

# Impact of Maternal Spinal Anesthesia-Induced Hypotension at Scheduled Cesarean Delivery on Risk Development of Transient Tachypnea of Newborn and Fetal Acidosis: A Prospective Study

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## ABSTRACT

**Background:** Maternal hypotension resulting from spinal anesthesia and the interval between anesthetic administration and delivery are considered potential modifiable risk factors contributing to the development of transient tachypnea of the newborn (TTN) and fetal acidosis. To investigate the precise impact of maternal hypotension triggered by spinal anesthesia and the duration of anesthesia-to-delivery intervals on neonatal outcomes, primarily the incidence of TTN and fetal acid-base imbalance among women undergoing planned cesarean section (CS) deliveries.

**Methods:** A pre-planned prospective cohort study was conducted over a one-year period at a tertiary care center, enrolling all women with non-anomalous singleton pregnancies between 37 and 41 weeks of gestation who delivered via elective CS under spinal anesthesia. The main outcome variables were the incidence of neonatal respiratory morbidity, specifically TTN, and fetal acidosis, evaluated in relation to maternal intraoperative hypotension and the interval from induction of anesthesia to delivery. Intraoperative maternal blood pressure profiles were systematically compared between neonates with TTN and those without.

**Results:** A total of 115 maternal-infant dyads were included in the final analytical cohort. Neonates who developed TTN were delivered by mothers who experienced more profound intraoperative hypotension, as evidenced by significantly lower minimum systolic blood pressure (SBP) ( $P = 0.006$ ), diastolic blood pressure (DBP) ( $P = 0.01$ ), and mean arterial pressure (MAP) ( $P = 0.003$ ). Notably, maternal baseline MAP demonstrated a considerable inverse correlation with umbilical cord pH ( $r = -0.473$ ,  $P < 0.001$ ), whereas minimum MAP correlated positively ( $r = 0.324$ ,  $P < 0.001$ ). Multivariable analyses identified both the severity and duration of maternal hypotension as significant independent predictors of TTN and fetal acidosis.

**Conclusion:** Implementing preventive measures against maternal spinal hypotension is a prudent strategy to minimize the likelihood of neonatal acidosis and reduce the risk of TTN in scheduled cesarean deliveries.

**Keywords:** Maternal hypotension, Neonatal acidosis, Scheduled cesarean delivery, Spinal anesthesia, Transient tachypnea of newborn

## Introduction

Transient tachypnea of the newborn (TTN) is a commonly encountered, self-resolving

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parenchymal lung disorder characterized by delayed reabsorption of fetal alveolar fluid in term neonates. Clinically, it presents as mild to moderate respiratory distress that typically resolves within 48 to 72 hours after birth (1, 2).

Neonates diagnosed with TTN often require admission to the neonatal intensive care unit (NICU), resulting in maternal-neonatal separation. Additionally, the need for respiratory support may lead to unnecessary antibiotic exposure, prolonged hospitalization, and increased healthcare expenditures (3–5).

Emerging evidence suggests that term neonates with TTN are at an increased risk of subsequent hospitalization due to respiratory syncytial virus (RSV) bronchiolitis during infancy (6, 7), as well as an inherent susceptibility to developing asthma later in childhood (8).

TTN represents a major contributor to respiratory distress among both term and late-preterm infants, with reported incidence rates from 4.0 to 5.7 per 1000 live births (3).

Prior studies have indicated that the risk of developing TTN in infants delivered via elective cesarean section (CS) is approximately 2 to 6 times higher compared to those born vaginally (9, 10).

While maternal-fetal determinants for TTN are well understood (11), the influence of preoperative variables, particularly in the context of elective CS deliveries, remains insufficiently explored (12).

Maternal hypotension, a frequent complication of spinal anesthesia during CS delivery, has been proposed as a potential contributing factor to adverse neonatal outcomes (13). Nevertheless, spinal anesthesia remains the preferred anesthetic approach for cesarean delivery due to several advantages, including a reduced risk of aspiration compared to general anesthesia, the opportunity for maternal awareness and presence at birth, and improved neonatal outcomes, as evidenced by higher APGAR scores (14).

