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A comparative clinical study on use of isoflurane and sevoflurane anaesthesia following dexmedetomidine premedication and propofol induction in dogs

Akarsh, R.[1], Jagan Mohan Reddy[2†], Madhav Rao, T.[3], Chandra Shekhar, E.L.[4] and Rajendranath, N.[5]

P.V. Narsimha Rao Telangana Veterinary University, Rajendranagar, Hyderabad-500030 (Telangana)

[1]MVSc Scholar, [4]Professor and University Head, [5]Professor and Head, Department of Veterinary Surgery & Radiology, College of Veterinary Sciences, Rajendranagar, [2]Assistant Prof. and Head, [3]Associate Dean, College of Veterinary Sciences, Korutla

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The objective of the study was to compare isoflurane and sevoflurane inhalation anaesthesia in dogs premedicated with dexmedetomidine and anaeshesia induced with propofol. The study was conducted in 12 dogs, divided in two equal groups, I and II, undergoing various orthopaedic procedures. All the animals of two groups were premedicated with dexmedetomidine (20 µg/kg body wt, i.v.) 15 min before induction of anaesthesia with propofol (3 mg/kg body wt, i.v.). Anaesthesia was maginated by isoflurance in group I and Sevoflurans in group II. There was a significant decrease in rectal temperature and respiratory rate and no change in heart rate during anaesthesia in both groups. Haemoglobin, PCV and TEC values did not change significantly, while TLC values showed a significant decrease in both groups. Serum creatinine and BUN levels were significantly decreased in both groups; however, ALT and AST levels were within the normal physiological range. No arrhythmia was recorded in both groups. In group I, the mean duration (in min) of extubation time, sitting time and complete recovery of animals were 8.73±0.34, 16.13±0.23 and 21.97±0.43, min, respectively. In group II, the values were 5.10±0.24, 9.37±0.23 and 15.47±0.17 min, respectively. Regaining of the reflexes in group II was faster than group I. Both isoflurane and sevoflurane provided excellent anaesthesia and recovery characteristics; however, sevoflurane had faster recovery compared to isoflurane.

Key words: *Clinical and pathological changes, Dexmedetomidine, Dog, General anaesthesia, Isoflurane, Orthopaedic surgery, Propofol, Sevoflurane*

Dexmedetomidine, a synthetic alpha-2 adrenoreceptor agonist is a popular drug in balanced anaesthesia due to its sedative, pain relieving and muscle relaxation properties. Dexmedetomidine shows greater affinity towards alpha-2 adrenergic receptors compared to similar agents such as xylazine and medetomidine, and has acquired interest in veterinary anaesthesiology over medetomidine (Kuusela *et al.*, 2000).

In small animals, propofol is injected as a single bolus for induction of anaesthesia to allow endotracheal intubation and initiation of inhalant anaesthesia.

In veterinary practice, isoflurane and sevoflurane are the most commonly used inhalant anaesthetics. Isoflurane is a structural isomer of enflurane, introduced into veterinary medicine in early eighties. The minimum alveolar concentration (MAC) for isoflurane is 1.15% and blood/gas partition coefficient is 1.4 (Eger, 1984). It is a halogenated compound which is non-explosive, highly stable and potent inhalational anaesthetic having less tissue toxicity (Eger *et al.*, 2003). Sevoflurane is an ether inhalation anaesthetic, which has low blood/gas solubility compared to isoflurane and other inhalant anaesthetics. The low solubility and absence of pungency facilitates rapid mask induction and recovery from anaesthesia (Patel and Goa, 1996). Both isoflurane and sevoflurane are having greater potency, less blood gas solubility and faster and smooth recovery periods in dogs (Basha and Ranganath, 2012). However, both these agents produce dose dependent depression of cardiovascular and respiratory systems, hence patient should be carefully monitored.

In the present study, efficacy of isoflurane and sevoflurane as maintenance agents was evaluated in dogs premedicated with dexmedetomidine and anaesthetized with proprfol and undergoing various orthopaedic surgical procedures.

