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Original article

Does sevoflurane preserve regional cerebral oxygen saturation measured by near-infrared spectroscopy better than propofol?

Le sévoflurane préserve-t-il mieux la saturation cérébrale régionale en oxygène mesurée par la spectroscopie infrarouge que le propofol ?

L. Valencia [a,*], A. Rodríguez-Pérez [a], B. Kühlmorgen [a,b], R.Y. Santana [a,c]

[a] *Department of Anaesthesiology, Hospital Universitario de Gran Canaria Doctor Negrín, Barranco de la Ballena s/n., 35010 Las Palmas de Gran Canaria, Spain*
[b] *Department of Anaesthesiology, Hospital el Ángel, Málaga, Spain*
[c] *Department of Traumatology, Hospital Universitario Insular de Gran Canaria, Las Palmas de Gran Canaria, Spain*

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ABSTRACT

Objective. – The aim of this study was to compare the effect of sevoflurane and propofol on cerebral oxygenation, using regional cerebral oxygen saturation (SrO_2) measured by near-infrared spectroscopy (NIRS).
Study design. – Prospective, randomized, controlled study.
Patients and methods. – Fifty-four patients aged between 18 and 65 years who underwent elective minor surgery (tumorectomy for breast cancer or inguinal hernia repair) were randomly assigned to receive sevoflurane or propofol anaesthesia. Exclusion criteria included pre-existing cerebrovascular diseases, anaemia, ASA >III, blood loss ≥200 mL, arterial hypotension, baseline pulse oximetry <97%, sign of sensor low quality of SrO_2 or bispectral index, and patients with a forehead area <6.5 cm. SrO_2, bispectral index, haemodynamic data and anaesthetic doses were recorded during surgery.
Results. – A total of 48 patients were included in the final analysis (24 in each group). There were no significant differences in mean, minimum and maximum SrO_2 between sevoflurane and propofol groups. The relative maximum decrease was higher in propofol anaesthesia than sevoflurane anaesthesia (9.6 ± 10.7 versus 4.2 ± 7.2%; $P = 0.048$). Cerebral desaturation (20% reduction from SrO_2 baseline during 15 seconds) occurred in 4 patients in propofol group exclusively ($P = 0.109$). SrO_2 adjusted for baseline was higher in the sevoflurane group than in the propofol group (67.3 ± 1.8% versus 64.2 ± 1.7%; $P = 0.018$). There were no significant differences in haemodynamic parameters between the two groups.
Conclusions. – Cerebral cortical oxygenation measured by NIRS may be better preserved with sevoflurane than with propofol. These findings suggest that sevoflurane anaesthesia could be a good option in patients with compromised cerebral oxygenation, given the absence of intracranial hypertension. Further studies with larger sample sizes are required to support our results.
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Mots clés :
Sévoflurane
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Monitorage

RÉSUMÉ

Objectif. – L'objectif de cette étude était de comparer l'effet du sévoflurane et du propofol sur l'oxygénation cérébrale, en utilisant la saturation cérébrale régionale en oxygène (SrO_2) mesurée par la spectroscopie infrarouge (NIRS).
Type d'étude. – Étude prospective, randomisée et contrôlée.
Patients et méthodes. – Cinquante-quatre patients âgés entre 18 et 65 ans programmés pour chirurgie élective mineure (tumorectomie pour cancer du sein ou réparation d'hernie inguinale) ont été assignés après randomisation à recevoir du sévoflurane ou du propofol.
Critère d'exclusion. – Antécédents de maladie cérébrovasculaire, anémie, score ASA > III, saignement ≥ 200 mL, hypotension artérielle, saturation artérielle en oxygène < 97 %, mauvais signal SrO_2 ou d'index bispectral, et une surface frontale < 6,5 cm. La SrO_2, l'index bispectral, les variables hémodynamiques et les doses anesthésiques ont été enregistrés au cours de la chirurgie.

* Corresponding author.
E-mail addresses: ori98es@yahoo.es, romenyone@hotmail.com (L. Valencia).

