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RESEARCH ARTICLE

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Assessment of anaesthetic quality and haemodynamic responses to dexmedetomidine-butorphanol-tiletamine-zolazepam-lignocaine continuous rate infusion in dogs

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This study aimed to evaluate the efficacy of a continuous rate infusion (CRI) protocol for total intravenous anaesthesia (TIVA) in animals chosen for various elective surgical procedures. A prospective clinical study was conducted on six healthy adult dogs of either sex, various breeds and ages, classified as ASA class I or II. All the dogs received intramuscular meloxicam (0.2 mg/kg) as pre-emptive analgesic. After 30 min, dexmedetomidine (5µg/kg), butorphanol (0.2mg/kg) and tiletamine-zolazepam (4mg/kg) were administered intramuscularly. The actual time of sedation was noted by attainment of lateral recumbency with the loss of righting reflex. After induction and successful intubation, a single intravenous bolus of lignocaine at 2mg/kg was administered as a loading dose. Maintenance of anaesthesia was achieved by CRI of dexmedetomidine (0.5 µg/kg/hr), butorphanol (0.2 mg/kg/hr), tiletamine-zolazepam (2 mg/kg/hr), and lignocaine (50 µg/kg/min) diluted in normal saline. The anaesthetic efficacy was evaluated based on clinical and physiological parameters as well as state entropy monitoring. The anaesthetic protocol facilitated smooth progression through induction, maintenance, and recovery phases by effectively providing adequate skeletal muscle relaxation, analgesia, and maintenance of cardiovascular and respiratory homeostasis.

Keywords: Butorphanol, Continupous rate infusion, Dexmedetomidine, Dog, Lignocaine, Tiletamine-zolazepam

Balanced anaesthesia involves administering small doses of several drugs, with each medication targeting a different component of the anaesthetic triad - unconsciousness, analgesia, and muscle relaxation (Brown *et al*., 2018). This method is widely preferred because it optimizes the desired effects while limiting adverse reactions, as the side effects of most drugs are dose dependent (Sooryadas *et al*., 2019). Total intravenous anaesthesia (TIVA), is the continuous delivery of an injectable anaesthetic to maintain an appropriate depth of anaesthesia during surgery. In this method, the concept of minimum alveolar concentration (MAC) used in inhalational anaesthesia is substituted with the minimum infusion rate (MIR), which refers to the infusion rate required to keep 50% of patients unresponsive to surgical stimuli (Joubert, 2009). TIVA offers several benefits compared to inhalational anaesthesia, such as enhanced haemodynamic stability, smoother recovery, and the avoidance of staff exposure to volatile anaesthetic agents (Bustamante *et al*., 2022).

This study investigates the effectiveness of a total intravenous anaesthesia (TIVA) regimen that includes dexmedetomidine, tiletamine–zolazepam, and butorphanol in canine patients. The sedative and analgesic action of dexmedetomidine, paired with the dissociative and tranquilising properties of tiletamine–zolazepam, along with the unique mixed agonist–antagonist activity of butorphanol, may collectively shape the depth of anaesthesia, cardiovascular and respiratory responses, and the overall quality of recovery. Analyzing the interactions of these drugs will help clarify whether this combination offers a consistent and well-balanced injectable anaesthetic approach for dogs, particularly in clinical scenarios where inhalation anaesthesia is not accessible. Such evaluation is crucial for confirming the safety and effectiveness of this injectable anaesthesia method under those specific conditions.

Materials and Methods

The study involved six clinically healthy dogs of both sexes, classified either as ASA class I or II, presented for elective surgical interventions. Each animal was assigned a sequential identifier from D1 to D6. The study population consisted of two males and four females, with age varying from 6 months to 12 years and with a mean body weight of 17.73±3.25 kg.

All the dogs received an intramuscular injection of meloxicam (0.2 mg/kg) as pre-emptive analgesic. After 30 min, balanced general anaesthesia was induced according to the study protocol. The dogs received a cocktail anaesthesia consisting of dexmedetomidine (5 µg/kg), butorphanol (0.2 mg/kg), and tiletamine-zolazepam (4 mg/kg) administered through intramuscular route. The time of sedation was noted by attainment of lateral recumbency with loss of righting reflex. After induction and successful intubation, a single intravenous bolus of lignocaine at 2 mg/kg was administered as loading dose. Maintenance of anaesthesia was achieved by CRI of dexmedetomidine (0.5

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μg/kg/hr), butorphanol (0.2 mg/kg/hr), tiletamine-zolazepam (2 mg/kg/hr), and lignocaine (50 μg/kg/min) diluted in normal saline.

The CRI of dexmedetomidine, butorphanol, tiletamine-zolazepam and lignocaine was prepared as per the steps mentioned below.

