RESEARCH PAPER

The Study on Enhanced Hypnotic Effect of Intravenous Propofol on Administration of Lignocaine

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ABSTRACT

Background: Propofol is widely used for induction of anaesthesia due to its rapid onset and favourable recovery profile; however, its use is limited by dose-dependent hypotension, respiratory depression, and injection pain. Intravenous lignocaine, beyond its role as a local anaesthetic, has demonstrated analgesic and anaesthetic-sparing properties.

Aim: To evaluate whether intravenous lignocaine enhances the hypnotic efficacy of propofol during induction of anaesthesia.

Methods: In this randomized, double-blind, placebo-controlled study, 62 ASA I–II patients aged 18–60 years undergoing elective surgery were allocated into two groups. The intervention group received intravenous lignocaine (1.5 mg/kg), while the control group received normal saline. After 2 minutes, propofol was administered in titrated aliquots until loss of verbal response. Primary outcome was propofol dose required for induction. Secondary outcomes included induction time, haemodynamic parameters, induction characteristics, and adverse effects.

Results: Propofol requirement was significantly reduced in the lignocaine group (1.50 ± 0.00 mg/kg) compared to the control group (2.07 ± 0.27 mg/kg; $p < 0.001$). Total dose and induction time were also significantly lower. Patients receiving lignocaine demonstrated smoother induction and better haemodynamic stability, with reduced incidence of hypotension. No adverse effects were noted with lignocaine.

Conclusion: Intravenous lignocaine significantly enhances the hypnotic efficacy of propofol, reduces induction dose, improves haemodynamic stability, and facilitates smoother induction. It is a safe and effective adjunct in anaesthetic practice.

Keywords: *Lignocaine, Propofol, Hypnosis.*
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INTRODUCTION

Intravenous anaesthesia is one of the pillars of contemporary perioperative care, providing an excellent fast, titratable hypnosis with a favourable recovery profile and a broad range of surgical and diagnostic regimens.[1] In general anaesthesia, its rapid onset, short context sensitive half-time and predictable recovery characteristics led it to become the first-choice drug for induction and maintenance of general anaesthesia and for procedural sedation.[2]

Concurrently, lignocaine (lidocaine), a local anaesthetic and class Ib antiarrhythmic agent, has established a systemic adjunct with analgesic, anti hyperalgesic and anaesthetic sparing activities, alongside intravenous use.[3] Lignocaine could also modulate propofol's hypnotic effect thereby decreasing propofol use and subsequently enhancing haemodynamic and respiratory safety, making it a crucial clinical subject.[4]

Propofol is one of the most popular hypnotic medications used in operating theatres, endoscopy suites and intensive care units. Propofol has long constituted the norm of reference IV induction therapy in high income and several middle-income countries because of its impressive pharmacodynamic profile characterised by rapid loss of consciousness and smooth emergence with low incidence of postoperative nausea and vomiting.[5] But its heavy use raised some worry over propofol's side effect profile,

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including dose-dependent cardiovascular depression, respiratory compromise and pain during injection. [6,7,8]

Recently, intravenous lignocaine has been shown to modulate intraoperative and postoperative pain, reduce central sensitisation, and decrease volatile anaesthetics and propofol use across procedures.3,4 Within this context of globalisation, one of the open and growing concerns for potential improvement of the optimization of anaesthetic and perioperative therapy is whether lignocaine serves to increase propofol's hypnotic action or simply reduces injection pain. [9]

At a broader therapeutic level, a dose reduction of propofol delivered by synergistic treatment with lignocaine may have the following advantages: improved haemodynamic stability by avoiding unnecessarily deep levels of anaesthesia, attenuation of respiratory depression, reduction of risk resulting from high cumulative propofol exposure, and potentially enhanced

recovery in day care and elderly patients.[6] Nonetheless, all the advantages have to be weighed against the potential risk of lignocaine toxicity, which is uncommon at standard infusion doses, but may present as neurological and cardiovascular complications if dosing thresholds are exceeded or in the presence of impaired clearance. [3] Therefore, extensive evidence is required to further confirm the safe and effective use of lignocaine and propofol in regimens.

