British Journal of Anaesthesia 1993; **70**: 311–316

PLASMA CATECHOLAMINES AND NEONATAL CONDITION AFTER INDUCTION OF ANAESTHESIA WITH PROPOFOL OR THIOPENTONE AT CAESAREAN SECTION

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SUMMARY

Increased maternal sympathetic nervous system activity may decrease placental perfusion and cause adverse neonatal effects. We have studied the catecholamine response and neonatal outcome in Chinese patients with uncomplicated, singleton pregnancies undergoing Caesarean section. Anaesthesia was induced with thiopentone 4 mg kg^{-1} (n = 32) or propofol 2 mg kg^{-1} (n = 30) followed by suxamethonium. Laryngoscopy was performed after 1 min and tracheal intubation completed by 2 min. Anaesthesia was continued with atracurium, nitrous oxide and isoflurane. Maternal venous blood samples were taken at 0, 1, 2, 3, 4 min and at delivery for assay of catecholamines. The increase from baseline values in mean arterial pressure after tracheal intubation was greater in the thiopentone group (29 (SD 15) mm Hg) compared with the propofol group (18 (14) mm Hg) ($P < 0.01$). The concentrations of noradrenaline and adrenaline increased in both groups after tracheal intubation. Maximum noradrenaline concentrations were greater in the thiopentone group (413 (177) pg ml^{-1}) compared with the propofol group (333 (108) pg ml^{-1}) ($P < 0.05$), but there were no differences between groups in adrenaline concentrations. Neonatal Apgar scores, neurobehavioural testing and umbilical catecholamine, blood-gas tension and oxygen content analysis were similar between groups. Propofol attenuated the hypertensive and catecholamine response associated with laryngoscopy and tracheal intubation but there was no improvement in neonatal outcome. (Br. J. Anaesth. 1993; **70**: 311–316)

KEY WORDS

Anaesthesia: obstetric. Anaesthetics, intravenous: propofol, thiopentone.

The uterine vascular bed during late pregnancy is considered to be maximally vasodilated, but responsive to stimuli causing vasoconstriction [1]. An increase in maternal concentrations of catecholamines can decrease uterine blood flow [2, 3] and this may affect the neonate adversely [4]. Plasma concentrations of catecholamines increased significantly after tracheal intubation in non-pregnant patients receiving thiopentone, but not in those receiving propofol [5, 6]. The prevention of this increase in catecholamine concentrations in obstetric patients may be beneficial for placental perfusion. In a sheep model, uterine blood flow decreased to less than 40% of control values in response to induction of anaesthesia with thiopentone and subsequent tracheal intubation, but there was no change in uterine blood flow when propofol was used [7]. Propofol has been shown to attenuate the cardiovascular response to laryngoscopy and tracheal intubation at Caesarean section [8]. However, that study assessed neonatal outcome only by Apgar scoring, which would have been unable to detect subtle depressant effects on the neonate.

We have compared the maternal catecholamine and cardiovascular response after rapid sequence induction of anaesthesia with propofol or thiopentone in patients undergoing elective Caesarean section. Neonatal outcome was assessed by neurobehavioural testing and umbilical cord blood-gas tension, oxygen content and catecholamine concentration analysis.

PATIENTS AND METHODS

The study was approved by the local Research Ethics Committee and informed consent was given by all patients. Sixty-one ASA I Chinese patients with uncomplicated, singleton pregnancies for elective Caesarean section were allocated randomly to receive either propofol or thiopentone for induction of anaesthesia. The indications for Caesarean section were breech presentation, cephalo–pelvic disproportion or previous Caesarean section. Patients were excluded if there was any evidence of intrauterine growth retardation or other fetal abnormality.

