Original Article

Propofol Pain Reduction by Pre-Administration with 6% Hes (Hydroxyethyl Starch): A Prospective Randomized Double-Blinded Study

Ananya Sukul[1], Anindita Saha[2], Saurendra Nath Mitra[3], Arnab Chakraborty[4], Ratul Kundu[5]

[1]Senior Resident, Department of Anaesthesia, Barasat Government Medical College and Hospital, Banamalipur, Kolkata, West Bengal, India. [2]Consultant, Department of Anaesthesia, Ruby General Hospital, 576, Anandapur Main Road, Kasba, Kolkata, West Bengal, India. [3]Senior Consultant, Department of Anesthesia, Ruby General Hospital, 576, Anandapur Main Road, Kasba, Kolkata, West Bengal, India. [4]Post Graduate Trainee, Department of Anaesthesia, IQ City Medical College and Hospital, IQ City Knowledge & Health Campus, Mohunbagan Ave, Durgapur, West Bengal, West Bengal, India. [5]Consultant, Department of Anaesthesia, Rabindranath Tagore International Institute of Cardiac Sciences, Narayana Health, Premises Number 1489, 124, Eastern Metropolitan Bypass, Mukundapur Market, Stadium Colony, Mukundapur, Kolkata, West Bengal, West Bengal, India.

Abstract

Background: Propofol is one of the most common intravenous induction agents used in day-to- day anaesthesia practice. It is often used for maintenance of anaesthesia and sedation as well. The study aimed to compare pain reduction during propofol injection following pre-emptive administration of 50 ml of 6% HES or 0.9% NS in ASA I–II patients aged 18–60 years undergoing elective surgery under general anaesthesia. While assessing pain severity, changes in blood pressure, heart rate following induction and postoperative recall of injection pain were also assessed. **Material and Methods:** This was a prospective, randomized, double-blinded study conducted in the Operation Theatre of Ruby General Hospital, Kolkata, from August 2022 to August 2023.A total of 140 patients were included in the study. **Results:** A total of 140 patients (70 HES, 70 NS) were studied. Baseline age, sex, ASA grading, and propofol dose were comparable between groups. From this study it was found that pre administration of 50ml HES prior propofol induction reduced the pain during intravenous propofol injection significantly at 30th, 40th, 50th and 60th sec after propofol induction and also helped to maintain the hemodynamics significantly after 2nd and 3rd min of propofol injection. It was also observed that none of the patients taken for this study, remembered propofol injection pain in the postoperative period. **Conclusion:** The study showed that pre administration of 50ml bolus of 6% HES significantly decreased the pain on injection with propofol in comparison with NS. It was also observed that 50ml bolus of 6% HES provided better hemodynamic stability.

Keywords: Injection Pain, Hydroxyethyl Starch, HES, Normal Saline, Pain Reduction, Haemodynamic Stability.

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Introduction

Propofol is one of the most common intravenous induction agents used in day-to- day anaesthesia practice. It is often used for maintenance of anaesthesia and sedation as well. Its wide acceptability among anaesthesiologist is mainly due to its rapid onset, short duration of action, favorable titration and predictable side effects. However, despite its positive attributes and wide acceptability propofol is known for various adverse effects. One such non- life-threatening complication is the pain on injection of propofol. The incidence of propofol pain during injection is as high as 30-90%.[1] 3 out of 5 experience pain on injection of propofol with 1 of them complaining of excruciating pain.[2] For some pain at induction may be the most unpleasant sensation which they recall in the postoperative period. Propofol injection pain is considered as seventh commonest post-operative problem following anaesthesia.[3] Pain because of propofol injection occurs due to a complex mechanism and is considered multifactorial. Propofol is composed of Glycerol, 2,6-diisopropyl phenol, soyabean oil and egg lecithin.[4] It is commercially available as a lipid emulsion. This 2,6-diisopropyl phenol causes irritation of vein immediately after injection and this factor is attributed for the rapid onset of pain just after injection.[5] The lipid component of propofol i.e.,1% propofol contain 10% lipid emulsion, activates the plasma kallikrein-kinin system and releases bradykinin which modifies the local vein. This may increase the contact between propofol (aqueous phase) and free nerve endings of vessels that causes delayed propofol induced pain.[5] Further, general anaesthetic drugs cause release of Transient receptor potential ankyrin 1 (TRPA1) and transient receptor potential vanilloid 1 (TRPV1) that activates peripheral nerve endings. TRPA1ion channels are present in plasma membrane of cells that senses for irritants pain, cold and stretch. Activation of these TRPA1 and TRPV1 following anaesthesia are considered as main mediators of propofol induced pain.[6]

