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Investigation of the effects of propofol and sevoflurane anesthesia on oxidant-antioxidant status and biochemical parameters in Turkish hair goats

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Objective
To investigate the effects of propofol and sevoflurane anesthesia on the oxidant-antioxidant status and biochemical parameters in Turkish hair goats.

Methods
The study was conducted from February 15 through March 15, 2024. Seven clinically healthy female Turkish hair goats aged 1 to 2 years with an average live weight of 29.95 (26.1 to 33.8) kg were included. A bolus of propofol (6 mg/kg) was injected IV. The goats were placed in the sternoabdominal position and administered 1% to 3% sevoflurane with 100% oxygen for 2 hours with the help of a vaporizer. Blood samples were collected before and after the administration of propofol and at the 15th, 30th, 60th, and 120th minutes after the start of sevoflurane administration in nonanticoagulated tubes. The oxidant-antioxidant status (serum total antioxidant status, total oxidant status, paraoxonase activity, and arylesterase activity) and biochemical parameters (levels of glucose, AST, GGT, ALT, ALP, LDH, creatine kinase, amylase, lipase, creatinine, urea, uric acid, high-density lipoprotein, low-density lipoprotein, total protein, albumin, total bilirubin, direct bilirubin, magnesium, chlorine, calcium, iron, sodium, phosphorus, and potassium) were evaluated. The heart rate, respiratory rate, and body temperature of the goats were measured using a bedside monitor.

Results
Arylesterase activity increased with sevoflurane administration. Changes were observed in heart rate and body temperature as well as in the levels of glucose, creatine kinase, urea, total bilirubin, and potassium at different time intervals of propofol-sevoflurane administration.

Conclusions
Propofol-sevoflurane anesthesia has no adverse effects on oxidant-antioxidant status in Turkish hair goats.

Clinical Relevance
The evaluation of heart rate and body temperature, as well as glucose, creatine kinase, urea, total bilirubin, and potassium levels, must be considered in propofol-sevoflurane anesthesia protocol.

Keywords: anesthesia, Turkish hair goat, oxidant/antioxidant status, propofol, sevoflurane

Anesthesia refers to insensitivity, numbness, and the temporary disappearance of sensations. Anesthetic drugs induce analgesia, sedation, narcosis, and hypnosis. There are 3 different ways to administer anesthetics drugs: general, regional, and local anesthesia.[1] For inducing general anesthesia, injectable anesthetics (eg, barbiturates, cyclohexamines, or propofol), inhalation anesthetics (eg, halothane, isoflurane, sevoflurane, or nitrous oxide), or their combination is used.[2]

In clinical veterinary practices, the search for anesthetic agents that can be safely administered for use in short- and medium-duration routine surgeries has continued rapidly in recent years. For this

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purpose, studies[3–5] have been conducted on anesthetic agents in a variety of animals, notably propofol and isoflurane on Angora goats, ketamine and ketamine-xylazine on sheep, and ketamine-xylazine on Bozova Greyhounds.

Propofol is an injectable anesthetic agent that has been successfully used in veterinary medicine and is preferred in outpatient surgeries. It metabolizes rapidly and allows the patient to return to normal activities in a shorter time[6]; this makes it preferable, especially in short-duration procedures, such as biopsy, castration, and ear washing.[7] In addition, propofol reportedly acts as a synthetic antioxidant by reacting with free radicals.[8]

Sevoflurane is an inhalational anesthetic that has been introduced for clinical use in recent years. Sevoflurane is a volatile anesthetic agent that provides rapid induction and recovery of anesthesia, is minimally metabolized in animals,[9] and is 95% to 98% eliminated via the lung.

Free radicals are reactive oxygen species that interact with macromolecules, such as lipids, proteins, and nucleic acids, causing disturbances in cell structure and organelles. Disruption of the balance between the body's antioxidant defense and free radical production that causes lipid peroxidation in cells is defined as oxidative stress.[10]

Surgical stress alone induces a pathological increase in the of free oxygen radical production. The formation of free oxygen radicals is affected by the type and duration of anesthesia and the surgeries performed, whereas the oxidant-antioxidant effects of anesthetic agents vary according to the tissues.[11]

In small ruminants, propofol can be used for the induction of anesthesia and sevoflurane for the maintenance of anesthesia.[12] A literature review revealed that no study has investigated the effects of propofol and sevoflurane anesthesia on oxidative stress and biochemical parameters in Turkish hair goats. This study aimed to investigate the effects of propofol and sevoflurane anesthesia on the oxidant/antioxidant status (total antioxidant status [TAS], total oxidant status [TOS], paraoxonase [PON], and arylesterase [ARES]) and biochemical parameters in Turkish hair goats.