Elective cesarean deliveries are generally associated with fewer instances of fetal distress and adverse neonatal outcomes compared to emergency procedures. Nevertheless, the interval between spinal anesthesia administration and fetal delivery represents a critical window during which potential fetal compromise may occur (15).

Episodes of maternal hypotension and prolonged surgical duration are potential contributors to adverse neonatal outcomes during the pre-delivery period; however, further research is needed to clarify their relative clinical

significance and establish definitive threshold values (16).

To our knowledge, few studies have investigated how maternal hemodynamic fluctuations under regional anesthesia and the anesthesia-to-delivery interval during planned cesarean sections contribute to the development of TTN and fetal acidosis in Egypt. Therefore, we hypothesized that pre-delivery maternal hypotension induced by regional spinal anesthesia, along with the interval between anesthesia administration and fetal extraction, influences the risk of developing TTN and fetal acidosis in full-term neonates delivered by elective CS.

The aim of this cohort study is to assess the relationship between the severity and duration of maternal hypotension and the time span from anesthesia-to-delivery with the risk of TTN and fetal acid-base imbalance.

## Methods

All term, non-anomalous singleton neonates ( $\geq 37$  weeks gestation) delivered via planned CS under spinal anesthesia between April 2023 and March 2024 at Benha University Hospital were prospectively enrolled in this study. The Human Subjects Review Committee of Benha Faculty of Medicine reviewed and approved the study protocol (RC# 18-4-2023). All procedures involving the cohort were conducted in strict accordance with international ethical standards, as stipulated by the World Medical Association's Declaration of Helsinki and relevant institutional regulations (17). This prospective clinical investigation was officially registered on clinicaltrials.gov (NCT# 06383624).

Exclusion criteria included multiple pregnancies, gestational age less than 37 weeks, neonatal malformations, missing arterial blood gas (ABG) data, maternal exposure to antenatal steroids, and a history of maternal comorbidities such as gestational or preexisting diabetes mellitus, as well as pregnancy-induced or chronic hypertension. Additionally, cases involving either spontaneous or induced labor, those managed with combined spinal-epidural anesthesia requiring epidural activation prior to delivery, were excluded and those who refused to participate in our cohort to be added before were excluded.

Spinal anesthesia was administered under complete aseptic standard technique with the patient in a flexed sitting position, utilizing an appropriate needle size that was inserted in the

L2-L3 or L3-L4 intervertebral space. Upon free flow of cerebrospinal fluid, the recommended dosages of Bupivacaine and Fentanyl were injected intrathecally. Maternal blood pressure was monitored at 3- to 5-minute intervals from the induction of anesthesia until fetal delivery, with any episode of hypotension recorded. The standard protocol at our institution for the management of maternal spinal anesthesia-induced hypotension was followed, which is consistent with guidelines for settings where phenylephrine is not the primary agent (18, 19). Accordingly, following the intrathecal anesthetic administration, all mothers received a rapid intravenous crystalloid co-load of 10-15 ml/kg. Maternal hypotension was defined as a systolic blood pressure (SBP) decrease of more than 20% from baseline or an absolute SBP of less than 100 mmHg. An initial intravenous bolus of ephedrine (5-10 mg) was administered based on our institutional policy and drug availability, and this bolus was repeated every 2-3 minutes if hypotension persisted.

Immediate postnatal umbilical ABGs were collected from a double-clamped segment of the umbilical cord for all neonates using a 1 ml pre-heparinized syringe to detect neonatal acidosis, defined as an arterial pH below 7.2 or a base deficit exceeding 12 mmol/L, which served as the primary outcome. Arterial samples were identified primarily based on anatomical characteristics, including smaller caliber and thicker wall compared to the umbilical vein, and standard sampling technique. Samples with extreme pO<sub>2</sub> values were excluded; however, paired arterial and venous sampling was not performed, and definitive confirmation of arterial origin could not be ensured. Therefore, classification of samples as arterial was based mainly on the sampling technique rather than pO<sub>2</sub> measurements.

