Deep sedation with dexmedetomidine administered by electrophysiologists during COVID-19 pandemic compared with propofol administered by anesthesiologists for transcatheter ablation of atrial fibrillation

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Abstract

Background COVID-19 pandemic, limiting the availability of anesthesiologists, has impacted heavily on the organization of invasive cardiac procedures such as transcatheter atrial fibrillation (AF) ablation. Objective We compared the safety and efficacy of deep sedation with dexmedetomidine administered by electrophysiologists without anesthesiologist supervision, against the standard protocol performed with propofol. Methods We retrospectively included all AF ablation procedures performed in 2020: 23 patients sedated with 1% propofol (2 ml bolus followed by infusion starting at 1 mg/Kg/h), 26 patients with dexmedetomidine (infusion starting at 0.7 mcg/Kg/h). Both groups additionally received 1 mcg/Kg of midazolam as a single bolus and 0.05 mg single boluses of fentanyl prior to ablation on each pair of pulmonary veins (PV). Primary outcomes were oxygen desaturation (<90%) or need for assisted ventilation/intubation, bradycardia (heart rate <45 bpm) and persistent hypotension (systolic blood pressure <90 mmHg). Results Baseline characteristics and hemodynamic variables did not differ between the two groups (all p>0.05). In 8/23 (35%) patients propofol infusion velocity reduction was necessary to maintain the hemodynamic values, compared to 7/26 (27%) with dexmedetomidine. Inter-group comparison of hemodynamic variables during the procedure showed no statistically significant difference, despite a trend in favor of dexmedetomidine (3 respiratory depressions and 3 persistent hypotension episodes with propofol vs. 0 with dexmedetomidine; p = 0.057). Conclusion Deep sedation with dexmedetomidine administered by electrophysiologists without anesthesiologist supervision is safe and effective for AF transcatheter ablation. A trend towards a lower incidence of hypotension and respiratory depression was noted when compared to propofol.

Title page

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Abstract

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COVID-19 pandemic, limiting the availability of anesthesiologists, has impacted heavily on the organization of invasive cardiac procedures such as transcatheter atrial fibrillation (AF) ablation.

Objective

We compared the safety and efficacy of deep sedation with dexmedetomidine administered by electrophysiologists without anesthesiologist supervision, against the standard protocol performed with propofol.

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We retrospectively included all AF ablation procedures performed in 2020: 23 patients sedated with 1% propofol (2 ml bolus followed by infusion starting at 1 mg/Kg/h), 26 patients with dexmedetomidine (infusion starting at 0.7 mcg/Kg/h). Both groups additionally received 1 mcg/Kg of midazolam as a single bolus and 0.05 mg single boluses of fentanyl prior to ablation on each pair of pulmonary veins (PV). Primary outcomes were oxygen desaturation (<90%) or need for assisted ventilation/intubation, bradycardia (heart rate <45 bpm) and persistent hypotension (systolic blood pressure <90 mmHg).

Results

Baseline characteristics and hemodynamic variables did not differ between the two groups (all $p>0.05$). In 8/23 (35%) patients propofol infusion velocity reduction was necessary to maintain the hemodynamic values, compared to 7/26 (27%) with dexmedetomidine.

Inter-group comparison of hemodynamic variables during the procedure showed no statistically significant difference, despite a trend in favor of dexmedetomidine (3 respiratory depressions and 3 persistent hypotension episodes with propofol vs. 0 with dexmedetomidine; $p = 0.057$).

Conclusion

Deep sedation with dexmedetomidine administered by electrophysiologists without anesthesiologist supervision is safe and effective for AF transcatheter ablation. A trend towards a lower incidence of hypotension and respiratory depression was noted when compared to propofol.

Keywords

COVID-19; atrial fibrillation; ablation; deep sedation; dexmedetomidine; propofol.

Introduction

The current Coronavirus disease (COVID-19) pandemic has brought many medical and organizational challenges to every healthcare system, creating a shortage of critical care and anesthesia staff. Anesthesiologists are playing a fundamental role in the fight against COVID-19 and the majority of them have been reassigned from the perioperative setting to intensive care units.

