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## Current Management of Pregnancy in the Diabetic: A Team Approach

Seth G. Kivnick, MD,\* J. David Fachnie, MD,\*\* and Chang Y. Lee, MD\*

*We reviewed details of 89 pregnancies in diabetic women who were delivered from 1980 through 1982. Data are presented on the obstetrical outcome and the level of diabetic control in patients with White classifications of B through R. Although the mean blood glucose levels of all groups failed to meet the criteria for ideal metabolic control (fasting less than 105 mg/dl, postprandial less than 120 mg/dl), the perinatal mortality rate of 4% compared favorably with reports from other centers. Fifty-six percent of our patients were delivered at 37 weeks gestation or later. The primary Caesarean section*

*rate was 37%, and the overall section rate was 50%. Reasons for high rates of preterm delivery and Caesarean section are analyzed. Management of diabetic pregnancies by a "high risk team" consisting of an obstetrician, diabetologist, dietitian, and neonatologist is described. Modern perinatal management, which strives to maintain maternal euglycemia and accurately monitor fetal well-being and maturity, can virtually eliminate the classic causes of increased perinatal morbidity and mortality in diabetic pregnancies. The challenge for the future is to decrease the incidence of congenital anomalies in the infants of diabetic mothers.*

Improvements in diagnosis and management of the diabetic pregnancy over the last 60 years have markedly reduced perinatal mortality rates. In 1922, the Joslin Clinic reported no live births to ketosis-prone diabetic women (1); but today, perinatal mortality rates in most centers are currently less than 10%. Such progress reflects an improved understanding of altered carbohydrate metabolism in pregnancy, greater insight into the effects of these alterations on the fetuses of diabetic mothers, and the development of effective methods for assessing fetal well-being.

Diabetes mellitus adversely affects the pregnant woman in several ways. Preclampsia-eclampsia is approximately four times more common in women with diabetes, with or without pre-existing vascular disease. The incidence of polyhydramnios and its associated problems is increased. Urinary tract infections are more common. During pregnancy, diabetic ketoacidosis occurs at much lower blood sugar levels than it does in nonpregnant diabetic patients. Exacerbation of pre-existing vascular disease, retinopathy, and nephropathy during pregnancy may cause permanent end organ damage (2). Since the fetus is frequently larger than average, dystocia and trauma to the birth canal may result. Fetal macrosomia and the

frequent need for preterm delivery require a Caesarean section more often than for normal pregnant women.

Infants of diabetic mothers have an increased risk for a number of antenatal and neonatal complications (3). The metabolic problems which such infants often encounter in the newborn period include hypoglycemia, hypocalcemia, hypomagnesemia, hyperbilirubinemia, polycythemia, and the respiratory distress syndrome. These problems are frequently compounded by the premature delivery necessitated by maternal or fetal compromise. The infant may be traumatized during difficult vaginal delivery. Intrauterine fetal death may occur secondary to maternal ketoacidosis or near term without apparent cause. Finally, diabetes increases the percentage of infants born with major congenital anomalies.

Improvements in the outcome of a diabetic pregnancy, both for the mother and the fetus, depend on aggressive

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perinatal management. Collaboration between obstetrician, diabetologist, dietitian, and neonatologist is essential to achieve excellent metabolic control of the pregnant diabetic woman, and close monitoring of fetal maturity and well-being, and to ensure atraumatic delivery necessary to optimize the management of these high-risk pregnancies.

### Materials and Methods

We have reviewed available records of all women with diabetes mellitus who were delivered at Henry Ford Hospital from January 1, 1980 through December 31, 1982. Each patient was categorized according to the White classification system (Table I). Because complications related to diabetes are difficult to demonstrate in gestational diabetic patients who do not require insulin, patients in class A (those controlled by diet alone) were excluded (4).

During initial evaluation by the high risk team, particular attention was given to the patient's cardiovascular and neurologic status. Electrocardiogram and 24-hour urine collection to determine total protein and creatinine clearance were performed on patients in classes C through R. Regardless of the duration of their disease, these patients were also evaluated by a retinal specialist (5), and those with retinopathy underwent regular ophthalmologic evaluation. Laser therapy was employed to arrest proliferative retinopathy. Glycosylated hemoglobin (Hb<sub>A1c</sub>) concentration was measured for each new patient.