The established diagnosis of TTN, the secondary outcome of interest, was based on diagnostic criteria within the first six hours postnatally, persisting for at least 12 hours, including a respiratory rate greater than 60 breaths per minute, and radiological findings on chest X-ray consistent with compliance alterations, such as fluid in the interlobar fissures and/or prominent central pulmonary vasculature. These neonates responded to oxygen therapy at concentrations  $\leq 40\%$ . Cases with other underlying causes of respiratory compromise, such as neonatal respiratory distress syndrome, meconium aspiration, congenital cardiac anomalies and/or pulmonary disorders, and

neonatal sepsis were excluded (20-22). All term infants diagnosed with TTN were admitted to and managed in a tertiary-level III neonatal intensive care unit (NICU) at Benha University Hospital.

### **Data collection**

Comprehensive maternal demographic and clinical data were collected, including age, body mass index (BMI), gravidity, parity, and indications for cesarean delivery (such as recurrent CS, breech presentation, maternal request, prior intrauterine fetal demise [IUFD], intraoperative time intervals, and maternal physiological status classified by the American Society of Anesthesiologists [ASA]). Neonatal parameters included gestational age (GA), sex, anthropometric measures (birth weight and length), APGAR scores at 1 and 5 minutes, NICU admission due to TTN, and umbilical cord blood gas analysis.

Anesthetic protocols were documented, detailing pharmacological agents administered, the spinal sensory dermatome level achieved prior to surgical incision, and the time intervals between spinal anesthesia induction and fetal extraction.

Hemodynamic monitoring encompassed baseline and minimum systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), as well as the magnitude of decline, duration of the most pronounced hypotensive episode, and encountered therapeutic interventions (intravenous fluid resuscitation versus vasopressor administration).

### **Estimation of sample size**

The required sample size was calculated using MedCalc software (version 18.2.1), based on Hassanin et al., who demonstrated a significant inverse correlation ( $r = -0.268$ ) between umbilical pH and the duration of prolonged hypotensive episodes (23). Assuming an alpha threshold ( $\alpha$ ) of 0.05 and power of 80%, respectively, the calculated minimum number of participants was estimated to be 110 participants to ensure adequate detection of clinically meaningful associations.

### **Statistics**

SPSS software version 28 (IBM, Armonk, NY, USA) was used for data processing and statistical analysis. Normality was assessed using the Shapiro-Wilk test, complemented by graphical evaluation through histograms and Q-Q plots. Continuous data were presented as means and standard deviations (SD) for normally distributed

variables, or medians and ranges for non-normal data. Categorical variables were described as frequencies and percentages (n, %). Comparisons between groups based on TTN occurrence were performed using the independent t-test or Mann-Whitney U test for continuous variables, depending on their distribution. Categorical variables were analyzed with the Chi-square or Fisher's exact test as appropriate. Correlations between umbilical cord pH and hemodynamic parameters (blood pressure) were evaluated using Pearson's correlation coefficient. Binary logistic regression was employed to predict TTN occurrence, with results expressed as adjusted odds ratios (aOR) and 95% confidence intervals (CI). Linear regression was used to predict umbilical cord pH, reporting regression coefficients (B) and 95% CI. Two-sided tests were applied, and p-values below 0.05 were considered statistically significant.

### Ethical approval

The study protocol was reviewed and approved by the Human Subjects Review Committee of Benha Faculty of Medicine (Approval Number: RC# 18-4-2023). All procedures were conducted in accordance with the ethical standards outlined in the World Medical Association's Declaration of Helsinki and relevant institutional guidelines. Informed written consent was obtained from all participating mothers prior to enrollment in the study.

## Results

### Neonatal and maternal characteristics

The study cohort demonstrated a 21.7% incidence of TTN, with significant maternal

demographic differences observed between TTN and non-TTN cases. Mothers of neonates with TTN were older (36±4 years) compared to the control group (31±5 years,  $P < 0.001$ ), had higher BMI (28.7±4.1 kg/m<sup>2</sup>) compared to the controls (26.2±3.4 kg/m<sup>2</sup>,  $P = 0.002$ ), and exhibited greater gravidity and parity ( $P < 0.001$  for each) (Table 1).

### Perinatal outcomes

Neonates developing TTN showed significantly higher birth weights (3.37±0.37 kg) compared to the control group (3.16±0.38 kg,  $P = 0.014$ ) and lower APGAR scores at the 1st and 5th minutes ( $P < 0.001$  for both) (Table 1).

