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Comparison of effect of inhaled anaesthetic (Sevoflurane) versus intravenous (Propofol) anaesthetic on core and peripheral body temperature during general anaesthesia: A randomised control study

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ABSTRACT

Background: The fall in core body temperature and peripheral body temperature following the administration of anaesthetic agent has been studied and demonstrated so far, along with the comparison of the same parameters following induction with propofol in contrast to that with sevoflurane. But no study so far, has compared the effects of TCI-propofol based anaesthesia with that of sevoflurane based anaesthesia for induction and maintenance, on core and peripheral body temperature and the gradient of temperature between the agents. The studies conducted so far, has shown more fall in core and peripheral body temperature from their respective baseline values when propofol was used for induction of anaesthesia in comparison to the use of sevoflurane.

Materials and Methods: A total of 60 adults were randomized into two groups of 30 each; Group 1 were induced with TCI- propofol at 8mcg/ml plasma concentration and maintained with TCI-propofol at 2-3mcg/ml plasma concentration and 66% nitrous oxide and 33% oxygen gas mixture. Group 2 were induced with intravenous thiopentone at 3-5mg/kg body weight and maintained with 1-1.5MAC sevoflurane, 66% nitrous oxide and 33% oxygen gas mixture. Core body temperature was measured inserting the temperature probe into nasopharynx and peripheral temperature was measured with the temperature probe inserted onto the ear eminence of either hand.

Results: We observed that both anaesthetic agents have caused similar fall in core temperature. However, peripheral temperature fell more with sevoflurane compared to TCI-propofol. In parallel with these observations, the increase in temperature gradient was higher in magnitude for sevoflurane based anaesthesia.

Conclusion: The core temperature was comparable between the groups from their respective baseline values. But, the fall in peripheral temperature was more in sevoflurane group, so was the temperature gradient. Thus, TCI-propofol when used in appropriate plasma concentration for induction and maintenance of anaesthesia causes less hypothermia in contrast to sevoflurane.

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1. Introduction

General anaesthesia inhibits the behavioural thermoregulatory compensations, leaving only autonomic

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compensation by dose dependent fashion.¹ It impairs the vasoconstriction threshold about three times as much as the sweating threshold. General anaesthetics linearly increase the warm-response thresholds.^{2,3}

In humans, maintenance of normal body temperature is essential for homeostasis. Hypothermia after the induction of anaesthesia is attributed to the temperature redistribution from core to the periphery. There is linear fall in thresholds for vasoconstriction and shivering with the use of propofol whereas, volatile anaesthetics decrease cold response thresholds in non-linear fashion.⁴

During general anaesthesia hypothermia is the most common peri-operative finding. Normally, heat loss and heat production form an equilibrium allowing a thermal steady state, which keeps core temperature constant. Temperatures of the peripheral tissues are lower than core temperature with a difference of 2°C to 4°C. This gradient is maintained by tonic thermoregulatory vasoconstriction resulting in an uneven distribution of heat. Thus, peripheral tissues is considered as a thermal buffer and hence redistribution of body heat results and heat distributes from the core compartment to the peripheral tissues under equilibration. During the first hour of anaesthesia this redistribution accounts for about 80% of the core temperature decline, but there is little net heat loss overall. Core temperature drops by about 1°C to 1.5°C, while peripheral tissue temperatures gain up to 2°C. After completion of the redistribution, core temperature usually decreases at a slower rate.⁵ This decrease is nearly linear and results from heat loss exceeding metabolic heat production.

Propofol causes profound peripheral vasodilation in contrast to sevoflurane. Vasodilation in the peripheral compartment of the body facilitate core to peripheral heat redistribution. Once heat is lost from the core, it cannot be recovered from the periphery because flow of heat up a temperature gradient would violate the second law of thermodynamics.⁶⁻¹⁰

These data thus suggest that periods of vasodilation during anaesthetic induction and maintenance may have substantial and prolonged effects on body temperature intra-operatively. Since the mechanism of actions of propofol and sevoflurane vary greatly with respect to thermoregulatory actions, different outcomes on core and peripheral temperatures was expected. In this study we hypothesise that both anaesthetic agents produce equal effects on temperature on individuals.

The aim of this study is to compare the effects of sevoflurane and propofol (TCI-TIVA) on core and peripheral body temperature, when used at clinical anaesthetic concentrations and to analyse which of the agents caused more hypothermia.