Except for particularly compliant patients with easily controlled diabetes, all were admitted to the hospital to

complete this evaluation and to achieve optimal glycemic control. Each was counseled by a dietitian about a diabetic diet that would contain 30-35 kcal/kg ideal body weight and 100-125 gm protein. A diabetes teaching nurse taught each patient how to inject insulin, to monitor her capillary blood glucose by intermittent finger pricks and chemstrip tests, and to test urine specimens for acetone.

Insulin therapy initiated during pregnancy consists only of purified pork insulin in order to minimize the development of insulin antibodies. Almost all patients were managed with two or more insulin injections daily. In most cases an algorithm was constructed for multiple injections based on preprandial and bedtime home blood sugar determinations. Subsequent adjustments were made in insulin dosage, as well as in the quantity and distribution of food intake as indicated by acetoneuria, adequacy of weight gain, blood glucose measurements, and symptoms of hypoglycemia or hyperglycemia. Patients with sufficient interest and comprehension were taught how to adjust their insulin dosages. Metabolic goals included fasting blood glucose concentration of less than 105 mg/dl, postprandial glucose of less than 120 mg/dl, no acetoneuria, and a weight gain of at least 10-12 kg over the entire pregnancy.

The purpose of obstetrical evaluation in the first trimester is to identify other obstetrical or genetic risk factors. Routine prenatal laboratory tests were obtained and vitamins prescribed. Estimated date of delivery was established as accurately as possible by traditional obstetrical landmarks (e.g., auscultation of fetal heart tones, fundal height measurement, quickening) as well as by an ultrasound examination made between 18 and 24 weeks of gestation.

Visits to the high risk clinic and to the diabetologist were made biweekly through 28 weeks gestation and weekly thereafter. Postprandial venous blood glucose levels were determined at each visit, and Hb<sub>A1c</sub> concentration was measured in each trimester. Some patients required interval admissions to the hospital to improve glycemic control, and most were hospitalized for that purpose at least one week before delivery.

Beginning at 28 weeks, each patient counted and recorded the number of fetal movements perceived in a given hour each day. This daily recording served as an index of fetal well-being. Ultrasound study was repeated in the third trimester if fundal height deviated from the standard growth curve. Nonstress testing (NST) (Fig. 1) was done weekly after 32 weeks. In this test, the fetal heart rate is continuously monitored for accelerations asso-

TABLE I

White Classification of Diabetes in Pregnant Women

A	Abnormal postprandial glucose; diet controlled
B	Requires insulin therapy; onset over 20 years old; duration less than 10 years; no vascular lesions
C,B	Requires insulin therapy; never on insulin before pregnancy
C <sub>1</sub>	Age 10-19 years at onset
C <sub>2</sub>	Duration 10-19 years
D <sub>1</sub>	Onset at under 10 years
D <sub>2</sub>	Over 20 years' duration
D <sub>3</sub>	Benign retinopathy
D <sub>4</sub>	Calcified leg vessels
D <sub>5</sub>	Hypertension
F	Nephropathy
H	Cardiopathy
R	Proliferative retinopathy
T	Renal transplant

ciated with fetal movements felt by the mother. A "reactive" NST (Fig. 1A), consisting of three or more such accelerations within a 10-minute period, is a highly specific indicator of fetal well-being. It assures with 99% certainty that intrauterine fetal death will not occur in the week following a reactive test (6). A "non-reactive" NST (Fig. 1B) fails to assure fetal well-being and requires further evaluation in the form of a contraction stress test (CST) (Fig. 2), which involves continuous observation of fetal heart rate response to uterine contractions stimulated by intravenous oxytocin. A negative CST (accelerations associated with uterine contractions) indicates the safety of prolonging the pregnancy for one week. A positive CST (persistent late decelerations with contrac-

tions) indicates uteroplacental insufficiency and the need for carefully monitored induction of labor or prompt Caesarean section. If CST demonstrated fetal distress, stimulation was continued, and delivery was accomplished promptly. When the fetal condition precluded labor, Caesarean section was performed.

