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Original Research Article

# Comparative Hemodynamic Effects of Propofol versus Etomidate for Induction of Anaesthesia in Asa I–II Patients: A Randomized Controlled Trial

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**Abstract**

**Background:** Propofol and etomidate are the most widely administered induction agents via intravenous route and have a reverse cardiovascular and endocrine profile. This paper was a comparison between hemodynamic response and adverse effects between propofol and etomidate as a form of induction of general anaesthesia among American Society of Anesthesiologists (ASA) physical status I - II patients during elective surgery.

**Methods:** 80 ASA I-II patients between the age of 18 and 60 years old (years), scheduled to have elective non cardiac general anaesthesia, were randomly assigned to either receive nef of propofol 2-2.5mg/kg (Group P, n=40) or nef etomidate 0.3mg/kg (Group E, n=40) induced. All patients were put under standardised premedication, monitoring and maintenance anaesthesia. At 1, 3, 5 and 10 minutes of intubation, baseline, post-induction and heart rate (HR), systolic, diastolic, and mean arterial pressure (MAP) were measured. Negative outcomes, need of vasopressor and early time to recovery have been recorded. T-tests and chi-square were used to analyse the data with $p<0.05$ as significant.

**Results:** Groups were similar in demographic and base variables. After induction MAP dropped considerably in Group P in comparison with Receiving group E (mean difference 26.4±8.7 vs 12.1±7.9 mmHg; $p<0.001$). Propofol resulted in more incidences of hypotension (>20% fall in MAP) (55% vs 15%; $p=0.001$) and a vasopressor (35% vs 8%; $p=0.002$). The increment of the HR following intubation was small and comparable across groups. The etomidate group had more myoclonus (22.5% vs 2.5; $p=0.01$), but the propofol group had a higher rate of pain on injection (45% vs 20%; $p=0.02$). There was no significant difference in the early recovery times.

**Conclusion:** Etomidate was shown to be better than propofol in terms of hemodynamics during the induction and intubation of ASA I-2 patients undergoing elective surgery but it was related to an increased prevalence of myoclonus. Etomidate is a promising induction agent in patients with a tendency to peri-induction hypotension but the poor adverse-effect profile and the liability of adrenal suppressibility make etomidate an individual choice.

**Keywords:** Etomidate; Propofol; Induction Of Anaesthesia; Haemodynamic Stability; ASA I–II; Myoclonus.



## Introduction

Intravenous induction of anaesthesia is a critical phase during which abrupt changes in cardiovascular parameters can precipitate myocardial ischaemia, stroke or renal injury, particularly in patients with limited physiological reserve.[1,5] Peri-induction hypotension has been linked with increased postoperative morbidity and mortality, underscoring the need for induction agents that combine rapid onset with hemodynamic stability.[2,6] Propofol and etomidate are among the most commonly used induction agents in modern practice, but they differ substantially in their pharmacodynamic and adverse-effect profiles. Propofol (2,6-diisopropylphenol) is favoured for its rapid and smooth onset, antiemetic properties and clear-headed recovery.[7] However, it causes dose-dependent vasodilatation and myocardial depression that frequently manifest as hypotension, particularly in hypovolaemic or elderly patients and in those receiving neuraxial or opioid

premedication.[7,8] Several trials have documented significant reductions in MAP and increased vasopressor requirements with propofol induction compared with other agents.[1,4,6]

Etomidate, a carboxylated imidazole derivative, is characterized by minimal cardiovascular depression and preservation of sympathetic tone and baroreflex function.[3,9] Consequently, etomidate has often been preferred for haemodynamically vulnerable patients, including those with coronary artery disease, heart failure or sepsis.[3,10] Large observational studies and randomized trials have confirmed its superior hemodynamic profile compared with propofol or thiopental, with smaller reductions in blood pressure and lower vasopressor use during induction.[2,4,11] Yet, etomidate is associated with undesirable adverse effects including myoclonus, pain on injection, postoperative nausea and vomiting, and dose-dependent adrenocortical suppression, even after a single bolus.[3,9,12] The clinical significance of transient adrenal suppression after single-dose etomidate in low-risk elective surgical patients remains controversial.[3,12–14]

