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Effect of Epidural Analgesia on Fundal Dominance during Spontaneous Active-Phase Nulliparous Labor

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Background: **The purpose of this investigation was to determine if epidural analgesia, established during active phase labor, results in elimination or reversal of fundal dominance (lower uterine segment pressure equal to or greater than fundal pressure).**

Methods: **Upper and lower uterine segment intrauterine pressures were prospectively evaluated for 50 min before and 50 min after epidural analgesia using 0.25% bupivacaine in 11 nulliparous women in spontaneous active labor. A total of 958 contractions were evaluated.**

Results: **No significant differences were found in the number of contractions in the interval before epidural analgesia compared to after epidural analgesia. Significantly greater pressure readings were recorded in the upper segment than in the lower segment (consistent with fundal dominance) both before and after epidural analgesia ($P < 0.01$). In addition, fundal dominance increased after epidural analgesia when compared to the preanalgesia period ($P < 0.01$).**

Conclusions: **Fundal dominance is present both before and after active phase epidural analgesia and is increased during the immediate 50-min postanalgesia period. (Key words: Analgesia: epidural. Anesthetics, local: bupivacaine. Intrauterine pressure: fundal dominance. Labor: active.)**

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FUNDAL dominance was first described in 1948 by Reynolds and colleagues[1] as a gradient of diminishing physiologic uterine contractile activity from the fundus to the lower uterine segment. This gradient was demonstrated by the placement of external tocodynamometers in three uterine zones. Since then, investigations of uterine activity have focused on intrauterine pressure during labor in an attempt to characterize those patients at risk for labor abnormalities and subsequent Cesarean delivery. These parturients may have either inadequate intrauterine pressure activity,[2] or incoordinate uterine activity as manifested by reversal of fundal dominance.[3] Because dystocia, or abnormal progress of labor, is the most common indication for Cesarean delivery in the United States, efforts to identify those at high risk may influence the rising Cesarean delivery rate.[4]

Epidural analgesia has been associated with prolonged first and second stages of labor, as well as a higher incidence of malposition and the need for operative vaginal delivery.[5] Only two randomized, prospective studies that included nonepidural control groups have been published evaluating the effect of epidural analgesia on labor and delivery outcome. One suggested that epidural analgesia may increase the risk of Cesarean delivery for dystocia, especially if initiated before 5 cm cervical dilation during nulliparous labor.[6] The second did not find a statistically significant increased risk of Cesarean section for dystocia in patients with epidural analgesia compared to the control group.[7] Other authors have advocated epidural analgesia as a method of resolving incoordinate uterine activity and protraction disorders.[8] Also, recent studies evaluating the effect of early *versus* late administration of epidural analgesia on spontaneous and induced/augmented nulliparous labor suggest that neither duration of labor nor mode of delivery are affected.[9,10]

The physiologic mechanisms of labor that may be affected by epidural analgesia have not been investigated completely. The purpose of this investigation was to determine if epidural analgesia, established during spontaneous active-phase nulliparous labor,

results in elimination or reversal of the intrauterine pressure gradient between the upper and lower uterine segments.

Materials and Methods

After obtaining approval of the Committee for the Protection of Human Subjects at the University of Texas Health Science Center and written informed patient consent, 11 parturient women at Hermann Hospital in Houston were enrolled into the study. Inclusion criteria were singleton, term (37 completed weeks gestation), nulliparous patients in spontaneous active-phase labor who were candidates for epidural analgesia, with cephalic presentation and clinically adequate pelvimetry. Exclusion criteria were intraamniotic infection, abruptio placenta, hydramnios, preeclampsia, suspected fetal macrosomia, and systemic or epidural opioid administration. Spontaneous active phase labor was defined as progressive cervical dilation at a rate of 1 cm or more per hour and cervical dilation of ≥ 4 cm and complete effacement at the time of the start of epidural analgesia. No patients received oxytocin infusion before or during the study period.

Under ultrasonographic guidance, two saline-filled intrauterine pressure catheters (Isoflow, Quest Medical, Dallas, TX) were inserted into each patient, one into the fundus and the other into the lower uterine segment. Amniotomy was performed at the time of catheter placement in all patients. Fundal placement was ensured by insertion of the catheter until the mark on the catheter was within the introitus (50 cm from the catheter tip). Lower uterine segment placement was performed by advancing the catheter into the uterus approximately 5–6 cm above the level of the internal cervical os. Each catheter was sonographically confirmed to be in different amniotic fluid pockets. Continuous pressure readings were recorded arbitrarily for 50 min immediately before and after induction of epidural analgesia requested by each parturient. Monitoring equipment used included the Hewlett-Packard 8040A fetal monitor.

