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CLINICAL RESEARCH

Propofol sedation administered by cardiologists for patients undergoing catheter ablation for ventricular tachycardia

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Aims Propofol sedation has been shown to be safe for atrial fibrillation ablation and internal cardioverter-defibrillator implantation but its use for catheter ablation (CA) of ventricular tachycardia (VT) has yet to be evaluated. Here, we tested the hypothesis that VT ablation can be performed using propofol sedation administered by trained nurses under a cardiologist's supervision.

Methods and results Data of 205 procedures (157 patients, 1.3 procedures/patient) undergoing CA for sustained VT under propofol sedation were analysed. The primary endpoint was change of sedation and/or discontinuation of propofol sedation due to side effects and/or haemodynamic instability. Propofol cessation was necessary in 24 of 205 procedures. These procedures (Group A; $n = 24$, 11.7%) were compared with those with continued propofol sedation (Group B; $n = 181$, 88.3%). Propofol sedation was discontinued due to hypotension ($n = 22$; 10.7%), insufficient oxygenation ($n = 1$, 0.5%), or hypersalivation ($n = 1$, 0.5%). Procedures in Group A were significantly longer (210 [180–260] vs. 180 [125–220] min, $P = 0.005$), had a lower per hour propofol rate (3.0 ± 1.2 vs. 3.8 ± 1.2 mg/kg of body weight/h, $P = 0.004$), and higher cumulative dose of fentanyl administered (0.15 [0.13–0.25] vs. 0.1 [0.05–0.13] mg, $P < 0.001$), compared with patients in Group B. Five (2.4%) adverse events occurred.

Conclusion Sedation using propofol can be safely performed for VT ablation under the supervision of cardiologists. Close haemodynamic monitoring is required, especially in elderly patients and during lengthy procedures, which carrying a higher risk for systolic blood pressure decline.

Keywords Ventricular tachycardia • Catheter ablation • Sedation • Propofol • Adverse events

Introduction

Catheter ablation (CA) is an established treatment option for refractory sustained ventricular tachycardia (VT), especially in patients with structural heart disease.[1] Although reduction of symptomatic VT episodes can be expected, the procedure may be uncomfortable for patients due to extended procedure duration. In addition, it is required that patients remain in a stable position throughout the procedure to avoid the risk of cardiac perforation, unintended catheter movement, and three-dimensional (3D) map shift. This can be achieved if patients are sedated using repeat bolus administrations of benzodiazepines and opiates. This sedation regime can be challenging for long-lasting procedures (≥90 min) due to the inevitable waxing and weaning levels of sedation that may lead to inadvertent movements or respiratory failure.

As an appropriate alternative, continuous application of propofol may be considered, which in intensive care has been shown to have a better and more predictable clinical response compared with benzodiazepines and is also more cost-effective than benzodiazepines and opiates.[2–5] Recovery time and risk of respiratory depression is significantly lower using propofol compared with benzodiazepines.[6] Consequently, propofol infusion with unassisted spontaneous ventilation has been widely preferred for procedural sedation.[7–9] In a large retrospective cohort study, Salukhe *et al.*

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What's new?

- In the cohort of 205 procedures, deep sedation without assisted ventilation using propofol alone with occasional addition of fentanyl appears to be a safe sedation option for patients undergoing CA for VT.
- Sedation with propofol had to be discontinued in 11.7% of the procedures, predominantly due to hypotension.
- Trained nurses and cardiologists' can safely apply this sedation regime, even in the setting of CA of patients with structural heart disease and impaired left ventricular function.

found that the use of propofol is safe and feasible for atrial fibrillation ablation. Furthermore, Sayfo *et al*. were able to emphasize the safety of nurse administered propofol sedation for internal cardioverter-defibrillator (ICD) implantation.[10,11]

Therefore, here we aimed to assess the safety and efficacy of continuous propofol sedation for VT ablation administered, monitored, and controlled entirely by the operating cardiologist and his team, without intubation or assisted ventilation.

