Sleep complaints are very common among pregnant women, but polysomnographic recordings systematically investigating the causes of these complaints are limited.1,2 In pregnancy, respiratory physiology is altered by physical and hormonal changes that can profoundly change breathing during sleep. Some of these changes may provide protection from sleep breathing disorders, whereas others may put women at risk.1,3–5 Changes during pregnancy with potentially protective effects decreases during late pregnancy in time spent in the supine position6 and in rapid eye movement (REM) sleep.7,8 The high circulating level of progesterone during pregnancy increases the ventilatory drive, which has a potentially protective effect.9

Obesity, as such, predisposes to sleep-related breathing disorders.10 Weight gain and increased nasal obstruction during pregnancy are considered potentially detrimental, leading to sleep breathing disorders.1 A decrease in arterial PO₂ has been noted during pregnancy when women change from a sitting to the supine position while awake, a decrease more important in obese patients.11 It may be that a combination of the supine position and sleep leads to worsened hypoxemia during pregnancy.
One of the potentially detrimental changes during pregnancy is the enlarging uterus, which alters diaphragmatic function. This results in reduced functional residual capacity, and in some patients a closing capacity greater than functional residual capacity, potentially causing shunting and hypoxemia; this decreased functional residual capacity reduces lung oxygen stores, which may lead to earlier occurrence of hypoxemia during hypoventilation in sleep. Nocturnal breathing and oxygenation have, according to one study, been well maintained in multiple pregnancies in nonobese mothers; however, that study included one mother with a slightly elevated body mass index (BMI), and she had significant partial upper-airway obstruction.

Two reports suggest that pregnancy may precipitate or worsen sleep apnea. One study looking at eight obese snoring pregnant women with a clinical diagnosis of sleep apnea found intrauterine growth retardation in all cases, but others have reported good fetal outcome. However, Crawford and Joel-Cohen and Schoenfeld noticed that even a small degree of hypoxemia during pregnancy may have profound effects on fetal development, resulting in intrauterine growth retardation.

This study was designed to evaluate sleep-related breathing in obese women during early (after 12 weeks) and late (after 30 weeks) pregnancy in comparison to that of normal-weight control subjects. Furthermore, we assessed obstetric and delivery data and data on the newborns.

**Materials and Methods**

**Subjects and Study Design**

Eleven obese but otherwise healthy pregnant women were recruited in the Helsinki region. Obesity was defined as a BMI of 20 to 25 kg/m² prior to pregnancy. Eleven healthy, nonobese (normal weight = BMI of 20 to 25 kg/m² prior to pregnancy) pregnant women were recruited in a similar manner. Subjects were ineligible if they were receiving any medication other than vitamin or iron supplements or if they had smoked > 20 pack-years.

All subjects were referred for the first polysomnography after their first routine obstetric ultrasound examination at 12 weeks of pregnancy. The second polysomnography was performed after 30 weeks. A prestudy decision was made to propose nasal continuous positive airway pressure to all mothers with an apnea-hypopnea index (AHI) > 10 events per hour. A follow-up polysomnography was planned 6 months postpartum for those with major pathologic findings. In connection with both polysomnographies, venous blood was drawn for estradiol and progesterone measurement. Furthermore, routine obstetric and delivery data were noted, as well as data on the newborn.

**Overnight Sleep Studies**

Prior to each sleep study, the subjects were asked to keep a sleep diary for 2 weeks. A sleep questionnaire based on the Basic Nordic Sleep Questionnaire was filled in by all before the first sleep study. A shorter follow-up questionnaire was used prior to the second study. In connection with both overnight recordings, daytime sleepiness was evaluated with the Epworth Sleepiness Scale.