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Résultats. – Quarante-huit patients ont été inclus dans l'analyse finale (24 dans chaque groupe). Aucune différence significative n'a été trouvée entre les groupes pour la SrO_2 moyenne, minimal et maximal. La baisse maximale de la SrO_2 basale était plus importante avec le propofol (9,6 ± 10,7 % versus 4,2 ± 7,2 % ; $p = 0{,}048$). Quatre épisodes de désaturation cérébrale (réduction de 20 % de la SrO_2 basale pendant 15 secondes) ont été observés dans le groupe propofol exclusivement ($p = 0{,}109$). La SrO_2 ajustée à la valeur basale était supérieure avec le sévoflurane par rapport au propofol (67,3 ± 1,8 % versus 64,2 ± 1,7 % ; $p = 0{,}018$). Aucune différence significative des paramètres hémodynamiques n'a été observée entre les deux groupes.

Conclusions. – L'oxygénation cérébrale corticale, mesurée par la NIRS, est probablement mieux conservée par le sévoflurane que le propofol. Ces résultats suggèrent que l'anesthésie au sévoflurane pourrait être une meilleure option chez les patients dont l'oxygénation cérébrale est compromise, à condition qu'il n'y ait pas d'hypertension intracrânienne. D'autres études plus larges sont nécessaires afin de soutenir nos résultats.

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1. Introduction

In several surgical procedures and in some clinical situations where there is a risk of cerebral hypoperfusion, the protection of the central nervous system becomes a priority for anaesthesiologists. Under these circumstances, adequate monitoring and the choice of the right anaesthesia can play a fundamental role in the outcome. Both sevoflurane and propofol have neuroprotective properties [1]. However, neither has proved clearly superior in the maintenance of cerebral oxygenation. Studies on human subjects have shown that although both drugs produce a similar decrease in Cerebral Metabolic Rate ($CMRO_2$), sevoflurane (MAC < 1.0) reduces cerebral blood flow (CBF) less than propofol [2–5]. This translates to a decrease of $CBF/CMRO_2$ ratio in the case of propofol, which remains constant when sevoflurane is used [6]. Thus, the cerebral oxygenation balance could be better preserved with sevoflurane than with propofol [7,8].

Near-infrared spectroscopy (NIRS) is a non-invasive technique that provides continuous monitoring of regional cerebral tissue oxygen saturation (SrO_2). Numerous studies have demonstrated its efficiency in discovering intraoperative silent ischaemic events and improve the results in high-risk surgery, such as cardiac [9], thoracic [10,11] and abdominal surgeries in elderly patients [12,13]. Because SrO_2 is capable of detecting small changes in cerebral oxygenation reflecting the $CBF/CMRO_2$ ratio, it is possible to evaluate how cerebral oxygenation is affected by sevoflurane and propofol.

The aim of this study was to determine if NIRS detects differences in SrO_2 between sevoflurane or propofol anaesthesia.

2. Patients and methods

Approval for this randomised, controlled study was provided by the Ethical Committee of the Hospital Universitario de Gran Canaria Doctor Negrín, Las Palmas de Gran Canaria, Spain (chairperson Professor Dr S. Ruiz) on 12 September 2009 (registration number 09/021). After written informed consent, 54 patients aged between 18 and 65 years who underwent elective minor surgery (tumorectomy for breast cancer or inguinal hernia repair) were randomly assigned to receive sevoflurane (group S) or propofol (group P) anaesthesia between September 2009 and February 2010. Simple randomisation was carried out using a random number table generator. Numbers were distributed in sealed, opaque envelopes which were opened at the beginning of anaesthesia. Non-inclusion criteria were pre-existing cerebrovascular diseases, ASA physical status ≥IV, anaemia according to WHO criteria (haemoglobin in females <12 g/dL and in males <13 g/dL) and patients with a forehead area <6.5 cm because it was impossible to attach both sensors. Exclusion criteria included baseline peripheral oxygen arterial saturation (SpO_2) less than 97%, low quality signs of SrO_2 or bispectral index (BIS), bleeding more than 200 mL or haemodynamic instability (decrease of 25% of baseline mean arterial pressure [MAP] for three minutes).

No premedication was administered. Monitoring included electrocardiography, SpO_2, non-invasive arterial pressure measurement (Cicero EM PM8060™, Dräger, Lübeck, Germany), bispectral index (BIS VISTA Aspect Medical Systems™, Massachusetts, USA) and SrO_2 using INVOS 5100C™ (Covidien, Dublin, Ireland). The SrO_2 sensors were placed in the superior supraorbital margins, on top of those BIS electrodes. Before recording the data, we checked for a good signal, free of artefacts on both monitors, using Signal Quality Index (ICS) for BIS and the Signal Strength Indicator (SSI) for SrO_2.

