In the United States, the proportion of the adult population classified as obese has steadily increased over the past 20 years. Recent estimates suggest that nearly 1 in 5 adults in the United States is obese, as defined by a body mass index (BMI) of 30.0 kg/m² or more. The general health burden this trend imposes upon society has been well described, with consistent associations between obesity and increased risks of chronic diseases such as hypertension, diabetes mellitus, and death from all causes. In pregnancy, maternal obesity has been associated with an increased risk of diabetes mellitus, pre-eclampsia, and cesarean delivery. The offspring of obese women are more likely to be excessively large, placing them both at risk for birth trauma. A quantitative assessment linking the increasing prevalence of obesity with its attributable perinatal risks has not been performed, in spite of these associations.

We used a computerized perinatal database, derived from an obstetric care system that has served pregnant women whose demographic profile has changed little for over 20 years, to assess temporal trends in maternal weight and calculate the increasing attributable risks of maternal obesity.

OBJECTIVE: In this study, we assessed the temporal trends and relative and attributable perinatal risks of maternal obesity over a 20-year period.

STUDY DESIGN: We conducted a retrospective cohort study between 1980 and 1999 by using a computerized perinatal database of all women who received prenatal care and delivered their infants within a regional health care system. The main outcome measures were as follows: (1) annual mean body weight and the percentage of women classified as obese at the first prenatal visit (primary definition ≥200 lb; secondary definitions ≥250 lb, ≥300 lb, body mass index ≥29 kg/m²); and (2) relative and attributable risks of obesity for selected maternal and perinatal morbidity in successive 5-year periods.

RESULTS: From 1980 to 1999, the mean maternal weight of women at the first prenatal visit increased 20% (144-172 lb), as did the percentage of women ≥200 lb (7.3-24.4), the percentage ≥250 lb (1.9-10.7), the percentage ≥300 lb (0.5-4.9), and the percentage with a body mass index >29 kg/m² (16.3-36.4), P < .01 for all. Controlling for maternal age, race, and smoking status, obese women were at increased risk at each period for cesarean delivery (range of adjusted relative risk, 1.5-1.8), gestational diabetes (range, 1.8-2.9), and large (>90th percentile) for gestational age infants (range, 1.8-2.2). From the earliest 5-year period (1980-1984) to the most recent (1995-1999), the percentage of obesity-attributable cesarean deliveries more than tripled from 3.9 to 11.6. Similar percentage increases were observed for the obesity-attributable risks for gestational diabetes (12.8-29.6) and large for gestational age infants (6.5-19.1). Trends for secondary obesity definitions were similar, although the magnitude of the increased attributable risks was smaller.

CONCLUSIONS: Efforts to reduce the frequency of certain perinatal morbidities will be constrained unless effective measures to prevent, or limit the risks of, maternal obesity are developed and implemented. (Am J Obstet Gynecol 2001;185:845-9.)

Key words: Maternal obesity, perinatal outcome, pregnancy


rians or nurse practitioners record the intrapartum and post partum data on standardized data forms. Dedicated personnel are then responsible for entry of the antepartum data into the database, as well as for conducting scheduled audits of it.

All of the women in this investigation received prenatal care through 1 of 9 clinics operated jointly by the Jefferson County, Alabama Health Department and the Department of Obstetrics and Gynecology at the University of Alabama, Birmingham, or at a tertiary referral clinic operated by the latter. The clinics are staffed by certified nurse midwives and nurse practitioners, obstetrics and gynecology residents, and obstetrics faculty. Inpatient care, including deliveries, was provided by residents under the supervision of faculty at one of two university-affiliated hospitals (The University of Alabama, Birmingham and Cooper Green Hospital).

To minimize the effect of the pregnancy-related weight gain, we included in the analysis only those women who sought prenatal care before the early third trimester, which was established by a standard obstetric dating algorithm. Sociodemographic data, including age, race, parity, reproductive history, education, and smoking status were obtained during the first prenatal visit. Maternal weight was recorded while the women were wearing light clothing and no shoes. Body mass index was calculated from the prenatal weight at the first visit and maternal height (obtained through recall).

