Preoperative dextrose does not affect spinal-induced hypotension in elective Cesarean section

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Purpose: To determine if preoperative intravenous dextrose affects the incidence and ease of treatment of spinal-induced hypotension in parturients having elective Cesarean section under spinal anesthesia.

Methods: In this prospective, double-blind study, following informed consent, 119 ASA I, II parturients for elective Cesarean section were randomized to receive intravenously either dextrose 5% in normal saline (Group D) or normal saline (Group NS) at 125 ml·hr⁻¹ for two hours prior to delivery. Following a bolus of 15 ml·kg⁻¹ normal saline iv, spinal anesthesia was induced with hyperbaric bupivacaine 0.75%, fentanyl and morphine. Hypotension (systolic blood pressure < 100 mm Hg or > 20% decrease) was treated with fluids and/or vasopressor. Data collected: demographics, blood glucose concentrations (fasting, time of spinal, delivery), blood pressure (baseline, one minute intervals from spinal to delivery), neonatal Apgar scores, umbilical blood gas analyses, glucose and lactate concentrations.

Results: There was no difference between the two groups in the rate of hypotension ($P=0.272$). All parturients who experienced hypotension received fluids, and there was also no difference between the groups in vasopressor requirement [mean dose of ephedrine: Group D = 21.6 mg (95% CI 15.1-28.2), Group NS = 16 mg (95% CI 12.0-20.5)].

Conclusion: The routine administration of dextrose 5% at a rate of 5.22 g·hr⁻¹ preoperatively does not affect the hypotension rate, or make it easier to treat.

Objectif : Déterminer si l'administration intraveineuse préopératoire de dextrose modifie l'incidence d'hypotension à induction rachidienne et la facilité de son traitement chez des parturientes qui subissent une césarienne électorale sous rachianesthésie.

Méthode : Ayant accordé leur consentement éclairé, 119 parturientes ASA I et II ont participé à une étude prospective à double insu. Réparties au hasard, elles ont reçu pendant deux heures avant la césarienne prévue une perfusion intraveineuse de dextrose 5 % dans une solution salée (Groupe D), ou d'une solution salée (Groupe S), de 125 ml·hr⁻¹. Après l'administration iv d'un bolus de 15 ml·kg⁻¹ de solution salée, la rachianesthésie a été induite avec de la bupivacaine hyperbare à 0,75 %, du fentanyl et de la morphine. On a traité l'hypotension (la tension artérielle systolique < 100 mmHg ou une baisse > 20 %) avec des liquides et/ou un vasopresseur. On a noté : des renseignements personnels, les concentrations de glucose sanguin (à jeun, au moment de la rachianesthésie, lors de l'accouchement), la tension artérielle (une mesure de base, des mesures à intervalles d'une minute de la rachianesthésie à l'accouchement), l'indice d'Apgar, la gazométrie ombilicale, les concentrations de glucose et de lactate.

Résultats : Il n'y a pas eu de différence intergroupe quant à la fréquence d'hypotension ($P = 0,272$). Toutes les parturientes qui ont souffert d'hypotension ont reçu des liquides. Il n'y a pas eu de différence intergroupe pour la demande de vasopresseur [dose moyenne d'éphédrine : Groupe D = 21,6 mg (IC 95 %; 15,1-28,2); Groupe NS = 16 mg (IC 95 %; 12,0-20,5)].

Conclusion : L'administration préopératoire de routine de dextrose à 5 % selon un débit de 5,22 g·hr⁻¹ n'a pas d'effet sur la fréquence d'hypotension et ne facilite pas son traitement.

