Total Intravenous Anesthesia with Target-Controlled Infusion of Remifentanil and Propofol for Ablation of Atrial Fibrillation

Fernando Squeff Nora, TSA 1, Maurício Pimentel 2, Leandro Ioschpe Zimerman 3, Eduardo B. Saad 4

INTRODUCTION

Ablation of atrial fibrillation (AF) is a relatively new procedure among us, although it is common in other centers. The choice of anesthesia, monitors, and anesthetic care for this procedure performed outside the surgical center has not been described. The objective of this report was to describe an anesthesia technique for ablation of AF as well as to present a brief review on the safety of the anesthetics used since some of them can change electrical conduction in the normal heart and anomalous conduction pathways, hindering the procedure and the evaluation of its efficacy.

CASE REPORT

This is a 49-year old female weighing 73 kg, height 155 cm, and ASA II due to hypertension. The patient was premedicated with oral midazolam 15 mg that was also used for sedation during the transesophageal echocardiogram to confirm the absence of intracavitary thrombi. In the hemodynamics room, a 16G Teflon catheter was used for peripheral venoclysis under local anesthesia with 1% lidocaine without epinephrine, and a 20G catheter was used for arterial puncture for invasive blood pressure determinations (MAP). Monitoring consisted of a 12-lead electrocardiogram, pulse oximetry, heart rate, bispectral electroencephalography to measure BIS, suppression rate (SR), and SEF 95, and MAP. Anesthesia was induced with intravenous target-controlled infusion (TCI) of propofol with a target of 4 µg.mL -1, intravenous IAC of remifentanil with a target of 3 ng.mL -1, and intravenous bolus of rocuronium 0.2 mg.kg -1. Shortly before induction, the patient was oxygenated, intubated, and placed on volume controlled mechanical ventilation to maintain $P_{a CO_2}$ between 30 and 35 mm Hg. Verbal contact was maintained during induction to detect the moment of loss of verbal response. Verbal response was lost when the concentration of propofol at the effector site reached 0.7 µg.mL -1 and of remifentanil 2 ng.mL -1. The bispectral index at this moment was 70 and SEF95 = 22, and the patient was ventilated with a face mask until further reduction of BIS was detected. Tracheal intubation was performed when the concentration of propofol at the effector site reached 2.2 µg.mL -1, BIS = 30, and SEF = 12. After induction, a duodecapolar catheter was inserted in the right internal jugular vein and placed inside the coronary sinus and the recording poles at the junction of the right atrium and superior vena cava. The right femoral vein was punctured twice for access of two sheaths for transeptal puncture. An intracardiac ultrasound probe was placed in the right atrium through the left femoral vein. The decapolar circular catheter (Lasso) and the ablation catheter were placed in the left atrium through two intracardiac echocardiogram-guided transeptal punctures. Systemic heparinization associated with monitoring of the activated coagulation time (ACT > 300 sec.) was instituted. The Lasso catheter was used for electrical mapping of the pulmonary veins. The pulmonary veins were disconnected from the left atrium by applying 30-watt radiofrequency with an irrigated catheter. Target-controlled infusion of propofol and remifentanil using infusion pumps with pharmacokinetic systems for each of the drugs was administered. We used the pharmacokinetic model described by Marsh for propofol, which was incorporated into the propofol PFS® pump. The pharmacokinetic model described by Minto for remifentanil was used and incorporated into the Alaris Pk® infusion pump. The concentration at the effector site, or biophase, corresponded to the information obtained through the infusion pumps and represented the predictive concentrations of both drugs at their sites of action. The concentrations of propofol and remifentanil were regulated according to BIS and MAP, respectively. Anesthesia was maintained with TCI of propofol at adequate concentrations to maintain BIS between 40 and 50. The mean arterial pressure and HR remained within normal limits during the procedure. The maximal and minimal concentrations of propofol at the effector site were 2.5 and 1.9 µg.mL -1, respectively. Remifentanil was titrated to maintain a maximal 20% variation in MAP. The maximal and minimal concentrations of remifentanil at the effector site were 4 and 2 ng.mL -1, respectively. The total propofol and remifentanil infusion time was 300 minutes. A mean infusion rate of 4.6 mg.kg -1.h -1 of propofol was used, while that of remifentanil was 0.1 µg.kg -1.h -1. Total doses of propofol of 1,697 mg and remifentanil of 2 mg were used. The initial MAP was 80 mmHg and it was maintained between 65 and 80 mmHg during the procedure with the administration of intravenous boluses of vasopressors in only two occasions. An esophageal thermometer was placed to determine the esophageal temperature when radiofrequency was applied close to the esophagus. The infusions of remifentanil and propofol were discontinued at the end of the procedure. The patient was extubated when the concentration of propofol at the effector site reached 1.2 µg.mL -1 and she was on sinus rhythm, and with stable vital signs and spontaneous ventilation after reversal of the neuromuscular blockade. The patient was then transferred to the post anesthetic care unit.