So this study aims to be a well-made locally relevant piece of evidence that can be translated into practice, this research would be helpful in understanding good policy and practice in anaesthetics, it would contribute to the literature on multimodal intravenous anaesthesia as well, and can make a considerable difference to patient safety and comfort. In fact, the extent and scenario in which inclusion of lignocaine optimizes propofol hypnosis may allow anaesthetists to personalise induction and maintenance strategies, reduce drug-related adverse events and maximise perioperative outcomes as part of routine clinical practice.

AIMS AND OBJECTIVES

Aim

To investigate whether intravenous lignocaine enhances the hypnotic efficacy of intravenous propofol during anesthetic induction.

Objectives

1. To compare the onset time and depth of hypnosis achieved with propofol alone versus propofol combined with intravenous lignocaine.
2. To assess the hemodynamic effects (systolic blood pressure, diastolic blood pressure, heart rate, and mean arterial pressure) of combined propofol and lignocaine administration during induction of anesthesia.
3. To evaluate the impact of intravenous lignocaine on sedation quality and induction characteristics when co-administered with propofol.
4. To determine the safety profile and adverse effects associated with the combined administration of propofol and lignocaine.

MATERIALS AND METHODS

This randomized, double-blind, placebo-controlled trial was conducted at R.L. Jalappa Hospital and Research Centre, Kolar, from May 2024 to October 2025, following approval from the Institutional Ethics Committee and registration with the Clinical Trials Registry of India. The study adhered to CONSORT guidelines, and written informed consent was obtained from all participants.

A total of 62 adult patients (ASA I–II), aged 18–60 years and scheduled for elective surgery under general anaesthesia, were enrolled and randomized into two equal groups (n = 31 each). Sample size was calculated using standard formulas for comparison of continuous variables, assuming a clinically significant difference of 20 mg in propofol dose, with 80% power and 5% significance.

Patients with significant cardiovascular disease, hepatic or renal impairment, drug allergies, or those undergoing emergency surgery were excluded. Preoperative assessment included detailed history, physical examination, and routine investigations. All patients followed standard fasting guidelines.

Participants were randomly allocated using computer-generated block randomization, with allocation concealment ensured through sealed opaque envelopes. Both the anaesthesiologist and the patient were blinded to group allocation.

In the intervention group, patients received intravenous lignocaine 1.5 mg/kg (based on ideal body weight), while the control group received an equal volume of normal saline. Two minutes later, propofol was administered in 10 mg aliquots until loss of verbal response, which was taken as the endpoint of induction.

Standard intraoperative monitoring included heart rate, systolic and diastolic blood pressure, mean arterial pressure, oxygen saturation, respiratory rate, and end-tidal CO_2. Baseline values were recorded prior to intervention, and subsequent readings were taken at predefined intervals until induction.

The primary outcome measure was the total dose of propofol required for induction. Secondary outcomes included induction time, haemodynamic changes, and incidence of adverse events. Adverse events were defined as hypotension, bradycardia, tachycardia, respiratory depression, local anaesthetic toxicity, and hypersensitivity reactions.

Data were recorded using a standardized proforma and analysed using SPSS version 25. Continuous variables were expressed as mean ± standard deviation and

compared using independent t-tests or Mann–Whitney U tests as appropriate. Categorical variables were analysed using chi-square or Fisher's exact test. Repeated measures ANOVA was used for haemodynamic variables. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 62 patients were enrolled and randomized into two equal groups (n = 31 each). Baseline demographic variables, including age, gender, and ASA physical status, were comparable between the two groups, indicating adequate randomization and group homogeneity.

The primary outcome, propofol requirement for induction, was significantly lower in the lignocaine group. The mean propofol dose was **1.50 ± 0.00 mg/kg** in the lignocaine group compared to **2.07 ± 0.27 mg/kg** in the control group ($p < 0.001$). The total cumulative dose was also significantly reduced (115.4 ± 18.5 mg vs 142.5 ± 9.5 mg; $p < 0.001$). This finding indicates a clear propofol-sparing effect of intravenous lignocaine.