2. Materials and Methods

2.1. Source of data

Patients aged between 18 to 60 years who are hospitalised at A.J institute of medical sciences and hospital research centre, Mangaluru, atleast a day prior to the day of surgery and who are expected to undergo various elective procedures of approximately two hour duration or more, were considered for the study.

2.2. Inclusion criteria

Patients who will be undergoing elective surgeries of expected duration of approximately 2 hours or more, belonging to American Society of Anaesthesiologists (ASA) grade 1-2, of either sex, between the age group of 18 to 60 years, with the Body Mass Index (BMI) between 18 to 25 under general anaesthesia.

2.3. Exclusion criteria

Patient refusal, patients belonging to ASA grade 3 or above, drug or alcohol abuse, allergy to Propofol, pregnant and lactating women, patients who are on sedatives, any psychiatric medications, patients with history of

1. Cerebral abnormalities (history of brain trauma, seizures)
2. Renal disorders, Renal function Tests above normal limits
3. Hepato-biliary -pancreatic disorders, Liver function Tests above normal limits
4. Cardiac diseases

2.4. Study design

Study was carried out for a period of twelve months in various patients who belonged to inclusion criteria of the study. This study was an observational, randomised control parallel group study. Subjects were assigned in two groups randomly based on random number allocation generated by random number generator applications.

1. Group 1 - Received intravenous agent, propofol using target controlled infusion device.
2. Group 2 - Received inhalational sevoflurane using vaporizer.

2.5. Sample size

Referring to a study on “Less Core Hypothermia when Anaesthesia is Induced with Inhaled Sevoflurane Than with Intravenous Propofol” conducted by Takheido Ikheda and colleagues¹¹ to observe a mean difference of 0.6 between the groups with standard deviation for each group being 0.5 and 0.8 respectively [α error of 0.05 and β error of 0.20, (power 80%)], the minimum sample size required was 27 for each

group. Thus, a total of 54 was considered. Assuming a drop out rate of 10% in each group, we included 60 subjects for our study.

3. Method of collection of data

This study was registered in institutional ethics committee. Prior to the day of surgery, pre-anaesthetic assessment, informed consent was obtained and demographic details and laboratory investigations were noted. Patients with ASA grades 1 and 2 were included in our study. A written informed consent was taken from all the included patients. They were premedicated with tablet ranitidine 150mg, orally and were advised to be nil per oral for solids for atleast a duration of 8 hours. Sedatives except opioids were used as pre-medicants. On the day of the surgery, patients were shifted to the operating room, positioned supine on the table, connected to Non Invasive Blood Pressure(NIBP), five lead Electrocardiogram (ECG) and saturation probe(SpO₂). For the purpose of temperature monitoring, monitor from the Spacelabs Healthcare (Spacelabs Healthcare, Inc. 35301 SE center street Snoqualmie, WA 98065 U.S.A.) monitor was used. After cannulating, intravenous fluid prewarmed to 37°C was started. Subjects were administered intravenous fentanyl 1-2mcg/kg. A baseline oral temperature (T_{BL}) was recorded using digital thermometer for all individuals for either groups.

Group 1 received intravenous propofol using TCI pump device of propofol at 8mcg/ml, plasma concentration for induction and 3-5mcg/ml for maintenance of anaesthesia along with oxygen 33% and nitrous oxide 66%. Group 2 received thiopentone at 5-7mg/kg body weight for induction and was maintained on inhalational sevoflurane at 1-1.5Iso-MAC concentrations (Datum vaporizer, MEDITEC England, Abbot ltd) at end tidal values for maintenance along with 33% oxygen and nitrous oxide 66%(11).

All subjects were be pre-oxygenated for 3 minutes with a pre-checked anaesthetic machine before induction. Tracheal intubation was facilitated using succinylcholine at the dose of 1.5mg/kg and ventilation was instituted to achieve normocarbida. Intermediate muscle relaxants were used to achieve desired neuromuscular blockade. Neuromuscular blockers used included rocuronium, atracurium, cisatracurium or vecuronium at their ED95 doses. After induction of anaesthesia, two temperature probes were placed for monitoring temperature of the subjects. The first, the nasopharyngeal temperature probe (NP probe) was inserted through nasal cavity into the posterior wall of the nasopharynx, posterior to the soft palate. The second temperature probe was fixed at the thenar eminence of the hand opposite to the cannulated upper extremity. Ambient temperature of the operating room was kept at 21°C. Relative humidity of 60-70% was maintained in the operation room. All the patients were covered with

