Identification of the High-Risk Gravida

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In the past, there has been considerable pessimism about our ability to identify the pregnant patient at risk. However, with the development of sophisticated diagnostic techniques these patients can be identified and with appropriate treatment their outcome can be improved. This paper presents the overall benefit of categorizing obstetrical patients, the method that has been developed at the Medical College of Virginia (MCV), and certain categories of high-risk obstetrical patients who continue to present problems and have unacceptably high complication rates.

A variety of systems has recently appeared in the literature designed to categorize the high-risk obstetrical patient.[1] To be successful such a system must be accurate and simple enough to understand. Some of the initial systems of categorization were simply too complicated. They involved the tabulation of multiple factors drawn from virtually every aspect of the patient's lifestyle, physical examination, and laboratory assessment. Consequently, they were too cumbersome to be practical. The goal of any identification system should be to separate patients into groups which can then be managed according to the common requirements of each group.

The classification system used at MCV is based on the premise that a given pregnancy may represent a progressive risk to the fetus or the mother and that this risk can be attenuated by appropriate care. Not included in the system are patients at risk for congenital anomalies. This group of patients undergoes comprehensive early antenatal evaluation. The results of this evaluation are then made available to the parents, who in turn determine what action is to be taken. On the other hand, there is a larger group of patients whose pregnancies are at progressive risk in utero. Identification, classification and appropriate intervention can improve perinatal outcome in this larger group.

The MCV identification system is divided into four categories. It was recognized at the outset that obstetrical patients could not simply be separated into normal patients and high-risk patients. If this were done, 90% of the patients would be in the high-risk category and only 10% in the normal category. Instead, it seemed more logical to group patients according to the severity of their problems.

Class IV *Critical care pregnancies*
- Eclampsia
- Severe preeclampsia
- Chronic hypertension with superimposed preeclampsia
- Chronic renal disease uncompensated (creatinine 1.2 mg/% or greater)
- Organic heart disease uncompensated (early signs of failure)
- Hemoglobinopathies in crisis
- Pyelonephritis, acute
- Premature rupture of the membranes
- Premature dilatation of the cervix in the second half of pregnancy
- Diabetes (ketonuria)
- Placental accidents (abruptio placenta

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Class III *High-risk pregnancy*
- Mild preeclampsia
- Diabetes without evidence of ketonuria
- Chronic hypertension
- Chronic renal disease compensated (creatinine less than 1.2 mg/%)
- Organic heart disease compensated (no signs of failure)
- Hemoglobinopathy, disease stable (hemoglobin less than 10 gm%)
- Rhesus negative, sensitized
- Previous intrauterine fetal demise in second half of pregnancy
- Proven intrauterine growth retardation
- Maternal weight loss
- Gestational age documented greater than 42 weeks
- Multiple pregnancy
- Maternal weight greater than 300 pounds
- Deficiency anemias (hemoglobin less than 10 gm/%)

Class II *At-risk pregnancy*
- Maternal weight between 250 and 300 pounds
- Hemoglobinopathy, trait (hemoglobin 10 gm/% or greater)
- Deficiency anemias (hemoglobin 10 gm/% or greater)
- History of urinary tract infections
- Bacteriuria
- Rhesus negative, unsensitized
- Suspected intrauterine growth retardation
- Inadequate maternal weight gain
- Previous cesarean section
- Previous premature baby
- Previous baby 10 pounds or greater

Class I *Normal pregnant patients*

Critical care pregnancies (Class IV) are those pregnancies in which there is an imminent possibility of decompensation. As is apparent from the diagnoses, there is a risk of death to the fetus or the mother. These patients should generally be cared for in the hospital.

High-risk pregnancies (Class III) include those that are not quite as critical as the Class IV type but whose diagnoses carry an unacceptably high perinatal loss. This is the category at which all the "new" antepartum testing techniques and methods of management have been directed. Specialized high-risk obstetrical clinics have been developed in referral centers to evaluate and closely follow patients in this category. The most substantial improvement in perinatal outcome can be realized in the Class III category.

At-risk pregnancies (Class II) need to be identified but do not require specialized surveillance. From the nature of the diagnoses it is apparent that all these patients have the capacity to decompensate and therefore require close supervision.