During the surgical procedure, the degree of neuromuscular blockade was logged (TOF Watch™, Organon Teknika, Durhman, USA), as well as the inspired and expired oxygen fractions, end-tidal carbon dioxide tension ($ETCO_2$), concentration of sevoflurane and tympanic temperature (Genius 2™, Covidien, Dublin, Ireland). The latter was maintained between 35 and 36 °C using a warming blanket (Warm Touch 5300™, Nellcor, UK).

After full preoxygenation, all patients in group S received fentanyl (2 μg/kg), before vital capacity induction with sevoflurane 8% (SIBI connector™, Ventitech Medical Device, Quebec, Canada). Cisatracurium (0.2 mg/kg) after loss of verbal response was administered. In group P, anaesthesia was induced with fentanyl (2 μg/kg), propofol (2 mg/kg) and cisatracurium (0.2 mg/kg). In all cases, orotracheal intubation was performed three minutes after injection of the neuromuscular blocker and connected to mechanical ventilation (workstation Dräger Cicero EM™, Lübeck, Germany), with the following parameters: tidal volume 7 mL/kg, respiration rate 12-c/min, inspiration–expiration ratio 1:2, oxygen/air mix (40/60%). Afterwards, minute volume was adjusted to obtain an $ETCO_2$ of between 30–34 mmHg.

Anaesthesia was maintained with propofol (4–8 mg/kg/h) or sevoflurane (1–2% end-tidal concentration) according to group. Propofol was administered to obtain BIS values of between 40 and 60. Both groups used remifentanyl with doses of between 0.05–0.3 μg/kg/min at the anaesthesiologist's discretion. Neuromuscular blockade was achieved with cisatracurium bolus, maintaining one or two responses in a train-of-four stimulation. Five milligrams of intravenous ephedrine was administered when the MAP decreased to less than 25% of baseline values.

Previous to induction, the following baseline values were recorded while the patient breathed room air: MAP, heart rate (HR), SpO_2, BIS and SrO_2. These variables were recorded every minute during the first five minutes after induction. Afterwards, until perfusion of propofol or sevoflurane was suspended at the end of surgery, the same data was collected every five minutes. Furthermore, the propofol dose used or expired fraction of sevoflurane was noted each minute. Cerebral oxygen desaturation

was defined as a decline in SrO_2 more than 20% from baseline for more than 15 s. Data were recorded by an independent investigator.

2.1. Statistical analysis

Sample size was chosen based on a pilot sample of 12 subjects. Differences in SrO_2 adjusted for a baseline value of three units was found (3.1 ± 1.5%). With these data, and assuming 80% strength and an alpha level of 0.05, a sample size for 24 patients in each group was calculated. We expected to detect a difference of 5% in a design with repeated measurements. Data from the pilot study were not included in the present study.

In the descriptive analysis of quantitative variables, the mean and standard deviation (S.D.) were used. The mean differences were analyzed using Student's *T* test for quantitative variables in those cases in which those variables followed a normal distribution. Distribution was evaluated by Kolmogorov-Smirnoff's *Z* test. Differences in proportion were carried out with contingency analysis using Chi-square and by Fisher's exact test when needed. A mixed-model repeated-measure analysis compared the effect of sevoflurane on SrO_2 adjusted for baseline over the surgery period. The *U* Mann-Whitney's test was used for the differences in MAP, HR, BIS and SrO_2 each time with respect to baseline value and between both groups. A P-value < 0.05 was considered statistically significant. Statistical analysis was performed using the SPSS-PC statistical software program (version 15.0; SPSS, Inc., Chicago, IL, USA).

3. Results

Fifty-four patients between 18 and 65 years scheduled for breast cancer tumorectomy (22 patients) or inguinal hernia repair (32 patients) were considered for the study. Four patients did not meet inclusion criteria (one because of pre-existing cerebrovascular disease, another due to anaemia, and two more because it was impossible to place the SrO_2 and BIS sensors). No patients declined to participate. After randomization, one patient in group S and another in group P bled more than 200 mL or suffered an episode of arterial hypotension for more than three minutes respectively. These patients were also excluded from the final analysis. Twenty-four patients in each group completed the study. There were no differences in demographic and intraoperative data between the groups (Table 1).