Address for correspondence: Dr. Ratul Kundu, Consultant, Department of Anaesthesia, Rabindranath Tagore International Institute of Cardiac Sciences, Narayana Health, Premises Number 1489, 124, Eastern Metropolitan Bypass, Mukundapur Market, Stadium Colony, Mukundapur, Kolkata, West Bengal, India
E-mail:drratulkundu@gmail.com

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Intravenous anaesthetic drugs also release neuropeptide from central and peripheral terminals of sensory neurons which in turn induces vascular leakage and may also be responsible for producing pain on injection. The study aimed to compare pain reduction during propofol injection following pre-emptive administration of 50 ml of 6% HES or 0.9% NS in ASA I–II patients aged 18–60 years undergoing elective surgery under general anaesthesia, while assessing pain severity, changes in blood pressure and heart rate, and postoperative recall of injection pain were also assessed.

Materials and Methods

Type of Study: A Prospective Randomized Double-Blinded Study

Place of Study: Operation Theatre of Ruby General Hospital, Kolkata

Study Duration: From August 2022 to August 2023

Sample Size: 140 patients

Inclusion Criteria:

- Age 18-60years
- Belonging to ASA physical status I and II
- Either Gender
- Undergoing elective surgery under General Anaesthesia

Exclusion Criteria:

- Emergency surgeries
- Allergic to propofol
- Hypertensive and diabetic
- Presence of Cardiac abnormalities
- Elevated serum creatinine
- Inaccessibility to forearm veins.
- IV access already secured with cannulas other than 18G.

Study Variables:

- Grades of pain caused during propofol injection were assessed by McCrirrick and Hunter scale till loss verbal response.
- Change in SBP, DBP, MAP and HR after propofol induction.
- Recalling of propofol injection pain in post-operative period in recovery.

Statistical Analysis: Data were entered into Excel and subsequently analyzed using SPSS and GraphPad Prism. Continuous variables were summarized as means with standard deviations, while categorical variables were presented as counts and percentages. Comparisons between independent groups were performed using two-sample t-tests, and paired t-tests were applied for correlated (paired) data. Categorical data were compared using chi-square tests, with Fisher's exact test applied when expected cell counts were small. A p-value of ≤ 0.05 was considered statistically significant.

Methodology: Propofol was given in patients of both groups by the allotted anaesthesiologists in pre-determined dose of 2 mg/kg body weight. The entire propofol was given over 60 secs. Pain was assessed by McCrirrick and Hunter scale, where 0 indicates no pain, 1 is mild pain, 2 is moderate pain and 3 is severe pain at interval of every 10 secs till 60 secs or till loss of verbal response. Allotted anaesthesiologist gave 50ml of HES or NS over 5 mins based on computer generated randomisation table before injecting propofol. Pain was assessed by the primary observer who was blinded to the type of HES or NS that was given pre emptively.

Results

Table 1: Comparison of Age, Sex and ASA grading between two groups (HES and NS)

Variable	Category	Hydroxyethyl Starch (HES)	Normal Saline (NS)	Total n (%)	p-value
Age group (years)	≤30	21 (30.0)	16 (22.9)	37 (26.4)	0.6908
	31–40	24 (34.3)	25 (35.7)	49 (35.0)	
	41–50	15 (21.4)	20 (28.6)	35 (25.0)	
	51–60	10 (14.3)	9 (12.9)	19 (13.6)	
Sex	Female	40 (57.1)	48 (68.6)	88 (62.9)	0.1617
	Male	30 (42.9)	22 (31.4)	52 (37.1)	
ASA grading	1	50 (71.4)	51 (72.9)	101 (72.1)	0.8504
	2	20 (28.6)	19 (27.1)	39 (27.9)	