No significant associations were found for GA ( $P = 0.124$ ), fetal presentation ( $P = 1.0$ ), length ( $P = 0.728$ ), or neonatal sex distribution ( $P = 0.215$ ), suggesting these variables were not clinically relevant predictors of TTN (Table 1).

### Anesthetic and hemodynamic correlates of TTN

Neonates developing TTN exhibited significant differences in spinal anesthesia parameters compared to controls. The TTN group received higher doses of bupivacaine (1.7±0.06 mg vs. 1.66±0.07 mg,  $P = 0.013$ ) and demonstrated greater prevalence of sensory blockade at the T5 dermatomal level (28% vs. 8.9%,  $P = 0.012$ ) (Table 2).

Hemodynamically, the TTN cohort experienced more pronounced hypotension, with lower minimum SBP (94±8 mmHg vs. 99±9 mmHg,  $P = 0.006$ ), DBP (64±7 mmHg vs. 68±7 mmHg,  $P = 0.01$ ), and MAP (74±6 mmHg vs. 78±7 mmHg,  $P = 0.003$ ) (Figure 1; Table 2).

These findings suggest that both anesthetic dose intensity and hemodynamic instability may

**Table 1.** General characteristics according to the occurrence of TTN

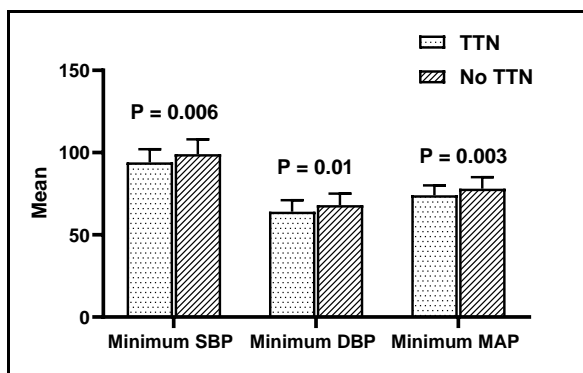
		Total	TTN		P-value
			Yes (n = 25)	No (n = 90)	
Age (years)	Mean ±SD	32 ±5	36 ±4	31 ±5	<0.001*
BMI (Kg/m <sup>2</sup> )	Mean ±SD	26.7 ±3.7	28.7 ±4.1	26.2 ±3.4	0.002*
Gravida	Median (Range)	3 (1 - 6)	3 (1 - 6)	3 (1 - 4)	<0.001*
Para	Median (Range)	2 (0 - 4)	2 (0 - 4)	1 (0 - 3)	<0.001*
GA (weeks)	Mean ±SD	38 ±1	38 ±1	38 ±1	0.124
Fetal presentation					
Breech	n (%)	18 (15.7)	4 (16)	14 (15.6)	1.0
Cephalic	n (%)	92 (80)	20 (80)	72 (80)	
Transverse lie	n (%)	5 (4.3)	1 (4)	4 (4.4)	
Neonatal sex					
Males	n (%)	61 (53)	16 (64)	45 (50)	0.215
Females	n (%)	54 (47)	9 (36)	45 (50)	
Birth weight (Kg)	Mean ±SD	3.2 ±0.39	3.37 ±0.37	3.16 ±0.38	0.014*
Length (cm)	Mean ±SD	49 ±2	49 ±2	49 ±1	0.728
APGAR 1 <sup>st</sup> min	Median (Range)	8 (5 - 9)	7 (5 - 8)	8 (7 - 9)	<0.001*
APGAR 5 <sup>th</sup> min	Median (Range)	9 (7 - 10)	9 (7 - 10)	9 (8 - 10)	<0.001*
pH	Mean ±SD	7.23 ±0.1	7.21 ±0.08	7.23 ±0.1	0.363

\*Significant P-value; SD: Standard deviation; BMI: Body mass index; GA: Gestational age; TTN: Transient tachypnea of the newborn