forced dry warm blanket and attached with forced dry air warmer which was maintained at 38 degree Celsius. The temperature were recorded immediately after induction, and was considered as T_1 . Temperatures were recorded every 15 minutes interval thereafter for upto two and a half hours duration. Any significant deviations with respect to hemodynamic parameters were noted. The duration of surgery, crystalloids and colloid transfusions, urine output and blood loss during the surgery were recorded. At the end of surgery, anaesthetics were discontinued. Neuromuscular blockade was reversed with 0.05mg/kg of neostigmine and 0.01mg/kg of glycopyrrolate. Extubation was done once extubation criteria were met. Patient was shifted to post operative intensive care unit. Immediate postoperative complications such as delayed awakening, shivering, opioid requirement for control of shivering was noted.

4. Results and Discussion

Our study demonstrates the effects of target controlled infusion of propofol (TCI-propofol) in comparison with sevoflurane based anaesthesia for changes in core and peripheral body temperature and gradient between the same scalars. We compared the trend of fall in core and peripheral body temperature in both groups and difference between the parameters i.e., temperature gradient (i.e., difference between and core and peripheral temperature) at each time intervals recorded at 15 minutes time gap.

In our study we have compared the change in core and peripheral temperature from pre-induction baseline value to all the readings at fifteen minutes time interval for a period of 180 minutes in both groups. Later the temperature gradient derived from the difference between the temperature parameters were calculated for each time interval in a group and compared with corresponding values in the other group. We observed that both anaesthetic agents have caused similar fall in core temperature; however, peripheral temperature fell more with sevoflurane compared to TCI-propofol. In parallel with these observations, the increase in temperature gradient was higher in magnitude for sevoflurane based anaesthesia.

Many studies done in the past have demonstrated the comparison of core temperature between the groups and maintenance of peripheral temperature following induction with sevoflurane and propofol. But the gradient between core and peripheral body temperature as a measure in target controlled infusion(TCI) of propofol anaesthesia throughout the surgery and sevoflurane based anaesthesia was not conducted.

In the study, “less core hypothermia when anaesthesia is induced with inhaled sevoflurane than with intravenous propofol”, by Ikheda et al.,¹¹ conducted in twenty individuals who underwent oral surgery demonstrated that, core temperature decreased significantly more after induction with propofol than with sevoflurane and showed

Table 1: Comparison of core temperature between the groups

Time	Propofol			Sevoflurane			P value
	Mean	SD	N	Mean	SD	N	
Baseline T1	36.1	0.521	30	36.1	0.584	30	>0.999999
T2	35.1	0.718	30	34.4	1.53	30	0.027034
T3	34.4	0.953	30	34.5	1.45	30	0.753390
T4	34.6	0.652	30	34.5	1.36	30	0.717806
T5	34.6	0.594	30	34.5	1.35	30	0.711721
T6	34.7	0.588	30	34.5	1.2	30	0.415715
T7	34.8	0.579	30	34.6	1.18	30	0.408027
T8	34.8	0.568	30	34.6	1.18	30	0.406318
T9	34.8	0.59	30	34.7	1.12	30	0.666856
T10	34.9	0.546	30	34.8	1.13	30	0.664143

The intergroup comparison of core temperature between the groups in terms of mean and standard deviation are as shown above. Multiple t test is used to analyse the data and p value calculated. All the p values are not statistically significant.

Interpretation: Core temperature between the groups have no significant difference. The fall in core temperature between the groups is comparable.

Table 2: Comparison of peripheral temperature between two groups

Time	Propofol			Sevoflurane			P value
	Mean	SD	N	Mean	SD	N	
Baseline T1	35.5	0.602	30	35.7	0.655	30	0.223158
T2	33.6	0.954	30	32.2	2.42	30	0.004604
T3	33.6	0.823	30	32.7	1.71	30	0.011881
T4	33.7	0.71	30	32.9	1.64	30	0.017242
T5	33.9	0.8	30	32.9	1.66	30	0.004297
T6	34	0.755	30	33	1.61	30	0.003161
T7	34.1	0.753	30	33.1	1.5	30	0.001848
T8	34.2	0.718	30	33.2	1.52	30	0.001877
T9	34.3	0.688	30	33.2	1.48	30	0.000494
T10	34.4	0.684	30	33.2	1.52	30	0.000219

The intergroup comparison of peripheral temperature between the groups in terms of mean and standard deviation are as shown above. Multiple t test is used to analyse the data and p value calculated. All the p values are statistically significant except for the baseline peripheral temperature.