Table 2: Comparison of Mean dose (D) OF PROPOFOL (mg) between HES and NS

Variable	Group	Mean	SD	Minimum	Maximum	Median	P-value
Dose of Propofol (mg)	Hydroxyethyl Starch (HES)	130.2571	18.3632	90	170	130	0.1152
	Normal Saline (NS)	125.6429	15.9928	90	160	130	

Table 3: Pain Response (PR) at 10th, 20th, 30th, 40th, 50th and 60th seconds in both HES and NS groups

		Grade 0	Grade 1	Grade 2	Grade 3		Chi-square	P-value
Hydroxyethyl starch (HES)	PR-10	70 (100.0)	0	0	0		NA	NA
	PR-20	70 (100.0)	0	0	0		NA	NA
	PR-30	69 (98.6)	1 (1.4)	0	0		66.0571	<0.0001
	PR-40	69 (98.6)	1 (1.4)	0	0		66.0571	<0.0001
	PR-50	68 (97.1)	2 (2.9)	0	0		62.2285	<0.0001
	PR-60	58 (82.9)	12 (17.1)	0	0		30.2285	0.8401
Normal Saline (NS)	PR-10	67 (95.7)	3 (4.3)	0	0		58.5142	<0.0001
	PR-20	50 (71.4)	20 (28.6)	0	0		12.8571	0.0003

	PR-30	26 (37.1)	40 (57.1)	4 (5.7)	0		28.2285	7.4172
	PR-40	10 (14.3)	40 (57.1)	20 (28.6)	0		20.0000	0.0004
	PR-50	6 (8.6)	30 (42.9)	32 (45.7)	0		42.2285	0.3588
	PR-60	5 (7.1)	20 (28.6)	34 (48.6)	9 (12.9)		49.0000	0.5841

Table 4: Comparison of the mean pain response (MPR) between HES and NS groups at 10th,20th,30th,40th,50th and 60th seconds

		Number	Mean	SD	Minimum	Maximum	Median	P-value	T Statistic
MPR-10	Hydroxyehtylstarch (HES)	70	.0000	.0000	0.0000	0.0000	0.0000	0.0810	1.7577
	Normal Saline (NS)	70	.0429	.2040	0.0000	1.0000	0.0000		
MPR-20	Hydroxyehtylstarch	70	.0000	.0000	0.0000	0.0000	0.0000	<0.0001	5.2536
	Normal Saline	70	.2857	.4550	0.0000	1.0000	0.0000		
MPR-30	Hydroxyehtylstarch	70	.0143	.1195	0.0000	1.0000	0.0000	<0.0001	9.5109
	Normal Saline	70	.6857	.5784	0.0000	2.0000	1.0000		
MPR-40	Hydroxyehtylstarch	70	.0143	.1195	0.0000	1.0000	0.0000	<0.0001	14.4268
	Normal Saline	70	1.1429	.6435	0.0000	2.0000	1.0000		
MPR-50	Hydroxyehtylstarch	70	.0286	.1678	0.0000	1.0000	0.0000	<0.0001	15.0855
	Normal Saline	70	1.4571	.7743	0.0000	4.0000	1.0000		
MPR-60	Hydroxyehtylstarch	70	.1714	.3796	0.0000	1.0000	0.0000	<0.0001	13.9049
	Normal Saline	70	1.7571	.8754	0.0000	4.0000	2.0000		

Table 5: Comparison of mean of BSBP (basal systolic blood pressure) and SBP(systolic blood pressure) at 1st, 2nd, 3rd min after propofol induction: GROUP HES and NS

Variable	Group	Mean	SD	Minimum	Maximum	Median	P-value
BSBP (mm of Hg)	Hydroxyethyl Starch (HES)	139.2857	13.213	105	178	136.5	0.0964
	Normal Saline (NS)	143.4714	16.2157	107	174	144.5	
SBP-1 (mm of Hg)	Hydroxyethyl Starch (HES)	129.0286	15.5218	93	165	127	0.0986
	Normal Saline (NS)	124.6857	15.376	90	170	125	
SBP-2 (mm of Hg)	Hydroxyethyl Starch (HES)	122.2286	13.1504	89	157	121	0.0015
	Normal Saline (NS)	114.6286	14.5095	83	171	114	
SBP-3 (mm of Hg)	Hydroxyethyl Starch (HES)	120.1714	13.3275	84	157	118	<0.0001
	Normal Saline (NS)	109.1571	11.7912	73	146	107	