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ELECTIVE Cesarean section delivery is commonly performed under spinal anesthesia. A major side effect of this technique is hypotension which is usually treated with fluid loading and/or vasopressor. Hypotension prior to delivery can be detrimental to both mother and fetus and, hence, methods to decrease this side-effect are of interest to the obstetric anesthesiologist.¹ A case series reported by Marx *et al.*² suggested that blood glucose may play a role in the maintenance of cardiovascular homeostasis in pregnant women and that the presence of hypoglycemia increases the incidence and severity of hypotension associated with regional anesthesia.² Marx *et al.* also suggested that hypoglycemia is common in the parturient following preoperative fasting.² This prospective, randomized, double blind study was designed to determine if preoperative intravenous dextrose affects the incidence and ease of treatment of spinal-induced hypotension during elective Cesarean section.

Methods

Following ethics committee approval, parturients presenting for elective Cesarean section under spinal anesthesia were approached to participate in the study. To be eligible for inclusion in the study the parturient had to have been in hospital for at least two hours prior to surgery, have fasted for at least four hours, be ASA I-II, with a singleton pregnancy and able to speak English. Parturients were excluded if they were morbidly obese, had a history of glucose intolerance, or were on vasoactive medication or any medication known to alter glucose metabolism.

Following written informed consent, parturients were randomized using a computer generated table, into one of two groups. The active treatment group (Group D) received dextrose 5% in normal saline *iv*, while the control group (Group NS) received normal saline only. The intravenous solution was administered at a rate of 125 ml·hr⁻¹ for two hours prior to surgery and was continued until delivery. The solutions were enclosed in an opaque bag to ensure blinding. The parturient, the anesthesiologist involved with patient care and the principal investigator recruiting the patients and performing the glucose measurements were unaware of the solution the parturient was receiving. Prior to induction of spinal anesthesia, an additional bolus of 15 ml·kg⁻¹ normal saline *iv* was administered. Spinal anesthesia was performed in the sitting position using a 25 g pencil point needle. The block was induced with 1.4 ml hyperbaric bupivacaine 0.75% with added fentanyl (10 µg) and morphine (250 µg). The parturient was immediately placed in the supine, wedged position. A

baseline blood pressure measurement was obtained prior to spinal anesthesia. The blood pressure was then measured, using a non-invasive technique, every minute from insertion of the spinal until delivery. Hypotension was defined as a decrease in systolic blood pressure of greater than 20% or a systolic blood pressure of less than 100 mmHg prior to delivery. If hypotension occurred it was treated initially with intravenous fluid followed by vasopressor, according to the discretion of the anesthesiologist managing the case. The vasopressor used was ephedrine (5-10 mg boluses) followed by phenylephrine 50-100 µg if hypotension was refractory.

Maternal venous blood glucose concentrations were collected on three separate occasions and analyzed using a One Touch® II hospital glucose meter (Life Scan, Canada). A baseline fasting blood glucose measurement was performed prior to the commencement of the study solution (glucose 1). A second blood glucose measurement was obtained at induction of spinal anesthesia, following two hours of the study solution and the normal saline pre-load (glucose 2). A final glucose measurement was performed at the time of delivery (glucose 3).

Neonatal assessment included one, five and ten minute Apgar scores. Umbilical arterial and venous blood gases were analysed at the time of delivery using an International Laboratory 1306 pH-Blood gas analyzer. Whole blood umbilical arterial and venous lactate and glucose concentrations were measured using a YSI Model 2306 STAT glucose and lactate analyzer.

A baseline incidence of hypotension of 50% was expected in the control group.¹ In the absence of other research in this area, power analysis was based on an expected improvement in the incidence of hypotension of 50% following glucose administration. With an alpha error of 0.05 and power of 80% to detect a difference, a sample size of 58 parturients per group was required. Statistical analysis used a two-sided Pearson Chi squared test for hypothesis testing. Multiple Logistic Regression was used to isolate predictors for hypotension and adverse neonatal outcome. Adverse neonatal outcome was defined as an umbilical artery pH <7.20 or a 1-minute Apgar <7. A *P* value of <0.05 was considered statistically significant.

Results

One hundred and twenty parturients consented to be part of the study. In one parturient, maternal data collection was incomplete leaving 119 parturients for analysis (Group D=60, Group NS=59). Neonatal data were not complete due to technical problems with the equipment used for the analysis of the umbilical cord samples.