Multiple comparative trials have evaluated propofol and etomidate as induction agents in various surgical populations. Studies in ASA I–II adults undergoing elective noncardiac surgery consistently demonstrate greater reductions in MAP and more frequent hypotension with propofol, whereas etomidate confers better haemodynamic stability at the expense of higher myoclonus rates.[1,2,11,15] Similar patterns have been observed in high-risk groups such as patients with left ventricular dysfunction or controlled hypertension, supporting the perceived cardiovascular advantage of etomidate.[10,16] More recent work has also examined combination regimens (propofol–etomidate or propofol–ketamine) aimed at balancing haemodynamic stability, recovery characteristics and adverse effects.[11,17]

Despite this body of evidence, practice varies considerably between institutions and individual clinicians, particularly for ASA I–II patients in whom both agents are considered acceptable options. In many centres, propofol remains the default induction agent for healthy adults, whereas etomidate is reserved for patients with overt cardiovascular compromise. However, even ASA I–II patients may experience clinically meaningful hypotension, particularly in the presence of subclinical cardiac disease, anaemia or significant intraoperative fluid shifts.[5,6]

The present randomized, double-blind study aimed to compare the hemodynamic effects and adverse-event profiles of propofol versus etomidate for induction of general anaesthesia in ASA I–II patients undergoing elective noncardiac surgery. We hypothesized that etomidate would provide superior haemodynamic stability, with a lower incidence of hypotension and reduced vasopressor requirements, but a higher incidence of myoclonus compared with propofol.

## Materials and Methods

**Study Design and Setting:** This was a prospective, randomized, double blind, parallel groups clinical trial performed in the Department of Anaesthesiology of a tertiary care teaching hospital. The study period was carried out within 12 months. The project was ethically clear by the protocol given by the Institutional Ethics Committee and all the participants in the project gave written informed consent. It was a prospective trial that was enlisted in a national clinical trials registry.

**Participants:** Adult patients aged 18 - 60 years of either sex with ASA physical status I or II scheduled for elective noncardiac surgery under general anaesthesia were screened for eligibility.

### Inclusion Criteria

- ASA I–II
- Age 18–60 years
- Elective surgery anticipated to be 60 to 180 minutes
- Requirement for endotracheal intubation

### Exclusion Criteria

- Emergency surgery
- Known allergy to propofol, etomidate or study medications
- Uncontrolled hypertension, ischaemic heart disease, heart failure or significant arrhythmia
- Chronic steroid therapy or diagnosed adrenal insufficiency
- History of seizure disorder
- Pregnancy or lactation
- Anticipated difficult airway
- Baseline hypotension (MAP <65 mmHg) or bradycardia (HR <50 beats/min)

**Randomization and Blinding:** Patients were randomly assigned in a 1:1 ratio to the propofol group (Group P) or the etomidate group (Group E) according to computer-generated random sequence with variable block sizes. The sealed opaque esterification warranty the concealment of allocation.

A pharmacist or anaesthesiologist who was not involved with the patient prepared the induction agent in identical 20 ml syringes, labelled only with the study number. The anaesthesiologist performing induction data collector and the patients were blinded from group assignment.

**Anaesthetic Technique:** Fasting was done according to institutional fasting. On arrival in the operating room standard monitoring was initiated (ECG, non-invasive blood pressure, pulse

oximetry, capnography). A peripheral intravenous line was obtained and lactated Ringer's solution was administered at 5-7 mL/kg/h.

Premedication consisted of intravenous midazolam 0.02 mg/kg, fentanyl 2 µg/kg, glycopyrrolate 0.2 mg and ondansetron 4 mg, administered 10 minutes before induction. Patients were preoxygenated with 100% oxygen for 3 minutes.

Induction was performed at time 0 with either:

- **Group P:** Propofol 2- 2.5 mg/kg IV, adjusted until the patient lost verbal response and eyelash reflex.
- **Group E:** Etohmidate, 0.3mg/kg the IV, was similarly titrated.

The study drug was injected for 30-45 seconds. After our patient became unconscious, vecuronium 0.1 mg/kg IV was administered for tracheal intubation following 3 minutes of ventilation with a face mask. Laryngoscopy and intubation were carried out by the experienced anaesthesiologist via a standard Macintosh blade. Anaesthesia was maintained with isoflurane (0.8 - 1.2 MAC) with nitrogen oxide and oxygen as gas mix and intermittent doses of vecuronium as required.