A. Calculated total milligrams (or micrograms) of drug needed per hour

a. For lignocaine:

Amount of drug (mg) =

$$\frac{\text{CRI dose of drug (mcg/kg/min) x body weight (kg) x 60 (min)}}{\text{1000 (mcg)}}$$

b. For dexmedetomidine:

Amount of drug (mcg) =
CRI dose of drug (mcg/kg/hr) x body weight (kg)

c. For tiletamine–zolazepam and butorphanol

Amount of drug (mg) =
CRI dose of drug (mg/kg/hr) x body weight (kg)

B. The volume of fluid (CRI infusion solution) to be delivered per hour, i.e. mL/hr = rate of flow of CRI (mL/kg/hr) x body weight (kg)

(The rate of flow of CRI infusion solution was taken as 10 mL/kg/hr).

The amount of drug in milligrams (lignocaine tiletamine-zolazepam and butorphanol) or micrograms (dexmedetomidine) needed per mL of infusion was calculated as: mg (or mcg) of drug per hour ÷ mL of infusion per hour.

The milligrams or micrograms of the drug per mL of infusion obtained in the above step was multiplied by 100 or 500 in case of 100 mL or 500 mL NS bottles, respectively to get the amount of drug (in mg or mcg) required to be added. The total milligram or microgram of each drug to be added was divided by the presentation of the respective drugs to obtain the volume of each drug required for preparation of CRI. These volumes were totaled, and an equal amount of fluid from the 100 mL or 500 mL bottle was withdrawn and discarded. The volumes of dexmedetomidine, tiletamine-zolazepam, lignocaine, and butorphanol, were added to the infusion fluid to make up 100 mL or 500 mL of the CRI. This infusion was then administered at 10 mL/kg/hr using the volumetric infusion pump so that the patient received dexmedetomidine @ 0.5 mcg/kg/hr, lignocaine @ 50 mcg/kg/min, tiletamine-zolazepam @ 2 mg/kg/hr and butorphanol @ 0.2 mg/kg/hr.

All the animals were allowed to breathe 100% oxygen through a suitable breathing circuit connected to endotracheal tube. Nociception noticed during surgery was managed by bolus administration of the ongoing CRI. The volume of the CRI and quantity of the drugs administered as bolus was noted and recorded in all the animals during induction, maintenance and recovery to assess the effects of the anaesthetic drug combination. Physiological parameters (heart rate, pulse rate, respiratory rate and rectal temperature) were recorded before anaesthesia, immediately following induction and every 10 min thereafter till recovery.

Haematology was done before induction of anaesthesia and 15 min after induction. Serum biochemistry and blood gas analysis were done before induction and after recovery from anaesthesia. Electrocardiogram was recorded before induction, following induction and every 10 min thereafter till recovery from anaestheia. Electroencephalograph was monitored using state entropy and values were recorded following induction and every 10 min thereafter till recovery.

The data obtained were recorded and statistically analysed using SPSS version 24.0.

Results and Discussion

Signs associated with sedation (Table 1) included side to side head movements (n=3), head down, sternal recumbency and lateral recumbency (n=6). The time taken for head movements ranged from 1-4 min with a mean±SE of 5.00±1.03 min. The side to side head movements during sedation were noticed partly in

Table 1: Signs associated with sedation and time taken for sedation after administration of the anaesthetic combination in animals studied.

Dog ID	Salivation (min)	Ptosis (min)	Head movements (min)	Sternal recumbency (min)	Head down (min)	Time taken for sedation (attainment of lateral recumbency with abolition of righting reflex) (**min**)
D1	Not exhibited	Not exhibited	1	2	2	3
D2	Not exhibited	Not exhibited	4	6	9	10
D3	Not exhibited	Not exhibited	1	2	3	5
D4	Not exhibited	Not exhibited	Not exhibited	3	4	4
D5	Not exhibited	Not exhibited	Not exhibited	2	2	4
D6	Not exhibited	Not exhibited	Not exhibited	3	4	4
Mean±SE			1.00±0.63	3.00±0.63	4.00±1.06	5.00±1.03

Table 2: Recovery signs associated with recovery and their respective times (min) following cessation of CRI.

Dog ID	Return of eyeballs to centre (min)	Return of pedal reflex (min)	Return of jaw muscle tone (min)	Rejection of endotracheal tube (min)	Head lift (min)	Sternal recumbency (min)	Time taken for recovery (min)
D1	6	8	7	9	9	21	9
D2	24	26	27	30	45	57	30
D3	13	18	25	31	43	45	31
D4	39	44	51	54	106	125	54
D5	5	7	12	15	16	27	15
D6	19	21	34	36	47	55	36
Mean±SE	17.67±5.21	20.67±5.56	26.00±6.45	29.17±6.52	44.33±13.98	55.00±15.21	29.17±6.52

Table 3: Change in physiological parameters and state entropy at different stages of anaesthesia.

