

# Comparative evaluation of propofol or etomidate induction in dexmedetomidine- glycopyrrolate- butorphanol premedicated brachycephalic dogs

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*This study evaluated and compared the induction characteristics of propofol and etomidate in 20 brachycephalic dogs undergoing various surgical procedures and premedicated with dexmedetomidine, glycopyrrolate, and butorphanol. The dogs were randomly divided into two groups of 10 animals each: group P (propofol) and group E (etomidate). All animals were pre-anaesthetized with dexmedetomidine (5 µg/kg body weight), glycopyrrolate (0.01 mg/kg), and butorphanol (0.2 mg/kg) administered intramuscularly in a single syringe, followed by preoxygenation. Induction was achieved intravenously to effect using propofol in group P and etomidate in group E. Anaesthesia was maintained with isoflurane in 100% oxygen. The mean induction doses of propofol and etomidate were 1.80±0.81 mg/kg and 0.72±0.50 mg/kg, respectively. Intubation was more smoothly facilitated in group P. Group P also exhibited a more pronounced reduction in blood pressure and heart rate, although values remained within physiological limits. Recovery was faster in group E, with earlier return of palpebral reflex, head lifting, and sternal recumbency. Etomidate demonstrated superior cardiovascular stability and recovery quality, indicating that it may be the preferred induction agent in brachycephalic dogs to optimize anaesthetic safety and outcomes.*

**Keywords:** Brachycephalic dogs, Butorphanol, Dexmedetomidine, Etomidate, Glycopyrrolate, Propofol.

With over 450 recognized breeds worldwide (Ostrander *et al.*, 2019), including 24 brachycephalic breeds (Humane Society Veterinary Medical Association), the dog is the most phenotypically diverse domesticated species. Brachycephaly, derived from the Greek term meaning “short head,” is an intentionally selected trait in several breeds but is strongly associated with significant health challenges. Brachycephalic Obstructive Airway Syndrome (BOAS) is among the most common concerns, predisposing affected dogs to upper airway obstruction, respiratory distress, impaired thermoregulation, and ocular and dental abnormalities.

Dexmedetomidine, an  $\alpha_2$ -adrenergic agonist, exerts sedative and anxiolytic effects primarily through stimulation of  $\alpha_2A$  receptors, while activation of  $\alpha_2B$

receptors results in peripheral vasoconstriction (Basker *et al.*, 2009). At higher doses, its stimulation of peripheral  $\alpha_1$  and  $\alpha_2$  receptors may lead to transient hypertension followed by reflex bradycardia (Ebert *et al.*, 2000). Butorphanol, a mixed opioid agonist-antagonist acting on  $\mu$ ,  $\delta$ , and  $\kappa$  receptors, provides mild analgesia and contributes to balanced anaesthesia by reducing inhalant anaesthetic requirements.

Etomidate, an ultrashort-acting hypnotic agent that enhances GABA A receptor activity, is widely recognized for its excellent cardiovascular stability, making it particularly suitable for patients with cardiac compromise, shock, or advanced age (Heinrich *et al.*, 2014; Valk and Struys, 2021). Compared with propofol, etomidate causes minimal cardiopulmonary depression and is often preferred in high-risk patients (Masoudifar and Beheshitan, 2013). However, its limitations include transient adrenal suppression due to inhibition of 11- $\alpha$ -hydroxylase, as well as potential side effects such as myoclonus, injection pain, and postoperative nausea.

Given the unique anaesthetic challenges in brachycephalic dogs, including airway obstruction risk and sensitivity to cardiopulmonary depression, careful selection of induction agents is critical. Therefore, the primary objective of the present study was to evaluate the efficacy, safety, and clinical suitability of propofol versus etomidate for anaesthetic induction in brachycephalic dogs premedicated with a Dexmedetomidine–Glycopyrrolate–Butorphanol protocol.

## Materials and Methods

A total of 20 client-owned brachycephalic dogs presented for various surgical procedures were enrolled in the study. The dogs were randomly allocated into two equal groups: group P (propofol induction) and group E (etomidate induction). Food and water were withheld for 8 hr and 4 hr, respectively, before anaesthesia. Thoracic auscultation and

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preoxygenation were performed prior to drug administration. Baseline cardiopulmonary and haematobiochemical parameters were recorded before premedication.

All dogs were premedicated intramuscularly using a single syringe containing dexmedetomidine (5 µg/kg body weight), glycopyrrolate (0.01 mg/kg), and butorphanol (0.2 mg/kg). Induction was achieved intravenously to effect using propofol in group P and etomidate in group E. Anaesthesia was maintained with isoflurane in 100% oxygen for all dogs.