The overnight hospital recordings were performed with a computerized 24-channel polygraph (Alice 3; Healthdyne Technologies; Marietta, GA). This included a four-channel EEG (C3/A2, C4/A1, O2/A1, O1/A2), electro-oculogram, and submental and leg (two separate) electromyograms. Heart rate was monitored through standard leads. Airflow was detected by monitoring with a nasal and oral thermostor, thoracic and abdominal belts (Healthdyne effort sensor; Healthdyne Technologies) were used for respiratory movement detection, Pulse oximetry (BCI Oximetry 3100; BCI International; Wankeisha, WI) was included in every recording. A body position sensor was included to record body position: supine vs other. A skin microphone was attached to the throat for snoring detection. Furthermore, the study included an overnight video recording. A nurse trained in sleep medicine attended the recording.

Sleep stage was scored manually in 30-s epochs and was based on the criteria of Rechtschaffen and Kales. Respiratory and nonrespiratory events were both scored visually. An apneic event was defined as absence of nasal or buccal flow for ≥ 10 s. Hypopnea was scored as the diminution of flow amplitude of > 50% and for > 10 s associated either with an arousal or an oxygen desaturation ≥ 3%. The AHI was defined as the number of apneas and hypopneas per hour of sleep. An oxygen desaturation index of 4 (ODI₄) was automatically registered by the polygraph when there was a ≥ 4% drop in oxygen saturation during sleep. An arousal was defined as an EEG frequency shift to the α range for at least 3 s. A respiratory event was scored when diminution of flow amplitude was observed for > 10 s associated with paradoxical chest and abdominal movement sometimes with a crescendo pattern of snoring. A respiratory arousal (RA) was scored when an arousal was preceded by an apnea, a hypopnea, or a respiratory event. The RA index was calculated by dividing the total number of RAs by total sleep time.

Snoring was detected with a microphone attached to the subject’s throat, and the analog signal was transferred to the monitor screen. Another microphone was attached to the ceiling, 2 m from the patient’s head, to record sounds on a videotape. Subjects were asked to snore as loud as they could while lying supine during the calibration process before the start of the recording. The maximal snores signal during calibration was given a value on an arbitrary scale from 0 to 100. With a snoring signal of < 50, no snoring was heard on the videotape. A snoring event was scored visually if the signal was at least 50% of the calibration signal. A snoring episode included at least one snoring event and terminated when no snoring event was detected for two breathing cycles. The time spent in snoring episodes was divided by total sleeping time to give the figure for snoring time. Breathing irregularity was scored visually in epochs of 5 min. Irregular breathing was noted when either the respiratory frequency or the respiratory amplitude was changed for > 50% of the epoch. Periodic leg movement scoring was based on American Sleep Disorders Association Task Force recommendations. All respiratory parameters were calculated separately for REM and non-REM sleep as well as for the supine position.

**Statistical Analysis**

The data are expressed as mean ± SEM. The Mann-Whitney U test, z-adjusted for ties, and Yates corrected χ² test were used to compare findings between the obese and control groups. Wilcoxon's matched-pairs test was used to compare the findings.
within the groups at two separate assessment points. All computations were performed with a commercial statistical package (Statistica v.5; StatSoft; Tulsa, OK); p values $\leq 0.05$ were considered to indicate statistical significance.

Ethical Considerations

The study was approved by the Ethics Committee of the Helsinki University Central Hospital. Written consent was obtained from each subject prior to the study.

RESULTS

Subjects

Prepregnancy data on the obese and control subjects are given in Table 1. All study subjects were white and born in Finland. Mean age of the mothers was 31 to 32 years. There were a few smokers in either group, and all had smoked $< 10$ years. None reported smoking during pregnancy. Self-reported snoring prior to becoming pregnant was more common in obese subjects, but the difference was not statistically significant. In the obese group, two women were primiparas, but only four had a child or children. In the control group, eight women were primiparas and only one woman had children.

Changes in Mothers’ Weight and Health During Pregnancy

BMIs in the obese mothers at the times of the polysomnographies were $35.4 \pm 0.5$ kg/m$^2$ and $38.3 \pm 0.9$ kg/m$^2$; corresponding data for the control subjects were $23.8 \pm 0.5$ kg/m$^2$ and $26.9 \pm 0.7$ kg/m$^2$, respectively (Fig 1). All differences in BMI at any assessment point between the obese and control subjects were highly significant ($p < 0.001$). Mothers in the obese group gained $12.5 \pm 3.6$ kg and control mothers gained $15.9 \pm 2.2$ kg (not statistically significant).