While many elective procedures have been postponed or suspended during the pandemic, many urgent or semi-urgent interventional cardiology procedures were overall preserved.

It is now well known that arrhythmias are frequently associated with COVID-19 increased morbidity and mortality, with atrial fibrillation (AF) being the most frequent.[1] Transcatheter ablation has become the most common technique to treat patients.[2] However, AF ablation is often a relatively long procedure and usually requires sedation to avoid patient's discomfort and movements. Therefore, many centers in the United States and in Europe perform AF ablation procedures under general anesthesia (GA). Nonetheless, deep sedation, with an adequate level of analgesia, has become a safe and effective alternative, avoiding the risks related to GA.[3-5] Moreover, if the sedation protocol is overseen by cardiologists/electrophysiologists, anesthesiologists are free to be redeployed from the operating rooms to the care of COVID-19. Many combinations of drugs have been used for sedation during catheter ablation of AF. While propofol administration has shown to achieve a better and more predictable level of sedation than benzodiazepine and opiates, adverse sedative effects might still be present.[6] Dexmedetomidine, a highly selective alpha2-adrenoreceptor agonist, offers effective sedation and analgesia combined with the unique characteristic to cause no respiratory depression.[7-8]

The aim of our study was to compare the safety and efficacy of dexmedetomidine administered by electrophysiologists with those of propofol operated by anesthesiologists in patients who underwent transcatheter AF ablation.

Methods

Patient inclusion

This was a retrospective study conducted at the Maria Vittoria Hospital in Turin, Italy, during the pandemic of COVID-19 and was approved by the institutional review board. Informed written consent to sedation and ablation was obtained from all patients.

All consecutive patients with paroxysmal or persistent AF who underwent catheter ablation between January 1st, 2020 and December 31st, 2020, either at their first or second ablation procedure, were considered eligible for the study and were included in the analysis; there was no exclusion criteria for enrollment other than guideline-directed clinical contraindication to catheter ablation.[2]

Sedation protocol

In our center all AF ablation procedures are usually performed under conscious sedation; GA is not routinely used. During the first part of the year, AF ablation procedures were performed using propofol, which was administered and monitored by an anesthesiologist in the operating room (group 1). Induction of sedation was performed with a bolus of 2 ml of 1% propofol followed by an infusion starting at 1 mg/Kg/h and then titrated to the clinical response.

After the unexpected COVID-19 pandemic arrival, due to organizational challenges and given the shortage of available anesthesiologists, sedation was performed by the electrophysiology (EP) team (consisting of two operating electrophysiologists for each procedure, at least two dedicated nurses and a radiology technician) using dexmedetomidine (group 2) without anesthesiologist supervision, according to a shared protocol approved in advance. The EP medical and nursing staff were trained and certified in advanced life support. An on-call anesthesiologist was available from within the hospital in case of emergency.

In both groups, patient's monitoring started in the EP laboratory waiting room: non-invasive blood pressure, heart rate and rhythm and oxygen saturation were continuously recorded. End-tidal carbon dioxide ($etCO_2$) monitoring was used to detect early respiratory dysfunction. In the EP laboratory, a 12-lead electrocardiogram (ECG) was continuously recorded for every patient; respiratory rate was also assessed. Continuous infusion of dexmedetomidine was started at a rate of 0.7 mcg/Kg/h before venipuncture and titrated to clinical response during the procedure (maximum dosage 1.4 mcg/Kg/h) in order to obtain patient's sedation with cessation of body movements. In case of adverse events, dexmedetomidine infusion rate was reduced if deemed necessary. Oxygen supplementation was administered by nasal cannulas at 2 l/min at baseline in both groups.

At the beginning of the procedure every patient (either in the propofol or in the dexmedetomidine group)

received 1 mcg/Kg of midazolam as a single bolus. Moreover, a 0.05 mg single bolus of fentanyl was administered for analgesia prior to radiofrequency application on the left pulmonary veins (PVs), and the bolus was repeated before starting ablation on the right PVs. If the patient was in AF when entering the EP laboratory, electrical cardioversion (EC) was performed at the beginning of the procedure with an additional bolus of propofol 1 mg/Kg in both groups. In case of EC, the starting infusion rate in group 1 was reduced to 0.8 mg/Kg/h without any other bolus.