DISCUSSION

Ablation of intracardiac anomalous pathways is a relatively common procedure among us. It is more commonly performed under local anesthesia associated with several se-
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...such as the nature of the procedure, patient-related characteristics, premedication, and use of TCI. Propofol and remifentanil were chosen because these agents do not seem to induce changes in the results of intracardiac mapping and can constitute a good choice for the proposed procedure. As for monitoring, the use of BIS was important because it allowed adequate and safe titration of propofol, and it is possible that this monitor was important in determining a reduction in the infusion doses of propofol to rates below 6 mg.kg⁻¹.h⁻¹. Similarly, the use of TCI of intravenous drugs allowed more judicious titration through constant changes in plasma concentration according to the variations of BIS and MAP, since monitoring of the HR is not reliable due to the nature of the procedure. Patient-related characteristics, as well as premedication with midazolam, might have contributed for the low doses of anesthetics, making it impossible to state that BIS alone was responsible for this. It is important to emphasize the need of postoperative analgesia since pain severity associated with this procedure varies, and it can be initially high due to pericardial irritation. Ablation of AF is performed outside of the surgical center and can be safely conducted under total intravenous anesthesia and tracheal intubation. More conclusive publications on the safest anesthesia technique in this situation are rare, although most have mentioned the importance of using drugs that do not change the electrophysiology of the cardiac conduction tissue and accessory pathways involved in the maintenance of AF. Any drugs that change the electrophysiological expression of accessory pathways might interfere with the localization and subsequent assessment of the ablation results. Target-controlled infusion of intravenous drugs monitored with BIS and MAP facilitated maintenance of satisfactory anesthetic plane and influenced, among other aspects, the doses used, which might reduce costs. Target-controlled infusion in total intravenous anesthesia for ablation of AF can become an attractive alternative since it allows fast awakening that facilitates immediate neurologic evaluation. The publication of more encompassing clinical assays may or may not ratify this anesthetic choice.

REFERÊNCIAS - REFERENCES


06. Sharpe MD, Dobkowski WB, Murkin JM et al. - Propofol has no direct effect on sinoatrial node function or on normal atrioventricular and accessory pathway conduction in Wolff-Parkinson-White syndrome during alfentanil/midazolam anesthesia. Anesthesiology, 1995;82:888-895.


RESUMEN

Nora FS, Pimentel M, Zimmerman LI, Saad EB - Anestesia Venosa Total con Infusión Objetivo-Controlada de Remifentanil y Propofol para Ablación de la Fibrilación Atrial.

JUSTIFICATIVA Y OBJETIVOS: La ablación de fibrilación atrial (FA) es un procedimiento nuevo en nuestro medio, aunque sea común en otros centros. No han sido descritos la elección de la anestesia, los monitores y los cuidados anestesiológicos para ese procedimiento realizado fuera del bloque quirúrgico. El objetivo de este relato fue describir una técnica de anestesia para la realización de la ablación de FA.

RELATO DEL CASO: Paciente femenina, 49 años, 73 kg, 155 cm, ASA II por hipertensión arterial sistémica. El monitoreo tuvo un electrocardiograma con 12 derivaciones, oximetría de pulso, frecuencia cardíaca, electroencefalografía bispectral para medidas de BIS, tasa de supresión (SR) y SEF95, y presión arterial promedio (PAM). La inducción anestésica fue realizada con propofol por vía venosa, en infusión objeto-controlada (IAC), con objeto regulado en 4 µg.ml⁻¹, remifentanil por vía venosa, en IAC, con objeto de 3 ng.ml⁻¹, y rocuronio por vía venosa en bolos con dosis de 0,2 mg.kg⁻¹. El modelo farmacocinético de propofol utilizado fue el descrito por Marsh y fue incorporado a la bomba de propofol PFS®. El modelo farmacocinético de remifentanil utilizado fue el descrito por Minto y fue incorporado a la bomba de infusión Alaris PK®. Las concentraciones en el local efecto o biofase, correspondieron a informaciones obtenidas a través de las bombas de infusión, y representaron medidas de predicción de las concentraciones de los dos fármacos en los respectivos locales de acción. Las concentraciones de propofol y de remifentanil se regularon a tono con el BIS y la PAM, respectivamente.

CONCLUSIONES: La anestesia venosa total para la ablación de FA, puede ser una opción segura si tenemos en cuenta que no hay alteración de la electrofisiología de las vías accesoria. La literatura es escasa en ese sentido, y nuevas publicaciones podrán o no justificar esa modalidad de anestesia durante la ablación de FA.