No systolic BP $> 140$ mm Hg or diastolic $> 90$ mm Hg were measured in any subject during early pregnancy. Preeclampsia during late pregnancy developed in one subject in the obese group, but with oral $\alpha$- and $\beta$-blocker medication, her BP returned to normal. Gestational diabetes developed significantly ($p < 0.05$) more often in the obese subjects (8 of 11 women) than in the control group (1 of 11 women). However, no medication was needed for diabetes control in any of these subjects.

Sleepiness and Sleep Characteristics

Sleepiness was evaluated on the Epworth Sleepiness Scales. If we consider values $\geq 8$ of 24 to indicate abnormal sleepiness, two subjects in the obese group and one subject in the control group were exceptionally sleepless at the time of the first polysomnography. Only the same three subjects were sleepy at the time of the second recording.

The main sleep characteristics are shown in Table 2. Three obese and two control subjects refused to undergo a second polysomnography. When the early- and late-pregnancy polysomnographies were performed during $16.3 \pm 1.2$ weeks and $32.7 \pm 0.8$ weeks in the obese group and $15.3 \pm 0.7$ weeks and $34.7 \pm 0.7$ weeks of pregnancy in the control group, respectively, no significant differences appeared between the groups at these time points.

Both obese and control subjects slept somewhat $< 7$ h in the recording at 80% sleep efficiency. In the polysomnography during late pregnancy, both groups slept more than half an hour less, with a deterioration in sleep efficiency and more wakefulness. During early pregnancy, REM sleep comprised

Table 1—Prepregnancy Data in Obese and Control Subjects

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<tbody>
<tr>
<td>Obese</td>
<td>11</td>
<td>31.3 ± 2.0</td>
<td>33.5 ± 0.9†</td>
<td>3</td>
<td>5</td>
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<tr>
<td>Control</td>
<td>11</td>
<td>31.8 ± 1.1</td>
<td>22.5 ± 0.5†</td>
<td>3</td>
<td>2</td>
<td>1</td>
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</tbody>
</table>

*Data are presented as mean ± SEM unless otherwise indicated.
†Snorers = self-reported snoring more often than once a week (prior to pregnancy); apneics = self-reported sleep apneas (prior to pregnancy).

†Difference between groups statistically significant.
about 20% of sleep, and slow-wave sleep (SWS) of stages 3 and 4 was about 17% in both groups. Obese and nonobese subjects all slept significantly (p < 0.01) less in supine position during the second polysomnography. Except for this change in sleeping position, none of the sleep parameters shown in Table 2 differed significantly between the two groups. In addition, none of the women showed significant periodic leg movement.

Sleep-Related Breathing Parameters

Mean awake oxygen saturations were 95.8 ± 0.6% and 95.8 ± 0.6% in the obese subjects and 96.9 ± 0.5% and 96.3 ± 0.4% in the nonobese subjects during the two polysomnographies, respectively. In the latter recording, one obese subject slept 6% of the time (corresponding AHI, 0.2 events per hour), and three obese subjects and one control subject slept 1% of the time at an oxygen saturation < 90%; all other values were > 90%.

The obese woman with preeclampsia was found to have mild obstructive sleep apnea (AHI, 12 events per hour) during late pregnancy. Within 48 h, she was admitted to the sleep laboratory for initiation of treatment with nasal continuous positive airway pressure; however, on admission to the sleep laboratory, she was already in labor.

