

MECHANISM OF EXTENSION OF SPINAL ANAESTHESIA BY EXTRADURAL INJECTION OF LOCAL ANAESTHETIC

C. H. BLUMGART, D. RYALL, B. DENNISON AND L. M. THOMPSON-HILL

SUMMARY

We have examined the effect of extradural injection of 0.5% bupivacaine or normal saline on the progression of spinal anaesthesia in 28 patients undergoing Caesarean section. Three groups were studied. Subarachnoid anaesthesia was established in all patients. Group A (n = 10), the control, received no extradural injection for 20 min. Group B (n = 9) received extradural bupivacaine 10 ml and group C (n = 9) received extradural saline 10 ml 5 min after the subarachnoid injection. Sensory levels were compared at 5-min intervals and extension of the block was found to be similar in groups B and C and significantly faster than the control ($P < 0.05$). The quality of anaesthesia and incidence of adverse effects was similar for all three groups. We conclude that the mechanism of extension of spinal anaesthesia by extradural injection of local anaesthesia is largely a volume effect. (Br. J. Anaesth. 1992; 69: 457-460)

KEY WORDS

Anaesthesia: obstetric. Anaesthetic techniques, regional: extradural, spinal.

The combined spinal-extradural technique of providing regional anaesthesia for Caesarean section is becoming increasingly popular. Subarachnoid injection of local anaesthetic provides rapid onset and reliable sacral block, whilst the presence of an extradural catheter allows flexibility in extending the block and provision of postoperative analgesia [1-4].

The injection of a relatively small amount of 0.5% bupivacaine into the extradural space after a subarachnoid injection of a standard dose of bupivacaine results in rapid extension of the block [5, 6]. Both the degree and speed of onset of this extension cannot be adequately explained solely by the presence of a small volume of local anaesthetic within a localized area of the extradural space.

Several hypotheses have been advanced to explain this enhancement of the subarachnoid block. These include leakage of extradural bupivacaine into the subarachnoid space via the hole created by the subarachnoid puncture, the existence of a "subclinical" analgesia at a higher level which is enhanced and becomes evident by perineural or transdural spread of the extradural solution, or the continuing spread of the initial subarachnoid injection independent of the extradural injection [4-7].

The extradural injection, by causing compression of the subarachnoid space and spread of the local anaesthetic within, may be another mechanism to explain this effect. This study was designed to compare the effect of extradural injection of normal saline or bupivacaine with a control group receiving no extradural injection on the level of the subarachnoid block.

PATIENTS AND METHODS

We studied 28 patients undergoing elective Caesarean section under regional anaesthesia. All were at term and none had any medical or obstetric complications. The investigation was approved by the Ethics Committees of Newcastle and South Tees Health Authorities and informed consent was obtained from all patients.

All patients received oral ranitidine 150 mg 2 h before operation, 30 ml oral sodium citrate 0.3 mol litre⁻¹ on leaving the ward and a standard preload of 1500 ml of warmed Hartmann's solution i.v. With the patient sitting, the extradural space was located via the L2-3 or L3-4 interspace with a 16-gauge Tuohy needle (Portex), using loss of resistance to air. A 26-gauge spinal needle was inserted through the Tuohy needle and the subarachnoid space identified by the appearance of CSF at the hub of the needle. Using a technique similar to that described by Rawal [6], 0.5% hyperbaric bupivacaine was injected—1.6 ml if the patient was less than 163 cm tall and 1.8 ml if they were 163 cm or taller. The spinal needle was removed and a catheter inserted 3 cm into the extradural space.

The patient was then placed supine with a 15° left tilt. Care was taken to avoid caval compression as this has been shown to influence the spread of subarachnoid local anaesthetic [8, 9]. An i.v. bolus of ephedrine 6 mg was given as prophylaxis against hypotension. Oxygen was administered by face mask and ECG, SpO₂ and arterial pressure monitored throughout the time of the study.