Methods

The study comprised consecutive VT ablation procedures from January 2007 to October 2012. All procedures were performed at the University Heart Center, Hamburg, Germany under sedation using propofol infusion (Propofol-Lipuro 2%, 20 mg/mL, B. Braun Melsungen AG, Melsungen, Germany), with fentanyl bolus administration if required (Fentanyl Janssen, 0.05 mg/mL, Janssen-Cilag GmbH, Neuss, Germany). The electrophysiological team consisted of two laboratory nurses, the operating board certified electrophysiologist and an electrophysiology fellow. Propofol was administered and monitored by catheter laboratory nurses under the direct supervision and instruction of the operating electrophysiologist. The medical staff were repeatedly trained in advanced life support. A stand-by anaesthesiology support was available during all procedures.

Patient assessment and inclusion

Patients were pre-assessed in the outpatient department prior to scheduling and consenting for elective VT ablation. Patients being admitted via the emergency room or referred for VT ablation from an outside institution were pre-assessed after admission onto the ward. All patients eligible for VT ablation with an endo- and epicardial substrate and VT episode duration of at least 30 s were screened for inclusion in the study. The ASA physical classification system was used to assess and compare the physical status of the patients.[12]

Procedures primarily planned to be performed under general anaesthesia or if mechanical or inotropic support by norepinephrine, epinephrine, or dobutamine could be expected were excluded from this analysis. Informed written consent was obtained from all patients. The study was approved by the institutional review board.

Sedation protocol and monitoring

Patients were positioned fully recumbent on the catheter laboratory table. Induction of sedation was administered with a bolus of 0.8 up to 1.2 mg/kg of body weight propofol followed by a continuous infusion starting at 3.5–4 mg/kg of body weight/h. Thereafter, the infusion rate was titrated to clinical response. Sedation depth was considered adequate when patients did not move or respond verbally. If sedation was not adequate, an additional 2–4 mg of propofol boluses were administered. Continuous propofol infusion was adjusted in steps of 0.5 mg/kg of body weight/h if required.

Oxygen was administered via a face mask starting at 5 L/min. Peripheral oxygen saturation, heart rate, electrocardiogram, and blood pressure (BP) were monitored continuously. To measure the fluid balance, all patients with structural heart disease and severe depressed left ventricular (LV) function received pre-procedural an indwelling catheter. In case of fluid imbalance, bolus wise furosemide (20–40 mg Lasix®, 20 mg/mL, Sanofi Aventis, Frankfurt/Main, Germany) was administered. Our protocol for sedation for electrophysiological procedures has been previously described in detail.[11] If tolerated, an oropharyngeal airway tube was inserted from the outset of the procedure (Teleflex, CO Westmeath, Ireland).

In ablation procedures for focal VT, BP was monitored non-invasively with a brachial cuff at 3 min intervals. In all other procedures, continuous arterial pressure monitoring was used. All measurements were documented on a specifically designed sheet every 5 min with repeat clinical assessment of sedation level and respiratory effort using visual inspection and palpation. Acoustic and optical oxygen saturation alarms were set to 90%. Boluses of intravenous fentanyl (0.05 mg) were administered prior to the first application of radiofrequency current and whenever required, at the discretion of the operating electrophysiologist.

Access, cardiac instrumentation, and procedural details

Our VT ablation approach has been previously described in detail.[13–16] Briefly, all patients were brought to the laboratory in a fasted state. Access was gained via bilateral femoral venous cannulation. Right femoral arterial access was only disclaimed in case of a suspected right ventricular focal source as underlying mechanism of the VT. In cases of focal VT, a 3.5 mm irrigated-tip ablation roving catheter (ThermoCool or ThermoCool NaviStar, Biosense-Webster, Diamond Bar, CA, USA) was used for mapping and ablation, as described previously.[16] For VTs with an expected re-entry mechanism, an right ventricular (RV) catheter (Inquiry IBI, 6 F, St. Jude Medical, St. Paul, MI, USA) was additionally positioned in the RV. If the patients had a known structural heart disease, a 3D mapping system was used to determine low voltage areas and to facilitate catheter manipulation (CARTO, Biosense-Webster Inc., Diamond Bar, CA, USA; Ensite-NavX®, St. Jude Medical, St. Paul, MN, USA). Mapping and ablation were performed as previously described.[14,17]

A single bolus of 50 IU/kg of heparin was administered after vascular access. Thereafter, additional boluses of heparin were given to maintain an activated clotting time of 250–300 s.