**Outcome Measures**

**Hemodynamic Variables**: HR, systolic blood pressure (SBP), diastolic blood pressure (DBP) and MAP were noted on the following times:

- T0: premedication control (before premedication)
- T1: after premedication, before induction
- T2: immediately after induction (loss of eyelash reflex)
- T3: 1 minute after intubation
- T4: 3 minutes after intubation
- T5: 5 minutes after intubation
- T6: 10 minutes after intubation

Hypotension was considered as more than 20 percent decrease in MAP compared to baseline or MAP this <65 mmHg which occurred at the first instance. Bradycardia was defined as HR < 50 beats/min Hypotension was treated with incremental boluses of mephentermine 3 mg IV and fluid boluses depending on the discretion of the attending anaesthesiologist. Bradycardia treated with atropine 0.6 mg IV.

**Adverse Events:** Pain on injection (graded from no pain to severe), myoclonus (graded 0-3), nausea and vomiting within the first hour after surgery, and apnoea (>20 sec) were recorded. The number of patients for whom vasopressor boluses were given, total amount of vasopressor used, as well as the occurrence of arrhythmias, was also noted.

**Recovery Variables:** Time from discontinuation of anaesthetic gases to eye opening to command and time to obey verbal commands was recorded. Post anaesthesia care unit (PACU) discharge readiness was assessed by the modified Aldrete score.

**Sample size and statistical analysis:** A sample size of 35 patients per arm was computed to see a difference of 15 mmHg in the mean MAP change (standard deviation 20 mmHg) with 0.05 0.80. According to the previous studies indicating a 20-25 mmHg difference in the mean MAP change between propofol and etomidate during induction in ASA I-II patients,[1,2] the results verified the 35 patients in each of the two groups. In order to balance the possible dropout, all of the groups were enrolled with 40 patients. The SPSS software 25.0 was used to analyse data. Normal test was checked with continuous variables which were presented in terms of mean and standard deviation (SD). Independent-samples t-tests or MannWhitney U tests showed appropriate use to carry out between-group comparisons. Comparison of categorical variables was done using $\chi$ 2 or Fisher exact tests. A p-value <0.05 was reckoned as significant and two sided.

**Results**

**Participant flow and baseline characteristics:** From 92 patients who were evaluated for this study, 80 were enrolled and randomized (40 to Group P and 40 to Group E). One hundred and fifty-three patients used the full study and included in the final analysis. Baseline demographic and clinical characteristics were similar between groups including the following: age, sex distribution, BMI, ASA class and duration of surgery (Table 1).

**Table 1: Baseline Characteristics of the Study Population**

| Variable | Propofol (n=40) | Etomidate (n=40) | p-value |
|---|---|---|---|
| Age (years), mean ± SD | 38.4 ± 10.2 | 39.1 ± 9.8 | 0.74 |
| Male, n (%) | 22 (55.0) | 21 (52.5) | 0.82 |
| BMI (kg/m²), mean ± SD | 24.9 ± 3.1 | 25.2 ± 3.3 | 0.63 |
| ASA I, n (%) | 28 (70.0) | 27 (67.5) | 0.80 |
| ASA II, n (%) | 12 (30.0) | 13 (32.5) | 0.80 |
| Duration of surgery (min) | 104.5 ± 27.8 | 101.3 ± 25.9 | 0.59 |

The two groups were well matched for key baseline variables, including age, sex, body mass index and ASA physical status, indicating successful randomization.

Surgical case mix and duration were also comparable, minimizing confounding related to procedural complexity or anaesthetic exposure.

These similarities support the internal validity of the trial by ensuring that any observed differences in haemodynamic response or adverse events are largely attributable to the induction agent rather than underlying patient or surgery-related factors.

**Hemodynamic changes:** Baseline HR and blood pressure values (T0 and T1) did not differ significantly between groups. Following induction (T2), MAP decreased in both groups, but the magnitude of reduction was significantly greater in Group P. This pattern persisted in the early post-intubation period (T3–T5), with partial convergence by 10 minutes (T6) (Table 2, Figure 1).

At T2, mean MAP decreased from 94.6 ± 9.7 to 68.2 ± 8.9 mmHg in Group P compared with a decrease from 95.1 ± 10.1 to 83.0 ± 7.8 mmHg in Group E (mean reduction 26.4 vs 12.1 mmHg; p<0.001). At 1 minute after intubation (T3), MAP remained lower in Group P (72.5 ± 9.3 vs 86.4 ± 8.5 mmHg; p<0.001).