C. H. BLUMGART*, M.B., B.CH., F.R.C.ANAES.; D. RYALL, M.B., CH.B., F.R.C.ANAES.; B. DENNISON, M.B., CH.B., F.R.C.ANAES.; Department of Anaesthetics, South Cleveland Hospital, Marton Lane, Middlesbrough. L. M. Thompson-Hill, M.B., B.S., F.R.C.ANAES., Department of Anaesthetics, Princess Mary Maternity Hospital, Newcastle Upon Tyne NE2 3BD. Accepted for Publication: June 3, 1992.

*Present address, for correspondence: Department of Anaesthetics, Freeman Hospital, High Heaton, Newcastle Upon Tyne NE7 7DN.

TABLE I. Patient characteristics and surgical details (mean (range or SD)). No significant difference between groups

	Group A (Control) (n = 10)	Group B (Bupivacaine) (n = 9)	Group C (Saline) (n = 9)
Age (yr)	28.6 (21-37)	29.8 (22-35)	28.7 (22-42)
Weight (kg)	74.7 (5.7)	69.3 (6.7)	74.0 (7.3)
Height (cm)	161.8 (6.0)	163.4 (6.6)	162.6 (4.3)
Time to start of surgery (min)	36.5 (4.7)	32.2 (2.6)	31.7 (2.5)
Duration of surgery (min)	50.0 (4.1)	46.7 (5.0)	45.6 (9.2)
Time to first request for analgesia (min)	162.5 (48.7)	133.89 (22.3)	156.1 (64.0)

The patient was then allocated randomly to one of three groups: group A (control; 10 patients): no extradural injection within 20 min after the subarachnoid injection; group B (bupivacaine; nine patients): extradural injection of 0.5% bupivacaine 10 ml 5 min after the subarachnoid injection; group C (saline; nine patients): extradural injection of normal saline 10 ml 5 min after the subarachnoid injection.

The extradural injection of 10 ml at the 5-min interval was administered over 20 s by an operator blinded to the composition of the injection. Twenty minutes after the subarachnoid injection, the level of sensory block was extended to T4 or T5 by extradural injection of 0.5% bupivacaine if required.

The level of sensory block was tested by pinprick every 5 min and at the time of the first incision. Motor function in the lower limbs was tested at 5-min intervals and graded according to the Bromage classification (grade 4 = full power; grade 1 = no power) [10].

If the patient experienced pain or discomfort, analgesia was supplemented with one or more i.v. boluses of alfentanil, progressing to general anaesthesia if required. Surgical analgesia was assessed by the anaesthetist and graded as excellent if no supplementation was required, good if one dose of alfentanil was administered with good effect, fair if more than one dose was required or poor if general anaesthesia was necessary. The patient was asked to grade her experience subjectively on the same scale.

Any adverse event during operation was recorded. Hypotension was defined as a systolic arterial pressure less than 100 mm Hg or a decrease of 30% from baseline value, and was treated with 6-mg i.v. boluses of ephedrine.

The condition of the baby was assessed at birth and 5 min (Apgar score).

After operation, time to first request for analgesia was noted and the woman questioned about the presence of backache, headache, urinary problems and any delay in mobilization. Patients were asked if they would choose the technique again.

Statistical analysis was performed using the Fisher exact, Mann-Whitney *U* and Student's *t* tests as appropriate. Significance was assumed at the 5% level ($P < 0.05$).

RESULTS

The three groups were comparable in age, weight and height (table I).

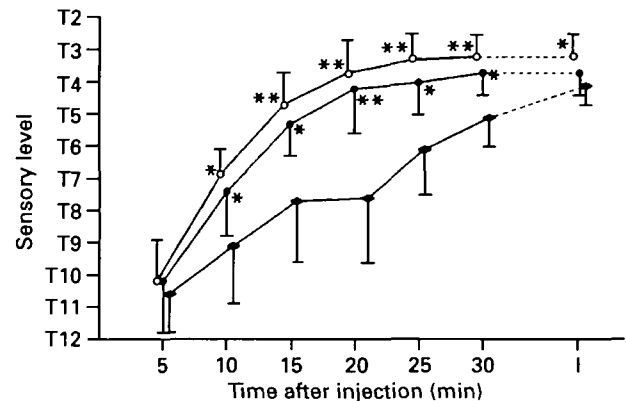


FIG. 1. Onset of sensory block (mean, SD). Group A, control (◆) received extradural bupivacaine at 20 min; group B (●) received extradural bupivacaine at 5 min; group C (○) received extradural saline at 5 min. I = First incision. * $P < 0.05$; ** $P < 0.01$ compared with control. No significant difference between groups B and C.