In patients with focal VT, the procedural endpoint consisted of abolition of ventricular premature beats and VT within a waiting period of at least 30 min, with and without provocation manoeuvres (pharmacological stimulation, reduction of the sedation) and secondary non-inducibility of VT, as described previously.[16]

In re-entrant VT, the procedure was considered successful if all VTs with cycle lengths equal to or longer than spontaneously documented or targeted VT were non-inducible using programmed stimulation with up to three extrastimuli.[17,18]

In case the clinical VT remained inducible, repeat ablation (e.g. epicardial) was planned or an intensified medical therapy was initiated.

Data analysis and study endpoints

Procedural data of all VT ablation procedures between January 2007 and October 2012 were analysed. Baseline clinical details including basic patient demography, starting BP, and oxygen saturations were recorded.

Procedural parameters, such as total procedure duration (induction of sedation to sheath removal) and overall dosage of propofol and fentanyl, were recorded. In case of propofol cessation, the cumulative midazolam dose, as well as BP and oxygen saturation, was recorded. The primary endpoint was change of sedation and/or discontinuation of propofol sedation due to side effects and/or persistent haemodynamic instability [systolic BP (SBP) <70 mmHg]. Patients with interrupted sedation were assigned to Group A and those with uninterrupted sedation to Group B. Temporary side effects of propofol sedation were considered secondary endpoints, including: (i) necessity of airway support by oro- or nasopharyngeal airway insertion if it was not established at the beginning of the procedure, chin-lift, bag-valve-mask ventilation; or (ii) hypersalivation. Further secondary endpoints included full recovery within 30 min, procedural completion and absence of any other complications.

Statistical analysis

Data are expressed as the mean value ± SD or as the median and first or third quartile if appropriate. Continuous measures were compared using the Student's *t*-test or the rank sum test and non-continuous variables using χ^2 or Fisher's exact test to a 0.05 level of significance.

Statistical analysis was performed and figures were generated with a commercially available software package (SPSS, Version 21, IBM SPSS Inc., Chicago, IL, USA).

Results

Patients and baseline physiology

Over a period of 58 months, 205 consecutive VT ablations in 157 patients (1.3 ablations/patient) were included in the analysis. Three ablation procedures were primary planned under general anaesthesia and, therefore, not included in this study. That three cases had severe heart failure complicated by recurrent VT led to pre-procedural haemodynamic instability and the need of anaesthesiology assistance with general anaesthesia and pre-procedural inotropic support.

The VTs were scar related in 135 cases, of focal origin in 51 cases (right ventricular outflow tract $n = 29$, left ventricular outflow tract $n = 2$, RV $n = 1$, LV = 19) and had a fascicular mechanism in 19 cases. One hundred and fifty-four (75%) cases were *de novo* ablation procedures, while 51 (25%) were repeat procedures either from our or an external institution.

The average age of patients was 65.6 (54.8–71.3) years and 85% were male. At screening, the median ASA score was 3 (3–4) and did not differ significantly between Groups A and B ($P = 0.43$) (*Table 1*).

Prior to sedation, the median SBP was 130 (115–150) mmHg, the median arterial pressure (MAP) was 93.3 (83.3–103.3) mmHg, and the mean oxygen saturation was 97.2 ± 2.3%. All patients were unselected and represented a typical referral cohort for VT ablation (*Table 1*). Except for a higher number of implanted ICDs in Group A (88 vs. 55%, $P = 0.003$), none of the baseline parameters differed significantly.