The main sleep-related breathing characteristics with arousal data are shown in Table 3. The difference in AHI (1.7 events per hour vs 0.2 events per hour, respectively) between the obese and nonobese subjects was significant (p < 0.05), although all indexes were < 10 events per hour. Even more highly significant (p < 0.005) was the difference recorded in ODI_4 (5.3 events per hour vs 0.3 events per hour). Obese subjects snored for one third of their sleeping time during the first recording, whereas control subjects snored only 1% of the time (p < 0.001). The RA indexes differed significantly (p < 0.001), with an index of 7.4 for the obese subjects and 0.8 for control subjects. When irregular breathing was scored as a percentage of REM sleep, non-REM sleep, or total sleep time, at all stages this finding was significantly (p < 0.05) more common in the obese women.

During the second polysomnography, the situation was essentially unchanged: AHI, ODI_4, and snoring time all had increased in the obese women and were

### Table 2—Sleep Characteristics in Polysomnographic Studies Performed During Early and Late Pregnancy in Obese and Control Subjects*

<table>
<thead>
<tr>
<th>Groups</th>
<th>TIB, min</th>
<th>TST, min</th>
<th>WASO, min</th>
<th>REM, %</th>
<th>SWS, %</th>
<th>Body Movement, %</th>
<th>Sleep Efficiency, %</th>
<th>REM Onset, min</th>
<th>REM Latency, min</th>
<th>Sleep Efficiency, %</th>
<th>REM Onset, min</th>
<th>Sleep Efficiency, %</th>
<th>WASO, min</th>
<th>REM, %</th>
<th>SWS, %</th>
<th>Body Movement, %</th>
<th>Sleep Efficiency, %</th>
<th>REM Onset, min</th>
<th>REM Latency, min</th>
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<tbody>
<tr>
<td>Early pregnancy</td>
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<tr>
<td>Obese</td>
<td>498 ± 16</td>
<td>401 ± 20</td>
<td>63 ± 11</td>
<td>21 ± 2</td>
<td>17 ± 3</td>
<td>4 ± 1</td>
<td>29 ± 8</td>
<td>122 ± 30</td>
<td>81 ± 3</td>
<td>14 ± 3</td>
<td>38 ± 5†</td>
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<tr>
<td>Control</td>
<td>502 ± 12</td>
<td>399 ± 17</td>
<td>66 ± 12</td>
<td>20 ± 2</td>
<td>17 ± 1</td>
<td>3 ± 1</td>
<td>37 ± 10</td>
<td>152 ± 19</td>
<td>80 ± 3</td>
<td>14 ± 3</td>
<td>47 ± 7†</td>
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<td>Late pregnancy</td>
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<tr>
<td>Obese</td>
<td>485 ± 14</td>
<td>377 ± 24</td>
<td>76 ± 10</td>
<td>19 ± 2</td>
<td>14 ± 2</td>
<td>3 ± 1</td>
<td>30 ± 12</td>
<td>176 ± 31</td>
<td>77 ± 3</td>
<td>17 ± 2</td>
<td>15 ± 4†</td>
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<tr>
<td>Control</td>
<td>472 ± 17</td>
<td>347 ± 29</td>
<td>77 ± 10</td>
<td>17 ± 2</td>
<td>15 ± 2</td>
<td>3 ± 1</td>
<td>40 ± 12</td>
<td>151 ± 20</td>
<td>73 ± 4</td>
<td>19 ± 2</td>
<td>25 ± 7†</td>
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</table>

*Data are presented as mean ± SEM. TIB = total time in bed; TST = total sleep time; WASO = wakefulness after sleep onset; I-S = inter-sleep wakefulness.
†Difference between early and late pregnancy recording statistically significant within each group.