Methods

Study design and animals

This study was initiated upon the approval of the Local Ethics Committee for Animal Experiments at Siirt University (January 30, 2024; No. 2024/01-05). The study included 7 clinically healthy female Turkish hair goats aged 1 to 2 years with a live weight of 29.95 (26.1 to 33.8) kg, obtained from a privately owned farm in the Siirt province. The animals were housed in 2 groups, consisting of 3 and 4 goats, respectively, in the small ruminant shelter of Siirt University Animal Health Practice and Research Hospital during the study. The goats were fed with concentrate feed and hay, and water was given ad libitum. The live weights of the goats were measured and recorded on the morning of the study. The animals were not allowed to eat or drink water for 12 hours before the experiment. Propofol (Dormofol 1%; Vem İlaç) was injected IV as a bolus at a dose of 6 mg/kg. An orogastric probe was inserted into the goats to prevent tympany. While the goats were in the sterno-abdominal position, the trachea was intubated (intubation tube No. 7.5, Kendall Curity Tracheal Tube, Sampran, Thailand). Sevoflurane (Sevorane; Abbott) was administered to the goats using a closed-circuit anesthesia device (SMS 2000 Classic Vent-V, SMS Medıcal Device, Limited Corporation, Ankara, Türkiye) for 2 hours with 100% oxygen at 1% to 3% concentration using a vaporizer.[3] The depth of anesthesia was determined based on the presence of palpebral and tail reflexes. Complications that may have developed due to anesthesia were recorded during the study period. The heart rate, respiratory rate, and body temperature of the goats were measured using a bedside monitor (uMEC12 Vet; Mindray).

Sample collection and analysis

Blood samples were collected from goats before propofol (BP) administration and immediately after propofol (AP) administration; goats were intubated, and then sevoflurane was administered. Blood samples were collected at the 15th, 30th, 60th, and 120th minutes after sevoflurane administration. Blood samples were collected from the vena jugularis, 5 mL in each repetition, into tubes without anticoagulant. The samples were kept at room temperature for 30 minutes, followed by a 10-minute centrifugation at 15,300 X *g*, after which the serum was transferred into Eppendorf tubes. Serum samples were stored at −20 °C and analyzed within 1 week **(Figure 1)**.

Measurement of TAS

Total antioxidant status levels were measured using commercially available kits (Rel Assay Diagnostics Kit; Mega Tıp), with the results reported as millimole Trolox equivalent per liter.[13] This automated method is based on the discoloration of the ABTS radical cation (2,2′-Azino-bis (3-ethylbenzothiazoline-6-sulfonic acid)) (ABTS; Invitrogen) by antioxidants; the assay has excellent precision values, which are lower than 3%.[13]

Measurement of TOS

Total oxidant status levels were measured using commercially available kits (Rel Assay Diagnostics Kit; Mega Tıp). In this method, oxidants present in the sample oxidize the ferrous ion-o-dianisidine complex to ferric ion, and the oxidation reaction is enhanced by the abundance of glycerol molecules in the reaction medium. The ferric ions produce a colored complex with xylenol orange in an acidic medium; subsequently, the color intensity can be measured spectrophotometrically, representing the total amount of oxidant molecules present in the sample. The assay is calibrated with hydrogen peroxide, and the results are expressed in terms of micromolar hydrogen peroxide equivalent per liter.[14]