Procedural data and endpoints

The acute procedural endpoint was achieved in 175 of 205 procedures (85%). There was no difference in terms of success rates comparing both study groups (A: 75% vs. B: 87%; $P = 0.935$). Ablation was performed in all procedures. In general, one VT mechanism was found per case. However, in 12 (6%) cases, two mechanisms were seen. Programmed stimulation was the dominant method for VT induction (87%). A median of two (one to three) VTs were induced with a cycle length of 423 ± 99 ms. An ablation during VT was impossible in 21% of cases because of a haemodynamic intolerance. An epicardial approach was necessary in six ablations (3%). In all cases with a focal origin, a sustained VT was previously documented. However, only in 20 procedures (39%), the sustained arrhythmia was intra-procedural reproducible, while in 61% non-sustained VTs and premature ventricular beats with identical QRS-axis were the ablation target. These findings did not differ

Table 1 Patient characteristics for Group A, Group B, and the entire cohort

Patient characteristics	Entire cohort	Group A (change of sedation)	Group B (no change of sedation)	*P*
Number of procedures (*n*)	205	24	181	
Age (years)	65.6 (54.8–71.3)	69.1 (60.2–72.7)	65.1 (52.7–70.5)	0.069
Male (*n* [%])	175 [85]	19 [79]	156 [86]	0.36
Body mass index (kg/m^2)	26.2 (24.2–29.4)	26.4 (25.5–30.89)	26.1 (24.2–29.4)	0.215
LV function (%)	36 (25–55)	38 (23.8–43.5)	35 (25–55.8)	0.168
Hypertension (*n* [%])	138 [67.5]	17 [70]	121 [67]	1
Diabetes mellitus (*n* [%])	35 [17]	3 [13]	32 [18]	0.772
Chronic lung disease (*n* [%])	14 [7]	3 [13]	11 [6]	0.21
Coronary artery disease (*n* [%])	120 [59]	17 [71]	103 [57]	0.27
Dilatative cardiomyopathy (*n* [%])	29 [14]	4 [17]	25 [14]	0.751
ARVD (*n* [%])	10 [5]	0	10 [6]	0.605
ICD implanted (*n* [%])	119 [58]	21 [88]	98 [54]	0.003
ICD as primary prevention (*n* [%])	85 [42]	12 [50]	73 [40]	0.94
ASA score	3 [3–4]	3 [3–4]	3 [3–4]	0.48
VT mechanism: re-entry (*n* [%])	135 [66]	16 [67]	119 [66]	1
VT mechanism: focal (*n* [%])	51 [25]	9 [38]	42 [23]	0.21
VT mechanism: fascicular (*n* [%])	19 [9]	0	19 [11]	0.137

between Groups A and B. Non-inducibility or completely suppressed spontaneous ventricular ectopy under propofol sedation was not seen in this collective.

The median procedure time was 180 (129.3–230) min (*Table 2*). One procedure was not completed due to acute pericardial effusion with a consecutively haemodynamic deterioration requiring vasopressor support, intubation, and an unexpected transfer to an intensive care unit.

A 3D mapping system was used in 84% of all procedures ($n = 172$). CARTO was provided in the majority of cases (63%; $n = 129$), while the Ensite-NavX® system was applied less frequently (21%; $n = 43$). The 33 procedures without a mapping system were exclusively focal VT and predominantly with an origin in the right outflow tract.

Sedation and analgesia

In all 205 procedures, sedation was initiated with a bolus of 0.8–1.2 mg/kg of body weight propofol, followed by a continuous infusion starting at 3.5–4 mg/kg of body weight/h, which was then titrated according to response. The mean propofol dose for the entire cohort was 3.7 ± 1.2 mg/kg of body weight/h. Fentanyl was used for analgesia in 75% of patients and was administered in repeated boluses as required. The median cumulative fentanyl dose was 0.1 (0.05–0.15) mg. This combination regimen resulted in a median intra-procedural decrease in SBP by 25 (13.8–40) mmHg (from 130 [115–150] to 100 [93.8–110] mmHg, $P < 0.001$) and a mean drop in MAP by 20 (11.7–31.7) mmHg (from 93.3 [83.3–103.3] to 71.7 [66.7–78.3] mmHg, $P < 0.001$) and no relevant change in oxygen saturation of 0 (−1.5 to 1%) (from 98% [96–99] to 98% [96.5–99], $P = 0.628$) (*Table 2*).

