none was found. In addition a second manual exploration did not produce hypertension. Because this period of hypertension correlated well with the autotransfusion, we suspected that this blood contained catecholamines, and were able to document their presence in extremely high levels (>10,000 times normal). The patient received a smaller amount of catecholamines than would be suggested by this assay because blood from the collection chamber was centrifuged, washed with saline, and centrifuged again before administration.

In summary, we suggest that catecholamine transfusion is a potential complication of autotransfusion during surgery for pheochromocytoma. Additional wash cycles before erythrocyte transfusion should decrease the catecholamine concentration. Hypertension should be anticipated with the use of autotransfusion in these individuals and could confuse the search for additional tumors.

REFERENCES

Spread of Epidural Analgesia in Early Pregnancy

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The mechanism for the facilitated spread of epidural analgesia in pregnant women at term has not been elucidated. Elevated intraabdominal pressure and inferior vena cava compression from the pregnant uterus have been proposed as likely causes during labor. Both factors may redirect part of the venous return via the vertebral system of veins leading to changes in epidural volume and pressure. In addition, the exaggerated lumbar lordosis of pregnancy may contribute to the increased cephalad spread of the anesthetic solution. We have observed a facilitated epidural spread in pregnant women even during the first trimester, at a time when mechanical factors are unlikely to play a significant role. The purpose of this study was to further document these findings.

MATERIALS AND METHODS

The study was approved by the Duke University Clinical Investigations Committee. Levels of epidural analgesia were studied in 37 women: 12 non-pregnant controls, and 23 patients pregnant in their first trimester (range 8-12 weeks). All patients were ASA physical status 1 and scheduled to undergo elective abortion or other gynecologic procedures. Each patient had requested epidural analgesia for the operation, was free of neurologic disease, local infection, sepsis, and bleeding abnormalities, and was not receiving heparin prophylaxis. No sedation was given. A standardized epidural technique was used in all cases. The epidural puncture was performed with an 18-gauge Tuohy needle at the second lumbar interspace with the patient in the lateral position. The epidural space was identified by the loss-of-resistance technique with an air-filled syringe. A 2% lidocaine solution premixed with epinephrine 1/200,000 was injected in two doses; a 2-ml test dose was followed after 1 to 2 min by 18 ml of the
remaining solution. The injection rate was kept at approximately 1 ml/s. Thus, each patient received 400 mg of lidocaine over a 3-min period. Immediately after the epidural injection, each patient was placed in the supine position.

Spread of analgesia was determined in 5-min intervals for 30–40 min. Maximum spread was defined as being achieved when dermatomal levels remained unchanged for two consecutive examinations or when 40 min had elapsed. Loss of sensation was tested using pin-scratch, always moving from an area of expected analgesia to an area of normal sensation. The skin was marked at the point at which the needle produced a sharp and painful sensation without further change of quality. Similarly, sensitivity to cold was tested with alcohol swabs, ice, or cold metallic objects. Examinations always started near the ventral midline and were repeated about every 2 inches moving laterally. Dermatome levels obtained for every 5-min period were recorded on dermatomal charts. Dose requirements per spinal segment were calculated. Data were analyzed with Student’s t test for unpaired samples and analysis of variance.

**RESULTS**

The average age of the control group was 26.3 ± 6.2 years (mean ± 1 SD), and the corresponding value for the pregnant group was 26.0 ± 6.4 years. The average heights for the two groups were 169.3 ± 5.8 cm and 163.2 ± 6.1 cm, respectively. Thus, both groups happened to be matched for age, but the non-pregnant group was significantly taller than the pregnant group (P < 0.001).

At completion of epidural anesthesia, dermatome levels tested with pin-scratch and cold always coincided; the lower level of analgesia included all sacral segments in every patient. In the pregnant patients, the average upper level of analgesia reached the fourth thoracic dermatome (mean 4.0 ± 1.8) and was significantly higher (28.8%) than the eighth thoracic dermatome in the control group (mean 8.1 ± 1.3, P < 0.001). The average epidural dose requirement per spinal segment in the pregnant group was 21.3 ± 2.1 mg lidocaine per segment, and the corresponding value for the control group was 27.1 ± 2.4 mg (P < 0.001).

The caudad spread of analgesia showed a similar pattern in both groups, with sacral analgesia being complete within 10 min in all patients. However, cephalad spread initially was faster in the pregnant group, and had reached significantly higher levels already 5 min after injection of the 2% lidocaine (P < 0.001). Further cephalad spread was parallel to that of the control group (fig. 1). The time to complete dermatomal spread, i.e., the time when the final dermatomal level was first reached, in the pregnant group was 28.3 ± 8.4 min (range 20–40), and in the control group, 20.5 ± 5.9 min (range 15–30). This difference proved to be significant by Student’s t test (P < 0.001) and analysis of variance (P = 0.0037).

**DISCUSSION**

Our results demonstrate a facilitated spread of epidural analgesia during the first trimester comparable in magnitude with the increased spread reported for patients at term.1–3 Time to achieve full segmental spread was increased significantly in our pregnant patients when compared with the control group (28.3 min vs. 20.5 min). This longer time to reach the final levels of analgesia during pregnancy may explain why some other studies using shorter observation periods failed to confirm an increased epidural spread in patients at term.6,7 Surprisingly, significantly higher levels of epidural analgesia were observed in our early pregnant patients as early as 5 min after induction (fig. 1). This finding could be explained by the observed difference

![Graph showing analgesia spread](image-url)
of 6.1 cm in patient heights. However, there is only a weak correlation between patient height and epidural segmental dose. For a 6.1-cm difference in height, the epidural dose per segment may be expected to increase by 0.06 ml of the 2% solution or 0.12 mg. We demonstrated a 5.9-mg difference in dose requirement per segment which, thus, is not likely to be explained by the observed difference in height between the groups.

