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Comparative evaluation of isoflurane and sevoflurane in etomidate induced anaesthesia in dogs

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Abstract
Anaesthesia plays a vital role in veterinary surgery, providing unconsciousness, analgesia and muscle relaxation. This study evaluated isoflurane and sevoflurane for maintenance of anaesthesia in dogs premedicated with atropine, butorphanol and diazepam, and induced with etomidate and maintained in one group on isoflurane and another group on sevoflurane. Both groups showed smooth induction, although laryngeal reflexes occasionally complicated intubation. Anaesthetic maintenance was stable in both groups, with no significant differences in depth or physiological reSpOnses. Recovery parameters such as extubation, head lift and sternal recumbency times were similar; however, standing and complete recovery were significantly faster with sevoflurane. Recovery quality was also non-significantly superior in the sevoflurane group. Physiological and haemodynamic variables remained within acceptable ranges, while haematological and biochemical values showed only minor fluctuations. Overall, both agents were safe and effective, though sevoflurane offered faster and smoother recovery, indicating clinical advantages over isoflurane.

Keywords: Anaesthesia, etomidate, diazepam, butorphanol, isoflurane, sevoflurane.

1. Introduction
Anaesthesia is a crucial component for the success and precision of any surgical procedure. General anaesthesia is considered a miraculous advancement in veterinary anesthesiology, as it induces unconsciousness, muscle relaxation, amnesia and analgesia. An ideal anesthetic should provide rapid induction of anaesthesia, quick recovery, no excitatory adverse effects and no significant cumulative effect with repeated administration (Chaudhary *et al*., 2022) [3].
Etomidate is a carboxylated imidazole derivative, an intravenously administered hypnotic agent used for anaesthesia induction in humans and animals. Its main advantage is its minimal impact on cardiovascular function, making it a suitable choice for hemodynamically unstable patients and those with advanced cardiac diseases (Keating *et al.*, 2020) [9].
Volatile general anaesthetics are commonly used, but their mechanisms of action are complex and not fully understood. The effects of the drugs on anaesthesia are mainly dependent on changes in GABA/glutamate neurotransmission induced by each drug. Gamma-aminobutyric acid-A receptors are widely expressed in the central and peripheral nervous systems and mediate fast postsynaptic inhibition. The distribution of GABA-A receptor subunits has only been investigated in nodose ganglion in dogs. Additionally, MAC remains a clinically useful measure, of the potency, of inhaled anaesthetic (Sirin *et al*., 2021) [15].

2. Material and Methods
The work was carried out in the Department of Veterinary Surgery and Radiology and the Veterinary Clinical Complex, College of Veterinary Science and Animal Husbandry, MHOW, Indore, Madhya Pradesh, India, during a period of six months from June 2024 to November 2024. A total of 12 dogs, aged 1 to 8 years and weighing between 10 to 30 kg, irrespective of sex and breed, presented for elective surgical procedures, were randomly divided into two equal groups of six animals each. All dogs underwent pre-anaesthetic evaluation, including physical examination and fasting, prior to anaesthesia. The anaesthetic protocol was similar in both groups and consisted of atropine sulphate (0.04 mg/kg IM), followed 15 minutes later by butorphanol tartrate (0.2 mg/kg IV) and diazepam (0.5 mg/kg IV after 5 minutes).

Ceftriaxone with tazobactam (15 mg/kg IV) and meloxicam (0.2 mg/kg) were given pre-operatively. Anaesthesia was induced with etomidate (0.2%) at 1 mg/kg IV until effect, and animals were intubated before beginning inhalation anaesthesia. In Group I, anaesthesia was maintained using isoflurane (1.0-2.5%) in 100% oxygen, while in Group II, sevoflurane (3.3-3.6%) in 100% oxygen was used. Both groups were maintained using a semi-closed circle rebreathing system with oxygen flow at 25-50 mL/kg/min until the end of surgery, followed by oxygen supplementation until recovery of swallowing reflex. Clinical observations included induction time, induction quality, duration and quality of maintenance (Table 1) and recovery characteristics (extubation, head lift, sternal recumbency, standing and complete recovery), along with recovery quality scoring. Physiological parameters recorded were rectal temperature, heart rate and respiration rate, while haemodynamic parameters included non-invasive blood pressure and SpO_2. All parameters were recorded at baseline (0 min), after premedication, post-induction, at 10-minute intervals up to 50 minutes and after complete recovery. Haemato-biochemical analysis was performed at 0 min, post-premedication, post-induction, every 15 minutes during maintenance and after full recovery. Haematological parameters included haemoglobin concentration, packed cell volume, total erythrocyte count, total leukocyte count and differential leukocyte count, while biochemical parameters comprised ALT, AST, serum creatinine and BUN. Statistical analysis of all variables was performed using Two-way ANOVA as per Snedecor and Cochran (1994) [16].