Vital signs were monitored throughout the procedure: peripheral oxygen saturation, respiratory rate, heart rate and rhythm were monitored continuously, while blood pressure was monitored with a brachial cuff at 3-minute intervals.

Adverse events such as oxygen desaturation ($< 90\%$), bradycardia (heart rate < 45 bpm) and hypotension (systolic blood pressure < 90 mmHg) were recorded. In case of airway compromise, the head tilt-chin lift maneuver was performed; in case of desaturation oxygen delivery was increased and/or the nasal cannula was changed to a facial mask with increased oxygen delivery capability with/without insertion of an oropharyngeal airway; in case of bradycardia, cardiac stimulation was performed via the catheters placed in the heart or by atropine administration; in case of significant hypotension, saline loading was performed with simultaneous check for mechanical complications by means of intracardiac echocardiography (ICE), routinely used during AF ablation procedures at our Institution.

Catheter ablation

All patients entered the EP laboratory after fasting for at least 8 hours and after a transesophageal echocardiography performed the day prior to the procedure had excluded the presence of left atrial thrombi. Right and left femoral veins were cannulated. A steerable catheter (Inquiry, Abbott, St. Paul, MN, USA) was positioned inside the coronary sinus. The ICE catheter (ViewFlex Xtra, Abbott, St. Paul, MN, USA) was positioned in the right atrium. The transeptal puncture was performed under radioscopic and ICE guidance. A multipolar mapping catheter (Advisor HD Grid, Abbott, St. Paul, MN, USA) was used for mapping. A 3.5 mm irrigated-tip radiofrequency ablation catheter (TactiCath, Abbott, St. Paul, MN, USA) was used to perform ablation. The NavX 3D electroanatomic mapping (EAM) system (Abbott, St. Paul, MN, USA) was used in every procedure to guide catheter ablation and reduce fluoroscopy use. PV isolation with wide antral circumferential approach was performed for every de novo ablation procedure. For redo procedures, conduction gaps were identified and the PV re-isolated. A vein was defined as isolated when PV potentials disappeared/were dissociated from the left atrium and exit block from the vein was demonstrated. Heparin was given to maintain an activated clotting time of 250–300 s throughout the procedure.

Endpoint

The primary outcome was the presence of adverse sedative effects that required cessation of the anesthetic drug: oxygen desaturation ($< 90\%$) or need for assisted ventilation/intubation, bradycardia (heart rate < 45 bpm) and hypotension (systolic blood pressure < 90 mmHg) requiring fluid administration. Secondary endpoints were procedural success and procedural and fluoroscopy time. The presence of any other complication was also recorded.

Statistical analysis

Categorical variables are expressed as frequencies (%), continuous variables are presented as means $\pm$ standard deviation (SD). Student's t-test was used to compare continuous variables, and chi-square test was used to compare categorical variables. A level of significance of 0.05 was used. All statistical analyses were conducted using SPSS version 20 (SPSS, Inc, Chicago, IL, USA).

Results

Forty-nine consecutive patients who underwent AF ablation were included in the analysis. Twenty-three belonged to the propofol group (group 1) and twenty-six to the dexmedetomidine group (group 2). Patient characteristics were similar between the two groups (Table 1). Eleven out of 23 patients (48%) had paroxysmal

AF in group 1 compared to 16/26 (61%) in group 2. Either patients with paroxysmal or persistent AF underwent PVI; 7 patients in the propofol group underwent EC at the beginning of the procedure compared to 11 patients in the dexmedetomidine group.

Baseline hemodynamic variables, including arterial blood pressure, heart rate and oxygen saturation were not significantly different between groups (Table 2).

Drug administration data are reported in Table 3. During the procedure, in 8 patients out of 23 (35%) in group 1, a reduction of the propofol infusion rate was necessary; 3 of them underwent EC at the beginning of the procedure. In group 2, 7 patients required dexmedetomidine infusion rate reduction during the procedure (27%); however, 5/7 were patients who underwent EC at the beginning of the procedure for which propofol had been used in combination.

