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The Effects of Isoflurane and Desflurane on Intracranial Pressure, Cerebral Perfusion Pressure, and Cerebral Arteriovenous Oxygen Content Difference in Normocapnic Patients with Supratentorial Brain Tumors

Marcial Fraga, M.D.,* Pablo Rama-Maceiras, M.D.,* Sara Rodiño, M.D.,* Humberto Aymerich, M.D.,* Pilar Pose, M.D.,† Javier Belda, Ph.D.‡

Background: Desflurane is a volatile anesthetic agent with low solubility whose use in neurosurgery has been debated because of its effect on intracranial pressure and cerebral blood flow. The purpose of this study was to determine the variations on intracranial pressure (ICP) and cerebral perfusion pressure (CPP) as well as on cerebral arteriovenous oxygen content difference (AVDO_2) in normocapnic patients scheduled to undergo removal of supratentorial brain tumors with no evidence of mass effect during anesthesia with isoflurane or desflurane.

Methods: In 60 patients scheduled to undergo craniotomy and removal of supratentorial brain tumors with no evidence of midline shift, anesthesia was induced with intravenous fentanyl, thiopental, and vecuronium and was maintained with 60% nitrous oxide in oxygen. Patients were assigned to two groups randomized to receive 1 minimum alveolar concentration isoflurane or desflurane for 30 min. Heart rate, mean arterial pressure, intraparenchymal ICP, and CPP were monitored continuously. Before and after 30 min of continuous administration of the inhaled agents, AVDO_2 was calculated.

Results: There were no significant differences between groups in heart rate, mean arterial pressure, ICP, and CPP. ICP measurements throughout the study did not change within each group compared to baseline values. Mean arterial pressure decreased significantly in all patients compared to baseline values, changing from 105 ± 14 mmHg (mean ± SD) to 85 ± 10 mmHg in the isoflurane group and from 107 ± 11 mmHg to 86 ± 10 mmHg in the desflurane group ($P < 0.05$ in both groups). CPP also decreased within each group compared with baseline values, changing from 95 ± 15 mmHg to 74 ± 11 mmHg in the isoflurane group and from 95 ± 16 mmHg to 74 ± 10 mmHg in the desflurane group ($P < 0.05$ in both groups). Cerebral AVDO_2 decreased significantly in both groups throughout the study, changing from 2.35 ± 0.77 mM to 1.82 ± 0.61 mM (mmol/l) in the isoflurane group ($P < 0.05$) and from 2.23 ± 0.72 mM to 1.94 ± 0.76 mM in the desflurane group ($P < 0.05$), without differences between groups.

Conclusions: The results of this study indicate that there are no variations on ICP in normocapnic patients undergoing removal of supratentorial brain tumors without midline shift, as they were anesthetized with isoflurane or desflurane. CPP and cerebral AVDO_2 decreased with both agents.

ONE of the objectives in neurosurgical procedures is to achieve an early recovery after the anesthesia to facilitate the neurologic evaluation of the patients. Desflurane is a volatile anesthetic agent with lower solubility than isoflurane[1] and with faster recovery after prolonged anesthesia.[2,3] Therefore, it may be an attractive choice for this type of procedures, especially when its duration is prolonged. However, the use of desflurane in neurosurgery has been debated because of its theoretical capacity to promote cerebral vasodilatation, which can jeopardize cerebral hemodynamics as it increases intracranial pressure (ICP) or lumbar cerebrospinal fluid pressure (CSFP). This increment of the CSFP has been demonstrated both in animals[4–6] and in hypocapnic patients.[7] Some studies in normocapnic patients also seem to confirm this theory, but the observed effects on CSFP were common to isoflurane and desflurane.[8]

The monitoring of cerebral arteriovenous oxygen content difference (AVDO_2) has been used as a parameter of assessment in the relation between cerebral metabolic oxygen requirement (CMRO_2) and cerebral blood flow (CBF): AVDO_2 = CMRO_2/CBF.[9,10]

The aim of this study was to determine the variations in ICP, mean arterial pressure, and cerebral perfusion pressure (CPP) as well as to determine the AVDO_2 in normocapnic and normothermic patients without midline shift in the cranial computerized tomography (CT) scan who were scheduled to undergo removal of supratentorial mass lesions during anesthesia with isoflurane or desflurane.