3. Results and Discussion

3.1 Clinical observations

There was no significant difference in induction time, anaesthesia induction quality, duration of maintenance of anaesthesia and quality of recovery from anaesthesia between the groups. Quality of maintenance of anaesthesia showed significant within-group differences across time (Table 02), indicating progressive anaesthetic depth and analgesia. No significant differences were observed between groups at any time. Etomidate causes hypnosis, amnesia, and curtails nociceptive reSpOnses mainly by acting on a specific type of neuronal ion channels called γ-Aminobutyric Acid Type A (GABA A) receptors (Forman, 2011) [5], which may explain the considerable alteration in the reflexes score. For recovery from anaesthesia times, extubation, head lift and sternal recumbency times showed no significant differences between groups. However, standing and complete recovery time was significantly faster in the sevoflurane group (Table 3). The group that was administered sevoflurane exhibited faster standing and recovery times compared to the group that received isoflurane, which can likely be attributed to the lower solubility of sevoflurane (Jadon *et al*., 2008) [7]. In canine patients, sevoflurane has a blood-gas partition coefficient of 0.68 and a Minimum Alveolar Concentration (MAC) of 2.36. This reduced solubility was noted to contribute to quicker recovery times (Basha *et al.,* 2018) [2].

3.2 Physiological observations

Rectal temperatures in both groups showed non-significant reductions after pre-anaesthetic administration, with further significant decreases observed post-induction. Temperatures declined progressively during surgery, reaching the lowest levels at 50 minutes. No significant differences were noted between groups at any time point. The values returned to near baseline levels after recovery. The observed trend of decreasing rectal temperature may be attributed to the reduced activity of the reticular activating system and depression of the thermoregulatory centre in hypothalamus (Seymour and Gleed, 1999) [14], as well as a decrease in the metabolic rate of body and reduced skeletal muscle activity during the induction and maintenance of anaesthesia (Hardman *et al*., 2001) [6]. Following pre-anaesthetic administration and induction, respiration rates decreased significantly within each group. During maintenance, rates stabilized at lower levels than baseline. Post-recovery, respiration rates significantly increased, nearing baseline levels, with no significant differences observed between groups at any time point. The significant decrease in respiration rate within both groups from baseline to pre-anaesthetic administration may be due to the administration of diazepam which has been shown to depress central respiratory activity, leading to decreased phrenic nerve activity and, consequently, a lower respiration rate (Al-Khudhairi *et al*., 1982) [1] and also administration of butorphanol, an opioid analgesic which acts as a partial agonist at mu-opioid receptors, has been shown to decrease the respiratory rate in dogs, primarily due to its central nervous system depressant effects (Santos *et. al.,* 2007) [13]. There were no significant between-group differences in heart rate at any time point, while within-group comparisons showed significant changes from baseline. In both groups, heart rate increased post-administration of pre-anaesthetics and induction of anaesthesia, followed by a gradual decline during maintenance, stabilizing upon recovery. An initial non-significant increase in the heart rate of animals in both groups after administration of pre-anaesthetic might be due to the vagolytic effect of atropine sulphate, as stated by Pereira *et al*. (2019) [12] and Tiwari *et al*. (2024) [17]. A significant increase in heart rate was noticed in comparison to the baseline value following induction of anaesthesia, and the increase may be due to etomidate, which does not effectively mitigate the stress reSpOnse associated with laryngoscopy and intubation (Wahab *et al*., 2020) [18].