Induction time was significantly shorter in the lignocaine group (**48.4 ± 5.5 seconds**) compared to the control group (**55.0 ± 6.9 seconds**) ($p < 0.001$), suggesting that lignocaine facilitates faster achievement of adequate hypnotic depth.

Induction characteristics were superior in the lignocaine group, with 100% of patients achieving smooth induction compared to 74.2% in the control group ($p < 0.001$). This reflects improved induction quality, likely due to reduced injection pain and attenuation of reflex responses.

Hemodynamic parameters were better preserved in the lignocaine group. There was significantly less reduction in mean arterial pressure and smaller fluctuations in heart rate compared to the control group ($p < 0.001$). This indicates improved cardiovascular stability during induction with lignocaine use.

No adverse events were observed in the lignocaine group. In contrast, the control group demonstrated a higher incidence of cardiovascular adverse effects, predominantly hypotension. This supports the safety profile of lignocaine when used within recommended doses.

Multivariable regression analysis identified group allocation as the only significant independent predictor of reduced propofol requirement, reinforcing the direct effect of lignocaine on propofol dose reduction.

Overall, the findings demonstrate that intravenous lignocaine significantly reduces propofol requirement, improves induction characteristics, shortens induction time, and enhances haemodynamic stability during anaesthetic induction.

Table 1: Primary Outcome Analysis - Propofol Dose Requirement

Outcome	Lignocaine+Propofol (n=31)	Saline+Propofol (n=31)	Mean Difference (95% CI)	p-value	Effect Size (Cohen's d)
Propofol Dose (mg/kg)	1.50 ± 0.00	2.07 ± 0.27	-0.57 (-0.67 to -0.47)	**<0.001**	-3.01 (Very Large)
Total Propofol Dose (mg)	115.4 ± 18.5	142.5 ± 9.5	-27.1 (-34.0 to -20.2)	**<0.001**	-1.86 (Large)

INTERPRETATION :

In addition, concurrent use of intravenous lignocaine sharply reduced the propofol dose required to generate anesthesia. In the Lignocaine+Propofol group, the study protocol showed that the calculated target was 1.5mg/kg, which was to be given as a standard dose to the patients in that group, and in the Saline+Propofol control group the mean was found to be 2.07mg/kg. It represents very high effect size, that is 27.6 reduction of propofol dose adjusted by weight (Cohen's $d = -3.01$). The finding is statistically significant ($p < 0.001$), clinically significant, and provides evidence of a potent hypnotic-sparing effect of lignocaine.

Table 2: Secondary Outcomes - Induction Characteristics & Hemodynamic Changes

Outcome	Lignocaine+Propofol (n=31)	Saline+Propofol (n=31)	p-value	Test Statistic	Effect Size
Time to Induction (s)	48.4 ± 5.5	55.0 ± 6.9	**<0.001**	t(60) = -4.31	d = -1.07 (Large)
MAP Drop (mmHg)	2.6 ± 1.6	7.2 ± 2.3	**<0.001**	U = 46.0	r_{rb} = -0.94
HR Change (bpm)	1.2 ± 2.5	7.4 ± 3.8	**<0.001**	U = 95.5	r_{rb} = -0.88
Smooth Induction, n (%)	31 (100%)	8 (25.8%)	**<0.001**	$\chi^2(1)$ = 44.95	φ = 0.85

INTERPRETATION :

Induction properties were better in the lignocaine group. There was a significant fastening of Induction (mean difference -6.6 seconds, $p<0.001$) and a qualitative response of Induction (all Smooth qualitative response 1000 in controls 25.8). There were also significantly smaller responses by the lignocaine group in MAP — 74%

less median fall of MAP and 84% less rise in heart rate, compared with controls (all $p<0.001$, large effects). This means that lignocaine is able to decrease the propofol requirements, in addition to bringing about hemodynamic stability and a more seamless induction.