TABLE I Patient demographics; mean $\pm$ standard deviation

	Group D (n=60)	Group NS (n=59)
Age (yr)	32.8 $\pm$ 5.2	33.5 $\pm$ 4.8
Height (cm)	158 $\pm$ 6.7	157.0 $\pm$ 12.8
Weight (kg)	73.5 $\pm$ 14.2	75.8 $\pm$ 12.7
Gestation (wk)	38 $\pm$ 0.8	38 $\pm$ 0.7

TABLE II Spinal information; mean $\pm$ standard deviation

	Group D (N=60)	Group NS (N=59)
Fasting Time (hr)	10.0 $\pm$ 3.4	9.5 $\pm$ 3.4
Study solution prior to spinal (ml)	209 $\pm$ 118	210 $\pm$ 131
Total fluid load prior to spinal (ml)	1323 $\pm$ 311	1366 $\pm$ 352
Total fluid prior to delivery (ml)	2215 $\pm$ 586	2175 $\pm$ 422
Dose of Hyperbaric Bupivacaine (ml)	1.37 $\pm$ 0.09	1.37 $\pm$ 0.11
Time from Spinal to Delivery (min)	25.13 $\pm$ 8.23	22.75 $\pm$ 5.98
Height of Block	T2.9 $\pm$ 1.2	T3.2 $\pm$ 1.2

TABLE III Umbilical venous and arterial values at birth
[mean $\pm$ standard deviation (number)]

	Group D	Group NS
Umbilical venous values		
pH	7.31 $\pm$ 0.05 (53)	7.31 $\pm$ 0.05 (54)
PO ₂ (mmHg)	32.6 $\pm$ 9.04 (53)	32.4 $\pm$ 7.91 (54)
PCO ₂ (mmHg)	46.4 $\pm$ 5.6 (53)	46.6 $\pm$ 7.4 (54)
Base excess	-2.33 $\pm$ 2.4 (53)	-2.40 $\pm$ 2.2 (52)
O ₂ saturation	70.12 $\pm$ 15.56 (42)	68.96 $\pm$ 17.08 (42)
Lactate (mmol·L ⁻¹)	1.05 $\pm$ 0.34 (40)	0.93 $\pm$ 0.19 (35)
Glucose (mmol·L ⁻¹)	3.90 $\pm$ 0.87 (55)	3.32 $\pm$ 0.41 (58)
Umbilical arterial values		
pH	7.25 $\pm$ 0.07 (45)	7.24 $\pm$ 0.06 (48)
PO ₂ (mmHg)	21.14 $\pm$ 18.63 (46)	16.37 $\pm$ 5.3 (48)
PCO ₂ (mmHg)	56.9 $\pm$ 9.5 (45)	59.2 $\pm$ 9.6 (49)
Base Excess	-2.65 $\pm$ 3.3 (45)	-2.12 $\pm$ 3.0 (47)
O ₂ saturation	33.11 $\pm$ 16.68 (47)	28.7 $\pm$ 13.74 (36)
Lactate (mmol·L ⁻¹)	1.29 $\pm$ 0.63 (28)	1.18 $\pm$ 0.31 (31)
Glucose (mmol·L ⁻¹)	2.87 $\pm$ 0.67 (51)	2.61 $\pm$ 0.39 (53)

Maternal demographic data were similar between the two groups (Table I). The most common reason for Cesarean section was previous Cesarean delivery. The fasting time, the quantity of study fluid received, total intravenous pre-load prior to spinal anesthesia, total fluids prior to delivery, total dose of bupivacaine 0.75% received, maximal height of block, and the time from spinal to delivery were similar between groups. Group D received a mean of 10.45 g dextrose (5.22 g·hr⁻¹) prior to the insertion of the spinal (Table II).