When the fetal condition was good and glycemic control satisfactory, delivery was planned for 38 weeks gestation. Amniocentesis was performed just before delivery to determine chemical indices which correlate with neonatal pulmonary maturity. These include the lecithin/sphingomyelin ratio, percentage of desaturated lecithin, phosphatidylinositol and phosphatidylglycerol. In these



Fig. 1

Nonstress Test (NST)

**A. Reactive nonstress test: Fetal heart rate accelerations associated with fetal movements indicate fetal well-being. B. Nonreactive nonstress test: Further evaluation indicated in form of contraction stress test (CST) or real-time ultrasound test.**

## Management of the Pregnant Diabetic

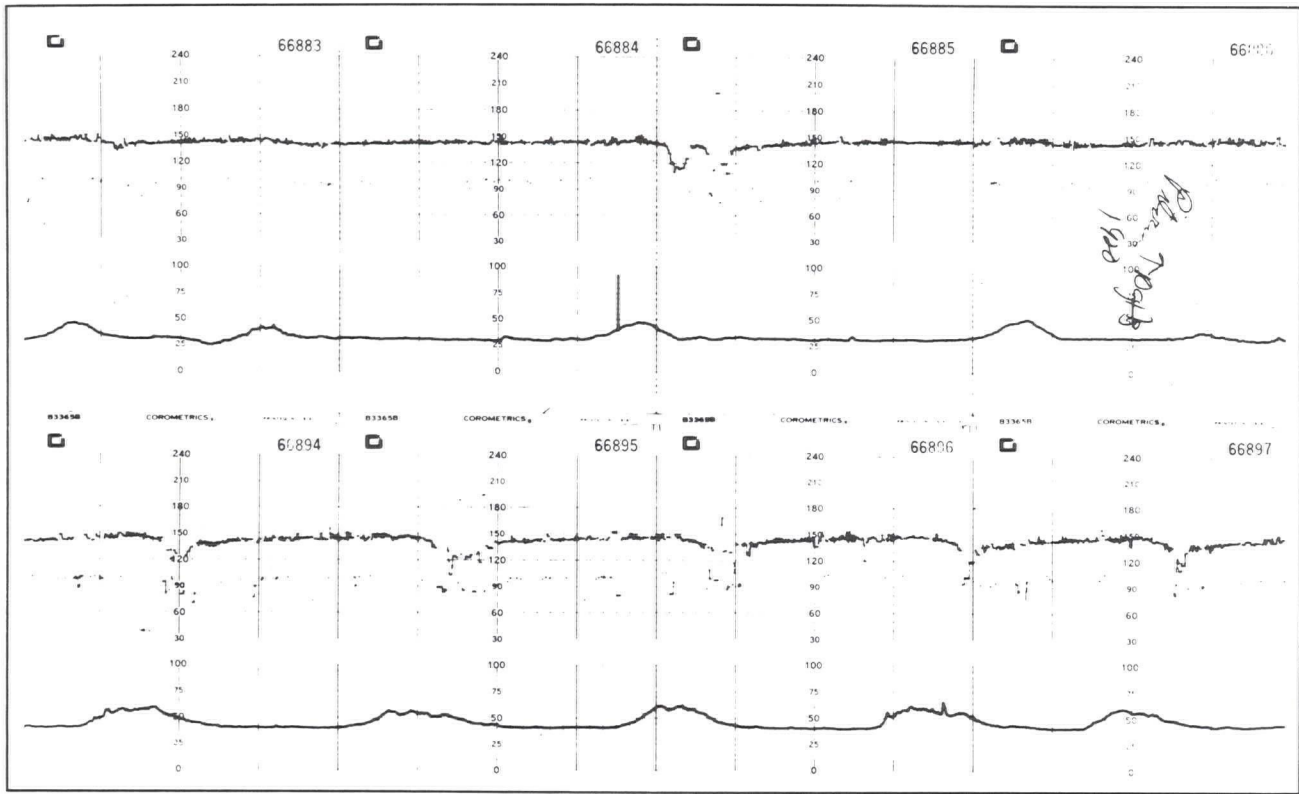


Fig. 2

Positive contraction stress test may indicate fetal compromise.

