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Original Research Article

Efficacy and Safety of Thiopentone for Anaesthetic Induction in Children Younger Than Two Years: A Cross-Sectional Observational Study

Supriya[1], Shilpi Singh[2], Damodar Sadashiv Patwardhan[3]

[1,2,3]Department of Anaesthesia and Critical Care, Chirayu medical College and Hospital Bhopal, Madhya Pradesh, India

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Corresponding author: Dr. Supriya

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Abstract

Background: Children younger than two years are physiologically distinct and unusually sensitive to intravenous anaesthetics. Although thiopentone remains widely used for induction in resource-limited settings, contemporary efficacy and safety data in this age band are scarce. We evaluated its efficacy and safety in this group.

Methods: In this single-centre, cross-sectional observational study, 40 children younger than two years (American Society of Anesthesiologists [ASA] physical status I–II) received weight-based intravenous thiopentone for induction. Induction characteristics, serial haemodynamic and respiratory variables, intra-operative adverse events and recovery were recorded. Serial data were analysed with repeated-measures ANOVA or the Friedman test and associations with the Fisher exact test ($P < 0.05$ significant).

Results: Mean age was 10.8 ± 5.84 months and mean thiopentone dose 4.96 ± 0.55 mg/kg. Median onset was 38 s (IQR 34–43); intubating conditions were good or satisfactory in 95.0% and anaesthesia adequate in 97.5%. Systolic and mean arterial pressures fell to a nadir at 1 min (76.6 ± 9.4 and 59.7 ± 6.3 mmHg) and recovered by 5 min, with a mild compensatory rise in heart rate (all $P < 0.001$); oxygen saturation was preserved ($P = 0.110$). At least one adverse event occurred in 20.0% of children, most commonly hypotension and desaturation, all self-limiting. Any adverse event was strongly associated with ASA II status and emergency surgery (both $P < 0.001$).

Conclusion: Thiopentone provided rapid, reliable induction with transient, self-limiting cardiorespiratory changes and preserved oxygenation, supporting its carefully monitored use in this vulnerable group.

Keywords: Anaesthetic Induction; Cardiovascular Stability; Infant; Recovery; Thiopentone.

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Introduction

Anaesthesia in the first two years of life remains one of the more exacting responsibilities in our specialty. Infants and toddlers are not small adults: immature hepatic and renal pathways, a larger proportion of total body water, reduced plasma-protein binding and a respiratory system with little functional reserve together reshape how anaesthetic drugs distribute, act and are cleared.[1] [2] [3] These maturational differences narrow the margin between adequate anaesthesia and cardiorespiratory compromise, which is why the choice and titration of an induction agent in this group warrants continual, evidence-based scrutiny.[1] Thiopentone sodium, an ultra-short-acting thiobarbiturate, has occupied a central place in this story for almost a century. By potentiating inhibitory neurotransmission at the gamma-aminobutyric acid type A receptor — and, at higher concentrations, acting directly on the chloride channel — it produces a rapid, smooth loss of consciousness. Its high lipid solubility allows swift passage across the blood–brain barrier, so hypnosis typically follows within 30–45 s of an intravenous bolus, characteristics well suited to rapid induction and short procedures in children.[4] The wider pharmacological landscape has shifted. Propofol and sevoflurane are now preferred in many centres for their cleaner recovery profiles.[5] Yet thiopentone remains familiar, inexpensive and widely available, and across much of the developing world it continues to serve as a frontline paediatric induction agent.[6] Its profile in the youngest patients is not entirely benign: dose-dependent respiratory depression, hypotension from venodilation and myocardial depression, and occasional airway events have all been described, and infants with limited reserve are particularly exposed.[6] [7] Safe practice therefore rests on

close monitoring of heart rate, blood pressure, oxygen saturation, respiratory rate and recovery.[1] Despite this long clinical history, robust contemporary data describing how thiopentone performs — in efficacy and safety terms — in children younger than two years are surprisingly limited; existing reports are dominated by sedation for imaging or by comparisons in older children.[5] [6] Because patient characteristics, the urgency and type of surgery, and local practice all shape outcomes, a real gap persists between received wisdom and current evidence. We therefore undertook a cross-sectional observational study to characterise the efficacy and safety of thiopentone for anaesthetic induction in children under two years undergoing surgery, examining its induction characteristics, the trajectory of haemodynamic and respiratory variables across the early induction period, the frequency of adverse events, and recovery, with the aim of generating pragmatic evidence to guide safe practice in this vulnerable group.

