Postanaesthetic Shivering—A Comparison of Thiopentone and Propofol

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Abstract

One hundred and sixty patients undergoing minor surgical procedures were randomly allocated to receive either thiopentone or propofol for induction of anaesthesia. All patients were assessed in the recovery period for the development of postanaesthetic shivering. Twenty patients (25%) in the thiopentone group and 8 patients (10%) in the propofol group developed postanaesthetic shivering (p <0.05). There was no statistically significant difference in tympanic temperature between shivering and nonshivering patients. Propofol as an induction agent is associated with a lower incidence of postanaesthetic shivering as compared to thiopentone.

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Key words: Anaesthetic complications, Body temperature, Pharmacology, Postoperative period, Tympanic membrane temperature

Introduction

The incidence of shivering following general anaesthesia varies from 5% to 65%.[1] Postanaesthetic shivering may increase tissue oxygen demand by as much as 500%[2] and accompanied by increases in minute ventilation and cardiac output to maintain aerobic metabolism. Failure to compensate may result in mixed venous desaturation, lactic acidosis and ischaemia of vital organs.[3] The elderly with their diminished cardiopulmonary reserves and patients with poor myocardial reserves are particularly at risk.[4,5]

A recent retrospective study using logistic regression analysis suggested that the incidence of postanaesthetic shivering was lower if propofol was used as the anaesthetic induction agent.[6] In this prospective study, we assessed the effect of the two commonly used induction agents, thiopentone and propofol, on the incidence of postanaesthetic shivering in our local patient population.

Patients And Methods

One hundred and sixty ASA I or II patients undergoing otorhinolaryngological, dental, gynaecological, orthopaedic or general surgery procedures were studied with approval of the local ethics committee. Patients were excluded if they had pre-existing history of fever, hypertension, ischaemic heart disease, or blood transfusion within 24 hours of the operation. No premedication was given. After informed consent was given, patients were randomly allocated into two groups according to a table of random numbers. The first group received thiopentone 4 $mg.kg^{-1}$ while the other received propofol 2.5 $mg.kg^{-1}$ as the intravenous induction agent. Anaesthesia was induced with intravenous fentanyl 2 $\mu g.kg^{-1}$ followed by the chosen induction agent. Endotracheal intubation was facilitated by suxamethonium 1 $mg.kg^{-1}$. Anaesthesia was maintained with 70% nitrous oxide and isoflurane 1% to 1.5% in oxygen via a circle system at a fresh gas flow rate of 3 $L.min^{-1}$. The minute ventilation was adjusted to maintain an end-tidal carbon dioxide concentration of 35 to 45 mmHg. Muscle paralysis was maintained by an infusion of atracurium at 0.5 $mg.kg^{-1}.h^{-1}$. At the end of the operation, residual paralysis was reversed with atropine 20 $\mu g.kg^{-1}$ and neostigmine 40 $\mu g.kg^{-1}$. Intravenous morphine 0.2 $mg.kg^{-1}$ was used in all patients during the surgery. Heart rate (HR), blood pressure (BP) and pulse oximetry (SaO_2) recordings were made prior to induction and at 5 minutes interval during anaesthesia. The tympanic membrane temperature, taken to represent core temperature, was measured using a Thermoscan® Pro-1 thermometer (San Diego, CA, USA) before induction of anaesthesia. Duration of anaesthesia was timed from the induction of anaesthesia to the cessation of the volatile agent.

All patients received an intravenous infusion of Hartman's solution at ambient temperature. The operating theatre temperature was maintained at 22 ± 0.5°C and a relative humidity of 55% to 58%. No warming blanket was used during the operative procedure and no blood or blood products given. All patients were covered with the standard gown and sterile drapes.

Upon transfer to the recovery unit, all patients re-

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ceived standard postoperative management including administration of oxygen 40% via a Ventimask. Oxygen saturation, heart rate, blood pressure and tympanic temperature were measured upon arrival at the recovery room and were repeated after 5 minutes and again at 15 minutes interval until discharge from the recovery room. The occurrence and duration of shivering were assessed by one of the investigators who was unaware of the induction agent given to the patient. Shivering was defined as any involuntary movements resembling those seen normally in thermoregulatory shivering.

Statistical analysis of the data was carried out using Chi-square and Student's *t*-tests where appropriate using the SPSS-PC+ package. A p value of less than 0.05 was taken as statistically significant.

Results

The two groups of patients were comparable in age, weight and duration of anaesthesia (Table I). Twenty-five per cent of the patients induced with thiopentone developed postoperative shivering compared to only 10% of those who received propofol as the induction agent. The difference was statistically significant ($p = 0.012$, Chi square test). The duration of shivering in the thiopentone group was longer than those in the propofol group although the difference was not statistically significant ($p = 0.07$, Student's *t*-test). The mean intraoperative decrease in temperature was 0.69 ± 0.63°C in the thiopentone group and 0.74 ± 0.67°C in the propofol group ($p = 0.68$, Student's *t*-test).