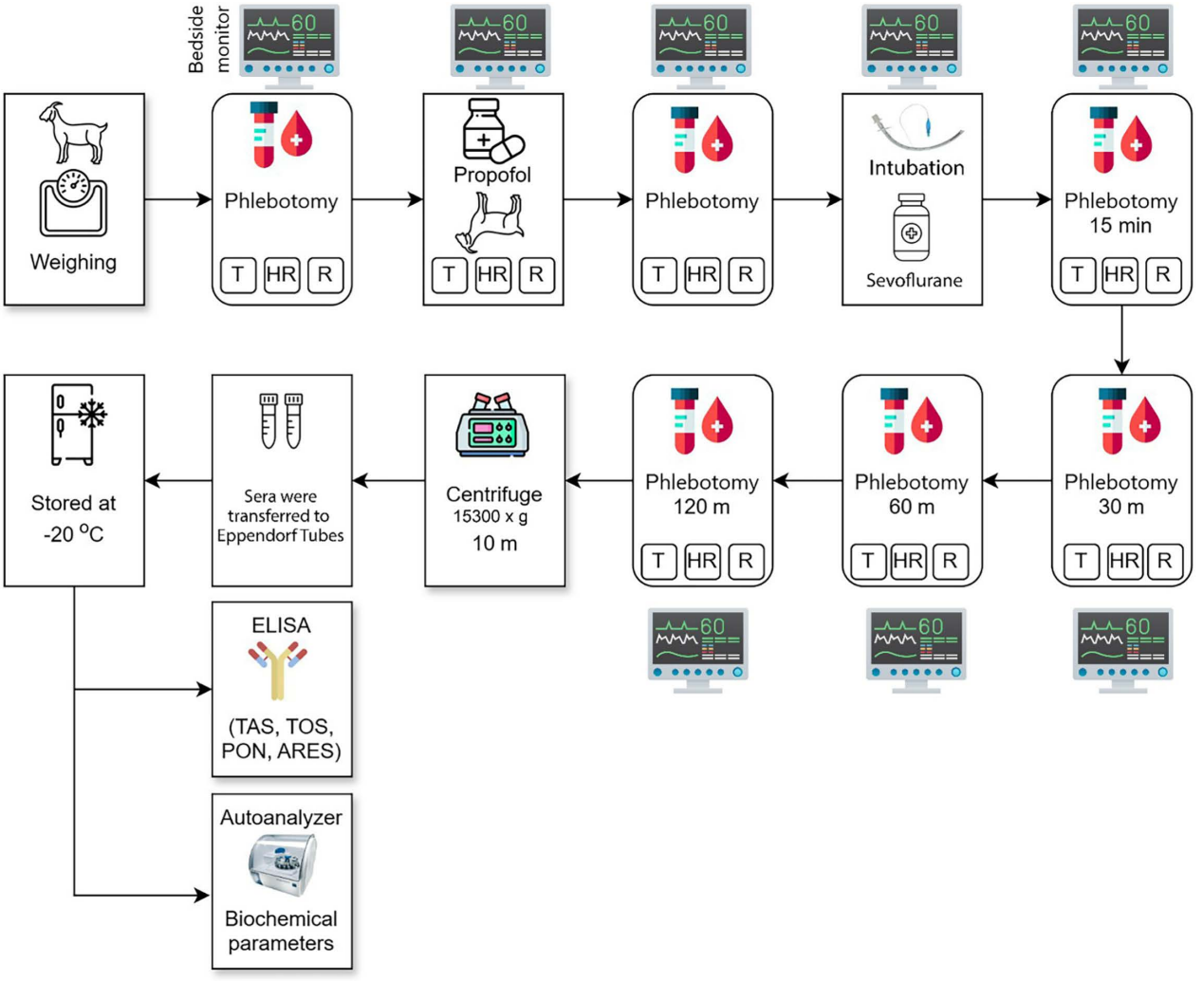

Figure 1—Schematic representation of anesthesia induction, blood sampling, and the analysis processes in Turkish hair goats anesthetized with propofol and sevoflurane. ARES = Arylesterase. HR = Heart rate. PON = Paraoxonase. R = Respiratory rate. T = Temparature. TAS = Total antioxidant status. TOS = Total oxidant status.

Determination of PON and ARES activity

The PON1 activity of the serum samples was examined using a fully automated method developed by Rel Assay Diagnostics (Mega Tıp). According to this method, PON activity is measured in 2 media with and without NaCl, representing salt-stimulated PON activity and basal PON activity, respectively. The hydrolysis of paraoxon (diethyl-p-nitrophenyl phosphate) is monitored by measuring the increase in absorbance at 37 °C and 412 nm. The amount of p-nitrophenol produced by hydrolysis is calculated using the molar absorption coefficient 17,000 Mj1cmj1 (at pH 8). The net enzymatic activity is calculated by subtracting the basal activity value from the salt-stimulated activity value. The results are expressed as unit per liter, which is equal to the hydrolysis of 1 µmol substrate in 1 minute and 1 L.[15]

The PON ARES activity of the serum samples was measured using a fully automated method developed by Rel Assay Diagnostics (Mega Tıp). In this method, phenylacetate is used as a substrate for the measurement of ARES activity, with the hydrolysis of phenylacetate resulting in phenol and acetic acid production. The resulting phenol joins to 4-aminoantipyrine and potassium (K) ferricyanide and is measured using a colorimetric method. The ARES enzyme activity is calculated from 4,000 Mj1cmj1, which is the molar absorption coefficient of the resulting colored complex. The results are expressed as unit per liter, which is equal to the hydrolysis of 1 µmol phenylacetate in 1 minute and 1 L.[15]

Analysis of biochemical parameters

Glucose, AST, GGT, ALT, ALP, LDH, creatine kinase (CK), amylase, lipase, creatinine, urea, uric acid, high-density lipoprotein, low-density lipoprotein, total protein, albumin, total bilirubin (TBIL), direct bilirubin, magnesium, chlorine, calcium, iron, sodium, phosphorus, and K levels were determined using an autoanalyzer (ADVIA 1800 Chemistry System; Siemens Healthcare Diagnostics).

Statistical analysis

Before conducting the study, G*Power (version 3.1.9.7, Heinrich-Heine-Universität Düsseldorf, Germany) was used to calculate the adequate sample size. By considering an effect size of 0.05, type 1 error of 0.05, and power of 0.8 and the number of groups as 1, the number of repeated measurements as 6, and the rmcorr as 0.6, the sample size was determined to be 7. Prior to statistical analysis, parametric test assumptions were assessed by the Shapiro-Wilk test for normality and the Mauchly test for sphericity. The Greenhouse-Geisser correction was used in cases where the assumption of sphericity was not met. Different time intervals were analyzed with repeated measures ANOVA. The Tukey test was used for paired comparisons where the within-variable difference was significant. The threshold of statistical significance was established at $P = .05$ in all statistical analyses. Statistical calculations were performed using SPSS Statistics, version 26 (IBM Corp).