Materials and Methods

After obtaining approval from the investigation committee of our institution (Complejo Hospitalario Juan Canalejo, A Coruña, Spain) and written informed consent, 60 patients with American Society of Anesthesiologists physical status II or III were studied. Patients who were aged between 18 and 70 yr, had supratentorial mass lesions, were scheduled for elective surgery (nonurgent), had 15 points on the Glasgow Coma Scale, and did not have a midline shift of greater than 5 mm on CT scan were included. Premenopausal women with positive pregnancy tests, patients with serious cardiovascular or respiratory diseases, obese patients (weight of over 25% of their recommended weight), those who con-

sumed drugs that might affect cerebral function, and those whose ICP at the moment of monitoring was higher than 20 mmHg were excluded from the study.

The patients were not premedicated. In the operating room, after electrocardiographic and pulse oximetry monitoring, a radial arterial cannula was placed to measure arterial blood pressure. Anesthesia was induced with intravenous fentanyl (3 μg/kg) and thiopental (5–7 mg/kg). Intravenous vecuronium (0.1 mg/kg) was administered to achieve muscle relaxation. After orotracheal intubation, anesthesia was maintained with 60% nitrous oxide in oxygen, with a fresh gas flow of 10 l/min. Ventilation was adjusted to maintain end-tidal carbon dioxide (ET_{CO_2}) between 33 and 36 mmHg, and normocapnia was confirmed with an arterial blood sample after stabilization of ET_{CO_2}. An oropharyngeal thermometer was placed to continuous temperature measurement.

Then, an intraparenchymatous cerebral transducer (OLM monitoring kit, Neurocare Group, Camino®; Staines, Middlesex, United Kingdom) was placed on the opposite side of the tumor to allow measurement of ICP. Arterial pressure and ICP transducer were set at zero at the level of the midcranium. The right internal jugular vein was entered between the two heads of the sternocleidomastoid muscle above the clavicle using an 18-gauge needle. Using the Seldinger technique, a 6-French percutaneous sheath introducer was placed in a retrograde fashion,[11] and a catheter for extraction of samples and determination of continuous jugular venous bulb oxygen saturation (Sj_{VO_2}) was placed *via* the sheath (Opticath®; Abbott, North Chicago, IL). The localization of the catheter was radiologically checked by means of a portable x-ray. The tip should be placed at the level of and just medial to the mastoid bone. The catheter was connected to a continuous pressurized flush system infusing heparinized saline solution at 3 ml/h. *In vivo* calibration of the oximeter (Abbott Oximetrix 3 SO_2/CO; Abbott Laboratories) was performed after signal stabilization. All painful procedures were performed after local infiltration of the punctured areas with 2% mepivacaine.

Heart rate and blood pressure were maintained within ±30% of the baseline value throughout the study by administering intravenous atropine (0.5 mg) in the case of bradycardia, intravenous ephedrine (0.1 mg/kg) in the case of hypotension, and intravenous fentanyl (1 μg/kg) in the case of tachycardia or hypertension as necessary. In the event of an increase in the ICP over 20 mmHg, a loading dose of intravenous mannitol (0.25 mg/kg) was administered. Patients requiring mannitol were excluded from the study.

The patients were randomly assigned to two groups, an isoflurane group (n = 30) and a desflurane group (n = 30), following a computer-generated chart, and they received the volatile agent at a concentration of 1 minimum alveolar concentration (MAC). The MAC of the isoflurane was defined as an expired fraction of 1.2

vol%,[12] and that of the desflurane was defined as an expired fraction of 6.0 vol%.[13] Desflurane was administered gradually (increments of 1% in the dial of the vaporizer every 20 s) to avoid a possible sympathetic response. Once the expired fraction corresponding to each agent was achieved, it was administered in a continuous and constant way throughout the 30 min of the study. End-tidal concentrations of carbon dioxide and volatile anesthetics were measured by means of the PM 8020® analyzer (Dräger Medizintechnik, Lübeck, Germany).

The recorded parameters in the patients were age, sex, weight, American Society of Anesthesiologists physical status, and required doses of fentanyl, atropine, ephedrine, and mannitol. Pharyngeal temperature, heart rate, ET_{CO_2}, mean arterial pressure, ICP, and CPP were registered before the beginning of administration of the volatile agent (T_{BAS}), once the expired concentration of 1 MAC of the agents was reached (T_0), and every 5 min throughout the 30-min study (T_5, T_{10}, T_{15}, T_{20}, T_{25}, T_{30}). CPP was calculated as mean arterial pressure minus ICP. The approximate time taken from anesthetic induction to the beginning of the study period was recorded.