Materials and Methods

The present study was conducted on 12 dogs presented for treatment of different long bone fractures. The dogs included in the study were of different breeds and age group (3-8 yr) and body weight (6-30 kg). All the dogs were thoroughly screened for their health status, by evaluating all the vital parameters, physical examination, clinical investigation and haematology. Physiological parameters like heart rate, respiratory rate, rectal temperature, colour of visible mucous membrane and capillary refill time were recorded preoperatively. Blood samples were collected for estimations of haematological parameters like Haemoglobin (Hb), Packed Cell Volume (PCV), Total Leukocyte Count

†Corresponding author; E-mail: drvrtjagan@gmail.com

(TLC), Total Erythrocyte Count (TEC) and Differential Leukocyte Count (DLC). The biochemical profile like Blood Urea Nitrogen (BUN), Creatinine, Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST) were estimated one day prior to surgery.

The dogs used in the study were randomly divided into 2 groups of six each, namely group I and group II. Preoperatively, water and food were withheld for 6 hr and 12 hr, respectively. Cephalic vein was catheterized with a suitable size of IV canula and normal saline (10 mL/kg body wt) was administered to each dog.

The animals of both groups were premedicated with dexmedetomidine (20 µg/kg body wt, i.v.) 15 min prior to induction of anaesthesia with propofol (3 mg/kg body wt, i.v.). Total amount of drug required for proper induction and time of administration of drug were noted. After connecting the machine to ET tube, oxygen flow rate was set according to the animal's tidal volume (50-80 ml/kg/min).

Anaesthesia was maintained with 2-4% isoflurane in 100% oxygen (initial vaporizer setting at 3-5%) in animals of group I, and 2-4% sevoflurane in 100% oxygen (initial vaporizer setting at 4-6%) in group II animals. The vaporizer settings were later increased or decreased as per the situation to maintain the surgical anaesthesia.

iM8 VET veterinary monitor (EDAN-USA, San Diego, CA 92117) was used for monitoring and recording of the vital signs of the animals during anaesthesia. The parameters were recorded at every 15 min interval i.e., immediately after induction, and at 0, 15, 30, 45 min of maintenance and during recovery. During the recovery from anaesthesia, different parameters such as extubation time, sitting time and complete recovery time (min) were recorded. The quality of recovery was evaluated in a score criteria of 0-4 (Pottie *et al.,* 2008) as: 0- unacceptable recovery; 1- vocalization, restless, paddling, urination or defecation; 2- whining, agitated, some paddling or trembling; 3- gradual, slow, moderate restlessness; and 4- gradual, smooth, quiet, rapid, comfortable.

The data were analyzed using standard tests-students 't' test and ANOVA. The significance was considered at $P<0.05$.

Results and Discussion

In the present study, dexmedetomidine was administered @ 20 µg/kg body wt, i.v. for premedication, as also reported by Kuusela *et al.* (2001). Dexmedetomidine is known to reduce the minimum alveolar concentration (MAC) of isoflurane for maintenance of anaesthesia (Bloor *et al.,* 1992). Santosh *et al.* (2013) observed that increasing the dexmedetomidine dose had no effect on the depth or duration of anaesthesia. Dexmedetomidine is reported to considerably reduce the cardiac output and heart rate, eventually increasing the arterial blood pressure (Kutter *et al.,* 2006; Pypendop *et al.,* 2017). Administration of dexmedetomidine as a premedicant in dogs caused a dose-dependent decrease in respiratory rate (Kuusela *et al.,* 2001).

Propofol @ 3 mg/kg body wt (i.v.) provided a rapid, smooth and excitement free induction as also reported by Heldman *et al.* (1999). Robinson and Borer Weir (2013) reported rapid central nervous system depression @ 4 mg/kg body wt in dogs facilitating anaesthetic induction within 20-30 seconds after commencement of intravenous administration. In the present study, relatively lower dose of propofol was reuired for induction of anaesthesia in dogs when compared to previous reports. This might be because of the use of dexmedetomidine as premedication agent, which might have helped to calm the patients by producing sedation and analgesia (Thurmon *et al.,* 1996; Salunke *et al.,* 2002). In the present study, a slight change in haematobiochemical parameters was observed after propofol induction. Khurana *et al.* (2014) and Anandmay *et al.* (2016) have reported reduction in PCV, Hb, AST, and TEC levels on administration of propofol. In the present study, after induction of anaesthesia with propofol, heart rate increased and there was slight decrease in arterial blood pressure, as also reported by Bufalari *et al.* (1996) and Suarez *et al.* (2012). In the present study, all the animals in both groups showed significant respiratory depression and one animal in each group showed apnoea, as also reported by Keates and Whittem (2012). Lerche *et al.* (2000) found respiratory depression and post induction apnoea with propofol and further stated that cyanosis was less commonly observed as a side effect. Cyanosis was not found in the present study.