### Table 3—Sleep-Related Breathing Parameters and Arousal Data in Polysomnographic Studies Performed During Early and Late Pregnancy in Obese and Control Subjects*

<table>
<thead>
<tr>
<th>Groups</th>
<th>AH1, Events/h</th>
<th>ODI_4 Events/h</th>
<th>Snoring, %</th>
<th>Arousal, Events/h</th>
<th>RA Index, Events/h</th>
<th>% REM</th>
<th>% Non-REM</th>
<th>% Total Sleep Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early Pregnancy</td>
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<tr>
<td>Obese</td>
<td>1.7 ± 0.7†</td>
<td>5.3 ± 2.8†</td>
<td>32.0 ± 7.7†</td>
<td>14.9 ± 2.8</td>
<td>7.4 ± 1.7†</td>
<td>71.6 ± 8.4†</td>
<td>27.1 ± 5.9†</td>
<td>36.2 ± 5.8†</td>
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<tr>
<td>Control</td>
<td>0.2 ± 0.1†</td>
<td>0.3 ± 0.2†</td>
<td>1.1 ± 0.6†</td>
<td>10.3 ± 0.8†</td>
<td>0.8 ± 0.3†</td>
<td>43.8 ± 9.3†</td>
<td>5.8 ± 3.1†</td>
<td>13.3 ± 4.2†</td>
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<tr>
<td>Late pregnancy</td>
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<tr>
<td>Obese</td>
<td>2.6 ± 1.5†</td>
<td>8.9 ± 3.1†</td>
<td>48.8 ± 11.9†</td>
<td>20.2 ± 4.4</td>
<td>13.6 ± 4.1†</td>
<td>89.0 ± 4.5</td>
<td>38.6 ± 8.0</td>
<td>48.4 ± 6.7†</td>
</tr>
<tr>
<td>Control</td>
<td>0.1 ± 0.1†</td>
<td>0.5 ± 0.4†</td>
<td>1.2 ± 0.5†</td>
<td>14.6 ± 1.0†</td>
<td>1.4 ± 0.3†</td>
<td>74.0 ± 5.9</td>
<td>20.6 ± 3.3</td>
<td>29.3 ± 3.4†</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SEM. Snoring = percent of sleeping time spent snoring.
†Difference between the groups statistically significant.
‡Difference between early and late pregnancy recording statistically significant within each group.
significantly (p < 0.01) more common. One obese mother had an AHI > 10 events per hour. At this time point, the obese group snored, as a mean, half of their sleeping time. With more advanced pregnancy, the number of arousals increased in both groups. The RA index increased in obese women, with the between-group difference reaching significance (p < 0.001). Irregular breathing increased in both groups, particularly as a percentage of REM sleep in the control group from 44 to 74%. At this time point, the difference in percentage of total sleeping time with irregular breathing was significant (p < 0.02), but differences between the two groups in either REM sleep (p = 0.06) or non-REM sleep (p = 0.054) with irregular breathing were not significant.

The sleep-related breathing parameters and arousal data within the groups at the two assessment points show several significant changes. With advancing pregnancy, the number of arousals only in the control group increased significantly (p < 0.05), but increase in snoring time was statistically significant (p < 0.05) only in the obese group. Increase in RA index in the obese group was also significant (p < 0.05). In the control group, irregular breathing as a percentage of sleeping time increased significantly in REM sleep (p < 0.01), non-REM sleep (p < 0.02), and also when calculated as a percentage of total sleeping time (p < 0.05).

**Hormonal Data**

The estradiol and progesterone levels are shown in Figure 2. At neither level did obese or control subjects show significant difference at any assessment point. The greatest difference was in progesterone level during late pregnancy, 298±39 nmol/L in the obese subjects and 437±57 nmol/L in the control subjects; as expected, within both groups, both hormone levels increased significantly (p < 0.05) between early and late pregnancy.

**Delivery and Newborns**

The babies were born at 274±4 days and at 281±3 days of pregnancy in the obese and control groups, respectively, with no significant difference between the groups. No baby had significant malformations.

Six of the babies were girls in the obese group, and five were girls in the control group. The babies’ weights were 3,506±223 g and 3,622±128 g in the respective groups. Their corresponding lengths were 49.2±0.8 cm and 50.5±0.7 cm. The mother with mild obstructive sleep apnea gave birth to a normal child with a relative body weight of −0.6 SD.24 The mother who spent 6% of her sleep time with an oxygen saturation < 90% in late pregnancy had a baby weighing 3 SD below the mean.