For analysis, pharyngeal temperature, heart rate, ET_{CO_2}, mean arterial pressure, ICP, and CPP recorded every 15 s were reduced to 1-min values. Baseline values and subsequent measurements (T_0–T_{30}) of these continuous variables were defined as the median values observed during 1 min before each measurement.

At the same time as T_{BAS} and T_{30}, jugular blood samples were drawn from the catheter at a rate not greater than 2 ml/min to minimize contamination with blood from extracranial vessels, after discarding the first 5 ml, and were analyzed in less than 2 min. Arterial blood samples were drawn from the radial catheter and processed at the same time, bearing out normocapnia of patients throughout the study. All the samples were analyzed using an automated blood gas analyzer Ciba-Corning 865® (Ciba Corning Diagnostics Corporation, Medfield, MA). Afterwards, AVD_{O_2} was calculated, using the equation

$$AVD_{O_2} = [Hgb \times (Sa_{O_2} - Sjv_{O_2}) \times 1.39] +$$
$$[(Pa_{O_2} - Pjv_{O_2}) \times 0.0031],$$

where Hgb is the hemoglobin concentration (g/dl), Sa_{O_2} is the oxygen saturation of hemoglobin in the arterial blood, Sjv_{O_2} is the oxygen saturation of hemoglobin in the jugular bulb venous blood; 1.39 is the milliliters of oxygen capable to be transported in 1 g hemoglobin, Pa_{O_2} is the pressure of oxygen in arterial blood (mmHg), Pjv_{O_2} is the pressure of oxygen in jugular bulb venous blood (mmHg), and 0.0031 is the solubility coefficient for oxygen in plasma. We applied a correction factor of 0.446 in the results obtained in vol% to obtain the values of AVD_{O_2} in mM (μmol/ml).[9] The study was completed

Table 1. Demographic and Clinical Characteristics of Study Patients

	Isoflurane (n = 30)	Desflurane (n = 30)
Age, yr	60 ± 10	55 ± 15
Gender, male/female	16/14	17/13
Weight, kg	71 ± 11	70 ± 11
ASA physical status, II/III	22/8	23/7
Fentanyl, μg/kg	3.2 ± 0.7	3.1 ± 0.9
Patients requiring atropine, n	3	5
Patients requiring ephedrine, n	12	14
Patients requiring mannitol, n	0	0
Arterial CO_2 after anesthetic induction, mmHg	41 ± 3	40 ± 4
Arterial CO_2 T_{BAS}, mmHg	39 ± 3	39 ± 3
Arterial CO_2 T_{30}, mmHg	40 ± 2	39 ± 3

Values are mean ± SD or number of cases (n). Total n = 60.

ASA = American Society of Anesthesiologists ; T_{BAS} = value prior the 30-min study period; T_{30} = values after the 30-min study period.

Table 2. Intracranial Pressure

	Isoflurane Group (n = 30)	Desflurane Group (n = 30)
T_{BAS}	11 ± 4 (3–17)	11 ± 5 (2–18)
T_0	10 ± 4 (3–18)	11 ± 5 (2–18)
T_5	10 ± 5 (3–19)	11 ± 5 (2–17)
T_{10}	11 ± 4 (4–19)	11 ± 4 (2–18)
T_{15}	11 ± 4 (4–19)	10 ± 4 (2–19)
T_{20}	11 ± 4 (5–18)	11 ± 4 (3–19)
T_{25}	11 ± 4 (5–18)	11 ± 4 (4–20)
T_{30}	11 ± 5 (5–19)	11 ± 5 (4–19)

Values are mean ± SD (range in parentheses). Intracranial pressures measured in mmHg.

T_{BAS} = values (1 min median) prior the 30-min study period; T_0-T_{30} = values (1 min median) every 5 min during the administration of 1 minimum alveolar concentration (MAC) of the volatile agent.

with patients in the supine position, before the surgical procedure was started.

Statistical Analysis

From a clinical point of view, the detection of variations in ICP of 3 mmHg or more was considered particularly relevant. For this, assuming an SD of 5 mmHg in the ICP values (previous results not published), with a power of 90% and a level of $P < 0.05$, 30 patients per group were required. Statistical analysis was performed using a t test for comparisons between groups of the parametric variables and the Fisher exact test and chi-square test for nonparametric variables. Values for pharyngeal temperature, heart rate, $ETCO_2$, mean arterial pressure, ICP, CPP, and $AVDO_2$ were compared with baseline values in each group using repeated-measures analysis of variance testing. Data are expressed as mean ± SD or number of cases. $P < 0.05$ identified statistical significance.