Interpretation: Fall in peripheral temperature in sevoflurane group is higher compared to propofol group

Table 3: Temperature gradient between core and peripheral body temperature

Time	Propofol		Sevoflurane		P value
	Mean	SD	Mean	SD	
Baseline T1	0.637	0.424	0.697	0.419	0.583543
T2	1.49	0.989	2.27	2.28	0.090943
T3	0.783	0.741	1.79	1.33	0.000615
T4	0.85	0.635	1.57	1.32	0.009261
T5	0.717	0.608	1.58	1.31	0.001796
T6	0.703	0.604	1.53	1.32	0.002813
T7	0.7	0.55	1.47	1.15	0.001615
T8	0.593	0.444	1.45	1.16	0.000374
T9	0.467	0.453	1.43	1.11	0.000047
T10	0.497	0.429	1.53	1.1	0.000012

The temperature gradient of core and peripheral body temperature of propofol group is compared with that of sevoflurane group. Multiple t test with statistical error calculated by HOL-SIDAK method is applied. The p value is significant for all the time intervals from T3 to T10.

Interpretation: The temperature gradient calculated as the difference between core and peripheral temperature at each time interval in both groups shows that there is significantly more fall in propofol group compared to sevoflurane group

that hypothermia persisted throughout the surgery. This greater difference in propofol induction is attributed to the systemic vasodilation along with blockade of central sympathetic tone.

In comparison to the study conducted by Ikheda et al., our study differs by following reasons, the first, TCI-Propofol was used for induction at plasma concentration of 5mcg/ml and maintained with the same at plasma concentration of 2-3mcg/ml in subjects randomized under the propofol group. This is in contrast with the study done previously where induction with 2mg/kg intravenous propofol was done and maintained with anaesthetic gas mixture of oxygen-nitrous oxide and volatile anaesthetic. Secondly, sevoflurane group subjects were induced with intravenous thiopentone injection at 3-5mg/kg body weight dosage and maintained with 33% oxygen, 66% nitrous oxide and 1-1.5 MAC sevoflurane in contrast to the previous study where in induction with 5% sevoflurane was done and then maintained with anaesthetic gas mixture of oxygen-nitrous oxide and sevoflurane.

K Leslie et al¹² studied that Propofol causes a dose-dependent decrease in the thermoregulatory threshold for vasoconstriction. They tested the hypothesis that propofol decreases the vasoconstriction threshold. Six healthy, male subjects were studied on 3 ordered days: as propofol, target propofol blood concentration 2 micrograms/ml, and target blood propofol concentration 4 micrograms/ml. Each day, epidural anesthesia (approximately T11 level) was induced, using 2% 2-chloroprocaine. Thermal manipulations were restricted to the legs, and they maintained upper-body (sensate) skin temperature constant. Propofol was infused by a computer-controlled infusion pump. Volunteers were warmed until sweating was observed, then cooled until fingertip vasoconstriction was observed. The vasoconstriction threshold was defined as the tympanic membrane temperature triggering a sustained reduction in fingertip blood flow to < 0.25 ml/min. Central venous blood was assayed for propofol blood concentration. Increasing propofol concentration produced a linear fall in the vasoconstriction threshold. Thus, they concluded that propofol reduces the vasoconstriction threshold.

In the study conducted by H. Anttonen et al., measured non-evaporative, cutaneous heat loss using heat flux transducers at eight skin sites in five children during anaesthesia and compared the data with basal metabolic heat production.¹³ They found that non-evaporative cutaneous heat loss exceeded basal metabolic heat production after induction of anaesthesia when patients were un-covered. Thermal radiation was the attributing mechanism for heat loss under physiological conditions, while evaporation, convection and conduction are less negligible causes. The highest regional heat fluxes were observed in the forehead. The relationship between regional heat flux and temperature difference was exponential and was attributed to increasing

radiation, as heat exchange by radiation is dependent on temperature to the fourth power. During anaesthesia the decrease in core temperature has been explained by redistribution of body heat via transfer from the core to the body surface, allowing the surface temperature to increase while the core temperature decreases.^{14,15} Based on their results, it can be concluded that cutaneous heat loss exceeded basal metabolic heat production after induction of anaesthesia, in pediatric age group unlike adult thermoregulation.