In both groups, ranitidine 150 mg was given the night before and on the morning of surgery, with 0.3-mol litre^{-1} sodium citrate 30 ml given 15 min before operation. Routine monitoring included end-tidal carbon dioxide and volatile agent concentration (Monitor 1304, Brüel & Kjaer). Arterial pressure was measured using an automatic oscillotonometer

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(Dinamap 1846SXP, Critikon) and the output recorded on the attached printer. Patients underwent preoxygenation for 3 min before rapid sequence induction of anaesthesia with thiopentone 4 mg kg^{-1} or propofol 2 mg kg^{-1} over 10 s followed by suxamethonium 1.5 mg kg^{-1}. Laryngoscopy was performed after the 1-min Dinamap recording and tracheal intubation completed before the 2-min reading. Anaesthesia was maintained with end-tidal concentrations of 50% nitrous oxide and 0.5% isoflurane in oxygen. Neuromuscular block was continued with atracurium 0.5 mg kg^{-1} and ventilation of the lungs controlled to maintain an end-tidal carbon dioxide concentration of 4.5%. The time taken for surgical preparation ensured that skin incision did not take place before the 4-min Dinamap recording. Hartmann's solution 500 ml was infused over the first 10 min. The induction to delivery (I–D) time and uterine incision to delivery (U–D) time were recorded by stopwatch. After delivery, oxytocin 10 u and morphine 0.2 mg kg^{-1} were given i.v.

Maternal venous blood samples (10 ml) were taken for assay of catecholamines immediately before induction of anaesthesia, at 1, 2, 3 and 4 min, and at the time of delivery. Umbilical venous (UV) and umbilical arterial (UA) blood samples were taken from a double-clamped segment of cord. Blood was put into lithium–heparin tubes containing metabisulphite as an antioxidant and placed in ice. These were centrifuged at 4 °C immediately after the last sample had been drawn, and the plasma separated and stored at −70 °C. Noradrenaline and adrenaline were measured by high pressure liquid chromatography within 2 weeks of collection. Catecholamines were extracted with alumina, analysed on a reversed-phase Ultrasphere IP C18 column (Beckman Instruments Incorporated, Altex Division, San Ramon, U.S.A.) and detected by an electrochemical method on an ESA 5100A coulometric detector (Environmental Science Associates, Bedford, U.S.A.). The within-day coefficients of variation for noradrenaline and adrenaline were 2.85% and 8.70%, respectively and the between-day coefficients of variation were 5.68% and 9.95%, respectively. The assay was linear to the lower limit of detection, which was 25 pg ml^{-1} for both noradrenaline and adrenaline.

UV and UA blood-gas tension and oxygen content analysis were carried out on a Ciba-Corning 288 Blood gas system (Ciba-Corning, Medfield, U.S.A.) and an IL-482 CO-Oximeter (Instrumentation Laboratory, Lexington, U.S.A.), with correction for 70% fetal haemoglobin.

Neonatal Apgar scores at 1 and 5 min and Neurological and Adaptive Capacity Scores (NACS) at 15 min and 2 h were assessed by a paediatrician who was unaware of the anaesthetic technique.

Statistical analysis was performed on a Macintosh IIci computer with the program Statview II, v1.04 (Abacus Concepts Inc, Berkeley CA, U.S.A.). Between groups, demographic data were compared using the unpaired *t* test, while time-related data, Apgar scores, NACS, blood-gas analysis data and catecholamine concentrations at delivery were compared using the Mann–Whitney test. Repeated measures analysis of variance was used to analyse the haemodynamic and catecholamine data after induction of anaesthesia. Peak catecholamine concentrations after tracheal intubation were taken as the greater of the 2- or 3-min values. Significant differences in the catecholamine values between groups were confirmed also using the Mann–Whitney test. $P < 0.05$ was considered significant. Kendall rank correlation was used to evaluate associations among I–D time, U–D time, catecholamine concentration at delivery, NACS and umbilical blood-gas values. Significant rank correlations were evaluated further by Pearson moment correlation, with log transformation of the time and catecholamine data because they were positively skewed, and $P < 0.01$ was considered significant because the sample size was relatively large. Results are presented as mean (SD).