Low-risk pregnancies (Class I) include patients with normal pregnancies. Frequently, the least number of patients are found in this category. These are patients with normal histories, normal physical examinations and normal laboratory values. Their pregnancies follow the projected course for fundal growth, maternal weight gain, blood pressure, and all other parameters of normal pregnancy.

It is important to note that the assignment of a classification does not mean that the patient is necessarily going to remain in that classification throughout the pregnancy. Patients may shift from one class to another as their status improves or worsens. For example, if a patient has an iron deficiency anemia with hemoglobin of 11 gm/% she is placed in the Class II category. She is counseled about nutrition and appropriate diet, and iron supplements are prescribed. If, however, her hemoglobin drops to 9 gm/% as her pregnancy progresses, she is then placed in the Class III category. This may mean that she is transferred from a routine obstetrical clinic to a high risk clinic with specialized surveillance.

There are several diagnoses contained in the classification system that are of special interest. One such diagnosis is maternal weight loss (Class III).

In the past, not enough attention has been paid to adequate weight gain in pregnancy. A great deal of emphasis has been placed on excessive weight gain yet poor weight gain or worse, weight loss, has not been so readily recognized. It is important to realize that the correlation between low-birth-weight infants and lack of maternal weight gain is greater than with any other single factor. When charts are examined retrospectively for factors such as parity, socioeconomic status, maternal weight gain, tox-

emia, renal disease, cigarette smoking, and number of children, the greatest influence on fetal weight gain is maternal weight gain.

Poor fetal weight gain is not necessarily due to the fact that the mother's nutritional status is inadequate. Maternal weight gain is predicted both on her nutritional status and on the size of her fetus. If there is a problem preventing the growth of the fetus, such as rubella, the mother will not gain weight. This mother can be fed an adequate diet, but the baby will not grow because of its limited potential. Decreased maternal weight may cause poor fetal growth, but the reverse is also true. Poor fetal growth may be responsible for limited maternal weight gain.

Suspected intrauterine growth retardation (IUGR) is another category that deserves special attention because of the difficulty of diagnosis. Suspected intrauterine growth retardation, as measured by biparietal diameter with ultrasound, is often an iatrogenic problem. Erroneous measurements, or studies done too frequently, may indicate that there is lack of growth in the biparietal diameter. This points to the possibility of placental or fetal compromise, although, in fact, it may be nothing more than laboratory error.

Intrauterine growth retardation diagnosed prior to 28 weeks gestation should be extremely suspect. Even in placental insufficiency syndromes, the fetal head usually continues to grow past 28 weeks, and it is extremely unlikely that the diagnosis of IUGR can be made from biparietal diameter data before that time. A biparietal diameter four or more weeks behind the dates prior to 28 weeks usually indicates "wrong dates."

Hypertensive disorders in pregnancy are important because of their frequency and because of the profound effects they have both on the mother and the fetus. They are divided into the toxemias (eclampsia and preeclampsia), chronic hypertension, gestational hypertension, and toxemia superimposed on hypertension. Gestational hypertension is hypertension that is unmasked in pregnancy but without the criteria for the diagnosis of toxemia.

Making the appropriate diagnosis of hypertensive disorders in pregnancy can be confusing. However, high blood pressure, regardless of etiology, has a deleterious effect on the end organs, be it the brain, the liver, the kidney, the cardiovascular system or the placenta. While it is important to establish a diagnosis, it is more important to realize that the magnitude of the blood pressure and the extent of end organ damage is directly proportional to fetal-maternal morbidity and mortality. In a series of hypertensives with proteinuria, the perinatal mortality rate was 37.9 per 1000 births. This compared with a rate of 17.2 per 1000 for normotensive patients without proteinuria.[2] In patients with diastolic blood pressure greater than 120, the perinatal mortality is 50%.[3]

Hypertensive syndromes in pregnancy continue to result in maternal mortality. The outcome is compromised particularly if accelerated hypertension occurs in the third trimester. Generally this is categorized as chronic hypertension with superimposed toxemia.[4] Chesley reports that hypertension is rarely aggravated in pregnancy unless there is significant cardiac, renal, or retinal pathology.[5] What must be emphasized, however, is that this type of pathology is fairly common in hypertensives and when these women become pregnant they are at increased risk.