Variables	Before induction	Immediately after induction	At the time of recovery	F-value (P-value)
Rectal temperature (ºC)	38.68^{a}±0.23	38.42^{a}±0.25	35.47^{b}±1.03	11.887* (0.017)
Heart rate (beats/min)	123.33^{a}±10.15	107.17^{a}±5.31	85.33^{b}±3.09	12.953** (0.002)
SPO_2 (%)	-	99.00±0.68	99.67±0.21	0.933^{ns} (0.394)
Respiratory rate (breaths/min)	Panting	8.17±0.79	23.00±3.65	3.896* (0.011)
$ETCO_2$ (mm Hg)	-	49.50±3.36	27.50±2.81	8.981** (<0.001)
Mean arterial pressure (mm Hg)	-	88.50±9.24	107.00±9.84	1.189^{ns} (0.288)
Sedative entropy	-	33.67±3.62	84.50±1.52	15.081**(<0.001)

*Means having different letter as superscript differ significantly within a row (*significant at 0.05 level, **significant at 0.01 level); ns: non-significant.*

accordance with the findings of Savas *et al.* (2001) and Chandramohan *et al*. (2023). These findings were aligned with the previous studies by Barletta *et al*. (2011) and Krimins *et al*. (2012b), who reported rapid sedation within 4-7 min following administrations of dexmedetomidine combined with butorphanol and tiletamine-zolazepam. The reduced time of onset of sedation was linked to the lipophilic nature of dexmedetomidine (Degroot *et al*., 2020; Manjusha *et al*., 2023), which crossed the lipid membranes of blood brain barrier and produced CNS depression. The pedal and palpebral reflexes remained absent for the entire duration of anaesthesia in all animals. This was mainly due to the synergistic effects of the drugs used in this protocol. Animals exhibited satisfactory muscle relaxation in the limbs, abdomen, and jaw throughout the study. The effective muscle relaxation observed in the current study was supported by the findings of Tiwari *et al*. (2024), who reported that alpha-2 agonists like dexmedetomidine inhibit neuronal impulse transmission within the central nervous system, leading to significant muscle relaxation. Ventromedial position of eyeball in all animals indicated surgical plane of anaesthesia. Mild, transient nociception was noticed in two of the six dogs while manipulating the skin during suturing. These findings were aligned with findings of Pelligand *et al*. (2016), who explained that somatic nociceptive signals originate from the skin and deeper tissues, rather than from visceral organs. The overall quality of anaesthetic maintenance was good based on the analgesia, muscle relaxation, absence of response to surgical stimuli and maintenance of physiological parameters. The recovery time was prolonged in this protocol with a mean±SE of 29.17±6.52 min (Table 3). The use of constant rate infusions for prolonged anaesthesia maintenance can increase the recovery duration (Gozalomarcilla *et al*., 2021). Two out of six animals in the group exhibited signs of transient delirium with shivering and hypothermia (Pottie *et al*., 2007). Anaesthesia was maintained through CRI for durations ranging from 46 to 219 min, with a mean±SE of 119.00±29.22 min.

There was a significant (P<0.05) reduction in body temperature after recovery compared to baseline values (Table 2). Dexmedetomidine with its α2-adrenergic agonism, can cause vasodilation and reduced heat production, which further contribute to hypothermia (Bisht *et al*., 2018) and cause reduced metabolism (Tiwari *et al*., 2024). Administration of a tiletamine zolazepam–dexmedetomidine combina-

Table 4: Change in serum biochemical parameters before induction and at the time of recovery.

Variables	Before induction	At the time of recovery	t-value (P-value)
ALT (U/L)	32.92±4.03	31.48±2.27	0.460^{ns} (0.665)
Total protein (g/dL)	6.02±0.46	5.67±0.50	0.889^{ns} (0.415)
Albumin (g/dL)	2.19±0.08	2.46±0.32	0.790^{ns} (0.465)
Glucose (mg/dL)	120.83±5.4	145.33±21.57	1.398^{ns} (0.292)
Globulin (g/dL)	3.83±0.45	3.20±0.62	1.418^{ns} (0.215)
Creatinine (g/dL)	1.050±0.108	0.858±0.101	2.339^{ns} (0.066)

Means do not differ significantly (P>0.05); ns: non-significant.

Table 5: Evaluation of blood gas analysis at different stages of anaesthesia.