**Follow-up Polysomnography**

A follow-up polysomnography was performed for the two mothers with abnormal findings in the second recording. At 6 months postpartum, sleep-
related breathing had normalized in both mothers, although the mother with mild obstructive sleep apnea during pregnancy still snored 59% of her sleep time.

**Discussion**

Our study found significantly more sleep-related disordered breathing in these obese but otherwise healthy pregnant women than in their normal-weight control counterparts, a finding not explained by differing sleep characteristics or sleep positions. These differences were observed even during early pregnancy and persisted thereafter. Preeclampsia and mild obstructive sleep apnea were diagnosed in one obese woman during late pregnancy, but she gave birth to a normal child. More importantly, one obese mother, who had spent in the latter recording 6% of her sleep time with an oxygen saturation < 90%, gave birth to a baby with growth retardation. Because obesity as a health problem is increasing in women in many parts of the world, these findings may have potentially serious implications for the health of their fetuses.

The sleep parameters in the pregnant women of the present study are in accordance with previous findings. Both groups of women slept more when monitored during early pregnancy, although this was not associated with marked sleepiness as evaluated by the Epworth Sleepiness Scale. As expected, during the last trimester all women spent less time in REM and SWS sleep. Their poorer sleep quality was further manifested in more arousals and more awake time during late pregnancy. However, periodic leg movement was recorded in neither group. As documented previously, both groups slept significantly less often in supine position during the third trimester.

Sleep-related breathing parameters differed significantly between our obese and control groups. The obese subjects had greater AHI and ODI values during early pregnancy, and these values increased, although not significantly, during late pregnancy. Although the means and the great majority of individual AHI and ODI values even in our obese subjects fell within current reference values, no findings and no change occurred in the control group. This pilot study was not planned to look at the effect of other possible risk factors, e.g., craniofacial characteristics, on sleep-related breathing and these issues need to be resolved in future, larger studies. Although our obese mothers were clearly overweight, they were not morbidly obese, and findings may be more prominent in cases of extreme obesity. Furthermore, our methodology with thermal sensors may have underestimated the number of hypopneas, which probably also explains the difference between AHI and ODI values.

Differences in sleep-related parameters between these obese and normal weight pregnant women are reflected even more with regard to snoring. The control group did not snore in either recording period, whereas the obese women spent one third and increasing to one half of their sleeping time snoring. In this study, the polygraph recorded snoring. Unfortunately, to our knowledge no data exist on the correlation between recorded and self-reported snoring. Studies looking at self-reported snoring have found it to be common in pregnancy (23% to 14%), and to be either a risk for growth retardation of the fetus or no risk for the newborn. Thus, the issue of snoring and any links with adverse effects on pregnancy await further studies.

The optimal weight or BMI for women who wish to become pregnant is unknown. That weight gain during pregnancy is poorly correlated with prepregnancy BMI was also reflected in our study. The normal weight group gained more, meaning that weight gain cannot explain the difference observed in sleep-related breathing parameters. However, the obese mothers experienced more complications. Only one obese subject had preeclampsia. Gestational diabetes developed in eight women in the obese group and one woman in the control group. Because gestational diabetes is not known to affect respiration, it is unlikely that the difference in its incidence had an effect on the differences observed in sleep-related breathing parameters. However, a larger study will be needed to clarify conclusively the role of these complications on sleep-related breathing during pregnancy.

The high circulating level of progesterone during pregnancy is suggested to prevent sleep-related disordered breathing by increasing ventilatory drive. The mechanism leading to snoring and sleep apnea in obese pregnant women is still unclear. Whether the smaller rise in serum progesterone levels in our obese women in late pregnancy favored their increased snoring needs further study. However, significant differences in sleep-related breathing parameters were observed at both assessment points.

The sleep-related disordered breathing in these obese mothers offers one possible explanation for the adverse outcomes of pregnancy reported for this group. Further, larger studies are needed to confirm our preliminary findings and to characterize potential additional risk factors.

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