Maternal outcomes assessed included cesarean delivery and the presence of gestational diabetes mellitus. Neonatal outcomes included preterm delivery (before 37 weeks’ gestation), small (<10th percentile) for gestational age (LGA) and sex according to the Brenner nomogram,16 macrosomia (>4500 g), fetal death (after 20 weeks’ gestation) and neonatal death (birth to 28 days of life).17 These outcomes were influenced by maternal body habitus and were measured and recorded objectively. The distinction between LGA infants and those with macrosomia within this analysis was important, because although they both reflect large infants, early intervention for delivery may be a confounding factor in the proportion of infants which are categorized as large when defined by an absolute birth weight threshold (such as >4500 g).

Throughout the study period, universal screening for gestational diabetes mellitus was performed by using a 50-g glucose solution given to women at or beyond 24 weeks’ gestation. An abnormal screening result was defined as a serum glucose level of at least 135 mg/dL. Women with an abnormal screening result underwent a 3-hour 100-g glucose tolerance test, and throughout the 20-year period, gestational diabetes was diagnosed according to the same standard criteria.18

Definition of obesity. A traditional definition of obesity is a maternal prepregnancy BMI of more than 29.0 kg/m².19 In the study clinics, maternal weight was universally measured, recorded, and subject to regular systematic reliability audits. Maternal height, however, was recorded from self-report and was available for only 70% of patients. For the purpose of this investigation, our primary definition of obesity was a first prenatal visit weight of at least 200 lb. Women who weighed <200 lb were considered to be at a normal weight. Among women weighing 200 lb, only those with a height of at least 5 ft 9 in had a BMI less than 29 kg/m². Within our population, that height threshold represents the 99th percentile; therefore, the 200 lb threshold is unlikely to include women with a BMI of less than 29 kg/m². In secondary analyses, the effect of alternate obesity definitions was evaluated, including a first prenatal visit weight of at least 250 to 300 lb and a BMI of more than 29.0 kg/m².

Statistical analysis. The temporal trend of obesity, as explained by the various definitions listed before, was calculated yearly. Because of the infrequent annual incidence of some of the perinatal outcomes of interest, the data were aggregated into successive 5-year periods. The earliest period ranged from January 1980 to December 1984, and the most recent period ranged from January 1995 to December 1999.

When applicable during each period, relative risks (RRs) and 95% CIs for obese women were calculated. As a control for the known confounding effects of maternal age, race, and smoking status,20-22 a logistic regression model was developed to calculate the adjusted RR. Because some women may have been included in the analyses more than once, separate analyses were performed with data from only nulliparous women.

The population-attributable risks, also referred to as the population etiologic fraction, for selected obesity-related perinatal outcomes were calculated by the method of Miettinen.23 They represent the proportion of an adverse outcome that can be attributed to exposure to a given risk factor in this case obesity. This differs from the concept of RR, which represents how much more often an outcome occurs in those with and without a given risk factor. The rationale for reporting the population-attributable risk, as opposed to the absolute risk or RR, is that it takes into account both the prevalence and the excess risk of a given factor.

Proportional data were compared by using a χ² or Fisher exact test, as appropriate. Continuous data were compared with the use of the Student t test. Data were analyzed with SAS Version 8.0 (SAS Institute, Cary, NC). Statistical significance was set at P < .05.