Materials and Methods

Study design and setting: This single-centre, cross-sectional observational study was conducted in the Department of Anaesthesiology of a tertiary-care teaching hospital over a ten-month period. The protocol was approved by the Institutional Scientific and Ethics Committee, and the study was conducted in accordance with the Declaration of Helsinki and the Indian Council of Medical Research National Ethical Guidelines. Written informed consent was obtained from a parent or legal guardian of every child. Reporting follows the STROBE statement for observational studies.[8]

Participants: We enrolled 40 consecutive children younger than two years of age, of ASA physical status I or II,[9] scheduled for elective or emergency surgery under general anaesthesia in which thiopentone was used as the induction agent. Children with known hypersensitivity to barbiturates, significant congenital heart disease, severe respiratory or airway anomalies, hepatic or renal impairment, or neurological disorders affecting consciousness were excluded, as were those who received an alternative intravenous induction agent.

Sample size: The sample size was derived from the expected ~20% prevalence of perioperative adverse effects associated with thiopentone in children, using $n = Z^2pq/d^2$ ($Z = 1.96$; $p = 0.20$; $q = 0.80$; d = allowable error). This yielded a minimum of 39 patients, rounded to 40.

Anaesthetic protocol and data collection: After a standard pre-anaesthetic evaluation, children were kept fasting per paediatric guidelines. On arrival in theatre, baseline heart rate (HR), non-invasive systolic and diastolic blood pressure (SBP, DBP), mean arterial pressure (MAP), respiratory rate (RR) and peripheral oxygen saturation (SpO_2) were recorded. Anaesthesia was induced with intravenous thiopentone in a weight-based dose (4–6 mg/kg) titrated to loss of the eyelash reflex.[10] Demographic data, the thiopentone dose, onset time (injection to loss of eyelash reflex), ease of intubation (graded good, satisfactory or poor), and adequacy of anaesthesia were documented as efficacy measures. Safety variables comprised the occurrence of intra-operative adverse events — hypotension, bradycardia, respiratory depression, apnoea, bronchospasm, laryngospasm and desaturation — together with recovery characteristics (time to spontaneous eye opening and to response to stimuli) and postoperative nausea, vomiting or delayed recovery. HR, SBP, DBP, MAP, RR and SpO_2 were recorded at baseline, immediately after induction, and at 1, 3 and 5 min after thiopentone administration.

Statistical analysis: Data were compiled in a master chart and analysed in R version 4.2.2. The distribution of each continuous variable was assessed for normality using the Shapiro–Wilk test (selected because $n < 50$), supported by skewness and kurtosis. Normally distributed variables are summarised as mean ± SD and non-normally distributed variables as median (IQR). Serial measurements were compared across time using repeated-measures ANOVA when the variable was normally distributed at all time points, or the Friedman test otherwise; each time point was compared with baseline using the paired t-test or Wilcoxon signed-rank test, with Bonferroni correction. Categorical variables are expressed as frequency (%), and associations were tested with the Pearson chi-square or Fisher exact test. A two-tailed $P < 0.05$ was considered statistically significant.

Results

Demographic and baseline characteristics: Forty children were studied [Table 1]. The mean age was 10.8 ± 5.84 months (range 2–22) and mean weight 7.72 ± 1.84 kg; 25 (62.5%) were male. Most were ASA I (29; 72.5%) and underwent elective surgery (34; 85.0%), with a median fasting duration of 5 h. Twenty-one distinct procedures were represented, predominantly herniotomy, orchidopexy, palatoplasty and hypospadias repair.

Tests of normality: On Shapiro–Wilk testing, age, weight, heart rate and recovery times were normally distributed, whereas thiopentone dose, onset time, systolic pressure, MAP, respiratory rate and SpO_2 were non-normal, guiding the choice of subsequent tests [Table 2].

Induction Characteristics (Efficacy): The mean thiopentone dose was 4.96 ± 0.55 mg/kg (total 38.35 ± 10.19 mg), and the median onset of anaesthesia was 38 s (IQR 34–43) [Table 3]. Intubating conditions were good in 29 children (72.5%) and satisfactory in 9 (22.5%) — good or satisfactory in 95.0% overall — and anaesthesia was judged adequate in 39 (97.5%). Induction was haemodynamically stable in 35 children (87.5%).

Haemodynamic and Respiratory Trajectory: All cardiovascular variables changed significantly over the induction period [Table 4; Figure 1]. Heart rate rose modestly from 113.0 ± 10.8 bpm at baseline to a peak of 121.4 ± 12.1 bpm at 1 min before settling toward baseline by 5 min (repeated-measures ANOVA, $P < 0.001$). Systolic pressure and MAP followed a characteristic U-shaped course, falling to a nadir at 1 min (76.6 ± 9.4 and 59.7 ± 6.3 mmHg — reductions of roughly 16% from baseline) and recovering to near-baseline by 5 min (Friedman test, $P < 0.001$ for each); diastolic pressure behaved similarly. Respiratory rate fell from 30.9 ± 4.8 to a nadir of 19.8 ± 4.2 /min at 1 min before recovering ($P < 0.001$), while oxygen saturation remained essentially unchanged across all time points (99.1% to 98.5–99.0%; $P = 0.110$) [Figure 2].