RESULTS

There were no differences in age, weight or height between groups (table I). Eight patients in the

TABLE I. *Maternal characteristics (mean (range or SD)) and neonatal data (mean (SD) or median (range))*

	Thiopentone	Propofol
Mothers		
Age (yr)	30.6 (23–39)	30.9 (21–42)
Weight (kg)	63.7 (8.5)	63.0 (8.8)
Height (cm)	155 (6.0)	154 (5.1)
Neonates		
Induction to delivery time (min)	13.2 (3.9)	12.5 (3.6)
Uterine incision to delivery time (s)	81.4 (59.4)	66.8 (43.4)
Gestation (weeks)	38.6 (0.9)	38.4 (1.1)
Birth weight (kg)	3.23 (0.39)	3.10 (0.42)
Apgar score at 1 min	9 (6–10)	8.5 (6–10)
Apgar score at 5 min	10 (8–10)	10 (9–10)
NACS at 15 min	33 (28–36)	33 (29–36)
NACS at 2 h	35 (26–37)	35 (31–38)

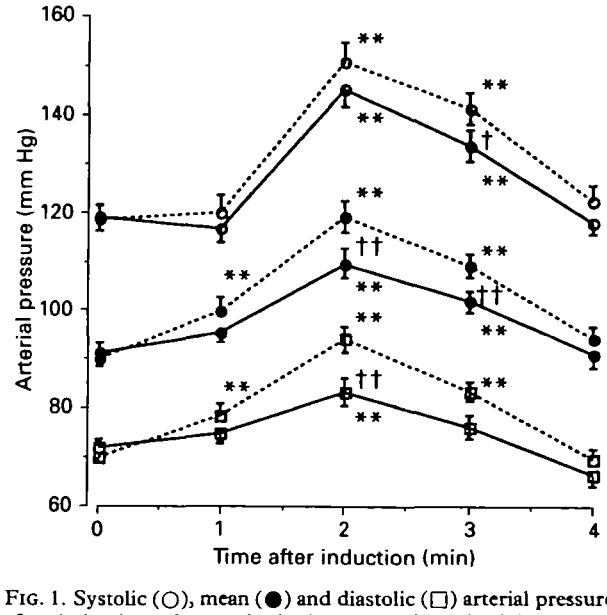

FIG. 1. Systolic (○), mean (●) and diastolic (□) arterial pressure after induction of anaesthesia (0 min) and tracheal intubation (1–2 min) after thiopentone (---) or propofol (——) ** $P < 0.01$ compared with initial values. † $P < 0.05$, †† $P < 0.01$ between groups.

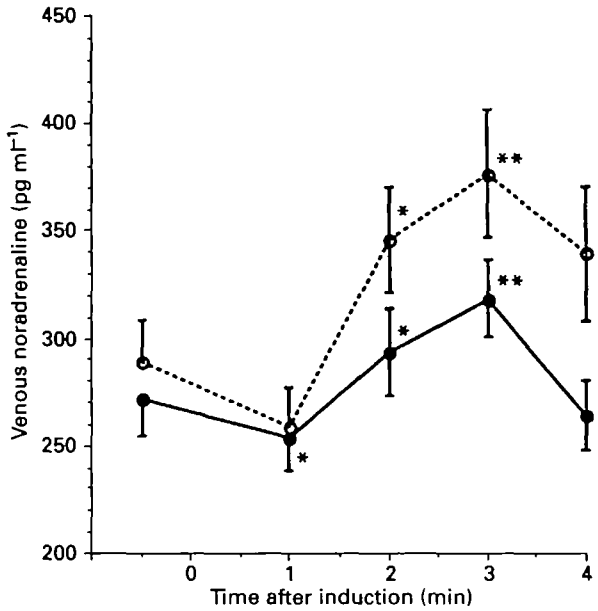

FIG. 2. Mean (SEM) venous concentrations of noradrenaline (pg ml^{-1}) after induction of anaesthesia (0 min) and tracheal intubation (1–2 min) after thiopentone (○) or propofol (●). * $P < 0.05$, ** $P < 0.01$ compared with initial values.