Results

The time between anesthetic induction and the beginning of the study was 29 ± 9 min for the isoflurane group and 30 ± 10 min for the desflurane group ($P =$ not significant). There were no patients excluded because ICP was over 20 mmHg throughout the study.

There were no differences between groups demographically nor in the requirements of fentanyl, atropine, ephedrine, or mannitol (table 1). Four patients in isoflurane group and three in desflurane group required fentanyl. Ephedrine doses ranged from 6 to 15 mg in isoflurane group and from 5 to 15 mg in desflurane group.

Arterial carbon dioxide data collected after anesthetic induction, at T_{BAS} and T_{30}, remained at normocapnic levels (table 1). ICP (table 2), pharyngeal temperature (table 3), heart rate (table 3), and $ETCO_2$ (table 3) re-

mained stable throughout the study in both groups, and differences between groups were not observed.

Mean arterial pressure and CPP values decreased significantly 5 min after administration of the volatile agents (T_5) compared with baseline values (T_{BAS}) and remained decreased throughout the study, but there were no differences between groups for either parameter at any time (table 3).

The cerebral $AVDO_2$ decreased in both groups throughout the study, changing from 2.35 ± 0.77 mM to 1.82 ± 0.61 mM in the isoflurane group ($P < 0.05$) and from 2.23 ± 0.72 mM to 1.94 ± 0.76 mM in the desflurane group ($P < 0.05$) but without differences between groups.

Discussion

In this study, it was observed that the ICP in normocapnic patients with supratentorial mass lesions without midline shift in the cranial CT scan is not affected by the administration of 1 MAC desflurane compared with isoflurane. Changes in ICP are not observed either, after the administration of the volatile agents, compared to the values measured before administration. A decrease in CPP, due to a decrease of mean arterial pressure, was observed with both agents, as well as a decrease in the cerebral $AVDO_2$. Despite this decrease, cerebral $AVDO_2$ remained in the normal range (1.3–3.0 mM).[9]

Previous studies performed in animals in normocapnia and hypocapnia[4–6,14] have reported a double effect of desflurane and isoflurane at the cerebral level: on one hand, an increase in CBF if the systemic arterial pressure remains constant due to a direct cerebral vasodilatation that causes an increase in the flow, and on the other hand, a decrease in cerebral metabolic rate that involves a parallel decrease in CBF. The net result of these phenomena is translated into a moderate increment of CBF that can give rise to an increment in CSFP.

The administration of 1 MAC desflurane to hypocapnic patients with supratentorial mass lesions and mass effect

Table 3. Temperature Data, EtCO$_2$, Hemodynamic Data, and Cerebral Perfusion Pressure

	Temperature, °C		EtCO$_2$, mmHg		HR, beats/min		MAP, mmHg		CPP, mmHg	
	ISO	DES	ISO	DES	ISO	DES	ISO	DES	ISO	DES
T$_{BAS}$	36.3 ± 0.3	36.2 ± 0.4	35 ± 3	34 ± 2	67 ± 15	66 ± 11	105 ± 14	107 ± 11	95 ± 15	95 ± 16
T$_0$	36.2 ± 0.4	36.3 ± 0.5	35 ± 3	34 ± 2	64 ± 12	62 ± 8	92 ± 11	95 ± 12	82 ± 13	84 ± 14
T$_5$	36.1 ± 0.5	36.2 ± 0.5	35 ± 2	33 ± 1	65 ± 11	62 ± 9	86 ± 10*	89 ± 12†	76 ± 12*	76 ± 15†
T$_{10}$	36.1 ± 0.5	36.2 ± 0.4	34 ± 2	33 ± 1	65 ± 12	62 ± 9	84 ± 9*	86 ± 10†	74 ± 12*	74 ± 11†
T$_{15}$	36.1 ± 0.5	36.2 ± 0.4	34 ± 2	33 ± 1	65 ± 11	62 ± 10	84 ± 9*	86 ± 9†	74 ± 11*	76 ± 10†
T$_{20}$	36.1 ± 0.7	36.2 ± 0.4	34 ± 2	33 ± 1	66 ± 12	62 ± 9	85 ± 9*	87 ± 8†	74 ± 11*	75 ± 9†
T$_{25}$	36.1 ± 0.6	36.2 ± 0.4	34 ± 2	33 ± 1	66 ± 11	62 ± 9	85 ± 9*	86 ± 8†	75 ± 10*	74 ± 9†
T$_{30}$	36.1 ± 0.6	36.1 ± 0.7	34 ± 2	33 ± 1	66 ± 11	61 ± 8	85 ± 10*	86 ± 10†	74 ± 11*	74 ± 10†

Values are mean ± SD.