Rectal temperature (RT) was significantly lower at 15 min, 30 min and at 45 min in both groups after propofol induction and during maintenance. The drop in RT could be due to decreased activity of the reticular activating system and depression of the thermoregulatory centre, as well as decreased metabolic rate and skeletal muscle activity during anaesthesia induction and maintenance. In both groups, the change in mean RT followed a similar pattern. However, during maintenance, the sevoflurane group had non-significantly lower temperature values than the isoflurane group. According to Mutoh *et al.* (1997), this could be due to a decrease in systemic vascular resistance and an increase in vasodilation caused by sevoflurane.

A significant decrease in the mean respiratory rate (RR) was observed in groups I and II after induction and during maintenance for up to 45 min (Table 1). Later, during the recovery, it increased close to the base value. Galloway *et al.* (2004) discovered that isoflurane and sevoflurane produced similar dose-related pulmonary depression; however, due to

its higher anaesthetic index, sevoflurane caused less respiratory depression at higher equipotent anaesthetic doses. On the contrary, during 5-30 min of maintenance, sevoflurane produced slightly more respiratory depression than isoflurane. This could be due to sevoflurane's lower blood:gas partition coefficient (0.68) and faster rise in alveolar concentration at 37°C than isoflurane (Kazama and Ikeda, 1988). There was dose dependent decrease in the RR, this could be attributed to the dose-dependent depressing effect of isoflurane and sevoflurane on higher respiratory centres (Galloway *et al.,* 2004).

There was progressive cardiovascular depression in both groups with higher doses of inhalation agents, hence dexmedetomidine was used in an attempt to reduce the requirement of inhalation agents and thus resulted in less cardiovascular depression (Gutierrez-Blanco *et al.,* 2013). Pottie *et al*. (2008) compared the potency of isoflurane and sevoflurane and concluded that their depressant effect on cardiovascular functions such as cardiac output and stroke volume in dogs was less than that of halothane. The change in heart rate (HR) followed a similar pattern in both groups, with no significant difference observed within or between groups at different time intervals compared to the baseline values, and a non-significant increase was observed during recovery, which was consistent with Jadon *et al.* (2008). The increase in HR at the end of isoflurane and sevoflurane inhalation anaesthesia maintenance could be attributed to increased baroreceptor reflex activity caused by decreased arterial blood pressure (Mutoh *et al.,* 1997).

In group I, arterial blood pressure (SAP, DAP, and MAP) increased significantly from induction up to 30 min of anaesthetic maintenance, then declined at 45 min interval and recovery, but remained significantly above baseline level. In group II, arterial blood pressure values decreased non-significantly from induction to 45 min of anaesthetic maintenance and then increased during recovery, but the values remained below the baseline. There was no significant difference in arterial blood pressure values between the groups. Sevoflurane reduced arterial blood pressure more than isoflurane (Table 2).

Sevoflurane was known to reduce arterial pressure, cardiac output, and peripheral vascular resistance in dogs in a dose-dependent manner. Sevoflurane's haemodynamic properties were nearly identical to those of isoflurane, with the exception that sevoflurane had a greater vasodilatory action than isoflurane (Bernard *et al.,* 1990). Isoflurane group showed significant increase in arterial blood pressure values from induction to 30 min of maintenance of anaesthesia and later it decreased until recovery. On the contrary, Mutoh *et al.* (1997) found a gradual decrease in systemic arterial blood pressure based on stage of anaesthesia. The isoflurane concentration required to achieve surgical plane anaesthesia was frequently associated with myocardial depression and significant vasodilation, which could result in hypotension and a significant decrease in arterial

Table 1: Changes in physiological parameters (mean±SE) in group I and group II (n=6)