The initial fasting glucose concentration (Glucose 1) was similar between the groups: Group D 4.37 mmol·L⁻¹

FIGURE 1

FIGURE 2

(95% CI 4.19-4.55); Group NS=4.38 mmol·L⁻¹ (95% CI 4.22-4.53). In Group D, the mean blood glucose concentration prior to spinal anesthesia [Glucose 2=4.87 mmol·L⁻¹ (95% CI 4.52-5.32)] and at the time of delivery [Glucose 3=5.28 mmol·L⁻¹ (95% CI 5.02-5.53)] increased in response to the dextrose 5% infusion. In contrast, in group NS the mean blood glucose concentration decreased initially in response to pre-loading [Glucose 2=3.98 mmol·L⁻¹ (CI 3.77-4.18)], but then increased at the time of delivery [Glucose 3=4.54 mmol·L⁻¹ (95% CI 4.33-4.75)] (Figure 1).

FIGURE 3

There was no statistically significant difference between the two groups in the incidence of hypotension ($P=0.272$) (Figure 2). There was also no difference between the two groups with respect to the treatment given for hypotension (Figure 3). All parturients who experienced hypotension received fluids and more than 80% in each group also received vasopressor. The mean dose of ephedrine used was: Group D = 21.6 mg (95% CI 15.1-28.2), Group NS = 16 mg (95% CI 12.0-20.5). Three parturients in each group had refractory hypotension and required phenylephrine. The distribution of blood glucose concentrations at induction of spinal anesthesia was similar in parturients who had hypotension ($n=79$) [mean blood glucose 4.4 mmol·L⁻¹ (95% CI 4.0-4.9)], compared with those who had no hypotension ($n=40$) [mean blood glucose 4.4 mmol·L⁻¹ (95% CI 4.2-4.7)]. Using multiple logistic regression, the only factor predictive of hypotension in our study was the level of the block: Odds Ratio 1.6 (95% CI 1.13-2.17) for each successive increase in the thoracic level of the block.

Maternal hypoglycemia (blood glucose concentration ≤ 3.33 mmol·L⁻¹) was present in four parturients following fasting (4/119=3.4%). The mean body mass index of these parturients was lower than the group mean (25.2 ± 1.12 vs 30.3 ± 7.37). Fasting times however were similar. Hypoglycemia during the perioperative period was more common in Group NS, with ten parturients (10/59=17%) in this group being hypoglycemic at the time of spinal insertion. Only one remained hypoglycemic at the time of delivery. In contrast, only four parturients (4/60=7%) were hypo-

glycemic at the time of spinal insertion in group D, one remaining hypoglycemic at the time of delivery.

Hyperglycemia (blood glucose ≥ 6.66 mmol·L⁻¹) only occurred in group D. One parturient was hyperglycemic following fasting, four additional parturients became hyperglycemic at the time of spinal insertion (5/60=8%), with four of these parturients remaining hyperglycemic at delivery.

Umbilical venous and arterial values at birth are shown in Table III. No neonates were born hyperglycemic. One neonate had hypoglycemia, with an umbilical arterial blood glucose level of ≤ 1.66 mmol·L⁻¹ at birth. This neonate was from group D. The maternal blood glucose concentration was elevated (10 mmol·L⁻¹) at the time of spinal anesthesia, but had returned to normal (4.5 mmol·L⁻¹) by delivery. Acidosis (umbilical arterial pH ≤ 7.20) occurred in 11 neonates, six from group D and five from group NS. Eight neonates in group D had a one minute Apgar score < 7 , while in group NS none had low initial Apgar scores. All neonates in group D had improved to a score of 7 or greater by five minutes. The maternal blood glucose levels were normal in these eight neonates.