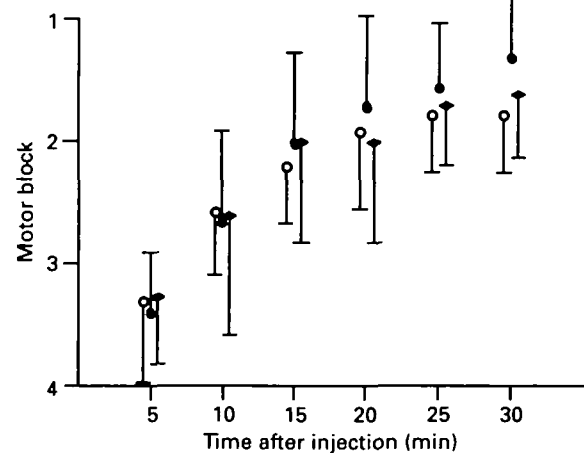


FIG. 2. Onset of motor block (mean, SD) (Bromage scale). Group A, control (◆) received extradural bupivacaine at 20 min; group B (●) received extradural bupivacaine at 5 min; group C (○) received extradural saline at 5 min. No significant difference between groups.

Levels of sensory blocks during operation are shown in figure 1. At 5 min, the levels in the three groups were similar. Progression of the level was rapid in both groups receiving extradural injections at 5 min and was significantly slower in the control group and had stabilized by 20 min. At this time, eight patients in group A, two in group B and one in

TABLE II. Total doses of bupivacaine and volumes injected into the extradural space (mean (SD) [range]). * $P < 0.05$ significant difference compared with control; ** $P < 0.01$ significant difference compared with group B

	Group A (Control) (n = 10)	Group B (Bupivacaine) (n = 9)	Group C (Saline) (n = 9)
Subarachnoid bupivacaine (mg)	8.5 (0.5)	8.7 (0.5)	8.4 (0.3)
Extradural bupivacaine (mg)	30.5 (21.2) [0-65]	54.4 (9.2) [50-75]*	1.7 (5.0) [0-15]***
Extradural volume (ml)	6.1 (4.2)	10.9 (1.8)*	10.3 (1.0)*
Final sensory level (thoracic dermatome)	4.1 (0.6)	3.7 (0.7)	3.2 (0.7)*

TABLE III. Incidence of adverse events. No significant difference between groups

	Group A (Control) (n = 10)	Group B (Bupivacaine) (n = 9)	Group C (Saline) (n = 9)
Hypotension	2	4	5
Pain	1	0	1
Discomfort	3	3	2
Nausea	1	2	2

TABLE IV. Quality of anaesthesia (see text). No significant difference between groups

	Group A (Control) (n = 10)	Group B (Bupivacaine) (n = 9)	Group C (Saline) (n = 9)
Anaesthetist			
Excellent	6	6	5
Good	2	2	2
Fair	1	1	2
Poor	1	0	0
Patient			
Excellent	6	6	5
Good	3	2	3
Fair	0	1	1
Poor	1	0	0

group C required further extradural bupivacaine to extend the block to T4 or T5 ($P < 0.05$).

The degree of motor block (fig. 2) was not related to height of sensory block or speed of progression and there was no significant difference between the groups.

The saline group received significantly less extradural bupivacaine to achieve comparable sensory levels, the final sensory level being related closely to the volume injected into the extradural space (table II).

Time to the first incision, duration of surgery and time to first request for analgesia were similar for all three groups (table I).