Table 2 shows the preoperative haemodynamic variables, SrO_2 and baseline SpO_2. There were no significant statistical differences between the groups. The end-tidal sevoflurane concentrations ranged between 3.2 and 2.4% in the group S during inhalation induction and between 1.3 and 1.7% during surgery. In group P, a maintenance dose of 4.7 ± 0.7 mg/kg/h was used. BIS (mean ± S.D.) did not differ between the groups (49.9 ± 4.2 in group S and 46.1 ± 6.1 in group P; $P = 0.13$). However, the BIS values were superior in group S in the induction (minutes 1, 2 and 3) and at minute 70 (Fig. 1).

Table 2
Preoperative cerebral oximetry values, baseline pulse oximetry and baseline haemodynamic data.

	Propofol ($n = 24$)	Sevoflurane ($n = 24$)	P-value
Baseline SrO_2 (%)	63.4 ± 9.9	61.4 ± 11	0.52
Mean arterial pressure (mmHg)	92.7 ± 12.2	98.1 ± 13.9	0.16
Baseline pulse oximetry (%)	99.3 ± 1	99.5 ± 1.1	0.32
Heart rate (b/min)	72.1 ± 12.9	77.9 ± 14	0.14

Data are mean ± S.D.

The mean, minimum and maximum values of SrO_2 reached were similar in both groups (Table 3). The maximum decrease of SrO_2 with respect to its baseline values was calculated, obtaining a higher decrease of five points in group P compared with group S (9.6 ± 10.7% versus 4.2 ± 7.2%; $P = 0.048$). Patients' SrO_2 data were compared between both groups using a mixed-model ANCOVA. We noted that SrO_2 depended on their baseline value, since patients with higher baseline levels tended to have higher means. When the SrO_2 values were adjusted for baseline, it was observed that the predicted mean SrO_2 for each patient did not depend only on the baseline SrO_2, but also on the anaesthetic used ($SrO_2 = 66.76 + 3.04 \times$ sevoflurane + 0.968 [baseline SrO_2–62.4]). The results of the covariance analysis showed the adjusted mean SrO_2 was lower in group P (64.2 ± 1.7%) than group S (67.3 ± 1.8%). This was a significant statistical difference ($P = 0.018$).

When comparing the groups' mean SrO_2 at each data collection point, there were no differences between the groups (Fig. 2). Up to the first 15 minutes in both groups significant superior SrO_2 values were found with respect to baseline value. These differences in the group S persisted until the 25th minute. Four patients in group P had episodes of cerebral desaturation while group S had none ($P = 0.109$). These events occurred during the anaesthetic maintenance phase, without regard to changes in MAP, HR or BIS.

Haemodynamic variables are represented in Figs. 3 and 4. In both groups, there was a decrease in MAP (16 ± 4.2% in group P versus 9 ± 17.2% in group S; $P = 0.06$) with respect to values previous to the induction. There were no significant statistical

Table 1
Demographic variables and intraoperative variables.

	Propofol ($n = 24$)	Sevoflurane ($n = 24$)	P-value
Age (years)	43.4 ± 12.9	50.6 ± 12.6	0.076
Body mass index (kg/m²)	27.2 ± 5.6	27.5 ± 5.6	0.78
Male/female	6/18	7/17	0.75
Haemoglobin (g/dL)	13.2 ± 1.4	12.8 ± 1.3	0.65
ASA physical status (I/II/III)	7/16/1	4/17/1	0.42
Underlying diseases			
Chronic obstructive pulmonary disease	4	4	1
Renal insufficiency	0	2	0.49
Arterial hypertension	4	10	0.06
Diabetes mellitus	1	4	0.35
Duration of anaesthesia (minutes)	96.1 ± 19.2	102.1 ± 16.2	0.24
Fluid administered (mL)	1095 ± 554	1050 ± 510	0.77

Data are mean ± S.D. or numbers.