As per the study titled, "Difference in Core temperature in response to propofol-remifentanyl anesthesia and sevoflurane-remifentanyl anesthesia" by Ui Jae Im et al¹⁶ the induction and maintenance of anesthesia with intravenous propofol to determine if it causes more core hypothermia than inhaled sevoflurane was studied. Core temperatures decreased in both the propofol-remifentanyl group and sevoflurane-remifentanyl group during surgical operation, but there was no significant difference between the two groups. Thus, they concluded that, anesthesia induced and maintained by propofol did not cause a greater degree of hypothermia than sevoflurane. In contrast to this study, in our study we found that, the fall in core temperature for both TCI-propofol and sevoflurane based anesthesia was comparable, but the peripheral body temperature was much less in sevoflurane group in comparison to the propofol group.

As per the observations of our study, there is fall in core and peripheral body temperatures in both groups from their respective baseline values. The fall in core temperature is due to redistribution of the heat from core to peripheral compartment as there is a normal gradient of about 2°C-4°C between them under general anaesthesia and the heat once lost from core compartment cannot be regained back as per the second law of thermodynamics which states that heat cannot flow itself from colder body to a hotter body. Much obviously the reason for fall in peripheral temperature from the baseline is due to the anaesthetic agent induced vascular tone alteration leading to generalized peripheral vasodilation. However, the fall in core temperature in sevoflurane group is more at time interval T3 which can be explained by combined effect of the induction with intravenous thiopentone and maintenance sevoflurane at 1-1.5MAC. The next finding in the study is that, the fall in core temperature was comparable between groups, while the fall in peripheral temperature was more for sevoflurane group in comparison to TCI-propofol. The third finding is that, the temperature gradient shows that there is significant fall in sevoflurane group in comparison to the TCI-propofol group. This can be substantiated by the fact that, propofol produces marked and linear decrease in vasoconstriction and shivering thresholds but, the volatile anaesthetics decrease the cold response thresholds non-linearly, i.e., the volatile anaesthetics inhibit

vasoconstriction and shivering less than propofol at lower concentration, but more than propofol at typical anaesthetic doses. Also, nitrous oxide which was used for maintenance of anaesthesia in both groups has the property to decrease vasoconstriction and shivering thresholds further less than equipotent concentrations of volatile anaesthetics in comparison to its effect on propofol.

The limitations of our study is that, the temperature recording was not done for the entire duration of surgery and hence the postoperative possibility of hypothermia, shivering, requirement of opioids could not be studied, however 5 patients in TCI-propofol group and 3 patients in sevoflurane group required forced air warming in postoperative period for shivering of which 2 patients in either group were administered intravenous meperidine 25mg for shivering. Another limitation is that, although we noticed hypothermia in both the groups intraoperatively, no intervention to correct mild to moderate hypothermia was undertaken. Our study excluded elderly and pediatric population, ASA-PS ≥ 3 , pregnant and nursing mothers, patients undergoing lower limb surgeries, the same findings cannot be extrapolated. The other confounding factors observed are non-uniformity of intravenous fluid temperature, blood transfusion in some cases, variable duration of surgery, various types of surgery with difference in body surface area exposed.

5. Conclusion

We conclude that, TCI-propofol and sevoflurane anaesthesia causes significant fall in core and peripheral body temperature from their baseline. However, the fall in core body temperature was comparable between the groups, but the fall in peripheral body temperature was significantly more in sevoflurane based anaesthesia. When the temperature gradient between core and peripheral was compared, the gradient was more observed in sevoflurane compared to TCI-propofol based anaesthesia. The hemodynamic parameters were comparable between the groups, so was the recovery rate. Thus, we can conclude that, sevoflurane anaesthesia causes more hypothermia when compared to TCI-propofol based anaesthesia. In view of significant differences in temperature between the groups, further studies including patients from different age groups, comorbid diseases and longer duration of surgery has to be carried out and are essential to ascertain an advantage of TCI-propofol based anaesthesia being superior to sevoflurane anaesthesia in minimizing the effects of hypothermia. Thus, for prolonged procedures, TCI-based propofol will offer better outcome as far as hypothermia is concerned in comparison to sevoflurane based anaesthesia, however its effect on delayed recovery cannot be commented at this point in time.

6. Source of Funding

None.


7. Conflict of Interest


None.


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