Safety Profile — Adverse Events: At least one intra-operative adverse event occurred in 8 children (20.0%) [Table 5; Figure 3]. The commonest were hypotension and desaturation (each 3; 7.5%) and bradycardia (2; 5.0%); respiratory depression, apnoea, bronchospasm and laryngospasm each occurred once (2.5%). All events were transient and resolved with routine measures without sequelae.

Recovery and Postoperative Course: Spontaneous eye opening occurred at 12.26 ± 2.25 min and response to stimuli at 15.46 ± 2.36 min [Table 6]. Postoperative nausea and vomiting each affected 4 children (10.0%), and delayed recovery 3 (7.5%).

Exploratory Associations: Adverse events clustered strongly in higher-risk children [Table 7]. None of the 29 ASA I children experienced an adverse event, compared with 8 of 11 ASA II children (72.7%; Fisher exact $P < 0.001$). Likewise, adverse events occurred in only 2 of 34 elective cases (5.9%) but in all 6 emergency cases (100%; $P < 0.001$).

Table 1: Demographic and baseline characteristics (N = 40)

Characteristic	Value
Age (months)	10.8 ± 5.84 (range 2–22)
Sex – Male	25 (62.5%)
Sex – Female	15 (37.5%)
Weight (kg)	7.72 ± 1.84 (range 4.3–10.8)
ASA I	29 (72.5%)
ASA II	11 (27.5%)
Elective surgery	34 (85.0%)
Emergency surgery	6 (15.0%)
Fasting duration, NPO (h)	median 5 (range 4–8)
Distinct surgical procedures	21 types (predominantly herniotomy, orchidopexy, palatoplasty, hypospadias repair)

Values are mean ± SD or n (%) unless stated. ASA = American Society of Anesthesiologists physical status; NPO = nil per os.

Table 2: Shapiro–Wilk tests of normality for key continuous variables

Variable	W statistic	p-value	Distribution
Age (months)	0.952	0.087	Normal
Weight (kg)	0.968	0.317	Normal
Thiopentone dose (mg/kg)	0.943	0.045	Non-normal
Onset time (s)	0.932	0.018	Non-normal
Heart rate – baseline	0.952	0.090	Normal
Heart rate – 1 min	0.969	0.338	Normal
Systolic BP – baseline	0.930	0.016	Non-normal
Systolic BP – 1 min	0.915	0.005	Non-normal
MAP – 1 min	0.928	0.014	Non-normal
Respiratory rate – after induction	0.932	0.019	Non-normal
SpO_2 – baseline	0.798	<0.001	Non-normal
SpO_2 – after induction	0.617	<0.001	Non-normal
Time to eye opening (min)	0.984	0.836	Normal
Time to response (min)	0.975	0.497	Normal

Shapiro–Wilk test; P > 0.05 indicates a normal distribution. Representative variables shown; all haemodynamic time points were tested. BP = blood pressure; MAP = mean arterial pressure; SpO_2 = peripheral oxygen saturation.

Table 3: Induction characteristics (efficacy parameters)

Parameter	Value
Thiopentone dose (mg/kg)	4.96 ± 0.55 (range 4.1–6.0)
Total dose (mg)	38.35 ± 10.19
Onset of anaesthesia (s)	median 38 (IQR 34–43)
Ease of intubation: Good	29 (72.5%)
Ease of intubation: Satisfactory	9 (22.5%)
Ease of intubation: Poor	2 (5.0%)
Adequacy of anaesthesia: Adequate	39 (97.5%)
Induction haemodynamic stability: Stable	35 (87.5%)
Induction haemodynamic stability: Unstable	5 (12.5%)

Values are mean ± SD, median (IQR), or n (%).

Table 4: Haemodynamic and respiratory parameters across the induction period (mean ± SD)

Parameter	Baseline	After induction	1 min	3 min	5 min	Test	P-value
Heart rate (bpm)	113.0 ± 10.8	118.8 ± 10.9*	121.4 ± 12.1*	117.6 ± 11.0*	114.4 ± 11.5*	RM-ANOVA	<0.001
Systolic BP (mmHg)	90.9 ± 9.4	79.0 ± 9.2*	76.6 ± 9.4*	83.8 ± 9.1*	88.6 ± 9.5*	Friedman	<0.001
Diastolic BP (mmHg)	60.7 ± 5.3	52.8 ± 5.4*	51.2 ± 5.6*	56.0 ± 5.6*	59.1 ± 5.2*	Friedman	<0.001
MAP (mmHg)	70.8 ± 5.9	61.6 ± 6.2*	59.7 ± 6.3*	65.2 ± 6.1*	68.9 ± 6.0*	Friedman	<0.001
Respiratory rate (/min)	30.9 ± 4.8	20.9 ± 4.2*	19.8 ± 4.2*	25.4 ± 4.2*	27.7 ± 4.3*	Friedman	<0.001
SpO_2 (%)	99.1 ± 0.8	98.2 ± 2.1*	98.5 ± 2.4	98.8 ± 0.8	99.0 ± 0.9	Friedman	0.110