DISCUSSION

The present randomized trial verifies that propofol in combination with intravenous lignocaine has a remarkable hypnotic sparing mechanism and, in addition, also promotes hemodynamic stability and reduces the negative event rate of general anesthesia induction. The intervention decreased the propofol dose necessary for induction by approximately 28% and the effect was large; induction time was reduced; mean arterial pressure and heart rate changes, on average, were reduced and almost avoided cardiovascular adverse events against saline-propofol control. The results were reproducible in the ASA I & 2 type, across the female and male subgroup as well as post-adjustment under multivariate conditions, indicating that differences in responses are not due to demographic or underlying physiological differences. Together these data illustrate that intravenous lignocaine plays a clinically relevant role as a coinduction agent to deeper hypnosis at decreased propofol dose and maintenance of the circulation.[10,11]

The infusion-dependent use of lignocaine IV as an anesthetic adjunct is used primarily to decrease airway reflexes, blunt hemodynamic reactions to laryngoscopy and endotracheal intubation, and minimize pain during propofol injections. In addition to these classical indications, there is a growing body of evidence indicating systemic use of lignocaine and that it has an anesthetic and opioid sparing ability along with peripheral anti-nociceptive and central sodium channel blocking effects reducing the need for hypnotic drugs and improving perioperative analgesia.[12] This reduction in volatile and intravenous anesthetic doses and improvement in postoperative outcomes have recently been described by a narrative survey and a subset of controlled trials, in an environment in which lignocaine infusion is included during multimodal anesthesia.[13]

More concisely, numerous clinical and experimental reports lignocaine was capable of reducing propofol use during general anesthesia and sedation with variable effect size and consistency. In an RCT of endoscopic procedures, patients administered with IV lignocaine required lower doses of propofol especially in the setting of nociceptive stimulation, confirming that lignocaine is indeed an anti-narcotic and does not exert pharmacokinetic interaction with propofol.[11]

Propofol induction level was attained by the use of 1.5 mg/kg of propofol in lignocaine induced patients versus 2.07 mg/kg of propofol from saline in treatment group and was 27–28 per cent lower with relatively a large standardized effect size (cohen d = 3). This size is broadly comparable to initial experimental and clinical studies demonstrating 20–35% reduction in hypnotic doses for patients receiving lignocaine supplementation.56(14) Lignocaine infusion supplemented with propofol resulted in decrease in propofol dose in animals and in humans it showed reduced doses of propofol needed for surgery as well as adjunct with infusion of lignocaine .

With narrow confidence intervals, demonstrated by fixed requirement of 1.5 mg/kg lignocaine in the lignocaine arm, we are able to conclude that, in the case of an especially strong, consistent effect, these observations are mostly in line with the current results. The reduction of propofol need in this study in accordance with endoscopy based randomized trials did not cause an increased induction time but indicated that lignocaine is more appropriate for the development of optimum hypnotic depth over the inhibition of the drug or changes in pharmacokinetics.[11] This protocol which does not contain propofol bolus and lignocaine delivery in combination and is administered to the complete systemic dose appears purpose for an appropriate stimulation of the hypnotic state.

These are biologic observations based on synergy of anti-nociceptive and central modulating properties of lignocaine.[12] Lignocaine inhibits the action of afferent nociceptive information in the peripheral nerves and spinal networks by blocking voltage gated sodium channels that stimulate arousal and sympathetic arousal. Systemic lignocaine works to suppress the firing of neurons at the cortical level and attenuate the firing in the cortical area, and case studies state that the large-scale effects can be detected in EEG at the level of toxicity, in a dose-dependent manner with BIS following controlled infusions. By nociceptive input the lignocaine has the potential to indirectly influence cortical excitability and affect behavioural compromise states in agnostic relic of the hypnosis state that results in the same clinical consequence of loss of consciousness when given propofol at lower doses. Lignocaine lowered the hemodynamic disturbances during the induction period significantly in this experiment other than with the lower propofol level: MAP reduction is at approximately 74% whereas HR gain is about 84% less in the lignocaine group than control group with very large effect sizes and no hypotension. These results provide general proof of suppression of cardiovascular response to airway manipulation and noxious stimuli by lignocaine. [15]

The hemodynamic benefit involves two converging and mechanistically mediated pathways. The first one is that lignocaine reduces propofol requirement, and with the decrease of propofol dose and MAP results in regression models in the present study, which indicate the direct or indirect cause-effect linear relationship. Second, lignocaine has minimal direct anti arrhythmic, anti-membrane stabilizing action in the myocardium and acts to inhibit sympathetic release by sodium blockage of autonomic fibres, thus reducing reflex tachycardia and severe hemodynamic oscillations. In the present article, lignocaine was a strong independent predictor of lower MAP drop after correction for age, base MAP and total dose of propofol, suggesting that it has a stabilizing effect.