Inter-group comparison of hemodynamic variables during the procedure showed that there was no statistically significant difference between the two groups (Tables 2 and 4), despite a trend in favor of dexmedetomidine in terms of better blood pressure control and better oxygen saturation. Indeed, persistent hypotension (failure to maintain a systolic blood pressure > 90 mmHg) at a drug infusion rate required to achieve adequate sedation was observed in 3 patients in the propofol group and in none in the dexmedetomidine group ($p = 0.057$). It resolved with propofol infusion rate reduction and fluid challenge. Three patients in group 1 experienced respiratory depression (sustained oxygen desaturation $< 90\%$) compared to 0 patients in group 2 ($p = 0.057$). All but one resolved with temporary propofol cessation, whereas in 1 patient few minutes of bag-and-mask ventilation was necessary. Two patients out of 26 in group 2 experienced a transient reduction in the heart rate during ablation (< 45 bpm), which promptly resolved with pacing from the catheters inside the heart.

There was no other serious complication related to the ablation procedure (no death, stroke, phrenic nerve damage, atrio-esophageal fistula, pericardial tamponade or effusion, vascular complication, etc.).

Complete PVI was successfully achieved in every patient. Mean procedural time was 138.5 ± 36.6 minutes in group 1 and 154 ± 43.3 minutes in group 2 ($p = 0.184$). Mean fluoroscopy time was 10.7 ± 6.8 minutes in group 1 and 10.1 ± 7.4 minutes in group 2 ($p = 0.747$).

After the procedure, patients were observed in the recovery unit for 60 minutes and full recovery was obtained in all patients.

Discussion

Catheter ablation has become the first-line therapy for the treatment of AF.[2] During the procedure, pain may result from catheter insertion or the ablation delivery itself.[9]Moreover, it is usually a quite lengthy procedure requiring the patient to lie perfectly still to avoid complications and technical difficulties such as EAM shifts or acquisition errors. Therefore, GA or deep sedation are generally recommended, usually at the operator's preference. In recent years, deep sedation has showed to be a very viable alternative to GA, reaching a similar efficacy level and being characterized by a lower rate of potential drawbacks (less phrenic nerve or esophageal injury, lower need for inotropic drugs during the procedure, presence of patient's feedback, etc.).[10-11] Moreover, GA has higher costs and requires more planning and organization in the operating room.

Regarding deep sedation, many protocols have been tested over the years.[3-5]

Benzodiazepines such as midazolam have been used in repeated boluses. However, they lack a proper analgesic effect, and their main disadvantage is the waxing and waning level of sedation/consciousness which can jeopardize the success and the safety of the ablation procedure. To maintain a longer and steadier level of sedation, propofol has been used, especially through a continuous infusion.[4,12] For AF ablation, propofol has been tested against a combination of midazolam and fentanyl by Tang et al.[13] and showed to be associated with an increased risk of persistent oxygen desaturation reflecting in lower catheter stability due to airway obstruction, despite achieving a deeper level of sedation. Furthermore, hypotension is a common side effect

of propofol, mainly due to a reduction in systemic vascular resistance and a negative inotropic effect. This is why anesthesia support or back-up is usually necessary when propofol is used. In two large observational prospective studies, propofol has been used as the drug of choice for sedation for AF ablation without anesthesiologist supervision.[4,16]Hypoxia and hypotension were present in a percentage ranging from 1.5 to 2.3% in one study,[16] whereas 15.6% of patients in the other study required switching from propofol to midazolam due to persistent hypotension or respiratory depression.[4]

In our center, we historically performed AF ablation procedures with propofol and anesthesiologist supervision. In the first part of the year 2020, before COVID-19 breakthrough, all procedures were performed using propofol and we had a 13% of either persistent hypotension or respiratory depression, which luckily resolved without the need for intubation or advanced life support maneuvers. After the pandemic arrival, we were forced to manage AF ablation patients without the anesthesiology team, which was redeployed in order to deal with the COVID-19 emergency.