Table 6: Comparison of mean of BHR (basal heart rate) and mean of HR (heart rate) at 1st,2nd and 3rd min after propofol induction between HES and NS groups

Variable	Group	Mean	SD	Minimum	Maximum	Median	P-value
BHR (bpm)	Hydroxyethyl Starch (HES)	88.2286	12.0713	64	120	88	0.059
	Normal Saline (NS)	92.2714	13.0361	65	123	92.5	
HR-1 (bpm)	Hydroxyethyl Starch (HES)	87.5429	10.9143	58	115	87	0.5315
	Normal Saline (NS)	86.3714	11.1799	66	118	85	
HR-2 (bpm)	Hydroxyethyl Starch (HES)	84.5	10.1706	61	116	83	0.3994
	Normal Saline (NS)	82.9	12.1395	65	121	80	
HR-3 (bpm)	Hydroxyethyl Starch (HES)	82.2857	9.8865	62	115	81.5	0.0126
	Normal Saline (NS)	77.6857	11.569	59	110	76	

Table 7: Comparison of mean BMAP (basal mean arterial pressure) and MAP(mean arterial pressure) at 1st, 2nd, and 3rd min after propofol induction: GROUP HES and NS

Variable	Group	Mean	SD	Minimum	Maximum	Median	P-value
BMAP (mm of Hg)	Hydroxyethyl Starch (HES)	96.4429	9.579	75	114	97	0.835
	Normal Saline (NS)	96.7857	9.8627	73	121	98	
MAP-1 (mm of Hg)	Hydroxyethyl Starch (HES)	89.1286	10.9332	65	109	89.5	0.1221
	Normal Saline (NS)	86.2143	11.23	61	113	85	
MAP-2 (mm of Hg)	Hydroxyethyl Starch (HES)	86.9	11.4489	60	120	85.5	0.0004
	Normal Saline (NS)	79.6714	11.9245	52	120	79	
MAP-3 (mm of Hg)	Hydroxyethyl Starch (HES)	85.0143	10.4222	65	107	85.5	<0.0001
	Normal Saline (NS)	75.2571	9.5791	53	103	76.5	

A total of 140 patients were included in the study, with 70 patients in the Hydroxyethyl Starch (HES) group and 70 patients in the Normal Saline (NS) group.

[Table 1] shows that the age, sex, and ASA grading distributions were comparable between the HES and NS groups. The majority of patients were aged 31–40 years (49, 35.0%), followed by ≤30 years (37, 26.4%), 41–50 years (35, 25.0%), and 51–60 years (19, 13.6%). Females constituted 88 (62.9%) patients and males 52 (37.1%). Most patients were ASA grade I (101, 72.1%), while 39 (27.9%) were ASA grade II. There were no statistically significant differences between the groups with respect to age (p=0.6908), sex (p=0.1617), or ASA grading (p=0.8504), indicating good baseline comparability.

[Table 2] demonstrates that the mean dose of propofol administered was 130.26 ± 18.36 mg in the HES group and 125.64 ± 15.99 mg in the NS group. The median dose was 130 mg in both groups. The difference in mean propofol dose was

not statistically significant (p=0.1152), suggesting comparable propofol exposure between the two groups.

[Table 3] has depicted the pain response (PR) in HES group and NS group at 10th, 20th, 30th, 40th, 50th and 60th seconds. In HES group none of the patients at 10th and 20th seconds did report any pain (grade 0). At 30th and 40th seconds 69 patients did not complain of pain (grade 0) and 1 patient complained pain of grade 1. At 50th second 68 patients were with no pain (grade 0) and 2 patients were with grade 1. Similarly, at 60th second in HES group 58 patients were with no pain (grade 0) and 12 patients complained of grade 1. None of the patients had grade 2 or 3 pain response at any point of time in HES group.