Adverse effects of sedation

Primary endpoint

Adverse effects of sedation led to cessation of propofol and switch to midazolam in 11.7% of cases ($n = 24$). These cases were due to persistent hypotension in 10.7% ($n = 22$) of cases, while in one procedure (0.5%), we saw a desaturation caused by significant hypersalivation. In a further procedure, sedation was changed due to a combination of hypotension and hypersalivation (0.5%). Cumulative persistent hypotension occurred in 23 procedures.

The pre-procedural SBP (Group A: 125.6 ± 24.5 mmHg vs. Group B: 130.3 ± 21 mmHg, $P = 0.319$) and the pre-procedural MAP (Group A: 91.2 ± 15 mmHg vs. Group B: 94.5 ± 13.2 mmHg, $P = 0.253$) did not differ significantly between groups. We found no significant difference in SBP drop between groups (27.5 [17.5–42.5] vs. 25 [10–40] mmHg, $P = 0.559$) after starting the propofol sedation.

However, patients in Group A showed a significant lower SBP (95.4 ± 9.6 vs. 102.7 ± 14 mmHg, $P = 0.002$) and MAP (67.9 ± 5.4 vs. 72.7 ± 9.4 mmHg, $P = 0.001$) during the ongoing propofol sedation. Blood pressure recovered in all patients after propofol discontinuation (*Table 2*).

Internal cardioverter-defibrillator carriers were more likely to experience persistent hypotension requiring propofol cessation (Group A: $n = 21$; 88% vs. Group B: $n = 100$, 55%, $P = 0.003$). There was no significant difference with regard to other parameters such as age (Group A: 69.1 [60.2–72.7] vs. Group B: 65.1 [52.7–70.5] years, $P = 0.069$) and gender (female: 21% in Group A vs. 14% in Group B, $P = 0.36$) (*Table 1*).

Data analysis revealed a significant though weak correlation between age and mean drop in SBP ($R = 0.22$; $R^2 = 0.047$; $P = 0.002$) and an inverse weak but significant correlation between age and mean tolerated propofol infusion rate within the entire cohort ($R = 0.31$; $R^2 = 0.098$; $P < 0.0001$). The lower doses of propofol tolerated prior to the change of sedation by patients with persistent hypotension (3 ± 1.2 in Group A vs. 3.8 ± 1.2 mg/kg of body weight/h in Group B, $P = 0.004$) resulted in an increase in fentanyl dose (0.15 [0.13–0.25) vs. 0.1 [0.05–0.13] mg, $P < 0.001$). Furthermore, procedure duration was longer in patients not tolerating propofol sedation (210 [180–260] vs. 180 [125–220] min, $P = 0.005$) (*Table 2*).

Table 2 Procedural findings for Group A, Group B, and the entire cohort

Intra-procedural findings	Entire cohort	Group A (change of sedation)	Group B (no change of sedation)	*P*
Number of procedures (*n*)	205	24	181	
Procedure time (min)	180 (129.3–230)	210 (180–260)	180 (125–220)	0.005
Propofol (mg/kg of body weight/h)	3.7 ± 1.2	3.0 ± 1.2	3.8 ± 1.2	0.004
Cumulative Fentanyl dose (mg)	0.1 (0.05–0.15)	0.15 (0.13–0.25)	0.1 (0.05–0.13)	<0.001
Mean pre-procedural SBP (mmHg)	129 ± 21.4	125.6 ± 24.5	130.3 ± 21	0.319
Mean pre-procedural MAP (mmHg)	94.1 ± 13.5	91.2 ± 15	94.5 ± 13.2	0.253
Mean drop in SBP (mmHg)	25 (13.8–40)	27.5 (17.5–42.5)	25.0 (10–40)	0.559
Mean drop in MAP (mmHg)	20 (11.7–31.7)	22.5 (12.5–31.7)	20.0 (11.3–31.7)	0.633
Mean SBP on propofol (mmHg)	101.8 ± 13.7	95.4 ± 9.6	102.7 ± 14	0.002
Mean MAP on propofol (mmHg)	72.1 ± 9.1	67.9 ± 5.4	72.7 ± 9.4	0.001
Mean drop in oxygen saturation (%)	0 (−1.5–1)	0.1 (−1.4–1.2)	0 (−1.5–1)	0.72
Procedural success (*n* [%])	175 [85]	18 [75]	157 [87]	0.132
Epicardial ablation (*n* [%])	6 [3]	0	6 [3]	0.469
Hypersalivation (*n* [%])	5 [2]	2 [8]	3 [2]	0.106