CPP = cerebral perfusion pressure; DES = desflurane group (n = 30); EtCO$_2$ = end-tidal carbon dioxide; HR = heart rate; ISO = isoflurane group (n = 30); MAP = mean arterial pressure; T$_{BAS}$ = values (1 min median) prior the 30-min study period; T$_0$-T$_{30}$ = values (1 min median) every 5 min during the administration of 1 minimum alveolar concentration (MAC) of the volatile agent.

* $P < 0.05$ *versus* T$_{BAS}$ in isoflurane group.　† $P < 0.05$ *versus* T$_{BAS}$ in desflurane group.

produced a sharp increase of CSFP in comparison to another group of patients who were given isoflurane.[7] The authors explained that this was due to a vasodilator effect of desflurane with respect to isoflurane and to increases of CBF and cerebral blood volume with the first of the agents, bearing out the results of previous studies in animals. However, another study in which CBF was measured directly by means of ^{133}Xe injection did not demonstrate differences between isoflurane and desflurane in hypocapnic patients, leading to the conclusion that both agents lacked cerebral vascular adverse effects.[15] In the study of Muzzi *et al.*,[7] lumbar CSFP continued to increase throughout the study, suggesting a possible affectation in the mechanisms of production and absorption of the cerebrospinal fluid (similar to that observed with enflurane), which has not been confirmed yet.

Talke *et al.*[8,16] observed an increase in lumbar CSFP in normocapnic patients without cerebral mass effect who received 0.5 and 1 MAC isoflurane, desflurane, and sevoflurane associated with propofol, in comparison to a control group that only received propofol. In the same studies, lumbar CSFP in all the volatile anesthetic groups increased throughout the study compared with baseline values but not in the propofol control group. The authors did not compare the volatile agents among themselves, and although they did not evaluate CBF, they also attributed the results to a cerebral vasodilator effect of all the inhaled agents.

In our study, we did not observe changes of the ICP between groups, or within each group compared with baseline values. The explanation for these differences regarding the studies mentioned before was probably methodological. The study of Muzzi *et al.*[7] was performed in cases of hypocapnia in patients with cerebral lesions with serious mass effect, allowing surgical stimulation during the measurements; thus, the results are difficult to compare with these. As for the studies of Talke *et al.*,[8,16] the methodology is closely related to that

of our study, except for the measurement of cerebral pressure (CSFP in their study and ICP in ours) and the inclusion of propofol and nitrous oxide as a maintenance anesthetic in all the groups in their studies and only nitrous oxide in ours.

Another possible explanation for the differences that we observed among all these studies could be a different effect of the volatile anesthetics, depending on the patient's cerebral situation. Those with worse cerebral elastance due to the lesion they presented would be more prone to develop changes in CBF and ICP in response to the administration of inhaled agents with a theoretically vasodilatory effect, like desflurane, just as they would not respond as well to other noxious agents.

In the same way, patients with a theoretically maintained cerebral elastance, such as those in the current study, would preserve their capacity to maintain a constant CBF and ICP when these agents were administered. In fact, it seems that both isoflurane and desflurane, at the concentrations usually used in clinical practice, preserve the reactivity to carbon dioxide and to flow–metabolism coupling.[15,17]

In our study, we did not measure CBF in a direct way, but we calculated cerebral AVDo$_2$ before and 30 min after administration of the volatile agents. Cerebral AVDo$_2$ relates the cerebral metabolic consumption of oxygen and CBF (AVDo$_2$ = CMRo$_2$/CBF),[18] and it remained within normal values with both agents. This result could be interpreted as a possible preservation of the flow–metabolism coupling with both desflurane and isoflurane.[9] The decrease in cerebral AVDo$_2$ with both anesthetics could be attributed to a certain prevalence of the increase in the cerebral flow, to a reduction of the cerebral metabolism, or to both. In any case, and still assuming an increase in the CBF, this was not reflected in an increase of ICP.