Administration of epidural analgesia was performed using a standard protocol. An intravenous infusion of 1,000 ml lactated Ringers solution was administered before epidural placement. With the patient positioned in the left lateral decubitus position, an 18-gauge Tuohy needle was inserted into the L2-L3 or L3-L4 interspace using a midline approach with the loss of resistance technique. The epidural catheter was then inserted. After a test dose of 2 ml 0.25% bupivacaine, a therapeutic dose of 10–12 ml of the same anesthetic agent was administered through the catheter in a divided fashion to provide adequate analgesia (T8–T10) as determined by pinprick. Sensory levels were evaluated every 10 min for the duration of the study period. Maintenance of analgesia throughout the active phase of labor was achieved with a continuous infusion 10–12 ml/h 0.125% bupivacaine. The infusion rate of the local anesthetic was modified to maintain analgesia at a sensory level of T8–T10. Blood pressure was maintained between 80 and 100% of the original level by increasing fluid infusion, and/or ephedrine if necessary.

All contractions were evaluated using the mean active pressure method.[11] This technique converts area under the curve measurements into units of pressure (kilopascals or kPa and mmHg, 1 kPa = 7.5 mmHg). Area under the contraction curves were determined using a commercially available planimeter and software package (Sigma-Scan, version 3.0, Jandel Scientific, Sausalito, CA). Planimeter calibration was performed using a known standard before each use. The mean active pressure method of uterine activity analysis has been used previously to evaluate fundal dominance in both spontaneous and augmented labor.[3] Statistical analysis used included the two-by-two factorial within-subjects repeated-measures analysis of variance.[12] Data are presented as mean ± SD and $P < 0.05$ was considered significant.

Results

The mean cervical dilation at the time of epidural administration was 5.6 cm. After the administration of the epidural block, all patients had adequate analgesia. A total of 958 contractions were evaluated using the method described previously. The mean number of contractions (± SD) per patient before analgesia was not significantly different from the mean number after analgesia (21.73 ± 4.5 compared to 21.82 ± 4.3, P = NS). No parturients in this study had an active phase arrest disorder and in all but one the fetus was delivered vaginally. The one patient of whom the infant was delivered by Cesarean section had an arrest of descent in the second stage of labor. This infant weighed 4,130 g. Clinical estimates of fetal weight on admission did not suggest the presence of macrosomia. Evidence of fundal dominance was demonstrated in this patient both before and after epidural placement and no evi-

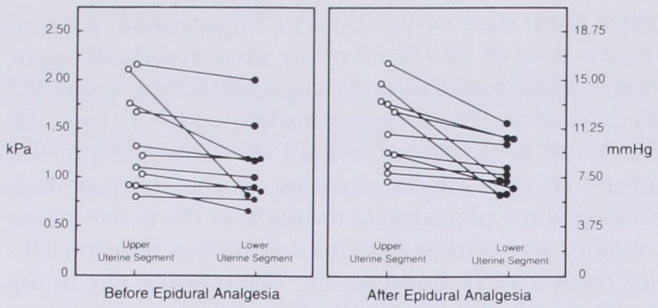

Fig. 1. Fundal dominance before and after epidural analgesia. Mean values of upper uterine segment pressures exceeded those of lower segment pressures both before and after epidural analgesia ($P < 0.01$). Data points for each individual patient are presented in kilopascals and mmHg. $P < 0.05$ is considered significant.

dence of active phase protraction or arrest disorder was noted.

Figure 1 displays upper uterine segment mean active pressures compared to lower uterine segment mean active pressures in kPa and mmHg. Mean intrauterine pressure measurements in the upper uterine segment exceeded lower uterine segment pressures both before and after epidural analgesia (before analgesia: 1.35 ± 0.47 kPa, 10.13 ± 3.53 mmHg *vs.* 1.09 ± 0.39, 8.18 ± 2.93 mmHg, $P < 0.01$; after analgesia: 1.49 ± 0.39 kPa, 11.18 ± 2.93 mmHg *vs.* 1.12 ± 0.26 kPa, 8.40 ± 1.95 mmHg, $P < 0.01$). Therefore, fundal dominance is demonstrated before and after epidural analgesia. No patient "lost" fundal dominance during the 50 min after the induction of epidural analgesia.

Data comparing the effect of epidural analgesia on fundal dominance are summarized in figure 2. Fundal dominance is significantly increased after epidural analgesia compared to the interval evaluated before analgesia ($P < 0.01$). This increase caused by a greater differential increase in fundal segment pressures compared to lower segment pressures after epidural analgesia (0.14 kPa, 1.05 mmHg *vs.* 0.03 kPa, 0.23 mmHg). No elimination or reversal of the intrauterine pressure gradient is demonstrated.

