Minute Ventilation and Oxygen Consumption during Labor with Epidural Analgesia

Magnus Hägerdal, M.D.,* Charles W. Morgan, M.D.,† Anne E. Sumner, M.D.,‡ Brett B. Gutsche, M.D.§

Oxygen consumption (\(\text{VO}_2\)) and minute ventilation (\(\text{VE}\)) were measured between and during uterine contractions in the first stage of labor before and after lumbar epidural analgesia (LEA) in 11 women who served as their own controls. \(\text{VO}_2\) and \(\text{VE}\) between contractions were essentially unchanged by LEA to a T10 or higher sensory level. Before LEA, both \(\text{VO}_2\) and \(\text{VE}\) were increased significantly during contractions by 63% and 74% respectively, whereas following LEA there was no significant increase in \(\text{VO}_2\) or \(\text{VE}\) during contractions.

In the second stage of labor, \(\text{VO}_2\) and \(\text{VE}\) were measured in seven patients electing to have no analgesia or sedation and in 10 patients having complete pain relief produced by LEA. Measurements were obtained 5–10 min before delivery. During contractions with pushing, \(\text{VO}_2\) and \(\text{VE}\) were decreased by 25% and 31%, respectively, in patients having LEA as compared with patients having no analgesia or sedation.

These results suggest that the increase in \(\text{VO}_2\) and \(\text{VE}\) are due primarily to pain associated with uterine contractions and that LEA decreased the work of breathing and the oxygen consumption of the parturient in both the first and second stages of labor. (Key words: Anesthesia: obstetric. Anesthesia techniques: epidural. Oxygen consumption. Pregnancy: labor. Ventilation.)

LUMBAR EPIDURAL ANALGESIA (LEA) is a safe and effective means to alleviate maternal pain and anxiety during parturition. It reduces maternal hyperventilation and lactate accumulation.1–4 Sangoué et al.1 found that both LEA and paracervical block decreased oxygen consumption by 14% during the first stage of labor but did not differentiate the effect of these forms of analgesia during and between contractions. They did not investigate the effects of LEA on these variables in the second stage of labor. It has been postulated that the pain of uterine contractions is the primary cause of the increased oxygen consumption and marked hyperventilation during labor.4 This study was designed to quantitate the effects of LEA on both oxygen consumption (\(\text{VO}_2\)) and minute ventilation (\(\text{VE}\)) during and between contractions in the first and second stages of labor.

Methods

Twenty-eight healthy women with uncomplicated pregnancies at term (38–40 weeks gestation) undergoing uncomplicated labor and delivery were studied. Each patient granted informed consent for participation in the study, which was approved by the institutional Human Studies Committee. All parturients receiving LEA had continuous electronic monitoring of the fetal heart rate and uterine contractions. All measurements were made with the patient in a semisitting position with the uterus displaced to the left by means of a roll of sheets elevating the right hip 10–12 cm.

Eleven patients who received LEA were studied in the first stage of labor (Group 1). With the onset of active, painful labor, usually at a cervical dilatation of 4–6 cm, control measurements of \(\text{VO}_2\) and \(\text{VE}\) were obtained between and during contractions. Expired gases were collected with a tightly fitting mask and led to the mixing chamber of a Beckman Metabolic Cart.6 This apparatus contains a turbine for volume measurement, a polarographic oxygen sensor, an infrared carbon dioxide analyzer, a barometer, a thermometer, and a computer that was programmed to give a printout of the calculated \(\text{VO}_2\) and \(\text{VE}\) every 30 s. Measurements were made with left uterine displacement assured and the patient breathing room air. After obtaining this control set of measurements, a catheter was placed in the peridural space at L2-3 or L3-4. Eight to twelve milliliters of 0.25% bupivacaine were injected to produce an upper sensory level of at least T10. When satisfactory pain relief was obtained, \(\text{VO}_2\) and \(\text{VE}\) were measured again. \(\text{VO}_2\) and \(\text{VE}\) were measured continuously for 3–5 min, during which time at least two contractions occurred. As values for \(\text{VO}_2\) and \(\text{VE}\) were computed at 30-s intervals, measurements used for final calculations represented the time during a contraction and the time between contractions. Typically, at least two determinations were recorded during each contraction and four between contractions. In each patient, \(\text{VE}\) varied on the average ±7% and \(\text{VO}_2\) ± 6% from one contraction to the next, and values for \(\text{VE}\) and \(\text{VO}_2\) obtained between contractions varied ±2%. The mean value of the contraction giving the highest \(\text{VO}_2\) and \(\text{VE}\) was chosen. The mean value of two measurements taken just preceding but not including any portion of the contraction was used to calculate \(\text{VO}_2\) and \(\text{VE}\) between contractions.