Parameters	Group	Base	Ind	T5	T15	T30	T45	Recy
Rectal	I	102.13±0.11	101.78±0.08*	101.31±0.07*	100.58±0.17*	99.91±0.22*	99.21±0.15 a*	100.01±0.11*
Temperaturte (°F)	II	102.23±0.13	101.91±0.10*	101.16±0.04 *	100.15±0.14*	98.38±0.16*	97.71±0.16 a*	99.76±0.08*
Respiratory Rate	I	32.16±1.35	20.16±0.70*	16.16±1.19*	9.16±0.47*	8.16±0.47*	9.00±0.36*	25.16±0.13
(breaths/min)	II	31.66±1.33	19.83±0.40*	12.33±0.66*	9.66±0.55*	7.83±0.47*	9.50±0.42*	28.66±0.88
Heart Rate	I	112.0±8.56	119.33±6.96	117.50±2.30	112.50±6.81	111.67±5.25	114.0±4.23	120.33±5.74
(beats/min)	II	109.0±8.58	115.33±5.51	118.83±2.90	113.00±4.95	109.67±3.81	111.83±2.93	127.17±3.54

Where **Base:** 10 min, prior to pre-medication, **Ind:** Immediately after induction, **T5:** 5 min after maintenance, **T15:** 15 min after maintenance, **T30:** 30 min after maintenance, **T45:** 45 min after maintenance, **Recy**: Immediately after recovery;
*Represents significant change within a group compared to the base value; a Represents significant change between group I and group II

Table 2: Changes in cardiovascular parameters (mean±SE) in group I and group II (n=6).

Parameters	Group	Ind	T5	T15	T30	T45	Recy
SAP(mm Hg)	I	107.83±2.08	118.16±1.92*	163.33±2.07*	154.50±2.23*	132.16±1.30	130.16±1.24
	II	122.0±1.06	123.50±1.05	122.33±1.42	110.50±1.52	106.33±1.87	117.33±0.88
DAP(mm Hg)	I	62.83±0.87	68.33±1.11	96.66±1.66	101.16±0.70*	91.33±1.22	85.66±2.06*
	II	73.83±1.85	68.83±1.01	67.66±0.91	64.50±0.95	62.00±0.57	72.33±0.80
MAP(mm Hg)	I	76.33±1.97	82.16±1.10*	111.50±0.76*	119.50±0.99*	106.0±1.6	101.33±1.45*
	II	87.16±1.10	84.33±1.11	85.66±1.45	79.50±1.92	75.33±1.99	84.83±1.44
SpO_2 (%)	I	97.67±0.42	98.0±0.45	98.33±0.21	97.83±0.31	98.17±0.17	96.50±0.49
	II	97.33±0.33	98.17±0.31	98.33±0.21	98.33±0.21	98.67±0.21	96.83±0.40

Where **Base:** 10 min prior to pre-medication, **Ind:** Immediately after induction, **T5:** 5 min after maintenance, **T15:** 15 min after maintenance, **T30:** 30 min after maintenance, **T45:** 45 min after maintenance, **Recy**: Immediately after recovery.
* Represents the significant change within a group compared to the induction value.

blood pressure. The change in oxygen saturation followed a similar pattern in both groups, the oxygen saturation per cent in blood showed no significant changes from induction to recovery. At different time intervals, no significant difference was observed between the groups. These findings agreed with those of Pypendop *et al.* (2019).

During anaesthesia maintenance, the QT interval was prolonged in both groups while the QRS interval remained unchanged. Jadon *et al.* (2008) recorded increased QT intervals in puppies anaesthetized with isoflurane and sevoflurane. An increase in the amplitude of the T wave was observed (Conti-patara *et al.*, 2009). There were no arrhythmias observed with both drugs, as also reported by Andreza *et al.* (2009).

All the haematological variables remained within normal limits and there was no significant change observed during maintenance of anaesthesia in both groups (Table 3), as also observed by Jadon *et al.* (2008) and Suthar *et al.* (2018).

In both groups, there was a significant increase in neutrophils percentage from baseline to recovery, and a significant decrease in lymphocytes percentage from 5 min of maintenance of anaesthesia up to recovery. The percentages of lymphocytes and neutrophils did not differ significantly between groups, also the changes in neutrophils and lymphocytes remained within the normal physiological range. Tomihari *et al.* (2015) have also reported a significant decrease in lymphocyte count after isoflurane and sevoflurane anaesthesia. The percentage of eosinophils, basophils, and monocytes changed insignificantly. According to Jadon *et al.* (2008), sevoflurane anaesthesia caused minimal haematological changes compared to isoflurane anaesthesia.