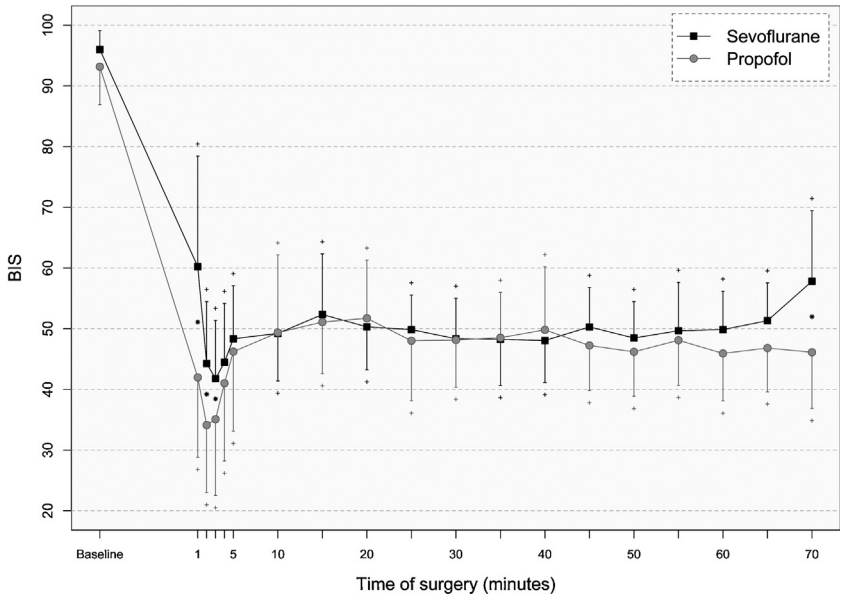

Fig. 1. BIS in patients under sevoflurane or propofol anaesthesia. Values are mean ± S.D. Preinduction values are shown at time O. + *P*-value < 0.05 versus baseline. *P-value < 0.05 between sevoflurane and propofol groups. The numbers of patients decreased in both groups from 24 at baseline to 22 at 70 minutes. BIS: bispectral index.

Table 3
SrO_2 values in propofol and sevoflurane groups.

	Propofol ($n = 24$)	Sevoflurane ($n = 24$)	*P*-value
Mean SrO_2 (%)	64.8 ± 11.5	65.9 ± 10.4	0.73
Minimum SrO_2 (%)	57.4 ± 12.3	58.6 ± 10.6	0.71
Maximum SrO_2 (%)	75.3 ± 11.2	76.4 ± 10.1	0.74
Relative maximum decrease SrO_2 (%)	9.6 ± 10.7	4.2 ± 7.2	0.04
Mean value of SrO_2 adjusted for baseline (%)	64.2 ± 1.7	67.3 ± 1.8	0.018

Data are mean ± S.D.

differences when comparing mean MAP values between the groups (81 ± 9.1 in group S and 81.7 ± 8.1 in group P; $P = 0.79$). At 1 and 2 minutes, MAP was statistically higher in group S than group P, while at 40 minutes it was lower than group P (Fig. 3). Significant differences in recorded MAP were found in both groups with respect to baseline values during data collection. Mean ± S.D. heart rates were very similar in groups S and P (66.7 ± 11.5 and 67.4 ± 8.1, respectively) without statistical differences ($P = 0.791$). At minutes 10, 30 and 40, heart rate was significantly higher in group P (Fig. 4).

4. Discussion

The results obtained in the present study indicate that SrO_2 values adjusted for baseline during sevoflurane anaesthesia were significantly higher than those observed during propofol anaesthesia. Also, the maximum decrease in SrO_2 with respect to baseline values was more pronounced in group P than group S. In group P, there were 4 episodes of cerebral desaturation, while there were none in group S. These results highlight the fact that sevoflurane decreases CBF less than propofol compared with the