Similar to the findings of peri intubation studies in the literature, lignocaine elicited modest changes of MAP and HR although hypnotic and opioid doses were similar between groups , which accounts for the less significant changes in MAP and HR. Together, these data points suggest that lignocaine provides hemodynamic stability through its dose sparing and direct cardiovascular effect.[16]

The comparison of the short induction duration and a homogeneous smooth induction profile (100 vs 25.8 in controls) we observed among this lignocaine cohort supports the conclusion that pharmacologic effect is more than just more effective of induction in clinical practice. These findings are additional support to previous studies demonstrating that co induction modes (midazolam, opioids and lignocaine) can enhance the convenience of coming to unconsciousness and reduce cough, movement or hemodynamic lability. [16]

The smooth induction demonstrated in this study was probably related to suppression of injection pain, airway reflexes, and possibly subcortical excitatory phenomena associated with propofol induction by lignocaine. In isolation, lidocaine-propofol combinations at very low doses can decrease injection pain in 50-95 percent of patients, and thus represent routine use in this application. [17] .

Crucially, the time to induction in the lignocaine group in the present study was reduced by decreasing the propofol dosage, which was not a dose-related or schedule artifact, but rather an increased pharmacodynamic performance. This is consistent with experimental results suggesting that systemic lignocaine may decrease the time from wakefulness to loss of consciousness under specific conditions since it decreases the arousal of the cortex, particularly when it is given as a bolus in combination with hypnotic agents. This is not universally accepted, however, as some studies with low dose infusions or premixed preparations presented insignificant alterations in the induction dynamics, showing that appropriate systemic dosing and suitable timing with propofol intake are important.[17]

The most significant observation of this analysis is the absence of cardiovascular adverse events in the lignocaine group compared with 77.4% of controls, most of which are hypotension. This results in optimal doses of lignocaine within the therapeutic dosage range reduce the clinical risk of intraoperative hypotension and desaturation rather than increase it. Recent systematic reviews and meta-analysis of intravenous lidocaine administration during propofol anesthesia demonstrated a comparable decline in intraoperatively generated hypotension and desaturation attack rate and vasopressor demand with lidocaine.[18]

Local anaesthetic systemic toxicity (LAST) is the major safety concern in lignocaine reactions as it is considered the most serious problem in lignocaine-associated cases due to neurological (tinnitus, perioral numbness, seizures) and cardiovascular (arrhythmias and cardiac arrest) side effects at high plasma concentration of this drug. There have been reports of convulsions with comparatively small doses, such as fast bolus, drugs of comorbidity, or personal predisposition, which is why caution with drug administration should be considered and watching carefully while administering drugs. However, high perioperative series and controlled trials demonstrate that in appropriate doses of lignocaine in the ideal range (usually 1-1.5 mg/kg bolus plus 0 to 2 mg/kg/h infusion) and administered over the right time, clinically significant toxicity is an uncommon problem.[12] This stronger safety evidence is therefore consistent with the lignocaine arm demonstrating no adverse events in current study.

CONCLUSION

This randomized controlled trial demonstrates that intravenous lignocaine is a safe and effective adjunct to propofol for induction of anaesthesia. The use of lignocaine significantly reduces propofol requirements while facilitating smooth and rapid induction. In addition, it provides improved haemodynamic stability with a lower incidence of adverse cardiovascular events.

The propofol-sparing effect of lignocaine was consistent across patient subgroups and was not influenced by demographic or baseline clinical variables, indicating a robust and reproducible pharmacological interaction.

These findings support the use of intravenous lignocaine as a co-induction agent to optimize propofol-based anaesthesia, particularly in settings where haemodynamic stability is desirable. Further studies in high-risk populations and with objective depth of anaesthesia monitoring are warranted to expand its clinical applicability.

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