*P < 0.05 versus baseline (Bonferroni-corrected paired t-test or Wilcoxon signed-rank test). Overall P from repeated-measures ANOVA (heart rate) or the Friedman test (all others). BP = blood pressure; MAP = mean arterial pressure; SpO_2 = peripheral oxygen saturation.

Table 5: Frequency of intra-operative adverse effects (N = 40)

Adverse effect	n	%
Hypotension	3	7.5%
Bradycardia	2	5.0%
Respiratory depression	1	2.5%
Apnoea	1	2.5%
Bronchospasm	1	2.5%
Laryngospasm	1	2.5%
Desaturation	3	7.5%
Any adverse effect	**8**	**20.0%**

Patients could experience more than one event; the final row counts patients with ≥ 1 adverse effect.

Table 6: Recovery characteristics and postoperative complications

Parameter	Value
Time to spontaneous eye opening (min)	12.26 ± 2.25
Time to response to stimuli (min)	15.46 ± 2.36
Postoperative nausea	4 (10.0%)
Postoperative vomiting	4 (10.0%)
Delayed recovery	3 (7.5%)

Values are mean ± SD or n (%).

Table 7: Association of any adverse effect with ASA status and surgery type

Subgroup	Patients with ≥1 adverse effect	Test	P-value
ASA I	0 / 29 (0%)	Fisher's exact	<0.001
ASA II	8 / 11 (72.7%)		
Elective	2 / 34 (5.9%)	Fisher's exact	<0.001
Emergency	6 / 6 (100%)		

Fisher's exact test used because expected cell counts were < 5. The odds ratio was not estimable (zero events in one subgroup). ASA = American Society of Anesthesiologists physical status.

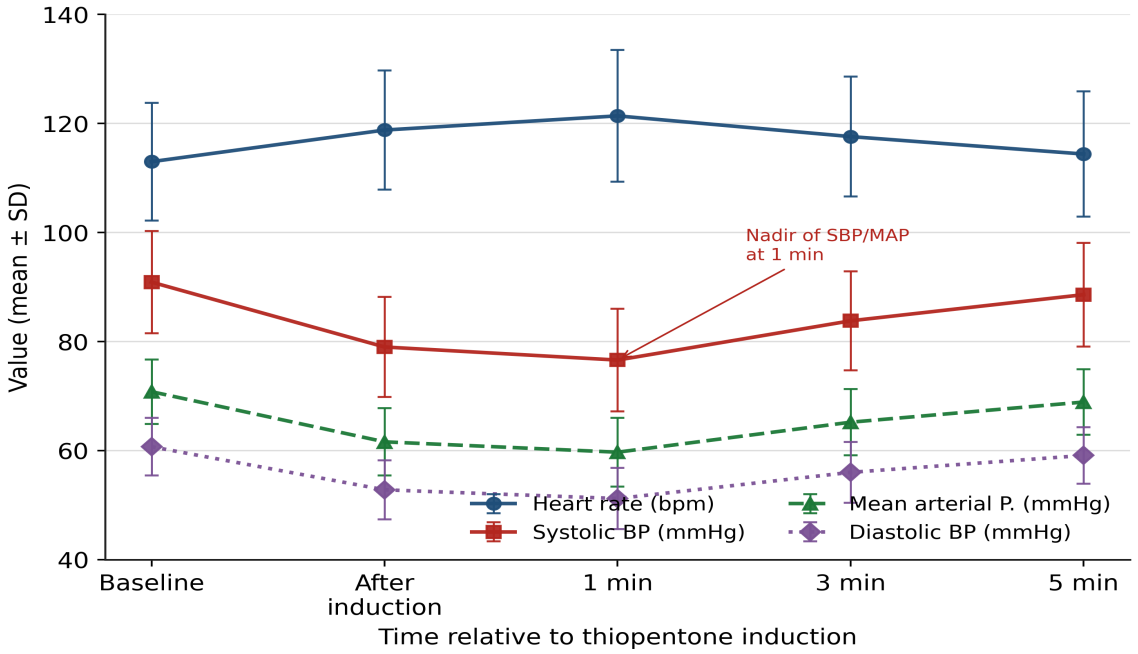

Figure 1: Haemodynamic trajectory (heart rate, systolic, diastolic and mean arterial pressure) across the induction period.