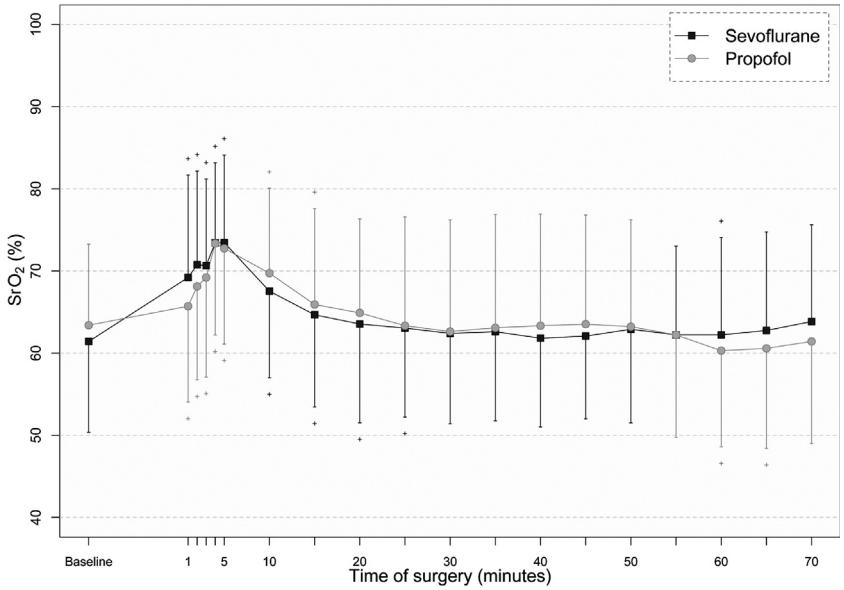

Fig. 2. SrO_2 in patients under sevoflurane or propofol anaesthesia. Values are mean ± S.D. Preinduction values are shown at time O. + *P*-value < 0.05 versus baseline. *P-value < 0.05 between sevoflurane and propofol groups. The numbers of patients decreased in both groups from 24 at baseline to 22 at 70 minutes. SrO_2: cerebral oximetry values.

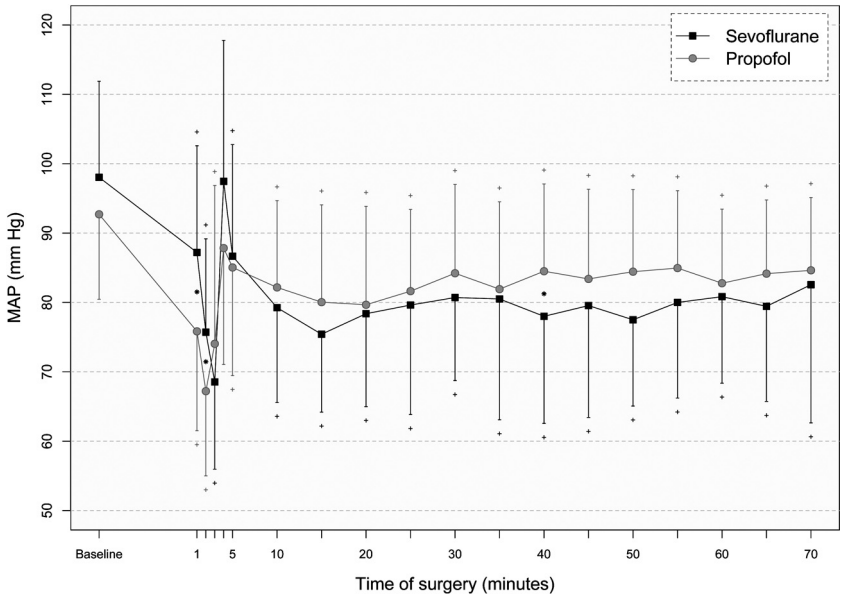

Fig. 3. MAP in patients under sevoflurane or propofol anaesthesia. Values are mean ± S.D. Preinduction values are shown at time O. + *P*-value < 0.05 versus baseline. **P*-value < 0.05 between sevoflurane and propofol groups. The numbers of patients decreased in both groups from 24 at baseline to 22 at 70 minutes. MAP: mean arterial pressure.

decrease in $CMRO_2$ that both produce. Inhalational anaesthetic agents alter the cerebral autoregulation in a dose-dependent way, and are not able to maintain the coupling between the CBF and $CMRO_2$ [14]. However, today we know that sevoflurane at concentrations lower than 1.5 MAC maintains the coupling between CBF and $CMRO_2$, the same as what occurs with propofol [15,16]. Not only is the CBF/$CMRO_2$ ratio more well preserved with sevoflurane, but its coupling at anaesthetic levels is also guaranteed.

The global effect of sevoflurane and propofol on cerebral oxygenation has already been studied on other occasions by monitoring jugular venous bulb oxygen saturation (SjO_2). The results of these studies have shown a tendency to obtain higher values of SjO_2 with the use of sevoflurane. The majority of authors agree with us that sevoflurane maintains a constant ratio of CBF/$CMRO_2$ and thus maintains oxygenation in cerebral tissue [6–8,17,18]. SjO_2 and SrO_2 are sensitive and specific instruments for detecting cerebral ischaemia [7]. However, studies that have used SrO_2 have not found any differences between sevoflurane and propofol. Both Yoshitani et al. [7] in patients who underwent total hip arthroplasty and Jeong et al. [19] in arthroscopic shoulder surgery in beach chair position detected no differences in SrO_2 with different anaesthetic agents. This could be due to various reasons. First, in both studies nitrous oxide was added to sevoflurane during the maintenance phase. It has been noted that when nitrous oxide is added to sevoflurane, important changes occur in cerebral haemodynamics, restoring CBF to levels similar to a waking state [20]. Moreover, high concentrations of nitrous oxide (70%) are also capable of increasing $CMRO_2$ [2]. Therefore, although the effect of nitrous oxide on SrO_2 has not been studied concretely, it seems evident that the use of nitrous oxide influences SrO_2 values. Second, a study in 2011 showed that