For the second stage of labor it was not possible to use each patient as her own control before and after initiation

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of LEA. Therefore, in the second stage of labor, seven patients who requested no analgesia or sedation (Group 2) and 10 patients with LEA established (Group 3) were studied. All patients in Group 3 were afforded complete perineal analgesia with 10–12 ml 3% 2-chloroprocaine injected 10–15 min before delivery. The patients in Groups 2 and 3 all were bearing down with contractions, during which time \( \dot{V}O_2 \) and \( VE \) were measured as previously described shortly before delivery.

Changes in \( \dot{V}O_2 \) and \( VE \) in Group 1 patients who served as their own control were evaluated using a two-way analysis of variance with Newman-Keuls test. The differences in \( \dot{V}O_2 \) and \( VE \) between Groups 2 and 3 in the second stage of labor were analyzed using Student’s \( t \) test for unpaired observations. A value of \( p < 0.05 \) was taken as significant.

**Results**

All patients had satisfactory pain relief with a sensory level of T10 to T7 except for one patient with a sensory level of T3. Age, weight, and height of patients in the three groups are given in table 1. Following initiation of LEA in Group 1 patients, both \( \dot{V}O_2 \) and \( VE \) were decreased significantly during contractions by 30% and 45%, respectively (table 2). Between contractions, there was no significant change in \( \dot{V}O_2 \) and \( VE \) after initiation of LEA. Before LEA, both \( \dot{V}O_2 \) and \( VE \) were significantly greater during contractions than between contractions (63% and 74%, respectively). There were no significant differences in \( \dot{V}O_2 \) and \( VE \) between and during contractions after LEA.

The frequency and the intensity of the contractions did not change after epidural anesthesia. One patient was excluded from the study because she stated she had no pain or discomfort during contractions but had requested LEA because she was afraid of having pain as labor progressed. She had no significant decrease in \( \dot{V}O_2 \) or \( VE \) during contractions following LEA.

In the second stage of labor, it was not possible to obtain any reliable measurements of \( \dot{V}O_2 \) and \( VE \) between contractions because the patients usually became apneic for variable periods of time after contractions. Patients with LEA (Group 3) had significantly lower values for both \( \dot{V}O_2 \) and \( VE \) than patients without LEA (Group 2) during contractions (table 3).

**Discussion**

The results of this study confirm the findings of Sangoul _et al._ in that both \( \dot{V}O_2 \) and \( VE \) were decreased following LEA initiated in the first stage of labor. Sangoul _et al._ found, following LEA or paracervical block, a decrease in \( \dot{V}O_2 \) and \( VE \) of 14% and 36%, respectively. During contractions in the first stage of labor, we found a 30% decrease in \( \dot{V}O_2 \) and a 45% decrease in \( VE \). The technique used by Sangoul _et al._ required a 7-min period of measurement, during much of which time the patient was not experiencing pain associated with contractions. By the use of the Beckman Metabolic Cart we were able to obtain values of \( \dot{V}O_2 \) and \( VE \) in 30 s, and thus we could measure these variables both during and between contractions. We found that LEA resulted in no significant changes between contractions, which could explain the greater decrease we found in \( \dot{V}O_2 \) and \( VE \) during contractions. Furthermore, Sangoul _et al._ used epinephrine-

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**Table 1. Physical Characteristics of the Patients**

<table>
<thead>
<tr>
<th></th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>23 ± 7</td>
<td>86 ± 22</td>
<td>163 ± 5</td>
</tr>
<tr>
<td>Group 2</td>
<td>23 ± 3</td>
<td>74 ± 19</td>
<td>161 ± 5</td>
</tr>
<tr>
<td>Group 3</td>
<td>27 ± 5</td>
<td>81 ± 11</td>
<td>166 ± 6*</td>
</tr>
</tbody>
</table>

Values are means ± SD.
* Statistically different from Group 2, \( p < 0.05 \).