Another possible explanation for this early facilitated spread may be venous distension secondary to the increased blood volume associated with pregnancy. However, blood volume changes during the first trimester are small, and the main increase occurs later in pregnancy. Therefore, although venous distension may contribute to the higher epidural levels in our pregnant group, it seems insufficient to explain a spread that is similar in magnitude to that observed at full term.

A non-mechanical explanation may be provided by the hormonal changes present already during the first trimester. For instance, dose requirements for inhaled anesthetics are decreased during both early and late phases of pregnancy. Since progesterone and other steroids have known anesthetic properties, perhaps the increased hormone levels during pregnancy are responsible for this phenomenon. Presuming the applicability of these observations to local anesthetics in the epidural space, a decrease in dose requirement per spinal segment could be possible during pregnancy. Hormonal mechanisms, however, fail to account for the observed prolonged time to complete spread of analgesia in the pregnant group. Instead, the apparent parallel spread of epidural analgesia after the first 5 minutes implies similar uptake mechanisms for both pregnant and non-pregnant groups. Therefore, it seems unlikely that hormones have significant additive effects on local anesthetics during pregnancy.

The facilitated spread of epidural analgesia at term has been attributed to a number of mechanical factors. Increased intraabdominal pressure during labor, a space occupying and massaging effect by distended epidural veins secondary to compression of the inferior vena cava and pelvic veins, increased epidural pressures in the parturient patient, and an exaggerated lumbar lordosis at term have all been implicated. Recently, however, uterine contractions have been reported not to influence the spread of local anesthetic solutions in the epidural space. Furthermore, mechanical factors apparently do not play important roles during the early phase of pregnancy. This view is supported further by a recent report on delayed pharmacokinetics of intravenous lidocaine during pregnancy.

Changes in distribution volumes and an increased half-life of the post-distributive phase for intravenous lido-

caine led to a slower decline in blood levels in pregnant sheep as compared with a non-pregnant control group. Therefore, we propose a biochemical explanation for the exaggerated epidural spread of local anesthetics during early pregnancy.

Pregnancy induces a state of hyperventilation beginning early in the first trimester. The lowered carbon dioxide tension leads to an alkalosis which is compensated by lowered bicarbonate levels. Local anesthetic agents are salts that need buffer to convert to the base form for transport across membranes into CNS and circulation. Decreased buffer capacity may allow the local anesthetic to remain a salt for a longer time and, therefore, stay longer in the area of injection (epidural space). This by itself will increase time to complete analgesia. In addition, the local anesthetic will have time to spread farther within anatomic tissue planes, and further increase dermatomal spread. Thus, the decreased buffer capacity during pregnancy may explain both increased epidural spread and increased time to achieve it.

We conclude that first trimester pregnancy is associated with an increased epidural spread of local anesthetics similar to that seen in pregnant women at term. Time to complete spread is prolonged. We suggest that biochemical changes of pregnancy, especially the decreased buffer capacity, may account for these observations.

REFERENCES


A Hazard of Continuous Flush Systems for Vascular Pressure Monitoring in Infants

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Indwelling arterial and central venous catheters have become indispensable in the care of critically ill patients for continuous monitoring of cardiovascular function and for rapid procurement of blood samples. Systems designed to deliver approximately 3 ml/h of a heparinized saline solution are used frequently to prevent clotting and consequent loss of catheter patency (fig. 1).1,2 However, exact quantitation of fluid delivered by this system is difficult, which may complicate fluid management in small children. A worrisome complication during a recent case stimulated an investigation of the accuracy of fluid delivery for four commonly used brands of continuous flush devices.

REPORT OF A CASE

An 18-month-old 11-kg boy was scheduled for repair of a large ventricular septal defect and a patent foramen ovale. After sedation with rectal methohexital, a radial arterial catheter and an internal jugular central venous pressure (CVP) catheter were inserted percutaneously without difficulty. Both the arterial and CVP catheter were connected to pressure transducers and to continuous flush systems that included a pressure bag inflated to 300 mmHg, a 250-ml bag of 0.9% saline with 500 units of heparin, intravenous tubing, and a continuous flow device designed to administer 3 ml/h (Continuous Flush Device, American Pharmaceutical Laboratories, Glendale, California) (fig. 1). Surgical repair was accomplished successfully, and the patient was transported to the Pediatric Intensive Care Unit (PICU) where the continuous infusion systems for both the arterial and CVP catheters seemed to be working well; both pressure bags were inflated properly to 300 mmHg. Approximately 4 hours after admission to the PICU and 8 hours after the arterial and CVP catheters were inserted, the CVP flush solution bag was noted to be empty. The patient had received an excess of approximately 225 ml of fluid through the CVP catheter. Furosemide, 0.5 mg/kg, iv, was administered, resulting in diuresis of 160 ml urine over the next hour. A new continuous flush system was connected to the still-perfused CVP catheter. The new infusion system functioned normally, and the patient had an otherwise benign postoperative course.

The continuous flush device, when tested in vitro with a system similar to that shown in figure 1, delivered 25 ml/h.

METHODS

Twenty continuous flush devices of each of four commonly used brands† were attached to flush systems consisting of an intravenous infusion set and a 250-ml bag of normal saline pressurized to 300 mmHg with a pressure bag. The distal end of the continuous flow device was placed in a graduated cylinder, and flush solution was collected over an eight-hour period. An average hourly flow rate for each continuous flush device was calculated. The rapid flush mechanism was activated for 10 s, and flush solution was collected and measured; the flow rate per second was calculated for the rapid flush mechanism of each continuous flush device.

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