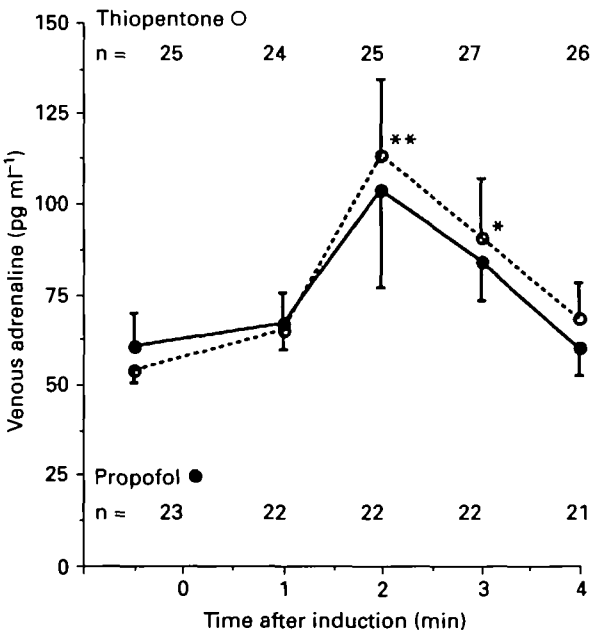

FIG. 3. Mean (SEM) venous concentrations of adrenaline (pg ml^{-1}) after induction of anaesthesia (0 min) and tracheal intubation (1–2 min) after thiopentone (○) or propofol (●). * $P < 0.05$, ** $P < 0.01$ compared with initial values. n = Sample size at each time.

thiopentone group and seven in the propofol group had fetal breech presentation.

Baseline arterial pressures were similar between groups. There was no change in arterial pressure after induction of anaesthesia and the greatest arterial pressures were recorded immediately after tracheal intubation (fig. 1). The increase in MAP from baseline values after tracheal intubation was greater in the thiopentone group (28.8 (15.2) mm Hg) than in the propofol group (18.0 (14.2) mm Hg) ($P < 0.01$).

Changes in heart rate were similar between groups. The heart rate increased immediately after induction of anaesthesia—from 84 (11) beat min^{-1} to 103 (15) beat min^{-1} in the thiopentone group and from 85 (13) beat min^{-1} to 102 (11) beat min^{-1} in the propofol group—and remained increased for at least 4 min.

Maternal concentrations of noradrenaline decreased after induction of anaesthesia in the propofol group and increased after tracheal intubation in both groups (fig. 2). The increase in noradrenaline from initial concentrations was greater in the thiopentone group ($P < 0.05$), with peak concentrations of 413 (177) pg ml^{-1}, compared with 333 (108) pg ml^{-1} in the propofol group ($P < 0.05$).

Analysis of the maternal adrenaline concentration data was more difficult because there were nine patients in the thiopentone group and 12 in the propofol group in whom the concentration was less than the lower limit of detection at some time during the first 4 min. These points were excluded from the comparison between groups. Concentrations of adrenaline were similar in both groups and increased after tracheal intubation (fig. 3). Peak concentrations were 110 (104) pg ml^{-1} in the thiopentone group ($n = 30$) and 114 (118) pg ml^{-1} in the propofol group ($n = 26$).

The I–D and U–D times were similar between groups (table I). There were two neonates in the thiopentone group and one in the propofol group with U–D times greater than 180 s. Exclusion of these neonates did not affect the conclusions, so their data have been included in the results. There were no differences in gestation or birth weight between groups (table I).