**Table 2.** Spinal anesthesia characteristics according to the occurrence of TTN

		Total	TTN		P-value
			Yes (n = 25)	No (n = 90)	
Bupivacaine (mg)	Mean ±SD	1.67 ±0.07	1.7 ±0.06	1.66 ±0.07	0.013*
Fentanyl (mcg)	Mean ±SD	15 ±4	15 ±4	15 ±4	0.662
Sensory dermatome level					
T4	n (%)	100 (87)	18 (72)	82 (91.1)	0.012*
T5	n (%)	15 (13)	7 (28)	8 (8.9)	
Baseline SBP (mmHg)	Mean ±SD	129 ±9	128 ±10	129 ±8	0.377
Minimum SBP (mmHg)	Mean ±SD	98 ±9	94 ±8	99 ±9	0.006*
Baseline DBP (mmHg)	Mean ±SD	85 ±10	87 ±13	85 ±9	0.535
Minimum DBP (mmHg)	Mean ±SD	67 ±7	64 ±7	68 ±7	0.01*
Baseline MAP (mmHg)	Mean ±SD	100 ±9	100 ±12	100 ±9	0.793
Minimum MAP (mmHg)	Mean ±SD	77 ±7	74 ±6	78 ±7	0.003*
Lowest hypotensive episode (min)	Mean ±SD	2.77 ±1.04	3.11 ±1.09	2.68 ±1.01	0.066
Spinal to delivery (min)	Mean ±SD	30.6 ±6.03	31.59 ±5.32	30.33 ±6.21	0.359
IV fluids (milliliters)	Mean ±SD	1499 ±251	1720 ±171	1438 ±235	<0.001*
Ephedrine (by use)	n (%)	58 (50.4)	20 (80)	38 (42.2)	<0.001*
Dose (mg)	Mean ±SD	19 ±7	24 ±6	16 ±5	<0.001*

\*Significant P-value; SD: Standard deviation; NICU: Neonatal Intensive Care Unit; TTN: Transient tachypnea of the newborn; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure; IV: Intravenous; PH: Potential of hydrogen



**Figure 1.** Correlation between TTN and maternal blood pressure parameters

contribute to TTN pathophysiology.

The TTN group received significantly greater volumes of intravenous fluids (1720±171 mL vs. 1438±235 mL,  $P < 0.001$ ) and required more frequent ephedrine administration at higher doses ( $P < 0.001$  for both) compared to the non-TTN group (Table 2).

Notably, no significant differences were observed in fentanyl dosage ( $P = 0.662$ ), baseline hemodynamic parameters (SBP:  $P = 0.377$ , DBP:  $P = 0.535$ , MAP:  $P = 0.793$ ), duration of profound hypotension ( $P = 0.066$ ), spinal-to-delivery interval ( $P = 0.359$ ), or umbilical pH ( $P = 0.363$ ) (Table 2).

These results implicate perioperative fluid loading and vasopressor requirements as potential modifiable risk factors for TTN, independent of baseline hemodynamic status or anesthetic duration.

### Correlation between maternal blood pressure parameters and umbilical cord pH

The study revealed several significant

correlations between pH and various hemodynamic variables. Baseline SBP showed a strong negative correlation with pH ( $r = -0.484$ ,  $P < 0.001$ ). Conversely, minimum SBP demonstrated a positive correlation with pH ( $r = 0.357$ ,  $P < 0.001$ ). Similarly, baseline DBP and minimum DBP also showed significant correlations with pH, with baseline DBP having a negative correlation ( $r = -0.439$ ,  $P < 0.001$ ) and minimum DBP a positive one ( $r = 0.252$ ,  $P = 0.007$ ). Baseline MAP negatively correlated with pH ( $r = -0.473$ ,  $P < 0.001$ ), while minimum MAP was positively correlated ( $r = 0.324$ ,  $P < 0.001$ ) (Figure 2).