cases, the route chosen for delivery was based on obstetrical factors. When the cervix was favorable, labor was induced with administration of oxytocin, and the patient was delivered vaginally. Throughout labor, blood glucose concentration was maintained below 120 mg/dl by means of continuous intravenous infusions of 5% dextrose in normal saline and insulin, along with frequent capillary blood glucose determinations by chemstrips. Caesarean section, when chosen for other than emergency obstetrical indications, was performed at 8:00 am after half the usual dose of intermediate acting insulin had been administered. Delivery was attended by a team consisting of a neonatologist and special care newborn nursery personnel who assigned Apgar scores, performed necessary resuscitative maneuvers, and initiated surveillance for hypoglycemia and other metabolic perturbations characteristic of infants of diabetic mothers. In the immediate postpartum period, maternal blood glucose goals were relaxed. Patients usually required only one half to one third of the antepartum insulin dose.

### Results

Of 89 patients whose charts were reviewed, 46 were placed in White classes B through R (Table II). At deliv-

ery, 26 of 46 (56%) were at 37 or more weeks gestation, as compared to 90% of all obstetrical patients at our institution. Eight were at 36 weeks gestation. Of the remaining 12, indications for preterm delivery were premature rupture of membranes in two cases, intrauterine fetal death in two cases, fetal distress in one case, worsening maternal hypertension in three cases, and elective delivery of an infant with laboratory evidence of pulmonary maturity in four cases.

TABLE II

#### Obstetrical Data for 46 Diabetic Pregnant Women

Number of patients (46)	White class	Mean age (years)	Mean weeks gestation at delivery	% C-section Primary/Total
27	B	31	36.3	41/48
8	C	26	36.1	22/33
9	D	28	36.2	38/67
2	R	29	35	50/50

The primary Caesarean section rate was 37%, while the total rate including repeat Caesarean sections was 50%. Overall rate of performance of this operation at Henry Ford Hospital during the study period was 9.13% primary

and 14.2% total. The indications for primary section were failure of induction in four cases (at 32.5, 36.5, 37, and 38 weeks gestation), fetal distress in three cases, cephalopelvic disproportion in three cases, breech presentation in two cases, and one case each of hypertension, transverse lie, chorioamnionitis, and fetal prematurity.

Data on the relationship between maternal metabolic control and fetal outcome (Table III) indicate that consistent, ideal glycemic control (fasting glucose less than 105 mg/dl and postprandial levels less than 120 mg/dl) was not achieved in any White class, although mean glucose levels in the two Class R patients were within stated metabolic goals.

**TABLE III**  
**Comparison of Maternal Metabolic Control and Fetal Outcome in 46 Diabetic Pregnant Women**

White class	Mean FBS mg/dl	Mean RBS mg/dl	Mean birth weight (gms)	% over 4000 gms	% 5 min Apgar <8	IUFD*
B	115	157	3336	11	7	1
C	97	146	3335	25	13	1
D	118	160	3150	11	0	0
R	80	110	3180	0	0	0

\*Intrauterine fetal death

Twelve percent of the infants weighed more than 4000 gm at birth. Apgar scores of lower than eight at five minutes of life occurred in 5% of Class B, none of Class C, and 22% of Class D offspring.

Two perinatal deaths occurred in the 46 cases reviewed (4%). One child was born with congenital anomalies incompatible with life. Another was already dead in utero when its mother presented in ketoacidosis for initial evaluation at 30 weeks gestation.