Results

Anesthetic induction in goats was rapid and uneventful. The animals were laid on their sternum within 10 to 25 seconds of anesthesia administration, and tracheal intubation was easily accomplished in all goats. During the continuation of anesthesia, salivation was detected in 2 animals, whereas signs of awakening (palpebral reflex, tail wagging) were observed in 3 animals at 12, 18, and 24 minutes, respectively. Deep anesthesia was continued by raising the sevoflurane concentration from 2% to 3%. After the termination of anesthesia, the endotracheal tubes were removed from the goats that had swallowing reflexes within 1 to 3 minutes. The animals awoke completely and stood up within 8 to 12 minutes after anesthetic termination.

Total antioxidant status and TOS levels, as well as PON and ARES activities in goat serum samples, were compared between different timepoints: BP and AP administration and at 15, 30, 60, and 120 minutes after starting sevoflurane administration. No differences in TAS and TOS levels or PON activities were found across time. However, a significant increase in ARES activity was observed at the 15th minute of sevoflurane administration compared to the activity level immediately AP administration ($P < .05$; **Table 1**).

When the heart rate, respiratory rate, and body temperature of the goats were compared between different timepoints, we found that the heart rate was not statistically different between the measurements BP and AP administration; however, a significant increase was found at the 15-minute mark, followed by a significant decrease at the 30-minute mark ($P < .001$); there were no statistical differences between heart rate measurements at the 30th, 60th, and 120th minutes of sevoflurane administration. Body temperature remained statistically unchanging across the measured timepoints except for a significant decrease at the 120th minute of sevoflurane administration ($P < .05$). Respiratory rates did not show any significant differences across the measurement timepoints **(Table 2)**.

Glucose levels were significantly higher AP administration than those BP administration and at the 15th, 30th, 60th, and 120th minutes after sevoflurane administration ($P < .01$). Creatine kinase level changes were significant ($P < .05$), increasing more than 2-fold, from 71.57 ± 10.75 U/L BP administration to 148.71 ± 33.84 U/L at the 120th hour. The differences in mean urea levels were significant between the 15th, 60th, and 120th minutes ($P < .05$). Total bilirubin levels were higher AP administration than those at the 30th, 60th, and 120th minutes ($P < .001$). A significant difference in K levels was found between BP administration and the 15th, 30th, and 120th minutes ($P < .001$; **Table 3**).

Table 1—Time-dependent changes in total antioxidant status (TAS) and total oxidant status (TOS) levels and paraoxonase (PON) and arylesterase (ARES) activities in Turkish hair goats anesthetized with propofol and sevoflurane.

Propofol sevoflurane

	BP	AP	15th minute	30th minute	60th minute	120th minute	*P* value
TAS (mmol/L)	1.13 ± 0.01	1.15 ± 0.04	1.11 ± 0.04	1.11 ± 0.03	1.04 ± 0.03	1.34 ± 0.29	.446
TOS (µmol/L)	6.56 ± 0.28	6.63 ± 0.4	6.26 ± 0.3	6.31 ± 0.49	5.73 ± 0.45	6.02 ± 0.27	.444
PON (U/L)	265.25 ± 7.21	267.23 ± 6.85	254.14 ± 31.95	253.63 ± 28.73	313.08 ± 5.2	284.77 ± 6.77	.247
ARES (U/L)	393.82 ± 3.57[a,b]	385.88 ± 5.18[b]	404.69 ± 4.14[a]	397.47 ± 4.58[a,b]	390.14 ± 2.27[a,b]	395.22 ± 3.2[a,b]	.048

AP = After propofol. BP = Before propofol.

[a,b]Superscripts in the same row indicate the differences between the groups.

Table 2—Time-dependent changes in the heart rate, respiratory rate, and body temperature of Turkish hair goats anesthetized with propofol and sevoflurane.

Propofol sevoflurane

	BP	AP	15th minute	30th minute	60th minute	120th minute	*P* value
Heart rate (min)	80.57 ± 7.88[a]	90.86 ± 4.76[a]	121.86 ± 8.22[b]	106.86 ± 6.49[c]	103.71 ± 3.97[c]	106.71 ± 2.62[c]	< .001
Respiratory rate (min)	21.14 ± 3.23	23 ± 3.58	27.14 ± 2.86	27.71 ± 2.98	23.71 ± 3.18	23.29 ± 2.55	.338
Body temperature (°C)	38.03 ± 0.2[b]	38.4 ± 0.2[b]	38.19 ± 0.24[b]	38.26 ± 0.21[b]	38.13 ± 0.17[b]	37.53 ± 0.18[c]	.034

[a–c]Superscripts in the same row indicate the differences between the groups.

Table 3—Time-dependent changes in some biochemical parameters of Turkish hair goats anesthetized with propofol and sevoflurane.