TABLE I: PATIENT CHARACTERISTICS

	Thiopentone (n = 80)	Propofol (n = 80)
Sex: male/female	30/50	28/52
Age (y)	35.9 (12.1)	34.9 (13.0)
Height (cm)	164.3 (6.6)	165.7 (7.4)
Weight (kg)	60.1 (10.8)	62.3 (18.1)
Duration of anaesthesia (min)	75.5 (37.3)	75.6 (50.75)
Baseline temperature (°C)	36.5 (0.37)	36.9 (0.53)
No. shivered	20	8
Duration of shivering (min)	20.9 (12.5)	15.0 (7.1)

Results are expressed as mean (SD)

TABLE II: MONITORED PARAMETERS ON ARRIVAL TO RECOVERY ROOM

	Thiopentone (n = 80)	Propofol (n = 80)	*t*-test
HR (beat/min)	82 (14)	78 (15)	$p = 0.15$
Systolic BP (mmHg)	129 (20)	130 (21)	$p = 0.9$
Diastolic BP (mmHg)	74 (14)	76 (13)	$p = 0.46$
SaO_2 (%)	98.1 (1.5)	98.6 (1.4)	$p = 0.17$
Temperature (°C)	36.3 (0.9)	36.3 (0.7)	$p = 0.87$

Results are expressed as mean (SD)
HR: heart rate; BP: blood pressure

On admission to the recovery room, there were no significant differences between the two groups of patients with regard to heart rate, blood pressure and oxygen saturation (Table II). No difference was noted in the temperature readings of those patients who shivered in the thiopentone group when compared to those in the propofol group (Table III).

The mean temperature values of patients who developed shivering and those who did not showed no significant difference at baseline and upon arrival to the recovery room (Table IV). At the time of discharge, those patients who had shivering had higher tympanic temperature readings than those who did not. The increase in the temperature in patients who shivered (0.83 ± 0.7°C) was however, not statistically significant from the patients who did not shiver (0.69 ± 0.6°C) ($p = 0.29$, Student's *t*-test).

Discussion

The finding that our local patients induced with propofol had a lower incidence of postoperative shivering as compared to those given thiopentone is consistent with the results by Crossley[6] and Singh et al.[7] However, in contrast to other studies,[7,8] we found that patients who shivered had a greater increase in tympanic temperature readings than those patients who did not. In this instance, postoperative shivering may be useful in accelerating the rewarming rate, the efficacy of which as a thermoregulatory response has been shown by other authors.[9,10]

While the aetiology of postanaesthetic shivering is still unclear, the adverse effects that may arise from it are well documented.[3,11-13] Opinions still differ as to whether

TABLE III: MEAN TEMPERATURE VALUES ON ARRIVAL TO RECOVERY ROOM

	Thiopentone group (n = 80)	Propofol group (n = 80)	*t*-test
Shivering patients	36.4 (0.9)	36.3 (0.8)	$p = 0.87$
Non-shivering patients	36.2 (0.9)	36.2 (0.7)	$p = 0.95$

Results are expressed as °C mean (SD)

TABLE IV: MEAN (SD) TYMPANIC TEMPERATURE IN SHIVERING AND NON-SHIVERING PATIENTS

	Shivering (n = 28)	Non-shivering (n = 132)	*t*-test
Baseline	37.2 (0.6)	36.7 (0.3)	$p = 0.32$
Entering recovery	36.3 (0.8)	36.2 (0.8)	$p = 0.43$
At discharge	36.8 (0.8)	36.3 (0.7)	$p = 0.003$

Results are expressed as °C mean (SD)

postoperative shivering is a true thermoregulatory response to the hypothermia sustained during the course of anaesthesia or it is due to the effects of residual anaesthetics inhibiting descending cortical control and causing spontaneous tremors by spinal reflex activation.[14] Not uncommonly, surgical patients become hypothermic due to an internal redistribution of heat from the core to the periphery due to anaesthetic-induced vasodilatation and as a result of heat loss to the environment exceeding metabolic heat production.[15,16] Cold thermoregulatory responses (vasoconstriction, nonshivering thermogenesis and shivering) would be activated as a result of the hypothermia.[17] However, the thresholds for these responses are known to be inhibited by anaesthetic drugs.[18-22] Recovery from anaesthesia in the presence of core hypothermia with the return of these cold responses thresholds towards normal would then stimulate thermoregulatory vasoconstriction and shivering.[23] Intuitively then, one would associate postanaesthetic shivering with the presence of hypothermia; thus implying that postanaesthetic shivering is thermogenic in nature. However, the exact aetiology remains debatable as a number of studies have shown a poor correlation between body temperature and the presence of postanaesthetic shivering.[6,7,24] In addition, a different electromyographic pattern of muscular contraction is seen in patients with postanaesthetic shivering as compared to hypothermia-induced shivering.[14] As such, it has been suggested that postanaesthetic shivering may represent some form of inhibition of cortical control by residual anaesthetics resulting in spinal hyperactivity and spontaneous postanaesthetic shivering. As the half-life of thiopentone is longer than that of propofol, it will therefore have a greater residual effect on cortical control than propofol, leading to spinal hyperactivity and consequently, shivering. In our present study, there was no difference in the tympanic temperature in those patients who developed shivering as compared to those who did not, thus supporting the view that body temperature per se may not be related to the development of shivering.

The incidence of postanaesthetic shivering may be decreased by a variety of means. The use of reflective blankets,[25] cutaneous forced-air warming devices,[26] warm humidified anaesthetic gases[27] and radiant heat[28] had been shown to be effective. Drug therapy offers an alternative solution.[1,29] Regarding this aspect, the use of opioids in the prevention and treatment of postanaesthetic shivering is well recognised.[1,6,12,17,29] Unfortunately, side-effects such as nausea, vomiting and respiratory depression may result. Our study, together with others,[6,7] demonstrated that the incidence of postoperative shivering can simply be influenced by the choice of induction agent. Even though the mechanism by which this occurs cannot be elucidated from this study, the lower incidence of shivering is beneficial with regard to the lower requirements for oxygen demand and cardiac output, thereby decreasing the risk of ischaemia in vital organs in the compromised patients.

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