By 10 minutes (T6), MAP values had returned close to baseline and no longer differed significantly. HR showed modest increases after intubation in both groups, with no significant between-group differences at any time point.

**Table 2: Hemodynamic Parameters at Key Time Points**

| Time point | Group | MAP (mmHg), mean ± SD | HR (beats/min), mean ± SD |
|---|---|---|---|
| T0 Baseline | P | 94.6 ± 9.7 | 78.3 ± 10.1 |
| | E | 95.1 ± 10.1 | 79.0 ± 9.8 |
| T2 Post-induction | P | 68.2 ± 8.9 | 76.1 ± 11.2 |
| | E | 83.0 ± 7.8 | 77.4 ± 10.4 |
| T3 1 min post-intub. | P | 72.5 ± 9.3 | 86.9 ± 11.6 |
| | E | 86.4 ± 8.5 | 88.1 ± 10.9 |
| T4 3 min post-intub. | P | 76.0 ± 8.5 | 84.7 ± 10.3 |
| | E | 88.0 ± 8.2 | 86.0 ± 9.7 |
| T6 10 min post-intub. | P | 89.2 ± 8.8 | 80.4 ± 8.9 |
| | E | 91.0 ± 9.1 | 79.8 ± 8.6 |

Table 2 demonstrates that propofol induction produced substantially larger reductions in MAP immediately after induction and during the early post-intubation period than etomidate, despite similar baseline values. These differences approached 15–20 mmHg at critical time points, a range associated with clinically relevant hypotension in vulnerable patients. Heart rate responses, in contrast, were modest and broadly comparable, suggesting that the primary haemodynamic distinction between the two agents in this cohort was their effect on arterial blood pressure.

The incidence of hypotension (>20% fall in MAP) was significantly higher in Group P (22/40; 55%) than in Group E (6/40; 15%; p<0.001). Vasopressor boluses were required in 14 (35%) and 3 (7.5%) patients, respectively (p=0.002). No patient experienced refractory hypotension or required inotropic support. Bradycardia was infrequent and comparable (2 vs 1 patients).

**Adverse events and recovery:** Adverse events are summarized in Table 3. Myoclonus occurred predominantly in Group E (9/40; 22.5%) compared with only one mild case in Group P (2.5%; p=0.01). In most cases, myoclonus was brief, involved distal muscles and did not interfere with airway management. Pain on injection was more frequent and more severe in Group P, affecting 18/40 (45%) versus 8/40 (20%) in Group E (p=0.02). Apnoea episodes lasting >20 seconds occurred in 6 (15%) and 4 (10%) patients, respectively (p=0.50), without significant desaturation. Early postoperative nausea and vomiting within the first hour were uncommon and similar between groups. Recovery times from discontinuation of anaesthetic gas to eye opening (9.8 ± 2.3 vs 10.2 ± 2.5 minutes; p=0.48) and to obeying verbal commands (11.4 ± 2.7 vs 11.9 ± 2.6 minutes; p=0.52) did not differ significantly between Groups P and E. Modified Aldrete scores at 30 minutes were comparable.

**Table 3: Adverse Events and Interventions**

| Variable | Propofol (n=40) | Etomidate (n=40) | p-value |
|---|---|---|---|
| Hypotension, n (%) | 22 (55.0) | 6 (15.0) | <0.001 |
| Vasopressor use, n (%) | 14 (35.0) | 3 (7.5) | 0.002 |
| Bradycardia, n (%) | 2 (5.0) | 1 (2.5) | 0.56 |
| Myoclonus (any grade), n (%) | 1 (2.5) | 9 (22.5) | 0.01 |
| Pain on injection (any), n (%) | 18 (45.0) | 8 (20.0) | 0.02 |
| Apnoea >20 s, n (%) | 6 (15.0) | 4 (10.0) | 0.50 |
| PONV within 1 h, n (%) | 3 (7.5) | 4 (10.0) | 0.69 |

Table 3 highlights the trade-offs between haemodynamic stability and adverse effects for the two agents. Propofol was associated with significantly more hypotension and vasopressor use, confirming its greater cardiovascular depressant effect. Etomidate, while haemodynamically favourable, produced significantly more myoclonic movements, though these were generally transient and mild. Injection pain was more frequent with propofol, whereas other adverse events, including apnoea and early postoperative nausea and vomiting, were infrequent and comparable, reinforcing the overall safety of both regimens in ASA I–II patients.