3.3 Hemodynamic observations

The SpO_2 values of both groups remained stable within clinically acceptable limits throughout the study. No significant differences were observed between groups. Isoflurane showed a significant increase in SpO_2 during anaesthesia maintenance, whereas sevoflurane exhibited no notable changes. Post-recovery, SpO_2 values returned to baseline levels in both groups. The significant increase during the maintenance phase may be due to inhalation anaesthesia often involving the administration of a high concentration of oxygen, which directly increases the partial pressure of oxygen (PaO2 mmHg) in the blood, leading to higher SpO_2 levels (Lopes *et al*., 2008) [11]. There were no significant differences in systolic, diastolic or mean arterial blood pressure between Group I and Group II at any time point. Both groups exhibited significant within-group variations at various time intervals when compared to baseline values. Systolic blood pressure decreased progressively during the maintenance phase, with recovery values approaching baseline. Diastolic blood pressure decreased in Group I during the maintenance phase, while Group II showed no significant change. MAP decreased in both groups during maintenance but remained stable after recovery. The decrease in SBP, DBP

and MAP during maintenance with isoflurane and sevoflurane may be due to the systemic vasodilation effect of both anaesthetics which is a significant contributor to the reduction in blood pressure. The vasodilating effect is mainly a result of the influence of anaesthetics on the smooth muscle of blood vessels, which causes a reduction in systemic vascular resistance (Chohan *et al*., 2013) [4].

3.4 Haemato-biochemical observations

Haemoglobin, TEC and PCV levels showed no significant differences between or within groups at any time point, remaining within normal physiological ranges despite minor fluctuations. Although these values slightly decreased during anaesthesia, Total Leukocyte Count (TLC) also exhibited a slight decrease after pre-anaesthetics and induction, with minimal variations during maintenance and a slight recovery post-anaesthesia. Neutrophil counts gradually increased while lymphocyte counts decreased during the maintenance but recovered afterwards; monocyte and eosinophil counts showed slight fluctuations yet remained within the physiological range. The slight non-significant decrease might be related to the transition of fluid from the extravascular space to the intravascular space to sustain normal cardiac output in animals, as noted by Kushwaha *et al.* (2012) [10], or possibly due to haemodilution resulting from ongoing and increased fluid therapy aimed at maintaining hydration and managing anaesthesia-induced hypotension, as pointed out by Kapil (2014) [8].

There were no significant differences in AST and ALT values within or between the isoflurane and sevoflurane groups at any time point, with fluctuations remaining within the normal physiological range. Similarly, BUN and serum creatinine levels showed no significant changes over time, although both groups experienced slight decline in BUN and minor increases in creatinine after pre-anaesthetic administration and induction, all still within normal limits.

Table 1: Numeric scoring system for recording various reflexes

Score	0	1	2	3	4
Jaw relaxation	Not allowing jaw to open and jaws are tightly closed	Marked resistance to opening of jaws and closes quickly	Mild resistance to opening the jaws and closes quickly	Moderate resistance to opening of jaws and closes slowly	No resistance to the opening of jaws and jaws remains open
Eyeball position	-	No rotation of eyeball (Centre position)	Slight downward rotation of the eyeball	Moderate downward rotation of the eyeball	Complete ventromedial rotation of the eyeball
Palpebral reflex	Strong reSpOnse to palpebral reflex	Intact but weak palpebral reflex	Very weak reSpOnse to palpebral reflex	Sluggish reSpOnse to palpebral reflex	Abolished palpebral reflex
Pedal reflex	Strong reSpOnse to pedal reflex	Weak reSpOnse to pedal reflex	Sluggish and occasional reSpOnse to pedal reflex	Very sluggish reSpOnse to pedal reflex	Abolished pedal reflex