In the past several years much attention has been directed at predicting the patient who will develop hypertension in pregnancy. Dalton looked at weight gain as an indicator.[6] She reported the incidence of preeclampsia to be 26% in patients who gained more than 1¾ pounds per week after the 30th week of gestation. The weight gain she is alluding to is not really caloric weight gain but rather fluid retention which is one of the triad of symptoms associated with preeclampsia. Edema, however, does not correlate well with increased perinatal morbidity and mortality. In fact, fluid retention is the least significant of the triad of hypertension, proteinuria and edema in relationship to perinatal mortality.[3] Chesley has demonstrated that the diastolic blood pressure is the most significant factor in patients who will develop hypertension in pregnancy.[5] In his study, diastolic elevation occurred as the first symptom in 58% of patients whereas proteinuria occurred as the first symptom in only 34%. Proteinuria is only a reflection of the end organ damage to the kidney in hypertensives. In toxemia the vascular changes resulting in decreased perfusion of vital organs is frequently long standing prior to a noticeable elevation in the diastolic blood pressure. Indeed, Gant and others[7] have demon-

strated that the vascular changes typical of toxemia occur as early as 24 weeks. Using 140/90 as a standard, the physician may not be able to appreciate an elevation until 35 or 36 weeks when end organ damage is already substantial. If the diastolic blood pressure is 75-85 mm Hg, the perinatal mortality is about 7 per 1000. As the diastolic pressure increases to 85-90 mm Hg the rate is 10 per 1000. When the diastolic is 90-104 mm Hg, the perinatal mortality triples what it was at 75 mm Hg and there is a progressive linear increase in perinatal mortality as the diastolic blood pressure continues to elevate.[3] A diastolic reading of 90 mm Hg at any point in pregnancy is distinctly abnormal.

Calculation of the mean arterial pressure (MAP) is the most sensitive method of predicting impending hypertension in pregnancy. This measurement is obtained using the following formula.

$$MAP = \frac{\text{Systolic} + 2\ \text{Diastolic}}{3}$$

A MAP greater than 90 in the second trimester or greater than 105 in the third trimester is prognostic of hypertension, either gestational hypertension or toxemia.[8]

Page and Christianson calculated MAP in the second and third trimesters and correlated it with outcome.[8] They found that when the MAP went from 90 to 95 or greater, the incidence of preeclampsia tripled. It is important to note that a blood pressure of 140/90 yields a MAP of well over 100. A blood pressure reading that may appear grossly normal is often abnormal when calculating its mean arterial pressure.

Diabetes in pregnancy, while it represents a much smaller proportion of patients than the hypertensives, continues to be a problem. Diabetic pregnancies carry a perinatal death rate four to five times higher than normal pregnancies.[9] The outcome depends largely on the severity of the diabetes and the amount of vascular disease present prior to pregnancy. Some of the common problems seen in these pregnancies are congenital anomalies (6%), oligohydramnios, premature rupture of membranes, macrosomia, toxemia (13% to 50%), urinary tract infections, increased incidence of cesarean section, birth trauma and intrauterine deaths. In addition, babies of diabetic mothers experience many problems in the newborn nursery. They include hypoglycemia, respiratory distress syndrome, and hyperbilirubinemia.

The key to improved outcome in diabetic pregnancies centers around early diagnosis and strict metabolic control. Patients with a family history of diabetes or macrosomic babies, (greater than 4000 grams) should have a glucose tolerance test to screen for diabetes. In women who have had a previous baby weighing more than 4000 grams, 10% have undiagnosed diabetes.[10] These women have high blood sugars which stimulate the fetal pancreas to produce insulin. Insulin acts like growth hormones in the fetus resulting in macrosomia. Glycosuria in pregnancy is another indicator to screen for diabetes. It cannot be dismissed as a decreased renal threshold for glucose or as galactosuria secondary to breast development. Any patient with glycosuria in pregnancy should be considered diabetic until proven otherwise.

Finally, patients with a poor obstetrical history should be screened for diabetes. This includes previous congenital anomalies, stillbirth, and repeated pregnancy loss.

In conclusion, the classification system developed at the Medical College of Virginia to identify the high-risk gravida has been presented with a discussion of some of the problems in pregnancy that carry a high perinatal mortality. The importance of classifying patients according to risk is emphasized so that appropriate management can ensue. In this way, pregnancy outcome can be improved.

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