### Discussion

The relationship between maternal glucose control and perinatal mortality in diabetic pregnancies was first demonstrated by Karlsson and Kjellmar (7). In 1972 they observed a perinatal mortality rate of 38 per 1000 if mean maternal glucose levels during the third trimester were kept below 100 mg/dl. If mean glucose levels exceeded 150 mg/dl, perinatal mortality was 236 per 1000. Since glucose diffuses freely across the placental interface, maternal hyperglycemia rapidly causes fetal hyperglycemia. Long-term hyperglycemia induces fetal islet cell hyperplasia and subsequent hyperinsulinemia. As a result, the deposition of proteins, glycogen, and fat

increases, as can be seen in the macrosomic infant of a poorly controlled diabetic mother. Such infants are likely to be traumatized at birth because of their large size. The abrupt withdrawal of maternal glucose at birth may result in life-threatening fetal hypoglycemia, since reduced insulin secretion by the neonatal pancreas occurs only gradually. Less marked hypoglycemia occurs in neonates whose mothers were well controlled.

Another major cause of perinatal mortality is intrauterine fetal death without ketoacidosis, which may also be related to poorly controlled maternal blood sugar. Shelley, et al, in a sheep model, demonstrated that fetal hyperglycemia, when accompanied by minimal hypoxia, produced fatal lactic acidosis (8). Beard and Oakley hypothesized that maternal hypoglycemia combined with fetal hyperinsulinemia caused intrauterine fetal death (9). Although much remains to be learned about the mechanisms of the diabetic environment, its deleterious effects on the fetus and newborn, as well as the correlation between good maternal glucose control and improved fetal outcome, are now well established.

The present study was limited in several ways. Our review of charts yielded insufficient data on maternal glycemic control to allow us to apply statistical tests of significance. Nor were we able to draw conclusions about the relationship between level of control and birth weight. This cohort of patients was heterogeneous with respect to the point in gestation at which each was referred to the high risk obstetrical clinic for management. The intrauterine fetal death which had occurred in the woman who presented herself initially at 30 weeks with ketoacidosis might have been prevented if tight metabolic control had been instituted earlier. The incidence of fetal death in ketoacidosis is 50% or more (10).

Home blood glucose monitoring provides great advantages over home urine glucose testing and also over periodic measurement of blood glucose at clinic visits. Patients use home blood glucose readings and algorithms constructed by the diabetologist to adjust insulin doses promptly. However, the cost of these techniques may limit their application; self-monitoring of blood glucose may cost up to \$1,000 annually. In addition to initial hospitalization or intensive outpatient education, these patients also require weekly phone contact, as the physician must be readily accessible to advise about insulin dose adjustments.

Modern techniques to evaluate fetal maturity and well-being account for much of the decrease in perinatal mortality rates seen in diabetic pregnancies over the past decade. As recently as the 1960s (11), fetal deaths not

associated with ketoacidosis were encountered in 10-30% of diabetic pregnancies. Since the incidence of such deaths increases after 36 weeks, it became common practice to deliver diabetic gravidas several weeks before term. This practice, however, often resulted in iatrogenic prematurity and subsequent neonatal deaths. More than 50% of such neonatal deaths were due to hyaline membrane disease (10), which is five to six times more common in infants of diabetic mothers in White classes A, B, and C than in the newborns of nondiabetic mothers (12). On the other hand, in classes D through R, maternal microangiopathy exposes the fetus to chronic stress, which seems to accelerate pulmonary maturity. Delayed lung maturity also appears to be caused by fetal hyperinsulinism, which inhibits the stimulation of pulmonary surfactant production by fetal cortisol (13). We perform biochemical analysis of the components of pulmonary surfactant in amniotic fluid to ascertain fetal lung maturity before elective delivery. Such fluid is obtained by amniocentesis as soon as preterm delivery seems indicated by either maternal or fetal compromise, other than in emergency situations. The ratio of lecithin to sphingomyelin, which is an extremely accurate index of pulmonary maturity in normal fetuses, has been reported to be less accurate in diabetic patients (14). Testing for the presence of phosphatidylglycerol (another component of surfactant) along with the lecithin/sphingomyelin ratio improves the accuracy of our prediction of pulmonary maturity to 99% (15).