The incidence of adverse events is summarized in table III. Hypotension occurred more frequently in groups B and C (ns).

The number of patients requiring alfentanil supplementation was similar in all groups (table IV). One patient in the control group, despite having loss of pinprick sensation to T4, required general anaesthesia because of severe pain.

All babies were vigorous at birth and had an Apgar score of 8 or more at 5 min.

After operation, there was no difference between the groups in the incidence of backache, headache, urinary problems and delay in mobilization. No post-spinal headaches occurred. One patient in the control group would not choose the same technique again.

DISCUSSION

It is well recognized that alterations in the contents of the spinal canal can influence the spread of local anaesthetic. It is thought that one of the reasons for the pregnant patient's decreased requirement of local anaesthetic agent for spinal anaesthesia in the latter stages of gestation is an increased vertebral venous volume and a reciprocal decrease in CSF volume at the caudal end of the subarachnoid space [7-9]. Compression of the inferior vena cava in non-pregnant patients, causing vertebral venous engorgement, increases the spread of subarachnoid local anaesthetic [8]. It has been shown that spread of subarachnoid local anaesthesia is slow in pregnant patients who are kept in the lateral position, with rapid progression when turned into a supine wedged position [11]. Hilt, Gramm and Link noted increases in intracranial pressure in head injured patients receiving lumbar extradural injection of local anaesthetic and similar increase with extradural injection of saline [12].

The evidence would suggest that compliance within the vertebral canal is dependent on a balance between vertebral blood volume and CSF volume. The injection of local anaesthetic or of any other fluid into the extradural space may be expected to result in initial shifts of one or both of these components before the fluid leaks away via the intervertebral foramina or is absorbed into the blood stream or CSF.

It has been noted that relatively small doses of extradural local anaesthetic, injected in the lumbar region, are required to extend a "fixed" subarachnoid block from T8 to T3-4 [5, 6]. The short space of time in which the extension occurs suggests a mechanism other than diffusion of the anaesthetic into the CSF or block of nerve roots within the extradural space.

This study has shown that rapid extension of spinal anaesthesia is possible by injecting either local anaesthetic or saline into the lumbar extradural space. We postulate that the rapidity of extension is caused by increased volume within the extradural

space, causing a decrease in CSF volume in the caudal subarachnoid space and cephalad shift of local anaesthetic within the CSF.

Time from injection of local anaesthetic to commencement of surgery was in excess of 30 min. In groups B and C this was partly, but not entirely, the result of the requirements of the study. For the purposes of the study, we used a technique similar to that described by Rawal, Schollin and Wesström [6], injecting a small amount of local anaesthetic to patients placed in the sitting position, and deliberately aiming to achieve inadequate anaesthesia which was extended higher by the extradural. It is our usual practice to inject 0.5% hyperbaric bupivacaine 2.5 ml into the CSF and we seldom need to extend the block with extradural anaesthetic. This results in a more rapid achievement of surgical anaesthesia and we would agree with Carrie [5] that this is preferable.

The incidence of adverse events was similar to that which may be expected for any regional technique for Caesarean section.

The need for opioid supplementation of analgesia was similar for all three groups and amounted to 40% of all patients studied. This compares poorly with the findings of Rawal, Schollin and Wesström (13%), who found the combined spinal-extradural technique better than extradurals alone [6] and is more in keeping with the findings of Alahuhta and colleagues, who showed an incidence of visceral pain of 50% for both spinal and extradural techniques for Caesarean section [13]. In our own experience, combined spinal and extradurals using 0.5% hyperbaric bupivacaine 2.5 ml results in a lower incidence of visceral pain [unpublished observations].

In this study we have shown that subarachnoid anaesthesia may be extended as effectively with extradural injection of normal saline as with bupiv-

acaine. It remains to be seen if the block can be extended if saline is injected at 20 min. This would provide important benefits, namely reducing the total local anaesthetic dose and obviating the need for a test dose which, at that time, would be difficult to interpret.

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