Physiological variables - including rectal temperature (°F), respiratory rate (breaths/min), heart rate (beats/min), oxygen saturation of haemoglobin (SpO<sub>2</sub>), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and any respiratory abnormalities - were monitored continuously from baseline (T0) and at 5-minute intervals post-induction (T5) up to 60 minutes (T60).

Depth of anaesthesia and analgesia was evaluated using standard clinical criteria, including jaw tone (Table 1), palpebral reflex (Table 2), pedal reflex (Table 3), and quality of intubation and induction (Tables 4 and 5). Recovery quality was assessed by recording the time of return of palpebral reflex, tongue withdrawal, first head lift, sternal recumbency, standing, and normal gait.

**Table 1:** Jaw reflex criteria (Mansi, 2018).

0	Did not allow opening of the jaws
1	Resistant to the opening of the jaws and closed slowly
2	Less resistance to opening the jaw and closed slowly
3	No resistance and jaw remained open

**Table 2:** Palpebral reflex score criteria (Mansi, 2018).

0	Intact and strong reflex (quick blink)
1	Intact but weak reflex (slow response)
2	Very weak (very slow and occasional response)
3	Abolished (no response)

**Table 3:** Pedal reflex score criteria (Mansi, 2018).

0	Intact and strong reflex (strong withdrawal)
1	Intact but weak reflex (animal responding slowly)
2	Intact but very light (very slow and occasional response)
3	Abolished completely (no response).

**Table 4:** Quality of intubation score criteria (Ahmad *et al.*, 2013).

0	Did not permit entry of tube into the mouth
1	Allowed entry but chewed
2	Allowed deeper entry but coughed
3	Difficult intubation without coughing
4	Easy intubation without coughing

**Table 5:** Quality of induction score criteria (Cleale *et al.*, 2009).

0	Did not permit entry of tube into the mouth
1	Allowed entry but chewed
2	Allowed deeper entry but coughed
3	Difficult intubation without coughing
4	Easy intubation without coughing

All objective data were analyzed using SPSS Statistics for Windows, Version 20.0. Results were expressed as Mean±SE. Statistical comparisons were performed using one-way analysis of variance (ANOVA), Tukey's post hoc test, and independent t-tests. A P-value of more than 0.05 was considered statistically significant.

**Results and Discussion**

Twenty client-owned brachycephalic dogs of both sexes presented for various surgical procedures were randomly allocated to two groups: group P (propofol) and group E (etomidate), with 10 animals in each group. The breed distribution in group P consisted of 40% Shih Tzu, 20% Pug, and 10% each of Chow Chow, Boxer, French Bulldog, and American Bully, whereas group E included 60% Shih Tzu, 30% Pug, and 10% American Bully.

The mean induction doses required were 1.80±0.81 mg/kg body weight IV for propofol and 0.72±0.50 mg/kg IV for etomidate, demonstrating a clear dose-sparing effect likely attributable to the premedication protocol. Induction with propofol facilitated smoother and easier intubation, with complete suppression of the cough reflex, in agreement with Kapil (2014) and Dar *et al.* (2019). In contrast, intubation following etomidate induction was slightly more challenging, possibly due to partially preserved laryngeal reflexes, as reported by Saini *et al.* (2020).

Both groups exhibited significant reductions in rectal temperature throughout the anesthetic period, resulting in intra- and postoperative hypothermia. This temperature decline is attributed to the combined depressant effects of dexmedetomidine, propofol or etomidate, and isoflurane. Sabbe *et al.* (1994) reported that alpha-2 agonists alter thermoregulation through their action on central α<sub>2</sub>-adrenoceptors.

Group P demonstrated a significant decrease in heart rate (HR) from baseline, which persisted until five minutes post-induction. In contrast, group E showed an initial rise in HR, reaching its peak at T30 before gradually returning to baseline values. Inter-group comparison indicated that group P recorded higher HR at T1 and T60, whereas group E showed slightly lower HR at T5, T15, T30, and T45. The cardiovascular pattern observed in the etomidate group aligns with its known minimal myocardial depressant effects and preservation of baroreceptor reflexes (Wauquier *et al.*, 1978; Kawakubo *et al.*, 1999).

Both groups exhibited a significant reduction in respiratory rate (RR) from baseline between T1 and T5, with values continuing to decline - yet remaining within the normal reference range - throughout the anaesthetic period. Group E consistently showed a slightly higher RR than group P, though this difference was not statistically significant except at T1, where a significant difference was observed. The marked reduction in RR following propofol induction is attributed to its depressive effects on central inspiratory drive and ventilatory responses (Goodman *et al.*, 1987).