Discussion

In 1950, Alvarez and Caldeyro-Barcia introduced direct measurement of intrauterine pressure *via* a transabdominally placed catheter.[13] Subsequently, in 1953, Hoff and Bayer performed direct intrauterine measurement of uterine activity in two regions of the uterus.[14] These investigators found that contractions of the uterine fundus were stronger than those of the isthmus. Since then, the number of patients evaluated using intrauterine pressure measurement in multiple regions of the uterus has been limited.[3]

In contrast to this limited work, many data have been published using external tocodynamometers to describe the "triple descending gradient" or "fundal dominance" that is demonstrated in normal spontaneous labor.[1,15,16] The normal contractile activity required for spontaneous vaginal delivery in nulliparous labor also has been evaluated by several investigators.[17–19] Cowen and colleagues[19] have demonstrated a strong correlation between uterine activity measured in Montevideo units and uterine activity measured in the same patients *via* the mean active pressure method. Although the values required to achieve cervical dilation may vary widely, it is likely that during active phase labor, a minimum of 1.33 kPa or 9.98 mmHg (approximately 180 Montevideo units) is associated with a cervical dilation rate of >1 cm/h.[18] In addition, a minimum of 2.0 kPa or 15 mmHg (200–224 Montevideo units) has been suggested to fully test the cephalopelvic relationship before the diagnosis of active phase arrest.[17,19] Upper uterine segment mean active pressures in the current study population are consistent with these findings and are confirmed by the clinically normal, spontaneous active phase labor.

With the use of two intrauterine pressure catheters, Margono and colleagues[3] demonstrated that parturients with active phase arrest disorders had lower segment pressures that exceeded upper segment pressures at the time that the arrest disorder was clinically diag-

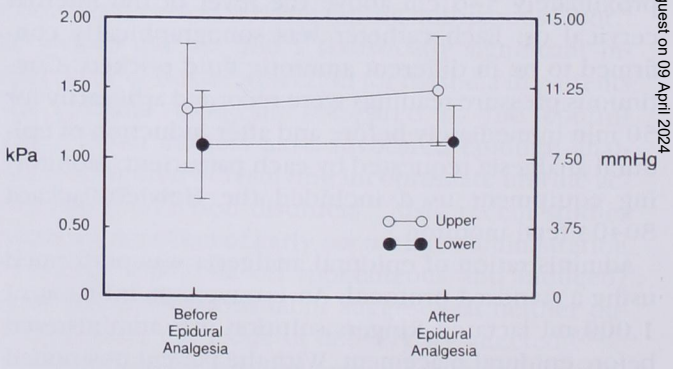

Fig. 2. The effect of epidural analgesia on fundal dominance. A significant increase in fundal dominance is demonstrated following epidural analgesia ($P < 0.01$). Data presented as mean ± SD in kilopascals and mmHg. $P < 0.05$ is considered significant.

nosed (reversal of fundal dominance). In addition, oxytocin augmentation was not found to correct this abnormal pressure gradient. They suggested that the use of two intrauterine pressure catheters in parturients with active phase arrest disorders may provide clinicians with information about hypotonic uterine activity as well as the presence or absence of fundal dominance.

To maintain fundal dominance, spontaneous rather than induced or augmented labor may be important, because patients who require oxytocin for induction or augmentation in early labor are at an increased risk of Cesarean section for dystocia.[17] Therefore, any effect of regional analgesia on fundal dominance should be evaluated separately in patients with spontaneous labor from those requiring oxytocin before analgesia. Cervical/lower uterine segment compliance also may contribute to the persistence of fundal dominance throughout labor. Gee and associates[20] and Olah and colleagues[21] suggest that the greater the cervical compliance, the lower cervical/lower uterine segment wall tension will be, thereby increasing the intrauterine pressure gradient.

Studies by Chestnut and colleagues[9,10] appear to refute the possible adverse effects of early epidural analgesia when compared to delayed epidural analgesia in nulliparous patients during spontaneous or induced/augmented labor. Based on our data, epidural analgesia using 0.25% bupivacaine followed by a continuous infusion of 0.125% bupivacaine, and established during active phase labor, does not eliminate fundal dominance. In contrast, these data show that fundal dominance increases during the immediate 50-min interval after epidural analgesia. Finally, because different agents and concentrations used to provide epidural analgesia during labor produce markedly variable analgesic and clinical effects on labor,[22,23] the effect of each on the course of labor and mode of delivery should be evaluated separately. Patients receiving opioids were excluded because of possible effect on the course of labor.[24]

The current study evaluated only those patients in active phase labor, and did not determine normal intrauterine pressure gradients throughout spontaneous labor without analgesia. Therefore, no comment can be made about the effect of epidural analgesia on intrauterine gradients compared to those generated in the absence of analgesia. Normal ranges of intrauterine pressure gradients for latent and active phase labor need to be measured in a large number of patients before it can be determined if epidural analgesic agents affect this gradient compared to patients without analgesia. In addition, determining and standardizing the exact distance between intrauterine pressure measuring points needs consideration because although sonography determined that each catheter was placed in upper and lower uterine segments, respectively, this exact distance was not determined and was probably not the same from one patient to the next. This point may be one explanation for the range of gradients seen in the current study.

In conclusion, the data presented in our study suggest that during the 50-min interval immediately after epidural analgesia in patients in spontaneous active phase labor, no elimination or reversal of intrauterine pressure is demonstrated. Fundal dominance is present both before and after epidural analgesia and is increased during the immediate postanalgesia interval. Finally, further studies using the aforementioned technique may be useful in evaluating the physiology of the entire labor course with and without analgesia.

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