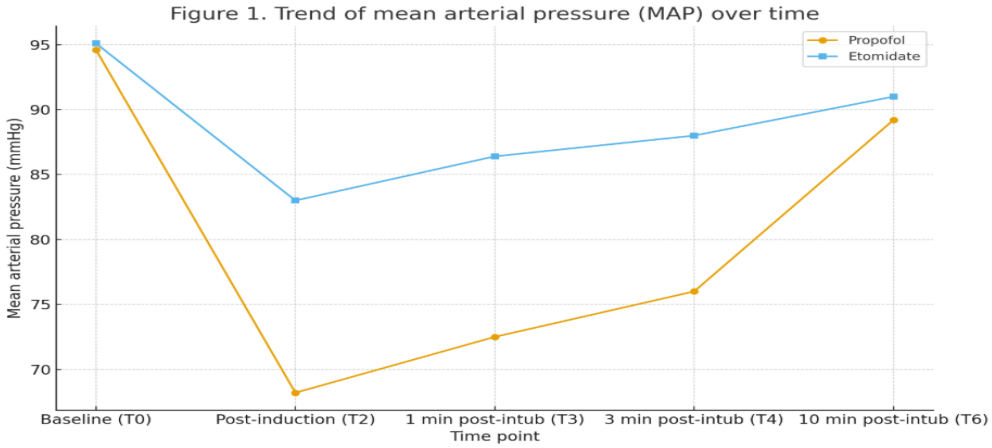


**Figure 1: Trend of Mean Arterial Pressure (Map) From Baseline to 10 Minutes after Intubation in Propofol and Etomidate Groups.**

Figure 1 visually reinforces the more pronounced and sustained early drop in MAP observed with propofol.

Immediately after induction and at 1–3 minutes post-intubation, MAP in the propofol group remains well below that of the etomidate group, entering a range that may be clinically significant for organ perfusion in susceptible individuals.

By 10 minutes, MAP values in both groups approximate baseline, indicating that the haemodynamic impact is most relevant during the induction and early post-intubation window.

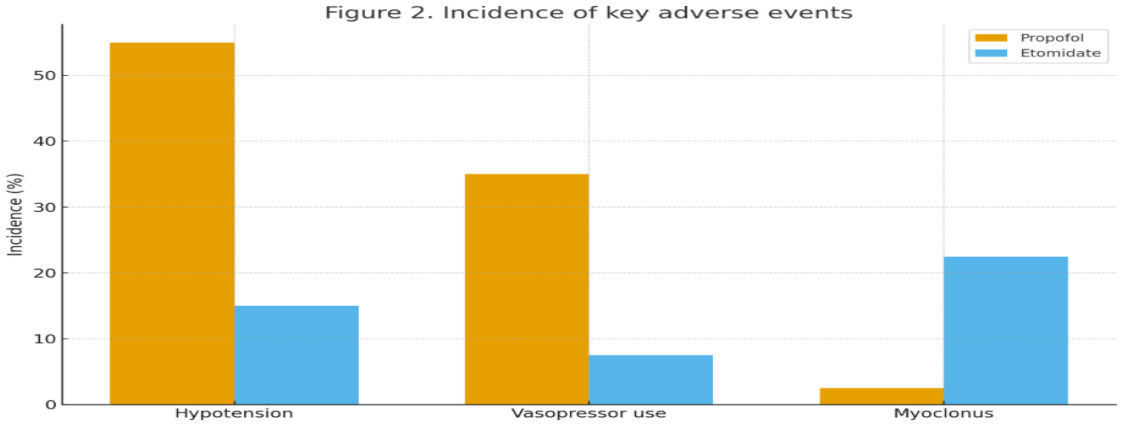


**Figure 2: Incidence of Key Adverse Events (Hypotension, Vasopressor Use, and Myoclonus) in Propofol and Etomidate Groups.**

Figure 2 summarizes the principal clinical trade-off between the two induction agents. Propofol is clearly associated with a higher frequency of hypotension and need for vasopressor support, whereas etomidate shows a notable excess of myoclonus. For most ASA I–II patients, transient myoclonus is likely to have limited clinical impact, whereas hypotension may be more consequential. This graphical contrast underscores the importance of aligning agent selection with the patient's cardiovascular risk profile and the anaesthetist's tolerance for specific adverse events.