In group P, both systolic arterial pressure (SAP) and mean arterial pressure (MAP) decreased significantly from baseline. In contrast, group E showed no significant deviations in SAP or MAP, remaining near baseline values, although SAP and diastolic arterial pressure (DAP) were consistently higher than those in group P. Dexmedetomidine typically produces a biphasic blood pressure response - an initial transient hypertension followed by hypotension due to reduced sympathetic outflow (Pypendop and Verstegen, 1998). The absence of this hypertensive phase in the present study may be attributed to intramuscular administration, which results in lower peak plasma concentrations (Monteiro *et al.*, 2010). Propofol-induced hypotension is primarily related to vasodilation and decreased cardiac contractility (Cullen and Reynoldson, 1997), whereas etomidate is known for its minimal cardiovascular depression (Forman, 2011).

Jaw, pedal, and palpebral reflexes were completely abolished in both groups (P and E), with consistent scores of  $3.00 \pm 0.00$  from T1 to T60, indicating a sustained absence of reflex activity throughout the anaesthetic period (Table 6). The persistence of reflex abolition following induction and subsequent maintenance with isoflurane confirmed that animals remained at a surgical plane of anaesthesia. Jaw relaxation was also used as an indicator of muscle relaxation. The loss of these reflexes can be attributed to the combined effects of dexmedetomidine, the induction agents, and isoflurane. Dexmedetomidine, like other  $\alpha_2$ -agonists, is known to produce profound muscle relaxation (Lemke, 2007). Propofol also provides effective muscle relaxation in dogs (Robertson, 1992). Similarly, etomidate induction has been reported to cause loss of jaw tone, palpebral reflex, and pedal reflex, along with adequate muscle relaxation, consistent with the observations of Perk *et al.* (2002) and Saini *et al.* (2020).

The mean induction doses for propofol (group P) and etomidate (group E) were  $1.80 \pm 0.81$  mg/kg and  $0.72 \pm 0.50$  mg/kg IV, respectively, following premedication with dexmedetomidine-glycopyrrolate and butorphanol. The reduced induction requirements observed in both groups are attributable to the synergistic sedative and anaesthetic-sparing effects of

dexmedetomidine and butorphanol. This is consistent with the findings of Panzer *et al.* (2009), who reported that dexmedetomidine significantly decreases the doses of both opioids and induction agents. Similarly, Raszplewicz *et al.* (2013) documented propofol induction doses ranging from 1–3 mg/kg IV after premedication with dexmedetomidine and butorphanol, aligning closely with the values obtained in the present study.

Group P showed significant intraoperative and postoperative decreases in Hb, PCV, and TEC, though all values remained within the normal reference range. In group E, Hb and PCV decreased significantly during surgery, while TEC showed a non-significant decline. These changes are likely attributable to splenic erythrocyte pooling, haemodilution from fluid therapy, and minor surgical blood loss (Kinjavdekar *et al.*, 2010; Anandmay *et al.*, 2012). Mild reductions in leukocyte counts may be related to splenic sequestration, underlying trauma, or hypothermia (Shekhawat, 2019; Biermann, 2012). No significant inter-group differences were noted.

Both groups exhibited non-significant decreases in TLC and platelet counts. Group P showed mild, non-significant hyperglycemia, whereas group E exhibited a significant increase; however, glucose levels in both groups remained above baseline, with group E consistently higher. This hyperglycemia may be associated with increased hepatic glucose output, reduced peripheral utilization, and decreased insulin release due to  $\alpha_1$ -adrenoceptor activation (Burton *et al.*, 1997). Propofol may further contribute by inhibiting glucose metabolism (Maeda *et al.*, 2018).

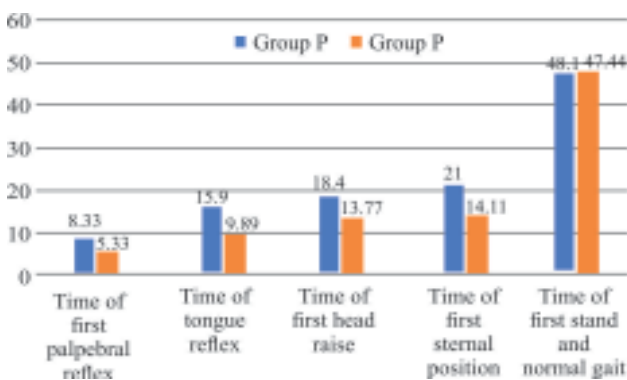
Group P showed a non-significant decrease in total protein, while group E demonstrated a significant intraoperative reduction. BUN and creatinine increased non-significantly during anaesthesia but returned near baseline postoperatively, remaining within physiological limits. These transient elevations may reflect temporary reductions in renal blood flow and glomerular filtration rate during anaesthesia (Jain *et al.*, 2007; Dewangan *et al.*, 2024). Dexmedetomidine may have mitigated more pronounced changes by stimulating  $\alpha_1$ -adrenoceptors, promoting diuresis, and supporting renal perfusion (Kuusela *et al.*, 2003; Arcangeli *et al.*, 2009) (Table 6).