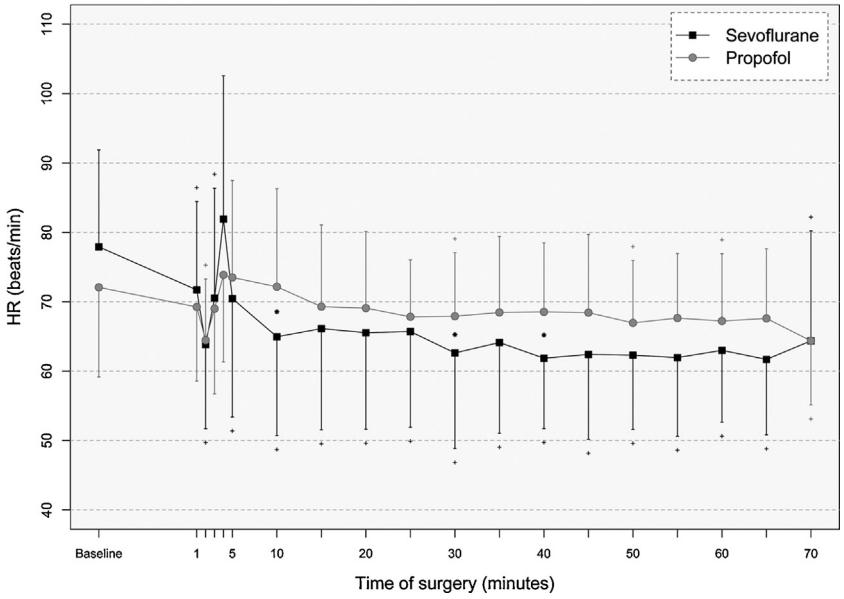

Fig. 4. HR in patients under sevoflurane or propofol anaesthesia. Values are mean ± S.D. Preinduction values are shown at time O. + *P*-value < 0.05 versus baseline. **P*-value < 0.05 between sevoflurane and propofol groups. The numbers of patients decreased in both groups from 24 at baseline to 22 at 70 minutes. HR: heart rate.

propofol has a differential effect on cerebral blood vessels, conserving cerebral oxygen saturation in the frontal cortex [21]. These findings suggest that cerebral metabolic demand may be reduced by propofol administration in cortical regions [22]. Third, in both studies there were clinical circumstances that could affect the values of SrO_2, masking the effect of the anaesthetic drugs on cerebral oxygenation. In the Yoshitani et al. [7] study, the loss of haemoglobin revealed a significant decrease in SrO_2. Surgery in beach chair position in the Jeong et al. [19] study produced a decrease in CBF, consequently altering recorded values of SrO_2.

In this study, we controlled the variables that influence SrO_2, including the type of anaesthetic used and the necessary dosage to obtain an adequate hypnotic level through BIS [23]. Group S received sevoflurane for both induction and maintenance of anaesthesia. We did this to avoid mixing hypnotics that could skew the results. Although vital capacity inhalatory induction has not been popular due to its adverse respiratory effects, sevoflurane permits a rapid and well-tolerated induction [24].

The remaining variables that could have an influence on SrO_2 were precisely those that affected the balance of supply and demand of cerebral oxygen. In this way, we excluded circumstances that did not guarantee a supply of oxygen to the brain, such as procedures that caused bleeding, haemodynamic instability and changes in surgical position. By doing this, we optimized some adequate levels of inspired oxygen content and arterial carbon dioxide concentrations, monitoring pulse oximetry and $ETCO_2$. With respect to factors that modulated the demand of cerebral oxygen, we recorded anaesthetic depth and temperature.

One of the limitations of SrO_2 is the lack of normal universal threshold. The most common limit is a relative decrease of 15–20% with respect to baseline [25] or SrO_2 <50 or 55% in absolute values [26]. The results of our study are expressed in values that refer to baseline SrO_2 since we used a relative oximeter (INVOS 5100C™, Covidien, Dublin, Ireland). However, FORE-SIGHT™ (CAS Medical Systems, Brandford, USA), another SrO_2 monitor, offers better technology for providing absolute real values [27]. Unfortunately, there are very few published studies on this monitor [11].