Secondary endpoints

Three (1.5%) patients experienced respiratory depression resulting in sustained oxygen saturation of <90%. Two patients required reduction of propofol, transient manual chin-lift, support by oro- or nasopharyngeal airway tube insertion and bag-valve-mask ventilation before resumption of spontaneous breathing. In a third patient, endotracheal intubation, mechanical ventilation, and the attendance of an anaesthetist were required due to haemodynamically and respiratory instability caused by pericardial effusion. In this procedure, propofol was continued as general anaesthesia was required.

Hypersalivation without the necessity to change the sedation was not seen in this study.

Recovery

All patients, except the one case being intubated during the procedure, were observed in a recovery unit for 120 min before returning to the ward. A physician assessed the patients' status after 30 min. Full recovery of psychomotor and cognitive function was assessed in all patients after 60 min. All patients were routinely kept under surveillance on the main ward after ablation for at least 1 day prior to discharge.

Complications

Serious procedural complications occurred in 2.4% of procedures ($n = 5$). Serious adverse events consisting of pericardial effusions emerged in two cases. In one case, the procedure was continued. The second procedure had to be aborted due to persistent haemodynamic instability in a patient with dilatative cardiomyopathy with a severe depressed LV function of 10% and a scar-related VT re-entry (cycle length 440 ms).

One patient had to undergo resuscitation due to fast VT but was eventually successfully treated using a substrate-guided ablation approach. These recognized complications of VT ablation were not related to the sedation regimen.

One patient had a transient ischaemic attack after the procedure and one patient died 4 days after the initial successful VT ablation due to an electrical storm.

Discussion

Main findings

In our cohort of 205 procedures, deep sedation without assisted ventilation using propofol alone with occasional addition of fentanyl appears to be a safe sedation option for patients undergoing CA for VT. In the present study, sedation with propofol had to be discontinued in 11.7% of the procedures, predominantly due to hypotension. During the sedation start, the respective patients of Group A presented a trend towards a lower BP combined with an insignificant deeper drop of the BP. This combination led to a significantly lower BP in Group A during propofol sedation and the necessity to change the sedation regime.

This study shows that trained nurses and cardiologists' can safely apply this sedation regime, even in the setting of CA of patients with structural heart disease and impaired LV function. The present data report two important findings: (i) sedation with propofol for VT ablation is safe and possible without anaesthesia support and, thus, likely raises the efficacy because of independent procedure planning and performing of anaesthesia and (ii) hypotension or desaturation under propofol infusion can be managed without interrupting the procedure by cessation of propofol and change of sedation regime.

Vasopressors and ventricular tachycardia

Catheter ablation of VT remains challenging mainly due to the fact that most patients present with an impaired left ventricular function. Additionally, mapping of the critical isthmus is often required during sustained clinical VT, which further complicates the procedure with respect to haemodynamic stability. It is of utmost importance to balance between the necessity to maintain VT and thereby deteriorate the patient's haemodynamics and on the other hand to keep BP values sufficiently high to warrant peripheral oxygen supply.

