	7.3	8.3	-	-	-

It is important to recognise that currently used criteria for glucose screening tests and glucose tolerance tests in pregnancy have not been determined by any assessment of fetal risk. The approach to defining these thresholds has rested on the distribution of glucose values in the normal pregnancy, the numbers of women who can be accommodated within the available obstetric-diabetic services and on the predictive value of GD for the later development of NIDDM.

#### Maternal risks of gestational diabetes

Gestational diabetes poses no significant short term risk to maternal mortality. Its impact on maternal morbidity is debatable. Some studies have shown that good control of GD, by reducing the risk of macrosomia, can reduce the requirement for emergency cesarean section for cephalopelvic disproportion. However, the cesarean section rate is influenced by the knowledge that diabetes is present, so that detection of GD could increase the rate of cesarean section. Arguably, the main short term problems for the mother relate to the inconvenience and expense of attending clinics and, in some cases, the hazards of surveillance and interventions which may be unnecessary. However, there are no studies reporting the incidence of episiotomy or tears resulting from the delivery of macrosomic babies.

Although a recent study has shown that a subsequent pregnancy *after* the first diagnosis of GD may be an independent risk for developing NIDDM (8), the relationship between parity and the risk of NIDDM is weak. In the main, GD is simply a marker for future NIDDM rather than a cause of it. The important risk factors for GD (age, ethnicity and obesity) are identical to the main risk factors for NIDDM and, in order to be diagnosed as having GD, a woman in effect, needs to 'fail' two glucose tolerances tests - the 50 g screening test and the formal glucose tolerance test. Given the lack of reproducibility of glucose tolerance test results (9) it is to be expected that someone giving high results on two tests has a strong probability of truly being in the upper ranges of glucose tolerance, and therefore at significant risk of developing NIDDM. If a third test is done, postpartum, then the predictive value is even higher (10). Of course, in communities with a high prevalence of diabetes, the postpartum test will often demonstrate that NIDDM is already present.

#### Fetal risks of gestational diabetes

Early studies from the Boston group demonstrated that women with gestational diabetes had greater than expected fetal losses (3) but as indicated earlier, this finding cannot necessarily be attributed to glucose intolerance. Women with GD tend to be older, multiparous, overweight, and from economically and educationally disadvantaged backgrounds - all factors which may influence rates of fetal loss. This is particularly true of maternal age, which is strongly linked to increased perinatal mortality (11). In this context, it is interesting to note that all cause perinatal mortality in women with NIDDM attending the Diabetes Pregnancy Clinic at National Women's Hospital, over the past ten years has been substantially higher than that in women with IDDM, despite the fact that the women with NIDDM achieve as good, or better, glycaemic control in the last trimester. Perinatal death is, fortunately, an infrequent event and it is likely to be impossible to determine the exact contribution of glucose intolerance to its occurrence. Studies to examine this outcome would need to be extremely large and there would be ethical difficulties in leaving some subjects untreated, as there are other important fetal morbidities which may result from poorly controlled GD. Neonatal polycythaemia, jaundice and hypoglycaemia are amongst the most important of these. These complications commonly necessitate admissions to neonatal special care units. A number of studies have suggested that such complications are significantly more prevalent at the more severe end of the GD spectrum (12-14). That is, there is a relationship between the degree of hyperglycaemia and the occurrence of these complications.

There is also a strong association between GD and accelerated fetal growth or macrosomia. Macrosomia is associated with difficult delivery (particularly shoulder dystocia) and a higher rate of obstetric intervention. Perhaps because it is the outcome most readily modifiably by insulin therapy, some authorities believe that the rate of macrosomia should be the main measure of outcome in gestational diabetes (6). This view is arguable since macrosomia is a measure, not a disorder and there is moreover, no agreed definition. Birth weight is the most commonly employed indicator of macrosomia, but this has its difficulties. The association between birth weight and ambient blood glucose levels is a continuous one and there is no threshold value for blood sugar which will result in macrosomia (6). Birth weight is also significantly affected by variables other than blood glucose; in particular, it is reduced by maternal cigarette smoking and increased by maternal obesity (15).

Ultrasonography can be used to detect macrosomia developing in utero. Differential growth of the fetal abdominal circumference is the most frequently recognised manifestation, but this is not unique to GD and is present in the fetuses of obese but non-diabetic mothers (16). Nonetheless, accelerated growth of the abdominal circumference is often used as an aid to the decision whether to begin insulin therapy in women with gestational diabetes.