With this aforementioned definition of an episode of cerebral desaturation, we find an 8% incidence. Compared to other works, the percentage of these events was lower. Casati et al., in two prospective studies on patients of advanced age during major abdominal surgery, obtained a cerebral desaturation of between 20 and 26% [12,13]. In arthroscopic shoulder surgery, the figures increased to 80% at the moment that the patient was placed in the beach chair position [28,29]. Also, in thoracic surgery during one-lung ventilation, the percentage is around 80% [30]. Some authors used a different threshold than ours to determine an episode of cerebral desaturation. Casati et al. [12,13] defines it as when SrO_2 falls below 25% of baseline value or 20% when the SrO_2 record is less than 50%. In arthroscopic shoulder surgery, Moerman et al. used a decrease of 20% with respect to baseline SrO_2 or a 50% absolute value of SrO_2 [28]. Another discrepancy is that in some studies the baseline SrO_2 is not indicated as previous to induction. It is considered as when the patient is anaesthetised [30]. Finally, not all studies define the duration of the decrease of SrO_2 as an episode of cerebral desaturation, although many specify a duration of 15 s. Apart from the definition of chosen cerebral desaturation, the majority of these studies involved elderly patients and surgeries with a high probability of neurological complications. Given that our patients were less than 65 years old and underwent surgery with low risk of cerebral hypoperfusion, it is reasonable that the number of episodes of cerebral desaturation was lower than what has been reported.

As to haemodynamic variables, our results showed no clear differences in MAP and HR between the two groups. Up to now, neither propofol nor sevoflurane have demonstrated a superior haemodynamic stability, and there is a lot of variability. As to HR, the studies are not clear either, although some have shown that propofol generated a decrease in HR compared to sevoflurane [31].

The present study has various limitations. First, we did not measure carbon dioxide values in arterial blood because we based our procedure on $ETCO_2$ levels. Minimal changes in arterial blood CO_2 concentrations caused large variations in CBF, and as a consequence in the cerebral oxygenation. However, we decided not to place an arterial catheter due to its invasiveness and potential complications. Second, our main objective was the SrO_2 values, without evaluating other results with high clinical relevance such as postoperative neurocognitive deficits. There are studies that relate a lower intraoperative SrO_2 with the appearance of postoperative cognitive deficits [11]. In our study, surely the magnitude of SrO_2 changes observed in groups P and S would not have been large enough to be clinically relevant. Nevertheless, these small differences could be significant in other circumstances where the oxygen supply is critical. Third, although there were no significant differences in mean BIS, in the first minutes BIS values were higher in group S. Inhalatory induction can be slower than when done with propofol. Therefore, low deep anaesthesia levels increase cerebral metabolism and the $CMRO_2$, thus increasing the SrO_2 values [32]. Fourth, the use of target-controlled infusion (TCI) in the case of propofol, or age-adjusted MAC in the case of sevoflurane, would have permitted us to control the anaesthesia dosage in a more exact way. Finally, cerebral oxygenation monitoring with NIRS is not widely accepted because it presents some limitations. There is a wide variability in normal values because of extra-cerebral contamination (scalp, cranium and cerebrospinal fluid). Because is a regional monitor, may fail in detecting ischemia areas distant to the site of sensor placement [26].

5. Conclusion

This is the first study demonstrating that, when measured by NIRS, cerebral cortical oxygenation may be better preserved with sevoflurane than with propofol. Therefore, sevoflurane anaesthesia may provide cerebral oxygenation better than propofol. The same evidence was discovered using SjO_2. The agreement of the results of two different cerebral oxygen monitoring methods using different technology is an important step in reaching a causal link, which supports our hypothesis.

These findings suggest that sevoflurane offers a margin of safety in patients whose cerebral oxygenation is compromised. Therefore, sevoflurane presents a favourable cerebral haemodynamic effect. This could be a better anaesthetic option in situations where the balance between oxygen supply and demand is affected. However, we should emphasize that in our study patients with endocranial hypertension were not included. For that reason, our results do not extend to patients with brain injury, especially those with endocranial hypertension. In those cases, propofol is preferable to sevoflurane, because it respects cerebral haemodynamics without affecting intracranial pressure [33].

More studies with larger sample sizes are needed to confirm our hypothesis. It would also be interesting to study the small incidences of cerebral desaturation episodes observed with sevoflurane which reflect a decrease in postoperative cognitive deficits, at least in patients at risk for cerebral hypoperfusion.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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