Dexmedetomidine was seen as a viable alternative to propofol. Dexmedetomidine is a selective alpha2-adrenoreceptor agonist characterized by anxiolytic, sedative and analgesic effects with minimal risk of respiratory depression,[15-18] therefore easier to be managed by electrophysiologists. It has been safely used in combination with other drugs to achieve deep sedation.[19-22] In the management of sedation for AF ablation, dexmedetomidine has been evaluated in two randomized controlled trials. The first one randomized dexmedetomidine and remifentanil versus midazolam and remifentanil.[23]Dexmedetomidine was associated with a deeper level of sedation but a lower incidence of respiratory depression; there was a non-significant trend towards a higher rate of hypotension and transient bradycardia. The second trial compared dexmedetomidine to thiamylal, a barbiturate, reporting fewer body movements and apneic events and a similar incidence of bradycardia and hypotension.[24]

To our knowledge, our study is the first comparison between dexmedetomidine and propofol in patients undergoing AF ablation. Despite not reaching statistical significance, likely due to the small sample size of our population, we observed a trend in favor of dexmedetomidine in terms of less hypotensive and hypoxic episodes. On the other hand, dexmedetomidine was characterized by a slightly higher number of bradycardia episodes compared to propofol (2 versus 0). They both happened during ablation of the ganglionated plexi in the left atrium and promptly resolved with pacing from the catheters inside the heart.

Procedural time did not statistically differ between the two groups. Complete PVI was successfully achieved in every patient, there were no procedure-related complications and every patient fully recovered from deep sedation.

Limitations

This is a retrospective study, with all the limits related to this type of design.

The small sample size of our population likely prevented reaching statistically significant results. This was due to the fact that, during the COVID-19 pandemic, unlike a "normal" year, the majority of elective AF ablation procedures have been canceled to allow the conversion of our Cardiology ward to a COVID-19 ward, with the deployment of cardiologists and electrophysiologists to the care of COVID-19. However, we believe that our results show that the use of dexmedetomidine without anesthesiologist support is feasible and safe, maybe safer than propofol. Our data need confirmation in a larger study.

Conclusions

Dexmedetomidine infusion administered by electrophysiologists without anesthesiologist supervision is a safe and effective deep sedation drug for catheter ablation for AF. A trend towards a lower incidence of hypotension and respiratory depression was noted when compared to propofol infusion.

References

1. Kwenandar F, Valeriani Japar K, Damay V, Hariyanto TI, Tanaka M, Pratama Hardjo Lugito N, Kurniawan A. Coronavirus disease 2019 and cardiovascular system: A narrative review. Int J Cardiol

Heart Vasc 2020 Jun 3; doi: 10.1016/j.ijcha.2020.100557.