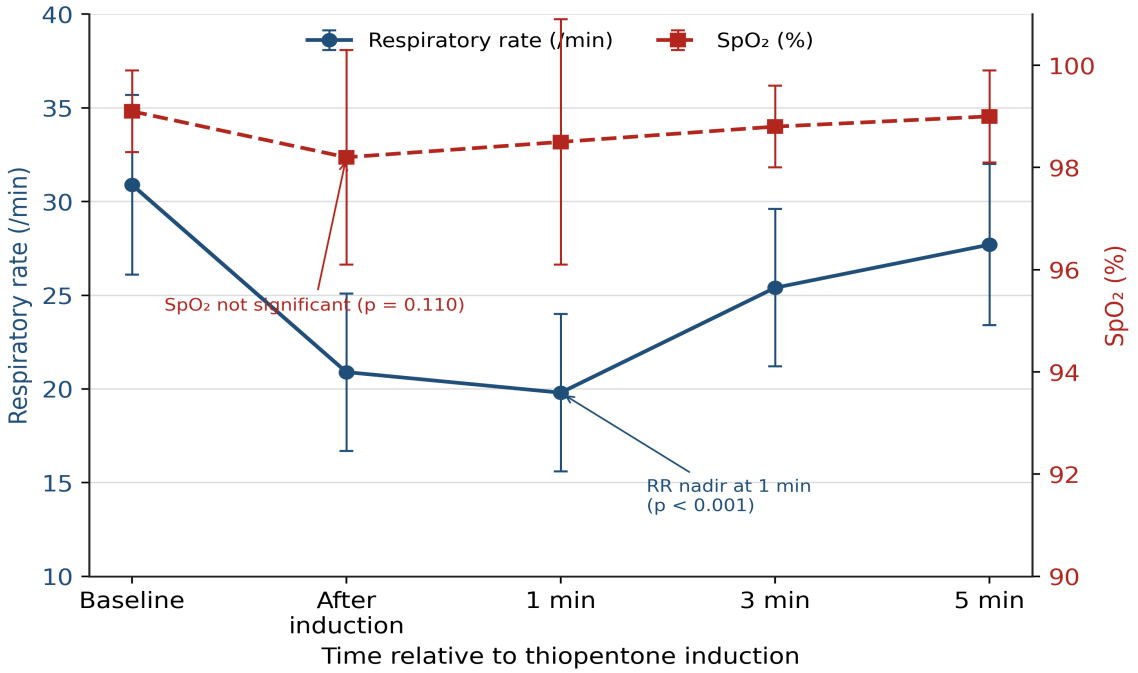

Figure 2: Respiratory rate and peripheral oxygen saturation (SpO_2) across the induction period.

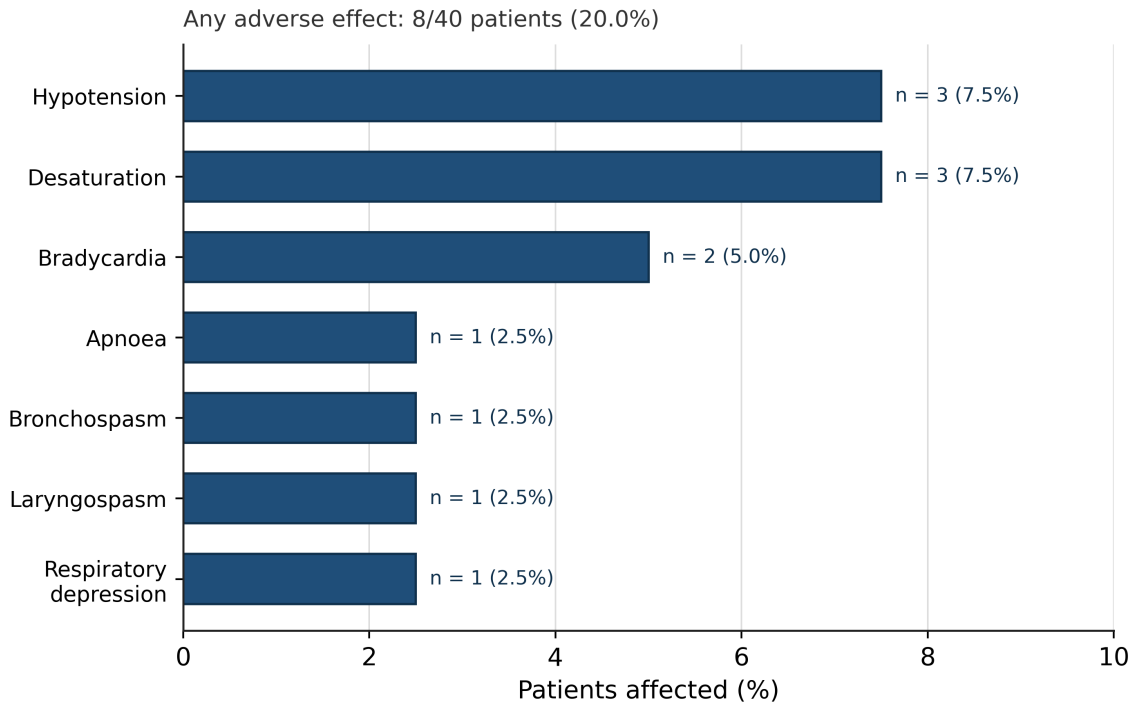

Figure 3: Frequency of intra-operative adverse effects (% of 40 children)