Results

Data from 53,080 women were available for the analysis. The two clinically important demographic changes over the 20 years were an increase in the proportion of the race category “other” (because of a rise in the number of pregnant Hispanic women) and a decline in the rate of smoking (Table 1).
From 1980 to 1999, the mean weight at first prenatal visit increased progressively from 144 lb to 172 lb. The percentage of women classified as obese by a first prenatal weight $\geq 200$ lb increased from 7.3 to 24.4. Obesity percentages as defined by a first prenatal weight $\geq 250$ lbs (1.9-10.7), $\geq 300$ lbs (0.5-4.9), or BMI $>29$ kg/m$^2$ (16.3-36.4) increased over the same period, respectively (Figure). Although the absolute rate of cesarean delivery did not increase from the first period (16.9%) to the fourth (16.3%), the rate of gestational diabetes increased nearly 25% (2.1-2.6) as did the percentage of LGA infants (11.5-13.9). The rate of SGA infants decreased from 10.2% to 6.2%. In every 5-year period, after adjustment for age, race, and smoking status, obese women were at significantly increased risk for cesarean delivery, gestational diabetes mellitus, and LGA newborns or those with macrosomia, and they were at decreased risk for delivering SGA newborns. Although obese women were at increased risk for fetal death, the risk increased significantly in only 1 of the 4 periods (Table II). Analyses confined to nulliparous women yielded point RR estimates essentially identical to the overall analysis but with a wider 95% CI. For example, among obese ($\geq 200$ lb), nulliparous women, the RR (95% CI) for cesarean delivery in each successive period was 1.7 (range, 1.3-2.2), 1.5 (range, 1.2-1.9), 1.9 (range, 1.6-2.2), and 1.6 (range, 1.3-1.9). The RR (95% CI) for LGA infants was 1.6 (range, 1.1-2.2), 1.8 (range, 1.4-2.5), 2.1 (range, 1.8-2.6) and 2.2 (range, 1.8-2.7).

Population-attributable risks were determined for those obesity-related perinatal outcomes in which the adjusted RRs were significantly increased in all 4 periods (ie, cesarean delivery, gestational diabetes, and LGA infants and those with macrosomia) (Table III). The trend was a progressive increase in the obesity-attributable risk with each successive period. For each of the perinatal outcomes, except macrosomia, the increase between the earliest and most recent periods was significant, as evidenced by the lack of overlap in the 95% CI. When the calculations of population-attributable risks were confined to nulliparous women, similar trends were observed. For example, the obesity-attributable risks for cesarean delivery in each of the successive periods were 3.6%, 4.5%, 8.9%, and 9.1%, and those for LGA infants were 2.9%, 6.2%, 10.2%, and 15.4%.

Comment

In this study, we examined the longitudinal trend of maternal obesity within our obstetric population over a 20-year period. Maternal weight obtained at the first prenatal visit and the percentage of women considered obese, regardless of the definition used, steadily increased. These dramatic trends mirror those from other reports on the general US population. In 1999, 4.9% of the pregnant women weighed at least 300 lb at their initial prenatal visit, a 10-fold increase in only 20 years. These trends were observed in spite of the fact that, on average, women in the most recent period (January 1995-December 1999) had their first prenatal visit almost 3 weeks earlier than women in the first period (January 1980-December 1984).

The increasing prevalence of maternal obesity in turn was associated with an increased absolute rate of several adverse perinatal outcomes, including gestational diabetes and LGA infants. The cesarean delivery rate was relatively stable over the course of the study (16.9% in the first 5-year period and 16.3% in the last). However, as opposed to the largely physiologic outcomes of gestational