Table 2: Mean (±SE) score of quality of maintenance of anaesthesia

Time interval	Jaw relaxation		Eyeball position		Palpebral reflex		Pedal reflex	
	Group I	Group II	Group I	Group II	Group I	Group II	Group I	Group II
After pre-anaesthetic	0.16^{a}±0.16	0.33^{a}±0.21	1.17^{a}±0.17	1.00^{a}±0.00	0.00^{a}±0.00	0.16^{a}±0.16	0.00^{a}±0.00	0.00^{a}±0.00
After induction	3.67^{b}±0.21	3.83^{b}±0.17	3.67^{b}±0.20	3.67^{b}±0.21	3.83^{b}±1.67	4.00^{b}±0.00	3.33^{b}±0.33	3.50^{b}±0.22
10 min	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{c}±0.00	4.00^{b}±0.00	3.67^{bc}±0.21	3.83^{bc}±0.17
20 min	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{c}±0.00	4.00^{b}±0.00	4.00^{c}±0.00	4.00^{c}±0.00
30 min	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{c}±0.00	4.00^{b}±0.00	4.00^{c}±0.00	4.00^{c}±0.00
40 min	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{c}±0.00	4.00^{b}±0.00	4.00^{c}±0.00	4.00^{c}±0.00
50 min	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{b}±0.00	4.00^{c}±0.00	4.00^{b}±0.00	4.00^{c}±0.00	4.00^{c}±0.00

Mean bearing different superscripts in rows and columns differ significantly ($p \leq 0.05$) and the same superscripts in rows and columns differ non-significantly (p>0.05) with each other (lower case stands for within group variation)

Table 3: Mean (±SE) time for recovery from anaesthesia

Time (minutes)	Group (N=6)	
	Group I	Group II
Extubation	9.05^{Aa}±1.16	6.57^{Aa}±0.92
Head lift	15.70^{Aab}±1.26	12.25^{Aab}±0.94
Sternal recumbency	26.72^{Ab}±4.58	25.01^{Ab}±2.50
Standing time	62.23^{Ac}±4.76	41.50^{Bc}±4.70
Completely recovery	88.07^{Ad}±6.81	62.12^{Bc}±6.70

Mean bearing different superscripts in rows and columns differ significantly ($p \leq 0.05$) and the same superscripts in rows and columns differ non-significantly (p>0.05) with each other (lower case stands for within group variation and upper case stands for between group variation)

Table 4: Mean (±SE) value of physiological observations (rectal temperature (°F), respiration rate and heart rate)

Time interval	Rectal Temperature		Respiration Rate		Heart Rate	
	Group I	Group II	Group I	Group II	Group I	Group II
Baseline	102.13^{a}±0.21	102.15^{a}±0.14	25.33^{a}±1.11	26.33^{a}±0.95	104.00^{a}±4.13	107.33^{a}±5.00
After pre-anaesthetic	101.70^{a}±0.18	101.97^{ac}±0.08	22.33^{b}±0.61	21.83^{b}±1.04	118.00^{ab}±6.04	121.17^{ab}±7.03
After induction	100.78^{b}±0.30	100.65^{c}±0.42	16.33^{c}±0.61	16.66^{c}±0.84	125.00^{b}±5.34	124.67^{b}±4.25
10 min	99.88^{c}±0.28	100.13^{cd}±0.41	12.33^{d}±0.61	12.17^{d}±0.40	123.33^{b}±3.68	123.50^{b}±3.95
20 min	99.37^{c}±0.18	99.55^{d}±0.33	12.5^{d}±0.62	12.5^{d}±0.62	118.33^{ab}±5.48	121.67^{ab}±4.74
30 min	99.12^{c}±0.20	99.07^{d}±0.16	13.00^{d}±0.44	12.17^{d}±0.54	111.67^{ab}±4.11	123.33^{ab}±4.61
40 min	99.23^{c}±0.22	99.53^{d}±0.30	12.66^{d}±0.99	12.67^{d}±0.42	110.67^{ab}±7.00	121.00^{ab}±7.39
50 min	99.30^{c}±0.25	99.53^{d}±0.24	12.67^{d}±0.42	12.33^{d}±0.67	109.00^{ab}±6.17	119.33^{ab}±7.04
Complete recovery	100.03^{b}±0.14	100.23^{cd}±0.29	24.00^{a}±0.73	24.66^{a}±0.84	115.00^{ab}±4.09	123.00^{ab}±4.52