Because of logistic difficulties, complete NACS were assessed for only 25 neonates in each group. There were no differences in Apgar scores, NACS or umbilical blood-gas analysis between groups (tables I, II, fig. 4). Retrospective power analysis ($\alpha = 0.5$, $\beta = 0.1$) estimated that the study would have been

TABLE II. *Umbilical venous (UV) and umbilical arterial (UA) blood-gas analysis (mean (SD))*

	Thiopentone		Propofol	
	UV ($n = 31$)	UA ($n = 26$)	UV ($n = 30$)	UA ($n = 29$)
[H$^+$] (nmol litre^{-1})	47.21 (4.07)	52.07 (4.19)	45.86 (2.75)	51.44 (3.16)
P_{CO_2} (kPa)	5.75 (0.70)	6.76 (0.81)	5.64 (0.09)	6.60 (0.73)
P_{O_2} (kPa)	4.51 (0.96)	2.72 (0.60)	4.73 (1.01)	2.96 (0.71)
Base excess (mmol litre^{-1})	−2.6 (1.6)	−2.6 (1.9)	−2.3 (1.3)	−2.8(1.8)
HCO_3^- (mmol litre^{-1})	22.8 (1.7)	23.8 (2.4)	22.9 (1.5)	23.3 (2.5)
Hb (g dl^{-1})	13.5 (2.2)	13.4 (2.3)	12.6 (1.7)	12.6 (1.6)
O_2 saturation (%)	70.4 (14.3)	41.2 (15.0)	72.4 (15.0)	44.1 (15.0)
O_2 content (ml dl^{-1})	13.1 (3.1)	7.5 (2.9)	12.9 (3.3)	7.6 (2 8)

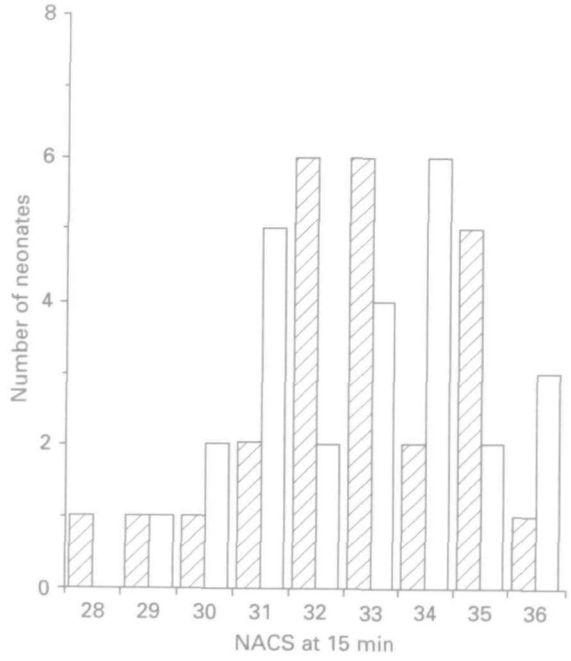

FIG. 4. Neonatal Neurological and Adaptive Capacity Scores at 15 min after delivery in the thiopentone (▨) and propofol (□) groups.

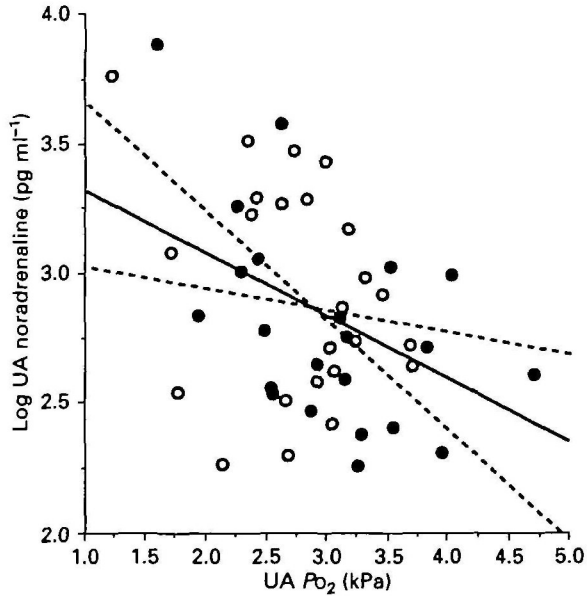

FIG. 5. Umbilical arterial (UA) P_{O_2} against log noradrenaline concentration in the thiopentone (○) and propofol (●) groups. —— = Linear regression line; --- = 95% confidence intervals for slope ($r = 0.43$, $P < 0.01$).

able to detect a difference between groups of two in the NACS. Neonates with breech presentation had a greater U–D time, smaller Apgar scores at 1 min and a lesser UA pH (all $P < 0.05$), but no differences were detected between the thiopentone and propofol groups.