Discussion

In this cohort of children younger than two years, thiopentone delivered rapid and dependable induction: a median onset of 38 s and good-to-satisfactory intubating conditions in 95% mirror the drug's well-described pharmacology of swift brain uptake and smooth hypnosis.[4] The mean effective dose of 4.96 mg/kg sits comfortably within the range expected for this age. Classic dose-finding work established that thiopentone requirements are inversely related to age — an ED50 of about 3.4 mg/kg in neonates rising to 6–7 mg/kg in infants and falling again in older children — reflecting maturational changes in distribution volume and clearance.[10] [11] [12] [13] Our observed dose is therefore physiologically coherent and reassuringly consistent with prior paediatric data.

The haemodynamic signature we recorded — a modest, baroreflex-mediated rise in heart rate alongside a transient fall in arterial pressure that reached its nadir at 1 min and recovered by 5 min — is the expected consequence of thiopentone-induced venodilation and mild myocardial depression. The roughly 16% reduction in systolic pressure and MAP aligns closely with comparative work in children, in which thiopentone lowered mean arterial pressure by approximately 14–21%, a smaller decrement than that seen after propofol (28–31%),[14] which in children is additionally associated with injection pain and movement on induction.[18] This relative cardiovascular stability is echoed in adult and mixed-age studies comparing the two agents for induction.[15] [16] Importantly, oxygen saturation was preserved throughout (P = 0.110) despite a measurable dip in respiratory rate, consistent with the brief, manageable respiratory depression characteristic of a single induction dose under attentive airway management.

Recovery times — eye opening near 12 min and response to stimuli near 15 min — are in keeping with thiopentone's redistribution-driven offset and are predictably slower than those reported for propofol, which is associated with faster, more complete early recovery in children.[17] The modest incidence of postoperative nausea and vomiting (10%) is unsurprising in this age group and broadly within the range anticipated for general anaesthesia.[24]

From a safety perspective, an overall adverse-event rate of 20% warrants careful interpretation. The events were individually uncommon (each ≤ 7.5%), uniformly transient, and resolved with routine intervention — a pattern that speaks to predictable, manageable physiology rather than to idiosyncratic harm. Set against contemporary epidemiology, this is reassuring: the APRICOT study reported severe critical events in 5.2% of paediatric anaesthetics, with respiratory events predominating and the youngest children at greatest risk,[20] and respiratory adverse events are consistently the leading source of perioperative morbidity in children.[19] [27] Critical incidents, including cardiac arrest, are well documented at tertiary paediatric centres,[22] although medication-related cardiac arrest has become rare in modern practice.[21] [28] Our adverse events were

dominated by hypotension and desaturation — precisely the physiology one would predict from thiopentone — rather than by unexpected complications. The most clinically instructive finding is the stark clustering of adverse events by baseline risk. No ASA I child experienced an adverse event, whereas nearly three-quarters of ASA II children did, and every emergency case had at least one event. Although the small subgroup sizes and the inevitable confounding between ASA status and surgical urgency demand caution, the direction of effect is consistent with established literature: emergency surgery, comorbidity and younger age are recognised determinants of perioperative respiratory and cardiovascular events.[19] [23] The practical message is not that thiopentone is unsafe, but that its safe use is conditional — a stable ASA I child tolerates it well, while a physiologically compromised or emergency patient deserves heightened vigilance, judicious dosing and pre-emptive readiness to support the airway and circulation. This aligns with broader evidence that pharmacokinetic immaturity in infants and neonates prolongs drug effect and amplifies haemodynamic sensitivity, mandating individualised titration.[3] [25]

Taken together, our findings support a measured view of thiopentone's continuing role. In settings where propofol and sevoflurane are readily available, the cleaner recovery profile of newer agents is attractive.[5] But where access, cost or familiarity favour thiopentone — as in many parts of the developing world — our data indicate that it remains an effective and acceptably safe induction agent in children under two, provided that monitoring and clinical judgement are applied with the rigour the age group demands.[6] [26]

Limitations

This study has limitations. It was a single-centre, observational study without a comparator arm, so the efficacy and safety of thiopentone cannot be directly benchmarked against propofol or sevoflurane within the same population. The modest sample of 40 children, while adequate for the primary prevalence estimate, limits the precision of subgroup analyses; the association between adverse events and ASA II or emergency status, though striking, rests on small numbers and an odds ratio that was not estimable because one subgroup had zero events. ASA status and surgical urgency were also confounded. Anaesthetic depth was assessed clinically rather than with processed electroencephalography, and follow-up did not extend beyond the early postoperative period. Larger, multicentre, comparative studies are needed to confirm these observations and to define risk-stratified dosing.