2. Hindricks G, Potpara T, Dagres N, Arbelo E, Bax JJ, Blomstrom-Lundqvist C, Boriani G, Castella M, Dan GA, Dilaveris PE, Fauchier L, Filippatos G, Kalman JM, La Meir M, Lane DA, Lebeau JP, Lettino M, Lip GYH, Pinto FJ, Neil Thomas G, Valgimigli M, Van Gelder IC, Van Putte BP, Watkins CL. 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). European Heart Journal 2020;42:373-498.
3. Mandel JE, Hutchinson MD, Marchlinski FE. Remifentanil-midazolam sedation provides hemodynamic stability and comfort during epicardial ablation of ventricular tachycardia. J Cardiovasc Electrophysiol 2011;22:464–466.
4. Ronan KP, Gallagher TJ, George B, Hamby B. Comparison of propofol and midazolam for sedation in intensive care unit patients. Crit Care Med 1995;23:286–293.
5. Pachulski RT, Adkins DC, Mirza H. Conscious sedation with intermittent midazolam and fentanyl in electrophysiology procedures. J Interv Cardiol 2001;14:143-146.
6. Salukhe TV, Willems S, Drewitz I, Steven D, Hoffmann BA, Heitmann K, Rostock T. Propofol sedation administered by cardiologists without assisted ventilation for long cardiac interventions: an assessment of 1000 consecutive patients undergoing atrial fibrillation ablation. Europace 2012;14:325–330.
7. Arcangeli A, D'Alò C, Gaspari R. Dexmedetomidine use in general anaesthesia. Curr Drug Targets 2009;10:687-695.
8. Guerra F, Stronati G, Capucci A. Sedation in cardiac arrhythmias management. Expert Rev Cardiovasc Ther 2018;16:163-173.
9. Alaeddini J,Wood MA, Parvez B, Pathak V,Wong KA, Ellenbogen KA. Site localization and characterization of pain during radiofrequency ablation of the pulmonary veins. Pacing Clin Electrophysiol 2007;30:1210–1214.
10. Aryana A, Heist EK, D'Avila A, HolmvangG, Chevalier J, Ruskin JN, Mansour MC. Pain and anatomical locations of radiofrequency ablation as predictors of esophageal temperature rise during pulmonary vein isolation. J Cardiovasc Electrophysiol 2008;19:32–38.
11. Di Biase L, Saenz LC, Burkhardt DJ, Vacca M, Elayi CS, Barrett CD, Horton R, Bai R, Siu A, Fahmy TS, Patel D, Armaganijan L, Tung Wu C, Kai S, Keong Ching C, Phillips K, Schweikert RA, Cummings JE, Arruda M, Saliba WI, Dodig M, Natale A. Esophageal capsule endoscopy after radiofrequency catheter ablation for atrial fibrillation: documented higher risk of luminal esophageal damage with general anesthesia as compared with conscious sedation. Circ Arrhythm Electrophysiol 2009;2:108–112.
12. Barrientos-Vega R, Mar Sanchez-Soria M, Morales-Garcia C, Robas-Gomez A, Cuena-Boy R, Ayensa-Rincon A. Prolonged sedation of critically ill patients with midazolam or propofol: impact on weaning and costs. Crit Care Med 1997;25:33–40.
13. Tang R, Dong J, Zhao W, Liu X, Kang J, Long D, Yu R, Hu F, Liu X, Ma C. Unconscious sedation/analgesia with propofol versus conscious sedation with fentanyl/midazolam for catheter ablation of atrial fibrillation: a prospective, randomized study. Chin Med J (Engl) 2007;120:2036–2038.
14. Kottkamp H, Hindricks G, Eitel C, Müller K, Siedziako A, Koc J, Anastasious-Nana M, Varounis C, Arya A, Sommer P, Gaspar T, Piorkowski C, Dagres N. Deep sedation for Catheter Ablation of atrial fibrillation: A prospective study in 650 consecutive patients. J Cardiovasc Electrophysiol 2011;22:1339–1343.
15. Bhana N, Goa KL, McClellan KJ. Dexmedetomidine. Drugs 2000;59:263–720.
16. Carollo DS, Nossaman BD, Ramadhyani U. Dexmedetomidine: a review of clinical applications. Curr Opin Anaesthesiol 2008;21:457–461.
17. Hall JE, Uhrich TD, Barney JA, Arain SR, Ebert TJ. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. Anesth Analg 2000;90:699–705.
18. Tan JA, Ho KM. Use of dexmedetomidine as a sedative and analgesic agent in critically ill adult patients: a meta-analysis. Intensive Care Med 2010;36:926–939.
19. Dere K, Sucullu I, Budak ET, Yeyen S, Filiz AI, Ozkan S, Dagli G. A comparison of dexmedetomidine

versus midazolam for sedation, pain and hemodynamic control, during colonoscopy under conscious sedation. Eur J Anaesthesiol 2010;27:648–652.
20. Arain SR, Ruehlow RM, Uhrich TD, Ebert TJ. The efficacy of dexmedetomidine versus morphine for postoperative analgesia after major inpatient surgery. Anesth Analg 2004;98:153–158.
21. McCutcheon CA, Orme RM, Scott DA, Davies MJ, McGlade DP. A comparison of dexmedetomidine versus conventional therapy for sedation and hemodynamic control during carotid endarterectomy performed under regional anesthesia. Anesth Analg 2006;102:668–675.
22. Tosun Z, Akin A, Guler G, Esmaoglu A, Boyaci A. Dexmedetomidine-ketamine and propofol-ketamine combinations for anesthesia in spontaneously breathing pediatric patients undergoing cardiac catheterization. J Cardiothorac Vasc Anesth 2006;20:515–519.
23. Cho JS, Shim JK, Na S, Park I, Kwak YL. Improved sedation with dexmedetomidine-remifentanil compared with midazolam-remifentanil during catheter ablation of atrial fibrillation: A randomized, controlled trial. Europace 2014;16:1000–1006.
24. Attanasio P, Huemer M, Shokor Parwani A, Boldt LH, Mügge A, Haverkamp *W,* Wutzler A. Pain reactions during pulmonary vein isolation under deep sedation: cryothermal versus radiofrequency ablation. Pacing Clin Electrophysiol 2016;39:452–457.