Variables	Before induction	Immediately after induction	At the time of recovery	F-value (P-value)
pH	-	7.31±0.04	7.36±0.01	5.672^{ns} (0.054)
P_vO_2 (mmHg)	40.08^{b}±3.91	76.02^{a}±7.19	78.52a±15.54	5.078^{*} (0.030)
P_vCO_2 (mmHg)	40.58±5.69	54.67±4.78	45.90±3.6	3.231^{ns} (0.083)
Bicarbonate (mmol/L)	26.27±1.75	27.22±0.69	24.45±1.26	0.981^{ns} (0.408)
Lactate (mmol/L)	2.50±0.41	1.60±0.34	1.74±0.34	2.328^{ns} (0.148)
Sodium (mmol/L)	148.67±3.04	149.33±2.23	149.67±2.12	0.368^{ns} (0.701)
Potassium (mmol/L)	3.70±0.35	3.63±0.14	3.78±0.16	0.154^{ns} (0.859)
Calcium (mmol/L)	1.26±0.02	1.29±0.05	1.29±0.03	0.508^{ns} (0.616)
Chloride (mmol/L)	116.33±1.56	116.00±2.15	115.50±1.29	0.155^{ns} (0.858)

*Means having different letters as superscript differ significantly within a row: *significant at 0.05 level; ns: non-significant.*

tion intravenously has been reported to lower the core body temperature (Manjusha *et al*., 2023), while tiletamine itself contributes to hypothermia by interfering with the body's thermoregulatory control mechanisms (Pereira *et al*., 2019). A significant (P<0.01) reduction in heart rate was observed in all six animals after induction and at recovery compared to baseline (Table 3). The reduction in heart rate was mainly due to the peripheral vasoconstriction of dexmedetomidine (Barletta *et al*., 2011). The animals showed a reduced respiration rate with elevated $ETCO_2$ due to the diminished alveolar ventilation and subsequent carbon dioxide build up immediately after induction. It could be due to the effect of butorphanol that potentiated the respiratory depressant effect of dexmedetomidine, leading to a significant (P<0.05) reduction in respiration (Ko *et al*., 2001). All the animals under the study exhibited stable SPO_2 values above 95% (Table 3), which indicated minimal vasoconstriction. At lower or steady-state dexmedetomidine concentrations, vasoconstriction is minimal and does not significantly reduce peripheral oxygen saturation (Talk *et al*., 2018). The non-invasive mean arterial pressure (MAP) of all animals was more than 60 mm Hg (Table 3), which indicated stable tissue perfusion (Hall *et al*., 2011). The stable mean arterial pressure observed in both groups aligns with the results of Kucharski *et al*. (2022), who reported that administering tiletamine-zolazepam as a CRI, maintained haemodynamic variables at consistently high yet stable levels without causing hypotension. The electrocardiographic evaluations remained within normal physiological ranges in all animals. Tiletamine's sympathomimetic action leads to mild increase in heart rate, without causing long lasting alterations in electrical activity (Pereira *et al*., 2019).

The serum-biochemical parameters like ALT, total protein, albumin, globulin, glucose, and creatinine (Table 4) did not show any significant (P>0.05) changes prior to induction and after recovery from anaesthesia. The ALT values remained within normal ranges, supporting adequate liver function during anaesthesia. The creatinine values prior to induction and after recovery were within normal range, which indicated stable renal function. On blood gas analysis, the pH , PvO2, PvCO2, lactate, sodium, potassium, calcium and chloride values (Table 5) did not show a significant (P>0.05) difference between pre-induction, after induction and post-recovery.

The degree of consciousness during anaesthesia was evaluated by state entropy (SE) monitoring. Mean± SE value at induction (0 min) was 33.67±3.62, indicating deep sedation (Figueroa *et al*., 2025). During maintenance the average mean entropy value was 49.5±5.5, which indicated the dimishined cortical activity of brain. Entropy increased to 84.50±1.52 during recovery (Table 3). Morgaz *et al*. (2011) and Brás *et al*. (2014) consistently demonstrated that deeper

anaesthesia leads to reduced SE values, reflecting diminished cortical activity. Figueroa *et al*. (2025) highlighted that during sedation in animals, SE decreased progressively as sedation deepened. Typically, SE values during normal sedation ranged from approximately 40 to 60. With deeper levels of anaesthesia, SE values could drop below 40 as recorded in the current study. The effective antinociception produced by the synergistic actions of drugs in the cocktail anaesthesia led to reduced cortical arousal and muscle activation (Thengchaisri *et al*., 2019).

In conclusion it was found that the anaesthetic regimen used in this study allowed for smooth induction, maintenance, and recovery, while providing enough muscle relaxation, pain relief, and cardiorespiratory stability. These results indicate that the protocol is safe and effective for various soft tissue and orthopaedic procedures.

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