Table I. Maternal characteristics

<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>(n = 11,126)</td>
<td>(n = 8,373)</td>
<td>(n = 16,632)</td>
<td>(n = 16,949)</td>
</tr>
<tr>
<td>Mean age (y)</td>
<td>23.4</td>
<td>24</td>
<td>22.8</td>
</tr>
<tr>
<td>Race (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>67.2</td>
<td>66</td>
<td>72.4</td>
</tr>
<tr>
<td>White</td>
<td>32.5</td>
<td>33.4</td>
<td>26.4</td>
</tr>
<tr>
<td>Other</td>
<td>0.3</td>
<td>0.5</td>
<td>1.2</td>
</tr>
<tr>
<td>Weight ($\geq 200$ lb) (%)</td>
<td>7.7</td>
<td>10.7</td>
<td>13.9</td>
</tr>
<tr>
<td>Education level &lt;12 y (%)</td>
<td>39.9</td>
<td>37.2</td>
<td>41.6</td>
</tr>
<tr>
<td>Smoking status (%)</td>
<td>29.6</td>
<td>30.1</td>
<td>22.9</td>
</tr>
<tr>
<td>Mean gestational age at first visit (wk)</td>
<td>15.9</td>
<td>15.2</td>
<td>14.7</td>
</tr>
<tr>
<td>Nulliparous (%)</td>
<td>54.9</td>
<td>58.1</td>
<td>45.1</td>
</tr>
</tbody>
</table>

Differences significant across time for all.
Table II. Perinatal outcomes associated with maternal obesity (first prenatal weight ≥200 lb) within each 5-year period for all women (multiparous and nulliparous)

<table>
<thead>
<tr>
<th>Period</th>
<th>CD</th>
<th>GDM</th>
<th>SGA</th>
<th>LGA</th>
<th>&gt; 4500 g</th>
<th>Fetal death</th>
<th>Neonatal death</th>
</tr>
</thead>
<tbody>
<tr>
<td>1980-84</td>
<td>1.5 (1.3-1.8)</td>
<td>2.6 (1.8-3.6)</td>
<td>0.7 (0.5-0.8)</td>
<td>1.9 (1.6-2.3)</td>
<td>3.3 (2.1-5.2)</td>
<td>1.5 (0.9-2.7)</td>
<td>0.8 (0.4-1.8)</td>
</tr>
<tr>
<td>1985-89</td>
<td>1.7 (1.4-1.9)</td>
<td>1.8 (1.3-2.5)</td>
<td>0.7 (0.5-1.0)</td>
<td>1.8 (1.5-2.2)</td>
<td>6.2 (3.9-9.8)</td>
<td>1.9 (1.1-3.1)</td>
<td>0.7 (0.3-1.4)</td>
</tr>
<tr>
<td>1990-94</td>
<td>1.8 (1.6-2.0)</td>
<td>2.9 (2.3-3.5)</td>
<td>0.7 (0.5-0.8)</td>
<td>2.1 (1.8-2.3)</td>
<td>3.2 (2.4-4.4)</td>
<td>1.4 (0.9-1.9)</td>
<td>1.8 (1.2-2.8)</td>
</tr>
<tr>
<td>1995-99</td>
<td>1.6 (1.4-1.8)</td>
<td>2.7 (2.2-3.4)</td>
<td>0.6 (0.4-0.7)</td>
<td>2.2 (1.9-2.4)</td>
<td>2.4 (1.7-3.6)</td>
<td>1.3 (0.9-1.9)</td>
<td>1.6 (0.9-2.9)</td>
</tr>
</tbody>
</table>

Adjusted relative risks (95% CI).
CD, Cesarean delivery; GDM, gestational diabetes mellitus; SGA, small for gestational age; LGA, large for gestational age; >4500 g = infant birth weight >4500 g.

*Adjustments made for maternal age, race, and smoking status.

Table III. Percentage (95% CI) of selected perinatal outcomes attributable to obesity within each 5-year period

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>CD</td>
<td>3.9 (2.4-5.0)</td>
<td>6.4 (4.8-7.7)</td>
<td>10.6 (8.4-11.1)</td>
<td>11.6 (9.5-13.4)</td>
</tr>
<tr>
<td>GDM</td>
<td>12.8 (9.8-15.1)</td>
<td>9.6 (4.6-13.1)</td>
<td>22.7 (19.9-25.1)</td>
<td>29.6 (24.9-32.9)</td>
</tr>
<tr>
<td>LGA</td>
<td>6.5 (5.2-7.8)</td>
<td>8.0 (6.1-9.6)</td>
<td>11.8 (10.7-13.0)</td>
<td>19.1 (16.9-20.8)</td>
</tr>
<tr>
<td>Macrosomia</td>
<td>16.2 (12.2-18.7)</td>
<td>39.1 (28.9-41.9)</td>
<td>25.2 (20.8-28.1)</td>
<td>25.7 (17.4-31.4)</td>
</tr>
</tbody>
</table>