One neonate in the thiopentone group with an NACS of 26 at 2 h was admitted to the neonatal intensive care for observation, but was discharged well without requiring treatment. There was only one neonate, from the thiopentone group, with a UA pH less than 7.2. This neonate had a breech presentation, a U–D time of 60 s, Apgar scores of 8 at 1 min, 9 at 5 min and NACS of 31 at 15 min and 33 at 2 h. The UA pH was 7.187, P_{CO_2} 8.87 kPa, P_{O_2} 1.22 kPa and oxygen content of 1.1 ml dl^{-1}. Concentrations of catecholamines were among the greatest, with noradrenaline concentrations in UA of 5793 pg ml^{-1} and in UV of 4090 pg ml^{-1} and adrenaline concentrations in UA of 533 pg ml^{-1} and in UV of 182 pg ml^{-1}. However, this neonate was well clinically and developed no problems after delivery.

There was insufficient blood to perform assay for catecholamines from six UA in each group and one UV in the thiopentone group. The concentration of adrenaline in the UV was below the lower limit of detection in most samples. Concentrations of noradrenaline and adrenaline were greater in the UA than in the UV and there were no differences between groups (table III). Noradrenaline and adrenaline data at delivery were positively skewed.

A prolonged I–D time was associated with greater maternal adrenaline concentration at delivery only in the thiopentone group ($r = 0.47$, $P < 0.01$). There were no significant correlations between I–D time and maternal noradrenaline concentration, umbilical catecholamine concentration, umbilical blood-gas analysis data or NACS.

A prolonged U–D time was associated with a lesser UA pH ($r = 0.64$), UV pH ($r = 0.51$), UA oxygen content ($r = 0.55$) and greater UA P_{CO_2} ($r = 0.60$) and UA noradrenaline concentration ($r = 0.53$) (all $P < 0.01$) in the propofol group. The correlation between U–D time and UA pH in the thiopentone group was $r = 0.39$ ($P = 0.048$). There were no significant correlations between U–D time and NACS.

There was no significant correlation between maternal concentrations of adrenaline and nor-

TABLE III. *Median (range) concentrations of noradrenaline (NA) and adrenaline (AD) at delivery in maternal venous (MV), umbilical arterial (UA) and umbilical venous (UV) blood. n.d. = Not detectable (< 25 pg ml^{-1}) and excluded; n = final sample size for listed results*

	Thiopentone group			Propofol group		
	n.d.	*n*	Concn (pg ml^{-1})	n.d.	*n*	Concn (pg ml^{-1})
MV–NA	—	32	290 (123–848)	—	30	281 (54–459)
MV–AD	1	31	119 (40–439)	3	26	160 (31–535)
UA–NA	—	26	899 (186–5793)	—	24	545 (183–7766)
UA–AD	—	26	149 (25–668)	2	22	99 (36–587)
UV–NA	—	31	240 (67–4090)	1	29	154 (25–969)
UV–AD	20	11	55 (28–186)	22	8	50 (28–112)

adrenaline at delivery and neonatal Apgar scores, NACS or umbilical blood-gas analysis data. Increased UV concentrations of noradrenaline were correlated with decreased UV pH ($r = 0.40$), increased UV P_{CO_2} ($r = 0.41$) and decreased UV P_{O_2} ($r = 0.54$) (all $P < 0.01$). Increased UA concentrations of noradrenaline were correlated with decreased UV and UA P_{O_2} (both $r = 0.43$, $P < 0.01$) (fig. 5).