Recent studies on the Pima Indians of Arizona have highlighted the potential importance of maternal diabetes as a risk factor for the later development of diabetes in the offspring. The children of mothers who had gestational diabetes developed obesity and NIDDM earlier in life than the children of mothers who subsequently developed diabetes, but did not actually have GD during their pregnancy (17,18). It has been argued that this represents a significant adverse effect of a diabetic intrauterine environment on postnatal life and there is animal data to support this hypothesis. Although this is an interesting and provocative idea, and certainly one which needs more research, it is important to remember that intervention in the GD pregnancy has not yet been shown to alter this outcome, and that there are other plausible explanations for the phenomenon.

#### Is screening for gestational diabetes justified?

Although the major risk factors for GD are well known (maternal age, obesity and ethnicity) numerous studies have demonstrated, unsurprisingly, that more women with abnormal glucose tolerance can be identified by screening than by risk factor analysis. It has, therefore, been argued that all pregnancies should be screened since the women affected may benefit from intervention (19). An opposing view is that the extra cost, inconvenience and 'medicalisation' of the pregnancy is unnecessary in many cases at the milder end of the GD spectrum and that the benefit of diagnosing mild cases is debatable. The Cochrane Collaboration reviews of treatment trials in GD show convincing reductions only in rates of macrosomia. Biochemical parameters such as hypoglycaemia, polycythemia and jaundice in the newborn also tended to improve but clinically meaningful sequelae (eg prolonged stay in hospital) were not reported (20).

If the concept is accepted that the fetal risks in GD are proportional to the degree of hyperglycaemia (and experience with IDDM would tend to support this) then it is particularly important to identify those women with the most severe hyperglycaemia. This group is reflected in those women who are shown to have diabetes, hitherto undiagnosed, on postpartum testing. One hundred fifty two such women were diagnosed in the ten years from mid 1985 - mid 1995, at National Women's Hospital, in Auckland. The ethnic breakdown of this group is instructive. Only 10 (6.5%) were of European origin, which represents only 0.023% of the women of European origin delivering at the hospital in this period. Of the ten patients, 7 (average age 31) were obese (BMI >30 kg/m<sup>2</sup>) and had NIDDM postpartum. The 3 other women proved to be in the prodromal phase of IDDM. It follows that severe GD is very uncommon amongst younger, non-obese women of European origin and the value of screening these women must be doubtful. The situation is different for other ethnic groups. With exposure to a western lifestyle virtually all other races : Maori, Pacific Island, Indian and, increasingly, Chinese, are very prone to developing glucose intolerance. For example, the prevalence of NIDDM amongst 40 year olds of Maori origin is four times greater than that of women of European origin (21). The value of screening non-European women for GD is higher, given the increased (and rising) background level of glucose intolerance in these communities. Reliance on risk factors alone to detect severe GD is unsatisfactory. For example, more than 40% of the women with newly confirmed NIDDM following pregnancy at National Women's Hospital in 1985-95 were detected only by the screening test at 26-28 weeks. These considerations argue in favour of a more selective

screening policy, which reflects the type of community being served rather than universal screening. Such a policy would be in accord with that advocated by the American College of Obstetrics and Gynaecology and recent editorials in both *The Lancet* and the *New England Journal of Medicine* (22-24).

### Conclusions

1. The prevalence and severity of GD in the community reflects the underlying prevalence of glucose intolerance. As currently defined, GD encompasses a range of abnormalities from the possibly trivial through to overt but undiagnosed DM.
2. GD is associated with other maternal characteristics (obesity, age and economic disadvantage) which compound the risks to the fetus. For some adverse outcomes, the precise contribution of diabetes itself is uncertain.
3. More stringent criteria for the diagnosis of GD capture a larger proportion of pregnant women at the lower extremes of glucose intolerance. However, there is, at present, insufficient evidence to justify the use of more stringent diagnostic criteria.
4. Clinical trials of intervention vs non-intervention in women at the milder end of the GD spectrum are required to determine the risks, benefits and costs of intervention.
5. Macrosomia is a common complication of GD. Despite its non-specificity, it does appear to be modifiable by dietary restriction and insulin therapy.
6. The low incidence of undiagnosed NIDDM in young, non-obese, European women means that screening for GD in this group may be unnecessary.
7. The prevalence of diabetes in the community is changing therefore screening policies need to be reviewed periodically as new data emerge.