CD, Cesarean delivery; GDM, gestational diabetes; LGA, large for gestational age infant; Macrosomia, infant with a birth weight >4500 g.

diabetes and LGA infants and those with macrosomia, the cesarean delivery rate is affected by many factors, only some of which are physiologic. Moreover, during the most recent 5-year period, we instituted strict labor management guidelines at the two study hospitals in an attempt to reduce the cesarean delivery rate. It is likely that use of these guidelines blunted some of the expected increase in the cesarean rate because of maternal obesity. Nevertheless, even under these guidelines, obese women had an RR of 1.5% for cesarean delivery in the earliest period and 1.6% RR in the most recent period.

Our calculation of the population-attributable risks, although not frequently used in the obstetric literature, does afford advantages over reporting either the absolute rates or RRs of selected outcomes. Because the calculation incorporates both prevalence and RR, it is particularly useful in highlighting changes of a given outcome over time, especially if either of these factors remains stable. In our series, because the RRs of adverse perinatal outcomes associated with obesity were more or less constant over the four successive 5-year periods, but the prevalence of maternal obesity rose steadily, the result was a commensurable rise in the population-attributable risks of obesity-related morbidities.

Viewed from a different perspective, these calculations provide an estimate in the potential reduction of obesity-related adverse events if maternal obesity was eliminated, or more realistically, reduced to some previous rate. If our patient population between 1995 and 1999 had the same body composition as the population between 1980 and 1984, the cesarean delivery rate for the later period would have been 15.1% instead of 16.3% (8.0% lower), 20.8% fewer women would have been diagnosed with gestational diabetes, and 11.6% fewer infants would have been LGA.

The number of obesity-attributable outcomes was purposefully limited in the investigation to those that were reliably coded and objectively measured. Because we were using a database that spans for more than twenty years, we strove to minimize the potential for mutable clinical criteria and subjectivity to bias our results. Gestational diabetes, LGA infants, and macrosomia are diagnoses made without consideration of, and without reference to, maternal body habitus. Because it is conceivable that maternal body habitus may have influenced the decision to perform cesarean delivery, given the known increased surgical risks posed by obesity, the clinical bias would have been toward not performing cesarean delivery in obese women.

We acknowledge that these data may not be generalizable to all pregnant women in the United States. Alabama does have one of the highest state rates of obesity, 20.3% between 1997 and 1999.1 Additionally, the higher rate of obesity within our obstetric population can be attributed to the facts that our patients were all publicly funded and primarily black, both of which are contrary to the general population characteristics in Alabama and are associated with increased rates of obesity.24 It stands to reason that systems and regions with lower rates of maternal obesity would have fewer obesity-attributable adverse perinatal outcomes.
outcomes. Thus, to the extent that obesity is over-represented in this study, we have overestimated obesity-attributable outcomes. We point out, however, that the rising rate of obesity in the United States, because it is more pronounced in certain regions and among lower socioeconomic groups, is a universal phenomenon that encompasses the entire societal spectrum.

Over a 20-year period within a well-characterized obstetric delivery system, we have demonstrated that the increasing prevalence of maternal obesity, along with the increased RRs of adverse perinatal outcome faced by obese women, has led to a dramatic increase in the population-attributable risks of obesity-related pregnancy complications. Our data demonstrate that maternal obesity is a direct countervailing force to the general improvements in pregnancy outcome achieved over the same period, and these data must be addressed if future pregnancy outcomes are to be optimal.

REFERENCES