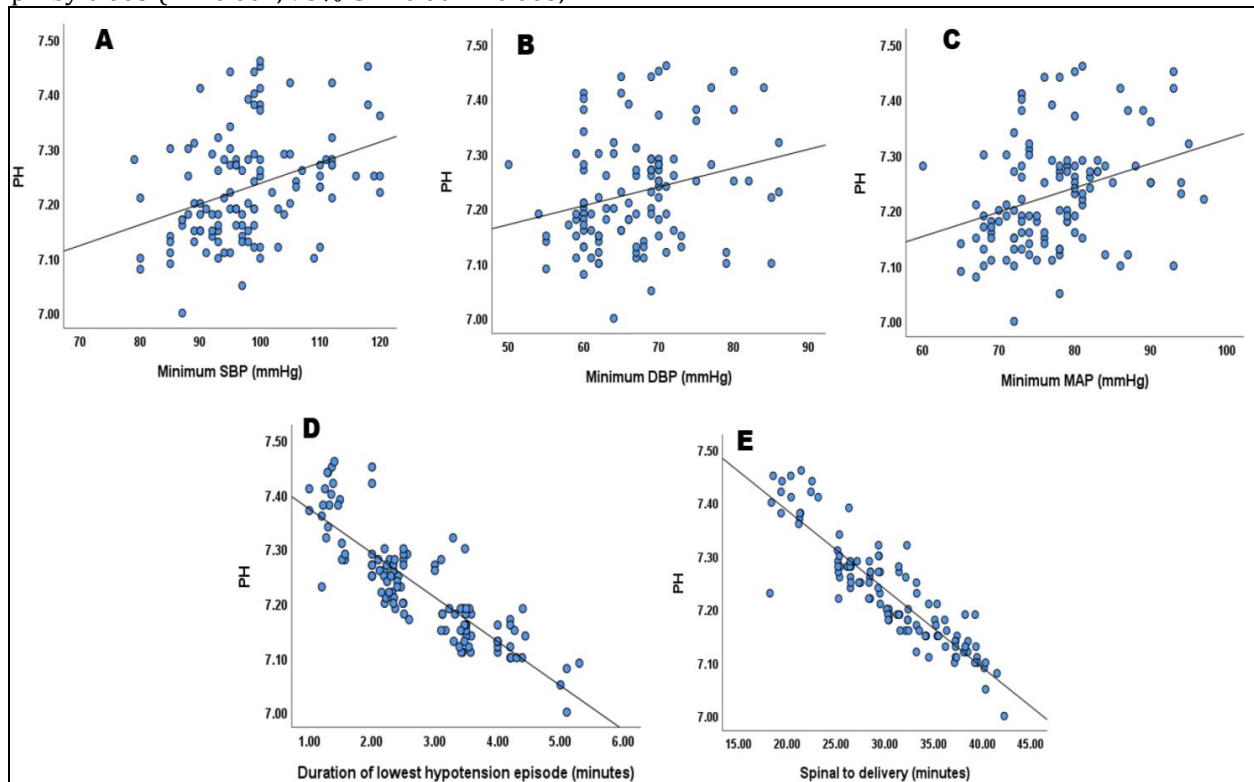
Moreover, the duration of the lowest hypotension episode exhibited a very strong negative correlation with pH ( $r = -0.852$ ,  $P < 0.001$ ), and the time from spinal anesthesia to delivery was also strongly negatively correlated with pH ( $r = -0.899$ ,  $P < 0.001$ ).

### Prediction of TTN and fetal pH

Multiple logistic regression analysis was done to predict TTN using blood pressure parameters, controlling for age, BMI, gravida, parity, GA, and gender. The analysis revealed that one mmHg increase in minimum SBP was associated with an 11.3% risk reduction of TTN (OR = 0.887, 95% CI = 0.824 – 0.955,  $P = 0.001$ ). One mmHg increase in minimum DBP was associated with a 12.6% reduced risk of TTN (OR = 0.874, 95% CI = 0.802 – 0.953,  $P = 0.002$ ). One unit increase in minimum MAP was associated with a 14.7% reduced risk of TTN (OR = 0.853, 95% CI = 0.777 – 0.936,  $P = 0.001$ ). Additionally, a minute increase in the duration of the lowest hypotension episode was associated with approximately 72% increased risk of TTN (OR = 1.719, 95% CI = 1.035 – 2.856,  $P =$

0.036) (Table 3).

Moreover, multivariable linear regression analysis was done to predict pH using blood pressure parameters, controlling for age, BMI, gravida, parity, GA, and gender. The analysis showed that every one mmHg increase in minimum SBP was associated with an increase in pH by 0.003 (B = 0.004, 95% CI = 0.001 - 0.005, P



**Figure 2.** Correlation between umbilical cord pH and A) minimum SBP; B) minimum DBP; C) minimum MAP; D) duration of lowest hypotensive episode; E) spinal-to-delivery time

**Table 3.** Multivariable logistic regression for blood pressure parameters to predict TTN

	OR (95% CI) †	P-value
Minimum SBP (mmHg)	0.887 (0.824 - 0.955)	0.001*
Minimum DBP (mmHg)	0.874 (0.802 - 0.953)	0.002*
Minimum MAP (mmHg)	0.853 (0.777 - 0.936)	0.001*
Lowest hypotension episode (minutes)	1.719 (1.035 - 2.856)	0.036*

\*Significant P-value; † Adjusted for age, BMI, Gravida, Parity, gestational age, and gender; OR: Odds ratio; CI: Confidence interval; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure.