Conclusion

In children younger than two years, thiopentone provided rapid, reliable anaesthetic induction with excellent intubating conditions and preserved oxygenation. The accompanying cardiovascular changes a transient fall in arterial pressure with a compensatory rise in heart rate — were modest, self-limiting and physiologically predictable, and adverse events, though present in one-fifth of children, were uniformly mild and concentrated among ASA II and emergency patients.

These findings support the continued, carefully monitored use of thiopentone for paediatric induction in this age group, particularly where newer agents are less accessible, while underscoring the need for individualised dosing and heightened vigilance in higher-risk children.

References

1. Sbaraglia F, Cuomo C, Della Sala F, Festa R, Garra R, Maiellare F, et al. State of the art in pediatric anesthesia: a narrative review about the use of preoperative time. J Pers Med. 2024;14(2):182. https://doi.org/10.3390/jpm14020182 (PMID: 38392615).
2. Gropper MA, Cohen NH, Eriksson LI, Fleisher LA, Leslie K, Wiener-Kronish JP, editors. Miller's Anesthesia. 9th ed. Philadelphia: Elsevier; 2020. Intravenous anesthetics.
3. Lu H, Rosenbaum S. Developmental pharmacokinetics in pediatric populations. J Pediatr Pharmacol Ther. 2014;19(4):262–76. https://doi.org/10.5863/1551-6776-19.4.262 (PMID: 25762871).
4. Kearns GL, Abdel-Rahman SM, Alander SW, Blowey DL, Leeder JS, Kauffman RE. Developmental pharmacology — drug disposition, action, and therapy in infants and children. N Engl J Med. 2003;349(12):1157–67. https://doi.org/10.1056/NEJMra035092 (PMID: 13679531).
5. Hong H, Hahn S, Choi Y, Jang MJ, Kim S, Lee JH, et al. Evaluation of propofol in comparison with other general anesthetics for surgery in children younger than 3 years: a systematic review and meta-analysis. J Korean Med Sci. 2019;34(15):e124. https://doi.org/10.3346/jkms.2019.34.e124 (PMID: 31001938).
6. Atalay YO, Leman T, Tobias JD. Efficacy and safety of intravenous thiopental for sedation during magnetic resonance imaging in pediatric patients: a retrospective analysis. Saudi J Anaesth. 2017;11(2):185–9. https://doi.org/10.4103/1658-354X.203086 (PMID: 28442957).
7. Vesselinova IV, Jensen KN, Hansen TG. Propofol and thiopental for intravenous induction in neonates: study protocol for a dose-finding trial. Acta Anaesthesiol Scand.