Group E exhibited faster recovery compared to group P. The time to first palpebral reflex was  $5.33 \pm 0.64$  min in group E versus  $8.33 \pm 0.42$  min in group P; tongue reflex appeared at  $9.89 \pm 0.65$  min versus  $15.90 \pm 0.42$  min; first head raise occurred at  $13.77 \pm 0.70$  min versus  $18.40 \pm 1.28$  min; and first sternal recumbency was observed at  $14.11 \pm 1.19$  min versus  $21.00 \pm 1.23$  min, respectively. Time to first stand and normal gait was comparable between groups (group E:  $47.44 \pm 1.38$  min; group P:  $48.10 \pm 2.99$  min). Mean recovery quality was higher in group E ( $4.00 \pm 0.00$ ) than in group P ( $3.75 \pm 0.58$ ) (Fig. 1). Recovery was smoother in the

**Table 6:** Mean±SE of various haematobiochemical parameters in the three groups at different time intervals.

Groups	Group P			Group E		
	Pre	Intra	Post	Pre	Intra	Post
Hb	13.70±0.49 <sup>a</sup>	13.70±0.49 <sup>a</sup>	13.70±0.49 <sup>a</sup>	14.15±0.54 <sup>a</sup>	14.15±0.54 <sup>a</sup>	14.15±0.54 <sup>a</sup>
PCV	43.90±0.53 <sup>a</sup>	43.90±0.53 <sup>a</sup>	43.90±0.53 <sup>a</sup>	39.53±1.02 <sup>a</sup>	39.53±1.02 <sup>a</sup>	39.53±1.02 <sup>a</sup>
TEC (X 10 <sup>9</sup> /L)	6.09±0.33	6.09±0.33	6.09±0.33	6.09±0.33	6.09±0.33	6.09±0.33
PLT (X 10 <sup>9</sup> /L)	248.30±9.05	248.30±9.05	248.30±9.05	258.80±4.58	258.80±4.58	258.80±4.58
TLC (X 10 <sup>9</sup> /L)	12.47±0.40	12.47±0.40	12.47±0.40	12.47±0.38	12.47±0.38	12.47±0.38
Glucose (mg/dL)	97.12±2.42 <sup>a</sup>	97.12±2.42 <sup>a</sup>	97.12±2.42 <sup>a</sup>	97.12±2.42 <sup>a</sup>	97.12±2.42 <sup>a</sup>	97.12±2.42 <sup>a</sup>
ALT (U/L)	33.36±1.04 <sup>a</sup>	33.36±1.04 <sup>a</sup>	33.36±1.04 <sup>a</sup>	31.15±0.51	31.15±0.51	31.15±0.51
AST (U/L)	31.58±0.84 <sup>a</sup>	31.58±0.84 <sup>a</sup>	31.58±0.84 <sup>a</sup>	33.57±1.58	33.57±1.58	33.57±1.58
TP (g/dL)	6.74±0.24 <sup>a</sup>	6.74±0.24 <sup>a</sup>	6.74±0.24 <sup>a</sup>	6.88±0.23 <sup>a</sup>	6.88±0.23 <sup>a</sup>	6.88±0.23 <sup>a</sup>
BUN (mg/dL)	22.85±0.79	22.85±0.79	22.85±0.79	25.40±1.37	27.26±0.90	27.26±0.90
Creat(mg/dL)	0.92±0.04	0.98±0.04	0.95±0.04	0.98±0.02	0.98±0.02	0.98±0.02

Values with different superscript alphabets (uppercase) differ significantly ( $P<0.05$ ) within the row at different time intervals  
 Values with different superscript alphabets (lowercase) differ significantly ( $P<0.05$ ) between columns at different time intervals

**Fig. 1:** Mean±SE values of recovery parameters of group P and group E.

etomidate group, whereas dogs in the propofol group showed struggling, vocalization, and standing ataxia. The slightly prolonged recovery in group P is likely due to the sedative effects of dexmedetomidine, which has been reported to extend recovery times, especially when combined with propofol (Ohtani *et al.*, 2008; Kuusela *et al.*, 2003).

The study concluded that premedication with dexmedetomidine (5 µg/kg body weight), glycopyrrolate (0.02 mg/kg), and butorphanol (0.2 mg/kg) produced a dose-sparing effect for both propofol and etomidate induction. In brachycephalic dogs, both induction agents, when combined with isoflurane, provided safe and effective general anaesthesia. Haematobiochemical and physiological parameters remained within normal limits, with no adverse clinical effects. Etomidate demonstrated superior performance compared to propofol, offering faster recovery and greater cardiovascular stability.

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