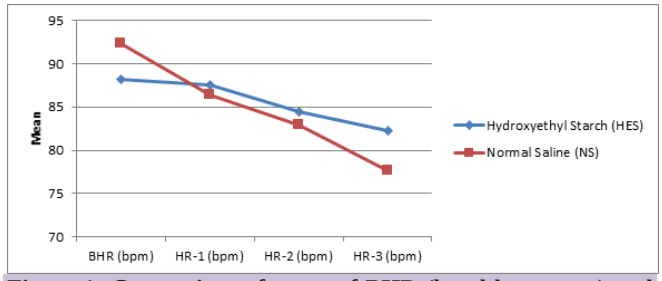


Figure 1: Comparison of mean of BHR (basal heart rate) and mean of HR (heart rate) at 1st, 2nd and 3rd min after propofol induction between HES and NS groups

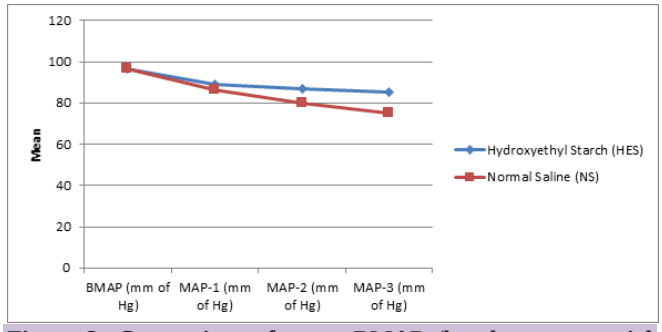


Figure 2: Comparison of mean BMAP (basal mean arterial pressure) and MAP(mean arterial pressure) at 1st, 2nd, and 3rd min after propofol induction: GROUP HES and NS

In NS group at 10th second 67 patients were with no pain (grade 0) and 3 patients complained of grade 1 pain. At 20th sec 50 patients were with no pain (grade 0) and 20 patients were with grade 1. Pain response at 30th second in NS group, were no pain (grade 0) in 26 patients, grade 1 in 40 patients and grade 2 in 4 patients. At 40th second 10 patients complained of no pain (grade 0), 40 patients complained grade 1 and 20 patients complained grade 2 with none of grade 3 pain. Similarly, at the 50th second in the NS group 6 patient were having no pain (grade 0), 30 were having grade 1, 32 were having grade 2 and none had grade 3. At 60th sec in NS group 5 patients did not experience pain (grade 0), 20 patients experienced grade 1, 34 patients had grade 2 and as many as 9 patients experienced grade 3 pain.

[Table 4] presents the comparison of mean pain response (MPR) between the HES and NS groups at 10th, 20th, 30th, 40th, 50th and 60th seconds. Consistent with the grade-wise pain response observed in Table 3, the mean pain response was lower in the HES group at all assessment points. At the 10th second, the mean pain response was 0.00 ± 0.00 in the HES group and 0.04 ± 0.20 in the NS group. The difference was not statistically significant (p=0.0810). At the 20th second, the mean pain response remained 0.00 ± 0.00 in the HES group, whereas it increased to 0.29 ± 0.46 in the NS group, with a statistically significant difference (p<0.0001). At the 30th second, the mean pain response was 0.01 ± 0.12 in the HES group compared with 0.69 ± 0.58 in the NS group, and the difference was statistically significant (p<0.0001). At the 40th second, the mean pain response remained 0.01 ± 0.12 in the HES group, while it increased to 1.14 ± 0.64 in the NS group (p<0.0001). At the 50th second, the mean pain response was 0.03 ± 0.17 in the HES group compared with 1.46 ± 0.77 in the NS group, showing a statistically significant difference (p<0.0001). At the 60th second, the mean pain response further increased to 1.76 ± 0.88 in the NS group, whereas it remained considerably lower at 0.17 ± 0.38 in the HES group (p<0.0001).

[Table 5] presents the comparison of systolic blood pressure. Baseline systolic blood pressure was comparable between the HES and NS groups (139.29 ± 13.21 vs. 143.47 ± 16.22 mmHg; p=0.0964). At 1 minute after induction, the mean SBP was 129.03 ± 15.52 mmHg in the HES group and 124.69 ± 15.38 mmHg in the NS group, with no statistically significant difference (p=0.0986). At 2 minutes, SBP was significantly higher in the HES group (122.23 ± 13.15 vs. 114.63 ± 14.51 mmHg; p=0.0015). Similarly, at 3 minutes, the HES group had significantly higher SBP than the NS group (120.17 ± 13.33 vs. 109.16 ± 11.79 mmHg; p<0.0001).