There are some questions requiring additional comments in our study. The use of opioids in the anesthetic induction could interfere with the transient sympathetic

discharge induced by desflurane, which would stimulate the cerebral metabolism and favor vasodilatation and the increase of CBF and ICP.[19,20] The doses of fentanyl used in this study were very low to suppress that possible response to desflurane, had it been produced, and they represent the usual clinical practice. In addition, fentanyl seems to affect ICP and cerebral perfusion less than other opioids in patients with supratentorial mass lesions.[21,22] In the study, all the patients requiring additional fentanyl doses needed it between T_{BAS} and the first 5 min after the addition of isoflurane or desflurane. Even if these requirements of additional fentanyl could be interpreted as a signal of light anesthesia or sympathetic response in the case of desflurane, and if we had excluded them, the results would not have changed.

The use of nitrous oxide in these patients could be questioned in the same way. Nitrous oxide was used for anesthetic maintenance during the placing of the ICP transducer and jugular cannulation and to allow baseline measurements before the administration of desflurane or isoflurane. We selected this anesthetic as in previous reports,[8,16] avoiding other drugs usually used, such as propofol, to prevent more pronounced hemodynamic changes. The presence of a certain degree of cerebral vasodilatation with nitrous oxide is possible,[8] but this effect is compensated by the administration of this agent at the same concentration in both groups.

As shown in table 1, the study was performed in normocapnia because the patients had no indications of hyperventilation. Normocapnia was confirmed by arterial carbon dioxide blood sample analysis after stabilization of ET_{CO_2} following anesthetic induction, at T_{BAS} and T_{30}. We maintained an ET_{CO_2} between 32 and 36 mmHg throughout the study. These values were taken as references because previous studies reported a difference between Pa_{CO_2} and ET_{CO_2} in neurosurgical procedures of approximately 7 mmHg.[23] In our study, we observed a difference between Pa_{CO_2} and ET_{CO_2} of 6 ± 3 mmHg. No one patient presented a Pa_{CO_2} less than 35 mmHg or higher than 45 mmHg in arterial blood samples obtained at the beginning and the end of the study period.

Vasoactive drugs in the study and their possible effects on CBF call for a commentary. Atropine was used in few patients, and in all cases, heart rate at the moment of administration was lower than 50 beats/min. We selected ephedrine sulfate for hemodynamic maintenance to avoid a great impairment of mean arterial pressure and CPP, undesirable for the pathology of the patients included in the study. Ephedrine was chosen because although it passes the blood–brain barrier, no direct effect of this drug on CBF has been proven,[24] especially at low doses as were used in this study. Published studies about the effects of inhaled anesthetics on CBF or ICP included a drug to maintain blood pressure in "normal" ranges, which was usually phenylephrine[7,8,16] or ephed-

rine.[25] We did not have available phenylephrine at our institution at the beginning of the study.

As a critique of our method, we analyzed a short period of time in the study, which may have made us underestimate a possible late effect of the studied anesthetics on the production and reabsorption of the cerebrospinal fluid.[7] However, the study was performed to reproduce clinical circumstances previous to surgical stimulation. Prolonging the measurements in time would delay the surgical procedure and the exposure of patients to the anesthetics, circumstances that would be questionable from an ethical point of view.

The measurement of ICP in all patients was performed on the opposite side of the mass lesion. ICP monitoring, instead of lumbar CSFP monitoring, is the usual clinical practice in our institution. The existence of an interhemispheric pressure gradient was possible, and the obtained values could have underestimated the real ICP. However, monitoring CSFP or ICP on the same side of the mass lesion would not guarantee the absence of these gradients,[9,26] and the latter could have interfered with the surgical procedure.

We did not perform direct measurements of CBF. Unfortunately, these were not within our reach because of their complexity and cost. However, the assessment of variations in CBF starting with AVD_{O_2} is perhaps the most frequently used method at the patient's bedside.[9,10,16]

It can be concluded that in normocapnic patients with supratentorial tumoral pathology without midline shift on the CT scan, administration of 1 MAC isoflurane or desflurane decreases CPP because of a decrease in mean arterial pressure. It does not increase ICP, and it produces a decrease, within normal values, of cerebral AVD_{O_2} that could be interpreted, together with the stability observed in ICP, as a possible preservation of the flow–metabolism coupling for these agents, at the concentrations used in this study.

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