= 0.001). Similarly, each one mmHg increase in minimum DBP was associated with an increase in pH of 0.003 (B = 0.003, 95% CI = 0.0005 - 0.006, P = 0.02). Moreover, one unit increase in minimum MAP was associated with an increase in pH by 0.004 (B = 0.004, 95% CI = 0.001 - 0.006, P = 0.002). Furthermore, each one-minute increase in the duration of the lowest hypotension episode

was associated with a reduction in pH by 0.082 (B = -0.082, 95% CI = -0.091 - -0.072, P < 0.001) (Table 4).

**Table 4.** Multivariable linear regression for blood pressure parameters to predict pH

	B (95% CI) †	P-value
Minimum SBP (mmHg)	0.003 (0.001 - 0.005)	0.001*
Minimum DBP (mmHg)	0.003 (0.0005 - 0.006)	0.02*
Minimum MAP (mmHg)	0.004 (0.001 - 0.006)	0.002*
Lowest hypotension episode (min)	-0.082 (-0.091 - -0.072)	<0.001*

\*Significant P-value; † Adjusted for age, BMI, Gravida, Parity, gestational age, and gender; B: Beta coefficient; CI: Confidence interval; SBP: Systolic blood pressure; DBP: Diastolic blood

### Discussion

In this prospective cohort of 115 term neonates born via planned CS under spinal anesthesia, a 21.7% incidence of TTN was

identified, which was significantly associated with maternal factors including advanced maternal age, higher BMI, parity, increased birth weight, lower APGAR scores, lower blood pressure parameters, higher sensory block level, greater anesthetic dosage, and prolonged hypotensive episodes.

Importantly, maternal hypotension, characterized by lower systolic, diastolic, and mean arterial pressures, as well as prolonged duration, emerged as a significant independent predictor of TTN and fetal acidosis. A robust inverse correlation between umbilical cord pH and the extent of maternal hemodynamic compromise further underscored the impact of intraoperative hemodynamic instability on neonatal acid-base status.

Remarkably, our study reinforces emerging evidence that scheduled cesarean deliveries under spinal anesthesia are associated with a heightened risk of TTN, potentially due to the absence of hormonal and mechanical stimuli intrinsic to vaginal delivery that promote fetal lung fluid clearance (1,3). Moreover, maternal hypotension further compounded this risk by compromising uteroplacental perfusion and fetal oxygenation, potentially leading to TTN and neonatal acidosis (11,24,25).

Our findings revealed that lower values of minimum SBP, DBP, and MAP significantly predicted TTN, with each unit drop in MAP increasing the odds of TTN by 14.7%. These results mirror prior studies, such as those by Hassanin et al. and Powell et al., who documented that both the severity and duration of intraoperative hypotension adversely affect umbilical pH and early neonatal outcomes (16,23).

The TTN group experienced longer durations of the lowest hypotensive episodes, necessitating greater administration of IV fluids and ephedrine. These findings suggest that, despite proactive intraoperative interventions, such measures may have been implemented reactively rather than preventively. This observation corroborates the work of Campbell and Stocks, who emphasized the importance of preemptive vasopressor use and adequate fluid preloading in mitigating the incidence and severity of spinal-induced hypotension and its potential fetal-threatening repercussions (13).

Interestingly, while the spinal-to-delivery interval was negatively correlated with pH, it did not significantly differ between TTN and non-TTN groups. This suggests that hemodynamic parameters may have a more direct influence on neonatal outcomes than timing alone, although

minimizing this interval remains critical. Rimsza et al. previously demonstrated that delays exceeding 30 minutes from spinal induction to delivery were independently associated with lower umbilical cord pH (15).

