

Title : SPREAD OF EPIDURAL ANALGESIA IN EARLY PREGNANCY

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**Introduction.** Increased spread of epidural analgesia has been reported for full term pregnant patients when compared to non-pregnant women.<sup>1</sup> This observation has been challenged in studies where dose requirements of pregnant and non-pregnant females were found to be similar.<sup>2,3</sup> In contrast we have observed a facilitated epidural spread even during the first trimester. To further document this unexpected finding the following study was undertaken.

**Methods.** Levels of epidural analgesia were studied in 23 women in early pregnancy scheduled for abortion, and compared with a non-pregnant control group (n = 12) scheduled for elective surgical procedures. The mean age of the pregnant patients was 26.0 years (range 18-45) and gestational age was 8-12 weeks as determined by date and size of fetus. The mean age of the control group was 26.3 years (range 18-35). Each patient had requested epidural analgesia for her operation. Included in the study were only women who were free of neurological disease, local infection, sepsis, bleeding abnormalities and not on subcoagulation heparin prophylaxis. No sedation was given. With the patient in the lateral position epidural analgesia was induced with a standard technique. The epidural space was entered with a 18 gauge Tuohy needle at the second lumbar interspace (L2-L3) and identified with loss of resistance. In all cases the local anesthetic consisted of 2% lidocaine with epinephrine 1:200,000 (commercially premixed): total dose was 20 ml. The patient was maintained in the lateral position and the bevel of needle pointed cephalad. A 2 ml test dose of the lidocaine was injected, followed 2 minutes later by the remaining 18 ml. The rate of injection was approximately 1 ml/sec. Immediately thereafter, the patient was placed in the supine position. Spread of analgesia was tested and recorded in 5 minute intervals until no further spread was observed. Loss of sensation was tested by using pin-scratch with a safety-pin; always moving from an area of expected hypalgesia to an area of normal sensation. In addition, sensitivity to cold was tested with alcohol swabs and cold metallic objects. The skin was marked at the point at which the needle produced a sharp painful sensation and cold objects were appreciated without further change of quality. Examinations always started near the ventral midline and were repeated and extended laterally. Maximal spread of analgesia for pin-scratch and cold was recorded on dermatomal charts. The time for complete spread was also noted. Data were analyzed using Student's t-test for unpaired samples.

**Results.** Levels for pin-scratch and cold always coincided. The lower level of analgesia included all sacral segments in every patient. In early pregnant patients the average upper level included

the 4th thoracic dermatome (range Th<sub>2</sub>-Th<sub>7</sub>). This corresponded to a mean dose of 21.3 mg lidocaine per segment. In the control group the mean upper level was Th<sub>8</sub> (range Th<sub>6</sub>-Th<sub>11</sub>) which corresponded to a mean dose of 27.1 mg lidocaine per segment. Thus, the pregnant group demonstrated a 28.8% higher spread, or a lower dose requirement per segment than the control group; the differences were significant (p<0.001). The mean time to reach maximal spread was 28.3 minutes (range 20-40) in the pregnant group whereas in the control group maximal spread was complete after 20.5 minutes (range 15-30). The time difference was also significant (p<0.001). The average rate of epidural spread, i.e. segments blocked per unit time, was similar in both groups and amounted to 0.67 and 0.68 segments per minute in pregnant and non-pregnant patients, respectively.

**Discussion.** Our results demonstrate a facilitated spread of epidural analgesia during the first trimester that is comparable in magnitude with the spread previously reported in patients near term.<sup>1</sup> Exaggerated spread near term has been explained on a mechanical basis due to distention of the epidural veins caused by partial obstruction of the inferior vena cava by the gravid uterus. Indeed, in dogs acute obstruction of the inferior vena cava has been shown to enhance the spread of epidurally injected contrast material.<sup>4</sup> Additionally, uterine contractions may massage more blood into the epidural veins. In the first trimester, however, these hemodynamic factors are not likely to play a role. We suggest that our observations may be explicable in terms of some other cause, and that for instance hormonal factors be considered. This study confirms that segmental spread of analgesia from epidural lidocaine may not be complete before 40 minutes. Hence, measurements of total spread are likely to be erroneous if they are completed within a shorter time frame. Our study also shows that the rate of segmental spread is similar in the two groups. We conclude that a facilitated spread of epidural analgesia in pregnant women appears to be present already during the first trimester.

#### References.

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