[Table 6] compares heart rate between the two groups. Baseline heart rate was comparable (88.23 ± 12.07 vs. 92.27 ± 13.04 bpm; p=0.059). At 1 minute, there was no significant difference in heart rate between the HES and NS groups (87.54 ± 10.91 vs. 86.37 ± 11.18 bpm; p=0.5315), and similarly at 2 minutes (84.50 ± 10.17 vs. 82.90 ± 12.14 bpm; p=0.3994). However, at 3 minutes, the mean heart rate was significantly higher in the HES group (82.29 ± 9.89 vs. 77.69 ± 11.57 bpm; p=0.0126).

[Table 7] shows the comparison of mean arterial pressure. Baseline MAP was comparable between the HES and NS groups (96.44 ± 9.58 vs. 96.79 ± 9.86 mmHg; p=0.835). At 1 minute, the difference was not statistically significant (89.13 ± 10.93 vs. 86.21 ± 11.23 mmHg; p=0.1221). However, at 2 minutes, MAP was significantly higher in the HES group (86.90 ± 11.45 vs. 79.67 ± 11.92 mmHg; p=0.0004), and this difference remained highly significant at 3 minutes (85.01 ± 10.42 vs. 75.26 ± 9.58 mmHg; p<0.0001). Overall, the haemodynamic findings indicate that both groups showed reductions in SBP and MAP following propofol induction, but the decline was significantly greater in the NS group at 2 and 3 minutes, while heart rate remained largely comparable except at 3 minutes.

Discussion

The present prospective randomized double-blinded study included 140 patients, with 70 patients in the 6% hydroxyethyl starch (HES) group and 70 patients in the 0.9% normal saline (NS) group. The baseline characteristics demonstrated good