Mean bearing different superscripts in rows and columns differ significantly ($p \leq 0.05$) and the same superscripts in rows and columns differ non-significantly ($p>0.05$) with each other (lower case stands for within group variation)

Table 5: Mean (±SE) value of hemodynamic observations (blood pressure and SpO_2)

Time interval	Systolic blood pressure		Diastolic blood pressure		Mean arterial pressure		SpO_2	
	Group I	Group II	Group I	Group II	Group I	Group II	Group I	Group II
Baseline	133.00^{a}±3.03	130.17^{a}±2.40	77.00^{a}±6.77	75.00^{a}±3.38	95.67^{ad}±4.13	93.39^{ab}±2.89	97.50^{a}±0.62	98.33^{ab}±0.67
After pre-anaesthetic	134.83^{a}±2.93	132.17^{a}±2.93	76.50^{ab}±5.19	76.67^{a}±2.35	95.94^{ad}±3.51	95.17^{a}±2.07	98.33^{ab}±0.33	97.83^{a}±0.48
After induction	132.17^{a}±2.40	129.17^{ab}±2.56	73.83^{abc}±4.18	74.83^{a}±3.64	93.28^{abd}±2.60	92.94^{ab}±2.24	97.83^{a}±0.48	97.33^{a}±0.71
10 min	125.67^{abd}±2.64	122.67^{bc}±1.52	67.33^{bc}±2.68	72.33^{a}±2.89	86.78^{bcd}±2.17	89.11^{ab}±1.85	98.67^{ab}±0.49	99.33^{a}±0.33
20 min	120.33^{bcd}±3.16	119.33^{c}±2.23	68.00^{bc}±4.00	70.50^{a}±3.74	85.44^{bcd}±3.41	86.78^{b}±2.38	98.33^{ab}±0.67	98.83^{ab}±0.60
30 min	117.50^{cd}±2.22	117.50^{c}±2.68	67.33^{c}±3.53	68.00^{a}±2.63	84.06^{cd}±2.40	84.50^{b}±1.41	99.67^{b}±0.21	98.67^{ab}±0.49
40 min	113.00^{c}±2.96	117.17^{c}±1.38	66.67^{c}±3.93	68.67^{a}±3.68	82.11^{c}±2.97	84.83^{b}±2.71	99.33^{b}±0.42	99.17^{b}±0.47
50 min	115.50^{c}±3.28	118.00^{c}±1.15	67.67^{c}±2.39	67.67^{a}±3.59	83.61^{cd}±1.96	84.44^{b}±2.31	99.17^{b}±0.54	99.17^{b}±0.48
Complete recovery	124.33^{d}±2.60	124.83^{c}±2.48	75.67^{a}±3.24	74.33^{a}±3.98	91.89^{ad}±1.90	91.17^{b}±2.60	98.83^{ab}±0.48	98.83^{ab}±0.47

Mean bearing different superscripts in rows and columns differ significantly ($p \leq 0.05$) and the same superscripts in rows and columns differ non-significantly ($p > 0.05$) with each other (lower case stands for within group variation)

Table 6: Mean (±SE) value of haematological parameters

Time interval	Haemoglobin(g/dL)		TLC (million/µL)		PCV (%)		TLC (thousand/µL)	
	Group I	Group II	Group I	Group II	Group I	Group II	Group I	Group II
Baseline	13.53±0.56	14.03±0.45	7.37±0.20	7.41±0.20	41.89±0.55	42.22±0.82	12.34±0.26	12.17±0.37
After pre-anaesthetic	13.37±0.53	13.78±0.45	7.30±0.20	7.32±0.20	41.47±0.47	42.01±0.78	12.12±0.27	11.98±0.39
After induction	13.08±0.33	13.33±0.48	7.19±0.20	7.17±0.19	41.25±0.44	41.48±0.63	11.96±0.29	11.81±0.31
15 min	13.13±0.32	12.73±0.35	7.15±0.22	7.11±0.21	40.83±0.44	40.97±0.60	11.83±0.23	11.78±0.29
30 min	12.63±0.40	12.60±0.52	7.10±0.22	7.04±0.21	40.70±0.32	40.72±0.59	11.68±0.21	11.73±0.33
45 min	12.45±0.38	12.57±0.98	7.06±0.21	6.94±0.22	40.76±0.38	40.67±0.60	11.75±0.27	11.88±0.33
Complete recovery	12.57±0.30	12.77±1.06	7.08±0.19	7.16±0.22	41.02±0.47	41.07±0.46	11.84±0.24	11.89±0.25