Discussion

Hypotension is a common complication of spinal anesthesia for Cesarean section. Hypotension can be detrimental to both the parturient and her neonate,¹ hence methods to treat hypotension are of interest to the obstetric anesthesiologist. Our study, looking at the effects of preoperative dextrose administration on hypotension during spinal anesthesia, was stimulated by a case series published by Marx *et al.*² In this series, Marx *et al.* reported hypotension associated with high regional anesthesia in three healthy parturients with hypoglycemia (blood glucose concentration < 3.33 mmol·L⁻¹). In these parturients, hypotension was not responsive to an intravenous fluid bolus and/or vasopressor, but did respond to an intravenous bolus of glucose. This suggested that blood glucose played an important role in cardiovascular homeostasis in pregnancy and that hypoglycemia may result in more profound hypotension during high regional anesthesia for Cesarean section. Based on these findings, the primary aim of our study was to look at the effects of routine pre-operative glucose administration on spinal-induced hypotension.

The incidence of hypotension in our study was 67%. This rate is consistent with that reported by other authors in the presence of left uterine displacement and adequate pre-loading.¹ Although the use of crystalloid preloading prior to spinal anesthesia has been recently questioned,^{3,4} it remains part of the routine practice in

most centres, including ours, and hence was used in this study. The incidence of hypotension in our study was not affected by the routine administration of preoperative dextrose (mean $5.2 \text{ g}\cdot\text{hr}^{-1}$). Despite the findings by Marx *et al.*,⁵ administration of dextrose 5% did not make the hypotension easier to treat. All hypotensive parturients received an additional fluid bolus and > 80% in each group received ephedrine. The common requirement for ephedrine as a vasopressor during elective Cesarean section has led to the use of prophylactic ephedrine infusions in some centres. Although earlier studies showed benefit with this technique,⁵ recent work suggests that its use may adversely affect neonatal blood gases and may not reduce the incidence of spinal-induced hypotension.⁶ This is an area requiring further research.

Jawalekar and Marx reported an incidence of preoperative hypoglycemia of 48% in parturients hospitalized on the night before surgery.⁷ Previously unpublished data by Marx *et al.*, presented along with their case series, reported an incidence of hypoglycemia of 68% if no evening snack was given to the hospitalized parturient on the night prior to surgery.² This high incidence of hypoglycemia led Marx *et al.* to suggest that measurement of blood glucose concentration should be performed prior to anesthesia.² Unfortunately, Marx *et al.* gave no information regarding the duration of fasting or the weight and the height of the parturients. Differences in these factors may account for the different hypoglycemic rates observed in our study in which the rate of hypoglycemia in parturients admitted on the day of surgery was only 3.4% (4/119). Our findings do not support the routine determination of blood glucose levels in parturients prior to spinal anesthesia.

The effects of preloading the parturient with dextrose containing fluid has also been the subject of research. Early work in the 1960s by Wollman and Marx recommended the use of dextrose 5% in normal saline to prevent spinal-induced hypotension.⁸ In 1982, Kenepf *et al.* questioned the safety of this technique.⁹ They performed a prospective, randomised study comparing the effects of three different glucose regimens for the prevention of hypotension prior to epidural anesthesia. Kenepf demonstrated that the use of large doses of glucose (25 g and 57.5 g) were of no benefit to the mother and had the potential to produce fetal hyperinsulinemia, with neonatal acidosis and rebound hypoglycemia. The authors recommended limiting the infusion of dextrose to $6 \text{ g}\cdot\text{hr}^{-1}$ prior to delivery in order to prevent adverse neonatal effects.⁹ This was the basis for limiting the amount of dextrose to $6 \text{ g}\cdot\text{hr}^{-1}$ in our study. Interestingly, in Kenepf's study the incidence of hypotension was similar in all parturients although

some parturients received larger doses of dextrose. Maternal hypoglycemia (blood glucose level $\leq 3.33 \text{ mmol}\cdot\text{L}^{-1}$) during the perioperative period is also of concern and is more likely in response to the dilution effects of non-glucose containing solutions given for pre-loading.⁸ In our study, 10/60 (17%) parturients became hypoglycemic at the time of insertion of the spinal in response to a non-glucose containing pre-load. A similar response to a preload of 1 L Ringer's lactate was observed by Grylack *et al.*⁹ In their study 4/20 (20%) parturients became hypoglycemic at the time of delivery. In contrast, Thomas *et al.* demonstrated a small increase in blood glucose concentration in response to preloading with 2 L Ringer's lactate solution.¹⁰