DISCUSSION

In this study, propofol was more effective than thiopentone in attenuating the cardiovascular and catecholamine responses to tracheal intubation, as has been noted previously [8]. Significant hypotension was not observed because of the coincident stimuli of cricoid pressure and laryngoscopy [8]. Previous workers who reported no increase in catecholamine concentrations in patients receiving propofol [5, 6] did not take blood samples 2 min after tracheal intubation and may have missed the peak concentrations. The sample sizes in those studies were smaller and this would have made it more difficult to detect any increase in catecholamines. Propofol has been shown to inhibit sympathetic neural outflow in man [9] and reduce noradrenaline release from sympathetic nerve endings in dogs [10]. The changes in concentrations of catecholamines after tracheal intubation in our thiopentone group were similar to those reported in another study with similar patients and methodology [11].

Induction of anaesthesia with thiopentone decreased placental perfusion in humans [12]. In a sheep mode, uterine blood flow decreased significantly, to less than 40% of control values, in response to induction of anaesthesia with thiopentone and subsequent tracheal intubation, but there was no change when propofol was used [7]. However, a preliminary report in humans showed no difference in umbilical artery velocity waveforms after induction of anaesthesia either between propofol and thiopentone or with respect to time [13].

We postulated that an increase in the concentration of maternal catecholamines may adversely affect neonatal outcome. We did not measure catecholamine concentrations between 4 min and delivery, so we cannot be sure that there were no differences between groups during this time, even though concentrations at 4 min and at delivery were similar between groups. Nevertheless, the differences between groups in catecholamines and arterial pressure following induction of anaesthesia were short-lived and appeared to have no effect on neonatal condition. The concentrations of catecholamines at delivery in our study were similar to those reported previously after general anaesthesia [11, 14, 15], but the umbilical concentrations were much smaller than after vaginal delivery or Caesarean section under regional anaesthesia. Increased concentrations of umbilical catecholamines are thought to be a response to the fetal stress associated with delivery, with the greatest concentrations found after forceps and breech deliveries [16]. Hypoxia is a stimulus for the release of catecholamines and our results showed a correlation between increasing umbilical concentrations of noradrenaline and decreasing umbilical P_{O_2}. A prolonged U–D time during regional anaesthesia has been associated with decreased UA pH and increased UA noradrenaline concentration [17] and we found a similar correlation in the propofol group. Our results do not suggest that neonates in the thiopentone group tolerated a prolonged U–D time better than those in the propofol group because there were no differences in UA pH or noradrenaline concentration between groups. A long U–D time has been associated with decreased UA pH after general anaesthesia using thiopentone [18] and we would expect similar results if we studied more patients, because this correlation in our study ($r = 0.39$, $P = 0.053$) was almost statistically significant.

It has been suggested that an increased concentration of catecholamines in the neonate is beneficial because it provides better adaptability for extrauterine life [14, 15, 17]. However, a smaller concentration of catecholamines could imply that the fetus has not been stressed unnecessarily and is not acidotic and hypoxic. There appears to be no firm evidence supporting either viewpoint.

We found no difference in neonatal outcome between groups and preliminary reports of neurobehavioural assessment from other studies are in agreement [13, 19, 20]. However, lesser Early Neonatal Neurobehavioural Scale values have been reported after propofol 2.8 mg kg^{-1} compared with thiopentone 5.0 mg kg^{-1} [21]. We believe that the former induction dose, based on pregnant body weight, was excessive. Although more neonates in that propofol group showed cortical depression, irritability and a decreased withdrawal reflex at 1 h, all neonates were said to be predominantly awake and the overall condition was the same between groups. An induction dose of propofol 2.0 mg kg^{-1} is used by current investigators and a dose ratio for propofol:thiopentone of 1:2 is thought to be appropriate clinically.

The neonatal elimination of propofol appears to be more rapid than that of thiopentone [22] and this should contribute to faster recovery. Two to three days after delivery, neonates from a propofol group had better nutritive sucking behaviour compared with those from a thiopentone group [23].

All published studies have examined neonates after elective Caesarean section. Propofol has potentially the most benefit if it affords the least neonatal depressant effects when the fetus is already compromised by prematurity or complications of labour. The present findings justify the evaluation of propofol during emergency Caesarean section before judging the place of propofol in obstetric anaesthesia compared with the other available induction agents.

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