### Recommendations

1. The NZSSD should continue to advise screening of all non-European women for gestational diabetes. European women over the age of 30 and/or with a prepregnancy BMI of over 30 kg/m<sup>2</sup> should also be screened. Women outside these groups should be offered screening if they have risk factors for diabetes. These risk factors include a family history of diabetes, previous GD, glycosuria, macrosomia or previous stillbirth.
2. The 50g glucose challenge test should continue to be used as the initial screening test.
3. The 75g fasting glucose tolerance test should continue to be used as the diagnostic test. The diagnostic criteria should remain unchanged from current NZSSD recommendations.
4. The NZSSD should liaise with other interested professional and lay groups to determine the acceptability of these recommendations and, if possible, to obtain consensus.
5. The NZSSD policies should be reviewed periodically as new information becomes available and the prevalence of glucose intolerance changes

### References:

1. Garner P. Type 1 diabetes mellitus and pregnancy. *Lancet* 1995 346 157-61.
2. Ales KL, Santini DL. Should all pregnant women be screened for gestational glucose intolerance? *Lancet* 1989 i 1187-91.
3. O'Sullivan JB, Gellis SS, Dondrow RV, Tenney BO. The potential diabetic and her treatment during pregnancy. *Obstet Gynecol* 1966 27 683-9.
4. Harris MI. Gestational diabetes may represent discovery of pre-existing glucose intolerance. *Diabetes Care* 1988 11 402-11.
5. Pettit DJ *et al*. Comparison of WHO and NDDG procedures to detect abnormalities of glucose tolerance during pregnancy. *Diabetes Care* 1994 17 1264-8.
6. Langer O. Gestational diabetes: a contemporary management approach. *The Endocrinologist* 1995 5 180-8.
7. Sacks DA *et al* Toward universal criteria for gestational diabetes : the 75 g glucose tolerance test in pregnancy. *Am J Obstet Gynecol* 1995 172 607-14
8. Peters RK *et al* Long-term diabetogenic effect of single pregnancy in women with previous gestational diabetes mellitus. *Lancet* 1996 347 227-30.
9. Sacks DA *et al* How reliable is the fifty-gram one-hour glucose screening test? *Am J Obstet Gynecol* 1989 161 642-5.
10. Kjos SL *et al* Predicting future diabetes in Latino women with gestational diabetes: utility of early postpartum glucose tolerance testing. *Diabetes* 1995 44 586-91.
11. Fretts RC *et al* Increased maternal age and the risk of fetal death. *N Eng J Med* 1995 333 953-7.
12. Weiner CP. Effects of varying degrees of "normal" glucose metabolism on maternal and perinatal outcome. *Am J Obstet Gynecol* 1988 159 862-70.
13. Maresh M. *et al* Factors predisposing to and outcome of gestational diabetes. *Obstet Gynecol* 1989 74 342-6.
14. Lucas MJ *et al* Class A1 gestational diabetes : a meaningful diagnosis? *Obstet Gynecol* 1993 82 260-5.
15. Cundy T. *et al* Determinants of birth-weight in women with established and gestational

- diabetes. Aust NZ J Obstet Gynecol 1993 33 249-54.
16. Roberts AB *et al* Fetal liver length in diabetic pregnancy. Am J. Obstet Gynecol 1994 170 1308-12.
  17. Pettit DJ *et al* Excessive obesity in offspring of Pima Indian women with diabetes during pregnancy N Eng J Med 1983 308 242-5.
  18. Pettit D J *et al* Congenital susceptibility to NIDDM role of intrauterine environment, Diabetes 1988 37 622-8.
  19. American Diabetes Association. Position statement on gestational diabetes mellitus. Am J Obstet Gynecol 1987 156 488-9.
  20. Walkinshaw SA. Diet and insulin vs diet alone for "gestational diabetes" and Dietary regulation for "gestational diabetes". In : Enkin MW Keirse MJNC, Renfre MJ, Neilson JP (eds). Pregnancy and childbirth module: Cochrane Database of Systematic Reviews. Reviews 06649 and 06650 April 1993. Update Software, Oxford, UK.
  21. Simmons D. *et al* Frequency of diabetes in family members of probands with non-insulin dependent diabetes mellitus. J Intern Med 1995 237 315-21.
  22. Dornhurst A, Girling JC. Management of gestational diabetes mellitus. N Eng J Med 1995 333 1281-3.
  23. Kopelman P. Gestational diabetes and beyond. Lancet 1996 347 208-9.
  24. American College of Obstetricians and Gynaecologists. Management of diabetes mellitus in pregnancy. ACOG Tech Bull 1986 92 1-2.