# SPREAD OF SPINAL ANAESTHESIA FOR CAESAREAN SECTION IN SINGLETON AND TWIN PREGNANCIES

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#### SUMMARY

We have compared the spread of spinal anaesthesia in parturients with singleton and those with twin pregnancies. Fifty-five unpremedicated patients with uncomplicated pregnancy scheduled for Caesarean section were allocated to two groups: group I = 35 singleton mothers; group II = 20 with twin pregnancy. Both groups received spinal anaesthesia with hyperbaric bupivacaine 10 mg (2 ml of 0.5%). Mean birthweight was 3290 (SD 452) g and 5008 (495) g in groups I and II (combined birthweights), respectively. We found a statistically significant difference in onset and maximal cephalad spread of spinal anaesthesia (group I median T5, range T8-T4; group II T3, range T6-T2). The mechanisms of higher cephalad spread of spinal anaesthesia in parturients may be a decrease in cerebrospinal fluid volume secondary to shunting of blood from the obstructed inferior vena cava to the extradural venous plexus and increased nerve sensitivity to local anaesthetics because of increased concentrations of progesterone. The twin pregnancy group had heavier, larger uteri and greater daily production of progesterone. (Br. J. Anaesth. 1993; 70: 639-641)

### KEY WORDS

Anaesthetic techniques: spinal. Pregnancy: singleton, twins, Caesarean section.

Pregnancy is known to cause higher cephalad spread of spinal analgesia [1, 2]. However, it is not known if there is a difference in the degree of spread between those with twin pregnancy and those with a singleton fetus. This study compared the level of spread of spinal analgesia between these groups of women undergoing elective Caesarean section.

#### PATIENTS AND METHODS

After obtaining Hospital Ethics Committee approval and written informed consent from the patients, and excluding those parturients with history of hypertension and pre-eclampsia, we studied 55 unpremedicated women, ASA I-II, undergoing elective Caesarean section. Patients were allocated to two groups: group I consisted of 35 parturients with singleton pregnancy; group II, 20 women with twin

pregnancy. All patients received spinal anaesthesia, through a 25-gauge disposable needle, comprising 0.5 % hyperbaric bupivacaine 10 mg (Marcaine Heavy, Astra) via the L3-4 interspace in left decubitus position. Patients were given lactated Ringer's solution 1000 ml and placed in a supine position with 15° left tilt immediately after the spinal injection. The level of analgesia was checked using pinprick at 2, 4, 10, 14 and 20 min. By 20 min, most of the newborns were delivered. Systemic arterial pressure was measured using an automatic monitor (Dinamap, Criticon) every 5 min. If the systolic AP decreased by more than 20% of the initial value, or to less than 100 mm Hg, ephedrine 12 mg was given i.v. Other variables measured included abdominal circumference and fundus-to-symphisis distance, recorded on admission, and birthweight and placental weight, measured at delivery.

The volume of amniotic fluid was disregarded because accurate measurement was difficult. All data are expressed as mean (range or SD), except for the level of spinal analgesia which is expressed as median (range). Data were analysed using the Mann-Whitney U test for the level of spinal spread, unpaired t test for parametric data, and chi-square test for the incidence of hypotension. P < 0.05 was considered statistically significant.

## RESULTS

Parturients with twins were significantly heavier (table I) and had significantly heavier (combined) infant birthweight (5008 (495) g vs 3290 (452) g) and placental weight (999 (128) g vs 678 (97) g) than those in the singleton group. Abdominal circum-

Table I. Patient data (mean (range or SD)) \* P < 0.05 compared with group I

(yr)	(kg)	(cm)
	65.3 (7.4)	157 (4.3) 159 (5.8)
	29 (19–36) 7.8 (23–41)	

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Table II. Birthweight of infants (BW), placenta weight (PW), fundus-to-symphisis distance (FSD) and abdominal circumference (AC) in the two groups (mean (SD)).  $\star P < 0.05$  compared with group I

	BW (g)	PW (g)	FSD (cm)	AC (cm)		
Group I Group II	3290 (452) 5008 (495)*	678 (97) 999 (128)*	35.8 (2) 40.5 (6.7)*	97.2 (5.7) 102 (9.1)*		

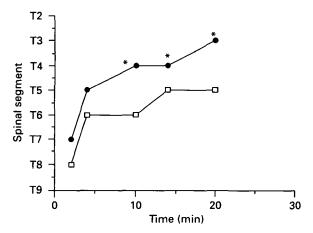


FIG. 1. Median cephalad spread of spinal analgesia in the singleton ( $\Box$ ) and twin pregnancy ( $\bullet$ ) groups. \*P < 0.05.

ference and fundus-to-symphisis distance were also larger in the twin group (table II). The number of patients who needed ephedrine did not differ between groups  $(57\% \ vs \ 65\%)$ .

After 10 min, the onset and cephalad spread of spinal analgesia in twin parturients were significantly different than those for singletons (P < 0.05) (fig. 1). Spread was to T4 (T10-T3), T4 (T8-T2) and T3 (T6-T2) in the twin group and to T6 (T8-T4), T5 (T8-T4) and T5 (T8-T4) in the singleton group at 10, 14 and 20 min, respectively.

#### DISCUSSION

We observed that parturients with twin pregnancy had a heavier and larger gravid uterus and more rapid onset and higher cephalad spread of spinal analgesia during Caesarean section compared with those with a singleton pregnancy. Compression of the inferior vena cava (IVC) secondary to increased intra-abdominal pressure has been shown to be an important factor in higher cephalad spread of spinal analgesia [1, 2]—the resulting engorgement of the lumbar and vertebral veins causes a decreased subarachnoid space and smaller CSF volume, and with less spinal fluid present in the subarachnoid space, the same amount of drug causes higher spread inside the narrower space [1, 2]. The fact that CSF volume is an important factor in modifying the spread of spinal analgesia was evident also in our previous study in which, by artificially reducing the CSF volume before administration of spinal anaesthesia, we produced a significant increase in cephalad spread of the spinal block [3].

The incidence of maternal hypotension which required treatment was 20/35 (57%) in the singleton

group and 13/20 (65%) in the twin group. The supine hypotension syndrome is not related to dose of anaesthetic or the level of spinal anaesthesia [4]. It may be caused by many factors such as occlusion of the IVC, direct compression of the aorta [5, 6], the number of venous collaterals [7], the level of compression [8], low pressure baroreceptors in the vena cava [9], and local sympathetic venoarteriolar reflex [10]. The failure to demonstrate a significant difference between the two groups in the incidence of hypotension does not imply that both groups had the same degree of IVC compression.

The sensitivity of nerve fibres to local anaesthetic agents during pregnancy has been studied extensively [11-13]. In the isolated intact vagus nerve of the pregnant rabbit, sensitivity was increased [12]. Pregnancy also increases peripheral median nerve sensitivity to lignocaine in humans [13]. Increased concentrations of progesterone during pregnancy are thought to be the cause of this increased nerve fibre sensitivity [13, 14]. The CSF concentration of progesterone is increased in parturients and a correlation has been found between the CSF concentration of progesterone and the dose requirement for intrathecal lignocaine [14]. Progesterone in human pregnancy is biosynthesized by syncytiotrophoblasts in the placenta [15]; parturients with twin pregnancy have a heavier placenta and seem to produce more progesterone than those with a singleton pregnancy. Thus nerve fibres in twin gestation may be more susceptible to local anaesthetics.

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