An increase in blood glucose at the time of delivery occurred in both groups in our study in response to surgery (i.e. between the time of spinal insertion and the time of delivery). This increase was also observed by Thomas *et al.* following the use of lactate containing solutions for intravenous preloading.¹² Thomas postulated that this increase may be in response to lactate acting as a gluconeogenic precursor. Previous work by Engquist *et al.* suggested that a neuraxial block from T₄ to S₅ inhibits the hyperglycemic response to surgery during hysterectomy.¹³ In our study no lactate was given, suggesting that the hyperglycemic stress response to surgery may still be present in the healthy parturient despite a neuraxial block to T₄.

Unfortunately, our neonatal data are incomplete. This limits the conclusions we can draw with respect to the effects on the neonate of our treatment regime. However, using multiple logistic regression and a definition of poor neonatal outcome of an Apgar of <7 at one minute or an umbilical arterial pH <7.2, the only factor found to be predictive was the dose of ephedrine required. The mean dose of ephedrine associated with good neonatal outcome was 8.0 mg (CI 5.8-10.2), while that associated with poor neonatal outcome was 26.3 mg (CI 12.6-40.0). This probably reflects the fact that parturients with severe hypotension require greater doses of ephedrine. This emphasises the importance of the rapid treatment of hypotension associated with spinal anesthesia. Maternal blood glucose concentrations were not associated with poor neonatal outcome.

We did not measure neonatal blood glucose concentrations after delivery. Rebound neonatal hypoglycemia usually occurs two hours following delivery and, hence, may have occurred and been missed due to our study design. However, neonatal hypoglycemia is only likely to occur if the maternal blood glucose concentration is $\geq 6.66 \text{ mmol}\cdot\text{L}^{-1}$ at the time of delivery.¹⁴ Hyperglycemia was only present in four parturients in

our study, all from group D. The umbilical artery blood glucose concentrations were normal in these cases, although the possibility of rebound hypoglycemia due to hyperinsulinemia cannot be excluded in these neonates. Fetal hypoglycemia (umbilical artery glucose $1.66 \text{ mmol}\cdot\text{L}^{-1}$) occurred on only one occasion in our study, and was not related to maternal hypoglycemia. Interestingly, the rate of neonatal acidosis observed in our study (11/119=9%) was lower than that reported by Roberts *et al.* in their retrospective review (55/231=24%).¹⁵

As with all studies in which no difference is found, we need to ask if this is a true negative finding. One possible reason we found no effect on the hypotension rate with the administration of a dextrose solution may be due to our low baseline hypoglycemic rate (3.4%). This was in contrast to what was expected based on the incidence of hypoglycemia reported by Marx *et al.* (68%).² However, using multiple logistic regression to examine the risk factors for predicting hypotension, blood glucose concentrations were not implicated. Despite this deficiency, our study clearly demonstrates that the routine use of an infusion of $5.22 \text{ g}\cdot\text{hr}^{-1}$ dextrose prior to spinal anesthesia for elective Cesarean section will not reduce the rate of hypotension or make it easier to treat.

A second reason we may have found no difference is that we used only a small dose of dextrose. We limited the dose of dextrose due to concerns regarding neonatal safety, and as mentioned previously, work by Kenepf *et al.* demonstrated no improvement in hypotensive rates when higher doses of dextrose were used.⁹

In conclusion, we have found that the routine administration of dextrose 5% at a rate of $5.24 \text{ g}\cdot\text{hr}^{-1}$ does not reduce the incidence of spinal-induced hypotension, or make it easier to treat. In addition, the low rates of hypoglycemia we observed following fasting are reassuring and do not support the routine determination of maternal blood glucose levels prior to spinal anesthesia for elective Cesarean section.

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