In general anaesthesia, maintenance of sufficient BP values is mostly achieved by administration of relatively high doses of vasopressors, such as epinephrine or norepinephrine. These agents, however, impact the ventricular myocardial re- and depolarization properties by altering calcium and potassium handling and increasing the sympathetic drive. These pharmacological effects may accelerate VT cycle length and therefore complicate mapping during VT. Furthermore, VT of unknown clinical relevance may arise and thereby lengthen the procedure since clinically irrelevant VTs may be targeted. The principally used endpoint for CA is programmed ventricular stimulation to test for inducibility. Ventricular tachycardia inducibility using programmed stimulation after ablation has been criticized for its lack of reproducibility and for the fact that parameters such as sympathetic tone (among others) may impact the inducibility of VT. Furthermore, there is no clear link between long-term outcome and this endpoint.[19] However, VT inducibility is a commonly accepted endpoint for CA of VT.[1,18] The use of vasopressors may further complicate interpretation of stimulation results.

In addition, short episodes of VT may be required for VT mapping. Even if the patient is unable to tolerate these episodes for a longer period of time, the recovery pattern after overdrive stimulation or cardioversion informs the operator about the haemodynamic situation and stability of the patient. These recovery patterns of rhythm, haemodynamic parameters, and oxygenation may be influenced and altered by mechanical ventilation and vasopressor support. For these reasons, high doses of vasopressors in a setting of general anaesthesia during CA of VT should be avoided. One possibility substitution for vasopressors could be a sedation regime without general anaesthesia to maintain a sufficient BP, as described in this study. However, low dose inotropic support is used in many EP-labs and can be helpful to overcome an intermittent hypotension. In our study, we used cafedrinhydrochlorid/theodrenalinhydrochlorid. These agents may have an impact on the cardiac electrophysiological properties; however, in contrast to high level inotropic support during general anaesthesia, the risk–efficiency ratio seems balanced and thereby represents a viable ablation strategy.

Propofol and ventricular tachycardia ablation

Altering the electrophysiological properties when using propofol in VT ablations is a major concern. Inducibility and reducibility of the

clinical VT is of utmost importance for a successful CA. It is well known that propofol has an impact on the cardiac conduction properties but it seems that atrial and AV-node tissue is most affected.[20] Lai *et al.*[21] underlines that aspect. Propofol had no negative impact on electrophysiological characteristics and arrhythmia inducibility except in atrial tachycardias in children. Comparable data have also been reported for VT inducibility and VT ablation.[22] Furthermore, Wutzler *et al.*[22] have shown in a retrospective study that a deep sedation with propofol/midazolam is feasible and safe.

The use of propofol for VT ablation without general anaesthesia is well established and has no negative impact on the reported VT-ablation outcome.[15,22,23]

In conclusion, we are aware that propofol can alter the electrophysiological properties of the human heart. However, other drugs necessary during VT ablation (e.g. catecholamine and anaesthetics) might also impact the conduction patterns, thus, it will be necessary to identify a sedation regime that is safe, feasible and allows successful ablation.

Risk of ventricular tachycardia catheter ablation

One of the main objectives of the present study was to evaluate whether the risk of periprocedural complications may be increased using analgosedation with propofol, administered and supervised by specially trained cardiologist, as has already been shown for CA of atrial fibrillation and device implantation.[10,11] However, in VT ablation, higher complication rates than in other procedures are accepted given the complexity of the procedure and the higher morbidity of VT patients.[24] In an analysis by Sacher *et al.*, the number of serious procedure-related complications was 3.7% (life-threatening events) and 4% (not life-threatening events), resulting in an overall event rate of 7.7% within 48 h after ablation. Overall, even severe complications have been lower in our study cohort, potentially indicating that the use of propofol may reduce the risk of such procedures by omitting tracheal intubation and mandatory vasopressor administration in a general anaesthesia setting.

Limitation

The study was observational by design and procedures were not randomized against a comparison group with an alternative form of sedation. However, the presented data from a fairly large VT ablation cohort indicate that the use of propofol in this setting is safe and feasible. A comparison study with a randomized controlled design should now be undertaken. Our procedures were limited to VT ablation and, therefore, we cannot assume how patients would tolerate propofol during longer cardiac interventions.

Conclusion

Sedation using propofol can be safely performed by a cardiologist and nurses specially trained for VT ablation procedures. However, close haemodynamic monitoring is required, especially in elderly patients and during lengthy procedures carrying a higher risk for SBP decline and an associated higher incidence of propofol interruption.

Conflict of interest: none declared.

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