Propofol sevoflurane

	BP	AP	15th minute	30th minute	60th minute	120th minute	*P* value
GLU (mg/dL)	60.71 ± 7.08[a]	71.71 ± 7.34[b]	75.14 ± 6.98[b]	69.29 ± 6.46[a,b]	69.86 ± 5.95[a,b]	66.57 ± 5.83[a,b]	.007
AST (U/L)	72.14 ± 2.69	74 ± 3.75	75 ± 3.44	72.43 ± 2.87	72.14 ± 3.62	72.29 ± 3.7	.69
GGT (U/L)	49 ± 6.31	50.71 ± 8.35	52.57 ± 7.72	50.14 ± 7.73	51.86 ± 7.5	49.71 ± 7.33	.211
ALT (U/L)	11.71 ± 1.3	12.71 ± 1.27	13.43 ± 1.19	12.57 ± 1.54	12.86 ± 1.3	12.57 ± 1.84	.357
ALP (U/L)	143.29 ± 60.14	149.57 ± 66.9	131.43 ± 53.03	155.71 ± 65.6	127.71 ± 50.28	99 ± 47.69	.185
LDH (U/L)	178.43 ± 16.74	183.43 ± 15.18	182.57 ± 13.84	170.86 ± 14.26	182.14 ± 11.51	181.57 ± 13.41	.499
CK (U/L)	71.57 ± 10.75[a]	77.57 ± 11.07[a,b]	85 ± 10.68[a,b]	84.14 ± 9.6[a,b]	122.14 ± 27.66[a,b]	148.71 ± 33.84[a]	.039
AML (U/L)	16.57 ± 1.29	16.71 ± 1.38	17.29 ± 1.39	17.57 ± 1.31	17.29 ± 1.15	16.14 ± 1.14	.45
LİP (U/L)	34 ± 1	32.71 ± 1.02	32.86 ± 1.12	32.57 ± 0.75	33.43 ± 1.27	35 ± 1.54	.06
CRE (mg/dL)	0.62 ± 0.03	0.59 ± 0.02	0.62 ± 0.03	0.6 ± 0.02	0.63 ± 0.02	0.65 ± 0.02	.057
UREA (mg/dL)	43.76 ± 3.87[a,c]	43.4 ± 3.65[a,c]	40.99 ± 5[c]	43.8 ± 3.64[a,c]	46.41 ± 3.94[a,b]	51.26 ± 4.22[a]	.016
UA (mg/dL)	0.06 ± 0.02	0.08 ± 0.02	0.08 ± 0.02	0.07 ± 0.01	0.08 ± 0.02	0.05 ± 0.01	.473
CHOL (mg/dL)	47.71 ± 4.8	48.14 ± 3.94	48.57 ± 5.24	49.57 ± 4.74	47.71 ± 5.06	46.71 ± 6.21	.642
HDL (mg/dL)	32.81 ± 3.31	31.73 ± 2.99	33.9 ± 3.5	31.6 ± 3.52	32.3 ± 3.47	34.19 ± 4.66	.308
LDL (mg/dL)	13.29 ± 1.46	13.86 ± 1.12	14.14 ± 1.24	13.57 ± 1.29	14 ± 1.35	13.43 ± 1.57	.315
TP (gr/L)	67.94 ± 3.48	69.03 ± 2.76	70.81 ± 2.68	68.01 ± 3.28	69.39 ± 2.99	65.01 ± 2.37	.219
ALB (gr/L)	21.01 ± 1.07	22.01 ± 1.16	22.57 ± 1.21	21.69 ± 1.18	22.03 ± 0.96	21.79 ± 1.54	.648
TBIL (mg/dL)	0.28 ± 0.03[a,b]	0.31 ± 0.03[b]	0.28 ± 0.03[a,b]	0.25 ± 0.03[a]	0.22 ± 0.02[a]	0.2 ± 0.02[a]	< .001
DBIL (mg/dL)	0.06 ± 0.01	0.12 ± 0.03	0.06 ± 0.02	0.09 ± 0.03	0.05 ± 0.01	0.03 ± 0.01	.064
Mg (mg/dL)	1.86 ± 0.1	1.85 ± 0.11	1.84 ± 0.1	1.89 ± 0.12	1.84 ± 0.12	1.92 ± 0.12	.385
Cl (mmol/L)	110.29 ± 2.08	109.57 ± 2.05	109.57 ± 1.36	111 ± 1.46	109.14 ± 1.1	110.43 ± 1.29	.556
Ca (mg/dL)	7.92 ± 0.3	7.96 ± 0.28	7.99 ± 0.27	7.92 ± 0.28	8.07 ± 0.25	8.15 ± 0.25	.055
Iron (µg/dL)	76.43 ± 9.97	75.71 ± 10.79	76 ± 10.62	74.57 ± 10.13	73.29 ± 9.27	69 ± 8.62	.175
Na (mmol/L)	145 ± 0.95	143.57 ± 1.38	144.43 ± 1.13	144 ± 1.4	143.43 ± 0.9	147.14 ± 1.72	.065
P (mg/dL)	3.72 ± 0.65	3.14 ± 0.57	2.84 ± 0.23	3.02 ± 0.15	2.4 ± 0.17	2.81 ± 0.33	.22
K (mmol/L)	4.39 ± 0.1[b]	4.27 ± 0.13[a,b]	3.87 ± 0.15[a,c]	3.82 ± 0.14[c]	3.96 ± 0.16[a–b]	3.91 ± 0.15[a,c]	< .001