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**Table 3. \( \dot{V}O_2 \) and \( VE \) With and Without LEA During Contractions and Pushing in the Second Stage of Labor**

<table>
<thead>
<tr>
<th></th>
<th>( \dot{V}O_2 ) (ml·kg(^{-1})·min(^{-1}))</th>
<th>( VE ) (ml·kg(^{-1})·min(^{-1}))</th>
<th>( n )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 2—no analgesia</td>
<td>6.5 ± 0.9</td>
<td>274 ± 31</td>
<td>7</td>
</tr>
<tr>
<td>Group 3—epidural analgesia</td>
<td>4.9 ± 0.4*</td>
<td>190 ± 19*</td>
<td>10</td>
</tr>
</tbody>
</table>

Values are means ± SEM.
* Statistically significant decrease compared to Group 2, \( p < 0.05 \).

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**Table 2. Group 1: \( \dot{V}O_2 \) and \( VE \) Before and After LEA, During and Between Contractions in the First Stage of Labor**

(Each Patient Served as Her Own Control)

<table>
<thead>
<tr>
<th></th>
<th>( \dot{V}O_2 ) (ml·kg(^{-1})·min(^{-1}))</th>
<th>( VE ) (ml·kg(^{-1})·min(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before LEA (1)</td>
<td>After LEA (2)</td>
</tr>
<tr>
<td>During contractions (a)</td>
<td>4.4 ± 0.6*</td>
<td>3.1 ± 0.4†</td>
</tr>
<tr>
<td>Between contractions (b)</td>
<td>2.7 ± 0.3</td>
<td>2.5 ± 0.2</td>
</tr>
</tbody>
</table>

Values are means ± SEM. Eleven patients were studied.
* Statistically significant decrease compared with “before LEA.” Compares columns 2 to 1 or 4 to 3, \( p < 0.05 \).
† Statistically significant increase in both \( \dot{V}O_2 \) and \( VE \) during contractions as compared with values between contractions before LEA. Compares lines a to b, \( p < 0.05 \).
containing solutions in the nerve blocks they performed. Epinephrine may have increased \( \dot{V}O_2 \)\(^{6}\) after the block in their study, thus giving a smaller difference in \( \dot{V}O_2 \) before and after LEA. Comparing \( \dot{V}O_2 \) and \( \dot{V}E \) during contractions in the second stage of labor, we observed a 25% decrease in \( \dot{V}O_2 \) and a 31% decrease in \( \dot{V}E \) in parturients having LEA compared with those without analgesia, changes quite similar to those observed in the first stage of labor.

Our findings suggest that the pain associated with uterine contractions is the primary cause for the increased \( \dot{V}O_2 \) and \( \dot{V}E \) observed during labor. This assumption is further supported by the lack of changes in \( \dot{V}O_2 \) and \( \dot{V}E \) between and during contractions in the first stage of labor after elimination of pain by LEA. Presumably, pain induces hyperventilation, which increases respiratory work and oxygen consumption. Kambatta and Sullivan\(^{4}\) have shown that respiratory alkalosis per se causes an increase in \( \dot{V}O_2 \). We did not measure arterial blood \( pH \) and therefore cannot evaluate the relative importance of this factor. Both our data and those of Sangoul et al.\(^{1}\) show a marked decrease in \( \dot{V}E \) following LEA, which would lead to a decrease in the work of breathing and could explain the decrease in \( \dot{V}O_2 \). Increased levels of catecholamines also will increase \( \dot{V}O_2 \).\(^{6}\) Sniider and co-workers\(^{8}\) have shown that LEA decreases levels of circulating catecholamines in human parturients during labor. Thus, lumbar epidural analgesia, by decreasing the work of ventilation, the respiratory alkalosis associated with hyperventilation, and the levels of circulating catecholamines, will decrease \( \dot{V}O_2 \).

While this decrease in \( \dot{V}O_2 \) may not be of great significance in the healthy parturient undergoing an uncomplicated delivery, it may be of benefit in the parturient with an inability to increase oxygen delivery, as in one with anemia or limited cardiac reserve. Our findings also would indicate that supplemental maternal oxygen administration during labor could be of benefit in the high-risk pregnant patient where effective analgesia is not employed.

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**References**