2023;67(6):820–8. https://doi.org/10.1111/aas.14238.
8. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596): 1453–7. https://doi.org/10.1016/S0140-6736(07)61602-X (PMID: 18064739).
9. Doyle DJ, Goyal A, Garmon EH. American Society of Anesthesiologists Classification. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK441940/ (PMID: 28722969).
10. Westrin P, Jonmarker C, Werner O. Thiopental requirements for induction of anesthesia in neonates and in infants one to six months of age. Anesthesiology. 1989;71(3):344–6. https://doi.org/10.1097/00000542-198909000-00004 (PMID: 2774260).
11. Jonmarker C, Westrin P, Larsson S, Werner O. Thiopental requirements for induction of anesthesia in children. Anesthesiology. 1987;67(1):104–7 (PMID: 3605730).
12. Coté CJ, Goudsouzian NG, Liu LM, Dedrick DF, Rosow CE. The dose response of intravenous thiopental for the induction of general anesthesia in unpremedicated children. Anesthesiology. 1981;55(6):703–5 (PMID: 7305057).
13. Stanski DR, Maitre PO. Population pharmacokinetics and pharmacodynamics of thiopental: the effect of age revisited. Anesthesiology. 1990;72(3):412–22. https://doi.org/10.1097/00000542-199003000-00003 (PMID: 2310488).
14. AlSaadawi MHH, Alkhafagy KAAHM, Al-jibawi MAIK. Comparative study of the effect of induction of general anesthesia with propofol versus thiopentone in pediatric age group on cardiovascular system. Cent Asian J Med Nat Sci. 2023;4(4):379–86. https://doi.org/10.51699/cajmns.v4i4.1709.
15. Aslan NA, Vural Ç, Yılmaz AA, Alanoğlu Z. Propofol versus thiopental for rapid-sequence induction in isolated systolic hypertensive patients: a factorial randomized double-blind clinical trial. Turk J Anaesthesiol Reanim. 2018;46(5):367–72. https://doi.org/10.5152/TJAR.2018.44442 (PMID: 30263860).
16. Thakur S, Sharma A, Kumar R, Pathania J, Thakur G. Comparative evaluation of propofol, thiopentone and etomidate on induction time, intubation response and recovery time during laparoscopic surgery under general anaesthesia. Indian J Clin Anaesth. 2023;10 (2):150–7.
17. Kamble VA, Shivakumar KG, Bhandi S, Mukund M. Recovery outcome of thiopentone vs. propofol as anaesthetic agent in paediatric patients of 2–10 years age group undergoing magnetic resonance imaging (MRI). Int J Med Anesthesiol. 2022;5(1):91–4.
18. Patel DK, Keeling PA, Newman GB, Radford P. Induction dose of propofol in children. Anaesthesia. 1988;43(11):949–52. https://doi.org/10.1111/j.1365-2044.1988.tb05659.x (PMID: 3266234).
19. von Ungern-Sternberg BS, Boda K, Chambers NA, Rebmann C, Johnson C, Sly PD, et al. Risk assessment for respiratory complications in paediatric anaesthesia: a prospective cohort study. Lancet. 2010;376(9743):773–83. https://doi.org/10.1016/S0140-6736(10)61193-2 (PMID: 20816545).
20. Habre W, Disma N, Virag K, Becke K, Hansen TG, Jöhr M, et al.; APRICOT Group of the European Society of Anaesthesiology Clinical Trial Network. Incidence of severe critical events in paediatric anaesthesia (APRICOT): a prospective multicentre observational study in 261 hospitals in Europe. Lancet Respir Med. 2017;5(5):412–25. https://doi.org/10.1016/S2213-2600(17)30116-9 (PMID: 28363725).
21. Bhananker SM, Ramamoorthy C, Geiduschek JM, Posner KL, Domino KB, Haberkern CM, et al. Anesthesia-related cardiac arrest in children: update from the Pediatric Perioperative Cardiac Arrest Registry. Anesth Analg. 2007;105(2):344–50. https://doi.org/10.1213/01.ane.0000268712.00756.dd (PMID: 17646488).
22. Lee JH, Kim EK, Song IK, Kim EH, Kim HS, Kim CS, et al. Critical incidents, including cardiac arrest, associated with pediatric anesthesia at a tertiary teaching children's hospital. Paediatr Anaesth. 2016;26(4):409–17. https://doi.org/10.1111/pan.12862 (PMID: 26956889).
23. Subramanyam R, Yeramaneni S, Hossain MM, Anneken AM, Varughese AM. Perioperative respiratory adverse events in pediatric ambulatory anesthesia: development and validation of a risk prediction tool. Anesth Analg. 2016;122(5):1578–85. https://doi.org/10.1213/ANE.0000000000001216 (PMID: 27101501).
24. Gan TJ, Belani KG, Bergese S, Chung F, Diemunsch P, Habib AS, et al. Fourth consensus guidelines for the management of postoperative nausea and vomiting. Anesth Analg. 2020;131(2):411–48. https://doi.org/10.1213/ANE.0000000000004833 (PMID: 32467512).
25. Larsson P, Anderson BJ, Norman E, Westrin P, Fellman V. Thiopentone elimination in newborn infants: exploring Michaelis-Menten

kinetics. Acta Anaesthesiol Scand. 2011;55(4): 444–51. https://doi.org/10.1111/j.1399-6576. 2011.02402.x (PMID: 21342153).
26. Tobias JD, Leder M. Procedural sedation: a review of sedative agents, monitoring, and management of complications. Saudi J Anaesth. 2011;5(4):395–410. https://doi.org/1 0.4103/1658-354X.87270 (PMID: 22144928).
27. Mamie C, Habre W, Delhumeau C, Argiroffo CB, Morabia A. Incidence and risk factors of perioperative respiratory adverse events in children undergoing elective surgery. Paediatr Anaesth. 2004;14(3):218–24. https://doi.org/ 10.1111/j.1460-9592.2004.01169.x (PMID: 14996260).
28. Ramamoorthy C, Haberkern CM, Bhananker SM, Domino KB, Posner KL, Campos JS, et al. Anesthesia-related cardiac arrest in children with heart disease: data from the Pediatric Perioperative Cardiac Arrest (POCA) registry. Anesth Analg. 2010;110(5):1376–82. https:// doi.org/10.1213/ANE.0b013e3181c9f927 (PMID: 20103543).