ALB = Albumin. AML = Amylase. AP = After propofol. BP = Before propofol. Ca = Calcium. CHOL = Cholesterol. CK = Creatine kinase. Cl = Chlorine. CRE = Creatinine. DBIL = Direct bilirubin. Fe = Iron. GLU = Glucose. HDL = High-density lipoprotein. K = Potassium. LDL = Low-density lipoprotein. LIP = Lipase. Mg = Magnesium. Na = Sodium. P = Phosphorus. TBIL = Total bilirubin. TP = Total protein. UA = Uric acid. UREA = Urea.

[a–c]Superscripts in the same row indicate the differences between the groups.

Discussion

Maintaining the balance between oxidant agents and antioxidant systems in the body is crucial for maintaining cell and tissue integrity. Total oxidant status refers to the total value of oxidative stress, whereas TAS indicates the total effect of all substances with antioxidant properties and can be used in clinical diagnosis.[16] Paraoxonase 1 and ARES are enzymes with similar active centers that are encoded by the same gene. Paraoxonase 1 has an antioxidant function due to its capacity to neutralize free radicals. Arylesterase activity can be considered an indicator of protein concentration, independent of changes in PON1 activity.[17]

A study[3] investigating the effects of propofol and isoflurane anesthesia on oxidative stress in Angora goats showed no statistically significant change in plasma malondialdehyde, vitamin A levels, erythrocyte superoxide dismutase, or catalase activities in blood samples collected before the administration of propofol, after the induction, and at the 15th, 30th, 60th, and 120th minutes as well as 24 hours after isoflorane administration. However, a statistically significant increase was observed in β-carotene levels following AP administration ($P < .05$). In this study, no statistical difference was found in serum TAS and TOS levels and PON activities BP administration, AP administration, and at the 15th, 30th, 60th, and 120th minutes after sevoflurane administration, whereas ARES activity significantly increased at the 15th minute after sevoflurane administration compared to that AP administration ($P < .05$). Propofol acts as a synthetic antioxidant by reacting with free radicals.[18] In this study, an increase in ARES activity is believed to be associated with the antioxidant effect of propofol.

Heart rate, respiratory rate, and body temperature are important parameters that need to be monitored before, during, and after anesthesia. A study[19] evaluating the effects of propofol and sevoflurane anesthesia on heart rate, respiratory rate, body temperature, and certain biochemical parameters in rabbits reported that the heart rate was 218.33 AP administration and increased to 226.67 at the 15th minute after sevoflurane administration, although this increase was not statistically significant. Hikasa et al[20] anesthetized 6 adult goats with halothane, isoflurane, and sevoflurane, maitaining the depth of surgical anesthesia for 3 hours. They reported that the heart rate increased significantly at the 30th minute after sevoflurane administration and continued to drop at the 60th, 90th, and 120th minutes. Similarly, in our study, the heart rate increased significantly at the 15th minute of sevoflurane administration and decreased thereafter. This may have been caused by a change in the position of the animals

under anesthesia. Adachi[21] reported that sevoflurane anesthesia caused reflex tachycardia when the patient's position was changed from supine to the head tilted upward. Kumandaş and Elma[22] compared the effects of isoflurane and sevoflurane on cardiovascular and respiratory systems during anesthesia in 7 healthy Angora goatsand measured the body temperature BP and AP induction and at the 5th, 10th, 15th, 30th, and 60th minutes of volatile anesthesia administration; they observed that body temperature dropped statistically significantly during anesthesia in both isoflurane and sevoflurane groups. This study also indicated that sevoflurane may cause a drop in body temperature, consistent with the finding of previous studies.[19,23] This drop is considered to be associated with a slower, linear drop in heat loss resulting from the metabolic heat production phase of heat loss.

Surgical stress can cause hyperglycemia due to neuroendocrine responses, insulin resistance, and increased hormone secretion. In Okwudili et al's[24] study, anesthesia was induced in West African dwarf goats using different combinations of propofol, xylazine, and ketamine, and blood glucose values increased significantly ($P < .05$) 30 minutes after inducing anesthesia in all groups compared to the values before induction and those of the control group. Similarly, in our study, glucose levels increased significantly AP administration compared to those BP administration ($P < .01$), which may have been caused by endocrine and metabolic changes that develop as a consequence of anesthesia and the accompanying surgical stress.