Table 1: Baseline data

	Propofol - Group 1 (n = 23)	Dexmedetomidine - Group 2 (n = 26)	P value
Age (years)	65.9 ± 10.4	52.5 ± 11.1	0.346
Sex (M)	18	18	0.674
Weight (Kg)	82.7 ± 15.5	82.3 ± 17	0.941
Hypertension	19	22	0.850
Diabetes	3	6	0.365
OSAS	1	5	0.113
COPD	3	6	0.365
History of cardiac disease	6	11	0.234
Ejection fraction (%)	56.3 ± 7.9	52.5 ± 11.1	0.179
Paroxysmal AF	11	16	0.336
Persistent AF	12	10	0.509
First ablation procedure	18	19	0.425
Second ablation procedure	5	7	0.674
Electrical cardioversion	7	11	0.130

Values are mean ± standard deviation. AF: atrial fibrillation; COPD: chronic obstructive pulmonary disease; OSAS: obstructive sleep apnea syndrome.

Table 2: Hemodynamic data

	Propofol - Group 1 (n = 23)	Dexmedetomidine - Group 2 (n = 26)	P value
Baseline systolic BP (mmHg)	130 ± 21.3	125.2 ± 14.2	0.353
Baseline diastolic BP (mmHg)	77.8 ± 13.1	72.5 ± 8.5	0.095
Baseline HR (bpm)	77.3 ± 21.2	78.2 ± 20.5	0.872

	Propofol - Group 1 (n = 23)	Dexmedetomidine - Group 2 (n = 26)	P value
Baseline O_2Sat (%)	98.2 ± 1.8	98.3 ± 1.3	0.768
Min systolic BP (mmHg)	107.3 ± 15.3	108.5 ± 14.7	0.796
Min diastolic BP (mmHg)	65.2 ± 9.8	61.9 ± 9.1	0.233
Min HR (bpm)	63 ± 8.8	58.5 ± 9.7	0.091
Min O_2Sat (%)	94 ± 3.1	95.5 ± 2.3	0.075

Values are mean ± standard deviation. BP: blood pressure; HR: heart rate; O_2Sat: oxygen saturation; Min: minimum.

Table 3: Sedative drugs

	Propofol - Group 1 (n = 23)	Dexmedetomidine - Group 2 (n = 26)	P value
Propofol for EC (mg)	81.7 ± 31.2	85.7 ± 15.3	0.759
Midazolam (mg)	0.83 ± 0.1	0.78 ± 0.2	0.410
Fentanyl (mg)	0.1	0.1	1
Propofol baseline vel (ml/h)	7.4 ± 1.4		
Propofol max vel (ml/h)	9.5 ± 2.1		
Propofol min vel (ml/h)	6.9 ± 1.1		
Dexmedetomidine baseline vel (ml/h)		14.2 ± 3.0	
Dexmedetomidine max vel (ml/h)		17.8 ± 5.2	
Dexmedetomidine min vel (ml/h)		13.3 ± 3.5	

Values are mean ± standard deviation. EC: electrical cardioversion; Max: maximum; Min: minimum.

Table 4: Adverse events

	Propofol - Group 1 (n = 23)	Dexmedetomidine - Group 2 (n = 26)	P value
Persistent hypotension (systolic BP < 90 mmHg)	3/23	0/26	0.057
Respiratory depression (O_2Sat < 90%)	3/23	0/26	0.057
Bradycardia (HR < 45 bpm)	0/23	2/26	0.174

BP: blood pressure; HR: heart rate; O_2Sat: oxygen saturation.