Biophysical methods to assess fetal well-being allow infants to be delivered closer to term without increased risk of antepartum fetal death. Recognition that the heart rate of a healthy fetus accelerates in association with vigorous (i.e., perceivable by the mother) activity led to the acceptance of nonstress testing as a primary means of antepartum fetal monitoring (16,17). As a biophysical screening method for fetal compromise, we have our patients at 28 weeks gestation begin to count and record the number of perceived movements in the same hour each day. The basis for this "kick sheet" is the observation that a decrease in fetal activity is an early indicator of fetal compromise (18). A kick count of fewer than five in two hours or a significant drop in kicks per hour requires immediate NST evaluation.

Many indirect biochemical methods to assess fetal well-being have been tested. The most successful are maternal urinary and plasma estriol determinations. Since the fetus plays an essential role in the biosynthesis of estriol (involving both fetal adrenal and liver functions), fetal compromise may decrease the concentration of estriol in maternal blood or urine (6). To be of use in making

decisions about obstetrical management, such measurements must be made daily on 24-hour urine collections. Normal daily variation in estriol production requires that each day's measurement be compared with the mean of the previous three. However, because the specificity and sensitivity of estriol as a predictor of fetal outcome have been questioned (19), and because of logistical problems with their use, estriol studies have been discarded in favor of direct, biophysical methods of assessing fetal well-being.

We are concerned about the overall Caesarean section rate of 50% in this group of patients. The rate for all patients delivered at Henry Ford Hospital during the study period was 14.2% (9.2% primary). Caesarean section without attempt at induction of labor was used liberally in the past for preterm delivery of diabetic women. Fetal kick counts or nonstress testing should increase the number of diabetic pregnancies which can be allowed to develop to 38 weeks gestation or longer. At that point, induction of labor is more likely to succeed. In this report, failure of induction was the most prevalent indication for abdominal delivery, and 75% of such failures occurred before 38 weeks. In addition, continuous fetal heart rate monitoring in labor now enables us to diagnose incipient but correctable fetal distress. Reducing the risks of unexpected intrauterine death and intrapartum fetal compromise by improved antepartum and intrapartum surveillance should diminish the incidence of elective sections before term and at term.

Nonetheless, this report reflects a continuing tendency toward preterm delivery as soon as amniocentesis demonstrates pulmonary maturity. The pressure to deliver soon after fetal lung maturity has been confirmed is especially great when managing patients with previous fetal losses at or near term. Such practice is likely to perpetuate relatively high section rates.

Our perinatal mortality rate of 4% (2/46) is similar to rates recently reported by other centers (7,20,21). Fetal and neonatal deaths due to prematurity, birth trauma, intrauterine death unassociated with ketoacidosis, and neonatal metabolic complications can now be prevented by careful metabolic control and accurate assessment of fetal maturity and well-being.

One of the major challenges facing perinatologists in the 1980s is to prevent congenital anomalies in the offspring of diabetic patients. In many series, birth defects are the leading cause of perinatal mortality (6). Such birth defects, which caused one of our two losses, occur in up to 20-36% of infants born to diabetic mothers. The most

common anomalies involve the heart and skeletal system. Recent data demonstrate an association between glycosylated hemoglobin levels at seven to eight weeks gestation and the incidence of congenital anomalies seen at delivery (22). These data indicate that the level of glycemic control at fertilization and during the period of organogenesis is a critical determinant of teratogenesis in these infants. Ideal care of pregnancies complicated

by diabetes must begin with tightening of glycemic control when a diabetic woman begins to contemplate conception. To realize this goal, all physicians who treat diabetic women in the reproductive age group must help. Only when aggressive management is practiced from before conception though the postpartum period are the diabetic woman's chances for successful pregnancy maximized.

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### Some Musculoskeletal Problems Associated with Diabetes

- Peri-capsulitis of the shoulders and hips
- Flexor tenosynovitis
- Carpal tunnel compression
- Diabetic cheiroarthropathy
- Diffuse idiopathic skeletal hyperostosis
- Dupuytren's contracture
- Neuropathic arthropathy (Charcot joint)

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