Serum creatinine and BUN levels in groups I and II were significantly lower from premedication to recovery (Table 5), with no significant difference between groups, as also reported in earlier studies (Jadon *et al.*, 2008. Fluctuations in serum creatinine levels during isoflurane and propofol anaesthesia were probably caused by the temporary inhibition of renal blood flow during anaesthesia, which resulted in decreased glomerular filtration rate (Robertson *et al.*, 1992).

ALT and AST values fluctuated within normal physiological range at different intervals in animals of both groups, and there was no significant difference between the groups. Basha and Ranganath (2012) also found no significant change in ALT and AST values during isoflurane and sevoflurane anaesthesia. The observations of the present study indicated that isoflurane and sevoflurane were nontoxic to liver and kidney.

The beginning of recovery phase of anaesthesia was marked from the point of cessation of anaesthesia. In group I animals, the mean duration (in min) of

Table 3: Changes in haematological parameters (mean±SE) in group I and group II (n=6)

Parameters	Group	Base	Ind	T5	T15	T30	T45	Recy
Hb (g/dL)	I	12.39±0.24	12.27±0.24*	12.21±0.23*	12.02±0.23*	11.80±0.22*	11.65±0.22*	11.55±0.21*
	II	12.50±0.24	12.40±0.23*	12.31±0.24*	12.18±0.23*	11.95±0.22*	11.79±0.22*	11.66±0.22*
PCV (%)	I	38.22±0.89	37.69±0.93*	37.17±0.94*	36.67±0.83*	36.07±0.78*	35.53±0.76*	35.13±0.79*
	II	38.99±1.08	38.73±0.95*	38.22±0.91*	37.62±0.86*	36.95±0.80*	36.67±0.78*	36.23±0.79*
TEC(x 10^6/μL)	I	6.42±0.16	6.31±0.16*	6.22±0.17*	6.13±0.16*	6.02±0.15*	5.95±0.14*	5.87±0.14*
	II	6.52±0.21	6.47±0.20*	6.37±0.20*	6.28±0.19*	6.15±0.17*	6.09±0.16*	6.03±0.15*
TLC(x 10^3/μL)	I	12.31±0.37	12.05±0.37*	11.83±0.37*	11.60±0.35*	11.41±0.34*	11.23±0.34*	11.46±0.36*
	II	11.96±0.35	11.74±0.36*	11.62±0.36*	11.52±0.37*	11.35±0.38*	11.21±0.35*	11.42±0.35*

Where **Base:** 10 min prior to pre-medication, **Ind:** Immediately after induction, **T5:** 5 min after maintenance, **T20:** 20 min after maintenance, **35:** 35 min after maintenance, **T50:** 50 min after maintenance, **Recy**: Immediately after recovery.
* Represents significant change within a group compared to the base value.

Table 4: Changes in differential leucocyte count (mean±SE) in group I and group II (n=6)

Parameters	Group	Base	Ind	T5	T15	T30	T45	Recy
Neutrophils (%)	I	67.83±0.79	68.83±0.70*	69.15±0.78*	70.83±0.95*	71.72±0.84*	72.50±0.67*	73.50±0.67*
	II	69.33±0.88	70.00±0.68*	70.84±0.77*	71.33±0.76*	72.08±0.67*	72.83±0.60*	73.67±0.67*
Lymphocytes (%)	I	25.33±0.33	25.17±0.75	24.27±0.79*	22.81±0.93*	22.00±1.06*	22.17±0.70*	20.17±0.65*
	II	24.00±0.86	23.50±0.50	22.73±0.82*	22.59±0.61*	22.50±0.72*	21.17±0.65*	20.33±0.84*
Monocytes (%)	I	3.82±0.31	3.56±0.24	3.38±0.23	3.52±0.38	3.57±0.22	3.27±0.26	3.35±0.28
	II	3.64±0.25	3.53±0.27	4.02±0.23	3.32±0.23	3.25±0.26	3.77±0.27	3.57±0.26
Eosinophils (%)	I	2.55±0.25	2.57±0.23	2.81±0.23	2.55±0.26	2.54±0.27	2.01±0.09	2.79±0.24
	II	2.74±0.26	3.04±0.06	2.38±0.27	2.86±0.33	2.25±0.28	2.58±0.23	2.58±0.26

Where **Base:** 10 min. prior to pre-medication, **Ind:** Immediately after induction, **T5:** 5 min after maintenance, **T15:** 15 min after maintenance, **T30:** 30 min after maintenance, **T45:** 45 min after maintenance, **Recy**: Immediately after recovery.
* Represents significant change within a group compared to the base value.