comparability between the two groups. There was no statistically significant difference in age distribution (p=0.6908), sex distribution (p=0.1617), or ASA physical status (p=0.8504), indicating that the two groups were adequately matched with respect to important demographic and clinical variables. The mean dose of propofol was also comparable between the HES and NS groups (130.26 ± 18.36 mg vs. 125.64 ± 15.99 mg, respectively; p=0.1152), suggesting that differences in subsequent haemodynamic responses were unlikely to be attributable to variation in propofol dose. The present study demonstrated a favourable haemodynamic profile following HES pre-administration. Baseline systolic blood pressure was comparable between the HES and NS groups (139.29 ± 13.21 vs. 143.47 ± 16.22 mmHg; p=0.0964), while SBP at 1 minute after induction was also not significantly different (129.03 ± 15.52 vs. 124.69 ± 15.38 mmHg; p=0.0986). However, at 2 minutes, SBP was significantly higher in the HES group than in the NS group (122.23 ± 13.15 vs. 114.63 ± 14.51 mmHg; p=0.0015), and this difference became more pronounced at 3 minutes (120.17 ± 13.33 vs. 109.16 ± 11.79 mmHg; p<0.0001), indicating better preservation of systolic blood pressure after propofol induction among patients receiving HES. A similar haemodynamic benefit has been reported in previous studies investigating HES before propofol administration. Misra et al,.[7] in a randomized placebo-controlled study involving ASA I–II patients, administered 6% HES or NS through an 18G cannula before propofol-lidocaine induction and demonstrated that HES significantly reduced propofol injection pain while maintaining a favourable haemodynamic profile. Their study reported pain in 28% of patients receiving HES compared with 53% receiving NS (p=0.004), supporting the beneficial effect of HES pretreatment.[7] More recently, Sahoo et al,.[8] in a randomized placebo-controlled trial involving 339 patients, found that pre-administration of 100 mL 6% HES resulted in a significantly greater proportion of patients experiencing no pain during propofol injection compared with both lidocaine and placebo groups, while haemodynamic variables were also assessed before and after induction.[8] Varghese et al,.[9] in a 2025 comparative study of 70 ASA I–II patients, similarly reported that pre-administration of 6% HES was associated with reduced propofol injection pain and acceptable haemodynamic stability when compared with 2% lidocaine.[9] The MAP findings in the present study further support the haemodynamic advantage of HES. Baseline MAP was almost identical between the groups (96.44 ± 9.58 mmHg in HES vs. 96.79 ± 9.86 mmHg in NS; p=0.835), confirming baseline haemodynamic comparability. At 1 minute, MAP remained statistically comparable (89.13 ± 10.93 vs. 86.21 ± 11.23 mmHg; p=0.1221); however, at 2 minutes, MAP was significantly higher in the HES group (86.90 ± 11.45 vs. 79.67 ± 11.92 mmHg; p=0.0004), and at 3 minutes the difference was highly significant (85.01 ± 10.42 vs. 75.26 ± 9.58 mmHg; p<0.0001). The simultaneous preservation of both SBP and MAP suggests that HES pre-administration may attenuate the reduction in arterial pressure commonly associated with propofol induction. This may be explained by the intravascular volume-expanding properties of HES, which can help maintain effective circulating volume during the vasodilatory phase of propofol induction. In contrast, NS provides substantially less colloid-mediated intravascular volume expansion, which may partly explain the greater fall in SBP and MAP observed in the NS group. Heart rate findings were generally comparable between groups. Baseline HR was 88.23 ± 12.07 bpm in the HES group and 92.27 ± 13.04 bpm in the NS group (p=0.059), while HR at 1 minute (87.54 ± 10.91 vs. 86.37 ± 11.18 bpm; p=0.5315) and 2 minutes (84.50 ± 10.17 vs. 82.90 ± 12.14 bpm; p=0.3994) showed no significant differences. At 3 minutes, however, HR was significantly higher in the HES group than in the NS group (82.29 ± 9.89 vs. 77.69 ± 11.57 bpm; p=0.0126). Despite statistical significance, the absolute difference was modest, and the mean HR in both groups remained within a clinically acceptable range. The relative preservation of HR together with significantly better SBP and MAP suggests that HES may provide greater cardiovascular stability during the early post-induction period. The findings are particularly relevant because propofol-induced hypotension is a well-recognized consequence of induction and is related predominantly to vasodilatation and reduction in vascular tone. The ability of HES to maintain arterial pressure may therefore represent an additional benefit when used before propofol administration in appropriately selected patients. Importantly, the recent study by Sahoo et al,.[8] also specifically evaluated pre- and post-induction haemodynamic variables and concluded that 6% HES was effective in reducing propofol injection pain while providing a favourable haemodynamic profile.[8] The analgesic mechanism proposed for HES is different from that of conventional local anaesthetic pretreatment. Propofol injection pain is thought to involve activation of vascular endothelial pathways and contact activation by nociceptive substances, while HES may inhibit endothelial membrane activation and thereby reduce the nociceptive response to propofol.[7,8] Overall, the findings of the present study indicate that pre-administration of 50 mL 6% HES was associated with better maintenance of SBP and MAP at 2 and 3 minutes following propofol induction, with largely comparable heart-rate responses and no significant difference in propofol dose between groups. These findings are consistent with recent evidence supporting HES as a potential strategy for reducing propofol injection pain and maintaining haemodynamic stability.[7–9] Nevertheless, the findings should be interpreted within the context of the study population, which consisted of relatively healthy ASA I–II patients undergoing elective surgery.[10] Given the recognized safety concerns associated with hydroxyethyl starch, particularly in patients with renal dysfunction, sepsis, or critical illness, further large-scale studies are warranted to establish the safety and generalizability of this approach.

Conclusion

To conclude pre administration of 50 ml 6% hydroxyethyl starch before induction with propofol is likely to reduce propofol injection pain. It can also be concluded that 50ml hydroxyethyl starch provides a better hemodynamic stability during propofol induction when compared to 50ml of normal

saline. However hemodynamic changes due to propofol induction did not need any interventions in ASA 1 and ASA 2 patients in this study. Lastly our study also depicts that none of the patients remembered propofol induction pain in the postoperative period and hence further evaluation will be required.

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Nil.

Conflicts of interest
There are no conflicts of interest.

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