Table 7: Mean (±SE) value of differential leukocyte count

Time interval	Neutrophil count (%)		Lymphocyte count (%)		Monocyte count (%)		Eosinophil count (%)	
	Group I	Group II	Group I	Group II	Group I	Group II	Group I	Group II
Baseline	70.17±3.11	68.00±3.04	22.83±1.82	23.50±2.47	3.33±0.61	3.83±0.70	2.67±0.67	2.83±0.60
After preanaesthetic	71.00±3.27	68.33±2.70	22.50±1.28	23.17±2.65	3.50±0.67	4.00±0.52	2.70±0.55	2.80±0.48
After induction	71.50±3.56	69.17±2.30	22.17±0.98	22.33±2.50	3.33±0.56	3.83±0.31	2.83±0.91	2.90±0.32
15 min	71.83±3.23	69.50±2.73	21.17±1.35	21.17±2.48	3.17±0.48	3.67±0.56	2.83±0.79	2.75±0.36
30 min	72.50±2.74	70.33±2.67	20.17±1.28	20.50±2.53	2.83±0.60	3.67±0.49	2.75±0.79	2.85±0.60
45 min	72.67±2.65	70.67±2.63	19.67±1.61	20.17±2.74	2.96±0.58	3.50±0.62	2.67±0.80	3.07±0.44
Complete recovery	72.83±2.75	71.50±2.73	20.83±1.40	21.67±2.60	3.33±0.61	3.67±0.71	2.58±0.49	2.83±0.31

Table 8: Mean (±SE) value Biochemical parameters

Time interval	AST (IU/L)		ALT (IU/L)		BUN (mg/dL)		Creatinine(mg/dL)	
	Group I	Group II	Group I	Group II	Group I	Group II	Group I	Group II
Baseline	20.67±2.50	18.50±2.22	42.67±6.58	38.50±7.52	21.93±2.58	20.64±3.33	0.90±0.13	0.84±0.16
After pre-anaesthetic	21.00±2.53	19.50±2.33	41.50±6.91	37.83±6.97	21.55±2.51	20.35±3.30	0.97±0.18	0.87±0.12
After induction	21.83±2.73	20.33±2.39	40.83±6.34	38.83±6.57	20.68±2.61	20.22±3.23	1.16±0.16	0.90±0.12
15 min	21.50±2.45	20.00±2.24	39.67±6.22	39.17±5.59	20.32±2.64	19.70±3.22	1.18±0.19	0.92±0.09
30 min	22.00±2.54	19.33±1.86	40.17±6.30	39.67±6.24	20.31±2.33	19.44±3.20	1.15±0.14	0.90±0.09
45 min	21.83±2.17	20.17±1.17	40.50±6.44	38.83±5.83	20.00±2.38	19.24±3.38	1.17±0.13	0.90±0.14
Complete recovery	23.17±2.70	21.33±1.78	40.17±6.98	39.00±6.57	20.13±2.45	19.30±3.32	1.11±0.15	0.93±0.20

4. Conclusions

Isoflurane and sevoflurane were found to be effective inhalant anaesthetics for dogs premedicated with diazepam-butorphanol and induced with etomidate, as both produced comparable anaesthetic depth, physiological reSpOnses and hemodynamic stability, with no significant differences in temperature, respiration, heart rate, SpO_2, blood pressure or haematological and biochemical parameters. Although both agents provided smooth and controlled anaesthesia, recovery from sevoflurane was notably faster than isoflurane, with significantly shorter, standing and complete recovery time. Etomidate induction was smooth and resulted in effective loss of consciousness; however, tracheal intubation posed some difficulty due to the persistence of laryngeal reflexes.

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