Table 5: Changes in serum biochemical parameters (mean±SE) in group I and group II (n=6)

Parameters	Group	Base	Ind	T5	T15	T30	T45	Recy
Creatinine	I	0.92±0.05	0.83±0.06*	0.80±0.06*	0.78±0.06*	0.74±0.06*	0.71±0.05*	0.74±0.05*
	II	0.90±0.03	0.82±0.03*	0.81±0.03*	0.79±0.03*	0.77±0.03*	0.76±0.04*	0.78±0.04*
BUN	I	18.30±1.19	17.72±1.14*	17.45±1.14*	17.58±1.14	17.70±1.14*	17.86±1.15*	18.10±1.14*
	II	18.25±0.67	17.36±0.66*	17.40±0.63*	17.51±0.64	17.58±0.63*	17.68±0.63*	17.95±0.62*
ALT	I	36.90±1.17	36.10±1.16*	35.61±1.17*	34.83±1.18*	35.71±1.19*	33.51±1.16*	35.51±1.01*
	II	37.08±1.26	36.20±1.20*	35.70±1.21*	34.81±1.18*	35.66±1.15*	33.48±1.16*	35.96±0.98*
AST	I	29.37±0.94	28.41±0.97*	29.38±0.96	29.41±0.84	29.60±0.81*	29.98±0.81*	32.93±0.80*
	II	29.61±1.01	28.40±0.98*	29.28±0.99	29.73±0.96	30.0±0.95*	30.31±0.92*	32.85±0.80*

Where **Base:** 10 min prior to pre-medication, **Ind:** Immediately after induction, **T5:** 5 min after maintenance, **T20:** 20 min after maintenance, **T 35:** 35 min after maintenance, **T50:** 50 min after maintenance, **Recy**: Immediately after recovery.
*Represents significant change within a group compared to the base value

extubation time, sitting time and complete recovery of animal were 8.73±0.34, 16.13±0.23 and 21.97±0.43 min, respectively. Reflexes returned quickly in group I animals, recovery was smooth, and the animals stood up with minimal effort. The number of attempts by animals to stand upright was small and appeared uncomplicated. This could be attributed primarily to the isoflurane's low solubility, which allowed for rapid elimination from the body.

The mean durations of extubation, sitting time, and complete recovery in group II animals were 5.10±0.24, 9.37±0.23 and 15.47±0.17 min, respectively. The recovery of reflexes in group II was faster than in group I. This could be because sevoflurane is less soluble than isoflurane (Steffey and Mama, 2007). In dogs, sevoflurane had a blood gas partition coefficient of 0.68 and a MAC of 2.36.

In present study, the average duration of anaesthesia required for completion of various orthopaedic procedures was 83.33±7.71 and 75.83±7.57 min in group I and group II animals, respectively. The mean vaporizer setting per cent for group I and group II were 3.39±0.02 and 3.83±0.04, respectively. The mean quantity of isoflurane consumed per animal was 13.85 mL, and the mean quantity of sevoflurane consumed per animal was 14.60 mL.

The recovery phase in animals of both groups was a relatively smooth transition from anaesthesia to full consciousness. There were no signs of struggling, tremors, or other abnormalities. One case in group I demonstrated slight pedaling, and in another case vocalisation was observed. Lip licking and licking of the floor, legs, and bandage were observed on a less frequent basis. In comparison, recovery was much smoother and faster in group II animals than in group I. Love *et al.* (2007) also reported that dogs given sevoflurane recovered significantly better than dogs given isoflurane.

It was concluded that both isoflurane and sevoflurane provided excellent anaesthesia and recovery characteristics; however, in comparison, sevoflurane provided faster recovery from anaesthesia than isoflurane. In terms of cost per surgery, isoflurane was 5-6 times less expensive than sevoflurane based on the quantity of inhalant anaesthetic consumed.

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