Creatine kinase is an enzyme found in skeletal and/or cardiac muscle cells, and skeletal or cardiac muscle damage may cause an increase in serum levels of CK. Hikasa et al[20] reported that changes in CK levels were not significant during anesthesia (at the 60th, 120th, and 180th minutes) in goats anesthetized with halothane, isoflurane, or sevoflurane; however, they tended to increase in all 3 anesthetic groups at the 60th minute after anesthesia. In this study, the mean CK level was 71.57 ± 10.75 U/L BP administration, whereas it increased to 148.71 ± 33.84 U/L at the 120th minute of sevoflurane administration ($P < .05$). Burtis and Ashwood[25] reported that CK levels may increase with IM injections, surgical manipulation, and as a result of certain drugs.

Serum urea levels can be significantly altered by protein catabolism, protein synthesis, or both.[26] Okwudili et al[24] observed decreases in BUN levels following propofol and xylazine (5 mg/kg propofol, IV, + 0.05 mg/kg xylazine, IV) administration. Hikasa et al[20] found that BUN levels in 6 adult goats anesthetized with halothane, isoflurane, or sevoflurane increased during anesthesia (at the 60th, 120th, and 180th minutes). Different studies[27,28] have reported an increase in urea levels in sevoflurane-treated rats and rabbits without any change in creatinine levels. Akbay et al[28] reported that changes in renal function in their sevoflurane group may be associated with decreased mean arterial pressure, toxic damage, or ventilator damage. In this study, urea levels dropped following propofol administration ($P > .05$), whereas urea levels increased from 40.99 ± 5 mg/dL at the 15th minute of sevoflurane administration to 46.41 ± 3.9 mg/dL at the 60th minute and to 51.26 ± 4.22 mg/dL at the 120th minute ($P > .05$). Moreover, no difference was found in creatinine levels, consistent with the findings of other studies.

It has been reported that TBIL levels do not change significantly in healthy goats after anesthesia with sevoflurane.[20] In this study, TBIL levels increased from 0.28 ± 0.03 mg/dL BP administration to 0.31 ± 0.03 mg/dL AP administration and significantly dropped at the 30th, 60th, and 120th minutes of sevoflurane administration ($P < .001$).

In a study[29] investigating the effect of propofol on acid-base balance and ionic composition of arterial and venous blood in clinically healthy goats, it was found that K levels were higher in the venous blood throughout the experiment. In this study, K levels were found to be statistically significantly lower ($P < .001$) at the 15th, 30th, and 120th minutes of sevoflurane administration compared to those BP administration.

In this study, we observed that propofol and sevoflurane used for anesthesia did not affect serum TAS, TOS, and PON activities, whereas ARES activity increased with sevoflurane administration in Turkish hair goats. In addition, changes occurred in physiologic parameters, including heart rate and body temperature, as well as biochemical parameters, including glucose, CK, urea, TBIL, and K levels, at different time intervals of propofol-sevoflurane administration. In conclusion, propofol-sevoflurane anesthesia did not have a negative effect on oxidant-antioxidant parameters, and it may be useful to evaluate the heart rate and body temperature, as well as the levels of glucose, CK, urea, TBIL, and K, in the anesthesia protocol.

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References

1. Alkan İ, Atasoy N, Taş A, et al. Anesthesia protocol in neonatal calves: anesthesia applications in surgical cases in calves. *Turk Klin Vet Sci Surg Spec Top.* 2019;5:24–28.
2. Topal A. *Veteriner Anestezi.* Nobel, Güneş Tıp Kitap Evi; 2005.
3. Pekcan Z, Çınar M, Gürkan M, Kumanda Ş. The effects of propofol and isoflurane anaesthesia on oxidative stress in Angora goats. *Atatürk Üniv Vet Sci J.* 2011;6:217–222.
4. Kamiloğlu NN, Kamiloğlu A, Beytut E. Changes in antioxidant system, lipid peroxidation, heart and respiratory rate and rectal temperature with ketamine and ketamine-xylazine anaestesia in Tuj rams. *Kafkas Univ Vet Fak Derg.* 2009;15:205–210.
5. Çamkerten İ, Şındak N, Özkurt G, et al. Effect of ketamine-xylazine anesthesia on some hematological and serum