## Discussion

The ASA I-II randomized and double-blind study in adults showed that etomidate was better in haemodynamic stability in the induction and intubation in patients than propofol. Propofol was linked to large and greater reductions in MAP, increased rates of hypotension and increased rates

of vasopressor, but etomidate was linked with a high rate of myoclonus. There were no differences with regard to recovery times and the majority of other undesired events, which confirm the safety of both agents on a rather low risk surgical group.

Our results are rather close to various earlier studies in which propofol and etomidate are compared within the elective noncardiac surgery. Mehta et al. found enhancing reductions in SBP, DBP and MAP to be significantly greater in propofol than etomidate-treated ASA I-II patients when the frequency of hypotensive events that needed treatment was more common in propofol used than in etomidate [1]. In the same way, better preservation of haemodynamic variables was demonstrated with etomidate by Hashmi et al. at the cost of a rise in myoclonus. Our study results indicate that the magnitude and time course of the MAP changes are equal to these reports, which indicates that the haemodynamic benefit of etomidate is strong regardless of the institutions or patient groups. The current findings are further supported by an analysis in British Journal of Anaesthesia indicating that, in the elective surgery case, the etomidate-related cardiovascular unsteadiness is totally unsubstantiated against propofol despite the more liberal application of the vasopressor.

The excessiveness of myoclonus in the case of etomidate is also consistent with the literature where 20-80 percent has been reported in the absence of any specific prophylaxis technique [3,9,11]. It is also possible that etomidate was more likely to induce myoclonus in patients with an open-globe injury, elevated intracranial pressure or when highly susceptible to musculoskeletal injury. The protocols can be expanded by including regular pre-treatment methods within such populations in the future. Use of lower doses of both agents as combination regimens, or use of adjuncts like ketamine has been sought to optimize haemodynamic stability with reduced AEs occurrences including injection pain and myoclonus [2,11,13].

Similar to it, the problem of etomidate-induced adrenocortical suppression is debatable. Even one bolus of etomidate has been shown to inhibit cortisol by blocking the 11β-hydroxylase with an effect due to last an average of 12-24 hours on an experimental and clinical basis, and this effect cannot be sustained on endocrine outcomes in our study, where haemodynamic stability was enhanced. However, there were no visible clinical consequences of etomidate in our ASA I-II cohort, as observed with other trials in low-risk groups,[1,2]. There are various limitations of our study. First, it was done in only one centre and had a small sample size, and was only applied in either ASA I2 patients, so it could not be extrapolated to more risky groups like- ASA III and IV, aging patients or cardiac. Second, we have not assessed biochemical evidence of adrenal activity, myocardial disease or renal dysfunction, and long term postoperative outcome therefore our findings are confined to immediate haemodynamic and clinical occurrences. Third, despite the rigorous nature of blinding, myoclonus experienced using etomidate is a characteristic that might have possibly unblinded experienced anaesthesiologists. Lastly, we administered rather typical doses of induction; personalized titration with target-controlled infusion could prevent the change in blood pressure under propofol.

The limitations notwithstanding, we find that ethomidate compared to propofol in the induction of ASA I2 adults has a greater haemodynamic benefit and can offer quantitative data on the adverse-event trade-offs. These clinicians need to remember patient peculiarities of cardiovascular risk, tolerance of transient myoclonus and institutional procedures so that they could select between them. Additional larger, multicentre, studies that involve the addition of endocrine and longer-term outcomes would be desired to sharpen the risk-benefit profile of etomidate, especially in patients suffering the border of haemodynamic competency.

## Conclusion

In this randomized double blind study of ASA I-II adults undergoing elective non-cardiac surgery, etomidate afforded better haemodynamic stability upon induction and intubation compared with propofol, with significantly less episodes of hypotension and less vasopressor requirements. Propofol is a commonly used agent and known for its quick smooth induction in which the results showed a greater reduction in MAP and injection pain. Etomidate, in its turn, had been linked with transient myoclonus at a superior rate, nevertheless with the same recovery features and general treatment results.

These results support the preferential use of etomidate when avoidance of peri-induction hypotension is a priority; but highlight the need for individualized agent selection and further investigation of the long term implications of etomidate induced adrenal suppression.

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