The lack of significant between-group differences in baseline BP values underscores that the most impactful determinants of adverse outcomes are the hypotensive nadir and its duration, rather than the initial hemodynamic status. Furthermore, the higher intrathecal doses of bupivacaine administered and the attainment of higher sensory block levels in the TTN group indicate a dose-dependent attenuation of sympathetic thoracolumbar outflow, contributing to more profound hypotension. Congruent with Ozden et al., who found a similar association between higher block levels and an increased incidence of neonatal respiratory distress (14).

Additionally, our study reinforces that fetal acidosis may still occur in the context of scheduled, controlled cesarean deliveries as a consequence of intraoperative events such as maternal hypotension. The observed correlation between fetal pH and both the minimum blood pressures and the duration of hypotension supports the utility of fetal acid-base status as a sensitive surrogate marker for assessing intraoperative perfusion adequacy (20).

Our interpretations highlight the imperative for implementing anticipatory anesthetic strategies aimed at preventing hypotension during scheduled CS deliveries. Such approaches may encompass reduced doses of intrathecal anesthetic, judicious co-loading with crystalloids, preemptive vasopressors, favoring phenylephrine over ephedrine, and vigilant BP monitoring during the perioperative period. Moreover, expeditious fetal extraction following spinal induction should be prioritized, especially in high-risk maternal cohorts such as those of advanced maternal age, obesity, or multiparity.

Reducing the risk of TTN is essential not only for optimizing immediate neonatal outcomes but also for averting downstream sequelae, including extended NICU stays, increased healthcare expenditures, and an increased risk of subsequent respiratory morbidities, including asthma and RSV hospitalization risks (6,8,26).

### **Strengths, Limitations and Recommendations**

To the best of our knowledge, this research represents one of the few prospective investigations conducted in Egypt that systematically scrutinizes the interplay between

maternal intraoperative blood pressure fluctuations and early neonatal respiratory and metabolic outcomes. Key strengths include continuous real-time blood pressure tracking, validated TTN diagnostic criteria, and the use of objective umbilical cord gas analyses.

Nonetheless, some limitations warrant consideration. First, the single-center design and relatively modest sample size may introduce selection bias and limit the generalizability and external validity of our findings to other settings with different patient demographics or clinical practices. Second, the exclusion of parturients with comorbid disorders such as diabetes mellitus or hypertension restricts applicability to higher-risk pregnancies. Third, the reliance solely on umbilical pH, without concurrent evaluation of lactate concentrations or base deficit status, may constrain the depth of secondary metabolic assessment. Fourth, although TTN diagnosis adhered to widely standardized clinical criteria, the absence of extended postnatal pulmonary follow-up precludes future incorporated insights into longer-term respiratory consequences. Furthermore, it is imperative to emphasize that our study interpretations were explicitly confined to scheduled CS deliveries performed under neuraxial anesthesia. Therefore, our results may not be directly generalizable to emergency CS, where the pathophysiological dynamics of emergency procedures, characterized by distinct maternal-fetal stress states, altered anesthetic considerations, and time-pressured decision-making, are fundamentally different. Caution should also be warranted when extrapolating these findings to other institutions utilizing markedly different anesthesia or vasopressor protocols.

Considering the adverse effects of sustained spinal hypotension associated with prolonged anesthesia-to-delivery time on neonatal outcomes, and the high risk of TTN and neonatal acid-base imbalance, our study supports the adoption of a preventive protocol for maternal hypotension. Future large-scale multicenter studies encompassing heterogeneous maternal risk profiles are warranted to further delineate optimal thresholds for hypotension and spinal-to-delivery time and to evaluate the potential superiority of prophylactic vasopressor infusions in optimizing neonatal outcomes.

## Conclusion

Maternal hypotension secondary to spinal anesthesia represents a significant yet potentially

modifiable determinant of adverse neonatal outcomes, notably TTN and neonatal acidosis, in the context of scheduled cesarean deliveries. Therefore, protocolized preventative hemodynamic interventions, integrating fluid optimization and timely vasopressor administration, along with precise synchronization of anesthesia-to-delivery intervals, may substantially attenuate neonatal morbidity, underscoring the significance of targeted peripartum strategies to improve neonatal respiratory and metabolic outcomes following cesarean births.

## Acknowledgments

None.

## Conflicts of interest

All authors provide consent for the publication of this manuscript.

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