biochemical values of Bozova Greyhounds. *Harran Üniv Vet Fak Derg.* 2013;2:27–31.
6. Duke T. A new intravenous anesthetic agent: propofol. *Can Vet J.* 1995;36(3):181.
7. Özaydın İ, Atalan G, Uzun M, et al. Assessment of anaesthetic properties and clinical, cardiovascular and respiratoric effects of medetomidine, propofol and ketamine combination in dogs. *Kafkas Üniv Vet Fak Derg.* 2001;7:71–76.
8. Murphy P, Bennett J, Myers D, Davies MJ, Jones JG. The effect of propofol anaesthesia on free radical-induced lipid peroxidation in rat liver microsomes. *Eur J Anaesthesiol.* 1993;10(4):261–266.
9. Bennett R, Fancy S, Walsh C, Brown AJ, Taylor PM. Comparison of sevoflurane and isoflurane in dogs anaesthetised for clinical surgical or diagnostic procedures. *J Small Anim Pract.* 2008;49(8):392–397. doi:10.1111/j.1748-5827.2008.00586.x
10. Mercan U. Importance of free radicals in toxicology. *Yüzüncü Yıl Üniv Vet Fak Derg.* 2004;15:91–96.
11. Riess ML, Eells JT, Kevin LG, Camara AKS, Henry MM, Stowe DF. Attenuation of mitochondrial respiration by sevoflurane in isolated cardiac mitochondria is mediated in part by reactive oxygen species. *Anesthesiology.* 2004;100(3):498–505. doi:10.1097/00000542-200403000-00007
12. Kumandas A, Çınar M, Pekcan M, Karslı B, Pekcan Z. The effects of propofol and sevoflurane anaesthesia on lipid peroxidation and the antioxidant system in Angora goats. *Int J Vet Anim Res.* 2019;2(1):16–19.
13. Erel O. A novel automated direct measurement method for total antioxidant capacity using a new generation, more stable ABTS radical cation. *Clin Biochem.* 2004;37(4):277–285. doi:10.1016/j.clinbiochem.2003.11.015
14. Erel O. A new automated colorimetric method for measuring total oxidant status. *Clin Biochem.* 2005;38(12):1103–1111. doi:10.1016/j.clinbiochem.2005.08.008
15. Irak K, Celik BA, Karakoc Z, et al. Oxidant/antioxidant status, PON1 and ARES activities, trace element levels, and histological alterations in sheep with cystic echinococcosis. *Iran J Parasitol.* 2018;13(3):448–456.
16. Gultekın M, Ural K, Pasa S, Balikci C, Asici GS. Oxidative status and lipid profile in mono-and co-infection with canine monocytic ehrlichiosis. *Medycyna Wet.* 2017;73(12):797–801. doi:10.21521/mw.5817
17. İnce Ü. *Preoperative Evaluation of Patients with Obstructive Sleep Apnea Syndrome: Effects of Anesthesia and Surgery on Inflammatory and Stress Parameters.* Akdeniz Üniversitesi Tıp Fakültesi; 2018.
18. Murphy P, Myers D, Davies M, Webster NR, Jones JG. The antioxidant potential of propofol (2,6-diisopropylphenol). *Br J Anaesth.* 1992;68(6):613–618. doi:10.1093/bja/68.6.613
19. Guzel O, Inal G, Cirakli ZT, Eraslan E, Aktas M. Effects of propofol and sevoflurane anesthesia on some physiological and biochemical parameters in rabbits. *Medycyna Wet.* 2006;62(12):1383–1386.
20. Hikasa Y, Hokushin S, Takase K, Ogasawara S. Cardiopulmonary, hematological, serum biochemical and behavioral effects of sevoflurane compared with isoflurane or halothane in spontaneously ventilating goats. *Small Rumin Res.* 2002;43(2):167–178. doi:10.1016/S0921-4488(02)00002-0
21. Adachi H. Sevoflurane anesthesia maintains reflex tachycardia on position change from supine recumbent to head-up tilt. *J Anesth.* 1996;10(2):129–132. doi:10.1007/BF02483349
22. Kumandaş A, Elma E. Comparison of sevoflurane and isoflurane effects on cardiovascular andrespiratory system during spontaneous ventilation in Angora goats. *Turk J Vet Anim Sci.* 2015;39(4):501–506. doi:10.3906/vet-1501-75
23. Iwata T, Inoue S, Kawaguchi M, et al. Comparison of the effects of sevoflurane and propofol on cooling and rewarming during deliberate mild hypothermia for neurosurgery. *Br J Anaesth.* 2003;90(1):32–38. doi:10.1093/bja/aeg016
24. Okwudili UC, Chinedu EA, Anayo OJ. Biochemical effects of xylazine, propofol, and ketamine in West African dwarf goats. *J Vet Med.* 2014;2014:758581.
25. Burtis C, Ashwood E. *Tietz Textbook of Clinical Chemistry.* Saunders Co; 1997.
26. Fox R. *Clinical Chemical Studies Applicable to Laboratory Animal Species and Their Interpretation: the Rabbit.* Pergamon Press; 1989:41–47.
27. Sezen G, Demiraran Y, Kocaman AB, Aksoy KA. Subanesthetic concentration of sevoflurane and desflurane were inhalated in rats; liver and kidney toxicities and behavioural effects. *Turk J Anaesthesiol Reanim.* 2007;35(2):83.
28. Akbay B, Ayhan B, Yilbaş S, et al. Evaluate of metabolic and biochemical effects of infusion of propofol in rabbits who undergoing mechanical ventilation. *Anestezi Derg.* 2011;19(3):162–176.
29. Sobiech P, Lew M, Lew S, Stopyra A. The effect of propofol on acid-base balance and ionic composition of venous and arterial blood in goats. *Pol J Vet Sci.* 2005;8(4):295–300.